



Standard Treatment Protocol



**Public Health Department
Government of Maharashtra**



STANDARD TREATMENT PROTOCOL

Public Health Department
Government of Maharashtra





सत्यमेव जयते

Dr. Deepak Sawant

Minister, Public Health & Family Welfare
Government of Maharashtra



Message

Maharashtra has made progress in the health care delivery which is evident from decreasing trend of Maternal and Infant Mortality over a period of years. Despite declining mortality and changing morbidity patterns, we still face the challenges of the widening disparities between various socio-economic classes in terms of access to quality health care services. Traditional infectious diseases still contribute to a heavy burden and at the same time, there has been a rise in the occurrence of chronic and newer emerging & re-emerging diseases due to the changing lifestyle, environmental changes and higher life expectancy.

With advent of newer drugs and technology, there has been a dramatic change in the outlook for prevention and management of diseases. Also, various newer Guidelines are issued by Government of India and International organisations. To keep the health care provider updated of these changes, a compendium of the treatment protocols to serve as a ready reference and establishing standard of quality care was the need of the hour. The Standard Treatment Protocols is one such important step of the Maharashtra towards attaining the Sustainable Development Goal no.3 by ensuring access to quality essential health-care services by empowering Health care providers.

I appreciate the amount of efforts put in by Dr. Archana Patil, Additional Director and her team for developing Standard Treatment Protocols book. I hope that the book would prove a valuable guide to health care providers in their everyday decision making.

(Dr. Deepak Sawant)



**MINISTER OF STATE FOR
PUBLIC HEALTH, LABOUR
TRANSPORT AND STATE EXCISE**

GOVERNMENT OF MAHARASHTRA
Mantralaya, Mumbai 400 032
www.maharashtra.gov.in

Date :




Message

There has been an increase in the expenditure on healthcare, especially in view of the growing rate of urbanisation and rise in the number of lifestyle diseases. Within the limited financial allocation in the Public Health Sector, health care services are needed to cater to majority of the population. This demands rational and efficient utilisation of the limited resources.

Standard treatment protocols aim to ensure efficient utilisation of essential drugs to treat majority of population in a standardised manner. Thus, more people can be treated, limiting the wastage of resources over unstandardized treatment modalities. It also ensures that treatment quality & standards are followed. They serve as a guide for health care providers in deciding the best possible approaches for diagnosis, management and prevention of the health issues.

I greatly appreciate the work done by the Public Health Department for coming up with the Standard Treatment Protocols for common health issues experienced by the people in the State. I hope that they help the care providers in rational use of treatment modalities and lay the standards for quality health services.


(Shri. Vijay Deshmukh)



सत्यमेव जयते

डॉ. विजय सतबीर सिंघ
अप्पर मुख्य सचिव

Dr. Vijay Satbir Singh

Additional Chief Secretary



महाराष्ट्र शासन
सार्वजनिक आरोग्य विभाग
मंत्रालय
कॉम्प्लेक्स बिल्डींग, गोकुळदास तेजपाल रुग्णालय कंपाऊंड,
१०वा मजला, मुंबई ४०० ००१
दुरध्वनी : कार्यालय - ०२२-२२६१७३८८ फॅक्स : २२६१७९९९
GOVERNMENT OF MAHARASHTRA
Public Health Department
Mantralaya

Complex Building, Gokuldas Tejpal Hospital Compound,
10th Floor, Mumbai - 400 001.
Phone : 022-22617388 Fax : 022-22617999



Message

Maharashtra ranks first among all States as per the IIMA report (2016) with respect to the inputs made available or created in the state. It is determined to sustain quality of healthcare services provided to all. Quality Health Care services imply that the standards of Healthcare delivery are maintained uniformly from the tertiary care unit to the primary health care unit. This will ensure that every patient gets safe affordable health care taking into consideration the "Time to Care approach".

Standard Treatment Protocols and Essential Drug List are important to ensure uniformity of the quality of care at all levels. This book will help the health care providers in determining the best possible choice for the patients. Vast quantum of literature is available in the world and it is difficult for accessing the same given the paucity of time. Through the book an earnest effort has been made to make concise, ready to use information available to the health care providers at their fingertips along with reference material for further reading.

I commend the work done by all the contributors who have given their valuable time in producing the book. I expect that all the Health Care providers in the department would use the book in maintaining the quality of services for the betterment of the people.

(Dr. Vijay Satbir Singh)





सत्यमेव जयते

Government of Maharashtra
Directorate of Health Services
Public Health Department



Dr. Satish Pawar

D.P.H., M.D., PH.D.

Director, Health Services

“Arogya Bhavan”, 8th Floor,

St. George’s Hospital Compound,

P.D’Mello Road, CST, Mumbai - 400 001



☎ : 022-22621006, 22620292

📠 : 022-22620234

✉ : dhs_2005@rediffmail.com

Website : <https://arogya.maharashtra.gov.in>

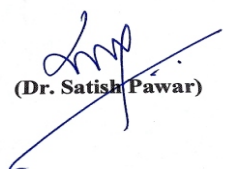


Message

Worldwide more than 50% of all medicines are prescribed, dispensed or sold inappropriately, while 50% of patients fail to take them correctly. Moreover, about one-third of the world's population lacks access to essential medicines. In Public Health Care system, which caters to the majority of the population and given the constraint of availability of drugs and commodities, rational use of medicine in treatment and management of illness has attained even more significance. Moreover, the quality of the treatment needs to be standardised for all the levels of health care. The health care provider must have updated knowledge about the latest developments in the treatment modalities and ascertain treatment regimen suitable for the disease.

This Standard Treatment Protocols book is meant for medical officers and health care providers from different levels in the public health care system, to refer information about the condition from a concise source. It is supposed to be a guide and aid for making decisions on diagnosis and management of conditions for majority of the population. The management of individual cases would ultimately depend on clinical judgement and skills of the health care provider.

I would like to express my appreciation to all those who have participated in various workshops, meetings and video conferences despite their demanding schedule for producing a valuable document. I am positive that this book will be useful to all Health care providers working in my department and ultimately contribute to attain positive health for the patients.


(Dr. Satish Pawar)

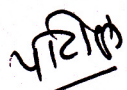
Foreword

Sustainable Development Goal 3 aims to ensure healthy lives and promote well-being for all ages. The strategic approach currently adopted is maintaining continuum of care throughout the various levels of Healthcare and over the life cycle of the individual. The Public Health Department has been working diligently with the goal of provision of effective and affordable quality health care which is easily accessible to all age groups. The work done is reflected in the SRS reports and the IIMA report for States. Still sustained and vigorous efforts are needed to reach the last mile.

To better utilise our resource pool in terms of drugs, laboratory services and human resources while maintaining the quality of care, there was a need of a standardised system. There are various guidelines and protocols available for all the diseases. To keep in pace with the recent changes in treatment regimens and technological progress, it was necessary to have Standard Treatment Protocols (STP) for all diseases of public health importance in the State.

With the extensive efforts from specialists working in the Public Health Department, Professors from Government, Private Medical Colleges as well as under the guidance of Dr. Prakash Doke, Ex-Director of Health Services, Ex-Bureau Chiefs and department heads, the Standard Treatment Protocols came into fruition. Over a period of 2 years, various deliberations were held in the form of meetings, workshops and video conferences to add the latest changes in the field and include references for further reading of the Health Care provider. I would like to highlight the guidance from our former Principal Secretary, Mrs. Sujata Saunik and our Additional Chief Secretary, Dr. Vijay Satbir Singh who shared their vision for developing this STP.

I sincerely hope that the Standard Treatment Protocols would be helpful for effective decision making and in day to day working of the Healthcare providers.


(Dr. Archana Patil)
Additional Director,
SFWB, Pune.

STP

Team No.1 Gen. Medicine, ICU, CCU, ICCU, ARV/ASV

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. N. J. Rathod (Team Leader)	Ex. Addl. DHS	Mumbai	9322890107
2	Dr. A.L. Kakrani	Prof. Medicine	Dr. D. Y. Patil Medical College, Pimpri	9823972424
3	Dr. Umakant Shetkar	Prof. Medicine	Smt. Kashibai Navale Medical College, Pune	9960011820
4	Dr. Mrs. Swati C. Aundhekar	Prof. Medicine	KIMS, Karad	9766535255
5	Dr. Priti Dave	Prof. Dept. of Medicine	Bharati Vidyapeeth Medical College, Pune	9822790490
6	Dr. (Col) P.K. Sharma	Asso. Prof. MD (Med)	AFMC, Pune	9823071758
7	Dr. Mrs. Priti P. Dhande	Asso. Prof. (Pharmacology)	Bharati Vidyapeeth Medical College, Pune	9922426840
8	Dr. Sujata Abhay Jadhav	Asso. Prof. M.D. Pharmacology	KIMS, Karad	9423262871
9	Dr. Madhulika Mahashabde	Asso. Prof. Dept. of Medicine	Dr. D. Y. Patil Medical College, Pimpri	9850015831
10	Dr. Kiran K. Khalate	Class I, Physician	D.H. Ratnagiri	9850493260
11	Dr. Shyamsundar H. Nikam	Physician, Medical Superintendent	RRH Amravati	9423124973
12	Dr. Sukrut V. Purandare	Lecturer, Medicine	Dinanath Mangeshkar Hospital, Pune	9881568748
13	Dr. Shingade Madhuri M.	Physician	T.C.M. Hospital, Pune	9763825105
14	Dr. Khalate	Physician	Civil Hospital, Ratnagiri	9850493260
15	Dr. Nikam	Medical Superintendent	VSSH, Amravati	9423124973
16	Dr. Miss Rande	Physician	District Hospital, Pune	

Team No.2 National Health Programme, NRHM

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. Dodwad	Ex. Addl.DHS	Pune	9421014106
2	Dr. Vandana A. Kakrani (Team Leader)	Prof. Community Medicine	Dr. D. Y. Patil Medical College, Pimpri	9373324064
3	Dr. Prasad D. Pore	Prof. Community Medicine	Bharati Vidyapeeth Medical College, Pune	9921073540
4	Dr. Abhay Mane	Professor MD (PSM)	Smt. Kashibai Navale Medical College, Pune	8975008663
5	Dr. Uddhav T. Kumbhar	Asst. Prof. MD (PSM)	KIMS, Karad	8983374099
6	Dr. Pavan Salve	Addl. MOH	PCMC	9922501584
7	Dr. Rahul D. Anerao	MO PHC, MD (PSM)	PHC Hasegaon, Latur	9881337338
8	Dr. Mangesh A Patil	MO, PHC MD (PSM)	Jalgaon	9403313482
9	Dr. Ganesh S. Lokhande	THO, MD (PSM)	THO Patoda, Beed	9922373120

Sr. No.	Name	Post	Place of post	Mobile No.
10	Dr. R.G. Chaudhari	MO PHC, MD (PSM)	Nashik	9403505910
11	Dr. Sanjiv Kamble	Jt. Director (TB & Leprosy)	TB & Lep. Office Pune	9869972056
12	Dr. Madhukar Pawar	Dy. Director (TB & Leprosy)	TB & Lep. Office Pune	9404194042
13	Dr. Pradeep Aawte	SSO	IDSP Pune	9423337556

Team No.3 General Surgery, Ortho., Burn & Trauma

General Surgery

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. Mrunal N. Ketkar	Prof. Surgery	Bharati Vidyapeeth Medical College, Pune	9623019743
2	Dr. D.P. Pande (Team Leader)	Prof. Surgery	Smt. Kashibai Navale Medical College, Pune	9422320164
3	Dr. Nitin Bilolikar	OSD to Hon. Health Minister	Mantralaya, Mumbai	9422189337
4	Dr. N.L. Vyas	Prof. Surgery	Navi Mumbai	9322956914
5	Dr. S.R. Kulkarni	Prof. Surgery	KIMS Karad	9423606439
6	Dr. K. Sahoo	Prof. Radiologist	KIMS Karad	9970173439
7	Dr. Sanjay Padale	Surgeon	YCMH Pimpri	9922501324
8	Dr. Bolde A.N.	Surgeon	CS Beed	9822195062
9	Dr. B.H. Shinde	Surgeon	DH Pune	9420202731
10	Dr. Ajit Gawali	Surgeon	CS Raigad	9422096363
11	Dr. Ashish Chitharanjan	Surgeon	Pune	9822931328
12	Dr. Shinde	Med. Superintendent	District Hospital, Pune	9420202731
13	Dr. Rathod	Addl. Civil Surgeon	Civil Hospital, Thane	9422877733
14	Dr. Sonar	Med. Superintendent	VSSH, Amravati	9422754136

Orthopedic

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. M.S. Diggikar	Jt. Dir (NVBDCP)	Pune	9325398011
2	Dr. Hemant Parekh	Asso. Prof. Ortho.	Smt. Kashibai Navale Medical College, Pune	9011020901
3	Dr. Chintamani Latkar	Asso. Prof. Ortho.	Bharati Vidyapeeth Medical College, Pune	9823028036
4	Dr. Piyush P. Jakkal	Ortho. Surgeon, C.I.I	D.H. Bhandara	8888865848

Sr. No.	Name	Post	Place of post	Mobile No.
5	Dr. Ranjit Deshmukh	Consultant	Dinanath Mangeshkar Hospital, Pune	9822058954
6	Dr. P.K. Dake	Med. Superintendent	Ortho. Hosp. Parbhani	9823529336
7	Dr. Anupam Hivlekar	Class I, Ortho Surgeon	D.H. Wardha	9422841044
8	Dr. Ravindra Gunaki (Team Leader)	Orthopedician	KIMS Karad	9422039510
9	Dr. Saurabh Kale	Orthopedician	YCMH Pimpri	9960902842
11	Dr. A. K. Bnajdr	Orthopedician	YCMH	9552595521
12	Dr. Sanjog Kadam	Orthopedician	DH Satara	9422606782
13	Dr. Santpure	Orthopedician	DH Pune	8308667603

Team No.4 Pediatric & Neonatology

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. Dilip N. Patil (Team Leader)	Ex. Dy. Director	Aurangabad	9421407574
2	Dr. C. D. Aundhekar	Prof. Paed.	KIMS, Karad	9960697814
3	Dr. Maya Borle	Assoc. Prof.	Pune	9890192458
4	Dr. Kavita Srivastava	Assoc. Prof. (Paed.)	Bharati Vidyapeeth Medical College, Pune	9850825791
5	Dr. M. V. Deshpande	DCH	YCMH, Pune	9922501322
6	Dr. Rohini Nagarkar	Paediatrician	YCMH, Pune	9922501571
7	Dr. Sagar Khedu	Paediatrician	D.H. Alibag	9224367253 8379006796
8	Damale Kalpana T.	Dietician	Osmanabad	9423073368
9	Dr. Lohare B.S.	M.O. Pediatrician	RH Panvel	8655007705
10	Dr. Sanjay Jaiswal	Paediatrician	Daga Hospital, Nagpur	9422150677
11	Dr. Anupama Nadkarni	Paediatrician	Dinanath Mangeshkar Hospital, Pune	9890599646
12	Dr. Ramdas N. Khune	Med. Superintendent	SDH Pen, Dist. Raigad	9271705557
13	Dr. Manoj Patil	Paediatrician	Dr. D. Y. Patil Medical College, Pimpri	9922417017
14	Dr. Tahsim F. Khan	Paediatrician Class-I	Maternity Hosp. U'nagar-4	9325329874
15	Dr. Kadam	Pediatrician	Civil Hospital, Hingoli	8007001525
16	Dr. Purushottam Darshne	Med. Superintendent	Rural Hospital Shikrapur, Pune	9823307703
17	Dr. Pankaj Gajre	Pediatrician	Civil Hospital, Nashik	9822965458
18	Dr. Chandrakala Jaiswal	Pediatrician	SFWB Pune	9822496233
19	Dr. Navale	Pediatrician	SFWB Pune	9326874228
20	Dr. Baviskar	ADHS	SFWB Pune	7738362899

Team No.5 Ophthalmic

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. Varsha N Kulkarni	Prof. Ophthalmology	Bharati Vidyapeeth Medical College, Pune	9423009019
2	Dr. Mrs. Smita D Javdekar	Asso. Prof. of Ophthalmology	KIMS, Karad	9422613592
3	Dr. Pramod M. Sonune	Civil Surgeon	D.H, Chandrapur	9561962697
4	Dr. Satish Kumar Sriwastav (Team Leader)	Ophthalmologist	Smt. Kashibai Navale Medical College, Pune	9881067666
5	Dr. Madhav J. Bhat	DMH Consultant	Dinanath Mangeshkar Hospital, Pune	9822323530
6	Dr. S.B. Andhare	Ophthalmic Surgeon Class-I	G.H, Wardha	9823248623
7	Dr. P. Jogewar	Ophthalmic Surgeon Class-I	DAGA Hospital Nagpur	9422131577

ENT

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. G. D. Mahajan	Prof. ENT	Dr. D. Y. Patil Medical College, Pimpri	9850038510
2	Dr. Havle Abhay D.	Prof. ENT	KIMS, Karad	9423263164
3	Dr. Sanjana Nemade	Asso. Prof. ENT	Pune	9923730525
4	Dr. Sarika Dhoot	Asst. Prof. ENT	Bharati Vidyapeeth Medical College, Pune	9326349006
5	Dr. N. B. Patel	M.S. (ENT)	RMO (OR) DH Beed	9370066114
6	Dr. Manoj Deshmukh	M.S. (ENT)	YCMH Pimpri	9922501564
7	Dr. S. M. Mahalle	Class-I, ENT Surgeon	C.H. Amravati	9423124812
8	Dr. Kamble A.G. (Team Leader)	MO ENT	C.H. Pune	9823174647
9	Dr. Vikram Oak	DMH Consultant	Dinanath Mangeshkar Hospital, Pune	9822031070
10	Dr. Yadav	Physician	VSS Hospital, Amravati	8308667603
11	Dr. Kamble	Physician	Civil Hospital Thane	9823776442

Team No.6 Obs/Gyn

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. G.S. Shekhawat	Prof. (Obst. &Gyn)	Smt. Kashibai Navale Medical College, Pune	9372897090
2	Dr. Aparna N. Shrotri (Team Leader)	(Retd.) Prof. OBGY. Consultant	BJMC, Pune	9422513482 020-25433233
3	Dr. Suchita Dabhadkar	Asst. Prof.OBGY	Bharati Vidyapeeth Medical College, Pune	9422304399
4	Dr. Dileep P. Javadekar	Asst. Prof MD (Gyn).	KIMS, Karad	9423871993
5	Dr. Mangal Santosh Supe	MD OBGY	YCMH Pimpri Pune	9604167167
6	Dr. Shubhang Mhasde	MBBS DGO M.O.	Govt. Maternity Hosp. & Dispensary, Ulhasnagar	7303170483
7	Dr. Vaishali P. Khedikar	Med. Superintendent	DAGA Hosp., Nagpur	9096724121
8	Dr. Ratna Raokhande	Obst. &Gyn	ADHS Thane	8080001530 9422030810
9	Dr. Arundhati Kanade	OBGYN Consultant	Dinanath Mangeshkar Hospital, Pune	9822614612
10	Dr. Miss Dikshit	Obst. &Gyn	District Hospital, Pune	9503486712
11	Dr. S.M. Sonawne	Obst. &Gyn	District Hospital, Satara	9422029973
12	Dr. D. Kangule	ADHS	SFWB, Pune	7875185846
13	Dr. M.V. Karnataki	UNICEF Consultant	SFWB, Pune	9324341580

Team No.7 Anesthesiology

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. Vithal K. Dhulkhed	Prof. Anaesthesia	KIMS, Karad	7588953396
2	Dr. Noopur Singh	Prof. Anaesthesia	Smt. Kashibai Navale Medical College, Pune	9011070600
3	Dr. Pooja N. Bhosle	Asso. Prof. Anesthesia	Bharati Vidyapeeth Medical College, Pune	9860835279
4	Dr. B. Nagaonkar	ADHS	RGJAY Mumbai	7738574849
5	Dr. Kore B.S.	Civil Surgeon	Latur	9011430402
6	Dr. Pradeep M. Kulkarni	M.D. Anaesthesia	Women Hosp. Jalna	9421448716
7	Dr. Patil Makarand P (Team Leader)	Anaesthesia	DH Alibag-Raigad	9822509077
8	Dr. Dhangekar	Anaesthesia	DH Pune	9822616930
9	Dr. Bhabhul Gaonkar	Anaesthesia	DH Thane	9892007704

Team No.8 Dental, Chest & TB

Sr. No.	Name	Post	Place of post	Mobile No.
	Dental			
1	Dr. Kharat (Team Leader)	Dental Surgeon	D.H. Beed	9423450077
2	Dr. Supriya Mahajan	Dental Surgeon	UH-3 Thane	8108715475
3	Dr. Dinesh Ashok Dhole	Dental Surgeon	D.H. Nashik	9923551519
	Chest & TB			
1	Dr. Ramkrishnan V. Ghubde (Team Leader)	Asso. Professor	Smt. Kashibai Navale Medical College, Pune	9822462536
2	Dr. Ramesh L. Pawar	M.O.	RRH Nashik	9420335958
3	Dr. Anita Kharat	Dental Surgeon	D.H. Jalna	9423450077
4	Dr. Zapkar	Dental Surgeon	D.H. Thane	9029083693
5	Dr. Shegavkar	Dental Surgeon	RH, Igatpuri, Nashik	9890317834

Team No.9 Psychiatry

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. Sharmishta S. Deshpande	Prof. (Psychiatry)	Smt. Kashibai Navale Medical College, Pune	9890381144
2	Dr. Jyoti Shetty	Prof. (Psychiatry)	Bharati Vidyapeeth Medical College, Pune	9822326019
3	Dr. Khandekar (Team Leader)	NRHM Psychiatrist	Thane	9821351712
4	Dr. Amit Nagarkar	Class I, Psychiatrist	Regional Mental Hospital, Nagpur	9579591936
5	Dr. Hemant Sonanis	Psychiatrist	D.H. Nashik	9922069966
6	Dr. Amol Gulhane	Psychiatrist	GH Amravati	9881424080
7	Dr. Divekar	Psychiatrist	D.H. Thane	9619388012
8	Dr. M. Bahale	Psychiatrist	D.H. Thane	9850411911
9	Dr. Shirsath	Med. Superintendent	D.H. Thane	9096726700

Team No.10 Nephrology, Haematology & Pathology

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. R. C. Nimbargi	Prof. & Head Pathology	Bharati Vidyapeeth Medical College, Pune	9422069392
2	Dr. Sunil V. Jagtap	Asso. Prof. Patho.	KIMS Karad	9960628672
3	Dr. Sunita M. Bhatawadekar	Assoc. Prof. Microbiology	Bharati Vidyapeeth Medical College, Pune	9922913622
4	Dr. Satish V. Shimpi	Pathologist	Nashik	9890304750
5	Dr. P. V. Puri	M.O.	Nashik	9850632960
6	Dr. Deepak N. Gosavi	Pathology	D.H. Alibag	9881671533
7	Dr. S.K. Pawar	Microbiologist	KIMS, Karad	9423034094
8	Dr. V.V. Jadhav (Team Leader)	Pathologist	G.H. Amravati	9763599173
9	Dr. Bhamre	Pathologist	DH, Nandurbar	9823100333
10	Dr. M.M. Kulkarni	Pathologist	DH, Pune	9623690256
11	Dr. Padmakar Kadam	Pathologist	DH, Satara	9822637176

Team No.11 Dermatology

Sr. No.	Name	Post	Place of post	Mobile No
1	Dr. N.D. Chaudhari	Prof. Dermatology	Dr. D. Y. Patil Medical College, Pimpri	9422331015
2	Dr. N.R. Gokhale	Prof. Dermatology	Smt. Kashibai Navale Medical College, Pune	9823154022
3	Dr. S.T. Vandav(Team Leader)	Dermatologist	D.H. Sindhudurg	9422052644
4	Dr. A.D. Ingale	MO, Class-I. Skin	DH Pune	9850556079
5	Dr. P.P. Ghushe	Prof. Dermatology	DH, Thane	9930661664
6	Dr. Vaishali Giri	Prof. Dermatology	DH, Nashik	8149904659

Special Thanks to Hon. Dean Seth G.S. Medical College and KEM Hospital Mumbai, Dr. Shinde (Prof and Head Department of PSM) and all Professors and their colleagues of the Medical College for their contribution for Standard Treatment Protocol Book. Without their contribution, it would have been not possible to develop this book.

Editors

Sr. No.	Name	Post	Place of post	Mobile No.
1	Dr. Archana Patil (Editor in chief)	Addl Director	SFWB, Pune	9869394115
2	Dr. Tanaji Mane	DDHS	SBHIVS	9422746567
3	Dr Umesh Shirodkar	ADHS	DHS, Mumbai	9892726618
4	Dr Madhusudan Karnataki	UNICEF Consultant	SFWB, Pune	9324341580
5	Dr Chandrakala Jaiswal	UNICEF Consultant	SFWB, Pune	9822496233
6	Dr Ravindra Bagal	UNICEF Consultant	SFWB, Pune	7720009948
7	Dr Aparna Deshpande	UNICEF Consultant	SFWB, Pune	9373089938
8	Dr Pushkar Deshmukh	UNICEF Consultant	Jalgaon	9028198508
9	Dr Neha Wagh	UNICEF Consultant	Aurangabad	9673527421
10	Dr Vinay Koparde	UNICEF Consultant	Nanded	9168849555
11	Dr Vaibhav Mahatme	UNICEF Consultant	Beed	9096309076
12	Dr Prashant Hingankar	UNICEF Consultant	Nandurbar	8879597487

MASTER INDEX

Sr. No.	Contents	Page No.
	Preface	i-x
1	Medicine	1-165
2	Pediatrics	166-269
3	Obstetrics and Gynaecology	270-335
4	Dermatology	336-362
5	Psychiatry	363-391
6	ENT	392-438
7	Ophthalmology	439-480
8	Surgery	481-529
9	Orthopaedics	530-571
10	Anaesthesiology	572-598
11	Pathology	599-626
12	Dentistry	627-661

Abbreviations Used

Sr. No.	Abbreviation	Forms
1	ASHA	Accredited Social Health Activist
2	AFB	Acid-Fast Bacillus
3	AFP	Acute Flaccid Paralysis
4	AIDS	Acquired Immuno Deficiency Syndrome
5	AMTSL	Active Management of Third Stage of Labour
6	ANC	Ante Natal Care
7	AOM	Acute Otitis Media
8	APH	Ante Partum Haemorrhage
9	ARDS	Acute Respiratory Distress Syndrome
10	ARF	Acute Renal Failure
11	ARM	Artificial Rupture of Membranes
12	ART	Anti-Retroviral Treatment
13	ARV	Anti-Rabies Vaccine
14	ASV	Anti-Snake Venom
15	ATT/AKT	Anti-Tuberculosis/ Anti-Koch Treatment
16	BERA	Brainstem Evoked Response Audiometry
17	BMI	Body Mass Index
18	BSL	Blood Sugar Level
19	BT	Bleeding time
20	BUN	Blood Urea Nitrogen
21	CAP	Community Acquired Pneumonia
22	CBC	Complete Blood Count
23	CCF	Congestive Cardiac Failure
24	CIN	Cervical Intraepithelial Neoplasia
25	CMV	Cyto Megalo Virus
26	COPD	Chronic Obstructive Pulmonary Disease
27	CPAP	Continuous Positive Airway Pressure
28	CPD	Cephalo-Pelvic Disproportion
29	CPK	Creatinine Phospho Kinase
30	CRF	Chronic Renal Failure
31	CRP	C-Reactive Protein
32	CRT	Capillary Refill Time
33	CS	Caesarean Section
34	CSOM	Chronic Suppurative Otitis Media
35	CT	Clotting time
36	CT Scan	Computerized Tomography
37	CTEV	Congenital Talipes Equino Varus
38	CVA	Cerebro Vascular Accident
39	D&C	Dilatation and Curettage

Sr. No.	Abbreviation	Forms
40	DHF	Dengue Haemorrhagic Fever
41	DIC	Disseminated Intravascular Coagulation
42	DMARDs	Disease Modifying Anti Rheumatic Drugs
43	DNS	Dextrose Normal Saline
44	DOTS	Directly Observed Treatment, Short Course
45	DUB	Dysfunctional Uterine Bleeding
46	EBM	Expressed Breast Milk
47	EBV	Epstein Barr Virus
48	ECG	Electro Cardio Gram
49	EDD	Expected Date of Delivery
50	EEG	Electro Encephalo Graphy
51	ELISA	Enzyme Linked Immuno-Assay
52	ENG	Electro Nystagmo Graphy
53	ESR	Erythrocyte Sedimentation Rate
54	FFP	Fresh Frozen Plasma
55	FHR	Fetal Heart Rate
56	FHS	Fetal Heart Sound
57	F-IMNCI	Faculty based Integrated Management of Newborn Child Illness
58	FNAC	Fine Needle Aspiration Cytology
59	FRU	First Referral Unit
60	GDM	Gestational Diabetes Mellitus
61	GERD	Gastro Esophageal Reflux Disease
62	GFR	Glomerular Filtration Rate
63	GIT	Gastro Intestinal Tract
64	HA	Health Assistant
65	HCC	Hepato Cellular Cancer
66	HCG	Human Chorionic Gonadotropins
67	HDCV	Human Diploid Cell Vaccine
68	HDL	High Density Lipids
69	HDN	Haemolytic Disease of Newborn
70	HE	Hepatic Encephalopathy
71	HIV	Human Immune Deficiency Virus
72	HPV	Human Papilloma Virus
73	HSG	Hystero Salpingo Graphy
74	HSV	Herpes Simplex Virus
75	HTN	Hypertension
76	IBS	Irritable Bowel syndrome
77	ICCU	Intensive Coronary Care Unit
78	ICTC	Integrated Counselling and Testing Centre
79	ICU	Intensive Care Unit
80	IDA	Iron Deficiency Anemia

Sr. No.	Abbreviation	Forms
81	IDDM	Insulin Dependent Diabetes Mellitus
82	IFA	Iron Folic Acid
83	IG	Immunoglobulin
84	IHBD	Intra Hepatic Biliary Duct
85	IHD	Ischemic Heart Disease
86	IMNCI	Integrated Management of Newborn and Childhood Illness
87	IPPR	Intermittent Positive Pressure Respiration
88	IUD	Intra Uterine Death
89	IUGR	Intra Uterine Growth Retardation
90	IUI	Intra Uterine Insemination
91	IVC	Inferior Vena Cava
92	IVF	In Vitro Fertilization
93	JVP	Jugular Venus Pressure
94	KFT	Kidney Function Test
95	KMC	Kangaroo Mother Care
96	LBW	Low Birth Weight
97	LDL	Low Density Lipids
98	LFT	Liver Function Test
99	LMP	Last Menstrual Period
100	LRTI	Lower Respiratory Tract Infections
101	LSCS	Lower Segment Cesarean Section
102	LVH	Left Ventricular Hypertrophy
103	MAM	Moderate Acute Malnutrition
104	MCHC	Mean Corpuscular Hemoglobin Concentration
105	MCV	Mean Corpuscular Volume
106	MDR-TB	Multi Drug Resistant Tuberculosis
107	MI	Myocardial Infraction
108	MMR Vaccine	Measles, Mumps, Rubella Vaccine
109	MOHFW	Ministry of Health and Family Welfare
110	MPW	Multi-Purpose Worker
111	MRI	Magnetic Resonance Imaging
112	MTP	Medical Termination of Pregnancy
113	MVA	Manual Vacuum Aspiration
114	NACP	National Aids Control Programme
115	NBCC	New Born Care Corner
116	NBSU	New Born Stabilization Unit
117	NGO	Non-Government Organization
118	NICU	Neonatal Intensive Care Unit
119	NSAID	Non-Steroidal Anti Inflammatory Drug
120	OAE	Oto Acoustic Emission
121	OCPs	Oral Contraceptive Pills

Sr. No.	Abbreviation	Forms
122	ORS	Oral Rehydration Salt
123	PCR	Polymerase Chain Reaction
124	PCV	Packed Cell Volume
125	PEEP	Positive End Expiratory Pressure
126	PEF	Peak Expiratory Flow
127	PEFR	Peak Expiratory Flow Rate
128	PEP	Post Exposure Prophylaxis
129	PET Scan	Positron Emission Tomography Scan
130	PID	Pelvic Inflammatory disease
131	PID	Prolapse Intervertebral disc
132	PMN	Poly Morpho-Nuclear
133	PNS	Para Nasal Sinuses
134	PPD	Purified Protein Derivative
135	PPH	Post-Partum Haemorrhage
136	PROM	Premature Rupture of Membranes
137	PT	Prothrombin Time
138	PUD	Peptic Ulcer Disease
139	RDT	Radical Drug Treatment
140	RF	Renal Failure
141	RL	Ringer Lactate
142	RNTCP	Revised National Tuberculosis Control Programme
143	RVH	Right Ventricular Hypertrophy
144	SAH	Sub Arachnoid Hemorrhage
145	SAM	Severe Acute Malnutrition
146	SGA	Small for Gestational Age
147	SLR Test	Straight Leg Raising Test
148	SNCU	Special New Born Care Unit
149	STD	Sexually Transmitted Disease
150	STI	Sexually Transmitted Infections
151	SVC	Superior Vena Cava
152	TIA	Transient Ischemic Attack
153	TSH	Thyroid Stimulating Hormone
154	UC	Ulcerative Colitis
155	URTI	Upper Respiratory Tract Infections
156	USG	Ultrasonography
157	VIA	Visual Inspection of Cervix with Acetic Acid
158	VLBW	Very Low Birth Weight
159	WHO	World Health Organization
160	XDR-TB	Extensive Drug Resistant Tuberculosis



Preface



I. HOW TO USE THE BOOK

The Standard Treatment Protocol booklet was developed to serve as a comprehensive index of protocols for the common conditions, syndromes and ailments. Each condition is individually dealt with clearly presented information on definitions, classification, symptoms, signs, investigation and advice on diagnosis and treatment. At the end of each chapter, books and journals referred by the specialists during the formulation are mentioned in bibliography along with additional reading material links and references are provided. Simplified Flowcharts, Algorithms and Tables have been used to depict contents in a comprehensive manner. The most effective and feasible therapy is chosen in case if several modalities of therapy are available. When to refer a case is also indicated. Drugs & dosages as well as alternatives are provided clearly. Photographs for reference are also included where required.

This booklet is intended as a ready reference for the clinical aspects of the conditions included in the book. It is meant for medical officers and health care providers from different levels in the public health care system, who wants to refer information about the condition from a concise source.

At the beginning of the booklet a list of conditions as per speciality are indexed for rapid referencing. Apart from this, section-wise contents are also provided in between sections. Abbreviations used throughout the booklet are also mentioned in the initial pages after master index. Introduction, concept of essential medicines, benefits of standard treatment protocol and rational prescription practices along with common prescription terminologies are listed next. Each section documents the nature and magnitude of the condition and provides basic approaches to the diagnosis and management. Bibliography mentions the source of information for the chapter along with further reading material sources for in depth reading for interested readers is provided at the end of each chapter of the booklet. The emphasis of the protocols is on rapid assessment and decision making, in order to prioritise the patients and the urgency of action required. It helps to easily identify the conditions that require referral to a higher level of health care.

This booklet is intended for health care providers who diagnose and treat patients at various levels of health care in a practical manner. The booklet is supposed to be guide and aid for making decisions on diagnosis and management of conditions for majority of the population. The management of individual cases would depend on clinical judgement and skills of the health care provider.

II. STANDARD TREATMENT PROTOCOLS

The terms standard treatment protocols, treatment guidelines, and prescribing policies are all used to indicate systematically developed statements to help practitioners or prescribers make decisions about appropriate treatments for specific clinical conditions. Treatment guidelines exist for different levels of health care, ranging from general treatment guidelines for sub centre to detailed protocols for Tertiary level healthcare institutes.

Advantages

Sr No.	Group	Benefits
1	Health Care Managers	<ul style="list-style-type: none">Deciding most effective, economical treatment for a specific setting
2	Health Care providers	<ul style="list-style-type: none">Gives opportunity to concentrate on correct diagnosis
3	Supply system managers	<ul style="list-style-type: none">Helps in calculation of demand based on the utilisation pattern of the drugs for treating conditions
4	Patients	<ul style="list-style-type: none">Ensures consistency among prescribers for treatment through provision of most cost-effective treatmentsEncourages adherence to treatment thus improving the availability of drugs and ensuring better treatment outcome
5	Overall	<ul style="list-style-type: none">Helps to integrate the technical advices of specialists from different disease programmes into an overall training policyCan be used as the basis for training, for supervisionFor medical audit to assess and compare quality of care

III. ESSENTIAL MEDICINES

The Alma-Ata declaration during the International Conference on Primary Health Care in 1978 reaffirms that health is a fundamental human right and the attainment of the highest possible level of health is a most important worldwide social goal. Provision of essential medicines is one of the eight essential components of primary health care outlined in The Alma Ata declaration. Medicines are integral parts of the health care and the modern health care is unthinkable without the availability of necessary medicines. The medicines are undoubtedly one of the weapons of mankind to fight disease and illness. They not only save lives and promote health, but prevent epidemics and diseases too. Accessibility to medicines is one of the fundamental right of every person.

The concept of essential medicines

World Health Organization (WHO) introduced the concept of essential medicines in 1977. **Essential** medicines, as defined by the World Health Organization (WHO) are those that satisfy the priority health care needs of the population. They are selected with due regard to public health relevance, evidence on efficacy and safety, and comparative cost-effectiveness. Essential medicines are intended to be available within the context of functioning health systems at all times in adequate amounts, in the appropriate dosage forms, with assured quality and adequate information, and at a price the individual and the community can afford. The implementation of the concept of essential medicines is intended to be flexible and adaptable to many different situations.

While the open pharmaceutical market is flooded with large number of medicines many of which are of doubtful value, an essential medicine list contains limited cost-effective and safe medicines. The concept of essential medicines has been worldwide accepted as a powerful tool to promote health equity and its impact is remarkable as the essential medicines are proved to be one of the most cost-effective elements in health care.

Several factors like *efficacy, safety, public health relevance and comparative cost-effectiveness of available treatments* determine the selection of essential medicines. Other factors which are also considered include factors such as local demography and pattern of disease, treatment facilities, training and experience of the available personnel, local availability of individual pharmaceutical products, financial resources and environmental factor. The generic name of the medicine is considered in selecting medicines for the List.

Lists of essential medicines also guide the procurement and supply of medicines in the public sector.

Bibliography

1. World Health Organization. Declaration of Alma Ata. International conference on primary health care, Alma-Ata, USSR, 6-12 September 1978. Geneva: WHO, 1978. (Cited 2016 June 28)
Available from: www.who.int/hpr/NPH/docs/declaration_almaata.pdf

Further reading

1. World Health Organization. The selection and use of essential medicines. Report of the WHO Expert Committee, 2002 (including the 12th Model List of Essential Medicines). Technical Report Series No 914. Geneva: WHO



IV. RATIONAL USE OF MEDICINE

Rational use of medicine is defined by WHO as "Patients receive medications appropriate to their clinical needs, in doses that meet their own individual requirements, for an adequate period of time, and at the lowest cost to them and their community."

Worldwide more than 50% of all medicines are prescribed, dispensed, or sold inappropriately, while 50% of patients fail to take them correctly. Moreover, about one-third of the world's population lacks access to essential medicines.

Rational use of medicine has attained more significance now-a-days in terms of medical, socio-economical and legal aspects. Factors leading to need for rational use of medicines are:

1. Drug explosion: Increase in the number of medicine available for a particular indication complicates choice of appropriate drug dosage.
2. Prevention of development of resistance: Irrational use of medicine may lead development of resistance to highly effective medicines prematurely
3. Growing awareness: With the rapid spread of technology, information about drug development, its uses and adverse effects can be accessed by any part of the world and is available at the fingertips of consumers
4. Increased cost of the treatment: Rational use of medicine can reduce the economic burden on the public as well as on the Government.
5. Consumer Protection Act (CPA): Extension of CPA in medical profession may restrict the irrational use of medicines.

Common types of irrational medicines use are:

- The use of too many medicines per patient (poly-pharmacy);
- Inappropriate use of antimicrobials, often in inadequate dosage, for non-bacterial infections;
- Over-use of injections when oral formulations would be more appropriate;
- Failure to prescribe in accordance with clinical guidelines;
- Inappropriate self-medication, often for prescription-only medicines.

Reasons for irrational use of medicines are:

- Lack of information
- Faulty and inadequate training and education of medical graduates
- Poor communication between health professional and patients
- Uncertainty of diagnosis
- Demand from the patient
- Defective drug supply system and ineffective drug regulation
- Promotional activities of pharmaceuticals

Irrational use of medicine may lead to

- Adverse, possibly lethal effects due to antibiotic misuse or inappropriate use of drugs
- Limited efficacy of treatment regimen
- Antibiotic resistance due to overuse as well as under-therapeutic dosage
- Drug dependence
- Risk of infections due to improper use of injections
- Waste of resources leading to medicine stock-outs and increased cost of treatment
- Exacerbation or prolongation of illness
- Distress and harm to patient leading loss of patient confidence in the health system

Bibliography

1. World Health Organization. Promoting Rational Use of Medicines: Core Components. [Internet] [cited 2016 June 27]
Available from: <http://apps.who.int/medicinedocs/en/d/Jh3011e/2.html>.
2. Brahma D, Marak M, Wahlang J. Rational Use of Drugs and Irrational Drug Combinations. The Internet Journal of Pharmacology. 2012: Vol 10; Number 1.

Further reading

1. World Health Organization. The selection and use of essential medicines. Report of the WHO Expert Committee, 2002 (including the 12th Model List of Essential Medicines). Technical Report Series No 914. Geneva: WHO



V. RATIONAL PRESCRIBING

WHO "Guide to good prescribing" provides step by step process of rational treatment.

Step 1: Define the patient's problem: A patient usually presents with a complaint or problem. Making a right diagnosis is crucial for starting a correct treatment strategy. This is based on integrating many pieces of information: the complaint as described by the patient; a detailed history; physical examination; laboratory tests; X-rays and other investigations.

Step 2: Specify the therapeutic objective: Before choosing a treatment it is essential to specify your therapeutic objective. Specifying therapeutic objective will prevent a lot of unnecessary drug use and helps to avoid unnecessary prophylactic prescribing. Discussing therapeutic objective with patient before starting treatment makes them an informed partner in the therapy and improves adherence to treatment.

Step 3: Selecting a suitable drug regimen: First confirm if drug is really needed. Very often, health problems can be resolved by a change in lifestyle or diet, use of physiotherapy or exercise, provision of adequate psychological support, and other non-pharmacological treatments; these have the same importance as a prescription of drug and instructions must be written, explained and monitored in the same way. Depending on the indication, suitable dosage schedule for suitable duration is selected taking into consideration factors like safety, efficacy, interactions and contra indications. If necessary the dosage form, the dosage schedule and the duration of treatment can be changed as per need of each patient. The selected strategy should be agreed with the patient; this agreement on outcome, and how it may be achieved, is termed concordance.

Step 4: Write a prescription: A prescription is an instruction from a prescriber to a dispenser. It is a medicolegal document which links prescriber, dispenser and patient. This step is covered in detail in the next section.

Step 5: Giving information, instructions and warning: This step is important to ensure patient adherence and helps create a good doctor-patient relationship This step is covered in detail in the next section.

Step 6: Monitoring treatment. Monitoring the treatment enables you to determine whether it has been successful or whether additional action is needed. Monitoring interval depends on the type of illness, the duration of treatment, and the maximum quantity of drugs to prescribe. At the start of treatment, the interval is usually short; it may gradually become longer, if needed.

Knowledge and ideas about drugs are constantly changing. New drugs come in the market and experience with existing drugs expands. Side effects become better known and new indications or ways of using existing drugs are developed. A physician is expected to know about developments in drug therapy.

Bibliography

1. World Health Organization. Guide to Good Prescribing - A Practical Manual. [Internet] [cited 2016 June 27]
Available from: <http://apps.who.int/medicinedocs/en/d/Jwhozip23e/5.html>



VI. PRESCRIPTION WRITING

A prescription is an instruction from a prescriber to a dispenser. The prescriber is not always a doctor but can also be a paramedical worker, such as a lady health visitor, an auxiliary nurse midwife or a staff nurse. The dispenser is not always a pharmacist, but can be a pharmacy technician, an assistant or a nurse. All prescriptions orders should be comprehensible, clear-cut, dated (and time in the case of chart order), and signed clearly for optimal communication between prescriber, pharmacist, and nurse. A good prescription or chart order should contain sufficient information to permit the pharmacist or nurse to avoid possible errors during dispensing or administering the drug.

Information on a prescription form

The most important requirement is that the prescription be clear. It should be legible and indicate precisely what should be given.

The following details should be shown on the form:

- Name, address, telephone of prescriber
- Date
- Generic name of the drug, strength
- Dosage form, total amount
- Label: instructions, warnings
- Name, address, age of patient
- Signature or initials of prescriber

The prescriber's name, address and telephone number - This will allow either the patient or the dispenser to contact the prescriber for any clarification or potential problem with the prescription.

Date of the prescription

Name, form, strength of the drug and duration of treatment.

- The Generic (non-proprietary) name of the drug should always be used. It also enables the pharmacist to maintain stock of drugs, or dispense the drug easily. If there is a specific reason to prescribe a special brand, the trade name can be added.
- The pharmaceutical form (for example 'tablet', 'oral solution', 'eye ointment') should also be stated.
- The strength of the drug should be stated in standard units using abbreviations that are consistent with the International System of Units (SI). 'Microgram' and 'nanogram' should not be abbreviated since abbreviated form ("µg") is very easily misread as "mg", a 1000-fold overdose. Also, 'units' should not be abbreviated.
- Try to avoid decimals whenever possible. If unavoidable, a zero should be written in front of the decimal point. Use ballpoint pen to avoid ink spread.

Specific areas for filling in details about the **patient including name, address and age**.

Directions

- Directions should be written out in full in English, many Latin abbreviations are still in use (some common terms are listed in Table 1). Knowledge of these abbreviations is essential for the dispensing pharmacist and often useful for the prescriber.
- The abbreviation “OD” should be used only to mean “the right eye” (if used); it has been used for “every day” and has caused inappropriate administration of drugs into the eye. Acronyms such as ASA (aspirin), 5-ASA (5-Aminosalicylic acid), PCM (paracetamol), CPM (chlorpheniramine), CPZ (chlorpromazine) etc., should not be used; drug names should be written out.
- Unclear handwriting can be lethal when drugs with similar names especially brand names but very different effects are available e.g., Daonil, Duodil and Diovol. In this situation, errors are best avoided by noting the indication for the drug in the body of the prescription e.g., “Daonil (Glibenclamide), for diabetes”.
- Directions specifying the route, dose and frequency should be clear and explicit; use of phrases such as ‘take as directed’ or ‘take as before’ should be avoided.
- For preparations which are to be taken on an ‘as required’ basis the quantity should be specified with maximum limit and minimum interval. It is good practice to specify the purpose of the medication (for example ‘every 6 hours as required for pain’, or ‘at night as required to sleep’).
- Where possible, usage directions should specify times (7 am, 3 pm, 11 pm) rather than simply frequency (thrice a day) and especially relationship to meals for orally consumed medication.
- Avoid units such as "teaspoons" or "tablespoons."
- The length of the treatment course may be stated
- For liquid preparations, the quantity should be stated in millilitres (abbreviated as ‘ml’) or litres (abbreviated as ‘L’, since the letter ‘l’ could be confused with the figure ‘1’).
- Explain the directions to the patient. The directions should be reinforced by the label on the medicinal product and by appropriate counselling by the dispenser.

Narcotics and controlled substances

Under Narcotic Drugs and Psychotropic Substances Act, 1985; the prescribing of a medicinal product that is liable to abuse requires special attention and may be subject to specific statutory requirements. In particular, the strength, directions and the quantity of the controlled substance to be dispensed should be stated clearly, with all quantities written in words as well as in figures to prevent alteration. Other details such as patient particulars and date should also be filled in carefully to avoid alteration.

Table 1. List of abbreviations used in medical prescriptions

Abbreviation	From the Latin	Explanation	Abbreviation	From the Latin	Explanation
a.c.	ante cibum	before meals	o.u.	oculus uterque	Both eyes
ad lib.	ad libitum	use as much as one desires	oz		ounce
bid	bis in die	twice a day	per	Per	by or through
bol.	bolus	as a large single dose (usually intravenously)	p.c.	post cibum	After meals
cap	capsula	capsule	prn	pro re nata	as needed
dil.		dilute	p.o.	per os	by mouth or orally
disp.		dispense	p.r.		By rectum
div.		divide	q	Quaque	every
D.W.		distilled water	q.a.m	quoque die ante meridiem	every day before noon. every morning
g		gram	q.d.	quaque die	every day
gr		grain	q.h, q1h	quaque hora	every hour
gtt (s)	gutta (e)	drop (s)	q.i.d.	quater in die	four times a day
hr		hour	q.s.	quantum sufficiat	sufficient quantity
h.s.	hora somni	at bedtime right eye	Rx		Take
ID		intra dermal	RL, R/L		Ringer's lactate
IM		Intramuscular (with respect to injections)	SC		subcutaneous
inj.	injection	injection	s.o.s.	si opus sit	if there is a need
IP		intra peritoneal	stat	Statim	at once
IV		intravenous	supp	suppositorium	suppository
liq	liquor	solution	susp		suspension
min	minimum	a minimum	syr	Syrupus	syrup
mcg		microgram	tab	Tabella	tablet
mEq		milliequivalent	Tbsp, T		tablespoon (always write out "15 ml")
mL		millilitre	tid		three times a day
noct.	nocte	at night	tsp		teaspoon (always write out "5 ml")
NS		Normal saline (0.9%)	t.i.d/t.d.s	ter in die/ ter die sumendum	Three times a day
o.d.	oculus dexter	Right eye	top.		topical
o.s.	oculus sinister	Left eye	U		units (always write out "units")

Bibliography

1. World Health Organization. Guide to Good Prescribing - A Practical Manual. [Internet] [cited 2016 June 27]
Available from: <http://apps.who.int/medicinedocs/en/d/Jwhozip23e/5.4.html>

Further reading

1. Johnston, Mike (2006). The pharmacy technician series: Fundamentals of pharmacy practice. Pearson Prentice Hall. p. 24
2. Aronson JK. A prescription for better prescribing. British Journal of Clinical Pharmacology. 2006;61(5):487-491.
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1885053/pdf/bcp0061-0487.pdf>
3. Scobie SD, Lawson M, Cavell G, Taylor K, Jackson SH, Roberts TE. Meeting the challenge of prescribing and administering medicines safely: structured teaching and assessment for final year medical students. Med Educ. 2003; 37: 434–7



Medicine



1. Medicine

Sr. No.	Contents	Page No.
1	Fever	1
2	Community Acquired Pneumonia (CAP)	6
3	Bronchial Asthma	9
4	Pleural Effusion	12
5	Chronic Obstructive Pulmonary Disease (COPD)	14
6	Bronchiectasis	16
7	Lung Abscess	18
8	Pneumothorax	20
9	Lung Cancer	23
10	Pulmonary Embolism	25
11	Hypertension	27
12	Heart Failure	32
13	Ischaemic Heart Disease and Acute Coronary Syndrome	34
14	Acute Rheumatic Fever	37
15	Infective Endocarditis	39
16	Diabetes Mellitus	43
17	Thyroid Disorders	55
18	Cerebrovascular Accidents	58
19	Subarachnoid Haemorrhage (SAH)	60
20	Pyogenic Meningitis	62
21	Tuberculous Meningitis	64
22	Viral Meningitis	65
23	Viral Encephalitis	67
24	Epilepsy	69
25	Guillain Barre Syndrome (GBS)	73
26	Aphthous Ulcers	75
27	Oesophageal Candidiasis	76
28	Dyspepsia	77
29	Gastro oesophageal Reflux	78

30	Peptic Ulcer Disease	79
31	Vomiting	82
32	Constipation	83
33	Irritable Bowel Syndrome	84
34	Ulcerative Colitis	85
35	Amoebic Liver Abscess	87
36	Pyogenic Liver Abscess	89
37	Intestinal Protozoal Infection	91
38	Ascites	95
39	Hepatitis	97
40	Hepatic Coma	99
41	Nephrotic Syndrome	101
42	Acute Nephritic Syndrome	102
43	Acute Renal Failure/Acute Kidney Injury	103
44	Chronic Kidney Disease	106
45	Malaria	108
46	Dengue	116
47	Leptospirosis	121
48	Influenza AHN (Swine Flu)	123
49	Diarrhoeal Diseases	126
50	Rheumatoid Arthritis	132
51	Snake Bite	134
52	Scorpion Sting	138
53	Dog Bite (Rabies)	140
54	Poisoning	143
55	Alcohol Intoxication / Alcohol Withdrawal	147
56	Anaemia	148
57	Heat Stroke	150
58	Tuberculosis (RNTCP)	154
59	Leprosy (NLEP)	163

1. FEVER

Body temperature is controlled by the hypothalamus. The normal core body temperature is 36.5-37.5°C (97.7-99.5°F). The morning temperature of >98.9°F and the evening temperature of >99.9°F defines fever.

1. Definition

Since an oral temperature is 0.5°F (0.3°C) to 1°F (0.6°C) lower than a rectal or tympanic temperature:

Rectal temperature $\geq 100.4^\circ\text{F}$ - Core temperature

Tympanic temperature $\geq 100.4^\circ\text{F}$ - Core Temperature

Oral temperature $\geq 99.5^\circ\text{F}$ -99.9°F

Axillary temperature $\geq 99.0^\circ\text{F}$ -99.5°F

Note:

- This is not absolute, remember that fever is a relative condition.
- Have a lower threshold for fever at 6am or 6pm.
- Keep antipyretics and recent intake in mind when considering fever.
- Feeling hot- does not necessarily imply fever.
- Rigors – profound chills accompanied by chattering of teeth & severe shivering implies a rapid rise in body temperature.

2. Causes

- Malaria
- Sepsis
- Abscess
- Brucellosis
- Lymphoma
- Night sweats– Characteristic of Tuberculosis, but sweating from any cause is usually worse at night.
- Recurrent fever– Cholecystitis, Cholangitis and Urinary tract infection with obstruction or calculi.
- Headache– Fever due to any cause can produce headache. If severe and with photophobia suspect- Meningitis.
- Delirium– Common in elderly and young ones.
- Muscle pain– Myalgia classical of viral fever, Influenza, Malaria, Leptospirosis and Brucellosis.

3. Evaluation of Febrile Patient

Although fever is a “normal response”, prolonged episodes can cause damage so always evaluate for stability of patient (regardless of what you think is the cause).

Table 1: Evaluation of fever patient

1.Temperature	Axillary temperature $>99^\circ\text{F}$
2.White Blood Cell Count	$>12,000$ or $<4,000$ or $>10\%$ bands
3.Heart Rate	>90 bpm*
4.Respiratory Rate	>24 bpm^ or $\text{PaCO}_2 < 32\text{mm Hg}$
Sepsis = SIRS** + infection	
Severe sepsis = SIRS** + infection + end organ damage	
Septic shock = Severe sepsis + refractory hypotension (<90 mm Hg or 40% below baseline)	
* beats per minute	
^ breaths per minute	
**SIRS- systemic inflammatory response syndrome	

3.1. Fever Pattern

It is important to note that the cycle of fever pattern is often not very helpful in determining the cause of the disease.

Possible exceptions are: Tertian and Quartan Malaria, Abscesses, Pel-Ebstein fever and drug fever.

3.2. Relation to Pulse

Liebermeister’s rule: For every one-degree Celsius rise of temperature above normal, the pulse will increase by 8-10 beats per minute.

Faget Sign: The exception to Liebermeister’s Rule.

This Relative bradycardia may be useful when present, although it is associated with a substantial

differential diagnosis, including Typhoid fever, Rickettsial diseases, Yellow fever, Legionnaire's disease, Psittacosis, Leptospirosis, Drug fever, Brucellosis, Mycoplasma infections, Neoplasm and Factitious fever.

4. Types of fever

- Continuous fever - Does not fluctuate more than 1°C in 24 hr. e.g. Lobar Pneumonia, Typhoid fever, Brucellosis, Urinary tract infection.
- Intermittent fever- Temperature elevation for a certain period then returning to normal. e.g. Malaria, Pyaemia, Septicaemia.
- Quotidian- Periodicity of 24 hours - Plasmodium Falciparum Malaria.
- Tertian fever- 48-hour periodicity - Plasmodium Vivax and Ovale.
- Quartan fever – 72-hour periodicity - Plasmodium Malaria
- Remittent fever- Temperature remains above normal throughout the day with fluctuations more than 1°C in 24 hours e.g. Infective Endocarditis.

5. Hints to be obtained from history

(Since Presentation can be non- specific)

- Detailed fever history
- Medication review
- Family illnesses
- Ethnicity
- Detailed history of past surgeries
- Recent sick contacts and TB contacts/risks
- Host factors (Immunocompromised)
- Recent travel
- Environmental exposures associated with jobs or hobbies
- Animal exposure
- Unusual dietary habits
- High risk behaviour
- Sexual history including Contraceptives
- Gynaecologic history
- Hypersensitivities to environmental agents / medicines or family history of such diseases

6. Common associated symptoms

- Fever only

- Neurologic
- Abdominal
- Pulmonary
- Rash
- Haemorrhage
- Bone and joint
- Gynaecologic

6.1 Fever only

- Malaria
- Typhoid fever
- Dengue
- Leptospirosis
- Rickettsia
- Relapsing fever
- Other viral illnesses
- HIV

6.2 Neurologic symptoms

Fever, headache, altered mental status, convulsions, coma

- Cerebral malaria
- Meningitis
- Encephalitis
- Chronic Meningitis: Tuberculous meningitis, Cryptococcal Meningitis
- Rabies
- Japanese Encephalitis
- West Nile Encephalitis
- HIV
- Toxoplasmosis
- HIV dementia
- Trypanosomiasis (Sleeping Sickness)

6.3 Abdominal symptoms

Fever, abdominal pain

- Typhoid
- Infectious colitis: *Shigella*, *E. coli*, *salmonella*, *Campylobacter*, *Amoeba*
- Amoebic liver abscess
- Abdominal TB
- Appendicitis, Pyelonephritis
- HIV

6.4 Fever and rash

Fever and skin rash

- Chicken pox
- Measles
- Dengue
- Other viral diseases

6.5 Haemorrhagic symptoms

Hematemesis, melena, epistaxis, petechiae, purpura, puncture site bleeding

- Dengue
- Relapsing fever
- Ebola, Lassa, Marburg
- Yellow fever

6.6 Bone and Joint

Fever with joint or bone pain

- Sickle cell disease
- Septic arthritis
- Osteomyelitis
- Pyomyositis
- Rheumatic fever
- Chikungunya
- Brucellosis

6.7 Gynaecological symptoms

Fever, pelvic pain, vaginal discharge

- PID
- Tubo-ovarian abscess
- Postpartum endometritis
- Septic abortion

7. Symptom Analysis of Fever

- Verify presence of fever- true/factitious
- Duration-acute/chronic
- Mode of onset- abrupt/gradual
- Progression- continuous/intermittent
- Severity- how it affects the daily work / physical activities?
- Relieving and aggravating factors
- Treatment received and outcome
- Associated symptoms- localizing features.

7.1. Respiratory tract symptoms

- Sore throat, nasal discharge, sneezing - URTI
- Sinus pain & headache- Sinusitis
- Cough, sputum, wheeze or breathlessness - LRTI

7.2. Genitourinary Symptoms

Frequency of micturition, loin pain, vaginal or urethral discharge suggesting

- Urinary tract infection
- Pelvic inflammatory disease
- Sexually transmitted disease

7.3. Abdominal symptoms

Diarrhoea with or without blood, weight loss and abdominal pain

- Gastroenteritis
- Intra-abdominal sepsis
- Inflammatory bowel disease
- Malignancy

7.4. Skin rash-appearance & distribution will give a clue

- Macular - Measles, Rubella, Toxoplasmosis
- Haemorrhagic - Meningococcal, Viral haemorrhagic fever
- Vesicular - Chicken Pox, Shingles, Herpes Simplex
- Nodular - Erythema Nodosum- TB & Leprosy
- Erythematous - Drug rash and Dengue fever.

7.5 Joint symptoms-

Joint pain, swelling or limitation of movement is suggestive of active arthritis

- Distribution-mono, oligo, polyarticular
- Appearance-Fleeting-Rheumatic fever
- Oligoarthritis-infective, Koch's
- Polyarticular- Rheumatoid arthritis, Osteoarthritis
- Axial skeleton involvement: Spondyloarthropathy, Psoriatic

8. Hints to obtain from examination

- Vital Signs: Monitor all of the vital signs for stability
- General appearance: Do they look sick? Anxious? Do they have altered sensorium? look for pallor, icterus, cyanosis, clubbing and lymphadenopathy.
- Oral examination: Oral cavity infections, dental examination, gum examination, sinuses
- Cardio vascular examination: Any murmurs
- Respiratory examination: Bronchial sounds, Decreased breath sounds, Adventitious sounds
- Central nervous system examination: Fundus examination, Mental Status, Encephalopathy, look for any neurological deficit.
- Per abdomen: Tenderness, Organomegaly, Ascites
- Skin: Rashes, Nail Exam, Wounds / Decubitus Ulcers
- Musculoskeletal examination: Joint examination, Muscle tenderness.

- Genital / pelvic examination and rectal examination.
- Look for indwelling devices.

9. Differential diagnosis

- **Infection** (TB, UTI/Prostatitis, Endocarditis, Abscess, Line Infection, Sinusitis, Meningitis, Arthritis, Osteomyelitis, Wound infectious, Diarrhoea)
- **Inflammatory** (Rheumatic Disorders, Vasculitis)
- **Drug Fever** (Beta - Lactam antibiotics, Amphotericin B, Chemotherapy, Drug Interactions)
- **Thrombotic** (DVT / PE / MI)
- **Neurologic** (Hypothalamic disorder, Spinal Cord Injuries, ICH)
- **Endocrine** (Thyrotoxicosis, Adrenal Insufficiency, Subacute Thyroiditis)
- **Gastrointestinal** (IBD, Pancreatitis, Cholecystitis)
- **Malignancy**

10. Fever Workup

10.1 Minimum in all patients

- CBC with differential and reticulocyte count
- Blood smear- for malarial parasites
- CXR PA and Lateral. Add Decubitus if needed. Infiltrates negative if dry
- Urine analysis (with Microscopy) and Urine Culture
- 2 sets of blood cultures + Cultures from any central catheter
- Electrolytes and Metabolic Panel, LFTs, Hepatitis Panel, HIV Test

10.2 Other Specific procedures/labs to obtain data

- Autoimmune Workup (RF, ANA, etc. as history guide, ESR, CRP)
- Specific Viral Serology
- Lumbar Puncture, Thoracentesis, Arthrocentesis, Paracentesis
- CT Scan of Head
- CT PE Protocol / Doppler of extremities
- Echocardiogram
- Stool Cultures - Gram Stain, Clostridium difficile toxin etc.
- Sputum Cultures
- Skin biopsy

10.3. ESR

Normal: Men = Age/2, Women = Age+10/2

Elevated in:

- Acute or Chronic Inflammation
- Infection
- Tissue Injury
- Thyroid Disease
- Azotaemia

An elevated ESR does not rule in or out disease.

As opposed to the ESR, the CRP increases more quickly with an acute process, and decreases for quickly when the underlying state resolves.

ESR > 100

- T - TB
- O - Osteoarthritis
- E - Endocarditis
- V - Vasculitis (Temporal Arteritis)
- A - Abscess
- N - Neoplasm (especially Lymphoma, Plasma Cell dyscrasias)

10.4. Blood cultures

- The bacteriologic burden is highest in the blood stream approximately 1 hour before fever spikes. So, collect blood cultures 1 hour before fever spike.
- If you are suspecting Endocarditis, tell the lab to continue following the cultures for at least 4 weeks.

11. Treatment of Fever Itself

- Give empiric Antibiotics when there is high suspicion of the source of infection or if the source is unknown and the patient is unstable.
- Tab Paracetamol 500 mg orally 4 times a day for most fevers with discomfort.
- Don't always lower the temperature so readily.

This decreases your ability to know when to draw cultures and may lower the patient's defence mechanism. Once workup has been performed (and possibly repeated) then temperature can be lowered. However, have low threshold for lowering the temperature when there is a hypermetabolic state that would be damaging (i.e. concurrent MI, CVA) or if the patient is very symptomatic.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S et al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015
4. National Treatment Guidelines for antimicrobial use in infectious diseases – Version 1.0 (2016), National Center for Disease Control, Ministry of Health and Family Welfare. [Cited 2016 July 7]
Available from: http://www.ncdc.gov.in/writereaddata/linkimages/AMR_guideline7001495889.pdf

Further reading

1. WHO Informal Consultation on Fever Management in Peripheral Health Care Settings a Global Review of Evidence and Practice [Internet]. [cited 2016 Jul 4]
Available from: http://apps.who.int/iris/bitstream/10665/95116/1/9789241506489_eng.pdf



2. COMMUNITY ACQUIRED PNEUMONIA [CAP]

1. Introduction

- Pneumonia is an infection of pulmonary parenchyma that causes them to function abnormally
- Classified as typical or atypical, although the clinical presentations are often similar. Approximately 20-33% of episodes result in hospitalization

Typical: Up to 70% usually caused by *Streptococcus pneumoniae*

Atypical: 30-40% (“My Lungs Contain Viruses”)

- *Mycoplasma pneumoniae*
- *Legionella pneumophila*
- *Chlamydia pneumoniae*
- Viruses: Influenza, Adenovirus

2. Clinical features

Symptoms:

- Cough, fever, chills, fatigue, dyspnoea, rigors, and pleuritic chest pain
- Cough may be persistent and dry or it may produce sputum
- Other presentations may include headache and myalgia
- Certain aetiologies, such as legionella, also may produce gastrointestinal symptoms

Signs -

- Tachycardia
- Tachypnea
- Dullness to percussion of chest, crackles or rales on auscultation, bronchial breath sounds, tactile fremitus, and Egophony (“E” to “A” changes)
- Patients with typical pneumonia are more likely to present with dyspnoea and bronchial breath sounds on auscultation

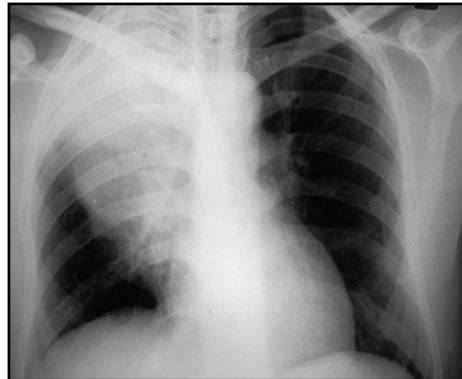
3. Radiological imaging

3.1 Chest x ray (PA and Lateral)

- Lobar consolidation – more common in typical pneumonia
- Bilateral, diffuse infiltrates – commonly seen in atypical pneumonia

If performed early in the course of the disease, may be negative

Figure 2.1 - Chest X-Ray PA VIEW - CAP



3.2 CT scan- Could be performed in patients with a negative chest radiograph when there is a high clinical suspicion for pneumonia and to rule out other pathologies.

4. Laboratory Diagnosis

Complete blood count, sputum, gram stain and cultures, blood sugars, blood urea, serum creatinine.

5. Treatment

Initial treatment of CAP is based on physical examination findings, laboratory results and patient characteristics. After examination, you must decide whether to treat patient on OPD basis or to admit the patient.

Patients with any one of following features must be admitted

- Respiratory rate >30/min
- Systolic BP \leq 90 mmHg or diastolic \leq 60 mmHg
- New onset confusion or impaired level of consciousness
- Comorbid illness- Diabetes, Ischaemic Heart Disease, Alcoholics, Immunocompromised, Multilobar pneumonia

Therapy for pneumonia is empiric because specific pathogens usually are not identified at the time treatment is initiated.

5.1. Duration of therapy

- *S. pneumoniae*: 7-10 days or until afebrile for 3 days
- *Mycoplasma/Chlamydia pneumoniae*: 10-14 days, up to 21 days
- *Legionella*: 10-21 days

5.2. CAP, not hospitalized with No comorbidities

- Cap Amoxicillin 500 mg three times a day x 5 days
- Tab Azithromycin 500 mg PO x 1, then 250mg once a day -5 days OR
- Tab Clarithromycin 500 mg twice a day - 5 or 7 days OR
- Cap Doxycycline 100 mg twice a day -10 days

5.3. CAP, not hospitalized with comorbidities

- Cap Amoxicillin 1gm thrice a day x 5 days plus Tab Azithromycin / Clarithromycin as above doses OR
- Cap Amoxicillin and Clavulanate 2 gm twice a day x 5 days plus a Tab Azithromycin / Clarithromycin as above doses

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015
4. National Treatment Guidelines for antimicrobial use in infectious diseases – Version 1.0 (2016), National Center for Disease Control, Ministry of Health and Family Welfare. [Cited 2016 July 7] Available from: http://www.ncdc.gov.in/writereaddata/linkimages/AMR_guideline7001495889.pdf

OR

- Tab Levofloxacin 750 mg once a day, OR Moxifloxacin 400 mg once a day, OR Gemifloxacin 320 mg once a day x 5-7 days OR
- Tab Cefpodoxime 200 mg BD or Tab Cefuroxime 500 mg BD Plus Macrolide antibiotics

5.4. Inpatients

- I.V. Cefotaxime 1-2 gm 8 hourly OR
- I.V. Ceftriaxone 1-2 gm once a day OR
- I.V. Ampicillin 1-2 gm 4-6 hourly OR
- I.V. Ampicillin Sulbactam 2 gm 8 hourly x 4 days plus
- Tab Azithromycin or in severe cases I.V. Azithromycin 1 gm on day one and then 500 mg once a day for next 4 days or Tab Levofloxacin, Moxifloxacin as above.
- After clinically stable (T<100.0°F, HR<100 beats/min, RR<24/min, SBP>90mm of Hg, O2 saturation>90%) and able to tolerate oral intake, may be switched to oral antibiotics for remainder of therapy
- PPV23 vaccine is recommended for all adults \geq 65 years of age and in younger patients with a number of conditions that increase the risk of invasive pneumococcal disease.

6. Complications

Lung abscess, pleural effusion, empyema. These patients need to be referred to district hospital.

Further reading

1. Gupta D, Agarwal R, Aggarwal AN, Singh N, et al. Working Group Guidelines for Diagnosis and Management of Community and Hospital Acquired Pneumonia in Adults: Joint ICS/ NCCP (I) Recommendations [Internet]. Lung India 2012;29: S27-S62. [cited 2016 Jul 4]
Available from: <http://medind.nic.in/iae/t12/i4/iaet12i4p267.pdf>



3. BRONCHIAL ASTHMA

1. Introduction

Asthma is defined as a chronic inflammatory disease of airway that is characterized by increased responsiveness of tracheobronchial tree to a multiplicity of stimuli.

2. Precipitating factors

- Childhood infections – Respiratory syncytial virus
- Allergen exposure – Allergy to feathers, animal danders, dust mites, molds
- NSAID, Aspirin, Beta blocker, Sulphite containing topical ophthalmic solution, food preserving agent
- Wood and vegetable dust, industrial chemicals and plastic
- Exercise, emotional stress.

3. Symptoms

- Dyspnoea, cough and wheezing
- Sense of constriction in the chest
- Cough that produces thick, stringy mucus. Increase mucus production, typically tenacious mucus.

4. Signs

Tachypnea, tachycardia, mild systolic hypertension

Respiration become audibly harsh, rhonchi heard on auscultation.

In severe cases - Accessory muscle become visibly active, Paradoxical pulse, Cyanosis, Silent chest. In some severe cases patients may land in to respiratory failure.

5. Investigation

- Sputum and blood examination for eosinophilia
- Chest X-ray showing hyper inflated lungs

- Simple spirometry shows air flow limitation with decreased FEV₁, FEV₁ / FVC and PEF.
- Peak expiratory flow- by using peak expiratory flow meter >20% of diurnal variation on ≥3 days in a week for 2 weeks.
- Diagnosis of asthma is established by demonstrating reversible airway obstruction. Reversibility is defined as a ≥ 12% increase in FEV₁ 15 minutes after two puffs of a β adrenergic agonist (salbutamol) on spirometry.

6. Treatment

Drug treatment: Classified in to:

6.1. Controller: To be taken on long term basis to control asthma through their anti-inflammatory effects

(a) Inhaled corticosteroids (ICS) –

Beclomethasone 200 mcg/metered dose twice a day OR

Budesonide –200-400 mcg/metered dose twice a day OR

Fluticasone- 100-250 mcg/metered dose twice a day

(b) Systemic corticosteroids

Oral Prednisone or Prednisolone 40-60 mg once daily for 5 to 10 days

(c) Leukotriene modifier –

Tab Montelukast 10 mg once a day X 5 days.

(d) Long acting inhaled β₂ agonist-

-Salmeterol (MDI 21 mcg/puff, 2 puff 12 hourly OR

- Formoterol (1 to 2 puffs 12 hourly)

(e) Theophylline-Tab Deriphyllin 150 mg twice a day X 5 days.

6.2. Reliever: Used on or as needed basis to quickly relieve symptoms by their bronchodilator properties. These are:

Short acting inhaled β ₂ agonist (SABA) Salbutamol 100 mcg/metered dose 1-2 puffs, Levosalbutamol 50 mcg/metered dose 1-2 puffs as needed, Terbutaline

Systemic glucocorticoid - Tab Prednisolone 40-60mg/day
--

Short acting oral β_2 agonist	- Tab Salbutamol 2-4mg/day
Anti-cholinergic	- Ipratropium inhaler
Theophylline	- 100-300 mg three times a day

6.3. Combinations available as inhaler and Rotacaps

Salmeterol / Fluticasone 1-2 puffs twice daily

Formoterol / Budesonide 1-2 puffs twice daily

Salbutamol / Beclomethasone.

Inhaled drugs are preferred over oral due to less dose, less side effects, quick onset of action.

		LABA	LABA	OCS
	ICS Low dose	ICS Low dose	ICS High dose	LABA ICS High dose
Short acting β_2 agonist as required for symptom relief				
Mild intermittent	Mild persistent	Moderate persistent	Severe persistent	Very severe persistent

Figure-3.1: Step wise approach to asthma therapy according to the severity of asthma and ability to control symptoms.

ICS = Inhaled corticosteroid;

LABA = Long acting β_2 agonist

OCS = Oral corticosteroid

7. Acute severe asthma: (Status Asthmaticus)

Can be fatal and must be treated promptly.

7.1. It is characterized by

- Severe dyspnoea
- Respiratory rate ≥ 25 /min
- Heart rate ≥ 110 /min.
- Inability to complete sentence in single breath.
- PEF (peak expiratory flow) 35-50% predicted

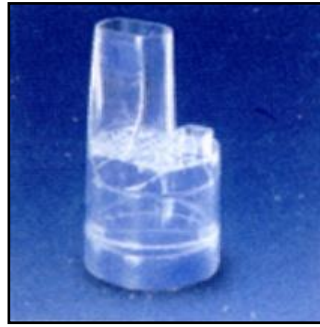
7.2. Life threatening features

- PEF < 33% predicted
- SpO₂ < 92% silent chest
- Cyanosis
- Normal or raised PaCO₂ (suggests impending respiratory failure)
- Feeble respiratory effort
- Bradycardia or arrhythmia
- Confusion, coma.

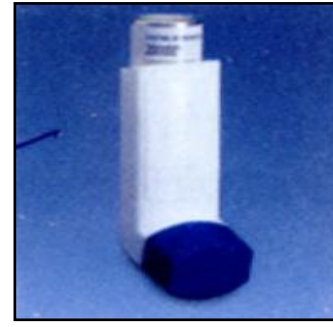
7.3. Treatment

- Oxygen 40-60 % nasally to achieve oxygen saturation >90%
- Inj. Hydrocortisone-200 mg I.V. stat and then 100 mg 8 hourly
- Salbutamol (2.5-5 mg) and Ipratropium Bromide (0.5 mg) alternately inhaled through the nebulizer.
- Inj. Aminophylline 500 mg in 500 ml of 5% Dextrose over 12 hours if patient is not receiving theophylline previously.
- Inj. Ampicillin 500 mg 6 hourly till patient is stable and then oral Ampicillin 500 mg TDS X 5 days.
- In very severe cases patient may require ventilatory support.

Refer to higher center with above life threatening signs.



Rotahaler Spacer



Metered dose inhaler



Figure 3.2 – Types of Devices used in Bronchial Asthma

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Agarwal A, Dhooria S, Aggarwal AN et al. Guidelines for diagnosis and management of bronchial asthma: Joint ICS/NCCP (I) recommendations. Lung India [Internet]. 2015 Apr; 32(Suppl 1): S3–S42. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4405919/>
2. WHO Guidelines, factsheets regarding Asthma [Internet]. [cited 2016 Jul 5]
Available from: <http://www.who.int/respiratory/asthma/en/>
3. Yash Pal Munjal, Surendra K. Sharma, A. K. Agrawal, R.K. Singal, Pritam Gupta, Shyam Sundar et al. API Textbook of Medicine. 10th edition. JP Brothers.2015



4. PLEURAL EFFUSION

A pleural effusion is an abnormal excess amount of fluid in the pleural space

1. Symptoms

- Breathlessness
- Cough
- Fever
- Pleuritic chest pain

2. Signs

- Decreased breath sounds
- Stony dullness on percussion
- Decreased vocal resonance

3. Common Causes

- Tuberculosis
- Pneumonia
- Cancer
- Liver disease (Cirrhosis)
- End-stage renal disease
- Nephrotic syndrome
- Congestive heart failure
- Pulmonary embolism
- Constrictive pericarditis
- Lupus and other autoimmune conditions

4. Investigations

4.1. Chest X-ray film

PA view, lateral decubitus, lateral view

Blunting of CP angle, Ellis S shaped in large effusions



Figure 4.1: Chest radiograph showing left-sided pleural effusion



Figure 4.2: Massive right pleural effusion with shift of mediastinum towards left

4.2. Thoracentesis

Once a pleural effusion is identified on imaging, a fluid sample is usually taken to determine the pleural effusion's character and seriousness, a procedure called Thoracentesis.

A sample of fluid is removed with a needle inserted between the ribs

Pleural fluid tests: routine, microscopy, cytology and ADA (Adenosine deaminase) levels

There are two different types of fluid

Transudative or Exudative

i. Transudative: clear fluid

Clear fluid, low protein content, cell count is low.
e.g. Congestive Cardiac Failure, Liver Cirrhosis, Nephrotic Syndrome.

ii. Exudative:

- Straw coloured, high protein content, cell count is high.

By the gross characteristics of the fluid.

- Frankly purulent fluid indicates an empyema.
- A putrid odour suggests an anaerobic empyema.
- A milky, opalescent fluid suggests a chylothorax, resulting most often from lymphatic obstruction by malignancy or thoracic duct injury by trauma
- Grossly bloody fluid result from trauma, malignancy,

5. Treatment

Management of common pleural effusions

5.1. Tuberculosis

Tuberculosis is the commonest cause of pleural effusion

- Straw coloured effusion
- Cell count - lymphocyte predominance
- Pleural fluid protein > 3 g/dl
- Cob web formation
- ADA level is high.

Tubercular pleural effusion is treated as per RNTCP guidelines

5.2. Para pneumonic effusion

- Chest X-ray consolidation features along with effusion

- Cell count predominantly neutrophilic
- Appropriate antibiotics
- Thoracentesis to ensure that empyema has not developed

5.3. Malignant effusion

- Cytology positive
- Refer to higher centre

Treatment of underlying cause in heart failure, nephrotic syndrome, liver cirrhosis

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. Jaypee Brothers. 2015

Further reading

1. WHO Guidelines for Respiratory tract diseases [Internet]. [cited 2016 Jul 5]
Available from: http://www.who.int/topics/respiratory_tract_diseases/en/



5. CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

1. Introduction

COPD is a chronic lung disease characterized by airflow limitation that is not fully reversible.

It includes chronic bronchitis and emphysema.

1.1. Chronic Bronchitis

This is a condition associated with excessive tracheobronchial mucus production sufficient to

cause cough with expectoration on most days for at least 3 months a year for more than 2 consecutive years.

1.2. Emphysema

It is defined as distension of air spaces distal to the terminal bronchiole with destruction of alveolar septa.

Table-1: Differentiating features between Emphysema and Bronchitis

Features	Predominant emphysema (Pink puffer)	Predominant bronchitis (Blue bloater)
1. Age of onset	6 th decade	5 th decade
2. Cough	After dyspnoea	Before dyspnoea
3. Dyspnoea	Severe	Mild
4. Sputum	Scanty, mucoid	Copious, purulent
5. Infections	Less common	Common
6. Respiratory insufficiency	Often terminal	Repeated attacks
7. Chest x-ray	Hyperinflation +/- bullous changes; small heart	Increased bronchovascular marking; large heart
8. PaCO ₂ (mm Hg) PaO ₂ (mm Hg)	35-40 65-75	50-60 45-60
9. Pulmonary hypertension	Mild	Moderate to severe
10. Cor pulmonale	Pre terminal stage	Common
11. Diffusing capacity	Decreased	Normal to slight reduction

Table-2: Differences between Pink puffer and Blue bloater

	Pink puffer	Blue bloater
1. Course	Progressive dyspnoea	Intermittent dyspnoea
2. Sputum	Scanty	Profuse
3. Polycythaemia	Uncommon	Common
4. X-ray	Attenuated peripheral vessels	Normal peripheral vessels
5. pCO ₂	Normal	Increased
6. Alveolar gas transfer	Reduced	Normal

2. Aetiology

Smoking (active and passive), Smoke from biomass fuel (firewood, burnt plastics)

3. Signs

- Sitting and bending forward with hands on knees (tripod position).
- Pursed lip breathing
- Cyanosis (Ominous sign)
- Forced expiratory time Normal - 4 secs, COPD - 6 secs and above
- Barrel chest

- Hyper resonant chest
- Diminished breath sound and bilateral wheeze
- Spirometry showing obstruction (FEV1/ FVC < 70%) even after bronchodilator confirms the diagnosis of COPD.

4. Treatment

4.1. Non pharmacological

- Rehabilitation
- Exercise
- Nutrition
- Education
- Avoid smoking

4.2. Pharmacological

4.2.1. Oxygen therapy

4.2.2. Antibiotics

Antibiotics are required as infection often precipitates acute attacks. Inj. Benzyl Penicillin 10 lakhs units 6 hourly 5-7 days **or** Inj. Ampicillin 500 mg 6 hourly 5-7 days

4.2.3. Bronchodilators

Inhaled bronchodilators are preferred to oral formulations in view of better efficacy and lesser side effects.

Inhaled bronchodilators include -

- Short acting beta agonists - Salbutamol, 1-2 puffs three times a day, Terbutaline 1.5 mg three times a day

- Long acting beta agonist (Salmeterol, Formoterol), 1-2 puffs twice a day
- Short acting anticholinergics (Ipratropium) 1-2 puffs, 2-3 times a day
- Long acting anticholinergic (Tiotropium) 1-2 puffs once a day.

4.2.4. Theophylline

- Inj. Aminophylline 250 mg in 500 ml of 5 % Dextrose slowly over 8-10 hours in acute attacks.
- Tab Theophylline 100-300 mg three times a day
- Side effects: Tachycardia, nausea, arrhythmias and convulsions

4.2.5 Glucocorticoids

- Inhaled corticosteroids should be given in severe COPD or in those with repeated exacerbation.
- Fluticasone 1-2 puffs twice a day
- Budesonide 1-2 puffs twice a day
- Systemic corticosteroids should be given only in patients with acute exacerbation of COPD.
- Tab Prednisolone 30-40 mg /day for 8-10 days in tapering doses

5. Complications

- i. Pneumothorax
- ii. Respiratory failure
- iii. Cor pulmonale

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015

Further reading

1. Gupta D, Agarwal R, Aggarwal AN, et el. Guidelines for diagnosis and management of Chronic Obstructive Pulmonary Disease: Joint recommendations of ICS and NCCP India, Special issue The Indian Journal of Chest Diseases and Allied Sciences, Vol 56 [Internet] – 2014. [cited 2016 Jul 5] Available from: <http://medind.nic.in/iae/t14/s1/iaet14s1.pdf>
2. World Health Organization (WHO) Guidelines and factsheets for COPD. [cited 2016 Jul 5] Available from: http://www.who.int/topics/chronic_obstructive_pulmonary_disease/en/



6. BRONCHIECTASIS

1. Definition

Bronchiectasis is chronic, irreversible dilation and distortion of the bronchi caused by inflammatory destruction of the muscular and elastic components of the bronchial walls. It may be focal or diffuse. It is categorized as cylindrical, tubular, varicose or cystic.

2. Aetiology

Conditions associated with the development of bronchiectasis

2.1. Post infection

- Bacterial pneumonia
- Tuberculosis
- Pertussis
- Measles
- Influenza
- Fungus

2.2. Proximal airway obstruction

- Foreign body aspiration
- Benign airway tumours

2.3. Abnormal host defence

- Ciliary dyskinesia (Kartagener's syndrome)
- Alpha 1 antitrypsin deficiency

2.4 Immunodeficiency

- HIV, Hypogammaglobinaemia

2.5 Genetic disorders

- Cystic fibrosis

3. Symptoms

- Cough with production of large quantities of purulent and often foul-smelling sputum.

- Fever, generalized malaise, weight loss, haemoptysis
- Dry bronchiectasis; usually involve the upper lobes
- Recurrent pneumonia

4. Signs

- Early phases or dry variety: Normal
- Severe or secondary infection: Persisting crackling rales in the same part of lung
- Later stage: Emphysema and Cor Pulmonale.
- Moist crackles at lung bases
- Halitosis, skin pallor

5. Laboratory tests

- Sputum for Gram stain, C&S, and acid-fast bacteria (AFB)
- CBC with differential (leucocytosis with left shift, anaemia).
- Serum protein electrophoresis to evaluate for hypogammaglobinaemia.
- Antibody test for Aspergillosis.
- Sweat test in patients with suspected Cystic Fibrosis.

6. Evaluation

6.1. Chest x-ray:

Increase in size and number of bronchovascular markings (quiet nonspecific). Presence of "Tram-track" indicates dilated airways suggestive of bronchiectasis.

6.2. CT or HRCT:

High sensitivity and specificity

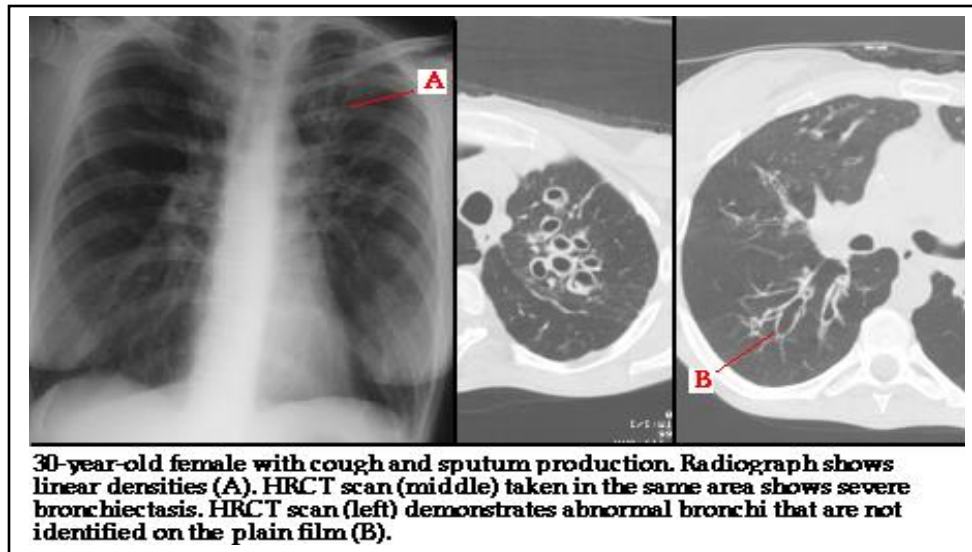


Figure 6.1- HRCT showing Bronchiectasis

Tram track sign: The bronchial wall is thickened and visible; the bronchi lose the trend of narrowing from proximal end to distal end.

Signet ring sign: Dilated bronchi appear as ring structures with internal diameters greater than those of them accompany pulmonary artery branches.

7. Differential diagnosis

Differentiate from:

Chronic bronchitis, Lung abscess, Tuberculosis, Congenital pulmonary cyst.

8. Treatment

8.1. Non-Pharmacologic Therapy

- Chest physiotherapy helps the Postural drainage and enhances removal of respiratory secretions.
- Adequate hydration, mucolytic administration

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



8.2. Acute General Treatment

- Supplement oxygen for hypoxemia.
- The choice of antibiotics should be accurate by the results of sputum culture and drug sensitivity test.
- Empirical therapy - Antipseudomonal antibiotics, Ciprofloxacin and Gentamicin or
- Antibiotic therapy is based on the results of sputum, Gram stain, and C&S
- Bronchodilators are useful in patients with demonstrable airflow obstruction.

8.3. Chronic Treatment

- Avoidance of tobacco.
- Maintenance of proper nutrition and hydration
- Prompt identification and treatment of infections.
- Pneumococcal vaccination and annual influenza vaccination

7. LUNG ABSCESS

1. Introduction

- A lung abscess is an infection of the lung parenchyma resulting in a necrosis and cavitation of lung.
- Commonest site is right lung and involves the posterior segment of the right upper lobe, the superior segment of the lower lobe, or both
- The bacterial infection may reach the lungs in several ways that most common is aspiration of oro-pharyngeal contents.

2. Microbiology

- The most common anaerobes are *Peptostreptococcus*, *Bacteroids*, *Fusobacterium species* & *Microaerophilic streptococcus*.
- Other organisms that may infrequently cause lung abscess include *Staphylococcus aureus*, *Streptococcus pyogenes*, *Streptococcus pneumoniae* (rarely), *Hemophilus influenza*, *Actinomyces species*, *Nocardia species*, & Gram negative bacilli (*Pseudomonas*)
- *Mycobacterial tuberculosis* is a common cause.
- Non-bacterial pathogens may also cause lung abscesses. Parasites [*Paragonimus*, *Entamoeba*] Fungi [*Aspergillus*, *Cryptococcus*, *Histoplasma*, *Blastomyces*, *Coccidioides*] seen in immunocompromised patients.

3. Clinical presentation

- Symptoms are generally insidious and prolonged, occurring for weeks to months
- Fever, chills, and sweats
- Cough
- Sputum production (purulent with foul odour)
- Pleuritic chest pain
- Haemoptysis
- Dyspnoea
- Malaise, fatigue, and weakness
- Tachycardia and Tachypnea

Dullness to percussion, whispered pectoriloquy, and bronchophony

4. Lab Studies

- CBC- leucocytosis
- Sputum for gram stain, culture & sensitivity.
- If T.B. is suspected, acid fast bacilli stain & mycobacterial culture is requested.

- Blood culture may be helpful in establishing the aetiology.
- Obtain sputum for ova & parasite whenever a parasitic cause for lung abscess is suspected.

5. Radiological imaging

5.1. CXR

Lung abscesses are most commonly found in the posterior segment of the right upper lobe. They appear as irregularly sharp cavity with an air-fluid level inside.



Figure 7.1- X-Ray Showing Lung Abscess

5.2. CT Scan

An abscess is rounded radio-lucent lesion with a thick wall & ill-defined irregular margins.



Figure 7.2- CT Showing Lung Abscess

6. Medical care

Antibiotic therapy:

- IV Clindamycin 600 mg 3 times a day, till afebrile then oral 300 mg four times a day for 7 days.

- Alternative is IV Amoxicillin / Clavulanate 1.2gm thrice a day or I.V. Ampicillin / Sulbactam 1.5gm thrice a day then to oral Amoxicillin / Clavulanate for 7 days, with I.V. Metronidazole 500 mg 8 hourly is an effective drug against anaerobic bacteria for 7 days.
- In hospitalized patients who have aspirated and developed a lung abscess, antibiotic therapy should include coverage against *Staphylococcus aureus* and *Enterobacter* and *Pseudomonas* species and as per the identified pathogen.
- Cephalosporin that have gram-positive, gram-negative, and anaerobic coverage, may be used when a polymicrobial infection is suspected as cause of lung abscess.
- Duration of therapy is generally for 4-6 weeks to as long as 14 weeks.

Antibiotic treatment should be continued until the chest radiograph has shown either the resolution of lung abscess or the presence of a small stable lesion.

Patients with poor response to antibiotic therapy include bronchial obstruction with a foreign body or neoplasm or infection with resistant bacteria, Mycobacteria, or fungi.

7. Complication

Rupture into pleural space causing empyema, pleural fibrosis, bronchopleural fistula.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015
4. National Treatment Guidelines for antimicrobial use in infectious diseases – Version 1.0 (2016), National Center for Disease Control, Ministry of Health and Family Welfare. [Cited 2016 July 7]
Available from: http://www.ncdc.gov.in/writereaddata/linkimages/AMR_guideline7001495889.pdf

Further reading

1. Kuhajda A, Zarogoulidis K, Isirgogianni K, et.al. Lung abscess – etiology, diagnostics and treatment options, Ann Transl Med [Internet].2015 Aug; 3(13): 183. [Cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4543327/>



8. PNEUMOTHORAX

1. Definition

The presence of air within the pleural cavity.

2. Classification

2.1. Spontaneous

- Primary.
- Secondary.

2.2. Traumatic

- Non-iatrogenic.
- Iatrogenic.

3. Spontaneous Pneumothorax

Pneumothorax occurring in the absence of trauma may be described as spontaneous.

Presents in 3 ways:

- **Open** Pneumothorax- air moves freely in & out of pleural space during breathing.
- **Closed** pneumothorax- no movement of air from the pleural space due to closure of the communication, air slowly gets absorbed & the lung re-expands.
- **Tension** pneumothorax- a check – valve mechanism is produced; this allows air to enter pleura & accumulate to raise the intrapleural pressure above the atmospheric pressure and leads to compression on lung & shifting of mediastinum to opposite side.

3.1. Primary spontaneous pneumothorax

- Commonly occurs in healthy subjects with no h/o of pre-existing lung disease.
- Disease of young adult.

3.2. Secondary spontaneous pneumothorax

- Coexisting structural or functional abnormality in the lung.
- Stature.

4. Causes of Pneumothorax

4.1. Primary spontaneous

- Apical Blebs (90%)

- Stature

4.2. Secondary spontaneous - less common

- Chronic bronchitis & emphysema, (35%).
- Asthma (0.8).
- Suppurative pneumonia like *Staphylococci*, *Klebsiella*, HIV (2-4%).
- TB of lungs.

4.3. Traumatic Iatrogenic

- Paracentesis thoracis (28%).
- Central venous cannulation, (22%).
- Barotrauma (mechanical ventilation).
- Tracheostomy.

4.4. Traumatic Non-Iatrogenic Pneumothorax

- Open & closed chest injury, (road traffic accident).
- Stab or gunshot wounds.
- Rib fractures.

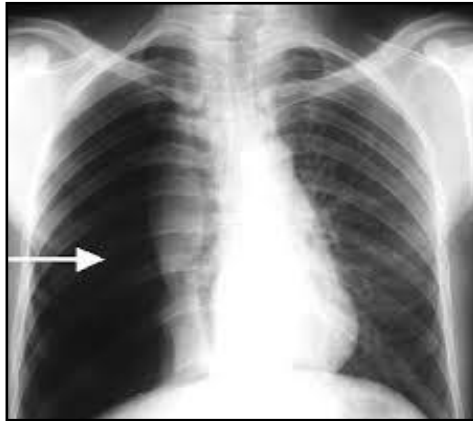
5. Symptoms

- Small pneumothorax is asymptomatic.
- Chest pain - Sharp unilateral associated with shortness of breath is commonest presentation.
- Sharp & stabbing Chest pain exacerbated by deep inspiration & postural change.
- Anxious, restless, tachypnoeic, struggling for breath, rapid low volume pulse & hypotension.
- May large pneumothorax produce respiratory distress, signs of shock.
- Closed pneumothorax –usually does not produce severe symptoms.
- Tension pneumothorax –medical emergency.

6. Physical signs

- Small pneumothorax – Difficult to detect on physical examination.
- Absence or diminished breath sounds on affected side.
- Chest movement diminished on affected side
- Decreased vocal fremitus.
- Hyper resonant percussion notes.

- Ipsilateral enlargement of chest due to decrease elastic recoil of the collapsed lung.
- Shift of mediastinum on opposite side.
- Increased JVP.
- Respiratory distress.
- Diaphoresis
- Cyanosis.
- Hypotension.
- Crepitus is seen if there is associated subcutaneous emphysema.



7. Diagnosis

ECG- Diminished anterior QRS amplitude.

Radiographic appearances.

X-ray chest-sharply defined lung edge convex outwards separated from chest wall by translucency with no lung markings & mediastinal displacement depending upon the extent of pneumothorax.

Plain X-ray film of the chest showing hyperlucency without any lung markings on right side of the chest (Pneumothorax)

Figure 8.1 – X-Ray Showing Pneumothorax

8. Differential diagnosis

- Transmural myocardial infarction-ECG changes & left sided pneumothorax changes resolve once re-expansions.
- Emphysema confused with pneumothorax but x-ray is main diagnostic tool.
- Massive emphysematous bulla or congenital cyst, when ruptures may be confused with pneumothorax but previous x-ray, lateral decubitus view is helpful in differentiating upper lobe bulla/cyst.

9. Complications

Recurrence, Haemopneumothorax, Pyopneumothorax and Respiratory failure –when tension pneumothorax present.

10. Treatment

Treatment depends on cause, size, degree of physiological derangement. Primary pneumothorax-smaller without pleural air leak may resolve spontaneously.

- If pneumothorax small but patient mild symptomatic, admit the patient & administer high-flow oxygen, resulting nitrogen gradient will speed resorption.
- If pneumothorax larger than 15% to 20% or more than mildly symptomatic, insert a thoracostomy tube.
- Secondary pneumothorax - Patients are symptomatic & require lung re-expansion.
- Often bronchopleural fistula persists & larger thoracostomy tube & suction are required.
- Iatrogenic pneumothorax -- Due to barotrauma from mechanical ventilation always persistent air leak & should be managed with a chest tube & suction.
- Tension pneumothorax--decompress the affected hemithorax immediately with a 14-gauge needle attached to a fluid filled syringe, release of air with clinical improvement confirms the diagnosis. Seal chest wound with an occlusive dressing & arrange the placement of a thoracostomy tube.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



9. LUNG CANCER

1. Definition

Uncontrolled growth of malignant cells in one or both lungs and tracheo-bronchial tree.

2. Epidemiology

- Lung cancer was initially thought to be infrequent in India.
- Rare below age 40.
- Increasing until age 80 after which rate tapers off.
- Probability developing lung cancer approximately 8% & 6% in males & females.

3. Risk factors

- Smoking: Smokers have 10 fold or greater increase in risk.
- Radiation Exposure.
- Family history: 1st degree relatives – 2 to 3-fold increase risk.
- Environmental/ Occupational Exposure: Asbestos, Arsenic, Chromium, Mustard gas, Nickel, Radon, Polycyclic Hydrocarbons.
- Scarring.

4. Signs and Symptoms

Table No. 1

Signs & symptoms	Range of frequency
Cough (persistent for > than 2 weeks)	8-75%
Weight loss	0-68%
Dyspnoea	3-60%
Chest pain (poorly localized deep chest discomfort)	20-49%
Haemoptysis (seen more in central tumours)	6-35%
Bone pain	6-25%
Clubbing	0-20%
Fever	0-20%
Weakness	0-10%
SVC syndrome	0-4%
Dysphagia	0-2%
Wheezing & stridor	0-2%

5. Types of Lung Cancer

5.1. Non-small cell carcinoma

- Squamous Cell Carcinoma (25-40%, epidermoid derived).
- Adenocarcinoma (25-40%, bronchial, acinar, papillary, solid, bronchoalveolar).

- Large cell Carcinoma – 10-15%, undifferentiated, giant or clear cell

5.2. Small cell Carcinoma (20-25%, oat, intermediate cell)

6. Diagnosis

History and Physical exam: Physical signs like clubbing, lymphadenopathy, hoarseness of voice (vocal cord palsy on indirect laryngoscopy) along

with chest x-ray signs of mass lesion and collapse which are pointers towards the diagnosis of non resolving pneumonia in an elderly individual or in smoker, lung malignancy needs to be excluded.

6.1. Diagnostic tests

- Chest X-ray – Identifies nodules usually >1cm
- HRCT Chest - Mass lesion along with its morphology and vascularity can be better visualised
- Bronchoscopy - It is the most useful investigation.
- Pleural tapping - Cytological examination of the pleural fluid is necessary to establish the diagnosis.

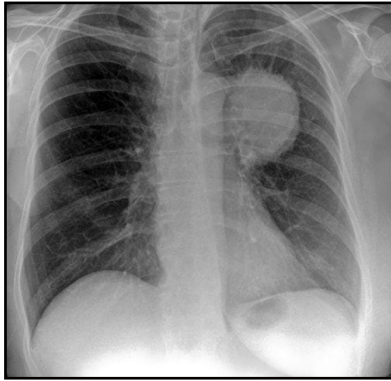


Figure 9.1: Chest X ray showing Left Para hilar lung mass

6.2. Staging tests

- CT chest/abdomen.
- Bone scan.
- Bone marrow aspiration.

malignancy.

- PET scan – helpful in staging to determine degree of metastases
- MRI/CT brain – useful in looking at CNS involvement.

7. Clinical findings suggestive of metastatic disease

- Lymphadenopathy [>1 cm]
- Bone tenderness
- Hepatomegaly [>13 cm span]
- Focal neurologic signs, papilledema
- Soft tissue mass
- Haematocrit < 40% in men, < 35% in women.
- Elevated alkaline phosphatase, GGT, SGOT, calcium levels.
- Oesophageal compression – Dysphagia.
- Laryngeal nerve paralysis – Hoarseness.
- Symptomatic nerve paralysis - Horner's syndrome.
- Cervical/thoracic nerve invasion - Pancoast tumour.
- Lymphatic obstruction - Pleural effusion.
- Vascular obstruction - SVC syndrome.
- Pericardial / cardiac extension - Effusion, tamponade.

8. Treatment

Medical, surgical and radiation therapy modalities considered according to the type and stage of the cancer.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Malik PS, Raina V. Lung Cancer – Prevalent trends and emerging concepts, Indian J Med Res [Internet]. 2015 Jan; 141(1): 5–7. [cited 2016 Jul 5]

Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4405940/>



10. PULMONARY EMBOLISM

1. Introduction

Thrombosis that originates in the venous system and embolizes to the pulmonary arterial circulation. DVT in veins of leg above the knee (>90%), DVT elsewhere (pelvic, arm, calf veins, etc.), Cardiac thrombi.

Risk factors - Obesity, smoking, OC pills, surgery, trauma, malignancy, thrombophilia.

2. Signs and symptoms

Massive PE – Severe dyspnoea, hypotension, cyanosis, tachycardia.

Moderate PE- Cough, pleuritic pain, haemoptysis, fever. Other signs are anxiety agitation, raised JVP, loud P2, right ventricular heave.

Sometimes patients are asymptomatic.

Differential Diagnosis - Myocardial Ischemia - Angina, Myocardial Infarct, Pneumonia, Pericarditis, Congestive Heart Failure etc.

3. Investigations

- **Chest x-ray**- Atelectasis, Westermark sign – increased lucency in area of embolism, Hampton's hump - peripheral wedge shaped density above diaphragm, pleural effusion.
- **ECG**- Sinus tachycardia, Classic S1Q3T3 pattern, signs of RV Strain-R in V1 V2 with t inversion.
- **ABG** - Hypoxia, hypocapnia, respiratory alkalosis Normal does not rule out PE.

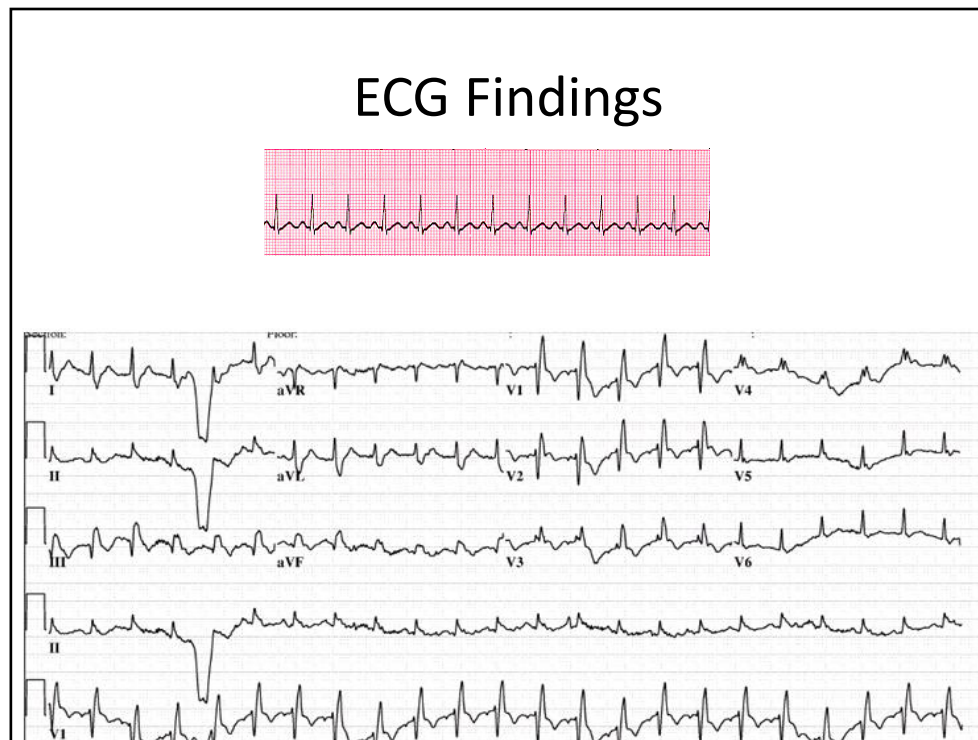


Figure-10.1: ECG changes seen in Pulmonary Embolism

- D-dimer is raised (high sensitivity but poor specificity).
- CT Pulmonary Angiography is gold standard.
- V/Q Lung scan identifies areas of lung that are ventilated but not perfused.
- US Venous Doppler to detect deep venous thrombosis.

4.Treatment

- Thrombolysis in massive PE. Inj. Streptokinase 2.5 lakhs bolus, then 1.0 lakh per hour.
- Unfractionated heparin 80 U/kg bolus, then 18 u/kg/hour with goal of PTT 46-70 secs.
- LMWH- Inj. Enoxaparin 1 mg/kg 12 hourly. To overlap with warfarin and to continue it for 3-6 months.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Dalal J J, Amin P. Management of Acute Pulmonary Embolism, Journal of The Association of Physicians of India Vol. 63 [Internet]. December 2015. [cited 2016 Jul 5]
Available from: http://www.japi.org/december_2015/07_oa_management_of_acute.pdf



11. HYPERTENSION

Blood pressure is lateral pressure exerted by column of blood on the walls of artery when it flows through it.

1. Definition

- **Hypertension** - Defined as any one of the following:
- Systolic blood pressure ≥ 140 mmHg and / or Diastolic blood pressure ≥ 90 mmHg,
- Patient taking antihypertensive medications.
- **Essential HT:** When the cause is not known (90 to 95 % cases).
- **Secondary HT:** Specific organ dysfunction is detected (5 to 10 % cases).

2. Symptoms

- Often asymptomatic (silent killer).
- Due to Elevated pressure: Headache (Occipital), vomiting, giddiness, breathlessness, palpitations.
- Due to Vascular disease: Cerebrovascular accident, Acute Myocardial Infarction.
- Due to Underlying disease: symptoms of underlying organ affected.

3. Signs

- Blood vessels – Bruits over carotid.
- Abdominal Bruit – To rule out Renovascular hypertension.
- Spells of sweating, tachycardia – Pheochromocytoma.
- Tremors, neck swelling– Thyroid Disorder.
- Snoring, Daytime somnolence – Obstructive sleep, Apnoea.
- Asymmetry of pulses, Radiofemoral delay– Takayasu Disease, Coarctation of Aorta.

4. How to investigate?

4.1. Accurate BP measurement

The average of two or more seated blood pressure during each of two or more outpatient visits.

4.2. Basic investigations for initial evaluation: Always includes

- Haematocrit / Hb.
- Serum BUN, Creatinine.
- Serum potassium.
- Fasting blood sugar.
- Total cholesterol, S Triglycerides.
- Urine analysis for albumin, blood, glucose.
- ECG for left ventricular hypertrophy and ST-T changes.
- Fundoscopy for HT retinopathy.

4.3. Investigations usually included depending on cost & other factors

- i. TSH
- ii. Complete blood count.
- iii. HDL, LDL cholesterol.
- iv. Serum calcium, phosphorus.
- v. Serum Uric Acid.
- vi. Chest X-ray - Cardiomegaly.
- vii. USG renal system - Cortical scarring, shrunken size, obstructive uropathy.
- viii. Echocardiography – LVH, Diastolic dysfunction, Ejection Fraction.

4.4. Special tests to screen for secondary HT (only in indicated cases)

- Renovascular disease: Renal Doppler, MR angiography, DTPA scan.
- Renal parenchymal: Kidney biopsy.
- Pheochromocytoma: 24-hour urine metanephrine & catecholamine.
- Cushing's syndrome: Serum cortisol, Dexamethasone suppression test.

- Aldosteronism: Plasma aldosterone: renin ratio.

4.5. Annual tests in hypertensive subjects

- Haemogram
- Renal profile
- Lipid profile
- Urine analysis

- ECG
- Fundoscopy.

5. How to treat?

5.1. Goal BP – 150/90 mm Hg in elderly and 140/90 mmHg in all others (including DM, CKD).

5.2. Life style changes

Table 1: Lifestyle changes to manage hypertension

Modification	Aim
Weight reduction	Attain and maintain BMI < 25 kg/m ²
Adopt DASH eating plan	Diet rich in fruits, vegetables & low-fat dairy products with reduced content of saturated and total fat
Dietary sodium reduction	< 4.8-7.4 g NaCl/day
Physical activity	Regular aerobic activity- brisk walking for 30 min/day

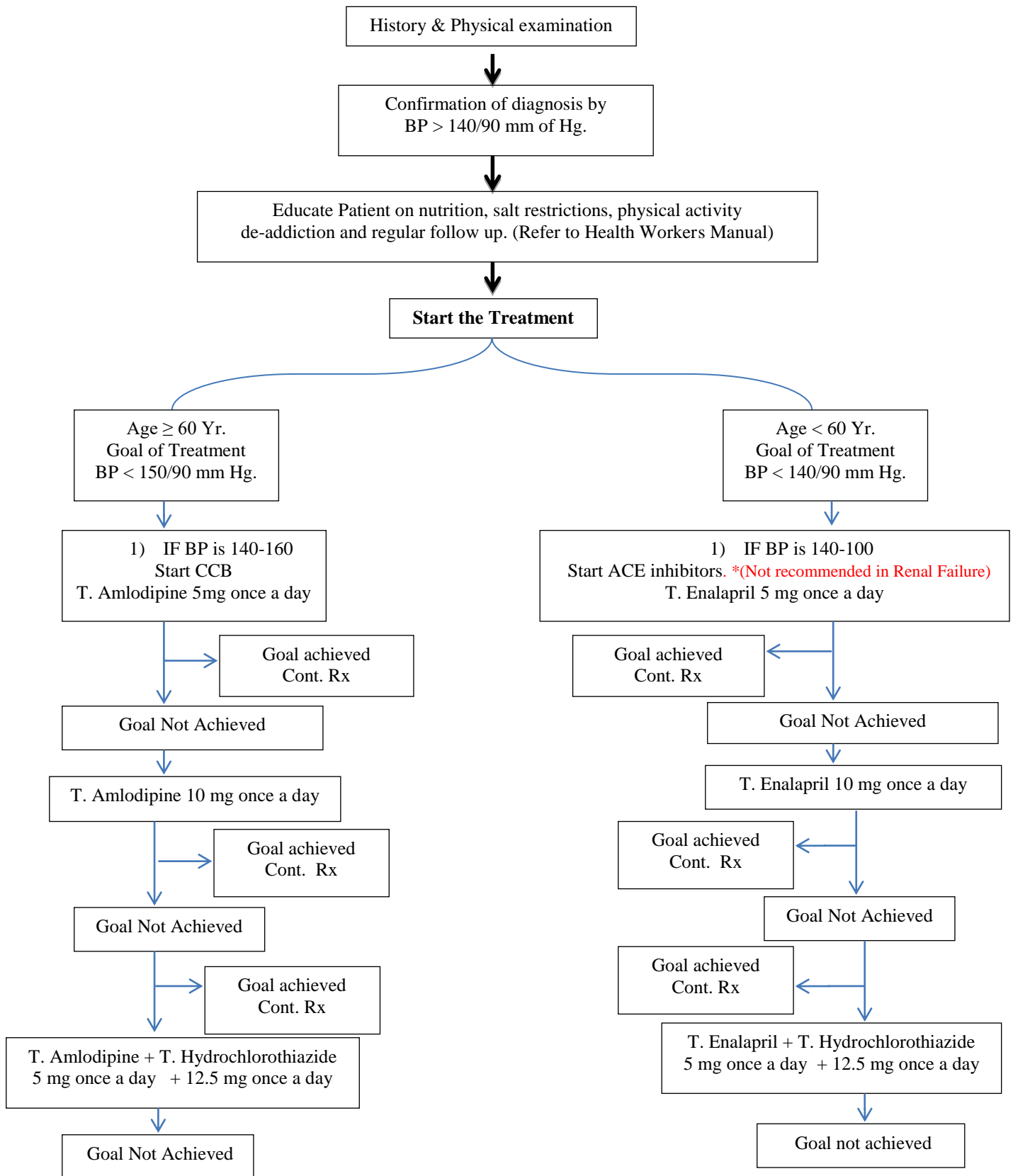
5.3. Guidelines for management

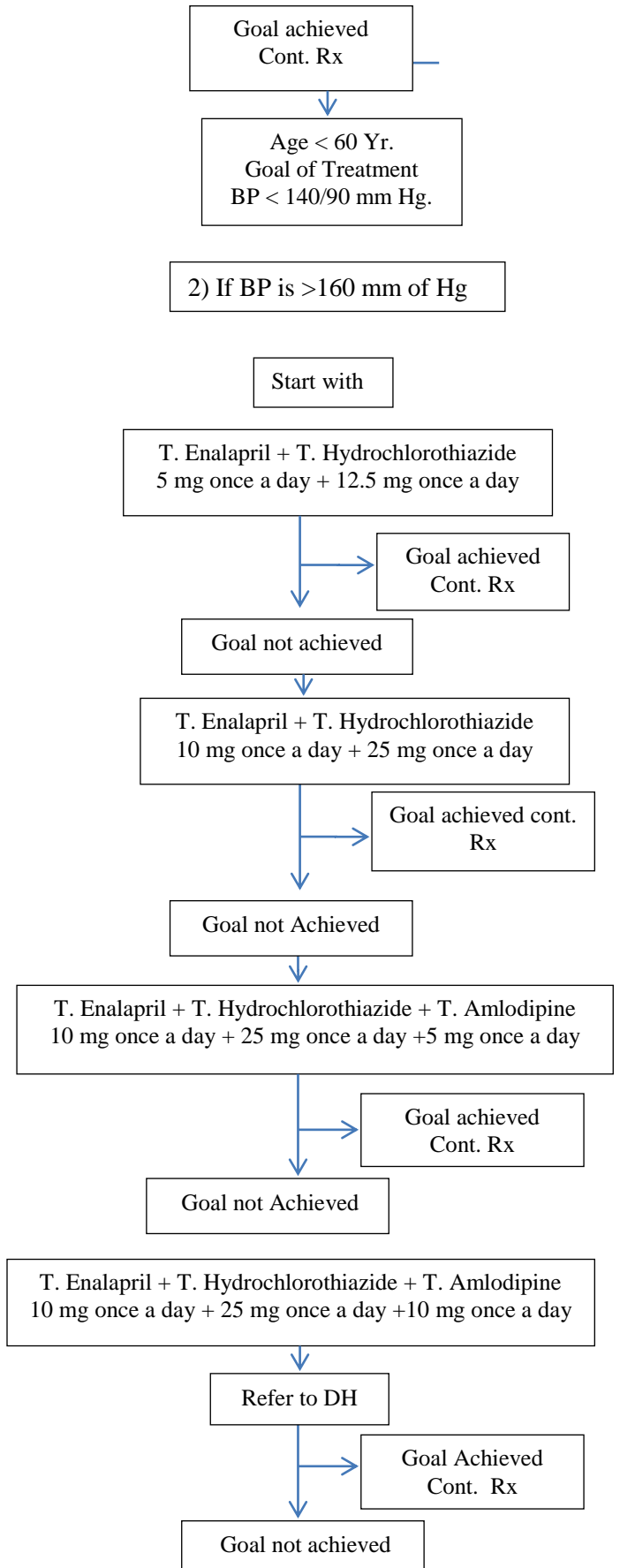
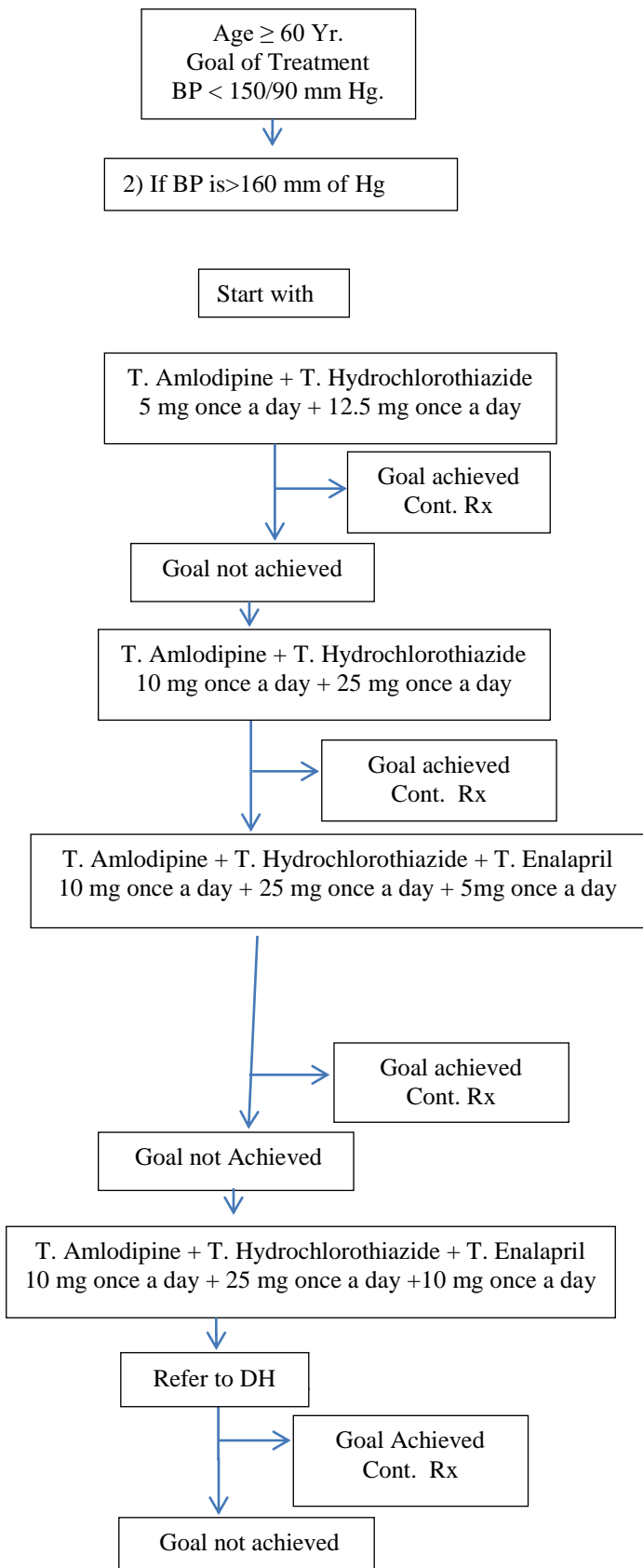
Table 2: Hypertension management guidelines

BP Classification	SBP mmHg	DBP mmHg	Lifestyle Modification	Drug Therapy
Normal	<120	and <80	Encourage	No
Prehypertension	120-139	or 80-89	Yes	No
Stage 1 Hypertension	140-159	or 90-99	Yes	Single Agent
Stage 2 Hypertension	≥ 160	or ≥ 100	Yes	Combo

5.4. Treatment protocol

Figure 11.1: Treatment Protocol for HT Age > 30 Years





- If age < 30 years & BP > 140/90 - immediately refer to DH (District hospital).
- When patient is on drug if systolic BP is < 100 mm of Hg withhold the drugs (Anti-Hypertensive drugs) and refer to DH.
- BP >160/100, Start min 2 Anti-Hypertensives
- If initial BP is > 200/100 refer to DH after a shot of Inj. Frusemide 60 mg stat.
- If Sr. Creatinine > 1.5mg% refer to DH.

5.5. Hypertension in Pregnancy

- Tablet Methyl Dopa – 500 mg-1000 mg/Day in three divided dose.
- Tablet Nifedipine Extended Release Preparation 30 - 60 mg OD/BD.
- Other drugs that can be given - Labetalol, Hydralazine, Beta Blocker.
- ACE inhibitors and ARBS avoided.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. India. Ministry of Health and Family Welfare. NHM Standard Treatment Guidelines for Hypertension. [cited 2016 Jul 5]
Available from: <http://nrhm.gov.in/images/pdf/guidelines/nrhm-guidelines/stg/hypertension.pdf>
2. Hypertension guidelines. World Health Organization (WHO) [Internet]. [cited 2016 Jul 5]
Available from: <http://www.who.int/topics/hypertension/en/>



6. Vigilance for End Organ Damage in hypertensive patients

- Congestive heart failure.
- IHD.
- Chronic Kidney Disease.
- Stroke.
- Hypertensive Retinopathy.

7. When to refer?

- Annual Work-up of known Hypertensive subjects.
- Young Hypertensive/ secondary HT.
- Resistant Hypertension (Target BP not achieved with 3 drugs including diuretics).
- Pregnant subjects.
- Hypertensive emergencies (BP > 180/ 110 mm Hg with e/o end organ damage).

12. HEART FAILURE

1. Introduction

Heart failure (HF), often referred to as congestive heart failure (CHF), occurs when the heart is unable to pump sufficiently to maintain blood flow to meet the body's needs

2. Types of heart failure

The heart consists of two distinct parts; right and left receiving blood from different venous system and perfusing distinct parts of the body; the right heart perfusing Heart Failure is a condition where the heart is unable to perform its functions optimally; leading to decreased perfusion of the tissues supplied by the lungs for gaseous exchange and the left heart perfusing the systemic circulation.

Hence there are two types of heart failure.

- Right heart failure.
- Left heart failure.

2.1 Right Heart Failure

The right receives blood from the systemic veins and pumps blood into the pulmonary artery through the pulmonary valve.

Hence the cause of Right heart failure would be; increased pressure in the pulmonary artery (Pulmonary Hypertension) or pulmonary valve stenosis.

2.1.1. Causes of Pulmonary Hypertension

- i. Chronic Obstructive Pulmonary Disease (COPD) seen commonly in smokers. Cor Pulmonale.
- ii. Mitral valve stenosis seen in Rheumatic heart diseases.

2.1.2. Clinical features of Right heart failure

As the right heart receives blood from the systemic veins, the main clinical features involves features of congestion in the systemic venous system.

- i. Raised Jugular Venous Pressure with engorged superficial neck veins.
- ii. Tender Hepatomegaly: Painful, soft liver palpable in the right hypochondrium.
- iii. Dependent Oedema: Pitting oedema demonstrable on the shin of the tibia and ankles.

2.1.3. When to Suspect Right Heart Failure

- i. Right heart failure should be suspected if the clinical features are present like swelling in the legs and engorged neck vein.
- ii. Also, once the features can be demonstrated on clinical examination, the underlying causes of right heart failure should be looked for; e.g. History of smoking and signs of COPD, or signs of Mitral Valve Disease.

2.1.4. Investigations for Right Heart Failure

The diagnosis of Right Heart failure is a clinical one. Investigations are basically to diagnose the underlying cause of right heart failure.

2.1.5. Treatment of Right Heart Failure

Right heart failure is not a medical emergency and does not cause immediate fatality. There is no direct treatment of Right Heart failure. This condition is alleviated by effective treatment of the cause of failure.

Hence attempts should be made to manage COPD effectively, like:

- Antibiotics in acute exacerbation.
- Effective bronchodilation using inhalers and oral long acting Xanthines (Deriphyllin).
- Low flow oxygen inhalation which is the treatment of choice for Cor Pulmonale.
- Effective treatment of Mitral stenosis including Low salt diet, Diuretics, and Digoxin.

2.1.6. When to Refer a Case of Right Heart Failure to Higher Centre

A case of right heart failure needs to be referred for specialized treatment only for management of non-responsive chronic obstructive airway disease. In case, the cause of right heart failure is Mitral valve disease which is not amenable to medical management, such cases should be referred to a cardiac centre for definitive management of the condition.

2.2. Left Heart Failure: [Pulmonary Oedema]

The left heart receives blood from the pulmonary vein and pumps blood in the systemic circulation.

2.2.1. Causes of left heart failure

- i. Systemic Hypertension is the commonest cause for left ventricular failure

- ii. Valvular heart diseases: Aortic stenosis, Aortic regurgitation and Mitral regurgitation are all causes of acute left heart failure.
- iii. Myocardial infarction involving significant part of left ventricle can cause L.V. failure.

2.2.2. Clinical features

- i. Sudden onset breathlessness is the main symptom.
- ii. There may be continuous cough with pink frothy sputum.
- iii. The patient will be very anxious and restless.
- iv. The patient will choose to be in the sitting position and unable to lie down.
- v. On auscultation of the chest, crepitation will be heard most prominently in the bases.
- vi. General examination will reveal high blood pressure which is the commonest cause.
- vii. Such patients are likely to present with cyanosis. (Bluish discoloration of tongue, lip, oral mucosa, fingers and toes).

2.2.3. Investigations

Left ventricular failure is a medical emergency and time should not be wasted in any investigation.

However, after stabilizing the patient investigations may be carried out to find the underlying cause.

2.2.4. Treatment

Left heart failure or Pulmonary oedema is a medical emergency and speed of administration of treatment is of paramount significance.

- i. The patient should be treated in the propped up position using a backrest or raised head end of the bed.
- ii. High flow Oxygen inhalation should be given.
- iii. Sedation preferably with Morphine 10 mg I.V or Pentazocin 30 mg is the management of choice.
If these powerful narcotics are not available, any form of sedation will be of help.
- iv. Intravenous Frusemide (Lasix) 60 mg I.V should be administered.
- v. Acute reduction of blood pressure must be done if the cause of left heart failure is Hypertension: I.V. infusion of Nitroglycerin (5 microgram/ kg) by BP monitoring, should be tried for the purpose.
- vi. Anti-platelets, statins to be continued.

2.2.5. When to Refer the Patient

Treatment of left heart failure must be attempted at the peripheral level. Once stabilized the patient may be referred to higher centre for management of the underlying cause.

Failure of the patient's breathlessness to resolve, may require, endotracheal intubation or non-invasive positive pressure ventilation and patient should be referred to such a centre if facilities for the same is not available.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Huffman MD, Prabhakaran D. Heart Failure: Epidemiology and prevention in India, Natl Med J India [Internet]. 2010 Sep-Oct; 23(5): 283–288. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3913650/>



13. ISCHAEMIC HEART DISEASE AND ACUTE CORONARY SYNDROME

1. Introduction

1.1. Ischemic heart disease (IHD)

It is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium. It typically occurs when there is an imbalance between myocardial oxygen supply and demand.

1.2. Patients with ischemic heart disease fall into two large groups.

- i. Patients with chronic coronary artery disease (CAD) who most commonly present with stable angina.
- ii. Patients with acute coronary syndromes (ACSs).
 - a. Patients with acute myocardial infarction (MI) with ST-segment elevation on their presenting electrocardiogram (ECG).
 - b. With unstable angina (UA) and non-ST-segment elevation MI.

2. Stable Angina

2.1. Clinical Presentation

Chest pain – Retrosternal, dull aching, constricting or burning, radiating to neck, jaw, shoulders or arms usually precipitated by exertion or stress and relieved by rest or nitrates. Angina is crescendo and decrescendo in nature and typically last for 2-5 minutes.

The physical examination is often normal in patients when asymptomatic. Examination during an anginal attack and transient left ventricular failure, there can be a third, fourth heart sound, and systolic murmur of mitral regurgitation.

2.2. Investigations

CBC, Urine, Blood sugar, Lipid profile, and X-ray chest PA view may be helpful.

ECG taken at that time may show ST elevation or depression or T inversion. In between anginal episodes, the ECG may be normal.

A treadmill stress test would confirm angina in over 95% of these cases in referral hospital.

Echocardiography to measure left ventricular function and rule out segmental dyskinesia suggestive of earlier myocardial infarction.

2.3. Treatment

Daily exercise, Stop smoking, Dietary modification – low cholesterol, low fat diet with high roughage.

Control of hypertension, Diabetes Mellitus and Dyslipidaemia.

Drug Treatment

- Tab Aspirin 75 mg once daily.
- Tab Clopidogrel 75 mg per day.
- Tab Atorvastatin 40 mg per day.
- Nitrates -Sublingual Glyceryl Trinitrate 300-500 microgram t.i.d. or Isosorbide Dinitrate 10 mg thrice a day. If there is headache lower dose of 5 mg twice or thrice daily can be tried.
- Beta-blockers - Tab Metoprolol 50 –200 mg / day (PO in divided dose).
- Potassium channel activators- Nicorandil 10mg BD.
- Calcium channel blockers like Tab Amlodipine 5-10 mg once a day.

Refer for coronary angiogram and revascularisation therapy.

3. Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction

UA is defined as angina pectoris or equivalent ischemic discomfort with at least one of three features:

- a. It occurs at rest (or with minimal exertion), usually lasting >10 minutes.
- b. It is severe and of new onset (i.e., within the prior 4–6 weeks); and/or
- c. It occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previously).

3.1. Clinical Presentation

Chest pain, typically located in the substernal region or sometimes in the epigastrium that radiates to the neck, left shoulder, and/or the left arm.

3.2. Electrocardiogram

ST-segment depression, transient ST-segment elevation, and/or T-wave inversion.

3.3. Cardiac Biomarkers

CPK-MB and troponin raised in Non-ST segment elevated MI.

3.4. Treatment

- Bed rest.
 - Sublingually Nitroglycerin .3-.6 mg stat can repeat after 5 min - 3 Doses.
 - If symptoms persist, intravenous Nitroglycerin infusion at dose of 5-10 microgram/min. once pain has resolved Oral Isosorbide Dinitrate 10 mg BD can be given.
 - Aspirin initial dose of – 325 mg followed by 150 mg/day lifelong.
 - Clopidogrel - Loading dose of 300 mg followed by 75 mg/ day for 2 years.
 - Tab Atorvastatin 40 mg / day lifelong.
 - Intravenous beta blocker – Metoprolol 5-15 mg over 5 mins followed by tab Metoprolol 50-100 mg/day in divided doses with BP check.
 - Unfractionated Heparin (UFH) bolus 60–70 U/kg (maximum 5000 U) IV followed by infusion of 12–15 U/kg per hour (initial maximum 1000 U/h) titrated to A PTT 50–70 s.
- or**
- Enoxaparin 1 mg/kg SC every 12 hr.

4. ST segment elevated Myocardial Infarction

4.1. Clinical Presentation

- Chest Pain- commonly occurs at rest, severe, and lasts longer. Typically, the pain involves the central portion of the chest and/or the epigastrium, and, on occasion, it radiates to the arms, abdomen, back, lower jaw, and neck.
- Sudden-onset breathlessness, Sweating, loss of consciousness, a confusional state.
- Sensation of profound weakness, the appearance of an arrhythmia, an unexplained drop in arterial pressure.

4.2. Laboratory Findings

- CBC, Blood sugar, lipid profile, X-ray chest.
- Cardiac markers - CPK-MB elevated.
- Troponin T and I released within 4-6 hours and elevated for 2 weeks.
- Electrocardiogram Convex ST – segment elevation with either peaked upright or inverted T waves. Q waves if necrosis occurs.
- Echocardiogram - regional wall motion abnormality.

4.3. Complications

- Arrhythmias.
- Acute Heart failure.
- Rupture of papillary muscle.
- Embolism leading to stroke.
- Ventricular remodelling.
- Ventricular aneurysm.

4.4. Treatment

- Bed rest.
- Oxygen therapy - 2-4 L/min.
- Sublingually Nitroglycerin .3-.6 mg stat can repeat after 5 min - 3 Doses if symptoms persist, intravenous Nitroglycerin infusion at dose of 5-10 microgram/min. once pain has resolved Oral Isosorbide Dinitrate 10 mg b.i.d can be given.
- Aspirin 325 mg and then 150 mg once a day.
- Clopidogrel 300 mg and then 75 mg BD.

- Atorvastatin 80 mg and then 40 mg HS.
- Reperfusion therapy: If presenting within 12 hours of chest pain with ECG showing ST elevation > 1 mm then give Inj. Streptokinase 1.5 million units over 1 hour (contraindications- a history of cerebrovascular haemorrhage at any time, a non-haemorrhagic stroke or other cerebrovascular event within the past year, marked hypertension at any time during the acute presentation, suspicion of aortic dissection, and active internal bleeding).
- Beta blockers: Metoprolol 25 to 50 mg b.i.d, Atenolol 25 to 100 mg one a day (if pulse rate > 60/mm, BP > 90/60 mm Hg, lung fields clear).
- ACE inhibitors – Enalapril 2.5 – 20 mg / day in divided doses twice a day.
- Stool softeners – Bisacodyl (**Dulcolax**) 10 mg at night.

Message

Acute myocardial infarction is an emergency whatever treatment possible should be started at the center and patient should be transferred to District Hospital or any hospital where ICU facility is available in a cardiac ambulance. Patient should not be allowed to walk for even short distances and absolute Bed Rest is important. Treatment of complications like Heart failure, Arrhythmias may need expert opinion.

Coronary Angiography and revascularisation therapy should be advised.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. Ministry of Health and Family Welfare. Guidelines for Acute Myocardial Infarction. [cited 2016 Jul 5]
Available from: <http://nrhm.gov.in/images/pdf/guidelines/nrhm-guidelines/stg/acute-myocardial-infarction.pdf>
2. Vamadevan S. Ajay and Dorairaj Prabhakaran. Coronary heart diseases in Indians: Implications of INTERHEART Study. Indian J Med Res.[Internet]. 2010 Nov; 132(5): 561–566. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3028954/>
3. WHO. Clinical management guidelines for CAD. SEARO: World Health Organization. [cited 2016 Jul 5]
Available from: http://www.searo.who.int/india/topics/cardiovascular_diseases/NCD_Resources_CLINICAL_MANAGEMENT_GUIDELINES_FOR_CAD.pdf?ua=1



14. ACUTE RHEUMATIC FEVER (ARF)

1. Introduction

Acute rheumatic fever (ARF) is a multisystem disease resulting from an autoimmune reaction to infection with group A streptococcus.

2. Criteria for diagnosis of ARF

Major manifestations	<p>Carditis (Pancarditis)</p> <p>Polyarthritis (Migratory)</p> <p>Chorea</p> <p><i>Erythema marginatum</i></p> <p>Subcutaneous nodules</p>
Minor manifestations	<p>Clinical: fever, polyarthralgia</p> <p>Laboratory: elevated erythrocyte sedimentation rate or leukocyte count</p> <p>Electrocardiogram: prolonged P-R interval</p>
Supporting evidence of a preceding streptococcal infection within the last 45 days	<p>Elevated or rising anti-streptolysin O or another streptococcal antibody, <i>or</i></p> <p>A positive throat culture, <i>or</i></p> <p>Rapid antigen test for group <i>A. streptococcus</i>, <i>or</i></p> <p>Recent scarlet fever</p>

Two major or one major and two minor manifestations plus evidence of preceding group A streptococcal infection.

3. Investigations

White blood cell count, Erythrocyte sedimentation rate, C-reactive protein, Blood cultures if febrile, Electrocardiogram, Chest x-ray if clinical or echocardiographic evidence of carditis, Echocardiogram (consider repeating after 1 month if negative). Throat swab (preferably before giving antibiotics)–culture for group *A. streptococcus*.

Anti-streptococcal serology: both anti-streptolysin O and Anti-DNase B titres, if available.

4. Treatment

- All patients with Acute Rheumatic Fever (ARF) should receive antibiotics sufficient to treat the precipitating group *A. streptococcal* infection.

Penicillin is the drug of choice and can be given orally [as Phenoxymethyl Penicillin, 500 mg (250 mg for children 27 kg) PO twice daily, or Amoxicillin 50 mg/kg (max 1 g) daily, for 10 days] or as a single dose of 1.2 million units (600,000 units for children 27 kg) IM Benzathine Penicillin G.

- Aspirin in initial dose of 80–100 mg/kg per day in children (4–8 g/d in adults) in 4–5 divided doses is used for the treatment of arthritis, arthralgia, and fever for 4 weeks.
- Inj. Benzathine Penicillin G 1.2 IU (6 lakhs units for children 27 kg) IM every 3 weeks as prophylactic dose up to the age of 25 years.

5. When to refer

Evidence of Congestive cardiac failure, Arrhythmia.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015
4. National Treatment Guidelines for antimicrobial use in infectious diseases – Version 1.0 (2016), National Center for Disease Control, Ministry of Health and Family Welfare. [Cited 2016 July 7]
Available from: http://www.ncdc.gov.in/writereaddata/linkimages/AMR_guideline7001495889.pdf

Further reading

1. Kumar RK, Tandan R. Rheumatic fever and rheumatic heart disease: Last 50 years, Indian J Med Res [Internet]. 2013 Apr; 137(4): 643–658. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3724245/>



15. INFECTIVE ENDOCARDITIS

1. Definition

Infective endocarditis is a form of endocarditis, or inflammation, of the inner tissue of the heart (such as its valves) caused by infectious agents. The agents are usually bacterial, but other organisms can also be responsible.

Infection most commonly involves heart valves either native or prosthetic.

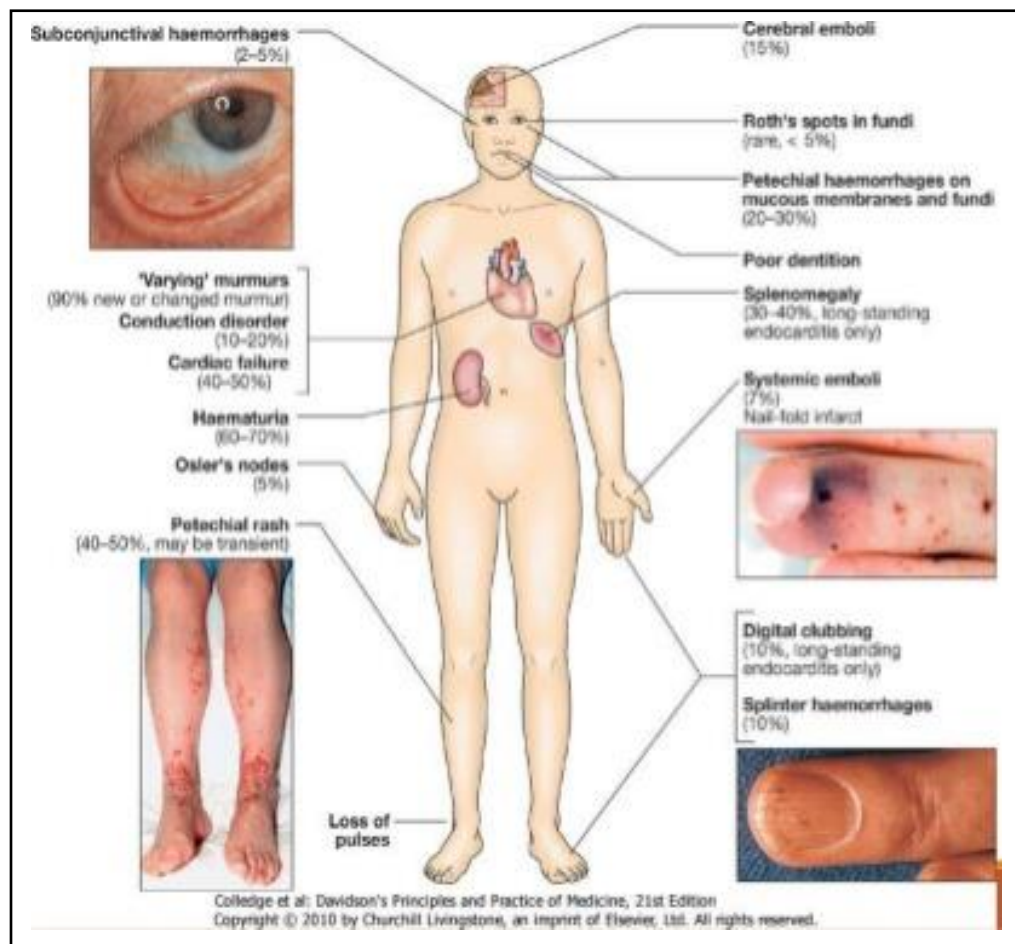
2. Organisms Causing Major Clinical Forms of Endocarditis

Streptococci, *Pneumococci* *Enterococci*,
Staphylococcus Aureus, Coagulase-negative
Staphylococci. Fastidious Gram-negative
coccobacilli (HACEK group i.e.
Haemophilus species, *Aggregatibacter Aphrophilus*,
Cardiobacterium, *Eikenella*, *Kingella*), Gram-
 negative bacilli, *Candida* spp.

3. Clinical Manifestations (Symptoms and signs)

Symptoms- Fever, chills, arthralgia, fatigue.

Figure 15.1: Signs of Infective Endocarditis



4. Diagnosis

Modified Dukes Criteria

4.1 Major Criteria

- i. Positive blood culture –
Two separate positive blood cultures with microorganism(s) typical for infective endocarditis: *Viridians streptococci*, *streptococcus bovis*, HACEK group, *Staphylococcus aureus*, community acquired *enterococci*
or
Persistently positive blood culture defined as presence of microorganism consistent with infective endocarditis from blood cultures drawn >12 hours apart.
or
Single positive blood culture for *Coxiella burnetii* or phase one IgG antibody titre of >1:800.
- ii. Echocardiographic evidence of endocardial involvement typical Valvular lesions; vegetations, abscess, or new partial dehiscence of a prosthetic valve New Valvular regurgitation.

4.2 Minor Criteria

- i. Predisposition; predisposing heart condition or intravenous drug use.
- ii. Temperature greater than 38.0 C.
- iii. Vascular phenomenon; major arterial emboli, septic pulmonary infarcts, mycotic aneurisms, intracranial haemorrhages, conjunctival haemorrhages, Janeway lesion

- iv. Immunological phenomenon;(glomerulonephritis; Osler nodes; Roth's spots; rheumatoid factor)
- v. Microbiological evidence positive blood cultures but not meeting major criteria or serological evidence of active infection with organism consistent with infective endocarditis.

Documentation of 2 major / one major and 3 minor /5 minor criteria allow a diagnosis Infective endocarditis.

5. Investigations

- CBC-Anaemia, Leucocytosis.
- Urine-Microscopic haematuria.
- Elevated ESR, Elevated CRP.
- Blood culture 3 sets of two bottle blood. Culture from different venepuncture site separated from one another by at least one hour over 24 hours.
- Echocardiography.

6. Complications

- Blood clots or emboli that travel to brain, kidneys, lungs, or abdomen.
- Brain abscess.
- Congestive heart failure.
- Glomerulonephritis.
- Jaundice.
- Neurological changes.
- Rapid or irregular heartbeats, including atrial fibrillation.
- Severe valve damage.
- Stroke.

7. Treatment

Table 1: Antimicrobial Therapy

Organism	Drug (Dose, Duration)
Penicillin-susceptible <i>streptococci</i>	Inj. Penicillin G (2–3 MU IV q4h for 4 weeks) or Inj. Ceftriaxone (2 g/d IV as a single dose for 4 weeks) plus Inj. Vancomycin (15 mg/kg IV q12h for 4 weeks)
Moderately penicillin-resistant <i>streptococci</i>	Inj. Penicillin G (4–5 MU IV q4h) or Ceftriaxone (2 g IV q.d.) for 6-weeks plus Inj. Gentamicin (3 mg/kg q.d. IV or IM as a single dose or divided into equal doses q8h for 6 weeks) Plus Inj. Vancomycin (15 mg/kg 12 hourly) as noted above for 4 weeks
<i>Enterococci</i>	Inj. Penicillin G (4–5 mU IV q4h) plus Inj. Gentamicin (1 mg/kg IV q8h), both for 4–6 weeks
<i>Staphylococci</i> (native valve)	Inj. Vancomycin (15 mg/kg IV q12h for 4–6 weeks)
<i>Staphylococci</i> (prosthetic valves)	Inj. Vancomycin (15 mg/kg IV q12h for 6–8 weeks) plus Inj. Gentamicin (1 mg/kg IM or IV q8h for 2 weeks) plus Rifampin (300 mg PO q8h for 6–8 weeks)

8. Prevention

High Risk cardiac lesions where antibiotic prophylaxis is needed.

1. Prosthetic heart valve.
2. Prior endocarditis.
3. Unrepaired cyanotic congenital heart disease.
4. Completely repaired cyanotic heart disease within 6 months.
5. Incompletely repaired cyanotic heart disease with residual defects.

Table: 2: Antibiotic Regimens for Prophylaxis of Endocarditis in Adults with High-Risk Cardiac Lesion

A. Standard oral regimen
1. Amoxicillin: 2 g PO 1 h before procedure
B. Inability to take oral medication
1. Ampicillin: 2 g IV or IM within 1 h before procedure
C. Penicillin allergy
1. Clarithromycin or azithromycin: 500 mg PO 1 hour before procedure
2. Cephalexin: 2 g PO 1 h before procedure
3. Clindamycin: 600 mg PO 1 h before procedure
D. Penicillin allergy, inability to take oral medication
1. Cefazolin or Ceftriaxone: 1 g IV or IM 30 min before procedure
2. Clindamycin: 600 mg IV or IM 1 h before procedure
Message- If any valvular heart disease or prosthetic valve patient develops fever, infective endocarditis should be thought of apart from other normal causes of fever.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015
4. National Treatment Guidelines for antimicrobial use in infectious diseases – Version 1.0 (2016), National Center for Disease Control, Ministry of Health and Family Welfare. [Cited 2016 July 7]
Available from: http://www.ncdc.gov.in/writereaddata/linkimages/AMR_guideline7001495889.pdf

Further reading

1. Ashish Gupta, Anu Gupta et el. Infective endocarditis in an Indian setup, Indian J Crit Care Med [Internet]. 2013 May-Jun; 17(3): 140–147. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3777367/>



16. DIABETES MELLITUS

1. Introduction

Diabetes mellitus is a clinical syndrome characterized by hyperglycaemia due to absolute or relative deficiency of insulin.

2. Classification of Diabetes Mellitus

2.1. Type 1 Diabetes Mellitus

Autoimmune Pancreatic B cell destruction; absolute insulin deficiency.

2.2. Type 2 Diabetes Mellitus

Characterized by peripheral tissue Insulin Resistance / Relative Insulin deficiency.

2.3. Other Specific Types

- Genetic defects of B cell function – Maturity Onset Diabetes in Young (MODY).
- Genetic defects of insulin action.

- Diseases of exocrine pancreas – Trauma, Pancreatitis, Pancreatectomy, Cystic fibrosis, Fibro-calculous Pancreatic diabetes, Haemochromatosis.
- Gestational Diabetes Mellitus (GDM) Onset / Recognition of glucose intolerance in pregnancy.

3. Diagnosis of Diabetes Mellitus

- Symptoms of Diabetes Mellitus and Random Blood Sugar > 200 mg% (mg / dl)
- Fasting blood sugar > 126 mg % on more than one occasion.
- 2 hours Plasma glucose > 200 mg% during oral glucose tolerance test with glucose – 75 g glucose.

Table 1: Criterion for Diagnosis based on blood glucose levels

	Normal Glucose	Pre-diabetic Impaired glucose tolerance	Diabetes mellitus
FPG	<100mg/dl	100-125 mg/dl	>126 mg/dl
2hr PG	<140mg/dl	141-199 mg/dl	>200 mg/dl
A1C (Glycosylated Haemoglobin)	<5.6%	5.7-6.4%	>6.5 mg/dl

4. Clinical symptoms

Common symptoms are

1. Polyuria (increased frequency of micturition),
2. Polydipsia (increased thirst),
3. Polyphagia (increased appetite),
4. Weight loss.

Other symptoms are tiredness, fatigue, pruritus vulvae, giddiness, burning over feet and can present with complications of diabetes.

5. Management

Table -2: Major differentiating features of Type 1 and Type 2 diabetes are as follows:

21.13 COMPARATIVE CLINICAL FEATURES OF TYPE 1 AND TYPE 2 DIABETES		
	Type 1	Type 2
Typical age at onset	< 40 years	> 50 years
Duration of symptoms	Weeks	Months to years
Body weight	Normal or low	Obese
Ketonuria	Yes	No
Rapid death without treatment with insulin	Yes	No
Autoantibodies	Yes	No
Diabetic complications at diagnosis	No	25%
Family history of diabetes	Uncommon	Common
Other autoimmune disease	Common	Uncommon

5.1 Type 1 Diabetes Mellitus

- Strict meal plan * Carbohydrate: 50 - 60% * Protein: 10 – 20% * Fat: 30% (If patient is dyslipidemic, fat should be 15%)

*Caloric intakes: 30 Kcal / kg

- Physical exercise
- Only Insulin

5.2 Type 2 Diabetes Mellitus:

- Strict meal plan
- Physical exercise
- Oral hypoglycaemic agents
- Insulin

Oral Hypoglycaemic Agents:

- a) Biguanides - Metformin
- b) Sulphonylureas - Glibenclamide, Glipizide, Glimpiride, Gliclazide
- c) Glinides (Nonsulphonylurea Secretagogue) - Repaglinide
- d) Thiazolidinediones –Pioglitazone

e) α Glucosidase inhibitors - Acarbose, Voglibose, Miglitol

f) DPP4 inhibitors- Sitagliptin, Vildagliptin

Parenteral agents

1. Insulin
2. GLP-1 receptor agonist- Liraglutide, Exenatide

(a) Biguanides

Dose- 500 mg-2000 mg/day.

Contra-indications to Biguanides therapy: 1. Renal failure when creatine clearance < 40 ml / min 2. Arteriography or intravenous urography – as intravenous iodinated products may precipitate lactic acidosis on patients with biguanides 3. Advanced liver cell failure 4. Alcoholism 5. Cardiac diseases 6. Diabetes with significant acute and late complications 7. Pregnancy 8. Old age > 70 years.

(b) Sulphonylureas

Mechanism of action- Increases insulin secretion from the beta cells through the ATP sensitive K channels.

Table-3: Sulphonylurea drugs with dosage and side effects

Drug	Dose	Side effects
Glibenclamide	1.25-20 mg	Hypoglycaemia, weight gain
Glimepiride	1-8 mg	Hypoglycaemia
Glipizide	2.5 -25 mg	Hypoglycaemia
Gliclazide	30-240 mg	Hypoglycaemia

Contraindications for sulphonylurea therapy

1. Insulin dependent diabetes mellitus (IDDM)
2. Pregnancy
3. Patients with severe infections
4. Allergic reactions
5. Significant liver and kidney disease
6. Patients undergoing surgery

(c) Glinides - (Nonsulphonylurea) Secretagogue-Increase insulin secretion

Dose-Repaglinide – 0.5-3 mg/day.

(d) Thiazolidinedione - These are insulin sensitizers. Pioglitazone is commonly used.

Dose- 15- 45 mg/day.

Side effects- Weight gain, congestive cardiac failure, fractures,

(e) α Glucosidase inhibitors – Decreases intestinal glucose absorption and reduce postprandial hyperglycaemia.

Dose- Acarbose - 25 -100 mg/day to be taken with food.

Voglibose - 0.2- 0.3 mg thrice a day

(f) DPP 4 Inhibitors

Mechanism of action – They inhibit the enzyme DPP 4 and increase endogenous GLP-1 action

thereby increasing insulin secretion only with the intake of food. They do not cause hypoglycaemia.

Dose- Vildagliptin 50 mg BD, Sitagliptin 100 mg OD.

Insulin

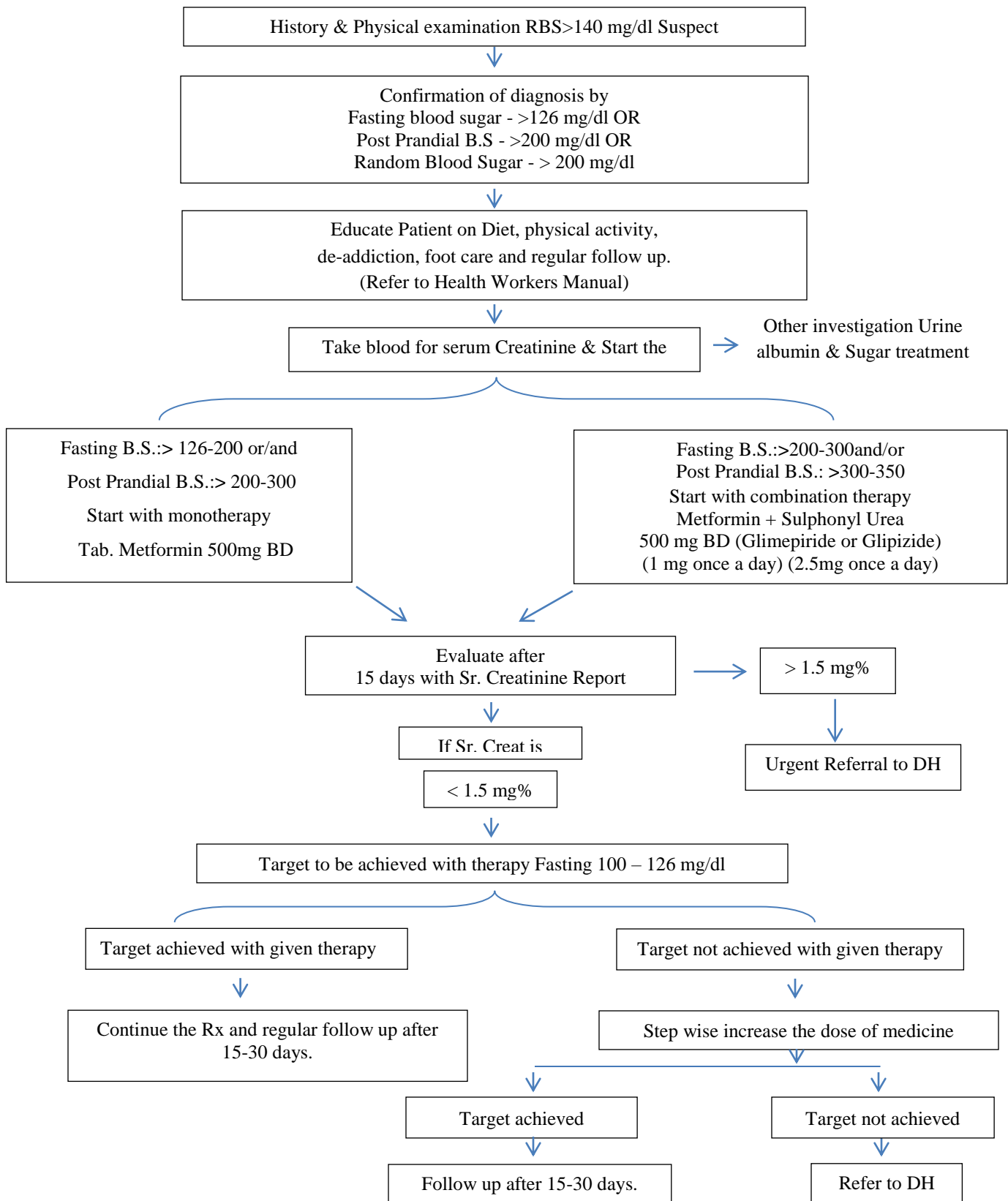
Consider insulin as initial therapy in patients with:

- Fasting plasma glucose > 250 - 300 mg/dl since more rapid glycaemic control will reduce glucose toxicity to islet cells, improve insulin secretion and possibly make oral hypoglycaemic agents more effective.
- Lean patients or those with severe weight loss.
- Underlying renal or hepatic disease.
- Hospitalized or acutely ill patients.
- If response to oral hypoglycaemics is not adequate. Consider insulin as initial therapy
- Pregnancy

Types of Insulin

1. Rapid-acting insulin: Aspart insulin and Lispro insulin
2. Regular or Short-acting insulin
Intermediate-acting insulin: Lente insulin and NPH insulin
3. Long-acting insulin: Insulin Detemir and insulin Glargine

Figure-15.1: Treatment Protocol for DM – Type II



Steps to increase dose in:

A) Monotherapy:

- I. Start with T. Metformin 500 mg BD
(If target not achieved)
- II. T. Metformin 750 mg BD
(If target not achieved)
- III. T. Metformin 1 gm BD
(If target not achieved)

Refer the patients to DH.

B) Combination therapy:

Metformin + Glimepiride or Glipizide.

- 1) 500mg BD + 1mg one a day or 2.5mg BD
(If target not achieved)
- 2) 750mg BD + 1mg once a day or 2.5mg BD
(If target not achieved)

- 3) 1 gm BD + 2 mg BD Or 5 mg BD (If target not achieved)

Refer the patients to DH.

* The main aim of this protocol is to control raised Blood sugar however not to complicate by hypoglycaemia.

- Instruction:
- Don't allow patients to fast while on medication.
 - Strict De-addiction state should be maintained.
 - If sudden rise in blood sugar observed then → urgent referral to District Hospital.

6. Check list

21.16 CHECKLIST FOR FOLLOW-UP OF PATIENTS WITH DIABETES MELLITUS

Body weight (body mass index)

Urinalysis

- Analyse fasting specimen for glucose, ketones, albumin (both macro- and microalbuminuria)

Glycaemic control

- Glycated haemoglobin (HbA_{1c})
- Inspection of home blood glucose monitoring record

Hypoglycaemic episodes

- Number of severe (requiring assistance for treatment) and frequency of mild (self-treated) episodes
- Time of day when 'hypos' experienced
- Nature and intensity of symptoms
- Ability to identify onset (awareness)

Blood pressure

Eye examination

- Visual acuities (near and distance)
- Ophthalmoscopy (with pupils dilated)
- Digital photography

Lower limbs

- Peripheral pulses
- Tendon reflexes
- Perception of vibration sensation, light touch and proprioception

Feet

- Callus skin indicating pressure areas
- Nails
- Need for podiatry
- Ulceration
- Deformity

7. Complications of Diabetes Mellitus

7.1 Acute complications

- Hypoglycaemia
- Diabetic Ketoacidosis (DKA)
- Diabetes and infections
- Hyperosmolar Hyperglycaemic coma (HHNKC).

7.2 Chronic complications

- **Microvascular complications –**
 - Eye disease- Retinopathy, Macular oedema.
 - Neuropathy- Sensory and motor neuropathy (mono and poly),
 - Autonomic neuropathy

- Nephropathy

- **Macrovascular complications –**

- Coronary heart disease
- Cerebrovascular disease
- Peripheral arterial disease

- **Other –** gastroparesis, sexual dysfunction, dermatologic

8. Hypoglycaemia

Hypoglycaemia is a clinical emergency occurring in diabetes characterized by either autonomic or neuroglycopenic symptoms (or) biochemically random blood sugar < 60 mg, due to antidiabetic agent, food and activity mismatch.

Table-4: Clinical presentation of Hypoglycaemia

Autonomic symptoms
<ul style="list-style-type: none"> ● Tremor ● Palpitation ● Sweating ● Sensation of hunger
Neuroglycopenic symptoms
<ul style="list-style-type: none"> ● Headache ● Fatigue ● Irritability ● Disturbed vision ● Mental confusion ● Personality changes ● Convulsion, night mares ● Coma

8.1 Management

- Draw blood sample immediately
- Dextrose supplementation - Conscious: Oral Glucose, Sugar, Fruit Juice, Unconscious: 50% Dextrose 100 ml IV Stat. Followed by 10% Dextrose then by 5% DNS maintenance (or) Inj. Glucagon 1 mg IM if not accessible to intravenous route
- Patient still remains unconscious - To rule out cerebral oedema, If present IV mannitol + Inj. Dexamethasone 8 mg IV
- Stop the antidiabetic agents for 3 days in Type 2 Diabetic mellitus patients and recheck blood sugars. In Type 1 Diabetic mellitus patients, recheck blood sugars after 6 hours and adjust insulin dose accordingly.
- Identify the cause of hypoglycaemia
- If recurrent hypoglycaemia, rule out - Renal function disorder, Liver function disorder
- Repeat blood sugar value after hypoglycaemia correction and monitor blood sugars

- Referral: If the patient remains unconscious even after dextrose administration refer the patient immediately to higher centre for further evaluation.
- Important Note Patient Education:
- Educate patient and his family members about low blood sugars and symptoms
- Never miss a meal after insulin / Oral hypoglycaemic agents
- Be cautious of unaccustomed physical activity
- To carry diabetic identity card
- Always carry simple sugar (biscuits and toffee) to avoid low sugar.

9. Diabetic Ketoacidosis (DKA)

Ketoacidosis is a major medical emergency and remains a serious cause of morbidity, principally in people with type 1 diabetes, which should be treated in hospital. Patient should be referred to tertiary health centre after the primary management.

9.1 Manifestation of DKA

Table 5

Table	Manifestations of Diabetic Ketoacidosis
Symptoms	<ul style="list-style-type: none"> Nausea/vomiting Thirst/polyuria Abdominal pain Shortness of breath
Precipitating events	<ul style="list-style-type: none"> Inadequate insulin administration Infection (pneumonia/UTI/gastroenteritis/sepsis) Infarction (cerebral, coronary, mesenteric, peripheral) Drugs (cocaine) Pregnancy
Physical Findings	<ul style="list-style-type: none"> Tachycardia Dehydration/hypotension Tachypnea/Kussmaul respirations/respiratory distress Abdominal tenderness (may resemble acute pancreatitis or surgical abdomen) Lethargy/obtundation/cerebral edema/possibly coma

9.2 Investigations

- Blood glucose [usually > 250 mg %]
- Blood urea [may or may not be ↑]
- Serum creatinine [may or may not be ↑]
- Serum electrolyte [Na ↑ or ↓, K ↑ or decreased]
- Serum bicarbonate < 10 mmol / l

- ABG for pH and bicarbonate
- Urine sugar [positive]
- Urine acetone [positive]
- Chest X-ray
- ECG
- Ultra-sonogram abdomen / KUB.

9.3 Management of DKA

<p>fluid replacement</p> <ul style="list-style-type: none">• 0.9% saline (NaCl) i.v.<ul style="list-style-type: none">◦ 1 litre over 30 minutes◦ 1 litre over 1 hr◦ 1 litre over 2 hrs◦ 1 litre over next 2-4 hrs• When blood glucose < 15 mmol/l (270 mg/dl)<ul style="list-style-type: none">◦ Switch to 5% dextrose, 1 litre 8-hourly◦ If still dehydrated, continue 0.9% saline and add 5% dextrose 1 litre per 12 hrs• Typical requirement is 6 litres in first 24 hrs but avoid fluid overload in elderly patients• Subsequent fluid requirement should be based on clinical response including urine output
<p>Insulin</p> <ul style="list-style-type: none">• 50 units soluble insulin in 50 ml 0.9% saline i.v. via infusion pump<ul style="list-style-type: none">◦ 6 units/hr initially◦ 3 units/hr when blood glucose < 15 mmol/l (270 mg/dl)◦ 2 units/hr if blood glucose declines < 10 mmol/l (180 mg/dl)• Check blood glucose hourly initially-if no reduction in first hour, rate of insulin infusion should be increased• Aim for fall in blood glucose of 3-6 mmol/l (~55-110 mg/dl) per hour
<p>Potassium</p> <ul style="list-style-type: none">• None in first litre of i.v fluid unless < 3.0 mmol/l• If plasma potassium < 3.5 mmol/l, give 40 mmol added potassium<ul style="list-style-type: none">◦ Give in 1 litre of fluid◦ Avoid infusion rate of > 20 mmol/hr• If plasma potassium is 3.5-5.0 mmol/l, give 20 mmol added potassium• If plasma potassium is > 5.0 mmol/l, or patient is anuric, give no added potassium
<p>21.21 ADDITIONAL PROCEDURES IN THE MANAGEMENT OF DIABETIC KETOACIDOSIS</p> <ul style="list-style-type: none">• Catheterisation if no urine passed after 3 hrs• Nasogastric tube to keep stomach empty in unconscious or semiconscious patients, or if vomiting is protracted• Central venous line if cardiovascular system compromised, to allow fluid replacement to be adjusted accurately• Plasma expander if systolic BP is < 90 mmHg or does not rise with i.v. saline

9.4 Referral

Refer the patient to a higher centre if:

- Patient is comatose
- Hypotension requiring inotropic support
- Anuric
- Elevated renal parameters
- Evidence of septicemia

9.5 Complications of Diabetic Ketoacidosis

COMPLICATIONS OF DIABETIC KETOACIDOSIS

- Cerebral oedema
May be caused by very rapid reduction of blood glucose, use of hypotonic fluids and/or bicarbonate
High mortality
Treat with mannitol, oxygen
- Acute respiratory distress syndrome
- Thromboembolism
- Disseminated intravascular coagulation (rare)
- Acute circulatory failure

10. Hyperglycaemic Hyperosmolar Non-Ketotic Coma (HHNKC)

It is an acute metabolic complication in middle aged and elderly diabetics with high morbidity and mortality.

10.1 Causes

Precipitated by

- Infection
- Trauma
- Burns
- Infarction
- Hyper-alimentation
- Drugs like - Thiazide, Cimetidine, Phenytoin and parenteral diuretics

10.2 Symptoms

- Polyuria
- Polydipsia
- Severe hyperglycaemia (Blood sugar > 600 mg%)
- Profound dehydration
- Elevated osmolality
- Hemianopia, muscle fasciculation, seizures
- Altered sensorium, coma

10.3 Treatment

- Fluid replacement: ½ normal saline at the rate of 2 litres in 1st 2 hours and 1 litre in another 2 hours
- Low dose insulin.
- Correction of electrolytes and hyper osmolality.
- Low-dose heparin to prevent vascular thrombosis intravascular coagulation.

10.4 Distinguishing features

Table-6: Distinguishing features between DKA and HHNKC

Distinguishing features between DKA and HHNKC		
Criteria	DKA	HHNKC
Age	Younger	Older
Respiration	Hyperventilation, deep	Normal, shallow
Dehydration	Around 10 %	Around 25 %
Consciousness	Diminished	Comatose
Temperature	Normal or low	May be raised
Blood glucose	>300 mg/dl	> 600 mg/dl
Blood urea	42 – 70 mg/dl	60 – 180 mg/dl
Sodium	125 – 140 mmol/l	130 – 155 mmol/l
Potassium	3 – 6.5 mmol/l	3 – 5 mmol/l
Bicarbonate	< 14 mmol/l	16 – 30 mmol/l
Ketones	+ + To + + +	0 to +

11. Medical Nutrition Therapy

A non-pharmacological mode of management of diabetes. Medical Nutrition therapy is individualized and should be a tailor made regimen. It is used as a compliment for an oral glucose lowering agent / insulin therapy.

Energy Recommendations:

- This depends on body weight and physical activity. 20 kcal/kg Ideal body weight – Sedentary worker 30 kcal/kg Ideal body weight – Moderate worker 40 kcal/kg Ideal body weight – For heavy worker
- In obese people - Reduce 500 kcal from the calculated energy requirement
- For underweight- Add 500 kcal to the calculated energy requirement of the total kcal, 45 - 65 % kcal from carbohydrate and 10-25 % kcal from proteins
- Fat recommendation - 500 ml / month of a blend of oils / individual Gingelly oil and any refined vegetable oil could be used or Rice brand oil and any refined vegetable oil

- Fibre recommendation – 14 gm/1000 kcal provided.
- Fluid recommendation - 8 to 10 glasses / day of water except in LVF, CKD, Cirrhosis etc.

11.1 MNT in Gestational Diabetes Mellitus

- 30 kcal / kg Instant Body weight in first trimester
- 30 kcal / kg Instant Body weight + 300 kcal/day in II and III trimester
- Protein - 1gm/kg Instant Body weight + 10gm daily throughout pregnancy
- Avoid hypocaloric diets in obese GDM
- Provide compulsory bed time and evening snack to avoid accelerated starvation and nocturnal hypoglycaemia

Gestational Diabetes mellitus:

- Strict meal plan
- Physical exercise
- Insulin

Other specific types

- Strict meal plan
- Physical exercise
- Insulin with or without oral hypoglycaemic agents

12. Gestational Diabetes Mellitus

Gestational Diabetes mellitus is defined as carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy.

12.1 Risk for gestational diabetes mellitus

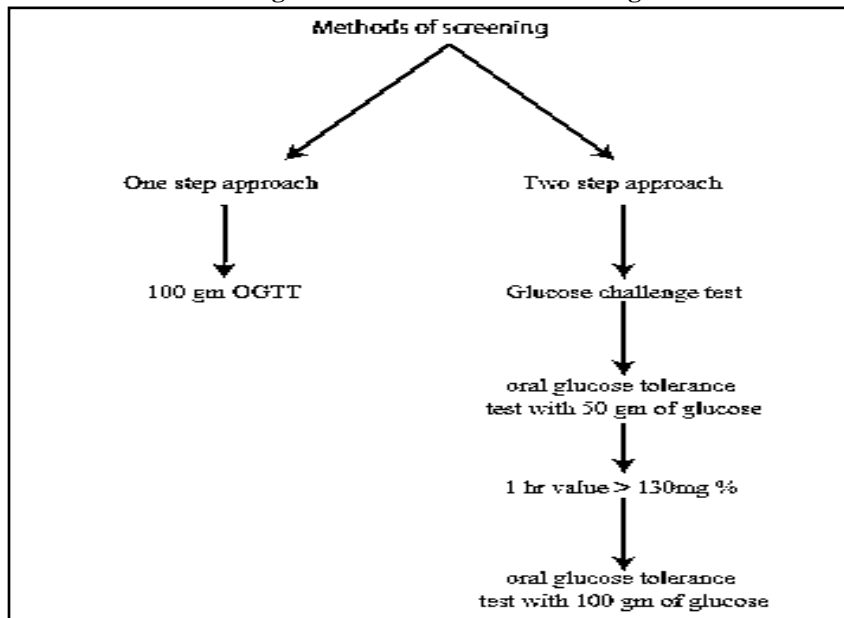
- Age more than 25 years
- Family history of diabetes mellitus
- History of unexplained Foetal loss
- History of baby being large for gestational age
- History of congenitally malformed infant
- Maternal obesity
- History of Polycystic ovarian disease
- Polyhydramnios
- Pre-eclampsia
- Unexplained intrauterine death

12.2 Methods of screening

- Spot test: Fasting < 90 mg% [normal 2 hr post-prandial < 120 mg%, random < 105 mg% values]

ADA recommendation

Figure-15.2: Methods of Screening



12.3 Diagnostic Criteria

WHO criteria (with 75 gm of glucose) F - 95 mg %
1 hour - 180 mg % 2 hour - 155 mg % If any two values equal or crosses normal value, it is termed as Gestational Diabetes mellitus.

Important Note OGTT value should never be treated.

12.4 Treatment

- Medical Nutrition Therapy [refer MNT]
- Insulin is essential if MNT fails to achieve euglycaemia

Target Glycaemic Level

- Fasting glucose – 90 mg %
- 2 hr postprandial – 120 mg %

Monitoring Glycaemic Control

- Blood glucose fasting and postprandial every three days till euglycaemia is achieved; then every fortnightly, throughout first and second trimester.
- Every week in third trimester.
- Glycaemic profile monitoring once in 1st and 2nd trimester and then every month.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier
3. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015

Further reading

1. India. Ministry of Health and Family Welfare. Guidelines for Diabetes Mellitus. [cited 2016 Jul 5] Available from: <http://nrhm.gov.in/images/pdf/guidelines/nrhm-guidelines/stg/diabetes-mellitus.pdf>
2. India. Ministry of Health and Family Welfare. Guidelines for Gestational Diabetes Mellitus. [cited 2016 Jul 5] Available from: http://nrhm.gov.in/images/pdf/programmes/maternal-health/guidelines/National_Guidelines_for_Diagnosis_&_Management_of_Gestational_Diabetes_Mellitus.pdf



17. THYROID DISORDERS

1. Hypothyroidism

1.1. Types

Hypothyroidism may be

1.1.2. Primary

Common causes of which are autoimmune, iatrogenic due to Iodine¹³¹, anti-thyroid or lithium treatment and thyroidectomy

1.1.2. Secondary

- i. Pituitary disease
- ii. Hypothalamic disease.

1.2. Symptoms

- Coarse dry skin
- Hoarseness of voice
- Facial puffiness, weight gain
- Cardiac enlargement and / or pericardial effusion,
- Goitre with or without prolonged relaxation phase of deep tendon reflexes.
- Myxoedema coma is a rare complication of severe hypothyroidism with hypothermia, hypoventilation, hyponatremia, hypoxia, hypercapnia and hypotension.

1.3. Diagnosis is confirmed by

- Low serum free T3 and T4
- Serum TSH raised

Other investigation – blood sugar level and lipid profile

1.4. Treatment

- Pharmacological
 - * Tab. L - Thyroxine- 1.6 mcg per kg body weight or Start with 50 – 100 mcg/day
 - * Dose to be adjusted based on TSH levels
 - * Goal is normal TSH (lower half of reference range

* Measure TSH levels after about 6 weeks of instituting therapy

* Adjust by 12.5 or 25 mcg increments if TSH is high; decrement of the same if TSH is suppressed.

* When full replacement is achieved then follow up measurement at annual intervals and later by a 2 - 3 yearly interval

* Ensure ongoing compliance.

1.5. Special treatment considerations

- A hypothyroid woman should be euthyroid prior to conception and during early pregnancy (effect on foetal neuronal development). Thyroid profile should be immediately done after confirmation of pregnancy and in second and third trimester.
- ✓ Dose of Thyroxine should be increased by 50% during pregnancy and return to previous level after delivery
- ✓ Elderly require less Thyroxine (less by up to 20%) especially those with coronary artery disease, starting dose 12.5 mcg/day with similar increments every 2 - 3 months until TSH level is normalized.
- ✓ In Hypothyroidism due to low TSH (suprathyroid cause is suspected) detailed investigations are required and patient should be referred to a tertiary care level
- ✓ Asses the response clinically and by serum TSH (serum T3 in suprathyroid type) at 8 weekly intervals
- ✓ Once euthyroid state is restored, follow-up at 6 - 12 monthly intervals.

1.6. Treatment of Myxoedema coma

- ✓ Warm blankets, mechanical ventilation for respiratory failure.
- ✓ Correction of metabolic disturbances and treat precipitating factors.
- ✓ Drugs
 - L-Thyroxine 500 mcg IV bolus, then 50-100mcg IV daily
 - If intravenous preparation not available, the same dose is administered through Ryle's tube.
 - Once acute phase is over, maintain L-Thyroxine as above.

- Inj. Hydrocortisone 100 mg IV stat, 25-50 mg 8 hourly.
- ✓ Caution: Avoid sedatives

1.7. Patient Education

- L-Thyroxine should be taken as a single daily dose, ideally on awakening, at least 30 minutes before breakfast.
- Fibre and bran products (e.g., Isaphghul husk) may impair absorption, as also Cholestyramine, Colestipol, Iron Sulphate, Sucralfate, Aluminium hydroxide
- Metabolism of L-Thyroxine is increased by Phenytoin, Rifampicin and carbamazepine.

2.1. Symptoms

Table Signs and Symptoms of Thyrotoxicosis (Descending Order of Frequency)	
Symptoms	Signs^a
Hyperactivity, irritability, dysphoria	Tachycardia; atrial fibrillation in the elderly
Heat intolerance and sweating	Tremor
Palpitations	Goiter
Fatigue and weakness	Warm, moist skin
Weight loss with increased appetite	Muscle weakness, proximal myopathy
Diarrhea	Lid retraction or lag
Polyuria	Gynecomastia
Oligomenorrhoea, loss of libido	

Grave's disease is characterized by diffuse goitre, Ophthalmopathy and Dermopathy in varying combinations.

Ophthalmopathy in Graves' disease; lid retraction, periorbital oedema, conjunctival injection, and proptosis are marked

2.2. Diagnosis

Diagnosis is confirmed by low to undetectable serum TSH and increased Serum free (FT3) and free (FT4)

Ultra-sonography of neck

Thyroid scan (if available)

2.3. Treatment

Pharmacological

- Adjunctive treatment * For adrenergic symptoms such as sweating, tremor and

- Explain to the patient that the treatment is lifelong. Do not modify dose or stop treatment with- out consultation.
- Over treatment may lead to decreased bone mineral density and adverse cardiac complications.

2. Hyperthyroidism

Thyrotoxicosis is defined as the state of thyroid hormone excess and is not synonymous with *hyperthyroidism*, which is the result of excessive thyroid function. However, the major aetiologies of thyrotoxicosis are hyperthyroidism caused by Graves' disease, toxic MNG, and toxic adenomas

tachycardia. Tab. Propranolol 40 – 120 mg a day or Tab. Atenolol 25 mg to 50 mg a day

- Anti-thyroid drug-
- Tab. Propylthiouracil 100 – 150 mg every 6 – 8 hours or Tab. Carbimazole 10 – 20 mg every 8 – 12 hours;
- After euthyroid state is achieved in 6 – 8 weeks once daily dosage. Review with serum TSH and FT3 after 3 – 4 weeks' treatment has been initiated.
- Once controlled reduce to the smallest effective dose or continue initial dose combined with L-Thyroxine
- Drugs are given for an average of 2 years.
- Definitive treatment is surgery/ablation of thyroid tissue
- Subtotal thyroidectomy in younger patients (<30 years) in whom anti-thyroid therapy has been unsuccessful and in very large goitres.

- Radioactive iodine (I131): Method of choice in Elderly Younger patients who have completed family with recurrent thyrotoxicosis following surgery or when surgery is refused or contraindicated. Caution Radioactive iodine should never be given in pregnancy. In woman of childbearing age if radioactive iodine is planned, a pregnancy test should always be carried out.

2.4. Pregnancy

- In pregnant woman, surgery should not be performed in 1st or 3rd trimesters
- Anti-thyroid drugs are less risky but may induce hypothyroidism in the foetus and should be used in the smallest necessary dose to keep serum TSH and FT4 in normal range.
- Propylthiouracil is preferred – usual maintenance is 200 mg/day. If > 300 mg/day required during 1st trimester, Subtotal thyroidectomy is indicated in 2nd trimester
- Propranolol should be avoided as it can cause foetal growth retardation and neonatal respiratory depression.

Ophthalmopathy: Refer to ophthalmologist. Initiate therapy in mild cases with elevation of head at night, diuretics to decrease oedema, use of tinted sun glasses and 1 % methyl cellulose eye drops to prevent drying and refer patients with severe and progressive exophthalmos to an ophthalmologist.

2.5. Toxic multinodular goitre

- Radioactive iodine is the treatment of choice.
- Large doses are usually required.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Ambika Gopalakrishnan Unnikrishnan and Usha V. Menon. Thyroid disorders in India: An epidemiological perspective, Indian J Endocrinal Metab [Internet]. 2011 Jul; 15(Suppl2): S78–S81. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3169866/>



18. Cerebrovascular Accidents

- Treatment with anti-thyroid drugs given till patient is euthyroid.
- Propranolol may be useful before and after radioactive iodine administration.

2.6. Thyrotoxic crisis or thyroid storm

- Refer to a tertiary care centre.
- Life threatening hyperthyroidism with fever, vomiting, diarrhoea, jaundice, delirium and coma.
- Usually precipitated by acute illness such as stroke, infection, diabetic ketoacidosis, trauma, patients undergoing surgery or radioactive iodine treatment in a poorly prepared patient:
- Tab. Propylthiouracil 600 mg loading dose, then 200 – 300 mg every 6 hours orally or through Ryle's tube. (or) Tab. Carbimazole 15 – 25 mg 6 hourly.
- 1 hour after the 1st dose of anti-thyroid drug, saturated solution of Potassium iodide (SSKI) 5 drops every 5 hours (or) Lugol's iodine 10 drops 3 times a day (or) Sodium iodide 1 g IV slowly.
- Tab. Propranolol 40 – 60 mg 4 hourly or 0.5 – 2 mg IV every 4 hours.
- Inj. Dexamethasone 2 mg IV 6 hourly
- Continue iodides and dexamethasone until normal metabolic stage is achieved and give supportive treatment such as cooling, antipyretics, antibiotics for infection, intravenous fluids, etc.
- Once euthyroid status is achieved, manage as already outlined.

1. Definition

A stroke, or cerebrovascular accident, is defined by the abrupt onset of a neurologic deficit that is attributable to a focal vascular cause.

Thus, the definition of stroke is clinical, and laboratory studies including brain imaging are used to support the diagnosis.

The clinical manifestations of stroke are highly variable because of the complex anatomy of the brain and its vasculature. TIA (Transient Ischemic Attack) is a brief episode of neurological dysfunction lasting 1 hour to 24 hours without residual deficit. Stroke in evaluation means when deficit worsens after patient first presents.

Cerebral ischemia is caused by a reduction in blood flow that lasts longer than several seconds. If the cessation of flow lasts for more than a few minutes, *infarction* or death of brain tissue results. When blood flow is quickly restored, brain tissue can recover fully and the patient's symptoms are only transient.

Focal ischemia or infarction, conversely, is usually caused by thrombosis of the cerebral vessels themselves or by emboli from a proximal arterial source or the heart.

Intracranial haemorrhage is caused by bleeding directly into or around the brain; it produces neurologic symptoms by producing a mass effect on neural structures, from the toxic effects of blood itself, or by increasing intracranial pressure.

2. Clinical features

- Most of the patients are elderly.
- There could be a history of Hypertension, Diabetes, Ischaemic heart disease
- History of cerebrovascular accident may be present.
- Hemiplegia or hemiparesis: Paralysis or weakness of right or left half of the body with deviation of one side of the face seen.
- Lower limb monoparesis or monoplegia.
- Faciobrachial stroke: involving right or left upper limb along with ipsilateral facial weakness.
- Rarely bilateral lower limb weakness in unpaired anterior cerebral artery thrombosis.
- Hemiplegia or hemiparesis may be accompanied by motor aphasia (patient unable to speak) or sensory aphasia (inability to comprehend) if there is cortical involvement of dominant hemisphere (right side in left handed people, left

side in 90% of right handed people).

- Transient or permanent loss of central vision due to involvement of ophthalmic artery a branch of internal carotid artery.
- Difficulty in swallowing, speaking due to bulbar involvement could be a presentation. In cases where patient has previously had cerebrovascular accident, fresh infarct now causing bilateral cortical involvement may lead to dysphagia and dysarthria causing what is known as pseudobulbar palsy. On examination, in these patients, jaw jerk is brisk.
- Midbrain lesions have ocular involvement.
- Sensory involvement occurs in stroke involving thalamus or medulla.
- The neurological deficit may be preceded by tingling numbness.
- TIA - Transient Ischaemic Attack is a neurological deficit that recovers completely within 24 hours.
- A focal deficit progressing over a period of hours and is characteristic of thrombotic. stroke.
- Any focal deficit preceded by a prolonged headache could be due to infarct with haemorrhagic conversion secondary to cerebral venous sinus thrombosis.
- Focal deficit could be accompanied by convulsions.

3. Examination

- Patient may have bradycardia if he is on beta blockers or due to raised intracranial pressure secondary to cerebral oedema.
- Blood pressure should be recorded and monitored.
- The power of all 4 limbs should be graded and any improvement or deterioration in the power should be recorded.
- Reflexes on the involved side are brisk and plantar reflex is extensor on the involved side.
- History of headache, altered sensorium, fever, vomiting.

4. Investigations

- Non-contrast CT scan brain to differentiate between infarct and haemorrhage and site of lesion.
- Routine investigations like HB, CBC, ESR, liver function test, renal function test, serum electrolytes, fasting and post prandial glucose, complete lipid profile.
- ECG, X- ray chest, 2D echo especially in young patients to rule out cardiac source of embolism.

- Ideally MR angiography in cases of infarction to look for the stenosis in intracranial vessels.
- SOS MR venography if any doubt of cerebral venous thrombosis.

5. Complications

- Aspiration pneumonia
- Deep vein thrombosis in lower limb complicating further as pulmonary embolism.
- Bed sore

6. Treatment

- Blood pressure, blood glucose and temperature of patients of acute cerebral infarction should be controlled.
- Anti-hypertensive medication. A blood pressure of 150/90 mm of Hg can be maintained initially to prevent Cerebral Ischaemia due to hypo perfusion.
- IV Mannitol 100 cc 8 hourly in cases of IntraCerebral Bleed, in large infarcts, brainstem infarction, Cerebellar lesions and all patient having altered Sensorium. Serum electrolytes and urine output should be monitored (on mannitol.)
- Tab Aspirin 75-150 mg Once a day after lunch should be started in cases of Cerebral Infarction. As Intracranial Bleed is ruled out.

- Tab Atorvastatin 40mg Once a day H.S.
- Antiepileptics (Inj. Phenytoin 100 mg t.d.s) if convulsions are present. But in case the bleed is large and if the patient is going to be shifted to a higher centre it may be advisable to give anti-epileptics as the patient's general condition might worsen if he gets a convulsion.
- Half an hour before Ryle's tube feeding Domperidone should be administered. Ryle's tube feeding in strict propped up position.
- Inj. Heparin (5000 IU IV bolus and then once a day to keep PT (INR) 2 to 3 times of normal value) for prophylaxis of deep vein thrombosis in cases of cerebral infarction (not in intracranial bleed) in patient of hemiplegia.
- Physiotherapy.
- Early detection and treatment of complications.

7. When to refer

- All young patients of stroke, intracranial bleed or infarction.
- Patients diagnosed with cerebral venous thrombosis.
- Subarachnoid haemorrhage if seen on the CT.
- Patient suspected to have AV malformation.
- Large intracerebral bleeds who might require evacuation to be transferred after stabilizing blood pressure.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Pandian JD, Sudhan P. Stroke Epidemiology and Stroke Care Services in India, J Stroke [Internet]. 2013 Sep; 15(3): 128–134. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3859004/>



19. SUBARACHNOID HEMORRHAGE (SAH)

Excluding head trauma, the most common cause of SAH is rupture of a saccular aneurysm. Other causes include bleeding from a vascular malformation (arteriovenous malformation or dural arterial-venous fistula) and extension into the subarachnoid space from a primary intracerebral haemorrhage.

1. Clinical features

Most unruptured intracranial aneurysms are completely asymptomatic. Symptoms are usually due to rupture and resultant SAH, although some unruptured aneurysms present with mass effect on cranial nerves or brain parenchyma. At the moment of aneurysmal rupture with major SAH, the ICP suddenly rises. This may account for the sudden transient loss of consciousness that occurs in nearly half of patients. Sudden loss of consciousness may be preceded by a brief moment of **excruciating headache**.

The most important characteristic is *sudden onset* or as a change in the patient's usual headache pattern. The headache is usually generalized, often with **neck stiffness** and **vomiting** is common.

Although sudden headache in the absence of focal neurologic symptoms is the hallmark of aneurysmal rupture, focal neurologic deficits may occur. The deficits that result can include hemiparesis, aphasia, and abulia.

Occasionally, prodromal symptoms suggest the location of a progressively enlarging unruptured aneurysm.

A **third cranial nerve palsy**, particularly when associated with pupillary dilation, loss of ipsilateral (but retained contralateral) light reflex, and focal pain above or behind the eye, may occur with an expanding aneurysm at the junction of the posterior communicating artery and the internal carotid artery. Visual field defects can occur with an expanding supraclinoid carotid or anterior cerebral artery aneurysm.

Aneurysms can undergo small ruptures and leaks of blood into the subarachnoid space, so-called *sentinel bleeds*. Sudden unexplained severe headache at any location should raise suspicion of SAH and be investigated, because a major haemorrhage may be imminent.

Bibliography

A detailed history should be sought for use of oral contraceptive pills in young females to rule out SAH secondary to cerebral venous thrombosis as could occur in any procoagulant state.

2. Investigations

CT brain, MRI angiography and MRI venography to rule out cerebral venous thrombosis which could present as subarachnoid haemorrhage.

3. Treatment: Subarachnoid Haemorrhage

- i. Protecting the airway.
- ii. Managing blood pressure before and after aneurysm treatment, with antihypertensive agent. Preventing re-bleeding prior to treatment.
- iii. Managing vasospasm, treating hydrocephalus.
- iv. Maintaining electrolyte balance and hydration
- v. IV Mannitol 100cc 8 Hourly to treat raised Intra cranial pressure.
- vi. Treatment with the calcium channel antagonist **Nimodipine** (60 mg PO every 4 h) improves outcome, perhaps by preventing ischemic injury rather than reducing the risk of vasospasm.
- vii. Volume expansion helps prevent hypotension, augments cardiac output, and reduces blood viscosity by reducing the haematocrit. This method is called "Triple-H" (Hypertension, Haemodilution, and Hypervolemic) therapy.
- viii. Acute hydrocephalus can cause stupor or coma. It may clear spontaneously or require temporary ventricular drainage. When chronic hydrocephalus develops, **ventricular shunting** is the treatment of choice.

4. When to refer

A patient diagnosed to be having SAH should be transferred to a higher centre at the earliest after stabilizing blood pressure for further management.

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Sodhi HBS, Savardekar AR, Mohindra S, et al. The clinical profile, management and overall outcome of aneurysmal subarachnoid haemorrhage at the neurosurgical unit of tertiary care center in India, J Neurosci Rural Pract. [Internet]. 2014 Apr-Jun; 5(2): 118–126. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4064174/>



20. PYOGENIC MENINGITIS

Meningitis is a serious disease in which there is inflammation of the meninges that cover the brain and spinal cord.

Bacterial meningitis can be deadly and contagious among people in close contact.

Viral meningitis tends to be less severe and most people recover completely.

Fungal meningitis is a rare form of meningitis and generally occurs only in people with weakened immune system.

1. Clinical Presentation

- Meningitis can present as either an **acute fulminant illness** that progresses rapidly in a few hours or as a **subacute infection** that progressively worsens over several days.
- The **classic clinical triad** of meningitis is **fever, headache, and nuchal rigidity**. Nausea, vomiting, and photophobia are also common complaints.
- **Seizures** occur as part of the initial presentation of bacterial meningitis or during the course of the illness in 20–40% of patients. Generalized seizure activity and status epilepticus may be due to hyponatremia, cerebral anoxia.
- **Raised Intracranial Pressure (ICP)** is an expected complication of bacterial meningitis and the major cause of obtundation and coma in this disease.
- **Signs of increased ICP** include a **deteriorating or reduced level of consciousness, papilledema, dilated poorly reactive pupils, sixth nerve palsy, decerebrate posturing**.
- **Cushing reflex (bradycardia, hypertension, and irregular respirations)**.
- The **rash of meningococcemia for meningococcal meningitis**, which begins as a diffuse erythematous maculopapular rash resembling a viral exanthema; however, the skin lesions of meningococcemia rapidly become petechial. Petechiae are found on the trunk and lower extremities, in the mucous membranes and conjunctiva, and occasionally on the palms and soles.
- **On examination** meningeal signs are positive.

Meningeal signs (Can be found in any type of meningitis)

- i. Neck stiffness: Stiffness of neck & resistance to passive movements, with pain & spasm on attempted motion. The chin cannot be placed upon the chest.
- ii. Kernig's sign:
With the hip flexed, the knee is extended. Normally it can be done up to 135 degrees. In meningitis it is restricted due to spasm of hamstrings.
- iii. Brudzinski's sign:
Neck sign- on flexing the neck, there is flexion of hips & knees.
Leg sign- on flexing one leg, other leg also flexes.
- iv. Straight leg raising test (SLR):
With the patient supine & both legs extended, one leg is passively flexed at the hip keeping the knee extended. Normally it can be lifted to 90 degrees. It is restricted in meningitis.

2. Diagnosis

CSF Studies

The classic CSF abnormalities in bacterial meningitis are (1) Polymorphonuclear (PMN) leucocytosis (>100 cells/L in 90%), (2) decreased glucose concentration [<2.2 mmol/L (<40 mg/dL) and/or CSF/serum glucose ratio of <0.4 in 60%, hence it is mandatory that a blood glucose levels be estimated simultaneously with the CSF study], (3) increased protein concentration [>0.45 g/L (>45 mg/dL) in 90%], and (4) increased opening pressure (>180 mmH₂O in 90%). CSF bacterial cultures are positive in $>80\%$ of patients, and CSF Gram's stain demonstrates organisms in $>60\%$

3. Treatment

- i. Intravenous fluids.
- ii. Inj Mannitol 100mg 8 hourly to 6 hourly.
- iii. Inj Dexamethasone 4mg 8 hourly but first dose to be given 20 minutes before first dose of antibiotics.
- iv. Antimicrobial therapy of

- (Ceftriaxone – 2 g twice a day intravenously, cefotaxime, or Cefepime) A 2-week course of intravenous antimicrobial therapy is recommended for *Pneumococcal meningitis*.
- Or Ampicillin IV 2 gm 4 hourly (total 12 gm per day) for at least 3 weeks. Gentamicin is added in critically ill patients (2 mg/kg loading dose, then 7.5 mg/kg per day given every 8 hours and adjusted for serum levels and renal function). The combination of Trimethoprim (10–20 mg/kg per day) and Sulfamethoxazole (50–100 mg/kg per day) given every 6 hours may provide an alternative in penicillin-allergic patients.
- Vancomycin 2 g 12 hourly is the drug of choice for methicillin-resistant staphylococci and for patients allergic to penicillin (3 weeks).
- For *gram-negative bacillary meningitis*, the third-generation Cephalosporins - Cefotaxime (2 gm 4 hourly i.e. total 12 gm per day), Ceftriaxone (2 gm 12 hourly), and Ceftazidime (2 gm 8 hourly) - are efficacious for the treatment of, with the exception of meningitis due to *P. aeruginosa*, which should be treated with Ceftazidime, Cefepime, or Meropenem. (2 gm 8 hourly). A 3-week course of intravenous antibiotic therapy is recommended for meningitis due to gram-negative bacilli.

4. When to refer

- i. Lumbar puncture requires to be done and facility not available.
- ii. Presence of any focal deficit.

Note: If meningitis is suspected empirical antibiotic treatment has to be started immediately.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



21. TUBERCULOUS MENINGITIS

1. Clinical Manifestations

- The disease often presents subtly as headache and slight mental changes after a prodrome of weeks of low-grade fever, malaise, anorexia, and irritability.
- If not recognized, tuberculous meningitis may evolve acutely with severe headache, confusion, lethargy, altered sensorium, and neck rigidity.
- Typically, the disease evolves over 1–2 weeks, a course longer than that of bacterial meningitis.
- Since meningeal involvement is pronounced at the base of the brain, paresis of cranial nerves (ocular nerves in particular) is a frequent finding, and the involvement of cerebral arteries may produce focal ischemia.
- The ultimate evolution is toward coma, with hydrocephalus and intracranial hypertension.

2. Diagnosis

CSF Studies

In adults, the mean white blood cell (WBC) count (range, 0-4000 cells/ μ L) with lymphocytic predominance. The mean protein level in adults averages 224 mg/dL (range, 20-1000 mg/dl). The proportion with depressed glucose levels (< 45 mg/dL or 40% of serum glucose).

3. Treatment

- IV fluids
- Inj. Mannitol 100 cc thrice a day to four times a day, should be tapered and stopped to be overlapped with oral glycerol 1oz three times a day. Patient may have worsening of headache or get signs of raised ICT, mannitol should be reinstated and tapered again.
- For treatment of tubercular meningitis, first-line drugs are Isoniazid (5 mg/kg) along with Pyridoxine 50mg, Rifampin (10 mg/kg) given before breakfast, Pyrazinamide (25 mg/kg), and Ethambutol (15 mg/kg).
- Duration of treatment is up to 9 months to 1 year.
- Steroids are given as Inj. Dexamethasone 4 mg three times a day for 3 weeks to be continued as Tab Prednisolone 1mg/kg and tapered and omitted over one-and-a-half-month duration.

4. Complications

- Hydrocephalous.
- Vasculitis induced stroke.
- Drug induced hepatitis.

5. When to refer

- Patient having any focal deficit.
- Diminution of vision.
- Patient not improving on treatment.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



22. VIRAL MENINGITIS

1. Clinical features

Immuno-compromised adult patients with viral meningitis usually present with.

- Headache, fever, and signs of meningeal irritation coupled with an inflammatory CSF profile.
- Headache is almost invariably present and often characterized as frontal or retro-orbital and frequently associated with photophobia and pain on moving the eyes.
- Constitutional signs can include malaise, myalgia, anorexia, nausea and vomiting, abdominal pain and/or diarrhoea.
- Patients often have mild lethargy or drowsiness; however, profound alterations in consciousness, such as stupor, coma, or marked confusion do not occur in viral meningitis and suggest the presence of encephalitis or other alternative diagnoses.

2. Etiology

Acute Meningitis	
Common	Less Common
Enteroviruses (Coxsackie viruses, echoviruses, and human enteroviruses 68–71)	Lymphocytic Choriomeningitis virus
Varicella zoster virus	Cytomegalovirus
Epstein-Barr virus	
Herpes simplex virus 2	
Arthropod-borne viruses	
HIV	
Acute Encephalitis	
Common	Less Common
Herpesviruses	Rabies
Herpes simplex virus 1	Powassan virus
Varicella zoster virus (VZV)	Enteroviruses
Epstein-Barr virus (EBV)	Mumps
Arthropod-borne viruses	
Cytomegalovirus	

Table No.1- Aetiology

3. Diagnosis

The most important laboratory test in the diagnosis of **viral meningitis** is examination of the CSF. The typical profile is a lymphocytic pleocytosis (25–500 cells/L), a normal or slightly elevated protein concentration [0.2–0.8 g/L (20–80 mg/dL)], a normal glucose concentration, and a normal or mildly elevated opening pressure (100–350 mm H₂O).

4. Treatment: Acute Viral Meningitis

- Treatment of almost all cases of viral meningitis is primarily symptomatic and includes use of analgesics, antipyretics, and antiemetics.

- Fluid and electrolyte status should be monitored. IV fluids should be administered.
- Oral or intravenous Acyclovir may be of benefit in patients with meningitis caused by Herpes simplex virus-1 or -2 and in cases of severe Epstein-Barr virus or Varicella zoster virus infection. Seriously ill patients should receive intravenous Acyclovir (15–30 mg/kg per day in three divided doses for 7–10 days i.e. which can be followed by an oral drug such as Acyclovir (800 mg, five times daily. Patients with HIV, meningitis should receive highly active antiretroviral therapy).

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



23. VIRAL ENCEPHALITIS

1. Definition

In contrast to viral meningitis, where the infectious process and associated inflammatory response are limited largely to the meninges, in encephalitis the brain parenchyma is also involved. Many patients with encephalitis also have evidence of associated meningitis (meningoencephalitis) and, in some cases, involvement of the spinal cord or nerve roots (encephalomyelitis, encephalomyelorradiculitis).

2. Clinical Manifestations

In addition to the **acute febrile illness** with evidence of meningeal involvement characteristic of meningitis, the patient with encephalitis commonly has an **altered level of consciousness** (confusion, behavioural abnormalities), or a depressed level of consciousness ranging from mild lethargy to coma, and evidence of either focal or diffuse **neurologic signs** and symptoms.

Patients with encephalitis may have hallucinations, agitation, personality change, behavioural disorders, and, at times, a frankly psychotic state. **Focal or generalized seizures** occur in many patients with encephalitis.

Virtually every possible type of focal neurologic disturbance has been reported in viral encephalitis; the signs and symptoms reflect the sites of infection and inflammation.

The most commonly encountered focal findings are aphasia, ataxia, upper or lower motor neuron patterns of weakness, involuntary movements (e.g., myoclonic jerks, tremor), and cranial nerve deficits (e.g., ocular palsies, facial weakness).

3. Laboratory Diagnosis

3.1. CSF Examination

- CSF examination should be performed in all patients with suspected viral encephalitis unless contraindicated by the presence of severely increased intracranial pressure (ICP).
- The characteristic CSF profile is indistinguishable from that of viral meningitis and typically consists of a lymphocytic pleocytosis, a mildly elevated protein concentration, and a normal glucose concentration. A CSF pleocytosis (>5 cells/L) occurs in >95% of immunocompetent patients with documented viral encephalitis.

- In rare cases, a pleocytosis may be absent on the initial LP but present on subsequent LPs. Patients who are severely immunocompromised by HIV infection, glucocorticoid or other immunosuppressant drugs, chemotherapy, or lymphoreticular malignancies may fail to mount a CSF inflammatory response.
- CSF cell counts exceed 500/L in only about 10% of patients with encephalitis. Infections with certain arboviruses (e.g., EEE virus or California encephalitis virus), mumps, and LCMV may occasionally result in cell counts >1000/L, but this degree of pleocytosis should suggest the possibility of non-viral infections or other inflammatory processes.
- Atypical lymphocytes in the CSF may be seen in EBV infection and less commonly with other viruses, including CMV, HSV, and enteroviruses.
- However, persisting CSF neutrophilia should prompt consideration of bacterial infection, leptospirosis, amoebic infection, and non-infectious processes such as acute haemorrhagic leukoencephalitis. About 20% of patients with encephalitis will have a significant number of red blood cells (>500/L) in the CSF in a non-traumatic tap. The pathologic correlate of this finding may be a haemorrhagic encephalitis of the type seen with HSV; however, CSF red blood cells occur with similar frequency and in similar numbers in patients with non-herpetic focal encephalitis.
- A decreased CSF glucose concentration is distinctly unusual in viral encephalitis and should suggest the possibility of bacterial, fungal, tuberculous, parasitic, leptospiral, syphilitic, sarcoid, or neoplastic meningitis.

3.2. MRI, CT, EEG

- Patients with suspected encephalitis almost invariably undergo neuroimaging studies and often EEG. These tests help identify or exclude alternative diagnoses and assist in the differentiation between a focal, as opposed to a diffuse, encephalitic process.
- Focal findings on MRI in a patient with encephalitis should always raise the possibility of HSV encephalitis. Approximately 10% of patients with PCR-documented HSV

encephalitis will have a normal MRI, although nearly 80% will have abnormalities in the temporal lobe, and an additional 10% in extra temporal regions.

4. Treatment

Vital functions, including respiration and blood pressure, should be monitored continuously and supported as required. In the initial stages of encephalitis, many patients will require care in an intensive care unit.

- i. Intravenous fluids.
- ii. IV Mannitol 100 cc 12 hourly for 3 days

Seizures should be treated with standard anticonvulsant regimens (Inj. Diazepam 10 mg, IV or Inj. Midazolam 2 mg), and prophylactic therapy should be considered in view of the high frequency of seizures in severe cases of encephalitis.

- iii. Acyclovir is of benefit in the treatment of HSV and should be started empirically in patients with suspected viral encephalitis, especially if focal features are present, while awaiting viral diagnostic studies.

Adults should receive a dose of 10 mg/kg of Acyclovir intravenously every 8 hours (30 mg/kg per day total dose) for 14–21 days.

CSF PCR can be repeated at the completion of this course, with PCR-positive patients receiving additional treatment, followed by a repeat CSF PCR test.

Prior to intravenous administration, acyclovir should be diluted to a concentration 7 mg/ml. (A 70-kg person would receive a dose of 700 mg, which would be diluted in a volume of 100 ml.) Each dose should be infused slowly over 1 hour, rather than by rapid or bolus infusion, to minimize the risk of renal dysfunction. Care should be taken to avoid extravasation intramuscular or subcutaneous administration. The alkaline pH of Acyclovir can cause local inflammation and phlebitis (9%). Dose adjustment is required in patients with impaired renal glomerular filtration. Penetration into CSF is excellent, with average drug levels 50% of serum levels. Complications of therapy include elevations in blood urea nitrogen and creatinine levels (5%), thrombocytopenia (6%), gastrointestinal toxicity (nausea, vomiting, diarrhoea) (7%), and neurotoxicity (lethargy or obtundation, disorientation, confusion, agitation, hallucinations, tremors, seizures) (1%).

- iv. Antipyretics for fever

5. Complications

Aspiration pneumonia, stasis ulcers and decubitus ulcer, contractures, deep venous thrombosis and its complications and infections of indwelling lines and catheters.

6. When to refer

Ideally all suspected patients of viral encephalitis after starting therapy as mentioned above and patients of viral meningitis not improving over or worsening over 48 hours should be referred to a higher centre.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



24. EPILEPSY

1. Definition

This is a condition characterized by recurrent episodes of seizures which are paroxysmal abnormal discharges at high frequency from an aggregate of neurons in cerebral cortex.

2. Types of epilepsy

- Grand mal epilepsy or Tonic-Clonic Seizures
Aura, epileptic cry, sudden fall due to tonic convulsions followed by clonic convulsions and then prolonged sleep and depression. The attack lasts for 1-2 min. Tongue bite; urinary incontinence may occur.
- Petit mal epilepsy or Absence seizures - No aura, no loss of consciousness. Prevalent in children and episode lasts for few seconds. There is momentary loss of consciousness hardly for 5 seconds without loss of postural control. Presence of freezing or staring in one direction.
- Partial seizures - Most common seizure types occurring in 80% of epileptic patients. Seizure activity is restricted to a discrete area belonging to one cerebral hemisphere only.
- Other types of seizures: Myoclonic seizures, atonic seizures, tonic seizures.
- Febrile seizures- Young children develop seizures during high fever.

3. Investigations

Apart from routine laboratory investigations, patients of epilepsy can be further investigated with an EEG and MRI. from routine laboratory investigations.

4. Treatment

- Correct any metabolic cause (hypo or hyperglycaemia, hypo or hypernatremia).
- Stop drugs like Theophylline (CNS Stimulant) which may cause seizures.
- Look for any structural CNS lesions like Brain tumour, Vascular lesions, Meningitis, CVST or abscess and treat them.
- Avoid precipitating factors like sleep deprivation, fasting, video games.

4.1 Antiepileptic should be started when

- A history suggestive of recurrent epileptic seizure is established.
- An abnormal neurological examination.
- Presenting as Status Epilepsy.
- Abnormal EEG suggestive of general seizure.
- Family history of epilepsy.

To start anti epileptics in single isolated seizure is not yet established. Monotherapy should be the mantra of treatment.

4.2 Anti-epileptic drugs

Table No.1: Antiepileptic Drugs & their Doses with type of seizure

Seizure	Drug	Dose
Generalized onset tonic clonic convulsions	Valproic acid	750-2000 mg/day (20-60 mg/kg/day) In divided doses
	Phenytoin	300-400 mg/day (3-6 mg/kg/day for adult, 4-8 mg/kg/day for Children) BD doses
	Levetiracetam	1 gm – 3 gm per day
	Lamotrigine	1000-3000 mg/day BD doses
	Topiramate	150-500 mg/day, BD doses
Focal onset tonic clonic convulsions	Carbamazepine Lamotrigine	600-1800 mg/day (15-35 mg/kg, child) in BD doses

	Levetiracetam Oxcarbazepine	Same as Above Same as Above 900-2400 mg/day (30-45 mg/kg, child) BD doses
Typical Absence	Valproic acid Ethosuximide	Same as Above 750-1250 mg/day in BD doses, 20 – 40 mg per kg divided in BD doses
Atypical Absence, myoclonic, Atonic Seizures	Valproic Acid, Lamotrigine, Topiramate	Same as Above Same as Above

5. Status Epilepticus

Status Epilepticus refers to continuous seizures or repetitive discrete seizures with impaired consciousness in the interictal period for duration of 15-30 minutes traditionally but practically for generalized convulsive Status Epilepticus the duration is 5 minutes.

- It is a medical emergency and must be treated immediately.
- Monitor vital signs. Maintain airway, breathing, and circulation. Administer supplementary Oxygen by mask.
- Laboratory analysis-glucose, electrolytes, calcium, magnesium, creatinine, CBC and urine analysis.
- Establish intravenous access.
- Lorazepam (0.1 mg/kg) IV over 1-2 min, Repeat if no response after 5 minutes.
- Phenytoin 15-20 mg/kg, at a rate not to exceed 50 mg/min (Never mix Phenytoin with a 5%

dextrose solution) Put it in a normal saline solution to minimize the risk of crystal precipitation or Fosphenytoin 20 mg/kg iv to maximum 150 mg/minute.

- Correct any metabolic imbalances. Control hyperthermia.
- If seizures continue after 20 minutes, give maximum rate 50 mg/minute or Fosphenytoin 7 to 10 mg/kg to maximum rate of 150 mg/minute.
- If seizures continue after 20 minutes, give Phenytoin (20 mg/kg IV at 60 mg/min)
- Other Antiepileptic Drugs which can be given IV are - Valproate sodium 30 mg/kg IV bolus or IV Levetiracetam 10-50 mg/kg
- If seizures continue, consider administering general anaesthesia with medications such as Propofol, Midazolam, or Pentobarbital.
- All the Patients after initial IV Antiepileptic Drug should be followed by maintenance doses with the oral antiepileptic drugs regularly.

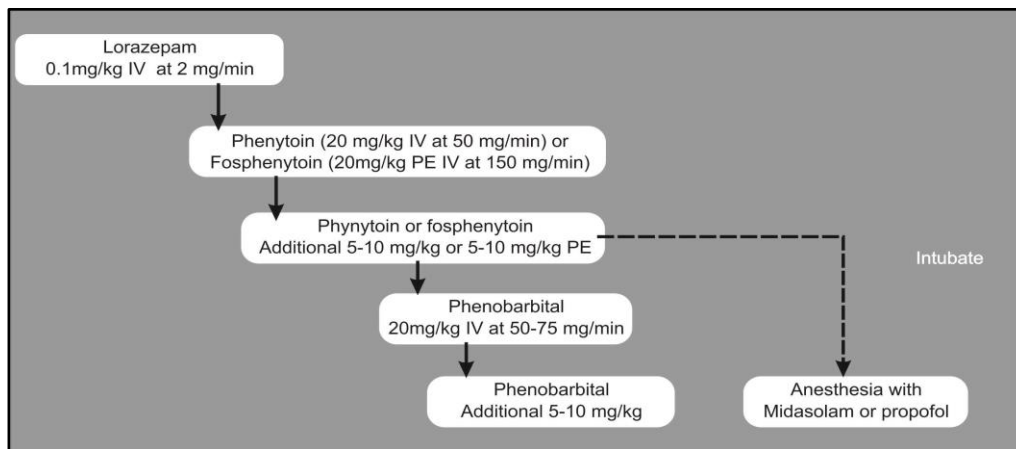
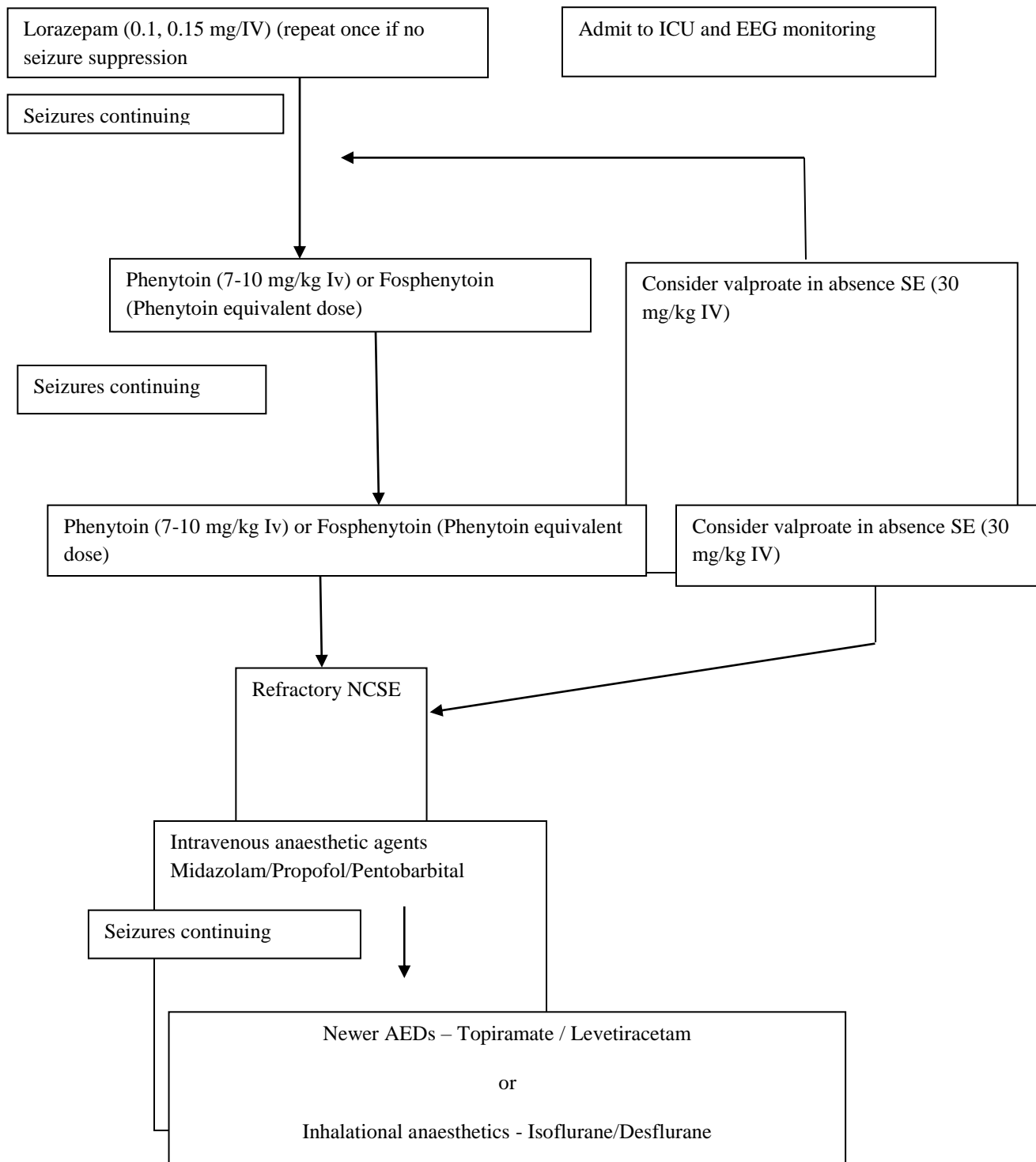


Figure-24.1: Management of Status Epilepticus algorithm

Figure 24.2: Pharmacological treatment algorithm of Non-Convulsive Status Epilepticus



Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Santhosh NS, Sinha S, Satishchandra P. Epilepsy: Indian Perspective, Ann Indian Acad Neurol. 2014 Mar; 17(Suppl 1): S3–S11. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4001222/>



25. GUILLAIN-BARRE SYNDROME (GBS)

1. Definition

Guillain-Barre syndrome (GBS) is an acute, frequently severe, and fulminant polyradiculopathy that is autoimmune in nature.

2. Clinical Features

GBS manifests as rapidly evolving areflexic motor paralysis with or without sensory disturbance. Usual pattern is an ascending paralysis. Weakness typically evolves over hours to a few days and frequently accompany by tingling dysesthesias in extremities. Legs are usually more affected than arms. Facial diparesis is present in 50% of affected individuals. The lower cranial nerves are also frequently involved, causing bulbar weakness with difficulty handling secretions and maintaining airway. Pain in the neck, shoulder or back is also common in the early stages.

Fever and constitutional symptoms are absent at the onset, and if present cast a doubt on the diagnosis. Deep tendon reflexes disappear within the first few days of onset. Bladder dysfunction may occur in severe cases but is usually transient. Autonomic involvement is common, usually presenting as wide fluctuation in blood pressure, postural hypotension & cardiac dysrhythmias.

Approximately 70% of cases of GBS occur 1-3 weeks after an acute infectious process, usually respiratory or gastro-intestinal.

3. Differential diagnosis

The diagnosis is made by recognizing the pattern of rapidly evolving paralysis with areflexia, absence of fever or other systemic symptoms, and characteristic antecedent events. Other disorders that may enter into the differential diagnosis include:

Acute myelopathies – prolonged back pain and sphincter disturbances.

Botulism – pupillary reactivity lost early.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015

Poliomyelitis–fever and meningismus is common.

4. Investigations

CSF analysis by lumbar puncture by the end of first week – Elevated CSF protein 100-1000 mg/dl without pleocytosis called as albuminocytological dissociation.

Can refer for electro-diagnosis- Demyelination or axonal involvement seen, according to the variant type

5. Treatment

Treatment should be initiated at the earliest. Each day counts. >2 weeks after the first motor symptoms, immunotherapy is no longer effective. Either high dose intra-venous immune globulin (IVIg) or plasmapheresis can be initiated, as they are equally effective.

- Intra-venous Immune globulin (IVIg): Total dose of 2 g/kg body weight divided into 5 doses given over 5 consecutive days.
- Plasmapheresis: 40-50 ml/kg plasma exchange four times over a week.

In the worsening phase of GBS, most patients require monitoring in a critical care setting, with particular attention to vital capacity, heart rhythm, blood pressure, nutrition, DVT prophylaxis and chest physiotherapy. Nearly 30% of patients with GBS require ventilator assistance.

6. When to refer: Any of the following

- Single breath count \leq 12/min
- Respiratory failure.
- Fulminant quadriparesis.
- Poor Gag reflex with risk of aspiration.
- Autonomic dysfunction
- Recurrent GBS.

2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Meena K, Khadilkar SV, Murthy JMK. Treatment guidelines for Guillain Barre Syndrome, Ann Indian Acad Neurol.[Internet] 2011 Jul; 14(Suppl1): S73–S81. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3152164/>



26. APTHOUS ULCERS

1. Introduction

Recurrent Aphthous stomatitis (RAS) is a common condition, restricted to the mouth, that typically starts in childhood or adolescence as recurrent small, round, or ovoid ulcers with circumscribed margins, erythematous haloes, and yellow or grey floors. A positive family history of similar ulcers is common, and the natural history is typically of resolution in the third decade of life.

2. Causes

- Some RAS cases involve a familial and genetic basis; approximately 40% of patients with RAS have a familial history, but inheritance may be polygenic with penetrance dependent on other factors.
- Most relevant studies have found hematinic (e.g. iron, folic acid, vitamin B-12) deficiencies in as many as 20% of patients with recurrent ulcers. In addition, deficiencies of vitamins B-1, B-2, and B-6 have been noted in some patient cohorts.

3. Symptoms

- Significant pain while chewing food. Minor aphthae are recurrent, painful, single or multiple, shallow surrounded by erythematous mucosa anywhere in the oral cavity. Small ulcers heal without scar, while larger and deep ulcers leave a scar.

- Fever, adenopathy, gastrointestinal symptoms are typically absent.

Important note –Repeated ulcers at the same site or slow healing ulcers with systemic symptoms e.g., uveitis, arthritis and fever adenopathy are worrisome. Malignancy should be excluded.

4. Treatment

4.1 Topical and systemic antibiotics

Tetracycline 250 mg dissolved in 180 ml of water and used as swish and spit 4 times a day for several days. Avoid in children and pregnancy.

4.2 Probiotics in powder form placed in oral cavity and swallowed 3 -4 times a day

4.3 Anti-inflammatory

Hydrocortisone pellets 5 mg kept at ulcer base and swallowed every 4 hours for 3-4 days. Triamcinolone 0.1 % applied to ulcers 2-4 times a day. Betamethasone (Betnesol) mouthwash - 0.5 mg tab dissolved in 5-10 ml of water for mouth wash 6 hourly during attack.

4.4 Immune modulators Levamisole

50 mg twice a day for three consecutive days for 4 weeks, no medication for 2 weeks; then Levamisole 150 mg tab, half tab twice daily for 3 days for 2 weeks.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Patil S, Reddy SN, Maheshwari S. Prevalence of Recurrent Aphthous ulceration in the Indian Population, J Clin Exp Dent. [Internet]. 2014 Feb; 6(1): e36–e40. [cited 2016 Jul 5] Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3935903/>



27. OESOPHAGEAL CANDIDIASIS

1. Introduction

Oesophageal candidiasis is an opportunistic infection of the oesophagus by *Candida albicans*. Occurs in immune compromised patients like AIDS, Post-

chemotherapy, uncontrolled Diabetes Mellitus, Chronic steroid therapy.



Figure 27.1: Endoscopic view of Oesophageal Candidiasis

2. Signs and symptoms

Oral lesions are painless but oesophageal lesions produce painful dysphagia. Discrete or confluent curdy plaques on the oesophageal mucosa.

3. Investigations

Demonstration of pseudo hyphae on wet smears and culture.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. America. Centers for Disease Control and Prevention (CDC) Information web-page. [cited 2016 Jul 5] Available from: <https://www.cdc.gov/fungal/diseases/candidiasis/thrush/>



28. DYSPEPSIA

1. Introduction

Nonspecific group of symptoms related to upper GI Tract. Also referred to as non-ulcer dyspepsia, functional dyspepsia, GERD.

2. Symptoms

Upper abdominal symptoms simulating an ulcer disease or heart burn with or without regurgitation, heaviness, post-prandial fullness or early satiety. Symptoms of gas in abdomen is common.

3. Red flag signs

Anorexia, weight loss, anaemia, dysphagia and mass in abdomen.

4. Investigations

History and upper GI-scopy.

5. Treatment

- Cap Omeprazole 20 mg once a day taken 45 min before breakfast for 4 weeks or Tab Ranitidine 150 mg twice a day before food 45 mins for 4-6 weeks.
- For those with dysmotility symptoms - Tab Domperidone 10 mg three times a day 30 mins before food.
- Antacids 2-3 teaspoon when symptomatic.
- Anti H. Pylori treatment is recommended for – those on long term NSAIDS, those with duodenal/gastric ulcers.

Treatment of H Pylori- Combination of

- Cap. Omeprazole 20 mg twice a day plus
- Cap. Amoxicillin 1gm BD or Tab. Metronidazole 500 mg BD for 14-days plus
- Tab Clarithromycin 500mg BD for 14 days

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Ghoshal UC, Singh R. Functional dyspepsia: The Indian Scenario. Supplement to JAPI. [Internet] March 2012, Vol 60. [cited 2016 Jul 5]
Available from:
http://www.japi.org/march_2012_special_issue_dyspepsia/02_functional_dyspepsia_the.pdf



29. GASTROESOPHAGEAL REFLUX

1. Introduction

GERD is a common disorder caused by retrograde flow of gastric contents through an incompetent gastroesophageal junction. There are two groups- erosive GERD and non-erosive GERD. Untreated may result in to oesophagitis, ulceration, stricture, and rarely adenocarcinoma.

2. Symptoms

Retrosternal pain, heart burn, regurgitation mostly after meals. Rarely present with chronic cough, laryngitis, bronchospasm, recurrent pulmonary infections, otitis media etc.

3. Treatment

- Antacid with or without alginate acid liquid or tab 2-3 chewed - 4-6 times a day 1/2-1 hour after a meal.
- Cap. Omeprazole 20 mg Once a day/BD OR Rabeprazole 20 mg Once a day or Pantoprazole 40 mg Once a day.
- Esomeprazole 40 mg Once a day, OR
- Lansoprazole 30 mg Once a day.
- All taken 45 mins before meals for 4-6 weeks.
- Add Prokinetic Domperidone 10 mg thrice a day 30 mins before meals if regurgitation is significant.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Poddar U. Diagnosis and management of gastroesophageal disease: An Indian perspective, Indian Pediatr. [Internet] 2013 Jan 8;50(1):119-26. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pubmed/23396785>



30. PEPTIC ULCER DISEASE

1. Introduction

Peptic ulcer is an important organic gastrointestinal disease.

2. Aetiology

Peptic ulcer disease (PUD) may be due to any of the following:

- H. pylori infection
- Drugs
- Lifestyle factors
- Severe physiologic stress
- Hypersecretory states (uncommon)
- Genetic factors

There is ulceration of the gastric and duodenal mucosa due to acid and pepsin.

3. Clinical features

- Obtaining a medical history, especially for peptic ulcer disease, H. pylori infection, ingestion of NSAIDs, or smoking, is essential in making the correct diagnosis. Gastric and duodenal ulcers usually cannot be differentiated based on history alone, although some findings may be suggestive.
- Epigastric pain is the most common symptom of both gastric and duodenal ulcers. It is characterized by a gnawing or burning sensation and occurs after meals—classically, shortly after meals with gastric ulcer and 2-3 hours afterward with duodenal ulcer. Food or antacids relieve the pain of duodenal ulcers but provide minimal relief of gastric ulcer pain.
- Duodenal ulcer pain often awakens the patient at night. About 50-80% of patients with duodenal ulcers experience nightly pain, as opposed to only 30-40% of patients with gastric ulcers and 20-40% of patients with non-ulcer dyspepsia (NUD). Pain typically follows a daily pattern specific to the patient. Pain with

radiation to the back is suggestive of a posterior penetrating gastric ulcer complicated by pancreatitis.

4. Alarm features that warrant prompt gastroenterology referral include the following

- Bleeding or anaemia
- Early satiety
- Unexplained weight loss
- Progressive dysphagia or odynophagia
- Recurrent vomiting
- Family history of GI cancer

5. Physical Examination

In uncomplicated PUD, the clinical findings are few and nonspecific and include the following:

- Epigastric tenderness (usually mild)
- Right upper quadrant tenderness may suggest a biliary aetiology or, less frequently, PUD.
- Guaiac-positive stool resulting from occult blood loss
- Malena resulting from acute or subacute gastrointestinal bleeding

6. Differential Diagnosis

- Acute Coronary Syndrome
- Aneurysm, Abdominal
- Cholangitis
- Cholecystitis
- Cholecystitis and Biliary Colic in Emergency Medicine
- Cholelithiasis
- Diverticular Disease

- Oesophageal Perforation, Rupture and Tears
- Oesophagitis
- Gastritis, Acute
- Gastritis, Chronic
- Gastroenteritis
- Gastroesophageal Reflux Disease
- Inflammatory Bowel Disease
- Viral Hepatitis

7. Approach Considerations

- Testing for *H. pylori* infection is essential in all patients with peptic ulcers. In most patients with uncomplicated peptic ulcer disease (PUD), routine laboratory tests usually are not helpful. Documentation of PUD depends on radiographic and endoscopic confirmation.
- If the diagnosis of PUD is suspected, obtaining CBC count, liver function tests (LFTs), amylase, and lipase may be useful. CBC count and iron studies can help detect anaemia, which is an alarm signal that mandates early endoscopy to rule out other sources of chronic GI blood loss.



7.1 *H. pylori* Testing

- Testing for *H. pylori* infection is essential in all patients with peptic ulcers.
- Endoscopic or invasive tests for *H. pylori* include a rapid urease test, histopathology, and culture. Rapid urease tests are considered the endoscopic diagnostic test of choice. The presence of *H. pylori* in gastric mucosal biopsy specimens is detected by testing for the bacterial product urease

7.2 Endoscopy

Upper GI endoscopy is the preferred diagnostic test in the evaluation of patients with suspected PUD. It is highly sensitive for the diagnosis of gastric and duodenal ulcers, allows for biopsies and cytological brushings in the setting of a gastric ulcer to differentiate a benign ulcer from a malignant lesion, and allows for the detection of *H. pylori* infection with antral biopsies for a rapid urease test and/or histopathology in patients with PUD. (See the images below.)

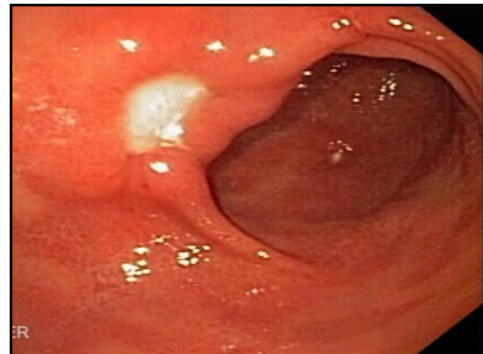


Figure-30.1: Endoscopic view of Peptic Ulcer

8. Treatment

Anti H. pylori treatment is recommended for patients on long term NSAIDS, bleeding peptic ulcer.

H. pylori treatment

Drugs	Dose(mg)	Frequency	Duration
Proton Pump Inhibitors (PPI)	20 mg	BD	14 days
Clarithromycin	500 mg	BD	
Metronidazole	400 mg	BD	
PPI	20mg	BD	14 days
Amoxicillin	1gm	BD	
Metronidazole	400 mg	TDS	
PPI	20 mg	BD	14 days
Amoxicillin	1 gm	BD	
Clarithromycin	500 mg	BD	

- Tab Ranitidine (150 mg) BD,
- Famotidine (40 mg) once a day equally efficacious, but takes longer time.
- Maintenance dose of PPI for patients on NSAIDS, IHD patients.

9. Do's and don'ts

- Avoid alcohol
- Stop smoking
- Avoid NSAIDs
- Prefer paracetamol, avoid foods that aggravate symptoms
- No role of bland diet
- Excess milk, meals at regular intervals.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Fashner J, GITU AC et al. Am Fam Physician. [Internet] 2015 Feb 15;91(4):236-242. [cited 2016 Jul 5] Available from: <http://www.aafp.org/afp/2015/0215/p236.html>



31. VOMITING

1. Introduction

Vomiting is the forceful expulsion of the gastric contents due to contraction of abdominal musculature and simultaneous relaxation of gastric fundus and lower oesophageal sphincter.

2. Causes

- i. Central (stimulation of vomiting centre) neurological disease raised intracranial pressure.
- ii. Vestibular system disorders
- iii. Drugs and toxins
- iv. Toxic and metabolic disorders such as ketoacidosis
- v. Systemic infections
- vi. Pregnancy
- vii. Psychogenic vomiting
- viii. Obstructive diseases of GIT
- ix. Acute gastritis, gastroenteritis
- x. Radiation exposure.

3. Investigations

Evaluation should exclude CNS causes and upper GI endoscopy to rule out upper GI pathology. Barium meal is recommended if upper GI scopy is normal. Psychogenic vomiting is considered after excluding organic cause.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



4. Treatment

- Intravenous fluids if dehydrated. Start oral fluids as soon as patient tolerates.
- Rule out gastric outlet obstruction then
- Inj. Metoclopramide 10 mg IM repeat after 6 hours if needed or
- Tab Mosapride 5 mg thrice a day, or
- Tab Domperidone 10 mg thrice a day or
- Tab Metoclopramide 10 mg thrice a day or
- Tab Prochlorperazine 5 mg thrice a day repeat after 4- 6 hrs if needed, or
- Tab/Inj. Ondansetron 8 mg stat dose and repeated 8 hourly
- In Pregnancy Tab/Inj. Promethazine 25 mg is safe in the first trimester.
- For motion sickness Tab Cyclizine 50 mg thrice a day.
- If vomiting is part of suspected acute abdomen, acute MI, refer for further evaluation.

32. CONSTIPATION

1. Introduction

Common causes of constipation are Diet deficient in roughage Ignoring the urge to defecate e.g. due to immobility, lack of exercise.

Other causes are neurological, Myxoedema, drugs like Atropine, Codeine, etc and Malignancy

2. Symptoms

Constipation itself is a symptom. When associated with inability to pass flatus, severe abdominal pain, or vomiting there may be the need for urgent referral to a surgeon.

3. Signs

Constipation, if associated with frequent high pitched bowel sounds or absent bowel sounds

4. Investigations

- Stool routine examination
- Stool for occult blood
- Sigmoidoscopy/Colonoscopy

A rectal examination with a short length colonoscopy is a must for all patients with recent onset of constipation irrespective of bleeding per rectum. When acute, the constipation may be a part of a serious illness such as acute bowel obstruction. These patients present with abdominal pain, vomiting and distension and non-passage of flatus. These patients should be referred immediately to a higher center after rectal examination, passage of rectal tube (for passage of flatus) and a plain X-ray abdomen.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Longstreth GF. Approach to adult with nausea and vomiting. Uptodate.com. Last updated May 12th, 2016. [cited 2016 Jul 5]
Available from: http://www.uptodate.com/contents/approach-to-the-adult-with-nausea-and-vomiting?source=outline_link&view=text&anchor=H23#H23



5. Treatment

Bisacodyl, oral, Adults 10-20 mg at night

Or

Psyllium (Isaphghul husk), oral, Adults 5-10 ml once or twice a day

Or

Liquid Paraffin, oral - 10-30 ml at night Or Glycerol Suppositories Adults 4 mg at night; 1 mg at night Or Lactulose liquid, oral, Adults 15-30 ml orally daily until response then 10-20 ml daily

- **Alternative treatment** - Magnesium Sulphate, oral, Adults 5-10 g in a glass of water, once or twice daily

6. Do's and Don'ts

- Advice patients to take plenty of fluids, high fibre diet – green leafy vegetables, fruits, avoid caffeinated drinks.
- Regular walk and exercise ½ to 1 hour daily, abdominal exercise.
- To use Indian closet as far as possible (this will straighten the anorectal angle).
- Avoiding suppression of urge to defecate, (making a regular habit).
- Avoid purgative frequently to treat constipation, as it may be habit forming.

Suppository or simple enema is preferred in IHD

33. IRRITABLE BOWEL SYNDROME

1. Definition

A constellation of gastrointestinal symptoms associated with lower bowel symptoms that occur in absence of an organic disease.

2. Symptoms

Clinically the diagnosis is made when continuous or recurrent symptoms of abdominal pain are associated with any of the three features viz. Relief by defecation and / or onset with change in stool frequency or consistency for at least 3 months. Supportive symptoms of IBS include passage of mucous, abnormal stool passage (straining, urgency, and feeling of incomplete evacuation) and feeling of abdominal fullness. Exclude IBS if individual has alarm symptoms such as fever, weight loss, bleeding per rectum or anaemia

3. Treatment

- Stool bulking agents – High fibre diet
- Antispasmodics – Anticholinergic drugs (Dicyclomine)

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Spiller R, Aziz Q, Creed F, Emmanuel A, et al. Guidelines on the irritable bowel syndrome: mechanisms and practical management, Gut. [Internet] 2007; 56:1770–1798. [cited 2016 Jul 5] Available from: http://www.bsg.org.uk/pdf_word_docs/ibs.pdf

- Antidiarrheals – Tab Loperamide 2 to 4 mg daily for several days
- Anti-depressants
 - IBD Diarrhoeal type –TCA (Imipramine)
 - IBD constipation type – SSRI (paroxetine)
- Calcium channel activators – Lubiprostone 8 micrograms for 3 months
- Any IBS patient with change in presentation e.g. change in bowel habit requires re-evaluation.

4. Do's and Don'ts

- Diet should contain high fibre and supplemented with bulk forming agents such as Isaphghul husk
- Avoid caffeine and alcohol
- Avoid milk and other dietary constituents, which worsens the symptoms
- Psychotherapy may be helpful in selected cases



34. ULCERATIVE COLITIS

1. Introduction

Ulcerative colitis is a chronic inflammatory bowel disease of unknown aetiology.

2. Symptoms

During the first attack the patient often presents with bloody diarrhoea, with systemic symptoms of low to moderate fever, backache, arthralgia. The first attack is a close mimicker of acute infective diarrhoea. A stool examination followed by sigmoidoscopy is mandatory, especially if the bloody diarrhoea persists for more than a month. It is important to exclude amoebic infection prior to institution of steroids. Rectum is uniformly involved in these patients. Frequency of stool can provide information on severity of the disease: mild (2 - 4 stools/day), moderate (4 - 6 stools/day) or severe (>6 stools/day). During remission, patient may be asymptomatic or may have extra-intestinal symptoms.

3. Treatment

These patients require a referral to a tertiary unit. Aim is induction of remission in acute stage and then maintenance of remission.

Therapeutic decisions depend on disease activity and extent. Patients with severe disease require hospital admission, whereas those with mild/moderate disease can generally be managed as outpatients.

- **Disease extent can broadly be divided into distal and more extensive disease.**
 - Distal disease (Proctitis/Proctosigmoiditis): Topical management is appropriate.
 - Extensive disease: Oral or parenteral therapy is the mainstay of treatment.

3.1 Choice of drugs

Mesalamine preparations and Steroid preparations

Treatment of active left sided, or extensive UC:

- Mesalazine 2 - 4 g daily or Balsalazide 6.75 g daily are effective first line therapy for mild to moderate active disease.

- Sulphasalazine has a higher incidence of side effects compared with newer 5-ASA drugs. Selected patients, such as those with a reactive arthropathy, may benefit.
- Prednisolone 40 mg daily is appropriate for patients in whom a prompt response is required, or those with mild to moderate active disease, in whom Mesalazine in appropriate dose has been unsuccessful. It should be reduced gradually according to severity of the patient.
- Long-term treatment with steroids is undesirable. Patients with chronic active steroid dependent disease should be treated with Azathioprine 1.5-2.5 mg/kg/day
- Topical agents (either Steroids or Mesalazine) may be added to the above agents. Although they are unlikely to be effective alone, they may benefit some patients with troublesome rectal symptoms.
- Severe UC: close monitoring at a tertiary centre.

3.2 Maintenance of remission

- Lifelong maintenance therapy is generally recommended for all patients, especially those with left sided or extensive disease, and those with distal disease who relapse more than once a year.
- Discontinuation of medication may be reasonable for those with distal disease who have been in remission for 2 years and are averse to such medication. However, there is some evidence that maintenance therapy reduces the risk of colo-rectal cancer.

For the maintenance of remission in UC:

- Most patients require lifelong therapy, although some patients with very infrequent relapses (especially if with limited extent of disease) may remain in remission without maintenance therapy.

- Oral Mesalazine 1 – 2 g daily or Balsalazide 2.5 g daily should be considered as first line therapy.
- Sulphasalazine 2–4 g daily has a higher incidence of side effects compared with newer 5-ASA drugs.
- Topical Mesalazine 1 g daily may be used
- Steroids are ineffective at maintaining remission.
- Azathioprine 1.5 – 2.5 mg/kg/day or Mercaptopurine 0.75 – 1.5 mg/kg/day are effective at maintaining remission in UC. However, in view of toxicity they should be reserved for patients who frequently relapse despite adequate doses of amino-salicylates, or are intolerant of 5-ASA therapy. It is a common practice to continue amino-salicylates with

azathioprine, but limited evidences that this is necessary.

Important Note

- Regular surveillance is necessary for UC lasting for more than 10 years.
- Explain to the patient the chronic nature of the disease and continuation of maintenance
- Treatment for life with regular follow-up. Risk of colonic cancer after 10 years of disease onset must be explained

3.3 Do's and Don'ts

Milk is preferably avoided during the acute phase of illness.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Kornbluth A, Sachar DB. Ulcerative colitis in adults. Am J Gastroenterol. [Internet] 2010; 105:500; doi:10.1038 / ajg.2010.52; published online 23 February 2010. [cited 2016 Jul 5]
Available from: <http://gi.org/guideline/ulcerative-colitis-in-adults/>



35. AMOEBIC LIVER ABSCESS

1. Introduction

Liver abscess is the commonest extra-intestinal form of amoebiasis, caused by *E. histolytica* infection but occurs in only less than 1% of *E. histolytica* infections. The disease usually affects young males, particularly chronic alcoholics, in endemic areas. Patients commonly affected are between 20 to 40 years of age with residence in or recent travel to or emigration from an endemic region.

2. Clinical Manifestations

2.1. History

The signs and symptoms of amoebic liver abscess often are nonspecific, resembling those of pyogenic liver abscess or other febrile diseases.

- Abdominal pain
- Weight loss
- Fever with rigors
- Diarrhoea/Dysentery

2.2. Physical Examination

- Fever is the most common sign and is found in as many as 99% of cases.
- Hepatomegaly is present in some cases.
- Signs of complications
- Signs of peritoneal irritation, such as rebound tenderness, guarding, and absence of bowel sounds, are present when the abscess ruptures in the peritoneal cavity. Peritonitis occurs in 2-7% of cases.
- Pericardial friction rub can be audible when the abscess extends into the pericardium. This sign is associated with very high mortality.
- Signs of pleural effusion are present when the abscess ruptures in the pleural cavity

3. Investigations

3.1. Laboratory Studies

Examination of the stool for hematophagous trophozoites of *E. histolytica* must be made on at least 3 fresh specimens because the trophozoites are very sensitive and may be excreted intermittently

3.2. Imaging Studies

- Ultrasonography is the preferable initial diagnostic test. It is rapid, inexpensive, and is only slightly less sensitive than CT scan (75-80% sensitivity vs 88-95% for CT scan).

4. Treatment

4.1 Medical Management

- Tab. Metronidazole 800 mg three times orally (or IV, if necessary) daily for 5-10 days or Tab. Tinidazole 600 mg 2 times a day for 7-10 days
- If the patient is very toxic, Inj. Metronidazole 500 mg given 8 hourly until patient improves. Switch over to oral therapy whenever possible. Followed by Diloxanide Furoate (luminal agent for cysts) 500 mg three times a day for 10 days
- Chloroquine 600 mg orally daily 2 days, followed by 300 mg daily for 2 weeks; dose is calculated as chloroquine base. Drug is active against *E. histolytica* trophozoites

4.2 Indications for drainage of an abscess

- If pyogenic abscess cannot be excluded
- No improvement with medical therapy in 72 hours
- Impending rupture of abscess (severe pain, pleuritic pain, hiccups) - one very close to the surface of the liver
- Large left lobe abscess, to prevent rupture in to the pericardium

4.3 Follow-up

- Monitor the patient for resolution of symptoms with medical treatment and aspirate if there is any indication.
- Abscess cavity may persist for several weeks even after cure of infection. Frequent US scan is unnecessary unless patient develops fever etc. Scan may be repeated after 4 - 6 weeks, after the patient becomes asymptomatic.

5. Do's and Don'ts

- Avoid taking alcohol, specifically if on treatment with Metronidazole
- Avoid contaminated food and drinking water. Vegetables should be cooked or washed well.
- Use boiled water (kills the cyst) or bottled water from a known source.

- Maintain good hygiene during food intake to prevent enteric infections

6. Refer

Patients with abscesses that are large or not responding to treatment will need to be referred to a physician or surgical specialist.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Brailita DM, Anand BS. Amebic Hepatic Abscesses. Medscape.com. Last updated April 15, 2015. [cited 2016 Jul 5]
Available from: <http://emedicine.medscape.com/article/183920-overview>



36. PYOGENIC LIVER ABSCESS

1. Introduction

Liver abscess constitutes about 48% of all visceral abscesses. Pyogenic liver abscess is usually caused by spread of infection from peritoneum, abdominal viscera such as appendicitis / diverticulitis / portal Pyaemia or diseases of biliary tract. It is most commonly caused by coliform organisms.

2. Clinical Manifestation

Fever, abdominal pain, toxemia, features of associated problems such as appendicular pain/ mass etc. Mostly abscesses are small and multiple.

3. Investigations

Diagnostic investigations include total counts, USG scan of the abdomen, blood culture, and pus culture. CECT and MRI is seldom indicated.

4. Treatment

4.1. Drainage

Percutaneous catheter or open surgical- remains the mainstay of treatment for a large abscess

Patient should be kept nil by mouth and given IV fluids if toxic and sick.

4.2 Recommended antibiotics

Metronidazole plus Ampicillin and Gentamicin, Ciprofloxacin, or a third-generation Cephalosporin

Standard dosage

- Ceftriaxone 2 g intravenously every 24 hours, or Cefotaxime 2 g intravenously every 8 hours.
- Initial empirical treatment should include broad spectrum antibiotics.
- Various combinations recommended are:
Metronidazole: 500 mg I/V three times daily
Ampicillin: 2 g I/V 6 hourly

Gentamicin: 2 mg/kg load, then adjust for renal function

Ciprofloxacin: 400 mg I/V 12 hourly for 10 days

or

Inj. Ceftriaxone 1-2 g IV every 12 hours 2 times a day

or

Inj. Cefotaxime 2 g 8-hourly for 10 days

- Combination of Amoxicillin + Ciprofloxacin +Metronidazole is also a recommended schedule
- In the elderly or those with renal impairment: A Penicillin (such as Amoxicillin) plus an injectable Cephalosporin (such as Cefotaxime or Cefuroxime) plus Metronidazole is recommended
- Inj. Penicillin – allergic patients: Ciprofloxacin plus Clindamycin
- Once the sensitivity is known, antibiotic therapy is amended accordingly. Duration of therapy is usually from 2–4 weeks or longer depending on number of abscesses and the clinical response.

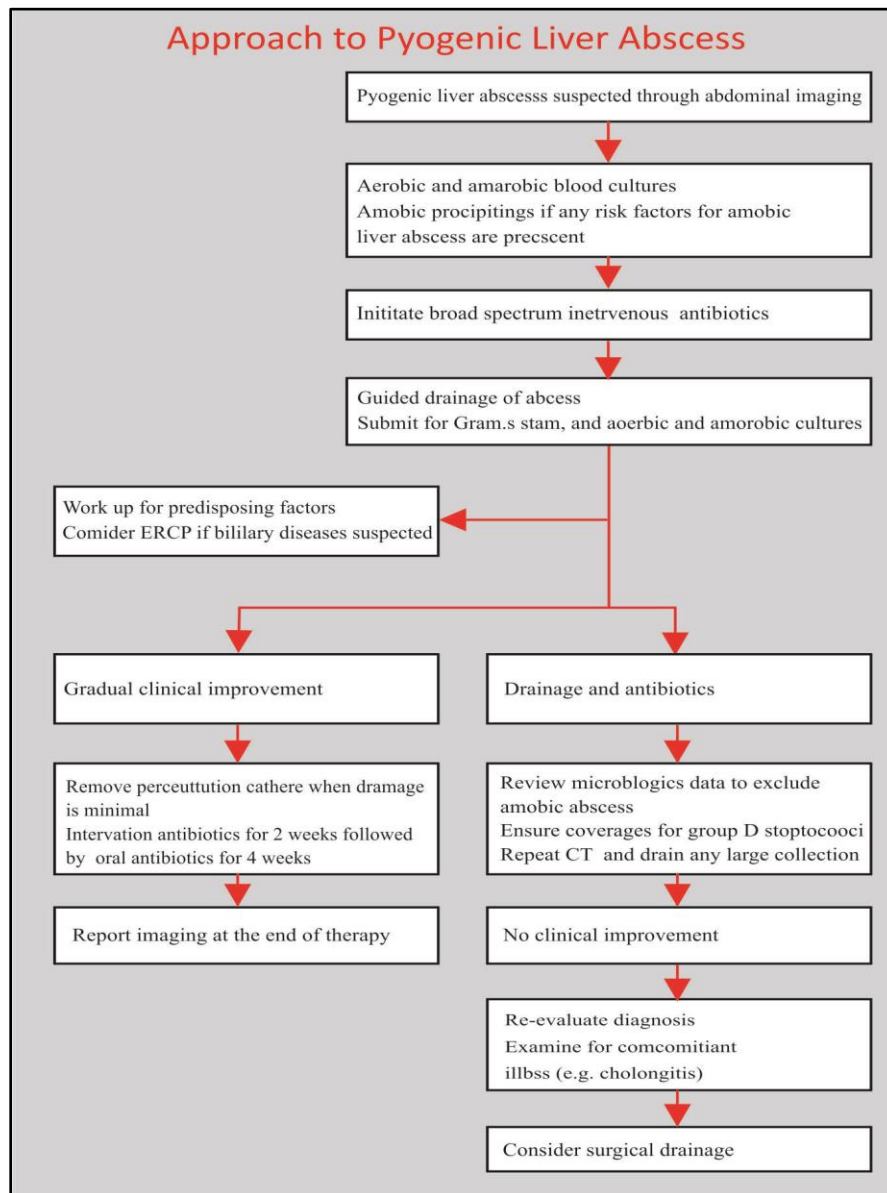
5. Follow-up

- Monitor for clinical improvement and modify the therapy based on culture sensitivity report.
- Abscess should always be drained.
- Surgery is considered if no improvement with medical treatment and percutaneous drainage in 4-7 days.

Important Note

1. Avoid taking alcohol, specifically if on treatment with Metronidazole.
2. Maintain good hygiene regarding food intake to prevent enteric infections.

Figure-36.1: Approach to Pyogenic Liver Abscess



Bibliography

1. Casper DL, Fauci AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Dutta A, Bandyopadhyay S. Management of Liver abscess, Medicine Update. [Internet] 2012, Vol. 22. [Cited 2016 Jul 5]
Available from: http://www.apiindia.org/pdf/medicine_update_2012/hepatology_04.pdf



37. INTESTINAL PROTOZOAL INFECTION

1. Amoebiasis

Entamoeba Histolytica is the most prevalent intestinal protozoa in India.

1.1 Clinical Features

Intestinal Amoebiasis / Amoebic Dysentery:

Patient with acute amoebic dysentery present with a 1-2-week history of abdominal pain, tenesmus, and frequent loose stools containing **blood and mucus**. Fever may or may not be present.

1.2 Investigations

Despite the presence of ulcerations and occult blood in stools, leucocytes may not be present in stools. A definite diagnosis is made by the demonstration of *E. Histolytica* cysts/ trophozoites in stools by a WET MOUNT PREPARATION.

1.3 Treatment

The drug of choice for amoebic colitis is Tinidazole/Metronidazole

Doses are:

- Tinidazole: 2 g/day with food for 3 days or
- Metronidazole: 750 mg TDS orally / IV for 5-10 days.

Treatment of intraluminal Amoebiasis is Diloxanide Furoate 500 mg PO-TID for 20 days or Paromomycin 30 mg/kg qd in 3 divided doses for 5-10 days.

2. Giardiasis

It is a parasitic infection caused by *Giardia Lamblia* a single celled organism.

2.1 Clinical Features

2.1.1 Acute symptoms include:

- Crampy abdominal pain
- Watery diarrhoea, vomiting and fever which last for few days.

2.1.2 In the chronic stage:

Patients have bloating, nausea, abdominal fullness, epigastric or substernal burning, malaise and fatigue.

Although severe forms of chronic giardiasis may occur in otherwise healthy individuals, they are common in patients with Hypoglobulinemia, particularly IgA deficiency in association with lymphoid hyperplasia of the bowel. Chronic

giardiasis may contribute to protein-energy malnutrition in children.

Protein-losing enteropathy has also been described.

2.2. Investigations

Diagnosis is made by stool examination and looking for cysts and trophozoites.

Parasites are best seen in fresh watery stools.

Duodenal aspirate is better for isolation but invasive.

2.3. Treatment

Treatment is Metronidazole 400 mg TID for 5 days orally or Tinidazole 2 gm PO once is equally effective.

Furazolidone 6 mg/kg q.i.d for 7-10 days is effective and well tolerated in children.

3. Spore forming protozoa

These include:

- *Cryptosporidium parvum*
- *Isospora belli*
- *Cyclospora cayetanensis*
- *Microsporidia*

The spectrum of diseases caused by these is as follows:

3.1 Asymptomatic infection

This can be seen in immunocompetent as well as immunocompromised patients.

3.2 Acute infectious diarrhoea in immunocompetent hosts

Acute watery diarrhoea with abdominal pain, with malaise with or without fever can be seen in all except microsporidia. (generally pathogenic only for immunocompromised patients)

3.3 Infections in immune compromised hosts

All spore-forming protozoa have a predisposition for more frequent and prolonged infections in patients who are immunodeficient.

Patients with HIV especially with low CD4 count (<50-100) are more prone.

Severe life-threatening watery diarrhoea, dehydration and chronic malabsorption leading to lethargy, failure to thrive, and malnutrition may occur.

3.4. Investigations

Stool examination with a wet mount preparation is required for isolation of the parasites.

Modified acid fast staining is used to identify *Cryptosporidium*, *Isospora* and *Cyclospora* as these are acid fast.

3.5. Treatment

PATHOGEN	TREATMENT
Cryptosporidium	Nitazoxanide 500 mg b.i.d for 3 days Paromomycin 30 mg/kg per day divided
Isospora	Cotrimoxazole (TMP/SMX,160/800 MG) four times a day for 10 days and then 3 times a day for 21 days
Microsporidia	No definite drug but Albendazole 400- 800 mg/day divided in b.i.d can be given
Cyclospora	Cotrimoxazole (TMP/SMX, 160/800 MG) twice daily for 7 days

Supportive treatment with ORS or iv fluids for fluid and electrolyte supplementation is necessary.

4. Intestinal helminthic (nematodes) infections

Nematodes are round worms infesting the intestines. The infective forms are the eggs/larvae transmitted by the feco-oral route.

Generally seen in low socio economic states with poor sanitation and hygiene.

Their life cycle is complex with stage of passage through lungs in some worms (ascaris, hookworm, strongyloides) leading to various manifestations like LOEFFLER'S SYNDROME.

Several clinical signs and symptoms can occur as follows:

Lung invasion - Loeffler's or Loeffler's like syndrome (ascariasis, hookworm infections, strongyloidiasis)

- Fever
- Cough
- Blood-tinged sputum
- Wheezing
- Rales
- Dyspnoea
- Substernal pain
- Pulmonary consolidations

- Eosinophilia
- Urticaria
- Asthma
- Angioneurotic oedema

Intestinal invasion:

- May be asymptomatic (small number).
- Abdominal pain (usually vague).
- Abdominal cramps/colic.
- Diarrhoea.
- Vomiting (rarely).
- Constipation (occasionally).
- Intestinal obstruction due to worm mass.

The common nematode infections are described below:

4.1 Ascariasis

Ascaris lumbricoides is the largest of the intestinal nematodes. Symptoms can be divided into 2 categories: early larvae migration and late mechanical effects.

In the early phase (4-16 hours after eggs ingestion), respiratory symptoms occur because of migration of larvae through the lungs with symptoms of eosinophilic pneumonia (Loeffler Syndrome). Patient may have fever, non-productive cough, dyspnoea or wheezing.

In the late phase (6-8 weeks after egg ingestion) gastrointestinal symptoms may occur. Patient may vomit out worms from mouth, nose.

Diffuse abdominal pain, nausea, vomiting, biliary and intestinal obstruction, appendicitis and pancreatitis.

Ascaris worms invade the biliary duct and cause pancreatic-biliary ascariasis. The most common presenting feature is abdominal pain, observed in 98% of patients. Less common features include ascending cholangitis, acute pancreatitis, and, rarely, obstructive jaundice.

4.1.1 Diagnosis

i. In early infection (larval migration):

- CBC may show peripheral eosinophilia
- Sputum may show Charcot laden crystals.
- Chest X Ray may show patchy infiltrates of eosinophilic pneumonia.

ii. In the late stage (adult worm):

- Stool microscopic examination shows eggs.
- USG may be used for ascariasis related biliary disease.
- CT abdomen may show adult worms or obstruction due to worm mass.

4.1.2 Treatment:

Albendazole 400mg one dose orally is the drug of choice OR Mebendazole 100 mg twice daily for 3 days. It is contraindicated in pregnancy.

4.2 *Ancylostoma Duodenale*: (Hookworm)

Many patients may be asymptomatic carriers.

4.2.1 Clinical features

The symptoms are classified into 2 phases:

In the migratory phase:

Pruritus and erythema and Vesiculation will occur once the filariform larvae have penetrated the skin of the feet and hands. This is called as “**ground/dermal itch**” in people who goes bare feet.

The pulmonary symptoms may develop with *A. Duodenale* during the phase of lung migration like mild transient pneumonitis.

In the intestinal phase:

Patients may present with abdominal pain (often epigastric).

Inflammatory diarrhoea, with eosinophilia.

Chronic hookworm infection:

Generally, presents with Iron deficiency anaemia, Hypoproteinaemia.

4.2.2 Diagnosis

Demonstration of eggs in stool examination. Sensitivity improved with stool concentration.

Microcytic hypochromic anaemia with proteinemia may be seen in chronic cases. Eosinophilia may be present.

4.2.3 Treatment

Albendazole 400 mg once / Mebendazole 500 mg once / Pyrantel Pamoate 11 mg/kg for 3 days.

Treat anaemia with iron supplements.

4.3 *Enterobiasis*

Enterobius vermicularis / pinworm is prevalent in most tropical countries.

The adult worms migrate to perianal area at night and release up to 10,000 eggs which after hatching are transmitted by hand to mouth passage.

4.3.1 Clinical features

The infection is generally asymptomatic.

The primary symptoms of pinworm infestation occur at night which include pruritus in the perianal region.

Heavy infection can cause abdominal pain and weight loss.

4.3.2 Diagnosis

Since pinworm eggs are not released in faeces, stool examination is not informative.

Detection of worms / eggs in perianal cellophane swab / cellophane tape applied in the early morning establishes diagnosis.

4.3.3 Treatment

Single dose Mebendazole 100 mg once / Albendazole 400 mg once.

4.4 *Strongyloidiasis*

Strongyloides stercoralis is the only intestinal nematode with the ability to replicate inside humans, thus leading to repeated **auto infection**.

In immunocompromised hosts it can lead to invasive and disseminated infection which can be fatal.

4.4.1 Clinical features

- Many patients are asymptomatic.

- Cutaneous manifestations include recurrent Urticaria involving buttocks and wrists.
- “**Larva currens**”: Cutaneous migration of larvae results in serpiginous eruption which is pruritic, erythematous.
- Burning or colicky **abdominal pain, often epigastric**, occurs and is associated with **diarrhoea** and the passage of mucus. Pain is aggravated by food.
- Some patients with strongyloidiasis report nausea, vomiting, and weight loss, with evidence of **malabsorption** or of protein-losing enteropathy.
- Massive larval invasion of the lungs and other tissues may occur with hyper infection, usually in immunocompromised hosts.
- Disseminated infection in immunocompromised hosts severe generalized abdominal pain, diffuse pulmonary infiltrates, ileus, shock, and meningitis or sepsis due to gram-negative bacilli may occur.

4.4.2 Diagnosis

- Stool examination for rhabditiform larvae is diagnostic.
- Duodenal aspirate may reveal the larvae if stool is persistently negative.
- In disseminated infections filariform larvae should be sought in stool, sputum, broncho-alveolar lavage etc.

Bibliography

1. Casper DL, Fauci AS, Harrison’s Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson’s Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Wright SG. Protozoan infections of the gastrointestinal tract, Infect Dis Clin North Am. [Internet] 2012 Jun;26(2):323-39. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pubmed/22632642>



4.4.3 Treatment

Ivermectin (200 mcg/kg daily for 2 days) is most effective. In cases with disseminated infection extend the duration for 5 to 7 days or up to clearance of parasites. Albendazole (400 mg daily for 3 days.) is also effective.

4.5 Trichuris Trichiura

- Also called as whip worm.
- Most infections are asymptomatic. Heavy infections can cause gastro intestinal symptoms.
- Common in low socio economic status with children especially affected.

4.5.1 Clinical features

- Usually mild symptoms with abdominal pain, anorexia, diarrhoea which can be bloody/mucoid.
- Rectal prolapse may occur in massive infections in children.

4.5.2 Diagnosis

- Stool examination for eggs.
- Proctoscopy for adult worms.

4.5.3 Treatment

Mebendazole 500 mg / Albendazole 400 mg daily for 3 days.

38. ASCITES

1. Causes

Cirrhosis is the common cause of ascites. Other causes are malignancy, cardiac failure, tuberculous ascites, pancreatitis, nephrotic syndrome, alcoholic hepatitis, acute liver cell failure etc.

2. Physical Exam

- Almost all patients with cirrhosis severe enough to cause ascites will have stigmata of cirrhosis – spider angiomas, palmar erythema, and caput medusae.
- Elevated JVP should raise suspicion of heart failure or constrictive pericarditis as a cause, although cirrhosis with tense ascites or pulmonary HTN may cause this.
- Sister Mary Joseph nodule with ascites may be caused by gastric or colon CA, HCC, or lymphoma. If found, FNAC can provide a rapid tissue diagnosis.

3. Investigations

- Ultra-sonography of abdomen is useful to confirm/refute presence of ascites, cirrhosis, splenomegaly, biliary obstruction, vessel patency, signs of portal hypertension, and cancer.
- CT-abdomen, tumour markers, HBsAg Anti HCV, serum amylase are other investigations.
- Abdominal paracentesis

Along with history and physical exam, ascitic fluid analysis helps in the diagnosis of aetiology. It is a safe procedure ascitic fluid analysis.

Appearance

- Turbid/cloudy – 98% sensitive but only 23% specific for SBP.
- Milky – Chylous ascites, TG level usually greater than serum TG level and >200 mg/dl.
- Bloody/ink – Usually a traumatic tap, but seen in 50% of patients with HCC and 22% with malignancy overall.
- Dark brown – If bilirubin level is higher than in serum, worry about ruptured gallbladder or perforated duodenal ulcer.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015

Lab tests

- In general, start with cell count and differential, TP and albumin when uncomplicated ascites due to cirrhosis is suspected. Culture is also usually sent.
- In patients with PMN >250, only 50% of cultures grow bacteria if sent down to lab in a syringe or plain tube. 80% grow bacteria if inoculated into blood culture vials at bedside (prior to antibiotics).
- Glucose < 50, LDH > upper limit of normal for serum, TP >1 and culture results can help differentiate secondary from spontaneous peritonitis.
- SAAG (serum albumin and ascitic albumin gradient) > or = 1.1 has ~97% accuracy for portal hypertension.
- TP > or = 2.5 can help differentiate cardiac from cirrhosis ascites.
- When PMN > or = 250, but less than 50% of WBC, consider peritoneal carcinomatosis and tuberculous ascites.

4. Management

- Goal is to minimize ascitic fluid volume and peripheral oedema without intravascular volume depletion.
- First line treatment includes 2 grams per day sodium restricted diet and oral diuretics (Spironolactone / Lasix).
- The usual diuretic regimen is single morning doses of 100 mg Spironolactone and 40 mg Frusemide (Lasix).
- The ratio of 100:40 generally maintains normokalemia. Usual max dose is 400 mg and 160 mg per day.
- Other specific management as per the cause of ascites.

5. When to Refer

Acute ascites, bleeding tendency, refractory ascites, hepatic encephalopathy.

2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Shah R, Katz J. Ascites. Medscape.com. Last updated: January 3rd, 2016. [cited 2016 Jul 5]
Available from: <http://emedicine.medscape.com/article/170907-overview>



39. HEPATITIS

1. Introduction

- Onset is gradual or sometimes rapid.
- There is fever, fatigue and nausea for a few days followed by jaundice accompanied by dark urine and sometimes clay - coloured stools.
- The severity of jaundice varies.
- On examination liver may be mildly enlarged with mild tenderness.
- This being the most common cause one must always make this clinical diagnosis after the exclusion of obstructive or haemolytic jaundice. In most cases this can be done on clinical grounds alone.
- There are five viruses that are commonly associated with hepatitis, called Hepatitis A, B, C, D and E virus. Of these two, Hepatitis A and E spread through faecal contamination of water. When there is an outbreak of jaundice in a village, it is probably this virus. Almost always this jaundice becomes alright on its own. Except in pregnant women where it can be life threatening. Three other types of viruses spread through the blood and through unprotected sexual contact (hepatitis B, C, D). These types are more severe, tend to worsen and have more long term complications.

2. Acute fulminant hepatitis

- Sometimes, especially with hepatitis B and D hepatitis turns severe due to cell necrosis.
- Patient develops confusion, stupor and then coma and it is difficult to save the life.
- Pregnant women are prone to develop this picture with all viruses but commonly with hepatitis E.

3. Chronic hepatitis

- Sometimes the liver disease goes on for years without becoming well.
- Such patients have frequent intermittent episodes of jaundice.
- Elevated liver enzymes over 6 months is enough to make this diagnosis.
- Eventually it can become well but more often leads to cirrhosis of the liver and portal

hypertension and end stage liver disease develops.

4. Obstructive jaundice

- Sometimes jaundice is due to obstruction to flow of bile and not due to a virus.
- On such cases the yellowing of eyes is very marked and may even be greenish.
- There is much itching and the stools are always whitish in colour.
- Usually the liver is enlarged.
- Ultrasound of the liver confirms obstruction best and the patients should be referred to a centre with ultrasonography facilities.
- Refer to higher centre as these cases need surgery.

5. Haemolytic jaundice

- Sometimes jaundice is due to increased breakdown of haemoglobin secondary to destruction of RBCs in a haemolytic anaemia.
- Jaundice is invariable light coloured, and urine is also normal in colour.
- Diagnosis needs to be established by blood smear examination, by ruling out hepatitis by liver function tests and by specific tests for haemolytic anaemia.

6. Alcoholic hepatitis

Excessive alcohol consumption is a significant cause of hepatitis and liver damage (cirrhosis). Alcoholic hepatitis usually develops over years-long exposure to alcohol. Alcohol intake in excess of 80 grams of alcohol a day in men and 40 grams a day in women is associated with development of alcoholic hepatitis. Alcoholic hepatitis can vary from mild asymptomatic disease to severe liver inflammation and liver failure. Symptoms and physical exam findings are similar to other causes of hepatitis. Laboratory findings are significant for elevated transaminases, usually with elevation of Aspartate Transaminase (AST) in a 2:1 ratio to Alanine Transaminase (ALT).

Alcoholic hepatitis may lead to cirrhosis and is more common in patients with long-term alcohol consumption and those infected with Hepatitis C.

Patients who drink alcohol to excess are also more often than others found to have Hepatitis C. The combination of Hepatitis C and alcohol consumption accelerates the development of cirrhosis.

7. Investigations for jaundice

These tests are needed to establish diagnosis & monitor improvement.

- Urine examination for bile salts & pigments.
- Serum bilirubin & Serum liver enzymes level.

8. Treatment for hepatitis

- Only supportive: Rest, hydration, correct but not specific diet.
- Avoid oily spicy foods that are ill tolerated.
- Avoid Corticotherapy. **NEVER** give steroids.
- There are no specific drugs to cure jaundice. Fortunately, most persons become well on their own.
- Remember many drugs commonly used are harmful when given to a person with jaundice.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. America. Center for Disease Control and Prevention (CDC). Sexually transmitted diseases treatment guidelines: Hepatitis A. [cited 2016 Jul 5]
Available from: <http://www.cdc.gov/std/tg2015/hepatitis.htm>
2. Kahn A and Higuera A. Hepatitis. Healthline.com. October 14, 2015. [cited 2016 Jul 5]
Available from: <http://www.healthline.com/health/hepatitis#Overview1>



8.1. Treatment for Acute fulminant hepatitis

- Patient needs hospitalization.
- Treatment is supportive & consists of maintaining parenteral fluids.
- Care is taken to treat infections or other precipitating factors like hematemesis.
- Gut sterilization with capsule amoxicillin and/or metronidazole may help.

8.2. Treatment for obstructive jaundice

- Refer for Surgery.

8.3. Treatment for Haemolytic anaemia

- Referral for further work up in higher centre with tests for type of haemolysis.

9. Vaccine

- Vaccine against hepatitis B is available.
- Hepatitis-B Vaccine is included in National Immunization Schedule.

40. HEPATIC COMA

1. Introduction

Hepatic Encephalopathy is a term used to describe a spectrum of neuropsychiatric abnormalities occurring in patients with significant liver disease and/or portosystemic shunting of blood.

2. Diagnosis

There are 2 major components of HE, altered mental status and generalized motor disturbance. Disturbances in awareness and mentation from forgetfulness and confusion to stupor and finally coma. Asterixis / flapping tremors is also commonly seen.

Search for precipitating factors – every advanced cirrhotic patient displaying a change in mental status is septic until proven otherwise. Common precipitating factors are GI bleeding, sepsis, hypokalaemia, high protein load, constipation, hyponatremia, Other causes include sedative drugs, superimposed liver injury.

3. Grade / Stage the severity of disease

Table-1: West-Haven Criteria for Hepatic Encephalopathy (HE)

Stage	Consciousness	Intellect and Behaviour	Neurologic Findings
0	Normal	Normal	Normal examination; if impaired psychomotor testing, consider MHE*
1	Mild lack of awareness	Shortened attention span	Impaired addition or subtraction; mild Asterixis or tremor
2	Lethargic	Disoriented; Inappropriate behaviour	Obvious Asterixis; Slurred speech
3	Somnolent but Arousable	Gross disorientation; Bizarre behaviour	Muscular rigidity and clonus; Hyperreflexia
4	Coma	Coma	Decerebrate posturing

*MHE, minimal hepatic encephalopathy.

4. Treatment

A. 4-pronged approach to treatment

Supportive care.

Search for and correct precipitating factors

Exclude and treat other causes of altered mental status.

Start empiric therapy for HE

- i. Lactulose: 30 cc b.i.d- q.i.d titrated to goal 2-4 soft BMs/day or retention enema 300 cc lactulose + 700 cc tap water retained for 1 hour reduces the ammonia production and absorption.

- ii. Rifaximin 400 mg PO t.i.d marginally better than lower doses (400 mg-550 mg b.i.d) or Neomycin 1 gm PO 6 hourly for up to 6 days (if used chronically, 1-2 gm/day).
- iii. Dietary protein restriction – No longer recommended.
- iv. L-ornithine-aspartate promotes waste nitrogen excretion.
- v. Antibiotics if sepsis suspected.

5. When to refer

Deteriorating sensorium, haematemesis, melena, bleeding tendencies.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further reading

1. Vilstrup H et al, Hepatic encephalopathy in chronic liver disease: 2014 Practice Guideline by the American Association for the Study of Liver Diseases and the European Association for the Study of the Liver, Hepatology. Volume 60, Issue 2. [Internet]. August 2014, Pages 715–735. [cited 2016 Jul 5] Available from: <http://onlinelibrary.wiley.com/doi/10.1002/hep.27210/abstract>
2. Hepatic Coma. Nmihi.com. [cited 2016 Jul 5] Available from: <http://www.nmihi.com/h/hepatic-coma.htm>
3. Mohamad R, Brendan MM. Current trends in the treatment of hepatic encephalopathy. Ther Clin Risk Manag. [Internet] 2009; 5: 617–626. Published online 2009 Aug 3. [cited 2016 Jul 5] Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2724191/>



41. NEPHROTIC SYNDROME

1. Definition

Nephrotic Syndrome is a clinical complex characterized by a number of renal and extra renal features, the most prominent of which are

- Proteinuria of 3-3.5 gm / 24 hour,
- Hypoalbuminemia,
- Oedema,
- Hyperlipidaemia, Lipiduria

2. Symptoms and Signs

Puffiness of face, increase in morning
Ankle pitting oedema with increasing severity to generalized anasarca Ascites, Pleural Effusion

3. Complications

Recurrent Infections, hypocalcaemia, anaemia, Thrombotic Tendencies

4. Investigation

- Urine Analysis for Proteinuria, Protein / Creatinine ratio
- Urine Microscopy for Deposit
- 24 Hour Urinary Protein
- Serum albumin
- Ultra-sonography abdomen and pelvis

- Renal Function Test: Blood Urea Level and Serum Creatinine Level
- Renal Biopsy: In Adult patients for a) Establishing a Definitive Diagnosis b) Guiding Therapy & c) Assessing Prognosis.

5. Treatment

- Steroid: Minimal change disease accounts for about 80% of Nephrotic Syndrome in children younger than 16 years and 20% of adult.
Adult: Tab Prednisolone 1-1.5 mg/kg body weight per day for 4 week followed by 1 mg/kg/day on alternate day for 4 weeks.
Check for urine protein after four weeks if nil no treatment needed and if present start with Prednisolone 1.5 mg for 2 weeks
- HMG-Co-A Reductase Inhibitor:
Tab Atorvastatin 10 mg Once a day
- Loop Diuretic
- Salt Restriction 1-2 Gram per Day.
- High Protein Diet
- Vitamin D Supplementation.

6. When to Refer

- i. For Renal Biopsy
- ii. When Dialysis is required.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. KDIGO. Clinical Practice Guideline for Glomerulonephritis. Kidney Int Suppl. [Internet] 2012; 2:209. [cited 2016 Jul 5]
Available from: http://www.kdigo.org/clinical_practice_guidelines/pdf/KDIGO-GN-Guideline.



42. ACUTE NEPHRITIC SYNDROME

It is Clinical Correlate of Acute Glomerular Inflammation Which May Be a Primary Disease OR Secondary to Systemic Process.

1. Definition

It is characterized by sudden onset (over days to weeks) of acute renal failure and Oliguria (400 ml/day of urine) renal blood flow and GFR fall as a result of obstruction of the glomerular capillary lumen.

2. Clinical Features

Tetrad of

- Oedema
- Hypertension mild
- Haematuria [macroscopic]
- Proteinuria (Sub-nephrotic Range < 3g/24 Hours)

Symptom:

- Puffiness of Face
- Oedema of Feet
- Cola Coloured Urine
- Dyspnoea If Pulmonary Oedema
- Hypertension
- Generalized Symptoms- Anorexia, Nausea, Vomiting, Malaise, Flank OR Back Pain.

3. Investigation

- Urine Microscopy: Dysmorphic RBCs and RBC Cast, Leucocytes
- Urine- Gross Haematuria (Red or Smoky Urine)
- Sub-nephrotic range Proteinuria

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. KDIGO Clinical Practice Guideline for Glomerulonephritis. Kidney Int Suppl. [Internet] 2012; 2:209. [cited 2016 Jul 5]

Available from: http://www.kdigo.org/clinical_practice_guidelines/pdf/KDIGO-GN-Guideline.



- Blood Urea and Serum Creatinine elevations.
- Decrease C3 Level, Normal C4 Level
- ASO Titre
- Ultra-sonography renal
- Renal Biopsy

4. Treatment

4.1 General Management

- Bed Rest
- Adequate Fluid Intake to ensure 400 ml Urine per Day
- Salt restriction especially if oedema present

4.2 Management of Renal Failure

- Tab Frusemide if volume overload hypertension
- Not to be given if no evidence of fluid excess
- Rarely require Dialysis

4.3 Antihypertensive Drug

4.4 Control of Infection

4.5 Renal Biopsy

If features not suggestive of Post Streptococcal Glomerulonephritis.

5. When to Refer

- i. For Renal Biopsy
- ii. When Dialysis is required.

43. ACUTE RENAL FAILURE / ACUTE KIDNEY INJURY

1. Definition

AKI is a syndrome characterized by rapid decline in glomerular filtration rate (hours to days), retention of nitrogenous waste products and perturbation of extracellular fluid volume and electrolyte and acid base homeostasis.

AKI is defined as any of the following (Not Graded):

- Increase in Sr. Creatinine by X 0.3 mg/dl (X26.5 $\mu\text{mol/l}$) within 48 hours; or
- Increase in Sr. Creatinine to X 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
- Urine volume 0.5 ml/kg/h for 6 hours

2. Classification

2.1. Prerenal:

a. Hypovolemia:

Haemorrhage, burns, dehydration, vomiting, diarrhoea, pancreatitis, peritonitis.

b. Low cardiac output:

CCF, Disease of myocardium, valves and pericardium,

Pulmonary Hypertension, Pulmonary emboli.

c. Altered renal systemic vascular resistance ratio;

Systemic vasodilatation- Sepsis, Antihypertensive drug

Renal vasoconstriction: Hypercalcemia

Cirrhosis with ascites- Hepatorenal syndrome.

2.2. Intrinsic renal

a. Renovascular obstruction:

Renal artery obstruction: atherosclerosis, thrombi, emboli, Renal vein obstruction: thrombi, compression

b. Disease of glomeruli:

Glomerulonephritis and vasculitis, Haemolytic uremic syndrome, DIC, Toxaemia of pregnancy, Accelerated Hypertension.

c. Acute tubular necrosis

Ischemia, Exogenous toxins: radiocontrast, chemotherapy, acetaminophen, Endogenous toxins: rhabdomyolysis, haemolysis, myeloma.

d. Interstitial nephritis

Antibiotic, NSAID, Sulphonamide, Beta lactam antibiotics

2.3. Post renal:

a. Ureter:

Calculi, blood clot, carcinoma

b. Bladder neck:

Prostatic hypertrophy, calculi, carcinoma

c. Urethra:

Stricture, phimosis.

3. Symptoms

3.1. Prerenal

- Evidence of true volume depletion- Thirst, postural or absolute hypotension and tachycardia, dry mucous membrane.
- Urine- Low volume, low sodium and high osmolality.

3.2. Renal

Symptom and sign of underlying disease affecting glomeruli or tubule.

3.3. Post renal

Abdominal or flank pain, palpable bladder.

3.4. Symptoms of uraemia

Anorexia, nausea, vomiting, Mental status changes, Pruritus, shortness of breath

4. Signs

Asterixis, Pericardial rub, Pedal oedema, Pulmonary oedema, Raised JVP

5. Investigation

- Urine analysis: Routine microscopy
- Routine blood chemistry: BUN, creatinine, electrolyte, Ca, PO₄
- Complete Haemogram
- Special test: Bence Jones protein
- Serology: ANA, ANCA, anti GBM, complement, ASO
- Imaging: Renal ultrasound, Plain radiograph of abdomen.
- Renal biopsy.

6. Staging of AKI

STAGE	Serum creatinine criteria	Urine output criteria
I	1.5 to 2-fold increase	<0.5 ml/kg/hr for > 6 hours
II	2 to 3-fold increase	<0.5 ml/kg/hr for >12 hours
III	>3-fold increase, absolute value > 4 mg/dl	< 0.3 ml/kg/hr for 24 hours or anuria for 24 hours.

7. Treatment

7.1. Prerenal

Restore blood volume (with isotonic saline 0.9%, or blood, plasma)
Treat underlying cause.

7.2 Renal

Eliminate toxins, Nephrology consultation

7.3. Post-renal

Relieve obstruction, Urology consultation

7.4. Hyperkalaemia

- calcium gluconate (10ml 10% solution)
- Inhaled β_2 agonist- Salbutamol
- IV glucose insulin infusion (100ml 25% dextrose with 10 unit of human Actrapid in 1 hour)
- Dialysis.

7.5. Treatment of metabolic acidosis

Inj. Sodium bicarbonate IV as per Base deficit

7.6. Dialysis: indication

Metabolic acidosis, Hyperkalaemia, Encephalopathy, Volume overload, Pericarditis

8. Complication

COMPLICATION	MANIFESTATION
1. Metabolic	Hyperkalaemia, hypocalcaemia, hyperphosphatemia, hypermagnesemia, hyperuricemia, metabolic acidosis.
2. Cardiovascular	Cardiac arrhythmia, pulmonary oedema, pericardial effusion, pericarditis
3. Gastrointestinal	Gastrointestinal haemorrhage
4. Neurological	Altered sensorium, seizures
5. Hematological	Anaemia, bleeding.

9. Prevention

- Management and prevention of sepsis
- Appropriate and adequate fluid management
- Optimization of hemodynamic

- Nephrotoxic drug should be stopped if patient is at risk of developing AKI.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl. [Internet] 2012; vol 2. [cited 2016 Jul 5]

Available from:

http://www.kdigo.org/clinical_practice_guidelines/pdf/KDIGO%20AKI%20Guideline.pdf



44. CHRONIC KIDNEY DISEASE

1. Definition

A. Presence of marker of kidney damage more than 3 months as defined by structural or functional abnormality of the kidney with or without decrease GFR manifested by either pathological abnormality or other marker of kidney damage.

B. GFR < 60 ml/min/1.73 m² for more than 3 months with or without sign of kidney damage.

2. Causes

- i. Diabetes
- ii. Hypertension

- iii. Nondiabetic glomerular disease- Nephritic and nephrotic presentation
- iv. Cystic kidney disease
- v. Tubulo-interstitial disease.

3. Staging

According to GFR (glomerular filtration rate) Cockcroft-Gault equation for calculation of creatinine clearance:

$$\frac{(140 - \text{Age}) \times \text{Body weight (kg)}}{72 \times \text{plasma creatinine (mg/dl)}}$$

Multiply by 0.85 for women.

Table-1: Staging of CKD

STAGE	DESCRIPTION	GFR (ml/min/1.73 m ²)
1	At increased risk of kidney damage with normal or raised GFR	90
2	Kidney damage with mildly decreased GFR	60-89
3	Moderately decreased GFR	30-59
4	Severely decreased GFR	15-29
5	Renal failure	<15

4. Symptoms and signs

Hypertension, proteinuria, anaemia, deep respiration (Kussmaul's respiration), tiredness, breathlessness, pruritus, anorexia, nausea, vomiting.

5. Risk factors

- Age > 65 years
- Diabetes
- Family H/O renal disease
- Autoimmune disease
- Systemic infection
- UTI, stone, urinary tract obstruction,
- HTN

6. Evaluation and management of patient of CKD

6.1. History and physical examination

H/O HTN, DM, use of analgesic Measure blood pressure

6.2. Laboratory investigation

- Haemoglobin level.
- Urine for proteinuria and haematuria
- Blood urea, serum creatinine

- Blood sugar level
- Serum electrolyte
- Serum calcium, phosphorus level

6.3. Imaging study

USG abdomen for the renal size and Cortico-medullary differentiation.

Table-2: To Delay Progression to Next Stage:

MEASURES	GOAL
1. ACE inhibitors / ARB	Proteinuria <0.5 g/day and GFR decline <2ml/min/yr.
2. Additional antihypertensive drug as needed	BP<130/80 if proteinuria <1 gm/day BP<125/75 if proteinuria >1gm/day
3. Dietary protein restriction	0.6 to 0.8 gm/kg/day
4. Glycaemic control	HbA1C <7 %
5. Anaemia correction	Target Hb 10 to 12 gm/dl
6. Cholesterol lowering agent	LDL <100 mg/dl
7. Dietary salt restriction	3 to 5 gm/day

6.4. Treatment

a. Slowing the progression of CRF:

Protein restriction 0.6 gm/kg/day

b. Slowing diabetic renal disease:

Glucose control – target HbA1c <7%

c. Managing complications of CRF:

Cardiovascular complication- Yearly screening of cardiovascular diseases, Anaemia: target Hb 11 gm/dl, use of iron and erythropoietin injection, Renal

osteodystrophy: Target Ca 8.8 – 9.7, phosphorus 3.5 – 5.5, Use of calcium carbonate, phosphorus restriction – avoid dairy product, meat, colas, oral calcitriol.

d. Renal replacement therapy:

Dialysis: refer to higher center, A-V fistula; if GFR <25ml/min Serum creatinine > 4 mEq/L.

Bibliography

1. Casper DL, Fauchi AS, Harrison’s Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson’s Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. Varma PP. Prevalence of Kidney disease in India – where are we heading? Indian J Nephrol.[Internet]. 2015 May-Jun; 25(3): 133–135. [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4446915/>



45. MALARIA

1. Introduction

Malaria is a protozoal disease caused by infection with parasites of the genus *plasmodium* and transmitted to man by female Anopheles mosquito. It is a very important public health problem in India particularly due to Plasmodium falciparum which is prone to various complications.

2. Agent

Malaria in man is caused by *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium Ovale* and *Plasmodium malariae*. Out of these, *Plasmodium vivax* and *Plasmodium falciparum* are very common in India including Maharashtra.

3. Mode of transmission

Direct – through blood or plasma.

Vectors – bite of female Anopheles mosquito.

4. Incubation Period

The duration varies with species of parasite.

- 12 (9-14) days for falciparum malaria.
- 14 (8-17) days for vivax malaria.

5. When to suspect

The typical attack comprises three distinct stages viz. cold stage, hot stage and sweating stage.

Cold Stage:

The onset is with lassitude, headache, nausea and chilly sensation followed in an hour or so by rigors. The temperature rises rapidly to 39-41°C. Headache is often severe and commonly there is vomiting. In early part of this stage, skin feels cold; later it becomes hot. Parasites are usually demonstrable in the blood. The pulse is rapid and may be weak. This stage lasts for ¼-1 hour.

Hot Stage:

The patient feels burning hot and casts off his clothes. The skin is hot and dry to touch. Headache is intense but nausea commonly diminishes. The pulse is full and respiration rapid. This stage lasts for 2 to 6 hours.

Sweating Stage:

Fever comes down with profuse sweating. The temperature drops rapidly to normal and skin is cool and moist. The pulse rate becomes slower; patient feels relieved and often falls asleep. This stage lasts for 2-4 hours.

The febrile paroxysms occur with definite intermittent periodicity repeating every third or fourth day depending upon the species of the parasite involved. The classical 3 stages (cold, hot and sweating) may not always be observed due to maturation of generations of parasite at different times. The disease has a tendency to relapse and is characterized by enlargement of the spleen and secondary anaemia.

In patients with *P. falciparum* infection, the primary fever in its first few days is usually irregular or even continuous and then the classical 48-hour periodicity becomes established or the fever may continue to be irregular and the hot and cold stages, so typical of other malarial infections are less clearly separated from one another, in persons with poor immunity. The paroxysms are associated with marked prostration. Headache, nausea and vomiting are usually more severe, and there is greater tendency towards the development of delirium, haemolytic jaundice and anaemia. The mortality is much greater than in other forms of malaria.

With *P. vivax* infection, symptoms are same but are usually milder and more regularly divided into “hot” and “cold” stages than in *P. falciparum* infections.

6. Complications

The complications of *P. falciparum* malaria are cerebral malaria, acute renal failure, liver damage, gastro-intestinal symptoms, dehydration, collapse, anaemia, black water fever etc. The complications of *P. vivax* infection are anaemia, splenomegaly, enlargement of liver, herpes, renal complications, ARDS etc.

7. Investigations

- Diagnosis of Malaria: One of the above clinical features, supported by blood smear examination for malarial parasites.
- Fever with splenomegaly in a patient with the above mentioned clinical features make diagnosis of malaria more likely.

- Confirmation of diagnosis always depends on seeing the parasite in the blood. In all cases, thick and thin smears should be examined.
- Blood smears may be negative in severe and chronic forms and this would need repeated smears.

8. Diagnosis of Malaria

It is stressed that all fever cases should be suspected of malaria after ruling out other common causes and should be investigated for confirmation of malaria by Microscopy or Rapid Diagnostic Kit (RDK) so as to

ensure treatment with full therapeutic dose with appropriate drug to all confirmed cases. Presumptive treatment of malaria with a single dose of chloroquine has been stopped.

All fever cases diagnosed as malaria by either RDT or microscopy should be promptly given effective treatment. The medicine chosen will depend upon whether the patient has vivax malaria or falciparum malaria as diagnosed by the blood test. The flow charts in different settings for diagnosis and drug selection for the treatment of malaria are mentioned below.

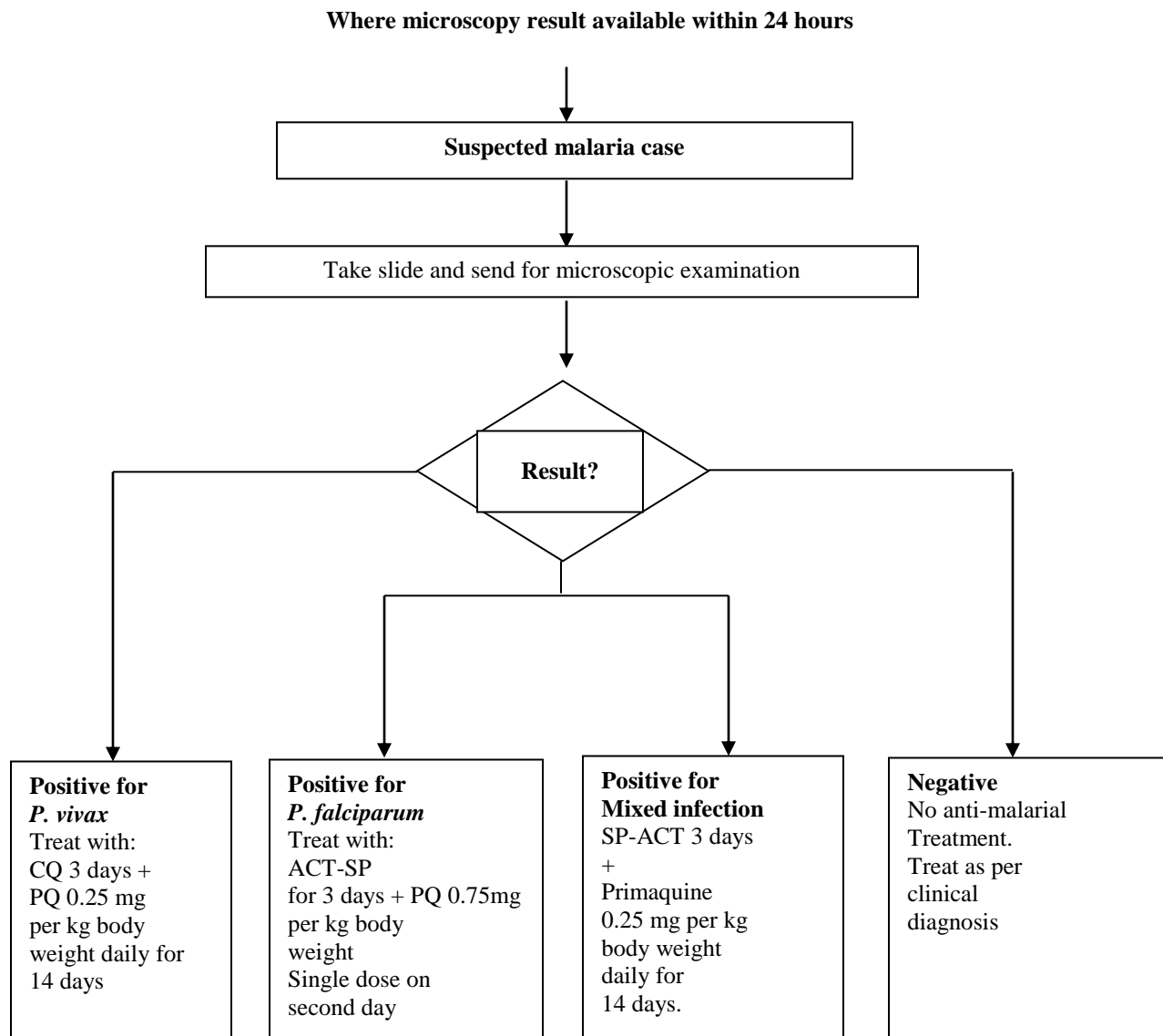


Figure-45.1: Management chart for suspected Malaria case

ACT-SP- Artemisinin-based Combination Therapy
(Artesunate + Sulfadoxine-Pyrimethamine)
CQ – Chloroquine PQ –Primaquine

Where microscopy result is not available within 24
hours and Monovalent RDT is used
TfR= Test falciparum rate

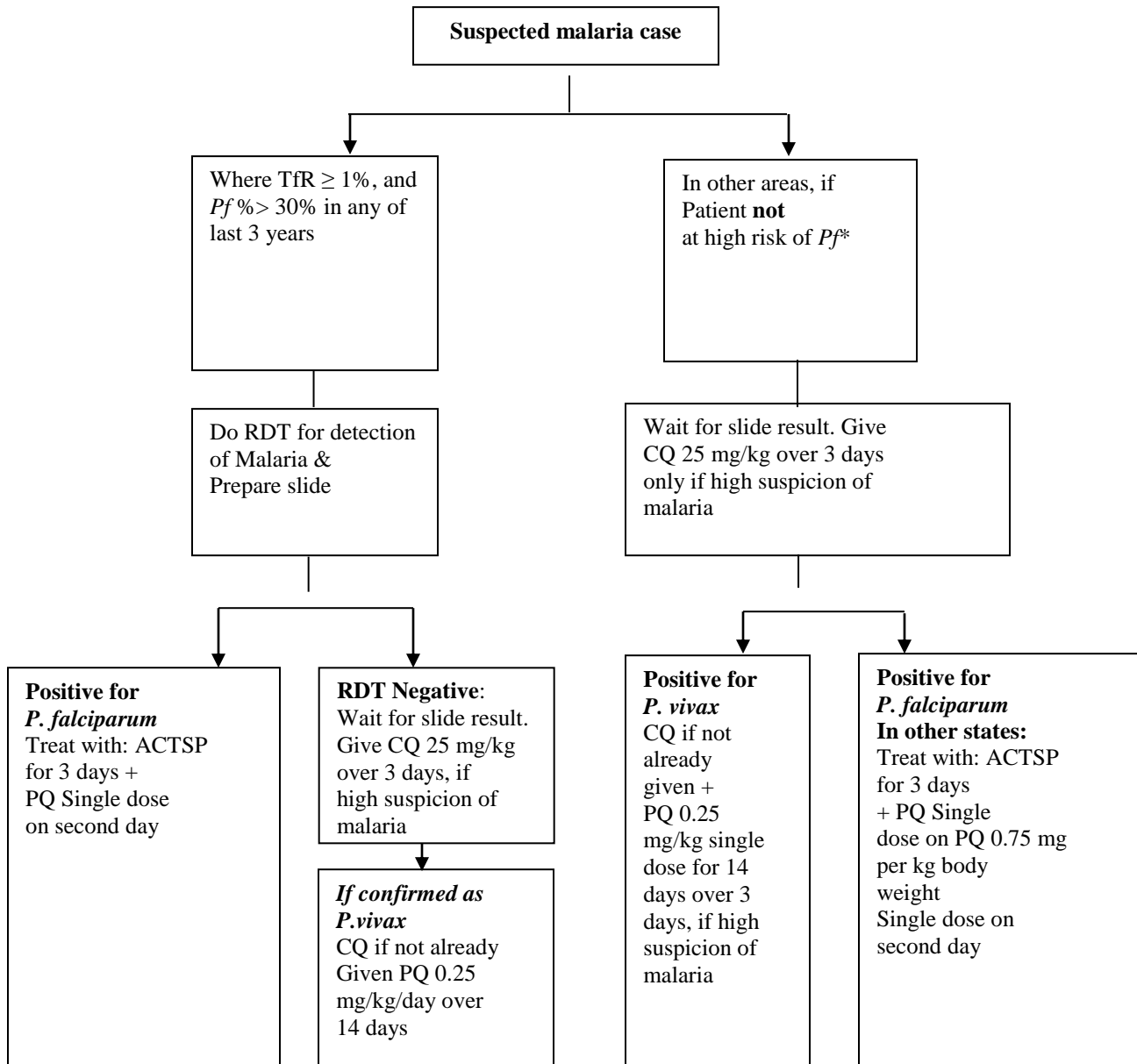


Figure-45.2: Management of suspected Malaria

Note: if a patient has severe symptoms at any stage, then immediately refer to a facility with indoor patient management.

Note: PQ is contra-indicated in pregnancy and in children under 1 year (Infant).

ACT-SP- Artemisinin-based Combination Therapy (Artesunate + Sulfadoxine-Pyrimethamine)

CQ –Chloroquine **PQ** – Primaquine.

Where microscopy result is not available within 24 hours and Bivalent RDT is used

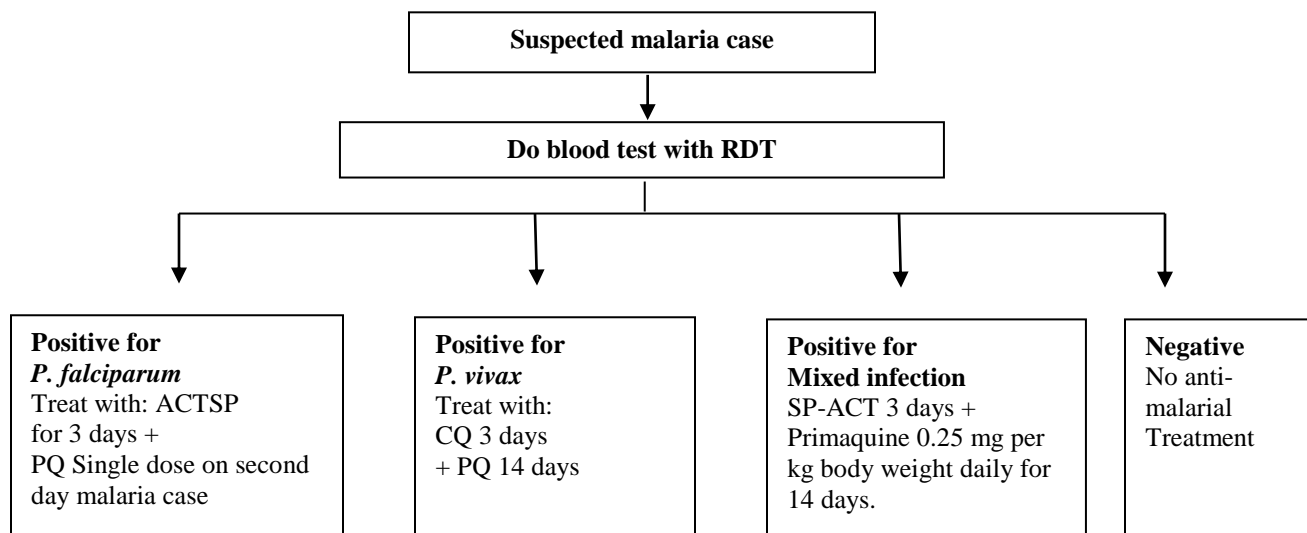


Figure-45.3: Suspected Malaria management based on RDT results

ACT-SP- Artemisinin-based Combination Therapy (Artesunate + Sulfadoxine-Pyrimethamine).

CQ –Chloroquine **PQ** - Primaquine

Differential Diagnosis:

Other causes of fever like – Dengue, UTI, URTI. All D/D can be excluded by using lab investigation (microscopy/RDT).

9. Treatment

9.1. Treatment of Vivax Malaria

Diagnosis of vivax malaria may be made by the use of RDT (Bivalent) or microscopic examination of the blood smear. On confirmation following treatment is to be given:

Drug schedule for treatment of P vivax malaria:

i. Chloroquine:

25 mg/kg body weight divided over three days i.e.

10 mg/kg on day 1,

10 mg/kg on day 2 and

5 mg/kg on day 3.

ii. Primaquine:

0.25 mg/kg body weight daily for 14 days.

Primaquine is contraindicated in infants, pregnant women and individuals with G6PD deficiency.

14-day regimen of Primaquine should be given under supervision.

Table-1: Dosage Chart for Treatment of Vivax Malaria

Age	Day 1		Day 2		Day 3		Day 4 to 14
	CQ (150 mg base)	PQ (2.5 mg)	CQ (150 mg base)	PQ (2.5 mg)	CQ (150 mg base)	PQ (2.5 mg)	PQ (0.25 mg)
Less than 1 year	½	0	½	0	¼	0	0
1-4 years	1	1	1	1	½	1	1
5-8 years	2	2	2	2	1	2	2
9-14 years	3	4	3	4	1 ½	4	4
15 years or more*	4	6	4	6	2	6	6
Pregnancy	4	0	4	0	2	0	0

Note: Chloroquine 250 mg tablet is having 150 mg base

9.2. Treatment of Falciparum Malaria

Diagnosis of falciparum malaria may be made by the use of RDT (Monovalent or Bivalent) or microscopic examination of the blood smear. It is imperative to start the treatment for falciparum malaria immediately on diagnosis. The treatment for falciparum malaria is as follows:

Dose schedule for Treatment of uncomplicated P. falciparum cases:

i. Artemisinin based Combination Therapy (ACT-SP) *

Artesunate 4 mg/kg body weight daily for 3 days Plus Sulfadoxine (25 mg/kg body weight) – Pyrimethamine (1.25 mg/kg body weight) on first day.

* ACT is not to be given in 1st trimester of pregnancy.

ii. Primaquine: 0.75 mg/kg body weight on day 2.

Treatment of uncomplicated P. falciparum cases in pregnancy:

- 1st Trimester: Quinine salt 10 mg/kg 3 times daily for 7 days. Quinine may induce hypoglycaemia; pregnant women should not take quinine on empty stomach and should eat regularly, while on quinine treatment.
- 2nd and 3rd trimester: ACT as per dosage schedule given above.

9.3. Treatment of mixed infections (P. vivax + P. falciparum) cases

All mixed infections should be treated with full course of ACT and Primaquine 0.25 mg per kg body weight daily for 14 days.

SP-ACT 3 days + Primaquine 0.25 mg per kg body wt. daily for 14 days.

Table-2: Treatment of mixed infections

Age	Day 1			Day 2		Day 3		Day 4 to 14
	As tablet (50 mg)	SP tablet	PQ (2.5 mg)	As tablet (50 mg)	PQ (2.5 mg)	As tablet (50 mg)	PQ (2.5 mg)	PQ (0.25 mg)
Less than 1 year	½	½	0	½	0	½	0	0
1-4 years	1	1	1	1	1	1	1	1
5-8 years	2	1 ½	2	2	2	2	2	2
9-14 years	3	2	4	3	4	3	4	4
15 years or more	4	3	6	4	6	4	6	6

All cases of mixed infection are to be treated as Pf plus Primaquine for 14 days.

When a patient fails to respond to treatment (symptoms fail to disappear, or they reappear), one should think of the possibility of drug resistance in such case refer patient to FRU. In the absence of any of these conditions, if a patient has completed full treatment and is still having symptoms after 72 hours, treatment failure may be suspected.

The course of action when a patient has persistent symptoms is:

- Ask the patient and the family a series of questions to help rule out some of the causes

(Did the patient get the drug from an authentic, designated provider? Did the patient get the right amount of the drug? Was all of it swallowed as prescribed? Was the drug vomited out? How many days has it been since drug treatment was begun (if it is not yet 72 hours, one can wait)? Can you see the packing to check the expiry date? Are there symptoms of other obvious causes of fever? If the symptoms had disappeared and then reappeared, how long was the interval (if more than 15 days, it could be a fresh infection)?)

- If it appears that the drug was not adequately taken or retained, a fresh course may be given unless the patient has symptoms of severe

malaria. Take a fresh blood smear (take two, for checking in different laboratories, if need be), and ask the nearest health care provider to keep an eye on the patient.

- Refer any patient who has symptoms despite taking and retaining a full course of treatment, or who has developed symptoms of severe malaria.

10. Severe and complicated malaria

10.1. Clinical Features

Severe manifestations can develop in *P. falciparum* infection over a span of time as short as 12-24 hours and may lead to death, if not treated promptly and adequately. Severe malaria is clinically characterized by confusion or drowsiness with extreme weakness (prostration) along with one or more of the following features:

- Impaired consciousness/coma
- Repeated generalized convulsions
- Renal failure (Serum Creatinine >3 mg/dl)
- Jaundice (Serum Bilirubin >3 mg/dl)
- Severe anaemia (Hb <5 mg/dl)
- Pulmonary oedema/acute respiratory distress syndrome
- Hypoglycaemia (Plasma glucose <40 mg/dl)
- Metabolic acidosis
- Circulatory collapse/shock (Systolic BP <80 mm Hg, <50 mm Hg in children).
- Abnormal bleeding and disseminated intravascular coagulation.
- Haemoglobinuria.
- Hyperthermia (Temperature >106°F or 42°C).
- Hyperparasitemia (<5% parasitized RBCs in low endemic and >10% in hyper endemic areas).
- Circulatory collapse/shock.
- Spontaneous bleeding and laboratory evidence of DIC.
- Macroscopic haemoglobinuria.

Foetal and maternal complications are more common in pregnancy with severe malaria; therefore, they need prompt attention.

10.2. In children

Febrile convulsions, repeated vomiting and dehydration are common if the temperature is high due to any cause. Therefore, these symptoms are not necessarily indicative of severe malaria. However, children with such symptoms should be managed as severe malaria in routine program situations, and a diagnosis of malaria should be confirmed at the earliest.

10.3. In pregnancy

Malaria, especially *P. falciparum* is a serious disease because with each bout of malaria, there is a reduction in haemoglobin and profound anaemia may develop rapidly. They are also at high risk of abortions or intrauterine growth retardation because sequestration of parasites in placenta restricts oxygen and nutrients flow to the foetus.

The management of severe malaria is possible in health facilities which are equipped with the following:

- Parenteral anti-malarials, antibiotics, anticonvulsants, antipyretics.
- Intravenous infusion equipment and fluids.
- Special nursing for patients in coma.
- Facilities for blood transfusion.
- Well-equipped laboratory.
- Oxygen respirator.

Often these items are not available at the PHC level. Under such circumstances, the Medical Officer, PHC and paramedical staff should be able to administer emergency treatment and refer the case without delay to other institutions where such facilities are available.

10.4. Treatment of severe malaria cases

Severe malaria is an emergency and treatment should be given as per severity and associated complications which can be best decided by the treating physicians. The guidelines for specific antimalarial therapy is as follows:

Parenteral artemisinin derivatives or Quinine should be used irrespective of Chloroquine resistance status of the area with one of the following options:

Table 3: Chemotherapy of severe and complicated malaria

Initial parenteral treatment for at least 48 hours: CHOOSE ONE of following four options	Follow-up treatment, when patient can take oral medication following parenteral treatment
Artesunate: 2.4 mg/kg IV or IM given on admission (time=0), then at 12 hours and 24 hours, then once a day.	Full oral course of Area-specific ACT: In other states: Treat with: ACT-SP for 3 days + PQ Single dose on second day
Quinine: 20mg quinine salt/kg body weight on admission (IV infusion or divided IM injection) followed by maintenance dose of 10 mg/kg 8 hourly; Infusion rate should not exceed 5 mg/kg per hour in normal saline. Loading dose of 20mg/kg should not be given, if the patient has already received quinine.	Quinine 10 mg/kg three times a day with: Doxycycline 100 mg once a day or Clindamycin in pregnant women and children under 8 years of age, - to Complete 7 days of treatment.

Note: The parenteral treatment in severe malaria cases should be given for minimum of 24 hours once started (irrespective of the patient’s ability to tolerate oral medication earlier than 24 hours).

Note:

- The parenteral treatment should be given for minimum of 48 hours.
- Once the patient can take oral therapy, give: Quinine 10 mg/kg three times a day with Doxycycline 100 mg once a day or Clindamycin in pregnant women and children under 8 years of age, to complete 7 days of treatment, in patients started on parenteral quinine.
- Full course of ACT to patients started on artemisinin derivatives.
- Use of Mefloquine should be avoided in cerebral malaria due to neuro psychiatric complications associated with it.

Supplementary treatment –

In unconscious patient, monitor blood glucose level every 4 to 6 hours and treat hypoglycaemia with IV dextrose. If blood sugar estimation is not available, one can presume hypoglycaemia in all cases of severe and complicated malaria especially cerebral malaria and treat with intravenous glucose – 100 ml of 25% glucose before giving quinine.

- All unconscious patients on quinine should receive a continuous infusion of 5 to 10% dextrose.
- Parasite count and haematocrit level should be measured every 6 to 12 hourly.
- Transfuse whole blood or packed cell concentrate when haematocrit drops to <20%.

- Renal function should be checked daily, institute early haemodialysis if necessary.
- Fluid management should be carefully done.
- Treat convulsions with IV Diazepam and shift the patient to an Intensive Care Unit with facilities for ventilator support.

10.5. Some don’ts in severe malaria case management

Do not use corticosteroids, give intravenous Mannitol, use Heparin as anticoagulant, administer Adrenaline or over-hydrate.

In recent years, increased attention has been drawn to severe malaria caused by *P. vivax*, especially in Indonesia and Papua New Guinea, where this parasite has become chloroquine resistant. Some cases have been found in India, and there is reason to fear that this problem will become more common in the coming years. Historically, *P. vivax* has been an important cause of death in India and in Europe, and this parasite can no longer be considered as “benign”.

10.6. When to refer: Any of the following

- Platelet count ≤ 20,000/cu.mm.
- Pregnancy with malaria.
- Any feature of complicated malaria.
- Renal failure.
- ARDS.
- Malaria with sepsis.
- Malaria with shock.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. Directorate of National Vector Borne Disease Control Programme. Diagnosis and treatment of Malaria 2013. Ministry of Health and Family Welfare. [cited 2016 Jul 5]

Available from: <http://nvbdcp.gov.in/Doc/Diagnosis-Treatment-Malaria-2013.pdf>



46. DENGUE

1. Introduction

In Maharashtra State, cases of Dengue Fever / Dengue Haemorrhagic Fever occur either in post-monsoon period when breeding of mosquitoes is highest due to accumulation of rainwater in discarded materials or during scarcity season due to Aedes breeding in stored water in cement tanks and earthen pots which are not emptied regularly.

2. Etiologic agent

DF/DHF is caused by a group B arbovirus (Flavivirus) and includes serotypes 1, 2, 3 and 4 (Den-1, Den-2, Den-3 and Den-4).

Infection with any one serotype confers lifelong immunity to the virus serotype, but no cross protection for other serotypes. It is caused due to bite of an infected female Aedes Aegypti mosquito.

Mode of Transmission It is transmitted through bite of Aedes Aegypti mosquito.

Incubation period: Varies from 5-10 days.

3. Diagnosis

(Table 1)

Table-1: Recommended diagnostic tool according to laboratory service level

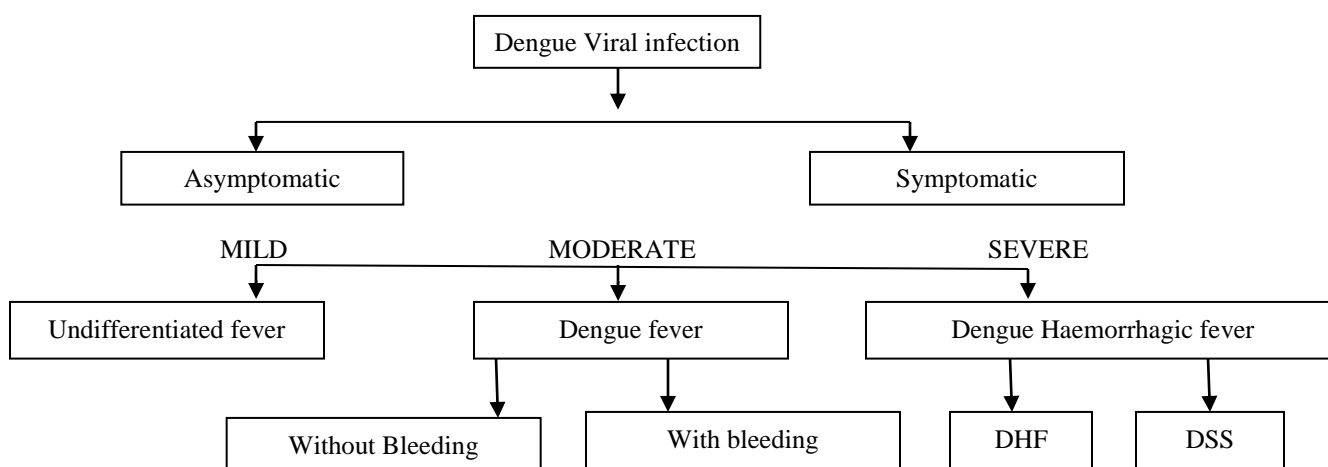
		Primary health care centres	District health centres	Reference centres
Genome Detection				Yes
NS1 Ag detection	Rapid tests	Yes	Yes	Yes
	ELISA		Yes	Yes
IgM detection	Rapid tests	Yes	Yes	Yes
	ELISA		Yes	Yes
IgG detection	ELISA			Yes
	IHA			Yes
	Neutralization assay			Yes

ELISA = Enzyme-linked immunosorbent assay; IgG = Immunoglobulin G; IgM = Immunoglobulin M; IHA = Indirect Haemagglutination; NS1 Ag = Non- Structural protein 1 antigen.

4. Clinical spectrum

Following is the spectrum of dengue viral infection.

Figure-46.1: Dengue Clinical Spectrum



5. Clinical management

Depending upon severity of infection, management of the cases differs. Early diagnosis & admission of DHF patients is important in order to reduce case fatality rates.

5.1. Dengue fever

Management of Dengue fever is symptomatic and supportive.

- Bed rest is advisable during acute febrile phase.
- Antipyretics and sponging is essential to keep body temperature of patient below 37°C. Do not prescribe Salicylates (Aspirin) to suspected DF patient. Paracetamol is preferred.
- Analgesic or a mild sedative may be prescribed for severe pain.
- ORS solution is recommended for patients with excessive sweating, nausea, vomiting or diarrhoea to prevent dehydration.
- Patients should be monitored in DHF area until they become afebrile & after platelet & hematocrit determinations are normal.

5.2 Dengue Haemorrhagic Fever

Management of Grade I DF/DHF

- Management during febrile phase is similar to that of DF.
- Patient should be monitored closely. Critical period for monitoring is transition from febrile to afebrile stage, which usually occurs after third day of illness.
- Platelet count & hematocrit estimation is essential.
- Drop in platelet count to <1,00,000/cu.mm i.e. 1-2 platelets per oil immersion field usually

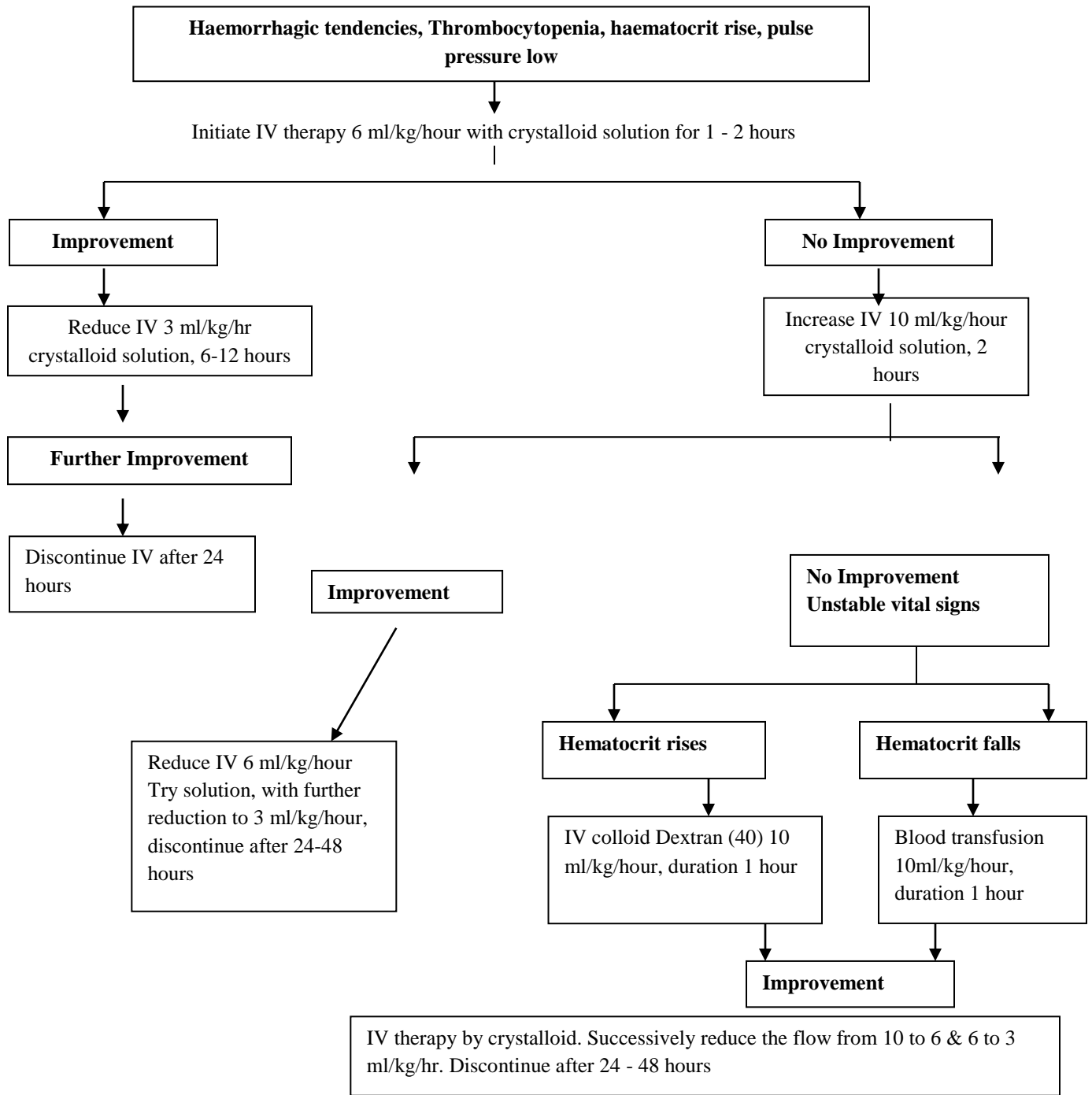
precedes rise in hematocrit. A rise of more than 20% indicates need for intravenous fluid therapy.

- If hematocrit determination is not possible, hemoglobin estimation may be carried out as an alternative.
- Hematocrit should be determined daily from the third day until the temperature remains normal for one or two days.
- Paracetamol is recommended to keep temperature below 40°C. Dosages of paracetamol recommended are: 1 - 2 years: 60-120 mg/dose, 3 - 6 years: 120 mg/dose & 7 - 12 years: 240 mg/dose.
- Plenty of fluids like ORS & or fruit juices should be given orally, to the extent patient tolerates.

Management of Grade II DF/DHF

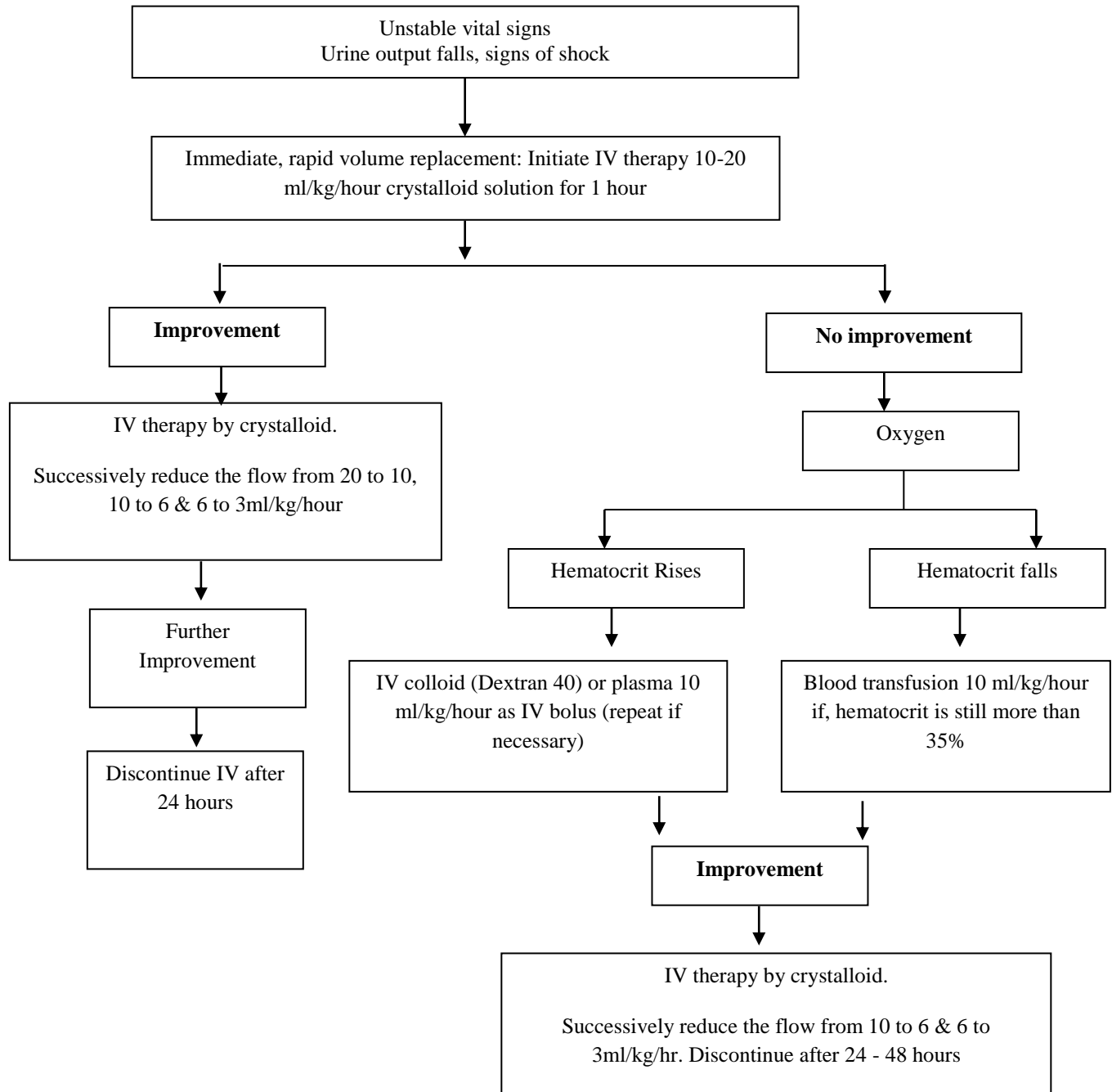
- Any person who has DF with thrombocytopenia & haemoconcentration & presents with abdominal pain, black tarry stools, epistaxis, bleeding from gums etc. needs to be hospitalized. Such patient should be observed for signs of shock.
- The critical period for development of shock is transition from febrile to afebrile. phase of illness, which usually occurs after third day of illness.
- A rise of hematocrit of 20% or more reflects need for IV fluid therapy.
- If despite of treatment, patient develops **features of shock**, management of **grade III & IV** should be started.
- Blood transfusion may be indicated in patients with severe shock, massive bleeding and DIC.

Figure-46.2: Management of grade I & II DHF - Volume replacement flow chart



- **Improvement:** Haematocrit falls, pulse rate & BP stable, urine output rises
- **No improvement:** Haematocrit, pulse rate rises, pulse pressure falls below 20 mm Hg, urine output falls
- **Unstable vital signs:** Urine output falls, signs of shock

Figure-46.3: Management of grade III & IV DHF - Volume replacement flow chart



Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. Standard Treatment Guidelines for Dengue. Ministry of Health and Family Welfare. [cited 2016 Jul 5]
Available from: <http://nrhm.gov.in/images/pdf/guidelines/nrhm-guidelines/stg/dengue.pdf>
2. India. Directorate of National Vector Borne Diseases and Control Programme. Guidelines for Clinical Management of Dengue 2008. Ministry of Health and Family Welfare. [cited 2016 Jul 5]
Available from: <http://www.nvbdc.gov.in/Doc/Clinical%20Guidelines.pdf>



47. LEPTOSPIROSIS

1. Introduction

Leptospirosis is an infectious disease caused by spirochetes *Leptospira interrogans*

Leptospira enter the host through abrasions in the skin or through intact mucosa, especially the conjunctiva and the lining of oro- and nasopharynx, when host come in contact with water contaminated with *Leptospira*.

2. High risk group

- Agricultural workers.
- Fisherman, sewer workers.
- Lorry drivers and masons.

Usually starts at the onset of rainy season and declines as the rains recede.

3. Anicteric leptospirosis

Accounts for 90% cases, usually recover completely with proper treatment.

- Fever with chills. Moderate to severe.
- Myalgia – Very characteristic finding. Calf, abdominal & lumbosacral muscles are very painful & severely tender. Increase in serum Creatinine Phosphokinase.
- Conjunctival Suffusion – Reddish colouration. Headache – commonly in frontal region.
- Renal involvement is invariable. Asymptomatic in the form of mild proteinuria with few casts in urine.
- Cough chest pain & in few cases haemoptysis.
- Bleeding tendency in few cases.
- All cases of fever with myalgia & conjunctival suffusion should be considered as a suspect case of leptospirosis.

4. Icteric leptospirosis

Weil's disease.

- Fever, Myalgia, Headache, Conjunctival suffusion.
- Oliguria, anuria, proteinuria.
- Nausea, vomiting, diarrhoea, abdominal pain.

- Hypotension & circulatory collapse.
- Jaundice – Important clinical feature. Mild to severe. Starts after 4 to 7 days of illness. Hepatomegaly liver tenderness usually present.
- Almost invariably present is renal involvement.
- ATN and interstitial nephritis are pathologic features.
- Haematuria and cola coloured urine with RBC casts.
- Oliguria and anuria.
- Oedema, facial puffiness, breathlessness, convulsions.
- Renal impairment worsens in 1st to 2nd week, recovers by the end of 4th week with treatment.
- Lung - Haemorrhagic pneumonitis with interstitial and alveolar haemorrhages.
- High mortality.
- Death occurs within few hrs to 2 days.
- Mild cases- Cough, chest pain and haemoptysis.
- Severe cases - Breathlessness increases and patient goes into respiratory failure.
- Pancreatitis and acalculous cholecystitis can occur.
- Cardiac involvement
- Hypotension shock- Cold clammy skin, tachycardia and hypotension due to dehydration and peripheral vasodilation.
- Arrhythmias- palpitations, syncope and irregular pulse, AV blocks and ST and T wave changes.
- CNS Meningitis usually present. Headache, irritability, restlessness, seizures and late stage is coma.
- Maculopapular erythematous skin lesions over face, trunk and extremities.
- Bleeding in leptospirosis is not directly related to the level of thrombocytopenia.

5. Investigations

- CBC Thrombocytopenia is characteristic.
- LFT – Direct hyperbilirubinemia., raised Alk.PO4, rise in bilirubin is very fast & reaches high level.
- Rise in enzyme level is not very high as compared to that in viral or alcoholic hepatitis.
- Raised CPK helps to differentiate from viral and alcoholic hepatitis.
- Raised serum creatinine levels.
- Urine albuminuria.

Table 1: Lab Investigations for Leptospirosis

CULTURE	MICROSCOPY	IMMUNOLOGICAL	MOLECULAR
Blood (10 days) Urine (10-30 days) CSF (5-10 days)	Dark Field Silver impregnation	MAT ELISA Latex agglutination tests	PCR

Laboratory diagnosis for current leptospirosis

- Culture –positive
- MAT- seroconversion/four-fold rise in the titre
- High titre
- ELISA/Latex agglutination positive.
- AVOID NEPHROTOXIC DRUGS in cases with renal involvement
- Severe cases- meticulous correction of hypovolemia and electrolyte imbalance, fluid management as per CVP.
- Patient with renal failure may require haemodialysis.

6. Treatment

6.1 Mild Case

- Tab. Doxycycline 100 mg BD for 7 days
- Cap. Ampicillin and Cap. Amoxicillin 30-50 mg/kg 5-7 days.
- Inj. Crystal Penicillin 20 lac units 6 hourly AST for 5-7 days or
- Inj. Ampicillin 500 mg-1 gm 6 hourly 5-7 days

6.2 Severe case

- Inj. Ceftriaxone / Cefotaxime or Erythromycin

7. Complications

Fluid overload, hyperkalaemia, acidosis

LUNG INVOLVEMENT - Continue O₂ therapy

- In patients with alveolar haemorrhages with ARDS, they require mechanical ventilation with low tidal volume and high PEEP.
- Mortality is very high.
- Supportive treatment with Platelet concentrate, FFP, Vitamin K.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. National Center for Disease Control (NCDC). Program for prevention and management of leptospirosis, National Guidelines, 2015. Ministry of Health and Family Welfare. [cited 2016 Jul 5] Available from: <http://www.ncdc.gov.in/writereaddata/mainlinkfile/File558.pdf>



48. INFLUENZA A- H1N1 (SWINE FLU)

We have faced pandemic of this Communicable air borne disease in 2009. This is commonly known as swine flu.

1. Epidemiological factors

Pandemic Influenza A (H1N1) 2009, currently the most common circulating strain of influenza virus globally, first caused illness in Mexico and the United States in March and April, 2009.

2. Agent factors

The causative agent is Influenza virus. It is an enveloped RNA virus and belongs to the family Orthomyxoviridae. The size of the virus is 80-200 nm /0.08 -0.12 micron in diameter. There are three types of influenza viruses, namely A, B & C which are characteristically distinct and bear no cross immunity. The virus contains two surface antigens H (hemagglutinin) and N (neuraminidase).

3. Host factors

The disease can occur in all ages and both sexes.

4. High Risk Groups

These risk groups include:

- Children younger than 5 years old;
- Adults 65 years of age and older;
- Chronic pulmonary condition (including asthma), cardiovascular (except hypertension), renal, hepatic, haematological (including sickle cell disease), neurologic, neuromuscular, or metabolic disorders (including diabetes mellitus);
- Immunosuppression, including that caused by medications or by HIV;
- Pregnant women;
- Residents of nursing homes and other chronic-care facilities;
- Obesity.

5. Mode of transmission

Influenza spreads from person to person by droplet infection created by sneezing, coughing or talking. The portal of entry is the respiratory tract.

6. Incubation period

It could range from one to seven days, and most likely from one to four days.

7. Signs and symptoms

An acute Respiratory Tract Infection (RTI), caused by Influenza virus, characterised by sudden onset of: Fever/chills, Headache, myalgia, Sore throat, Cough, Coryza, and Prostration.

Range of symptoms differs by age. Vomiting & diarrhoea in children/elderly. Fever alone in infants may be atypical in elderly. Serious complications can occur among high risk groups.

8. Diagnosis

The recommended test to confirm the diagnosis of H1N1 influenza A virus is real-time Polymerase Chain Reaction (RT-PCR) in designated laboratories. Also viral culture and four-fold rise in new influenza A (H1N1) virus-specific neutralizing antibodies can be done.

8.1. What sample to be collected?

- Nasopharyngeal/oropharyngeal swabs.
- Bronchoalveolar lavage.
- Tracheal aspirates.
- Nasopharyngeal / oropharyngeal aspirates as washes.
- Samples should be collected in VTM.

8.2. Transportation of samples:

- All samples should be kept at 2-8 degree Celsius until they can be placed at -70°C.
- Samples transported on dry ice in triple packaging.
- Clear labels with patient's complete information.
- Samples should be sent within 24 hrs.

9. Treatment

Treatment with Oseltamivir or Zanamivir is recommended for all people with suspected or confirmed influenza who require hospitalization. It is given in a dose of 75 mg Bid in adults.

All individuals seeking consultations for flu like symptoms should be screened at healthcare facilities both Government and private or examined by a doctor and these will be categorized as under:

9.1. Category- A

- Patients with **mild fever plus cough / sore throat** with or without body ache, headache, diarrhoea and vomiting will be categorized as Category-A. They **do not require** Oseltamivir and should be treated for the symptoms mentioned above. The patients should be monitored for their progress and reassessed at 24 to 48 hours by the doctor.
- **No testing of the patient for H1N1 is required.**
- Patients should confine themselves at home and avoid mixing up with public and high risk members in the family.

9.2. Category-B

1. In addition to all the signs and symptoms mentioned under Category-A, if the patient has high grade fever and severe sore throat, may require home isolation and Oseltamivir;
2. In addition to all the signs and symptoms mentioned under Category-A, individuals having one or more of the following high risk conditions shall be treated with Oseltamivir:
 - Children with mild illness but with predisposing risk factors.
 - Pregnant women;
 - Persons aged 65 years or older;
 - Patients with lung diseases, heart disease, liver disease, kidney disease, blood

disorders, diabetes, neurological disorders, cancer and HIV/AIDS;

- Patients on long term cortisone therapy.
- Obese persons.

No tests for H1N1 is required for Category-B (1) and (2).

All patients of Category-B (1) & (2) should confine themselves at home and avoid mixing with public and high risk members in the family.

Broad Spectrum antibiotics as per the Guideline for Community-acquired pneumonia (CAP) may be prescribed.

9.3. Category-C

In addition to the above signs and symptoms of Category-A and B, if the patient has one or more of the following:

- Breathlessness, chest pain, drowsiness, fall in blood pressure, sputum mixed with blood, bluish discolouration of nails;
- Children with influenza like illness who had a severe disease as manifested by the red flag signs (Somnolence, high and persistent fever, inability to feed well, convulsions, shortness of breath, difficulty in breathing, etc.).
- Worsening of underlying chronic conditions.

All these patients mentioned above in Category-C require testing, immediate hospitalization and treatment.

9.4. Oseltamivir Medication -Doses Details

Oseltamivir is the recommended drug for treatment.

In the current phase, if a person confirms to the case definition of suspect case, should be provided Oseltamivir.

Dose for treatment is as follows:

By Weight:

- | | |
|---------------------|---------------------|
| - For weight <15 kg | 30 mg BD for 5 days |
| - 15-23 kg | 45 mg BD for 5 days |
| - 24-<40 kg | 60 mg BD for 5 days |
| - >40 kg | 75 mg BD for 5 days |

10. Vaccine

A Vaccine is available for swine flu. It gives immunity for one year. It is given to pregnant

mothers, high risk persons (DM, HT, Obese, Respiratory diseases, Immunocompromised).

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. Ministry of Health and Family Welfare. H1N1 Guidelines. [cited 2016 Jul 5]
Available from: <http://mohfw.gov.in/index4.php?lang=1&level=0&linkid=372&lid=3066>



49. DIARRHOEAL DISEASES

Diarrhoeal diseases include acute diarrhoea, persistent diarrhoea (diarrhoea duration two weeks or more) and dysentery (blood stained stools with fever). Diarrhoeal diseases are one of the most common causes of epidemic in our State. Most of the deaths in diarrhoeal diseases are due to dehydration which is preventable by timely and adequate replacement of fluids.

- Acute diarrhoea – Cholera, Rota virus, food poisoning, gastrointestinal disorders and medications (rare).
- Persistent diarrhoea – Chronic bacterial infections, inflammatory bowel disorders, malabsorption syndrome.
- Dysentery – Amoebiasis, Giardiasis, Shigellosis.

1. Following are important causes of diarrhoeal diseases in rural areas

Table-1: Diagnosis of diarrhoea

Sign/symptom	Acute diarrhoea	Persistent diarrhoea	Dysentery
Frequency of stools/day	Three or more	Three or more	Three or more
Consistency of stools	Watery	Variable	Variable
Duration of diarrhea	Less than 2 weeks	Two or more weeks	Less than 2 weeks
H/o fever	No	Variable	Yes
H/o blood stained mucus	No	Variable	Yes
Effect on appetite	No	Loss of appetite	Loss of appetite
Dehydration	Important, may lead to severe dehydration if not treated in time.	Patient may have some dehydration.	Patient may have some dehydration
Treatment principle	Management of dehydration is priority	Start management of dehydration. Simultaneously find cause of persistent diarrhoea and treat accordingly.	Start management of dehydration. Simultaneously start appropriate antibiotics.
Long term effects	No long term effect for occasional episodes. Repeated attacks may lead to PEM.	If not treated correctly, child may get severe Protein Energy Malnutrition	Repeated attacks may lead to Protein Energy Malnutrition

Table-2: Dehydration Diagnosis Chart

Sign/Symptom	Severity of symptoms and signs (Encircle <i>the</i> finding)		
	No dehydration	Some dehydration	Severe dehydration
General condition of Patient	Patient well alert	Restless and irritable	Lethargic, unconscious, floppy
Presence of thirst	Normal/not thirsty	Thirsty, drinks water immediately when offered	Not able to drink
Dryness of mouth and tongue	Moist mouth and tongue	Mouth and tongue dry	Mouth and tongue very dry
Condition of eyes	Normal	Sunken	Very sunken, patient's face looks like old man's face.
Condition of tears	Tears appear while crying	Tears appear while crying	No tears, dry eyes even in crying child
Skin turgor	Normal. Pinch to skin immediately goes back to normal.	Pinch slowly goes back and takes some time to become flat.	Pinch remains as it is for 2-3 seconds and then slowly goes back.
Classification of dehydration	No dehydration	Some dehydration	Severe dehydration
Treatment of dehydration	Plan – A	Plan – B	Plan – C

2. Important diseases causing diarrhoea or dysentery in adults

2.1. Cholera

Cholera is the most important diarrhoeal disease which leads to rapid dehydration.

Etiologic agent

Cholera is caused by bacteria *Vibrio cholerae* which exists in two biotypes, Classical and El tor. Each biotype is further divided into two subgroups Inaba and Ogawa.

Clinical manifestations

Cholera is an acute infection of small intestine manifested as watery diarrhoea and vomiting. Clinical spectrum of cholera is broad, ranging from in apparent infection to cholera gravis, which may be fatal in few hours. Incubation period of 24 to 48 hours is followed by abrupt onset of painless, profuse and watery diarrhoea associated with vomiting.

Symptoms and signs of cholera are entirely due to loss of large volume of isotonic fluid and resultant depletion of intravascular and extra vascular fluid leading to severe dehydration, metabolic acidosis and hypokalaemia. Patient develops thirst, cramps, and anxiety due to depleting isotonic fluid.

Diagnosis

Suspect cholera when patient has severe watery diarrhoea and vomiting. Collect stool sample of suspected cases in Cary Blair media and transport to District Public Health Laboratory. However, treatment and control measures should be started immediately on the basis of clinical symptomatology without waiting for laboratory confirmation.

Treatment

Carefully examine patient for signs of dehydration and treat as per dehydration status. Most important treatment of cholera is rehydration of patient with ORS and Ringer's Lactate. In addition to this, start one of the following antibiotics to patient -

- Cap Doxycycline 6 mg/kg/ day as a single dose for 3 days OR

- Cap Tetracycline 50 mg/kg/day in 4 divided doses for 3 days OR
- Tab Erythromycin 30mg/kg/day in 3 divided doses for 3 days.

3. Diagnosis of dehydration

Although number of organisms are responsible for causing diarrhoea, clinical presentation is same i.e. passage of watery stools leading to dehydration in all these cases. Therefore, assessment of dehydration status and correct management of dehydration by ORT is mainstay of diarrhoeal disease control programme.

4. Management of dehydration

Most important aspect in management of diarrhoeal diseases is correction of dehydration.

Treatment of dehydration is divided into three plans as follows -

- Plan-A: For patients with no dehydration – Principle is to prevent dehydration.
- Plan-B: For patients with some dehydration – Principle is treatment of some dehydration and preventing patient from going into severe dehydration.
- Plan-C: For patients with severe dehydration - This is lifesaving plan. Rehydrate patient as early as possible and prevent from going again into severe dehydration.

Description of treatment plans in details is as follows.

4.1. Plan-A

Plan-A is for patients who are having diarrhoea but no signs of dehydration.

4.1.1 Principle of treatment

As diarrhoea is continuing, there is continuous loss of water and electrolytes from body of patient which may lead to dehydration. Therefore, principle of Plan-A schedule is correction of whatever loss of water and electrolytes before the patient develops signs of dehydration. Plan-A can be advised at home to caretaker of patient. However, make sure that caretaker has understood danger signs of dehydration (like thirst). Following steps are recommended in Plan-A.

a. Home available fluids

- Advise to give Home Available Fluids (HAF) e.g. sorbet, lassi, vegetable soup, kheer, buttermilk, tea, coconut water, etc. i.e. any liquid available at home to patient as much he/she can drink.
- Continue breast feeding and feeding – If child is being breastfed, then breast-feeding should be continued. Regular feeding of non-breast fed child should also be continued.

b. ORS to prevent dehydration

- If frequency and amount of diarrhoea is not declining or amount of stool is large, then start ORS.
- Contents of WHO ORS are as follows – (New low osmolarity ORS).

Sodium chloride	-	2.6 grams
Potassium Chloride	-	1.5 grams
Trisodium Citrate	-	2.9 grams
Glucose	-	13.5 grams

Dissolve the packet in one liter of water to prepare ORS.

- Show caretaker how to prepare ORS.

Following steps should be carried out for preparation of ORS -

- Take clean pot of one and half liter capacity and one clean spoon.
- Pour 1 liter of clean drinking water in the pot. (No need to boil water).
- Add whole packet of ORS into one-liter of water and stir till all powder is dissolved. Now ORS is ready for use.
- Give ORS by cup or spoon to small children and by glass to older children and to adults as per indicated dose.
- If patient has vomiting, wait for 5 minutes and start again.
- Keep ORS covered. Once prepared ORS should be used within 24 hours. Do not use ORS beyond 24 hours, as there are chances of contamination.

- If child develops swelling on eyelids, stop ORS as it indicates overhydration.
- Ask her to give ORS in following doses after passage of each liquid stool.

Less than 6 months	-	50 ml
6 months to 2 years	-	50 - 100 ml
2 to 5 years	-	100 - 200 ml

4.2. Plan -B

Start Plan-B treatment to patients showing signs and symptoms of some dehydration as per dehydration diagnosis chart. Aim of this plan is to correct dehydration and prevent patient from going into severe dehydration.

4.2.1 Principle of treatment

Patient with some dehydration should be given ORS for correction of dehydration.

Dose of ORS: Dose of ORS is calculated, preferably according to weight of patient. Give ORS in a dose of 100 ml/kg in 4 hours. If weighing is not possible, calculate age wise ORS requirement for four hours as follows –

Table -3: Age wise ORS requirement for four hours

Age	< 4 months	4–11 months	12–23 months	2 – 4 years	5 – 14 years	15 + years
Dose	200-400 ml	400 – 600 ml	600 – 800 ml	800 – 1200 ml	1200 – 2200 ml	2200-4000 ml

Continue breast feeding and feeding – If child is being breastfed, then breast-feeding should be continued. Regular feeding of non-breast fed child should also be continued.

4.2.2 Re-examination of patient

Re-examine patient after every four hours for status of dehydration with the help of Dehydration. (Table 4)

Table-4: Management advice based on re-examination findings

Condition of patient on re-examination	Management advise
Patient improves, no signs of dehydration on examination and diarrhoea stops	Keep patient under observation for 24 hours. Continue HAF. Observe if diarrhoea and/or vomiting start again.
Patient improves, no signs of dehydration on examination but diarrhoea continues	Continue giving ORS in doses suggested in Plan-A, re-examine after four hours.
Dehydration status same	Continue with Plan-B. Check whether ORS is being given in correct dose. Re-examine after four hours.
Signs of severe dehydration appear	Switch on to Plan - C (start IV fluids). Continue to give ORS as much as possible.

4.3. Plan – C

If signs and symptoms of patient are suggestive of severe dehydration, start Plan – C. This is emergency plan. Incorrect or incomplete management of severely dehydrated patient may lead to death of patient. Medical Officer must personally examine patient and treat for severe dehydration.

4.3.1 Principles of management

Principle of management of severe dehydration is replacing fluid loss by giving rapid IV infusion. Only Ringer's lactate should be used as IV fluid and the dose is 100 ml/kg body weight.

Table -4: Details of Ringer’s Lactate administration

Age group	Intensive phase	Maintenance phase	Duration of treatment	Remarks
Infants (0-1 year)	30 ml/kg body wt. during first 1 hour.	70 ml/kg body wt. in next 5 hours.	6 hours	Assess patient after every 6 hours
Older children and adults	30 ml/kg body wt. in first half hour.	70 ml/kg body wt. in next 2½ hours.	3 hours	Assess patient after every 3 hours

4.3.2 Re-examination of patient

Re-examine patient after every six hours in infants and three hours in adults for status of

dehydration with the help of dehydration diagnosis chart and decide management plan as follows -

Table-5: Treatment advice based on condition of patient

Condition of patient	Treatment advise
Patient improves, no signs of dehydration on examination and diarrhoea stops	Keep patient under observation for 24 hours as patient may start diarrhoea/vomiting again
Patient improves, no signs of dehydration on examination but diarrhoea continues	Continue giving ORS (Plan-A)
Patient improves, signs of some dehydration on examination.	Stop IV fluids after required dose is administered. Continue giving ORS (Plan-B)
Dehydration status same	Continue with Plan-C. Check for any complications like anuria. If yes, carefully examine the patient and decide for referral. Continue giving IV during transportation of patient.

ORS or Oral rehydration therapy (in case ORS is not available), irrespective of type of dehydration.

5. Use of antibiotics and other drugs

Antibiotics are recommended only to suspected patients of cholera and dysentery. Other drugs like anti motility drugs, binding agents, anti-secretory agents and steroids are not of any use in management of diarrhea. They are harmful to patients and therefore not at all recommended for treatment. Judicious use of antibiotics is appropriate in selected patients. Severely ill patients with febrile dysentery can be treated with Ciprofloxacin 500mg BD for 3-5 days.

Use of Zinc Tablets

Zinc Dosage Recommendation:

Zinc is very safe drug and has a very large window of safety. Zinc dispersible tablets are to be given in each diarrhoeal episode along with low osmolality

Zinc administration as per age of child:

a) Children from 2-6 months:

Children aged between 2-6 months should be given 10 mg of elemental zinc per day for a total period of 14 days from the day of onset of diarrhoea. A tablet of zinc contains 20 mg of elemental zinc. Therefore, half tablet should be given to the children in this age group.

Zinc when supplied in the form of dispersible tablets, easily dissolves in breast milk or water. Therefore, in infants below 6 months of age, the tablet should be given by dissolving in breast milk and in infants above 6 months of age, it should be given by dissolving in breast milk or water.

b) Children above 6 months:

One full tablet (20mg) should be given to all children with diarrhoea above 6 months of age. It should start from the day of onset of diarrhoea and continued for a total period of 14 days.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. World Health Organization (WHO) Factsheet. April 2013. [cited 2016 Jul 5]

Available from: <http://www.who.int/mediacentre/factsheets/fs330/en/>

2. Keusch GT, Fontaine O, Bhargava A, Pinto CB, Bhutta ZA, Gotuzzo E et al. Diarrheal Diseases, Disease Control Priorities in Developing countries. 2nd Edition. Chapter 19. New York: Oxford University Press 2006. [cited 2016 Jul 5]

Available from: <http://www.ncbi.nlm.nih.gov/books/NBK11764/>



50. RHEUMATOID ARTHRITIS

1. Introduction

Rheumatoid arthritis (RA) is a chronic inflammatory disease of unknown aetiology marked by a symmetric, peripheral polyarthritis. It is the most common form of chronic inflammatory arthritis and often results in joint damage and physical disability.

2. When to suspect

- The incidence of RA peaks between 25 and 55 years of age.
- Early morning joint stiffness lasting more than 1 hour and easing with physical activity.
- Earliest involved joints typically the small joints of the hands and feet.
- The wrists, metacarpophalangeal (MCP), and proximal interphalangeal (PIP) joints stand out as the most frequently involved joints.
- Flexor tendon tenosynovitis is a frequent hallmark of RA.

Deformities are Swan-neck deformity, Boutonnière deformity, Z-line deformity.



Figure-50.1: Hand changes in RA

Extra-articular features –

- i. Neurologic-atlanto-axial dislocation, cervical myelopathy.
- ii. Haematological- Anaemia of chronic disease, neutropenia, splenomegaly, Felty's syndrome.
- iii. GI-vasculitis.
- iv. Skeletal- osteoporosis.
- v. Ocular- Episcleritis, Scleritis, Keratoconjunctivitis sicca.

- vi. Oral - Xerostomia, periodontitis.
- vii. Pulmonary – Pleural effusion, pulmonary nodules, interstitial lung disease, pulmonary vasculitis, organizing pneumonia.
- viii. Cardiac- Pericarditis, IHD, myocarditis, cardiomyopathy, mitral regurgitation.
- ix. Renal – Membranous nephropathy.
- x. Skin – Rheumatoid nodules, pyoderma gangrenosum.

3. Investigations

The clinical diagnosis of RA is largely based on signs and symptoms of a chronic inflammatory arthritis, with laboratory and radiographic results providing important supplemental information.

3.1 Rheumatoid factor

Serum IgM RF found in 75-80% of RA patients; therefore, the negative result does not exclude RA.

Found in other connective diseases as well.

3.2 Anti –CCP antibodies

Highly specific 95%

So, useful for distinguishing RA from other forms of arthritis.

3.3 Synovial fluid analysis-

Inflammatory state.

Synovial fluid WBC count ranges from 5000 to 50000 WBC/mm³.

Useful for confirming inflammatory arthritis, excluding infection or crystal induced arthritis.

3.4 X ray hands

Juxta articular osteopenia,

Other findings include soft tissue swelling, symmetric joint space loss, subchondral erosions.

3.5 MRI

Offers greatest sensitivity for detecting synovitis, joint effusions as well as early bone and bone marrow changes.

4. Treatment

Table-1: Treatment of RA

Non-pharmacologic treatment	Pharmacologic treatment
Counselling, physiotherapy, diet	NSAIDS
Stress reduction, exercise	Glucocorticoids
	DMARDS

4.1 NSAIDs

Tab. Ibuprofen (200 mg BD or TDS) / Tab. Diclofenac (50 mg BD) - Now considered to be adjunctive therapy for management of symptoms uncontrolled by other measures.

4.2 Glucocorticoids

May be administered in low-to-moderate doses to achieve rapid disease Control before the onset of fully effective DMARD therapy, which often takes several weeks or even months.

4.3 DMARDS

Hydroxychloroquine, Methotrexate, Leflunomide, Sulfasalazine, Biologicals, anti TNF agents, Rituximab.

5. When to refer

Severe case of rheumatoid arthritis not responding to first line therapy (Hydroxychloroquine, Methotrexate, NSAIDS), with extra articular manifestations should be referred to Higher institute for further work up.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015



51. SNAKE BITE

1. Introduction

Envenoming by poisonous animals (Snakes, Scorpions, Wasps, Ants and Spiders) is an occupational hazard often faced by farmers, farm labourers. Poisoning by venomous snakebite is a common acute life-threatening time-limiting medical emergency. The majority of current first aid methods adopted by the victims such as tourniquet, cutting and suction and herbal remedies are completely ineffective and dangerous. It is now recommended to adopt what has been called the 'Do it R.I.G.H.T.'

approach, stressing the need for Reassurance, Immobilization of the part, getting to hospital without delay and telling the doctor of any symptoms that develops.

2. Common Poisonous snakes in India

(1) Cobra (2) Krait (3) Russell's Viper (4) Saw Scaled Viper (5) Indian Pit Viper (6) Sea snake

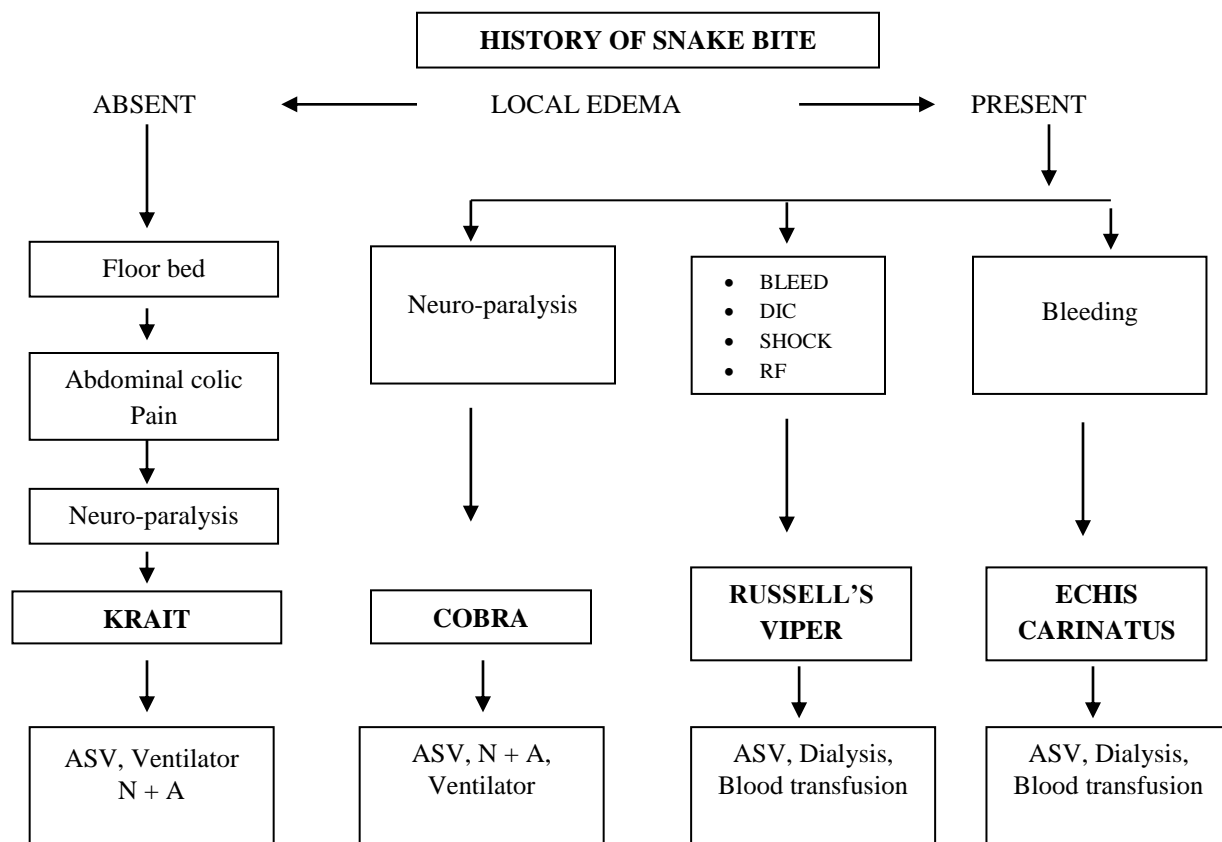


Figure 51.1. Snake Bite: Diagnostic Algorithm and Treatment

(ASV- Anti-snake venom, N- Neostigmine, A- Atropine, RF- renal failure, DIC- Disseminated intravascular coagulation)

3. Clinical manifestations

3.1. Common Indian Krait (Bungarus Caeruleus)

Local Names- Kala gandait, Kala taro, Kandar, Manyar, Chitti, Kattu Viriyan and Valla Pamboo. The common krait is regarded as the most dangerous species of venomous snakes in the Indian

subcontinent. Majority of krait bite cases are reported between 11 pm – 5 am.

3.1.1 Symptoms

- Mild pain at the site
- Paraesthesia or numbness
- Abdominal pain/vomiting/chest pain
- Difficulty in bringing tongue beyond teeth margin
- Slurred speech
- Difficulty in breathing

3.1.2 Signs

- Bradycardia, hypotension
- Bilateral ptosis, external ophthalmoplegia, dysphagia
- Paralysis, coma

3.1.3 Management

(a) First aid at home or place where bite happens:

If one succeeds in locating the bite site on the victim's body, clean the surface where venom is deposited by clean cloth or cotton. Keep the bitten part below heart level.

(b) At hospital:

Thorough clinical examination including neurological exam

Haemogram, urine exam, ECG, serum electrolytes, renal profile

Anti-snake venom (ASV):

- On arrival 100ml (10 vials) ASV to be added to 200 cc of normal saline run over 30-50 minutes.
- Repeat dose of 100 ml (10 vials) after 30 min if no improvement in neurological manifestation.
- Maximum dose 200 ml (20 vials).

Ventilation

Indicated if victim has pooling of saliva, unable to lift the neck from pillow, reduction in oxygen saturation, respiratory failure, abdominal-thoracic respiration, suffocation and signs of cerebral hypoxia.

Refer the patient to higher centre with intubation and Ambu bag ventilation

Acetylcholine esterase inhibitor (AChEI):

Indian common krait venom contains both pre and post synaptic blocker. Whether victim responds to AChEI or not can be confirmed by putting ice-filled glove finger over eyelid. Hypothermia sensitizes the

ACh receptors of Acetyl choline. If there is slight improvement in ptosis, you can try AChEI. Neostigmine 50 µg/kg over first hour & then 25 µg/kg in the next hour preceded by Atropine

3.2. Cobra Bite

Cobra bite tends to occur during day time.

3.2.1. Symptoms

- Pain at site
- Progressive swelling / ecchymosis
- Blurred vision

3.2.2 Signs

- Sinus bradycardia, hypotension
- Ptosis, bulbar palsy
- Respiratory depression

3.2.3 Management

Victim should not be allowed to walk or run and the bitten part should be kept below heart level.

On arrival 100 ml (10 vial) ASV to be added to 200 cc of normal saline run over 30-50 minutes.

Maximum dose 200-250 ml (20-25 vials).

Neostigmine 50 µg/kg over first hr. & then 25 µg/kg in the next hr preceded by Atropine

If patient develop respiratory paralysis intubate the patient and refer to higher centre

Local wound care is done by intravenous antibiotic, sterile dressing and skin grafting of all victims with non-healing wound.

3.3. Sea snakes

Sea snake bite cases are reported from coastal region. Fishermen accidentally handle sea snake resulting in envenoming. Its venom is neurotoxin, myotoxic and haematotoxic.

3.3.1 Symptoms

- Headache
- Nausea
- Vomiting
- Tingling numbness
- Foreign body sensation in throat
- Swelling of tongue
- Severe muscle pain
- Brown coloured urine

3.3.2 Signs

- Trismus
- Muscular paralysis

- Respiratory arrest
- Myoglobinuria

3.3.3 Management

On arrival 100 ml (10 vial) ASV to be added to 200 cc of normal saline run over 30-50 minutes.

Ventilator for respiratory failure. Refer to higher centre with intubation.

Correction of hyperkalaemia - Calcium gluconate, insulin glucose drip (10% dextrose 100 ml add 12-unit insulin for 6 hours), salbutamol nebulization or dialysis.

3.4. Russell's viper

It is found in South Asian Countries. In Pakistan, India, Sri Lanka, Bangladesh, Burma and Thailand it ranks amongst the most important causes of snakebite mortality. With protecting the paddy, wheat by containing rodents (rats), the Russell's viper kills many farmers unlucky enough to treat on it during harvesting.

3.4.1 Symptoms

- Severe local pain at the site of bite.
- Rapid swelling progresses to whole limb within six to eight hours.

3.4.2 Signs

- Regional lymphangitis with ecchymosed skin.
- Development of compartment syndrome characterized by swelling, hypotension and shock.

Renal dysfunction

20-40% cases subsequently develop anuria, oliguria, acute renal failure. Renal angle tenderness is most important clinical sign for early diagnosis of renal failure. There is serial rise in blood urea and serum creatinine with acidosis and hyperkalaemia. Generalized anasarca, renal failure is due to tubular damage by venom itself.

Haemoglobinuria, hypotension and micro thrombi in the kidney contribute to the acute tubular necrosis which is the most common cause of death. Ptosis, bulbar palsy, internuclear ophthalmoplegia and respiratory paralysis due to presynaptic neuromuscular block in a Russell's viper bite poisoning are often seen and reported from Kerala (South India) and Sri Lanka.

3.4.3 Management

- No tourniquet
- Bitten part should be kept below heart level
- No intramuscular injection unless 20 MWBCT is done and blood clots within 20 minutes.

20-minutes whole blood clotting test (20 MWBCT)

Before injection of ASV take 2-3 ml of blood in a new dry glass test tube which is not irrigated by any detergents. Keep the tube undisturbed for 20 minutes and then tip it off. If blood does not clot, it confirms hypofibrinogenemia. ASV – 100 ml (10 Vials) ASV diluted in 200 ml of 5% dextrose run over 30 minute by intravenous route. If external bleeding does not stop within 20-30 minutes one can repeat 50 ml of ASV. Thrombocytopenic, abnormal fragmented RBC's are a diagnosis of DIC. In addition, if victim is too late in such situation in addition to ASV one has to try plasma products and whole blood transfusion which is rarely required if ASV is administered in time with adequate dose. Hypotension is to be managed with fluid and inotropic agents. Severe hypotension due to bleed in adrenal and pituitary glands and abdominal bleed and endothelial dysfunction with capillary leak needs heavy doses of intravenous methyl-prednisolone and correction of electrolytes.

Renal failure

One should keep in mind and look for renal failure from the time of admission. Risk factors such as hypotension, hypovolemia can be corrected. Intravenous frusemide 80-100 mg and oral acetyl cysteine 600 mg three times a day may help to arrest the renal damage.

Refer to higher centres for haemodialysis, if needed.

3.5. Saw scaled viper or Carpet viper or Echis carinatus

Farmers, hunters, labourers and persons walking bare foot or in jungle and rocky areas are often bitten by this snake.

3.5.1 Clinical manifestations

Soon after the bite within one hour there is development of swelling over the bitten part. Swelling progresses in more than one segment. The victim experiences a painful lymphadenopathy at the drainage area of the bitten part.

At times if the patient remains untreated, bleeding persists for 1-2 weeks in the form of blood stain sputum, haematuria and disappears on its own. Such patients are markedly anaemic and report to hospital

for weakness or non-healing cellulitis with uncontrolled bleeding from cellulitis.

3.5.2 Management

Local wound care

ASV required is 30-50 ml

3.6. Green pit viper and bamboo pit (*Trimeresurus*)

Pit viper victims report during the monsoon season.

3.6.1 Clinical manifestation

It shows up in sudden rapid development of massive oedema without regional involvement. Rarely the victim manifests external bleeding or renal failure.

3.6.2 Management

Anti-venom should be administered as soon as signs of systemic or severe local swelling are noted.

The mean times between envenoming and death are

- 8 hours (12 minutes to 120 hours) in Cobras,
- 18 minutes (3-63 hours) in *Bungarus Caeruleus*,
- 3 days (15 minutes to 264 hours) in Russell's viper
- 5 days (25 to 41 days) for *Echis-Carinatus*.

The approximate serum half-life of anti-venom in envenomed victims ranges from 26 to 95 hours. Before discharge envenomed victims should be closely observed daily for minimum 3 to 4 days.

ASV doses and repeat doses

The recommended initial dose of ASV is 8 to 10 vials administered over 1 hour.

Repeat doses for neurotoxic species is based on 1 to 2-hour rule.

Repeat doses for haematotoxic species is based on the 6-hour rule.

The maximum recommended dose for neurotoxic bite is 20 vials of ASV.

The maximum recommended dose for haematotoxic bite is 30 vials of ASV.

ASV Reactions

No ASV test doses are to be administered.

At the first sign of an adverse reaction the ASV is halted

Adrenaline 0.5 ml is given SC

Steroid and antihistamine perform a secondary supportive function to Adrenaline.

(National Snakebite Management Protocol 2008)

Bibliography:

1. India. Directorate General of Health Services. National Snakebite Management Protocol 2009. Ministry of Health and Family Welfare. [cited 2016 Jul 5]
Available from: <http://164.100.130.11:8091/nationalsnakebitemanagementprotocol.pdf>

Further Reading:

1. Warrell DA. Guidelines for the management of snake bites. World Health Organization. [cited 2016 Jul 5]
Available from: http://apps.searo.who.int/PDS_DOCS/B4508.pdf?ua=1



52. SCORPION STING

1. Introduction

- Scorpion envenomation is an occupational hazard for farmers, farm labourers, migrating population and hunters.
- The endemic areas are western Maharashtra, Karnataka and Konkan Region.
- Severe Scorpion stings are due to Mesobuthus Tamulus species of scorpion.

2. Clinical features

- The venoms of genera Hadrurus, Vaejovis and Uroctonus has local effects only including sharp burning, swelling and discoloration at the bite site.
- The second type of venom produced by the genera of the poisonous varieties of Centruroides and Mesobuthus contain neurotoxins which block sodium channels. This leads to spontaneous depolarization of parasympathetic and sympathetic nerves which results in stimulation.
- In adults, the first time is rarely dangerous. But if the second time, the person may die if not treated soon. The body becomes allergic after the first sting. So it is important to find out if he had an earlier scorpion sting.
- Severe pain, redness and swelling at the site of the sting.
- Clinically “autonomic storm” evoked due to venomous envenoming is characterized by transient parasympathetic stimulation- vomiting, profuse sweating, ropy salivation, bradycardia, ventricular premature contraction, priapism in male, hypotension and prolonged sympathetic stimulation - cold extremities, hypertension, tachycardia, pulmonary oedema and shock.

3. Gradation

On basis of clinical manifestations at the time of arrival to hospital and according to severity they are graded in 4 grades.

Grade 1: Severe excruciating local pain at the sting site radiating along with corresponding dermatomes, mild local oedema with sweating at the sting site, without systemic involvement.

Grade 2: Pain, paraesthesia remote from the site of sting, in addition to local findings.

Grade 3: Either cranial nerve / autonomic dysfunction.

Cold extremities, tachycardia, hypotension

(Respiratory rate > 24 per minute, basal rales or crackles in lungs).

Grade 4: Combined cranial nerve / autonomic dysfunction and somatic nerve dysfunction.

4. Investigations

ECG- Tall T waves is a common finding. Others are atrial arrhythmias, non-sustained ventricular tachycardia, and various conduction defects seen.

Chest X-ray - shows pulmonary oedema.

5. Management

If it is for the first time in adult, do the following:

- Give Paracetamol if possible, put ice on sting.
- Infiltration of site with local anaesthetic may relieve pain and anxiety.
- Anti-histaminic tablets can be given
- If the sting is for the second time in adult, or is in children under 5, do the following
 - IV Fluid management
 - Inj. Scorpion anti-venom 30 ml in 200 ml of normal saline neutralises circulating venom.
 - If evidence of myocarditis and pulmonary oedema, strict bed rest and management of heart failure is indicated.
 - Prazosin 0.5 mg 3 hourly orally for first 2 days (or 0.25 mg in children and for adults) is acceptable therapy
 - Injection Frusemide 20 to 60 mg IV to control Pulmonary oedema
 - Inj. Dobutamine 5-20 microgram/kg/min IV given in heart failure, tachycardia, pulmonary oedema with warm extremities
 - Patients in Pulmonary oedema may need Inj NTG drip 5 microgram/kg

- At times with severe respiratory distress may need non-invasive ventilation or mechanical ventilation.

Stage- 1 (0-4 hours)	-	Stage- 2 (4-6 hours)	-	Stage-3 (6-10 hours)	-	Stage-4 (10-12 hours)	-	Stage-5 (>12 hours)	-
Sweating Salivation Mydriasis Priapism Hypertension Hypotension Cold extremities	-	Hypertension Tachycardia Cold extremities	-	Tachycardia Hypotension Pulmonary oedema Cold extremities	-	Massive pulmonary oedema	-	Warm extremities Tachycardia Hypotension Pulmonary oedema Grey pallor (warm shock)	-
ASV + Prazosin	-	ASV + Prazosin	-	ASV +Prazosin + Dobutamine	-	ASV + SNP or NTG + NIV/MV	-	Dobutamine	-

Figure 52.1 Scorpion sting: stages, clinical presentation and treatment

(ASV – Anti Scorpion Venom, SNP- Sodium Nitro Prusside, NTG- Nitroglycerine, NIV-Non-invasive ventilation, MV – Mechanical Ventilation)

Bibliography:

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015

Further Reading:

1. Bawaskar HS, Bawaskar PH, Scorpion Sting: Update. January 2012, Vol 60. [Internet] [cited 2016 Jul 5]
Available from: http://www.silae.it/files/08_scorpion_sting_update.pdf
2. Chippaux JP, Emerging options for management of scorpion sting, Drug Des Devel Ther. 2012; 6: 165–173. [Internet] [cited 2016 Jul 5]
Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3401053/>



53. DOG BITE (RABIES)

Rabies can be transmitted by dog bites or licks of rabid animals on abraded skin and intact mucosa. Other animals which can transmit rabies are cat, monkey, horse, sheep, goat, mongoose, jackal, fox, hyena and bat. Exposure to rodents, rabbits and hares seldom requires specific anti-rabies treatment.

1. Clinical features

Prodromal symptoms- such as headache, malaise, sore throat and fever last about 3-4 days. Pain and tingling at the bitten site.

Stage of excitation- Patient is intolerant to noise; bright light or a cold draught. Aerophobia may be present. Hydrophobia is a characteristic symptom of rabies. Examination shows increased reflexes, dilatation of pupils, increased sweating, lacrimation and salivation. Mental changes include fear of death, anger, irritability and depression. Convulsions may occur resulting in death. The last stage is that of paralysis and coma. The total duration of illness lasts for 2-3 days.

Treatment (post-exposure prophylaxis)

2. Categories of dog bite

- Category I – Touching or feeding animals, licks on the intact skin
- Category II – Nibbling of uncovered skin, minor scratches or abrasions without bleeding, licks on broken skin
- Category III – Single or multiple transdermal bites or scratches, contamination of mucous membrane with saliva from licks; exposure to bat bites or scratches

The WHO recommended classification of animal bite for post-exposure treatment should be followed. Every instance of human exposure to a suspected rabid or wild animal must be treated as a category III. The post-exposure treatment is a three-pronged approach. All three carry equal importance and should be done simultaneously:

Table-1: WHO Guide for post-exposure treatment against rabies

<p>Category Type of contact with a suspect or confirmed rabid domestic or wild animal or animal unavailable for observation</p> <p>I. Touching or feeding of animals Licks on intact skin</p> <p>II. Nibbling of uncovered skin Minor scratches or abrasions without bleeding Licks on broken skin</p> <p>III. Single or multiple transdermal bites or scratches Contamination of mucous membrane with saliva (i.e. licks)</p>	<p>Recommended treatment.</p> <p>None, if reliable case history is available</p> <p>Administer vaccine immediately. Stop treatment if animal remains healthy throughout an observation period of 10 days or if animal is killed humanely and found to be negative for rabies by appropriate laboratory techniques</p> <p>Administer rabies immunoglobulin and vaccine immediately. Stop treatment if animal remains healthy throughout an observation period of 10 days or if animal is killed humanely and found to be negative for rabies by appropriate laboratory techniques.</p>
<p>A. Exposure to rodents, rabbits and hares seldom, if ever, requires specific anti-rabies treatment</p> <p>B. If an apparently healthy dog or cat in or from a low-risk area is placed under observation, the situation</p>	

may warrant delaying initiation of treatment.

- C. This observation period applies only to dogs and cats. Except in the case of threatened or endangered species, other domestic and wild animals suspected as rabid should be killed humanely and their tissues examined using appropriate laboratory techniques.

3. Treatment of dog bite

3.1. Management of wound

Immediate washing of the wound is a priority.

Wound toilet must be done even if several hours or days have elapsed. The wound is immediately flushed and washed with plenty of soap and water (avoid direct touching of wounds with bare hands). Punctured wounds should be irrigated with the help of catheters followed by 70% alcohol or povidone iodine application. The application of irritants (like chillies, oil, turmeric, lime, salt etc.) is unnecessary and damaging.

Do not suture bite wounds immediately. If suturing is required, hold it for 24-48 hours, applying minimum number of stitches under the cover of anti-rabies Immunoglobulin locally. Anti-tetanus treatment can be given after local wound treatment.

3.2. Passive immunization with rabies immunoglobulin (RIG)

Local infiltration of RIG in category III rabies-RIG should be infiltrated in the depth and around the wound even if the lesion has begun to heal followed by administration of anti-rabies vaccine.

(Caution: RIG should never be administered in the same syringe or at the same anatomical site as vaccine).

Doses of rabies immunoglobulin (IG)

Human rabies immunoglobulin (HRIG) 20 IU/kg (max 1500 IU), available in concentration of 150 IU/ml, it does not require any prior sensitivity testing. **SHOULD NEVER BE INJECTED INTRAVENOUSLY.** The anti-rabies sera should always be brought to room temperature (20-25°C) before use.

Or

Equine Anti-Rabies Serum (ERIG) 40 IU/kg (max 3000 IU), available in concentration of 300 IU/ml, given after prior skin sensitivity testing, single dose on day 0. Half the dose is infiltrated around the bitten wound and the rest is given I.M.

(Caution: A negative skin test must never reassure the physician that no anaphylactic reaction will occur. Avoid alcohol, glucocorticoids and chloroquine during vaccination; avoid multiple needle injections into the wound. Must not exceed the total recommended dose of IG as it may reduce the efficacy of the vaccine).

If the calculated dose of IG is insufficient to cover infiltration in all wounds, sterile saline can be used to dilute 2 or 3 fold to permit thorough infiltration.

RIG is not indicated beyond the seventh day after administration of the first dose of vaccine.

3.3. Active immunization with anti-rabies vaccine:

Anti-rabies vaccine (ARV)

- **Intramuscular schedule.**

The course for post-exposure prophylaxis consists of five injections (days 0, 3, 7, 14 and 28) irrespective of severity of exposure. The 6th injection (day 90) is optional for immunologically deficient and extremes of age and on steroid therapy. The dose of vaccine per injection is 2.5 IU/dose/ml for HDCV. Preferable site is deltoid; anterolateral thigh in children (**Caution:** Must NOT be given into gluteal muscle).

- **Intradermal (ID) schedule.**

(i) The 2 site ID TRC schedule (2-2-2-0-1-1) to be administered: One ID injection of 0.1 ml per ID site over each right and left deltoid on days 0, 3, 7 and 0.1 ml at a single site on days 28 and

90 or as per updated TRC schedule (2-2-2-0-2) on days 0, 3, 7 and 28.

Note: Correct ID injection should result in a raised papule with an orange peel appearance. If a papule is not observed, the needle should be withdrawn and vaccine re-administered correctly nearby.

(ii) The 8-site ID method (8-0-4-0-1-1) for use with HDC/PCECV in emergency, when no RIG is available.

The intradermal route is preferred as it reduces cost but must not be used in case of immunocompromised patients, individuals receiving long-term corticosteroids or other immunosuppressive therapy or chloroquine.

Anti-rabies vaccine should be kept and transported at a temperature range of +2°C to 8°C. The reconstituted vaccine should be used immediately or within 6-8 hours of reconstitution.

3.4. Post-exposure treatment of persons previously vaccinated

Managing re-exposure following post-exposure treatment with tissue culture vaccine (TCV)

If re-exposed, persons who have previously received full post-exposure treatment with a potent cell-culture

vaccine should be given only two booster doses, intramuscular (0.5 ml/1 ml) / intradermal (0.1 ml at 1 site) on days 0 and 3, but no rabies immunoglobulin. Proper wound toilet should be done.

4. Pre-exposure prophylaxis

Indications: Laboratory staff working with rabies virus, veterinarians, animal handlers and wildlife officers. Three full IM or ID doses of tissue culture vaccine given on days 0, 7, and 28. Laboratory staff and others at high continuing risk of exposure should have their neutralizing antibody titre checked every 6 months. If it is less than 0.5 IU/ml, a booster dose of vaccine should be given. Such individuals on getting exposed to rabies virus after successful pre-exposure immunization require only two booster injections of vaccine given on days 0 and 3 without any anti-rabies serum/RIGs.

5. Patient education

Dog bite (category II and III) is an emergency and as a general rule rabies post exposure treatment should not be delayed or deferred.

Immediate washing with plenty of water and disinfecting with alcohol/iodine.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. National Institute of Communicable Diseases. National Guidelines for Rabies Prophylaxis 2007. Ministry of Health and Family Welfare. [cited 2016 Jul 5]
Available from: http://www.ncdc.gov.in/Rabies_Guidelines.pdf



54. POISONING

1. Introduction

Poisoning due to the pesticides is increased due to the accessibility to the pesticides used in agriculture. Of the total burden of acute pesticide poisoning the majority of deaths are from self-poisoning with organophosphorus pesticides, aluminium phosphides and Paraquat.

2. Classification of Pesticides

2.1. Insecticides

- Acetylcholinesterase inhibitors
- Organophosphates
- Carbamates
- Organochlorines
- Pyrethrins
- Pyrethroids

2.2. Herbicides

- Dipyridyl pesticides
- Paraquat and diquat
- Dichlorophenoxyacetate weed killers
- Bromoxynil, 2,4-D

2.3. Fungicides

- Substituted benzene
- Chloroneb
- Chlorothalonil
- Thiocarbamates
- Organomercurials
- Methylmercury
- Phenyl mercuric acetate
- Molluscicides – Metaldehyde

2.4. Rodenticides

- Aluminium phosphide
- Zinc phosphide
- Warfarin and super warfarin compounds
- Yellow phosphorus
- Heavy metal: Thallium – containing pesticides

2.5. Insect repellents

- Diethyltoluamide (DEET)

2.6. Miscellaneous

- Anilides

3. Stepwise Case Approach

- Diagnosis - Suspect and identify poison, if possible.
- Management includes basic principles, antidotes, symptomatic and supportive treatment.
- Anticipate complications, preserve evidence and prevent sequelae as well as recurrence

4. Organophosphorus Compounds

Broadly OP compounds can be divided in to

1] **Dimethyl compounds** - Dichlorvos, Fenthion, Malathion, Methamidophos, Dimethoate

2] **Diethyl compounds** – Chlorpyrifos, Diazinon, Parathion-ethyl, Quinalphos

4.1. Mechanism of toxicity

Organophosphorus compounds inactivate acetylcholinesterase by phosphorylation leading to accumulation of acetylcholine at cholinergic synapses.

The rate of spontaneous reactivation of AChE is very slow with diethyl OPs while it is relatively fast with dimethyl OPs. Further, there is ageing of the phosphorylated enzyme after which the enzyme cannot be reactivated by oximes. The half-life of ageing of dimethyl phosphorylated and diethyl phosphorylated AChE *in vitro* is 3.7 hours and 33 hours, respectively, and the therapeutic windows therefore are 13 and 132 hours, respectively (4 times the half-life).

4.2. Organophosphorus intoxication results in triphasic illness including

- (a) Acute cholinergic syndrome
- (b) Intermediate syndrome
- (c) Organophosphate-induced delayed polyneuropathy

4.2.1 Acute cholinergic syndrome

- Acute cholinergic syndrome may occur within minutes
- Pathognomonic features via muscarinic and nicotinic receptors-

Muscarinic effects– Include meiosis, bronchorrhea, salivation, lacrimation, pain in abdomen, bradycardia, urination, defecation

Nicotinic effects– Muscle fasciculation, muscle cramps, flaccid muscle weakness with reduced tendon reflexes, tachycardia, hypertension

Central nervous system effects– Headache, dizziness, confusion, convulsions, central respiratory depression, coma

4.2.2 Intermediate syndrome

- This begins 48 hours after poisoning in approximately 20% of patients but may be delayed for 72-96 hours.
- The onset is often rapid with progression of muscular weakness from ocular muscles to neck and proximal limbs to respiratory muscles over 24 hours.
- Endotracheal intubation and ventilation are to be done, if not instituted early, cyanosis, coma and death may follow rapidly.

4.2.3 Organophosphate induced delayed polyneuropathy

- This occurs 1-3 weeks after acute exposure and uncertain period following chronic exposure due to degeneration of long myelinated nerve fibres.
- Cramping muscle pain in the legs are followed by numbness and paraesthesia in distal upper and lower limbs.
- Symmetrical flaccid paralysis in distal muscles especially in the legs. The dominant hand may be more affected.

4.3. Investigations

Plasma cholinesterase (pseudo cholinesterase) is less reliable. Red cell cholinesterase level falls to 20% of normal when symptoms appear.

4.4. Treatment of Organophosphorus Poisoning

- i. Airway, breathing and circulation should be ensured and monitored.

- ii. Further contamination is prevented by removal from the site of exposure and of contaminated clothing. Skin should be cleaned thoroughly with water.
- iii. The airway is cleared and high-flow oxygen is administered.
- iv. Direct mouth to mouth and nose resuscitation must be avoided.
- v. Following ingestion, gastric lavage must be done within an hour of intake, followed by activated charcoal via nasogastric tube after establishing intravenous and airway protection.

Sample should be collected from the gastric lavage, sealed and handed over to the police registering the medicolegal case.

- vi. Convulsions are treated with intravenous diazepam 10 mg or midazolam 2 mg.
- vii. Monitoring of ECG, blood gases, temperature, blood urea and serum electrolytes, amylase and glucose is mandatory.

- **For muscarinic effects**

Injection Atropine 1.8-3mg bolus immediately – double the dose every 5minutes until atropinized. Once patient is atropinized give 20%- 30% dose required for atropinisation as infusion/hour [5 mg/hour]. The best guide to adequate atropinisation is to monitor features of cholinergic poisoning (Bradycardia, Sweating, meiosis, bronchorrhea and hypotension). A confused, agitated, febrile patient with no bowel sounds and a full bladder with urinary retention certainly has atropine toxicity, indicating the need to reduce or stop atropine temporarily.

- **For nicotinic effects**

Pralidoxime chloride- Cholinesterase reactivator which reverses the nicotinic effects and some CNS effects. It is given 1 gm bolus in 30 minutes then infusion at 0.5 gm/hour. (Loading dose of 30mg/kg and 10 mg/kg/hour infusion).

- **Treatment of the intermediate syndrome**

Early institution of ventilatory support, which may be required for a prolonged duration, is essential for management. Close monitoring of

respiratory function such as chest expansion, arterial blood gas monitoring and oxygen saturation is essential to identify the onset and monitor the progress of the intermediate syndrome. Some patients develop an offensive and profuse diarrhoea and it is important to maintain a close watch and a positive fluid balance. Recovery usually occurs without residual deficit.

5. Organochloride Compounds (OC)

The commonly used OC insecticides are Endrin, Aldrin, Benzene Hexachloride (BHC), Endosulphan, Dieldrin, Toxaphene, DDT, Heptachlor, Kepone, Dicofol, Methoxychlor, etc. DDT, the most toxic OC, is available in dry powder form or as a mixture with other pesticides in powder or liquid form.

5.1. Mechanism of toxicity

OC compounds impair nervous system function by depolarization of the nerve membranes they also cause sensitization of the myocardium to both endogenous as well as exogenous catecholamine and predispose to arrhythmias.

5.2. Clinical effects within minutes to hours

- Nausea
- Vomiting
- Agitation
- Fasciculation
- Paraesthesia of face and extremities
- Seizures dizziness
- Tremors
- Myoclonus
- Coma
- Respiratory depression and death

5.3. Complications

Hyperthermia, Rhabdomyolysis, Pulmonary oedema and Disseminated intravascular coagulation. Lindane is particularly toxic to the central nervous system. It can also produce alterations in the ECG including rhythm abnormalities and changes in ST-T waves

Bibliography:

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015

suggesting hyperkalaemia. Besides the features related to OCs, associated solvents may produce aspiration pneumonitis.

5.4. Management

- Nasogastric aspirate may be useful if liquid preparation has been taken and should be kept and handed over to medical official for medicolegal purposes.
- Activated charcoal is given within 1 hour of ingestion.
- Seizures should be treated with Benzodiazepines. (Diazepam 10 mg or Midazolam 2 mg I.V.)
- Patients should be kept on cardiac monitor. Use Dopamine instead of Epinephrine if patient has hypotension, as OC compounds sensitise the myocardium.

6. Carbamate Poisoning

Carbamate insecticides- Aldicarb, Carbofuran Methomyl inhibit a number of tissue esterases-AchE. Aldicarb, Benomyl, Carbaryl, Carbendazim, Carbofuran, Propoxur, Triallate, etc. are the commonly used carbamates.

6.1. Clinical features

Clinical features are similar and less severe compared to organophosphorus poisoning.

6.2. Complications

Pancreatitis and death.

6.3. Treatment

Nasogastric aspirate may be useful if liquid preparation has been taken and should be kept and handed over to medical official for medico-legal purposes.

Injection Atropine 0.5-1mg I.V. in small doses for an adult till atropinisation occurs.

Role of PAM is unclear.

Further Reading:

1. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet*. [Internet] 2008 Feb 16; 371(9612): 597–607. [cited 2016 Jul 5]

Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2493390/>



55. ALCOHOL INTOXICATION /ALCOHOL WITHDRAWAL

1. When to Suspect

Alcohol has CNS depressant effect; hence person intoxicated with alcohol will present with depressed level of consciousness, sometimes with respiratory depression, cardiac arrhythmia, or blood pressure instability.

If the patient agrees to stop drinking, sudden decreases in alcohol intake can produce withdrawal symptoms, many of which are the opposite of those produced by intoxication. Features include tremor of the hands (shakes); agitation and anxiety; autonomic nervous system over activity including an increase in pulse, respiratory rate, and body temperature; and insomnia. These symptoms usually begin within 5–10 hours of decreasing ethanol intake, peak on day 2 or 3, and improve by day 4 or 5. About 2–5% of alcoholics experience a withdrawal seizure.

2. Investigation

- History of Alcoholism
- Blood ethanol level (Avoid cleaning with spirit while collecting Sample)

- CBC, LFT, obtaining toxicology screens for opioids or other CNS depressants
- Neuroimaging

3. Treatment

- Adequate nutrition and oral or I.V. Vitamin B complex, including 50–100 mg of Thiamine daily for a week
- Inj. Dextrose 25% in patients of altered sensorium (dose 100 cc 25% dextrose)
- Administering any depressant in doses that decrease the agitation- 25–50mg of Chlordiazepoxide or 10 mg of Diazepam given PO every 4–6 hour on the first day, with doses then decreased to zero over the next 5 days.
- Rehabilitation

4. Whom to Refer

Those with poor Glasgow coma scale, Recurrent Seizure, Focal neurological deficits or with delirium tremens / Korsakoff psychosis.

Bibliography:

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015

Further Reading:

1. India. National Health mission. Standard Treatment Guidelines; Alcohol and Substance use. Ministry of Health and Family Welfare. [cited 2016 Jul 5]
Available from:
<http://nrhm.gov.in/images/pdf/guidelines/nrhm-guidelines/stg/alcohol-sustance-use-disorders.pdf>



56. ANAEMIA

1. Introduction

Anaemia is one of the most common disease a physician can come across in the community. It can be defined as HB < 13 g/dL for male and < 12 g/dL for females as per WHO.

Anaemia can be due to nutritional deficiency, blood loss, and increase in destruction of RBCs or due to disturbance in formation of RBCs in bone marrow. Detailed history and clinical examination is must to reach the diagnosis. Patients especially females don't report to clinics until they have severe anaemia. Chronic anaemia patients are usually well adjusted to HB as low as 5 g/dL.

2. When to suspect

Symptoms

- i. Easy fatigability
- ii. Breathlessness
- iii. Swelling of feet
- iv. Hypo menorrhoea, amenorrhoea
- v. Stunted growth in adolescent

3. History to be inquired for aetiology of anaemia

3.1. Acute

- i. H/O blood loss –hematemesis, haemoptysis or any other
- ii. H/O Fever or jaundice – acute blood loss due to either haemolysis or blood loss due to coagulopathy
- iii. H/O petechiae, ecchymosis or lymphadenopathy with fever – Acute Leukaemia
- iv. Recovering from recent surgery

3.2. Chronic

- i. Antenatal or postnatal female
- ii. H/O passage of worms in stool causing chronic blood loss
- iii. H/O Chronic blood loss– Haemorrhoids, Melena, Menorrhagia
- iv. H/O Chronic Alcoholism leading to Vitamin B12 deficiency
- v. H/O Anorexia or any GI complaints leading to malnutrition
- vi. Poor socioeconomic status leading to malnutrition

- vii. H/O chronic diarrhoea, malabsorption
- viii. H/O chronic illness e.g. chronic renal failure, TB or any other causing anaemia of chronic disease
- ix. H/O blood transfusion in the past
- x. H/O similar complaints in the past
- xi. H/O pure vegetarian diet causing Vit. B12 deficiency causing Megaloblastic Anaemia

4. Signs

- i. Pallor– Conjunctiva, mucous membranes, skin
- ii. Nails – Platonychia (flat) or koilonychias (spoon shaped) nails in iron deficiency anaemia (IDA)
- iii. Severe anaemia signs of hyper dynamic circulation e.g. tachycardia, flow murmurs (ejection systolic loudest at apex), cardiomegaly
- iv. Congestive Heart failure – with oedema feet, right hypochondriac tenderness, raised JVP and basal crepitation
- v. Other signs of aetiology may be found e.g. icterus, Lymphadenopathy, stigmata of TB

5. How to Investigate

5.1. Complete blood count

The most important investigation gives maximum information regarding diagnosis.
Mean Cell Volume (MCV) -Normal 76 -96

Low MCV (microcytic anaemia)

- Iron deficiency anaemia (IDA) most common
- Hereditary haemolytic anaemia e.g. Thalassemia

Normal MCV (normocytic anaemia)

- Acute blood loss
- Haemolyses
- Anaemia of chronic disease
- Chronic renal failure
- Pregnancy
- Bone marrow failure e.g. Aplastic anaemia
- Hypothyroidism

High MCV (Macrocytic anaemia)

- Vitamin B12 or folate deficiency (strict vegetarian diet, Pernicious anaemia)
- Alcoholism

- Myelodysplastic syndromes (MDS)
- Drug induced e.g. Phenytoin

5.2. Stool examination

Parasites, occult blood, malabsorption

5.3. Other Baseline investigations

FBS, Creatinine, Liver enzymes, reticulocyte count, LDH

5.4. Iron studies, serum folic / Vitamin B 12 levels

5.5. Bone marrow biopsy if malignancy suspected

6. Treatment

i. IDA –

Oral iron – Ferrous Sulphate (200 mg) thrice a day for 3 months.

Or (300 mg) BD Ferrous fumarate, Ferrous ascorbate can also be used

- Elemental Iron – 100 mg once daily can be increase up to 300 mg OD

Repeat HB at the end of 1 month to confirm response to treatment, if HB is increasing then repeat after 3 months.

- ii. Megaloblastic anaemia-
Oral supplements–Vitamin B12 (7.5 mcg) +Folate (0.75 mg) BD for 1 month.
- iii. Deworming - All Patients Tab Albendazole (400 mg) 1 stat, can be repeated after 2 weeks
- iv. Treat aetiology if possible
- v. Packed cell transfusion if haemoglobin is less than 7

7. When to refer

- Severe anaemia (HB < 4g /dL) requiring blood transfusion
- Signs of Heart failure
- No response to oral supplements of Iron or B12 and folate at the end of 2 months
- Any suspicion of Leukaemia, Lymphoma, MDS or Aplastic anaemia
- No apparent cause found

Bibliography:

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015

Further Reading:

1. India. National Health Portal. Anaemia. National Institute of Health and Family Welfare. [cited 2016 Jul 5]
Available from: <http://www.nhp.gov.in/disease/blood-lymphatic/anaemia>
2. India. National Health Mission. Iron Deficiency Anaemia. Ministry of Health and Family Welfare. [cited 2016 Jul 5]
Available from: <http://nrhm.gov.in/images/pdf/guidelines/nrhm-guidelines/stg/anaemia.pdf>
3. India. Ministry of Health and Family Welfare. Guidelines for control of iron deficiency anaemia. National Iron Plus Initiative. 2013. [cited 2016 Jul 5]
Available from: <https://groups.google.com/forum/#!topic/hrh-pediatrics/JIIMPXkEJmY>



57. HEAT STROKE

1. Definition

Heat Stroke is a syndrome of acute thermoregulatory failure in warm environments characterized by

central nervous system (CNS) depression, core temperatures usually above 40°C (105°F), and typical biochemical and physiologic abnormalities.

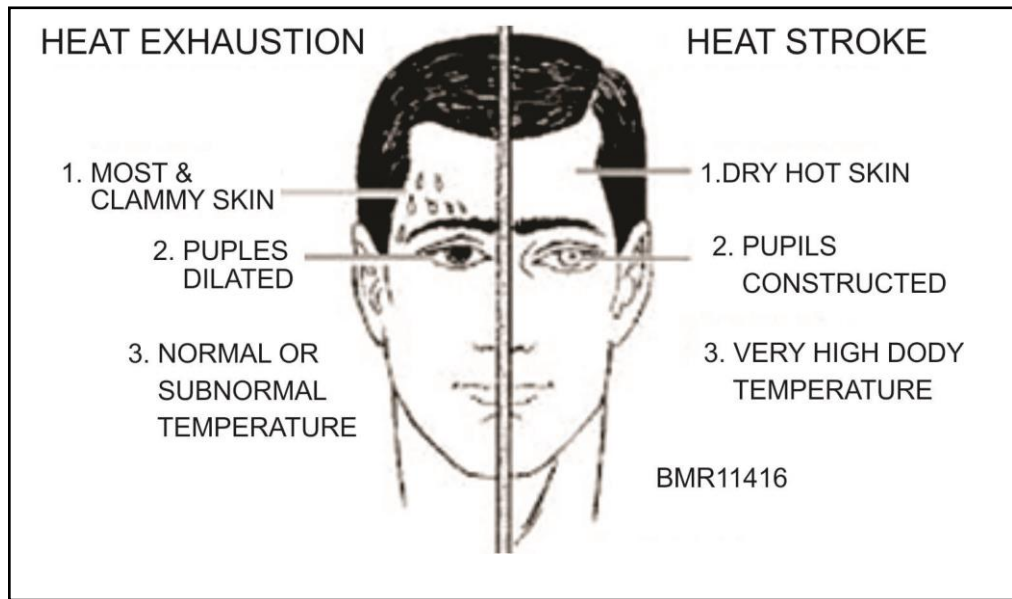


Figure 57.1: Effects of Heat Exhaustion and Stroke

- **Hyperthermia:** Hypothalamic set point is unchanged; does not respond to antipyretics. Distinct from fever (pyrogens change Hypothalamic temperature set point).
- Uncontrolled increase in body temperature that exceeds the body's ability to lose heat due to failed thermoregulation.
- Life-threatening medical emergency.
- Core temperature >40 °C (105 °F) & CNS disturbances in patients with a history of heat exposure.

2. Clinical spectrum of heat illness

- Heat Oedema
- Heat Rash (Miliaria)
- Heat Cramps
- Heat Tetany
- Heat Exhaustion
- Heat Stroke

3. When to Suspect Heat Stroke

- i. In any patient exercising in hot weather or in susceptible individuals; mainly elderly population.
- ii. Coma or profound stupor is nearly always present.
- iii. Diagnostic criteria for Heat Stroke should include
 - a. A core temperature above 40°C
 - b. Severely depressed mental status or Coma on Central Nervous System examination
 - c. Elevated Serum Creatinine and Serum Electrolyte levels (Hyperkalaemia)
 - d. Compatible historical setting.

4. Causes

- i. Increased Heat production- Exercise, Fever, Thyrotoxicosis, Amphetamines, Atropine toxicity.
- ii. Impaired Heat loss- High ambient temperature or humidity, Ineffective voluntary control, Lack

of acclimatization, Dehydration, Cardiovascular diseases.

- iii. Drugs- Anticholinergics, Phenothiazines, Butyrophenones, Thiothixene, Barbiturates, Anti-Parkinson's agent, Alcohol.
- iv. Debilitating conditions- Skin diseases, Cystic fibrosis, Central nervous system lesion, older age.

5. Clinical examination of patient with Heat Stroke

- Anhidrosis often present (but is no longer criteria for diagnosis)
- (Hot, dry skin)
- Altered mental State.
- Often missed in physically inactive patients.
- Baseline body temperature increased (core temperature).

Non-glass medical thermometer.

Rectum –preferred site

Axillary and inguinal sites are unreliable

6. Types

- **Exertional:** Typically seen in healthy young adults who over exert themselves in high ambient (Surrounding) temperatures or in a hot environment to which they are not acclimatized (To adapt). Sudden inability to dissipate / Lose body heat through perspiration (evaporation) or cutaneous vasodilatation (convection), especially after strenuous physical activity in hot weather. (Increased heat production).
- **Non-exertional (classic):** Usually affects elderly and debilitated patients with chronic underlying disease. Result of impaired thermoregulation combined with high ambient temperatures. Often due to impaired sweating.

7. Management

Primary therapy includes cooling and decreasing thermogenesis.

- i. Evaporative cooling methods involve placing a nude patient in a cool room, wetting the skin with water and encouraging evaporation by the use of fans.
- ii. Direct external cooling involves immersing the patient in water. Close monitoring of haemodynamics i.e. Pulse, Blood pressure, respiration and urine output is mandatory.

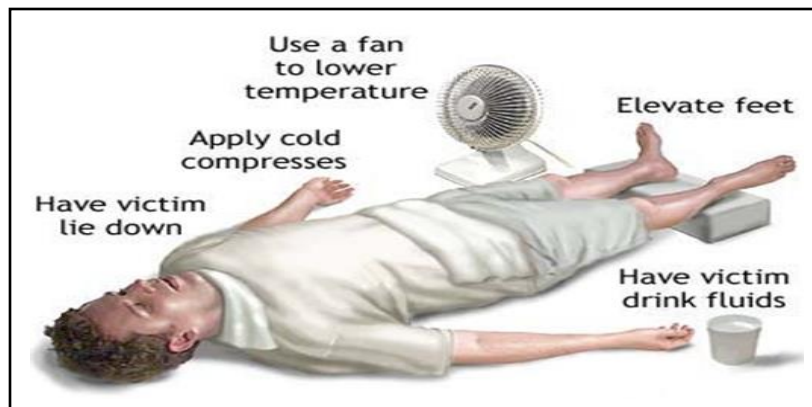


Figure 57.2: Primary therapy of hyperthermia

- Seek medical attention immediately-continue first aid to lower temperature until medical help takes over.
- DO NOT give any medication to lower fever- It will not be effective and may cause further harm.
- DO NOT use an alcohol rub
- It is not advisable to give the victim anything by mouth (even water) until the condition has been stabilized.
- Body cooling methods
- Body immersion in iced water
- Evaporative cooling: Spraying water and the use of fans over the patient facilitates evaporation and convection

- Gastric lavage with cold water / ice, bladder, or peritoneal lavage
- Immersion method of body cooling

Aggressive ice water immersion is gold standard for life threatening heat stroke

- Advantages: Cools patients faster; cooling rate of 0.20°C/min for iced water
- Disadvantages
 - May cause peripheral vasoconstriction (not clinically relevant in RCT)
 - Difficult in patients with reduced level of consciousness
 - For alert - It is uncomfortable and often intolerable
 - Shivering leads to worsening
- Other cooling methods
 - (a) Placement of ice packs in the axillae, groin, and neck
 - Easy method, slower cooling
 - (b) Gastric, peritoneal, and bladder lavage with cold water
 - Used in resistant cases
 - (c) Cooling gloves
- Supportive treatment
 - Treated in ICU settings
 - IV Fluids, treat electrolyte disturbance
 - Mechanical Ventilation

8. Preventing heat-related illness

- Wear loose, lightweight, light-coloured clothing. Light colours will reflect away some of the sun's energy.
- Wear hats or to use an umbrella.
- Drink water- Carry water or juice with you and drink continuously even if you do not feel thirsty.
- Avoid alcohol and caffeine, which dehydrate the body.
- Avoid foods that are high in protein, which increase metabolic heat.
- Stay indoors when possible.
- Take regular breaks when engaged in physical activity on warm days.

- Take time out to find a cool place.

9. Complications

- Acute renal failure
- Rhabdomyolysis
- Liver failure
- Disseminated intravascular coagulation(DIC)
- Acute respiratory distress syndrome (ARDS)
- CNS: Altered Mental state, confusion, delirium
- Seizure, decorticate posture
- Coma and Death
- Dehydration
- Caution – Over hydration
- Electrolyte Imbalance
- Hyponatremia
- Hyperkalemia
- Hypokalemia
- Localized muscle pain on active/passive flexion of limbs
- Urine- Haemoglobinuria, Myoglobinuria

10. Management of complications

- i. Benzodiazepines- Lorazepam (2 to 4 mg I.V. slowly) or Midazolam (2 to 5 mg I.V. slowly) Indicated in patients with agitation & shivering to prevent heat production. Also, given in patients with convulsions.
- ii. Rhabdomyolysis- Common in Exertional heat stroke & in patients with hypotension.

Signs & symptoms

- Dark urine
 - Acute renal failure
 - Treatment
 - I.V. fluids
 - Alkalinisation of urine – sodium Bicarbonate
- iii. Metabolic support- Correcting the electrolyte disturbances like hyponatremia, hypercalcemia, hypocalcaemia, hyperphosphatemia
 - iv. Hepatic injury
 - Monitor liver function tests
 - Avoid hypoglycaemia
 - Early recognition & treatment of DIC
 - Respiratory support

- v. Pulmonary injury- Pulmonary oedema is common complication.
ARDS- Patient should be referred for mechanical Ventilation.
- vi. Renal injury- ARF may develop due to many reasons. These patients may need haemodialysis & so referred to higher centre.

11. When to Refer

- Evaporative and direct external cooling methods fail to reduce the temperature.
- Arrhythmias, metabolic acidosis and cardiogenic failure complicate the early management of hyperthermia crisis.
- Those with evidence of renal failure, disseminated intravascular coagulation or superadded infections.

Bibliography:

1. Munjal YP, Sharma SK, Agrawal AK, Singal RK, Gupta P, Sundar S, et.al. API Textbook of Medicine. 10th edition. JAYPEE Brothers. 2015

Further Reading:

1. India. National Disaster Management Authority. Guidelines for preparation of Action Plan – Prevention and Management of Heat Wave 2016. Government of India. [cited 2016 Jul 5]
Available from: <http://ndma.gov.in/images/guidelines/guidelines-heat-wave.pdf>



58. TUBERCULOSIS (RNTCP)

1. Introduction

Tuberculosis is an infectious disease caused predominantly by *Mycobacterium tuberculosis*. Pulmonary tuberculosis is the most common form of TB (more than 85% of all TB cases), while extra pulmonary tuberculosis can affect almost any organ in the body. Transmission occurs by the airborne spread of infectious droplets and droplet nuclei containing the tubercle bacilli. The source of infection is a person with sputum smear-positive pulmonary TB. Transmission often occurs indoors, where droplets and droplet nuclei can stay in the air for a long time.

2. Epidemiology of Tuberculosis

- TB is a bacterial disease caused by *Mycobacterium tuberculosis*. These organisms are also known as tubercle bacilli or as acid-fast bacilli.
- Transmission of tuberculosis occurs by airborne spread of infectious droplets and droplet nuclei. Source of infection is patient of tuberculosis who spreads tuberculosis bacilli during coughing, sneezing, etc.
- Most common symptom of pulmonary TB is persistent cough for two weeks or more, usually with expectoration. Persistent cough for 2 weeks or more may be accompanied by one or more symptoms such as weight loss, loss of appetite, tiredness, evening rise fever, chest pain, shortness of breath and haemoptysis.
- Incidence of tuberculosis is usually similar in both sexes below 15 years of age, thereafter incidence is higher in males than females and difference is greatest in old people.
- Early detection of sputum positive tuberculosis case and conversion into sputum negative by effective treatment are most important measures for tuberculosis control.

3. Diagnosis of TB

• Identification of Tuberculosis Suspects

The most common symptom of pulmonary TB is persistent cough, usually with expectoration. Persistent cough may be accompanied by other symptoms such as weight loss, tiredness, fever

with evening rise, night sweats, chest pain, shortness of breath, anorexia and haemoptysis.

a. Pulmonary TB Suspects

A pulmonary TB suspect is defined as:

- An individual having cough of 2 weeks or more.
- Contacts of smear-positive TB patients having cough of any duration.
- Suspected/confirmed extra-pulmonary TB having cough of any duration.
- HIV positive client having cough of any duration.

b. Extra-Pulmonary TB Suspects

A patient with extra-pulmonary TB (EP TB) may have general symptoms like weight loss, fever with evening rise and night sweats. Other symptoms depend on the organ affected.

Examples of these symptoms are, swelling of a lymph node in TB lymphadenitis, pain and swelling of a joint in TB arthritis, neck stiffness and disorientation in a case of TB meningitis. Patients with EP TB, who also have cough of any duration, should have sputum samples examined. If the smear result is positive, the patient is classified as pulmonary TB and his/her treatment regimen will be that of a case of smear-positive pulmonary TB.

4. Screening for TB among high risk groups

- a) Contact investigation among the diagnosed smear-positive cases is to be systematically implemented and monitored, and it offers a major opportunity for early case detection.
- b) HIV care centres: Intensified TB case finding should be implemented in all facilities providing HIV care, like ICTCs, ART Centres, Care and support centres etc. Involve NGOs working with HIV programmes in TB case finding activities.
- c) Diabetic patient: regularly screening for TB in all diabetic patients at each visit.
- d) Elderly patients.
- e) Smokers.
- f) Other High risk groups: Malnutrition, patients with silicosis, patients on corticosteroids and other chronic diseases need to be screened for TB regularly.

5. Tools for diagnosis of TB

Following are RNTCP recommended diagnostics tests for TB

- Sputum smear microscopy.
- Histopathology/cytology/radiology.
- Solid/liquid culture and DST for diagnosis TB and Drug Resistant TB.
- PCR based newer rapid diagnostic tools for diagnosis of TB/drug resistant TB e.g. Line probe assay (LPA), CBNAAT.

6. Guidelines for collecting sputum

- The patient is given the sputum container with Laboratory Serial Number written on its side. The patient is instructed to inhale deeply 2–3 times with mouth open, cough out deeply from the chest, open the container and spit out the sputum into it. Sample should be at least 2 ml. If the quantity is less, then the procedure can be repeated. Once adequate quantity of sample is collected, the container should be closed. This is the **first spot specimen (A)**.
- The patient is given a labelled container with instructions to cough out sputum in to the container early in the morning after rinsing the mouth, before breakfast. This is the **early morning specimen (B)**.

The person collecting the sputum specimens should follow the guidelines specified below:

- If the sputum specimens are to be sent immediately to the laboratory, the person should put the container into a special box meant for transport.
- If the sputum specimens are not being sent immediately to the laboratory, these should be stored in a cool and shady place in the referring health facility.
- The person should wash hands thoroughly with soap and water every time when the material is handled.
- Patients should be told to come back to receive the results of sputum examination.

Alternatively, sputum results may be sent to the referring health facility by hand. Laboratory serial number (and/or specimen identification number) should be clearly written on the side of the sputum container.

7. Management of Patient after receiving the sputum results

7.1 Smear positive pulmonary TB.

Patients with at least one sputum positive smear result out of two samples are diagnosed by the physician as having smear-positive pulmonary TB. They are further classified as a new or re-treatment case based on their previous treatment history and appropriate regimen is prescribed.

7.2 Follow up of the sputum negative symptomatic

Patients, who are negative for AFB in both the samples, will be prescribed a course of antibiotics for duration of 10-14 days. In such cases antibiotics such as fluoroquinolones (Ciprofloxacin, Ofloxacin, Levofloxacin, and Moxifloxacin etc.), Clavulanate Macrolides, Rifampicin or Streptomycin, which are active against tuberculosis, are not to be used. Antibiotics of choice include Cotrimoxazole, Amoxicillin, & Doxycycline. Most patients are likely to improve with antibiotics if they are not suffering from TB. If the symptoms persist after a course of broad spectrum antibiotics, repeat sputum smear examination (2 samples) must be done for such patients.

However, if repeat sputum examination turns to be negative, they are subjected for chest x-ray examination. If chest x-ray is suggestive of pulmonary TB, they will be diagnosed as smear negative pulmonary TB and treated accordingly. If chest x-ray is not suggestive of TB, then they should be evaluated for other respiratory diseases.

For patients infected with HIV, antibiotic trial is not indicated and Chest X-ray needs to be taken to avoid delay in diagnosis of smear negative TB.

Patients with suspected EP TB should be referred to a competent medical practitioner / doctor / specialist for expert opinion. Diagnosis of such patients may be made using appropriate diagnostic procedures (such as FNAC/Biopsy/Radiology) as well as clinical methods.

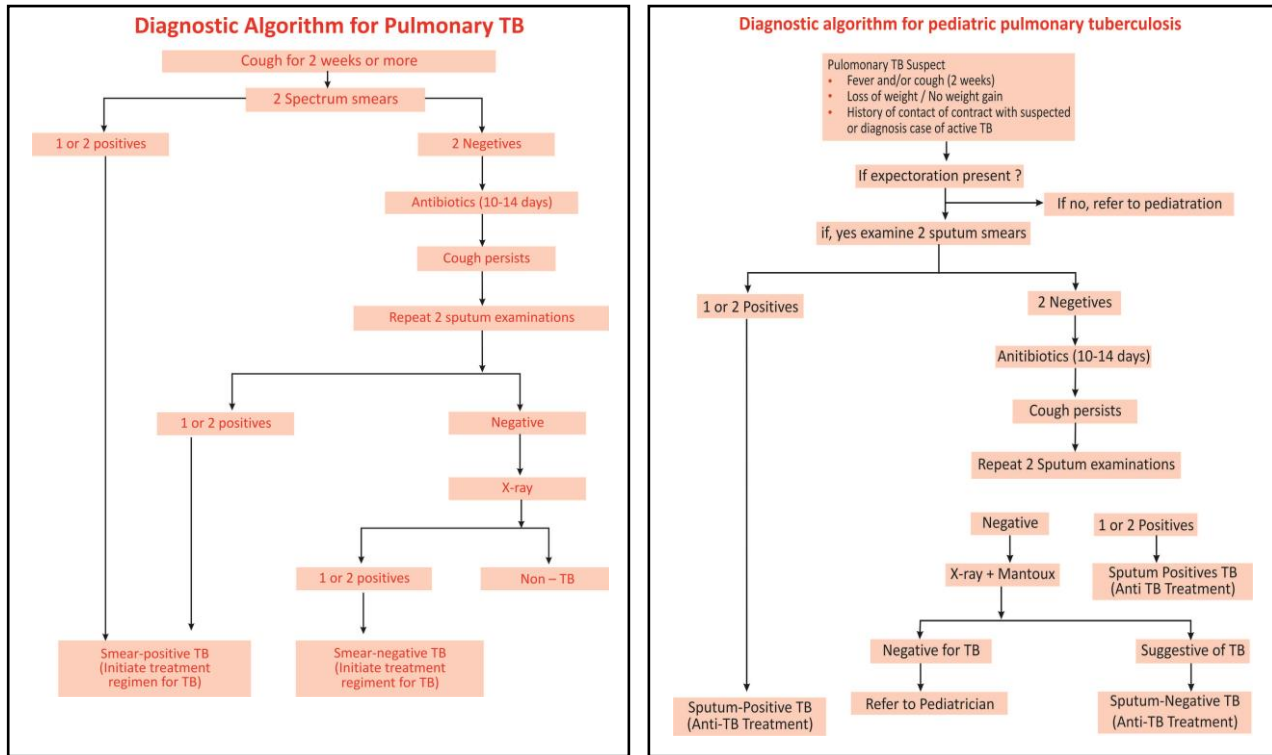
Diagnosis of TB by chest X-ray alone is unreliable because no radiological pattern is pathognomonic of pulmonary TB. Unless the prescribed algorithm is followed, a large number of patients who do not have TB will be falsely diagnosed and treated.

8. Diagnostic Algorithm

When the referring doctor receives the results of sputum examination, and it is decided to put the patient on chemotherapy, health education must be imparted to the patient. The patient is told about TB, how it spreads, precautions to be taken to prevent the spread, importance of directly observed treatment and

its duration, and the need for prompt evaluation of children under six years or contacts with cough of any duration living in the household. The patient should also be informed that his address would be verified by a competent person prior to the start of treatment.

Figure-58.1: Diagnostic Algorithms for Pulmonary Tuberculosis



9. Treatment of TB

MO must undertake detailed clinical examination and history before starting the TB treatment.

The following is required before starting treatment:

- I. History of patient, including history of any previous treatment for TB.
- II. Sputum smear examination results from an approved DMC.
- III. Chest X-ray report if the case warrants radiographic examination.
- IV. Other supporting investigation reports, if any.

V. Culture / Drug Susceptibility Test (DST) report from RNTCP certified laboratory (if available).

9.1. Directly Observed Treatment (DOT)

RNTCP Definitions: Case Definitions, Types of Cases and Treatment outcomes

Table-1: Definitions under RNTCP

DEFINITIONS: THE REVISED NATIONAL TUBERCULOSIS CONTROL PROGRAMME		
Case definitions	Type of cases	Treatment outcomes
<p>Pulmonary Tuberculosis, Smear Positive</p> <p>TB in a patient with atleast one smear positive for AFB out of the two initial sputum smear examination by direct microscopy</p>	<p>New A TB patient who has never had treatment for TB or has taken anti-TB drugs for less than one month</p> <p>Relapse A TB patient who was declared cured or treatment completed by a physician and who reports back to the health facility and is now found to be sputum smear positive</p>	<p>Cured Initially sputum smear positive patient who has completed treatment and had negative sputum smears on two occasions, one of which was at the end of the treatment</p> <p>Treatment completed Initially sputum smear positive patient who has completed treatment with negative smears at end of the intensive phase / two months in the continuation phase, but none at the end of the treatment is declared as treatment completed. or Initially sputum smear negative patient who has received full course of treatment and has not become smear positive at the end of the treatment or Extra pulmonary TB patient who has received full course of treatment and has not become smear positive during or at the end of treatment</p>
<p>Pulmonary Tuberculosis, Smear Negative</p> <p>A patient with symptoms suggestive of TB with two smear examination negative for AFB, with evidence of pulmonary TB by microbiological methods (culture positive or by other approved molecular methods) or Chest X-ray is classified as having smear negative pulmonary tuberculosis</p> <p>Extra Pulmonary Tuberculosis*</p> <p>Tuberculosis in any organ other than lungs (eg. pleura, lymph nodes, intestine, genitor-urinary tract, joint and bones, meninges of the brain etc).</p> <p>The diagnosis should be based on strong clinical evidence with the following investigations</p> <ul style="list-style-type: none"> • Smear / Culture from extrapulmonary sites • Histopathological examination or • Radiological examination or • Biochemical and cytological examination including FNAC 	<p>Transferred in A TB patient who has been received for treatment in a Tuberculosis Unit, after starting treatment in another TB unit where s/he has been registered.</p> <p>Treatment after default A patient, who has received treatment for TB for a month or more from any source and returns for treatment after having defaulted i.e., not taken anti-TB drugs consecutively for two months or more and found to be smear-positive</p> <p>Treatment failure Any TB patient who is smear-positive at 5 months or more after initiation of treatment.</p> <p>Chronic A patient who remains smear-positive after completing regimen for previously treated but not initiated on MDR-TB treatment</p> <p>Others A patient who does not fit into the any of the types mentioned above. The reasons for labeling a patient under this type must be specified in the Treatment card and TB Register#</p>	<p>Died Patient who died during the course of treatment regardless of any cause</p> <p>Failure Any TB patient who is smear positive at five months or more after initiation of the treatment and not put on MDR-TB treatment</p> <p>Defaulted A Patient after treatment initiation has interrupted treatment consecutively for ≥2 months</p> <p>Transferred out A patient who has been transferred to another TU and whose treatment outcome is still not available.</p> <p>Switched over to MDR-TB Treatment A patient who has been diagnosed as having MDR-TB by an RNTCP accredited laboratory, prior to being declared as "Failure", and is placed on the RNTCP MDR-TB treatment regimen</p>

* Pleurisy is classified as extra pulmonary tuberculosis. * A patient diagnosed with both smear positive pulmonary and extra pulmonary TB should be classified as pulmonary TB # Others can come both under new and previously treated.

9.2. Treatment Regimens

For the purpose of treatment regimen to be used, TB patients are classified into two groups, namely, “New” or “Previously Treated”, based on the history of previous treatment.

9.2.1. Regimen for New cases

This regimen is prescribed to all new pulmonary (smear-positive and negative), new extra pulmonary and new ‘others’ TB patients.

The regimen is 2H₃R₃Z₃E₃ / 4 H₃R₃.

Treatment is given in two phases. For “New” patients, the intensive phase consists of isoniazid (H), rifampicin (R), pyrazinamide (Z) and ethambutol (E) given under direct observation thrice a week on

alternate days and lasts for 2 months (8 weeks, 24 doses).

This is followed by the continuation phase, which consists of 4 months (18 weeks; 54 doses) of isoniazid and rifampicin given thrice a week on alternate days with at least the first dose of every week being directly observed. If the sputum smear is positive after 2 months of treatment, the intensive phase of four drugs (H, R, Z and E) are continued for another one month (12 doses), and sputa sent for culture and drug susceptibility testing (C&DST) to an accredited RNTCP C&DST laboratory. Treatment remains continued as per regimen if C&DST report is Rifampicin-sensitive. Sputum is examined after the completion of the extension of intensive phase. Irrespective of the sputum results after this extension of the intensive phase, the 4 months (18 weeks) of the continuation phase is started.

If the sputum smear is positive after 5 or more months of treatment, the patient is declared as a “**Failure**” and is placed on the “**Previously Treated**” treatment regimen afresh. If patient remains smear positive in any follow-up sputum examination, then sputum samples are sent for culture and drug susceptibility testing (C&DST) to a certified RNTCP C&DST laboratory. If the report indicates Rifampicin resistant, then the Cat I regimen is stopped and patient is counselled and referred to District / Drug Resistance TB centre for pre-treatment evaluation and treatment initiation. While treating TB meningitis(TBM) in “New” patients, streptomycin is to be used in place of ethambutol during the intensive phase (H₃R₃Z₃S₃ instead of H₃R₃Z₃E₃). The continuation phase of treatment for patients with TBM or spinal TB is for 7 months. Hence, the total duration of treatment will be for 9 months.

9.2.2. Regimen for Previously Treated cases

This regimen is prescribed for TB patients who have had more than one-month anti-tuberculosis treatment previously. These patients are at a higher risk of having drug resistance. Hence all such patients are also subjected to C&DST for identification of MDRTB. If C&DST report is expected beyond 7 days, then patients are initiated on Cat II regimen with 5 drugs in the intensive phase, and the total duration of treatment is 8 months. **Relapses,**

Treatment after Default, Failures and Others are treated with this regimen.

The regimen is IP: 2S₃H₃R₃Z₃E₃+ 1H₃R₃Z₃E₃
CP: 5 H₃R₃E₃.

Treatment is given in two phases. For “**Previously Treated**” cases, the intensive phase consists of two months (24 doses, 8 weeks) of isoniazid (H), rifampicin (R), pyrazinamide(Z), ethambutol (E) and streptomycin (S), followed by one month (12 doses, 4 weeks) of isoniazid, rifampicin, pyrazinamide and ethambutol, all given under direct observation thrice a week on alternate days. Patient is subjected for follow-up sputum examination at the end of three months. If the sputum smear is positive at the end of 3 months of treatment, the intensive phase drugs (H, R, Z and E) are extended for another one month (12 doses,4 weeks). Irrespective of the sputum results at the end extended intensive phase, 5 months (22 weeks) of continuation phase is started. If the sputum remains positive at the end of the extended intensive phase, sputum is sent to an accredited RNTCP C&DST laboratory for culture and drug susceptibility testing. The continuation phase consists of 5 months (22 weeks; 66 doses) of isoniazid, rifampicin and ethambutol given thrice a week on alternate days, with at least the first dose of every week being directly observed.

Table-2: Treatment regimen under RNTCP

Treatment groups (category)	Type of patient	Regimen ¹	
		Intensive phase (IP)	Continuation phase (CP)
New (Cat I: Red Box)	Sputum smear-positive Sputum smear-negative Extra-pulmonary Others	2H ₃ R ₃ Z ₃ E ₃	4H ₃ R ₃
Previously Treated** /Retreatment case (Cat II: Blue Box)	Smear-positive relapse Smear-positive failure Smear-positive treatment after default Others ²	2H ₃ R ₃ Z ₃ E ₃ S ₃ / 1H ₃ R ₃ Z ₃ E ₃	5H ₃ R ₃ E ₃

i. The number before the letters refers to the number of months of treatment. The subscript after the letters refers to the number of doses per week.

The dosage strengths are as follows: Isoniazid (H) 600 mg, Rifampicin (R) 450 mg, Pyrazinamide (Z) 1500 mg, Ethambutol (E) 1200 mg, Streptomycin (S) 750 mg.

- Patients who weigh 60 kg or more receive additional rifampicin 150 mg.
- Patients who are more than 50 years old receive streptomycin 500 mg. Patients who weigh less than 30 kg, receive drugs as per Paediatric weight band boxes according to body weight.

ii. In rare and exceptional cases, patients who are sputum smear-negative or who have extra-pulmonary

disease can have recurrence or nonresponse. This diagnosis in all such cases should always be made by an MO and should be supported by culture or histological evidence of current, active TB. In these cases, the patient should be typed as ‘Others’ and given treatment regimen for previously treated.

Note: - All doses in Intensive phase and at least first dose in Continuation phase should be directly observed by DOT provider.

9.3. Paediatric TB

Table-3: Paediatric patient wise boxes for new cases according to weight band

Paediatric patient wise boxes for new cases according to weight band			
Weight band	For New cases		Prolongation of IP
	IP	CP	
6-10 Kg	PC 13		PC 15
11-17 Kg	PC 14		PC 16
18-25 Kg	PC 13 + PC 14		PC 15 + PC16
26-30 Kg*	PC 14 x 2		PC 16 x 2

* For children weighing >30 kgs, adult PWB are to be used

Paediatric patient wise boxes for previously treated cases according to weight band			
Weight	For previously treated cases		Prolongation of IP
	IP	CP	
6-10 Kg	(PC 13+ PC15) + (24 vials inj sm*)	(PC 13 + 54 Tab E 200 mg) + (PC 15 without Z)	PC 15
	(PC 14 + PC16) + (24 vials inj sm*)	(PC 14 + 54 E 400 mg) + (PC 16 without Z)	
11-17 Kg	(PC 13 + PC 14 + PC 15 + PC 16) + (24 vials inj sm*)	(PC 13 + PC14 + 54 Tab E 600 mg) + (PC 15+ PC 16 without Z)	PC 15 + PC 16
	(PC 14 x 2)+(PC 16 x 2) + (24 vials inj sm*)	(PC 14 x 2+ 54 Tab E 800 mg) + (2 x PC 16 without Z)	

* Injection streptomycin 15mg/Kg body weight

INH Chemoprophylaxis

All children aged ≤ 6 years in contact with smear-positive pulmonary TB case are screened by MO to rule out TB. If suffering from TB, should be treated appropriately. Children not having TB are to be administered preventive chemotherapy with INH, 10mg/kg body weight for 6 months.

9.4. Monitoring of Treatment

Follow up of patient

Table-4: Follow up of DOTS patients

CAT	Follow up sputum	If smear + at the end of IP		Remarks
		Action	Follow up sputum	
CAT-I	At 2,4,6 months of treatment	Extend IP for another one month	3,5,7 months of treatment	If sputum +ve at 5 months, declare patient as failure and put on CAT-II
CAT-II	At 3,5,8 months of treatment	If +ve at 3 months then extend IP, 4 drugs for one more month	4,6,9 months of treatment	-

Default retrieval action

In spite of counselling and health education at the time of diagnosis and follow up by health workers, some patients remain irregular for treatment. Defaulter retrieval action is action taken to bring back patients for treatments who do not take medicines regularly. DOT provider should take defaulter action. Following actions are suggested under RNTCP for defaulter patients-

- If patient does not come as scheduled during Intensive Phase (IP) then health worker / DOT provider makes home visit immediately same day or next day and brings patient for regular treatment.
- If patient is in Continuation Phase (CP) home visit should be made and patient should be retrieved within a period of a week after missed dose.

In case of community DOT provider / ASHA / Anganwadi, the respective ANM/MPW of that areas should supervise the DOT provider fortnightly for ensuring DOT implementation and assist in ensuring patient adherence, timely follow-up.

9.5. Programmatic Management of Drug Resistant TB (PMDT)

MDR-TB Suspects

The following are the criteria to label a patient as MDR-TB suspect.

- All previously treated pulmonary TB cases at diagnosis.
- Any smear positive follow-up result in new or previously TB cases.
- HIV TB co-infected cases at diagnosis.
- All pulmonary TB cases who are contacts of known MDR TB case

Once the districts will have adequate laboratory capacity for DST, all TB patients will be provided DST at the time of diagnosis. Two fresh sputum samples (Spot and early morning) are to be collected from MDR TB suspects and transported from DMC/PHI to the RNTCP certified C & DST laboratory in cold chain by using transport mechanism (courier/post/human carrier) within 72 hrs.

Definitions

MDR-TB case: A TB patient whose sputum is culture positive for *Mycobacterium tuberculosis* and

is resistant *in-vitro* to isoniazid and rifampicin with or without other anti-tubercular drugs based on DST results from an RNTCP-certified Culture & DST Laboratory.

XDR TB case: An MDR TB case whose recovered *M. tuberculosis* isolate is resistant to at least isoniazid, rifampicin, a fluoroquinolone (Ofloxacin, Levofloxacin, or Moxifloxacin) and a second-line injectable anti-TB drug (Kanamycin, Amikacin, or Capreomycin) at a RNTCP-certified Culture & DST Laboratory.

Diagnosis of MDR-TB

Presently, 3 technologies are available for diagnosis of MDR TB viz. the conventional solid Egg-based Lowenstein-Jensen (LJ) media, the liquid culture (MGIT), and the rapid molecular assays such as Line Probe Assay (LPA) and similar Nucleic Acid Amplification Tests like Xpert MTB/Rif (CBNAAT). Molecular/genotypic tests are much faster than phenotypic tests, as molecular tests don't require growth of the organism, and *M. tuberculosis* is notoriously slow growing. The turnaround time for C-DST results by Solid LJ media is around 84 days, by Liquid Culture (MGIT) is around 42 days, by LPA is around 72 hours and by CBNAAT is around 2 hours. Currently LPA does the DST of INH and Rifampicin and CBNAAT gives the DST of Rifampicin. Liquid culture DST is used to do the DST of second line drugs – Ofloxacin / Kanamycin.

RNTCP MDR/XDR-TB Treatment

As per PMDT guidelines, a specialized centres named “Drug Resistant TB Centres” are been established at tertiary care hospitals like medical college/ TB hospitals/civil hospital for admission of MDR TB patients for pre-treatment evaluation and to initiate treatment. Patients should receive counselling at every level on 1) the nature and duration of treatment, 2) need for regular treatment, 3) possible side effects of these drugs and 4) the consequences of irregular treatment or pre-mature cessation of treatment.

Regimen for MDR-TB

This regimen comprises of 6 drugs - Kanamycin, Levofloxacin, Ethionamide, Pyrazinamide, Ethambutol and Cycloserine during 6-9 months of the Intensive Phase and 4 Drugs-Levofloxacin, Ethionamide, Ethambutol and Cycloserine during the 18 months of the Continuation Phase.

All drugs should be given in a single daily dosage under directly observed treatment (DOT) by a DOT Provider. All patients will receive drugs under direct observation on 6 days of the week. On Sunday, the oral drugs will be administered unsupervised whereas injection Kanamycin will be omitted. If intolerance occurs to the drugs, Ethionamide, Cycloserine and,

PAS may be split into two dosages and the morning dose administered under DOT. The evening dose will be self-administered. The empty blister packs of the self-administered doses will be checked the next morning during DOT. Pyridoxine should be administered to all patients on Regimen for MDR TB. Drugs are provided to the DOT provider in monthly patient wise boxes (PWB). PWBs are prepared at State Drug store as per the five weight bands- <16kg, 16-25Kg, 26-45kg, 46 -70kg and > 70kg.

If patient gains or loses >5 kg weight during treatment and crosses the weight band range, Committee may consider moving the patient in higher or lower weight band in next supply of drugs.

Follow-up investigations during MDR TB treatment

- One specimen for follow up culture at the end of 3,4,5,6,7,9,12,15,18,21,24 months of treatment.
- Monthly weight.
- Chest X-ray during pre-treatment evaluation, end of IP, end of treatment and whenever indicated.
- Physician evaluation every month for 6 months and then every 3months for 2 years.
- Serum Creatinine monthly for first 3 months and then every 3 months till inj. Kanamycin is given.
- TSH during pre-treatment evaluation and when indicated.
- Patients will be considered **culture converted** after having two consecutive negative cultures taken at least one month apart. Based on culture conversion patient is shifted from IP to CP.

Regimen for XDR TB

The **Intensive Phase** will consist of 7 drugs – Capreomycin (Cm), PAS, Moxifloxacin (Mfx), High dose-INH, Clofazimine, Linezolid, and Amoxiclav.

The **Continuation Phase** will consist of 6 drugs – PAS, Moxifloxacin (Mfx), High dose-INH, Clofazimine, Linezolid, and Amoxiclav.

Follow-up monitoring in XDR TB

XDR TB requires more intensive monitoring during follow-up.

- Complete Blood Count with Platelets Count: weekly in first month, then monthly to rule out bone marrow suppression and anaemia as a side effect of Linezolid.
- Kidney Function Test- monthly creatinine and addition of monthly serum electrolytes to the monthly creatinine during the period that Inj Capreomycin is being administered.
- Liver Function Tests: Every month in IP and for every 3 months during CP.
- CXR every 6 months.

No difference to follow-up Culture for patients on regimen for MDR TB and XDR TB.

M/XDR TB Treatment Outcome definitions

Standardised treatment outcome definitions are to be used following treatment of an MDRTB case. These definitions apply to patients with rifampicin resistance (who are taken to be MDR TB for management purposes), and XDR TB cases as well:

- **Cure:** A patient who has completed treatment and has been consistently culture negative (with at least 5 consecutive negative results in the last 12 to 15 months). If one follow-up positive culture is reported during the last three quarters, patient will still be considered cured provided this positive culture is followed by at least 3 consecutive negative cultures, taken at least 30 days apart, provided that there is clinical evidence of improvement.
- **Treatment completed:** A patient who has completed treatment according to guidelines but does not meet the definition for cure or treatment failure due to lack of bacteriological results.
- **Treatment failure:** Treatment will be considered to have failed if two or more of the five cultures recorded in the final 12-15 months are positive, or if any of the final three cultures are positive.
- **Death:** A patient who dies for any reason during the course of M/XDR-TB treatment.
- **Treatment default:** A patient whose treatment was interrupted for two or more Consecutive months for any reasons.
- **Transfer out:** A patient who has been transferred to another reporting unit (DR-TB Centre in this case) and for whom the treatment outcome is not known.

- **Switched to Regimen for XDR TB:** A MDR-TB patient who is found to have XDR-TB.

By an RNTCP certified C-DST laboratory, who subsequently switched to a regimen for XDR TB treatment initiated.

Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. Central Tuberculosis division. Revised National Tuberculosis Control Programme. Ministry of Health and Family Welfare. 2016 [cited 2016 Jul 5]
Available from: <http://tbcindia.nic.in/>
<http://tbcindia.nic.in/index1.php?lang=1&level=2&sublinkid=4573&lid=3177>



59. LEPROSY (NLEP)

1. Cardinal signs of leprosy

i. Hypo-pigmented / Erythematous anaesthetic patch/s.

ii. Thick and/or Tender nerve
Selection of Leprosy case on clinical sign & symptoms.

Figure-59.1: Algorithm for Confirmation

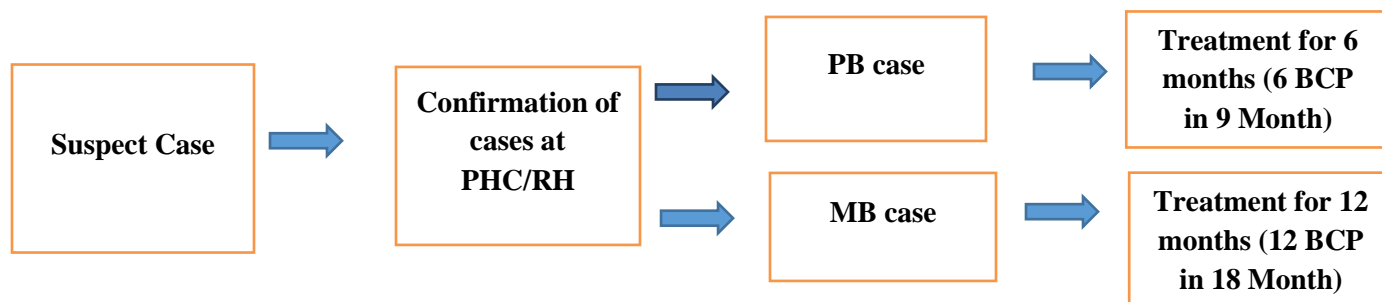


Table-1: Cardinal signs for classification of Leprosy Case

Clinical aspects	PB (Paucibacillary)	MB (Multibacillary)
Skin lesions	1 to 5 skin Patches with definite sensory deficit	6 & 6 + skin Patches with definite sensory deficit
Nerve involvement	And/or one definite thicken or tender peripheral nerve involvement	And/or More than one definite thicken or tender peripheral nerve

If any one or both signs present in suspected case, then it is diagnosed as leprosy.

2. Management

Table-2: Management of Leprosy case as per classification²

Multi Drug Treatment for PB leprosy

Drug	Adult	Children 10-14 years	Children below 10 years	Frequency
Rifampicin	600 mg	450 mg	300 mg	Once a month
Dapsone	100 mg	50 mg	25 mg	
Dapsone	100 mg	50 mg	25 mg daily or 50 mg alternate day	Daily/ alternate day

Multi Drug Treatment for MB leprosy.

Drug	Adult	Children 10-14 years	Children below 10 years	Frequency
Rifampicin	600 mg	450 mg	300 mg	Once a month
Dapsone	100 mg	50 mg	25 mg	
Clofazimine	300 mg	150 mg	100 mg	
Dapsone	100 mg	50 mg	25 mg	Daily
Clofazimine	50 mg (Daily)	50 mg (Alternate day)	50 mg	weekly twice

The appropriate dose for children under 10 years of age can be decided on the basis of body weight.

- Rifampicin: 10 mg per kilogram.
- Clofazimine: 6 mg per kilogram monthly and 1 mg per kilogram per body weight daily.

- Dapsone: 2 mg per kilogram body weight daily.

MDT Multi drug Treatment is safe and well tolerated by most of the patients.

3. Management of Reaction

Table-3: Features of Lepra Reaction

Features	Type – I	Type-II
Skin	Existing lesions suddenly become red, swollen, warm and tender. New lesions may appear Lesions when subsiding, may show scales on surface	Red, painful, tender, subcutaneous (deep) nodules (ENL) appear commonly on face, arms and legs. They appear in groups and subside within a few days.
Nerves	Nerves close to skin become enlarged, tender and painful with loss of nerve function.	Nerves may be affected but not as common as in Type-I reaction
Other organs	Not common	Fever, joint pains fatigue

Treatment of Lepra Reaction (Type-I and Type II)

- Assurance to the patient & family
- Early diagnosis & prompt treatment to prevent deformity.
- Removal of precipitating factor wherever possible.

Tab. Prednisolone

40 mg once a day for first 2 weeks.
30 mg once a day for first 3-4 weeks.
20 mg once a day for first 5-6 weeks.
15 mg once a day for first 7-8 weeks.

10 mg once a day for first 9-10 weeks.

5 mg once a day for first 11-12 weeks.

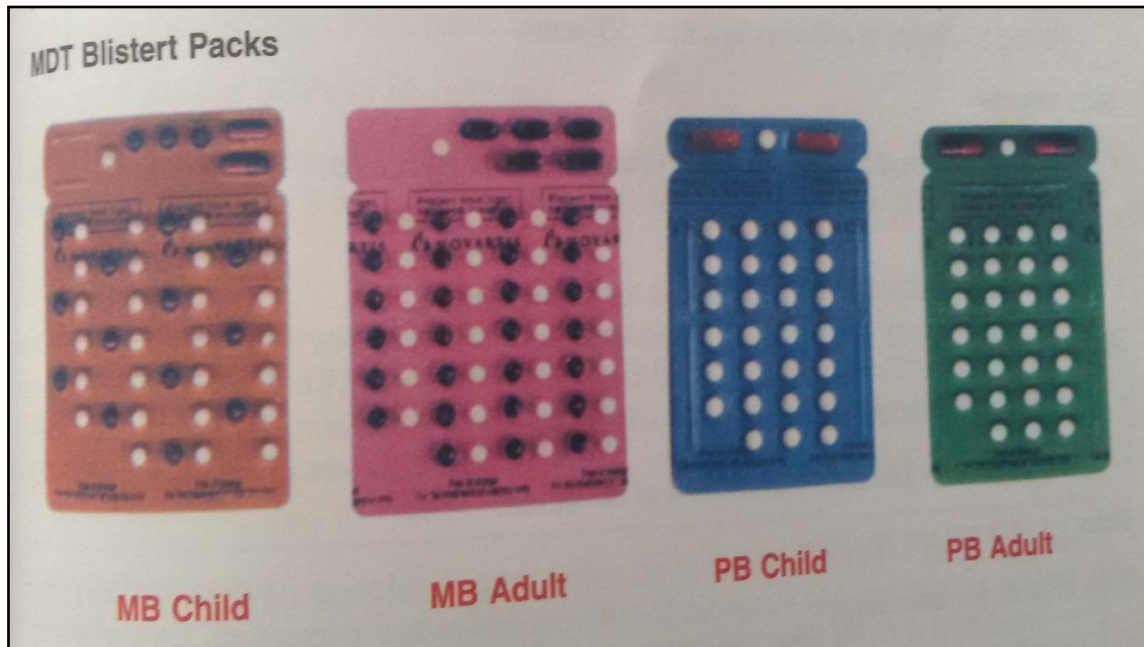
(The total duration should not exceed 12 weeks even in type 2).

Cap Clofazimine 100mg T.D.S. in type 2 reaction for 12 weeks, followed by 100 mg once a day for 12 weeks & 100 mg od for 12-24 weeks.

- Rest: Adequate rest to the affected nerve until symptoms clear, by applying a padded splint or any other suitable material to immobilize the joints near the affected nerve.

- Continuation of Anti Leprosy Treatment only if not completed. appearance of reactions with new development of the disease.
- It has been observed that many medical officers become panicky and start MDT again equating the

Figure-59.2: MDT Blister Packs



Bibliography

1. Casper DL, Fauchi AS, Harrison's Principles of Internal Medicine, 19th edition, McGraw Hill Professional. April 2015
2. Boon NA, Colledge NR, Walker BR. Davidson's Principles and Practice of Medicine, 20th edition, Churchill Livingstone Elsevier

Further Reading:

1. India. Directorate General of Health Services. National Leprosy Eradication Programme. Ministry of Health and Family Welfare. [cited 2016 Jul 5]
Available from: <http://nlep.nic.in/about.html>





Paediatrics



2. Paediatrics

Sr. No.	Contents	Page No.
1	Emergency Management in Paediatrics	166
2	Neonatal Resuscitation Guidelines	177
3	Guidelines for Management of Normal Newborn	179
4	Care of at Risk Neonates	187
5	Care of Sick Neonates	190
6	Management of Low Birth Weight Babies	194
7	Neonatal Sepsis	199
8	Treatment of Respiratory Distress in Newborn	203
9	Meconium Aspiration Syndrome	205
10	Bleeding Neonate	207
11	Jaundice in the Newborn	209
12	Management of Surgical Neonate	213
13	Vitamin A Deficiency	214
14	Rickets	215
15	Management of Children with SAM	217
16	Mumps	220
17	Case Management of Children with Sever Acute Malnutrition (SAM)	222
18	Fever with Rash	227
19	Enteric fever / Typhoid	230
20	Acute Meningoencephalitis	232
21	Tuberculous Meningitis	234
22	Acute Respiratory Infection	236
23	Bronchial Asthma	241
24	Breath holding spell	244
25	Bronchiolitis	245
26	Empyema	247
27	Approach to Fever	249
28	Acute Flaccid Paralysis (AFP)	250
29	Febrile seizures	254

30	Acute Nephritis	256
31	Nephrotic Syndrome	258
32	Congestive Heart Failure (CHF)	260
33	PICA	262
34	Nocturnal Enuresis	263
35	Thrush / Candidiasis	265
36	Congenital Hypothyroidism	266
37	Urinary Tract Infection (UTI)	268

1. Emergency Management in Pediatrics

In children under five years of age, Pneumonia, Diarrhea, Birth asphyxia, Low birth weight and neonatal infections are the most important causes of death. Many children suffer from more than one illness at a time and also many different diseases present with similar symptoms. This chapter describes a sequential process for managing sick young infants and children as soon as they arrive in the facility. Only the key protocols from Integrated Management of Childhood Illness (IMNCI) and Facility Based IMNCI are re-emphasized.

Efforts should be made to maintain euglycemia and euthermia while managing ABCD. Thus Blood sugars should be done for every sick Newborn, Infant and older child.

1. Emergency Management process of the sick child (0- 5 years)

The first step in assessing children referred to a hospital should be triage – The process of rapid screening to decide to which of the following group(s) a sick child belongs:

- First assess every child for emergency signs. Those with emergency signs require immediate emergency treatment.
- If emergency signs are not present, look for priority signs. Those with priority signs should alert you to a patient who is seriously ill and needs immediate assessment and treatment.
- Children with no emergency or priority signs are treated as non-urgent cases.

2. Triage

All sick children are assessed for Airway, Breathing, Circulation, Coma, Convulsions and severe Dehydration (ABCD). Table 1

Table-1: Assessment of Airway and Breathing



3. How to Keep baby warm

- Keep the infant dry and well wrapped.
- Cap, gloves and stockings are helpful to reduce heat loss.
- Keep the room warm (at least 25°C) making sure that there is no heat source directed straight at the newborn.
- Keep the baby under a radiant warmer and re-warm so as to bring the child's temperature to 36.5°C. Pay special attention to avoid chilling the infant during examination or investigation.
- Monitor temperature every half hourly for first 2 hrs and then every 2 hourly.

4. How to Treat Hypoglycemia

- Check for blood glucose in all children presenting with emergency sign, those with severe acute malnutrition and all sick young infants (0-2 months):
- If hypoglycemia detected defined as: < 45 mg/dl for young infants and < 54 mg/dl in older sick children beyond 2 months), give I/V bolus dose of 10% dextrose, in the dose of 2 ml/kg for young infants, and 5 ml/kg for older children.
- If you cannot measure blood glucose, give bolus dose as above. Refer the case to higher center.

5. Airway and Breathing:

5.1. Signs of severe respiratory distress

- **Respiratory rate in**
 - ✓ 0 -2 months > 60,
 - ✓ 2 months – 1 year > 50 &
 - ✓ >1 year – 5 years > 40.
- Severe lower chest in-drawing
- Head nodding
- Grunting
- Apnoeic spells
- Unable to feed due to respiratory distress
- Stridor in a calm child.

5.2. Management of airway in a child with gasping or who has just stopped breathing.

Table-2: Positioning to Improve the Airway when no neck trauma suspected.

Child Conscious	Child Unconscious
<ul style="list-style-type: none"> • Inspect mouth and remove foreign body, if present. • Clear secretions from throat using suction catheter. • Let child assume position of maximal comfort. • Give Oxygen. • Continue with further assessment. 	<ul style="list-style-type: none"> • Open the airway by Head tilt and Chin lift method. • Inspect mouth and remove foreign body, if present • Clear secretions from throat • Check the airway by looking for chest movements, listening for breath sounds and feeling for breath.

5.2.1 Head tilt-chin lift maneuver (Figure 1.1)

The neck is slightly extended and the head is tilted by placing one hand on to the child's forehead. Lift the mandible up and outward by placing the finger tips of other hand under the chin.

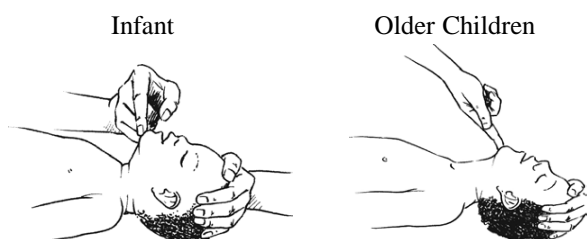


Figure-1.1: Position for opening airway

a) Positioning to improve the Airway when neck trauma suspected

To limit the risk of aggravating a potential cervical spine injury, open the airway with a jaw thrust while you immobilize the cervical spine. It is safe to use in cases of trauma for children of all ages.

b) Neck trauma suspected (possible cervical spine injury)

- Stabilize the neck, as shown in Figure 1.2.
- Inspect mouth and remove foreign body, if present
- Clear secretions from throat by suction catheter
- Check the airway by looking for chest movements, listening for breath sounds, and feeling for breath

5.2.2 Jaw thrust maneuver

The jaw thrust is achieved by placing two or three fingers under the angle of the jaw on both sides, and lifting the jaw upwards and outward. The jaw thrust maneuver is also used to open the airway when bag-mask ventilation is performed.



Figure-1.2: Using Jaw thrust without head tilt

- If after any of these maneuvers the child starts breathing, an oropharyngeal airway should be put and start oxygen.
- If the child is not breathing even after the above maneuvers or spontaneous ventilation is inadequate (as judged by insufficient chest movements and inadequate breath sounds), ventilate with a self-inflating bag and mask

5.3. Ventilation with Bag and mask

- Positioning (Figure 1.3)
- A “sniffing” position (padding under the shoulder to prevent excessive flexion of the neck that occurs when their prominent occiput rests on the surface on which the child lies without hyper-extension of the neck) is usually appropriate for children less than 2 years old.

In correct sniffing position, the opening of the external ear canal should be in line with or in front of (anterior to) the anterior aspect of the shoulder. Extreme hyperextension of the infant neck can produce airway obstruction.

- In children older than 2 years you may need to give padding under the occiput to obtain optimal airway position.



Figure-1.3: Difference in padding for an infant and older child

- Bags and masks should be available in sizes for the entire pediatric range (size 0, 1 and 2).
- It is important for the mask to be the correct size for the child; it must completely cover the mouth and nose without covering the eyes or overlapping the chin. The correct size and position are shown in the Figure 1.4 & 1.5.

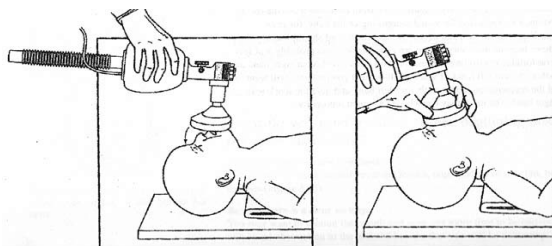


Figure-1.4: Bag and mask ventilation

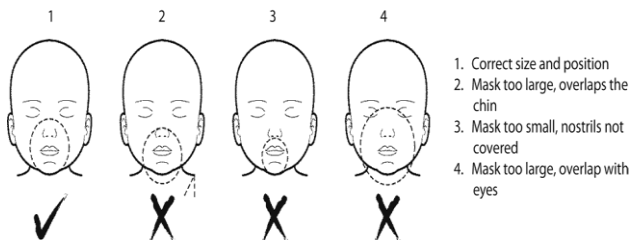


Figure 1.5: Choosing the correct mask size

- Self-inflating bags of minimum volume 450-500ml should be used. Use force and tidal volume just enough to cause the chest to rise visibly.

- Reservoir and oxygen (5-6 L/min) should be connected to the self-inflating bag during resuscitation.
- After two effective ventilations, check the pulse (femoral, brachial or carotid) for no more than ten seconds. If pulse is absent, the second person should start chest compression.

5.3. Chest compressions:

The techniques for chest compression vary for a child under 1 year and those between 1-8 years and are detailed below:

5.3.1 Chest compression in the infant (less than 1 year of age)

There are two techniques for performing chest compression. These techniques are:

- *Thumb technique*, where the 2 thumbs are used to depress the sternum, while the hands encircle the torso and the fingers support the spine

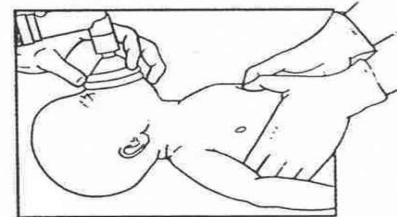


Figure-1.6: Thumb technique

- 2-finger technique, where the tips of the middle finger and either the index finger or ring finger of one hand are used to compress the sternum, while the other hand is used to support the baby's back (unless the baby is on a very firm surface).
- Using either method to give chest compressions, compress the lower half of the sternum but do not compress over the xiphoid. After each compression allow the chest to recoil fully because complete chest re-expansion improves blood flow into the heart.
- "Push hard": push with sufficient force to depress the chest approximately one third to one half the anterior-posterior diameter of the chest.
- "Push fast": push at a rate of approximately 100 compressions per minute.

- Release completely to allow a complete recoil of the chest by completely releasing the pressure but maintaining contact with the compression site.
- Minimize interruptions in chest compressions.
- During cardiopulmonary resuscitation, chest compressions must always be accompanied by positive-pressure ventilation.
- Avoid giving a compression and ventilation simultaneously, because one will decrease the efficacy of the other.
- Therefore, the 2 activities must be coordinated, with one ventilation interposed after every third compression (3 compressions followed by one ventilation), for a total of 30 breaths and 90 compressions per minute

5.3.2 Chest compressions for the child (1 to 8 years of age)

- Place the heel of one hand over the lower half of the sternum. Lift your fingers to avoid pressing on the ribs.

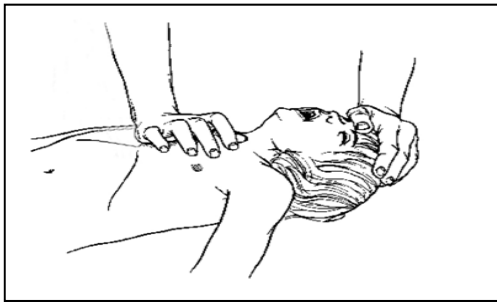


Figure-1.7: Chest compression for the child

- Depress the sternum 1/3 to 1/2 of the depth of the chest. This corresponds to a 1 to 1-1/2 inches.
- Compress at the rate of approximately 100 times per minute.
- The ratio of chest compressions and ventilation should be 15:2, (Fifteen compressions followed by two ventilation).
- Bag and mask ventilation is a very effective way of ventilation if done correctly.
- Setup an intravenous or an intraosseous line for use of any drugs, where needed.

6. Use of Adrenaline

Adrenaline 0.1 ml /kg (1: 10,000) intravenous can be used in a child who does not respond to initial ventilation and chest compressions and his pulses are absent. Two such doses can be used 3-5 minutes apart.

7. Giving Oxygen to a child with respiratory distress

- With a Head box (8-10 L/min) or a Face mask (5-6 L/min).
- Should be allowed to take a comfortable position of his choice and should be given oxygen.
- Continue giving oxygen continuously until the child is able to maintain a SaO₂ > 92% in room air. When the child is stable and improving, take the child off oxygen for a few minutes. If the SaO₂ remains above 92%, discontinue oxygen, but check again 1/2 hour later, and 3 hourly thereafter on the first day off oxygen to ensure the child is stable. Where pulse oximetry is not available, the duration of oxygen therapy is guided by clinical signs, which are less reliable.
- Any child who has been successfully resuscitated or any unconscious child who is breathing and keeping the airway open should be placed in the recovery position. This position helps to reduce the risk of vomit entering the child's lungs. It should only be used in children who have not been subjected to trauma. A child with cyanosis or severe respiratory distress should be allowed to take a comfortable position of his choice.
- Organize Urgent transfer to higher centers in ambulance and ensure doctor accompanies the sick child.

8. Circulation:

- After the Airway has opened, assess if a child has a circulation problem you need to know: The letter C in "ABCD" stands for Circulation, Coma and Convulsions.
- Assess the circulation for signs of shock
The most common cause of shock in children is due to loss of fluid from circulation, either through loss from the body as in severe diarrhoea or when the child is bleeding, or through capillary leak in a disease such as severe Dengue fever. In all cases, it is important to replace this fluid quickly. An intravenous line must be inserted and fluids given rapidly in children with shock without severe malnutrition.

- **Capillary Refill Time:** To assess the circulation, take the child's hand and feet in your own hand. If it feels warm, the child has no circulation problem and you do not need to assess capillary refill or pulse. If the child's hands and feet feel cold, you need to assess the capillary refill.

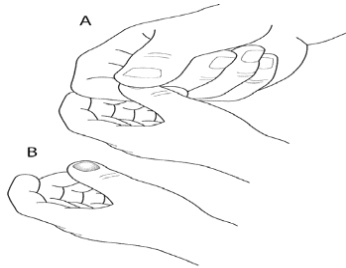


Figure-1.8: Capillary refill time

- Applying pressure to the nail bed for 3 seconds
 - Check the time to the return of the pink colour after releasing the pressure
 - The capillary refill time is the time from release of pressure to complete return of the pink color. If it is more than 3 seconds, the child may be in shock. Lift the limb slightly above heart level to assess arteriolar capillary refill and not venous stasis.
- **Weak & Fast Pulse:** Evaluation of pulses is critical to the assessment of systemic perfusion. The radial pulse should be felt. If it is strong and not obviously fast the pulse is adequate; no further assessment is needed.
 - Weak and fast pulse is defined as:
In Infants - >160/min
In Children - > 140/min.

Thus if the child has cold extremities, a capillary refill time more than 3 seconds, and a fast weak pulse, then he or she is in shock.

9. Treatment of Shock:

- If the child has any bleeding, apply pressure to stop the bleeding. Do not use a tourniquet.
- Give oxygen.
- Give fluids and other treatment for shock.

9.1. Young Infants:

- Fluid bolus of 20ml/kg of normal saline over 20-30 minutes. E.g. in a baby weighing 3 kg, 60 ml of normal saline should be infused over 20-30 minutes. If no or partial improvement (i.e. tachycardia and CRT still prolonged), repeat a bolus of 20 ml/kg of normal saline.
- If the signs of poor perfusion persist despite 2 fluid boluses, start vasopressor support, except in infants with severe dehydration who should be treated as per Plan C of diarrhea management.

9.2. Children above 2 months of age:

The recommended volumes of fluids to treat shock depending on the age/weight of child. If the child has severe malnutrition, you must use of different fluid and a different rate of administration and monitor the child very closely. Therefore, a different regime is used for these children.

10. Coma and Convulsion

- C also represents "Coma and Convulsion".
- Assess the child for coma and convulsion

10.1 Coma

For assessment of the conscious level of a child is, a simple scale (AVPU) is used.

- **A** - Is the child Alert? If not,
- **V** - Is the child responding to Voice? If not,
- **P** - Is the child responding to Pain?
- **U** - The child who is Unresponsive to voice (or being shaken) AND to pain is Unconscious.
- A child who is not alert, but responds to voice, is lethargic.
- An unconscious child may or may not respond to pain.
- *A child with a coma scale of "P" or "U" will receive emergency treatment for coma as described below.*

10.2. Convulsions

- The child must be seen to have a convulsion during the triage process for emergency treatment for convulsion.

- Convulsion are recognized by the sudden loss of consciousness associated with uncontrolled jerky movements of the limbs and/or the face. There is stiffening of the child's arms and legs and uncontrolled movements of the limbs. The child may lose control of the bladder, and is unconscious during and after the convulsion.
- Sometimes, in infants, the jerky movements may be absent, but there may be twitching (abnormal facial movements) and abnormal movements of the eyes, hands or feet.

10.3. Treatment of Coma & Convulsions are similar and is as follows:

10.3.1. Manage the Airway

a) Coma

Managing the airway is done in the same way as treating any child with an airway or breathing problem. This has been discussed earlier. Give oxygen for the emergency setting.

b) Convulsion

To manage the airway of a convulsing child gentle suction of oropharyngeal secretions should be done & child put in recovery position and oxygen started.

Do not try to insert anything in the mouth to keep it open.

10.3.2. Put the child in Recovery Position

Any unconscious child who is breathing and keeping the airway open should be placed in the recovery position.

This position helps to reduce the risk of vomit entering the child's lungs.

- **If neck trauma is not suspected**
 - Turn the child on the side to reduce risk of aspiration
 - Keep the neck slightly extended and stabilize by placing the cheek on one hand
 - Bend one leg to stabilize the body position

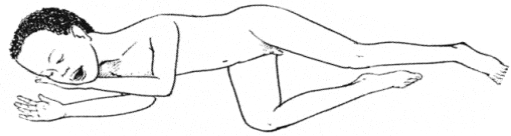


Figure-1.9: Position of Unconscious child (no trauma suspected)

- **If trauma is suspected**

- Stabilize the child while lying on the back.
- When the patient is not being moved, a sandbag placed on each side or a cervical collar can splint the neck.
- Use bottles or rolled towels in case sandbags are not available as shown in the figure 1. 10 below.



Figure 1.10 Position of unconscious child (trauma suspected)

- Use the "log roll" technique to turn the child on the side if the child is vomiting.

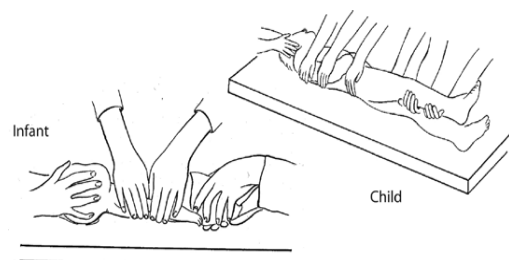


Figure 1.11 "Log roll" technique

- **Insertion of an oropharyngeal (Guedel) airway**
 - The oropharyngeal or Guedel airway can be used in an unconscious patient to improve airway opening.

- It may not be tolerated in a patient who is awake and may induce choking or vomiting.
- Guedel airways come in different sizes (Guedel size 000 to 4). An appropriate sized airway goes from the centre of the teeth (incisors) to the angle of the jaw when laid on the face with the convex side up.

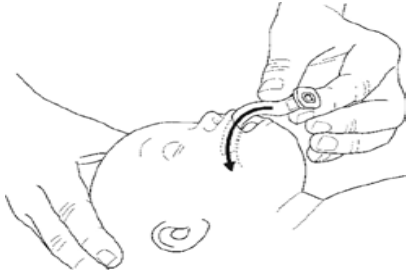


Figure 1.12 Inserting an oropharyngeal airway in an infant: convex side up

- Select an appropriate sized airway
- Position the child to open the airway as described above, taking care not to move the neck if trauma suspected.
- Using a tongue depressor, insert the oropharyngeal airway the convex side up.
- Re-check airway opening.
- Use a different sized airway or reposition if necessary.
- Give oxygen

10.3.3. Management of Convulsions in infant up to 2 weeks of age:

1. Secure IV access
2. If blood sugar < 45 mg/dl, give 2 ml/kg 10% dextrose
3. *If seizures continue:* IV 10% Calcium gluconate 2ml/kg to be diluted in distilled water in proportion 1:1 over 10 minutes to be diluted in while monitoring heart rate (in young infants).
4. *If seizures still continue:* IV Phenobarbitone 20 mg/kg in 5ml of 5% dextrose or saline over 20 min (Table 4)
5. *If no control:* Repeat Phenobarbitone 10 mg/kg till a total of 40 mg/kg
6. *If seizures continue:* Give phenytoin 20 mg/kg in 5 ml of normal saline over 20 min

Table 4: Dose of Phenobarbitone for young infants

Inj. Phenobarbitone intravenous dose (200mg/ml)		
Weight of Infant	Initial dose	Repeat dose
2 kg or less	0.2 ml	0.1 ml
2 to 4 kg	0.3 ml	0.15 ml

- *Caution: Do not use Diazepam for control of convulsions in Neonates < 2 weeks*

10.3.4. Management of Convulsions in infant more than 2 weeks of age:

- Diazepam is the first drug used to stop convulsions (anticonvulsant), if the child is convulsing in front of you. No drug should be given if the convulsion has stopped.
- Diazepam can be given by the rectal or intravenous route.
- Rectal diazepam dose is 0.5mg/kg (0.1ml/kg) by tuberculin syringe or a catheter acts within 2 to 4 minutes. Hold the buttocks together for a few minutes.
- Intravenous dose is 0.25mg/kg (0.05 ml/kg) over 1 minute. Diazepam can affect the child's breathing, so it is important to reassess the airway and breathing regularly.

Table 5: Dose of Diazepam

Age / weight	Diazepam given rectally 10 mg / 2 ml solution	Diazepam given IV 10 mg / 2 ml solution
	Dose 0.1 ml/kg	Dose 0.05 ml/kg
2 weeks to 2 months (<4 kg)	0.3 ml	0.15 ml
2 - <4 months (4 - <6 kg)	0.5 ml	0.25 ml
4 - <12 months (6 - <10 kg)	1.0 ml	0.5 ml
1 - <3 years (10 - <14 kg)	1.25 ml	0.6ml
3 - <5 years (14 - 19 kg)	1.5 ml	0.7ml

- If convulsions do not stop after 10 minutes of second dose of diazepam.
- Inj. Phenytoin can be given intravenously if access has been achieved. 15 - 20 mg/kg Phenytoin is diluted in about 20 ml of saline and given slowly (Not more than 1 mg/kg Phenytoin per minute).
- Alternatively, Phenobarbitone can be used in a dose of 15- 20mg/kg IV (in 20 ml 5% dextrose or saline) or IM.
- At this stage, seek help of a senior or more experienced person, if available

11. If there is high fever:

- Sponge the child with room-temperature water to reduce the fever.
- Do not give oral medication until the convulsion has been controlled (danger of aspiration)

12. Dehydration

The letter D in the ABCD formula stands for Dehydration.

Assess for severe dehydration. To assess if the child is severely dehydrated ask for:

- Lethargic
- Child have sunken eyes
- Skin pinch take longer than 2 seconds to go back

If child has diarrhea with any two of the above signs he is classified to have severe dehydration.



Figure 1.13 Skin pinch

12.1 Treatment of severe dehydration in an emergency setting

12.1.1 Severe dehydration (without severe acute malnutrition)

- Start IV fluid immediately. If the child can drink, give ORS by mouth while the drip is set up. Give 100 ml/kg Ringer's lactate solution (or, if not available, normal saline), divided as follows:

Table 6: IV fluids for severe dehydration

AGE	First give 30 ml/kg in	Then give 70 ml/kg in
Infants (Under 12 months)	1 hour*	5 hours
Children (12 months up to 5 years)	30 minutes*	2 ½ hours

* Repeat once if radial pulse is still very weak or not detectable.

- Reassess the child every 15-30 minutes. If hydration status is not improving, give the IV drip more rapidly.
- Also give ORS (about 5 ml/kg/hour) as soon as the child can drink: usually after 3-4 hours (infants) or 1-2 hours (children).

Table 7: Volume of ORS per hour

Weight	Volume of ORS solution per hour
< 4 kg	15 ml
4 - <6 kg	25 ml
6 - <10 kg	40 ml
10 - <14 kg	60 ml
14 - 19 kg	85 ml

If IV treatment not possible, give ORS 20 ml/kg/hour for 6 hours (120 ml/kg/day) by NG tube

- Reassess an infant after 6 hours and a child after 3 hours. Classify dehydration. Then choose the appropriate plan (A, B, or C) to continue treatment as you have learned in IMNCI / F-IMNCI.
- Give oral antibiotic for cholera if child 2 is years or older.
- If possible, observe the child for at least 6 hours after rehydration to be sure that the mother can maintain hydration by giving the child ORS solution by mouth.

12.1.2. Severe dehydration with severe acute malnutrition

It is difficult to determine dehydration status in a severely malnourished child, as the usual signs of dehydration (Such as lethargy, Sunken eyes) may be present in these children all of the time, whether or not they are dehydrated.

Signs of Dehydration

- Lethargy
- Restless, irritable
- Sunken eyes
- Thirsty
- Skin pinch goes back slowly



Figure 1.14 Severe dehydration with severe acute malnutrition

Treatment of dehydration in the children with SAM without shock

- If the child has had watery diarrhea or vomiting, assume dehydration and give ORS. WHO recommends use of **ReSoMal**, which is not available commercially. Use either WHO-low Osmolarity ORS with potassium supplements (15 ml of potassium chloride syrup added to one litre ORS) as mentioned in step 4 or ReSoMal prepared from WHO-low Osmolarity ORS
- Calculate amount of ORS to give

Table-8: Give ORS as follows in amounts based on the child's weight

How often to give ORS	Amount to give
Every 30 minutes for the first 2 hours	5 ml/kg body weight
Alternate hours for up to 10 hours	5-10 ml/kg
The amount offered in this range should be based on the child's willingness to drink and the amount of ongoing losses in the stool. Starter (diet is given in alternate hours during this period until the child is rehydrated.	

- BEFORE Starting any rehydration treatment:
 - WEIGH the child (The weight should be taken on admission)
 - MARK the edge of the liver and the costal margin on the skin
 - RECORD the respiration rate.
- In addition, the following should be recorded:
 - Pulse rate.
 - The capillary refill time
- In malnourished child look out for:
 - Clinical signs of improvement and
 - Clinical signs of over-hydration.

13. Management of Shock in children with SAM

Give this treatment only if the child has signs of shock and is lethargic or has lost consciousness

13.1 Weight the child Estimate the weight if child cannot be weighed or weight not known

13.2 Give oxygen

13.3 Make sure child is warm

13.4 Insert an IV line and draw blood for emergency laboratory

13.5 Broad spectrum antibiotic should be administered

Immediately to all SAM with septic shock packed RBCs 10ml/kg should be given over 4-6 Hours if HB is less than 4 gm/dl or active bleeding. If there is no improvement with fluid bolus start dopamine at 10µg/kg/min if there is no improvement in next 24-48 hours upgrade antibiotics.

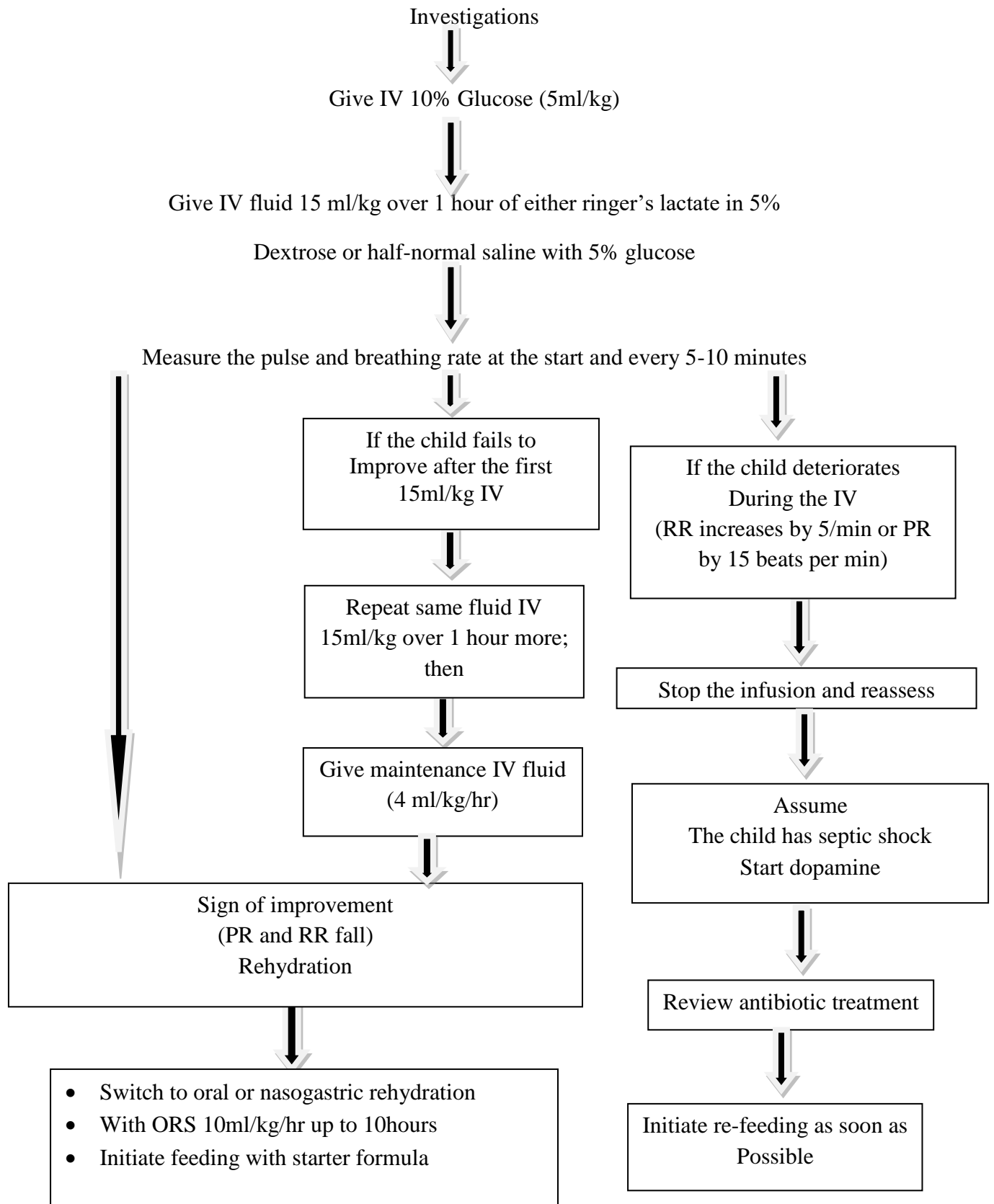


Figure-1.15: The management of shock in child with severe acute malnutrition is given

13.6 How to give Dopamine (By infusion pump)

- Amount of dopamine (mcg) to be added = weight in kgX6
- To convert this dose into amount to ml of dopamine divided by 40 (1 ml of dopamine= 40mg of dopamine)
- Add this amount of dopamine (ml) to make 10 ml of total fluid.
- 0.1 ml/hour of this fluid gives 1 mcg/kg/minute
- To give 10 mcg/kg/minute gives infusion at the rate on 1ml /hr

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
3. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
4. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012
5. India. Navjaat Shishu Suraksha Karykram, Basic Newborn Care and Resuscitation Program Training Manual. Ministry of Health and Family Welfare. [cited 18 July 2016]
Available from: <http://www.nihfw.org/pdf/NCHRC-Publications/NavjaatShishuTrgMan.pdf>

Further reading:

1. Hashim, Muhammad. Et al. Common issues in the care of sick neonates. *American family physician*. 2002; 66 (9): p.1685.
2. Bhimani, Salima. Et al. Streamlining Pediatric Emergency Medicine at a Tertiary-care Hospital of a Low- to Middle-income Country. *Indian pediatrics*. 2015; 52: 1021-1024.



2. NEONATAL RESUSCITATION GUIDELINES

1. What is Neonatal Resuscitation?

- Neonatal resuscitation means to revive or restore life to a baby from the state of asphyxia.
- The following guidelines are intended to neonates undergoing transition from intrauterine to extra uterine life.

2. A rapid assessment of the following 4 characteristics:

- Was the infant born full-term gestation
- Is the amniotic fluid clear of meconium and evidence of infection
- Is the infant crying or breathing, colour of baby?
- Does the infant have good muscle tone?

If the answer is to all 4 of these question is yes, the infant does not need resuscitation and should not be separated from mother.

Observation of breathing, activity, and colour should be ongoing.

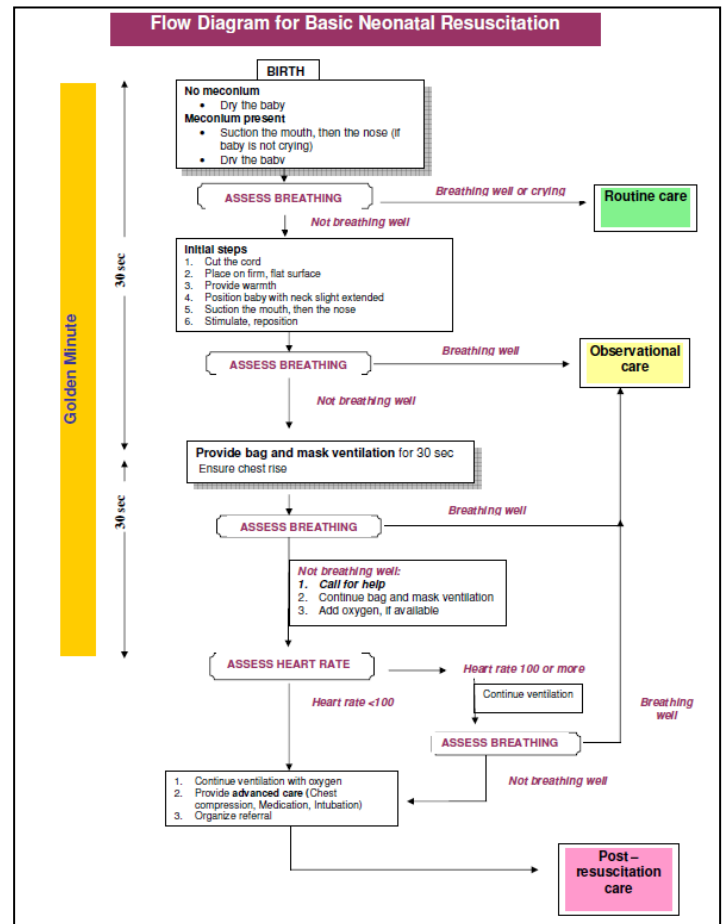
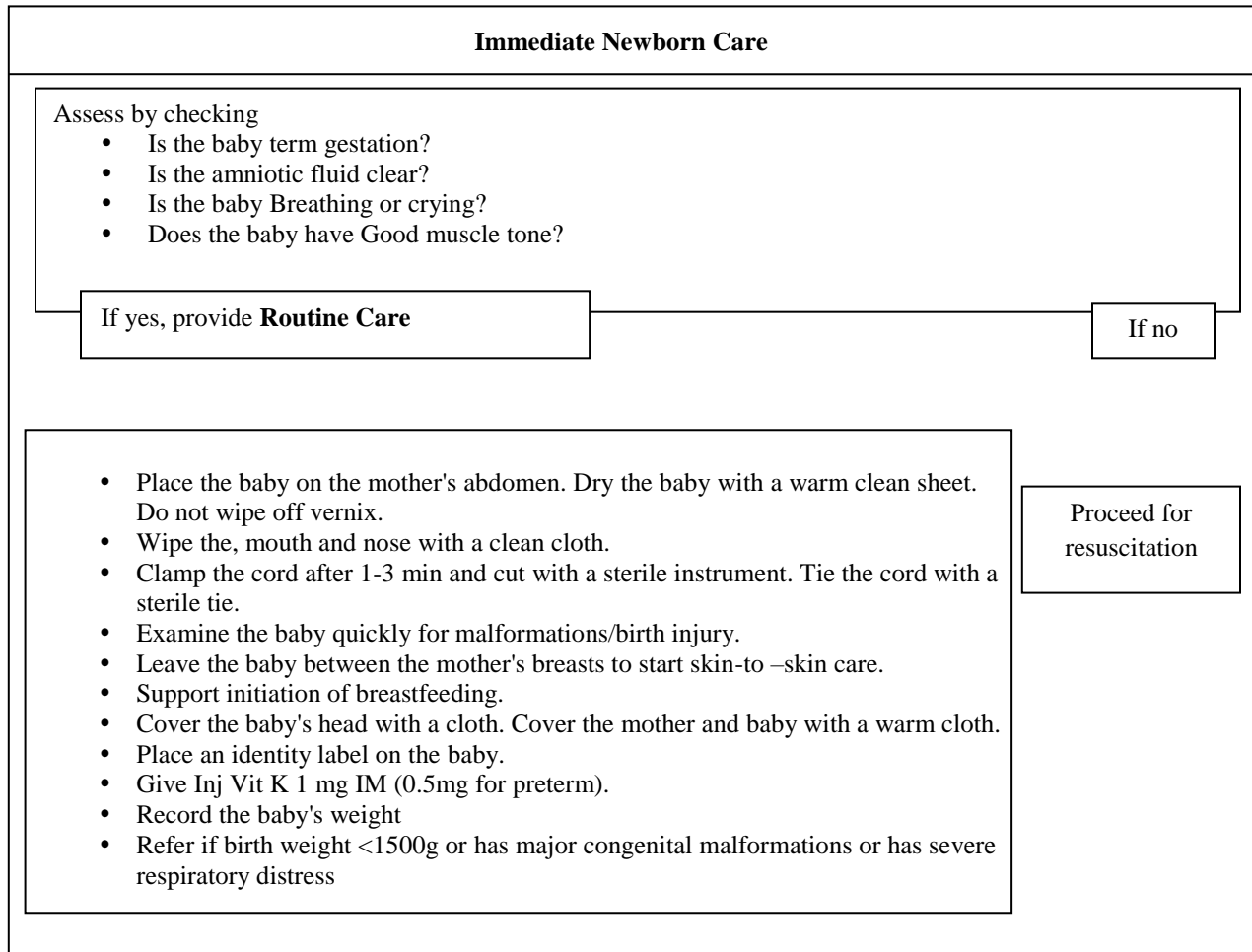


Figure-2.1: Neonatal Resuscitation Protocol

Table-1: Immediate Newborn Care



Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson’s Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
3. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
4. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012
5. India. Navjaat Shishu Suraksha Karykram, Basic Newborn Care and Resuscitation Program Training Manual. Ministry of Health and Family Welfare [cited Jul 18th 2016]
Available from: <http://www.nihfw.org/pdf/NCHRC-Publications/NavjaatShishuTrgMan.pdf>

Further reading:

1. American Heart Association (AHA) Guidelines for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiovascular Care (ECC) of Pediatric and Neonatal Patients: Pediatric Basic Life Support. 2006; volume 117: Issue 5.



3. GUIDELINES FOR MANAGEMENT OF NORMAL NEWBORN

1. Care at Birth:

The four basic needs of ALL newborns at the time of birth and for the first few weeks of life are:

- i. To be warm
- ii. To breathe normally
- iii. To be protected (prevent infection)
- iv. To be fed

2. Newborn Care Corner

This is a space within the delivery room for facilitating immediate care of the newborn. This area is mandatory for all health facilities where deliveries take place.

2.1 Equipment and supplies that should be available in the corner:

2.1.1 Equipment:

- Radiant warmer with bassinet
- Suction equipment
- Weighing machine
- Self-inflating resuscitation bag (500 ml) with masks (size 0,1)
- Oxygen source
- Laryngoscope (straight blade, size 0,1)
- Wall Clock
- Room thermometer

2.1.2 Supplies:

- Clean baby sheets
- Sterile cord ties
- Sterile Gloves
- Sterile blade/scissors
- Mucus extractors
- Suction catheters (10F, 12F)
- Feeding tube (6F, 8F)
- Endotracheal tubes (3, 3.5 mm)
- IV cannula (24G)
- Drugs (Inj. Epinephrine, Normal saline, Inj. Vitamin K1)

3. Immediate Newborn Care of a Normal Newborn at the time of Birth:

- Most babies would require routine care; 5-10% may need assistance to establish adequate breathing and therefore will need resuscitation.
- Deliver the baby onto a warm, clean and dry towel or cloth and keep on mother's abdomen or chest (between the breasts).
- Dry the Baby with a warm clean sheet. Do not wipe off vernix
- Wipe both the eyes separately with sterile swab.
- Clamp and cut the umbilical cord after 1 to 3 minute, if baby is breathing well.
- Assess the baby's breathing while drying.
- Examine the baby quickly for Malformation and birth injury.
- Leave the baby between the mother's breasts to start skin-to-skin care for at least an hour.
- Cover the baby's head with a cap. Cover the mother and baby with a warm cloth.
- Place an identity label/band/tag on the baby and mother
- Encourage mother to initiate breastfeeding (within half an hour of birth).

4. Ensuring 'WARM CHAIN'

4.1. At delivery

- Ensure the delivery room is warm (25-28° C), with no draughts of air
- Dry the baby immediately; remove the wet cloth.
- Put the baby on the mother's abdomen.
- Cover the baby and mother with clean dry cloth
- Keep the baby in skin to skin contact with mother on chest or abdomen
- Postpone bathing/sponging for at least 12 hours or next day

4.2. After delivery

- Keep the baby clothed and wrapped with the head covered.
- Avoid bathing especially in cool weather or for small babies.

- Keep the baby close to the mother
- Use kangaroo care for stable LBW babies and for re-warming stable bigger babies
- Show the mother how to avoid hypothermia, how to recognize it, and how to re-warm a cold baby.
- The mother should aim to ensure that the baby's feet are warm to touch

4.3 If mother and baby's separation is necessary, do the following

- Wrap the baby in a clean dry warm cloth and place under a radiant warmer. If warmer is not available, ensure warmth by wrapping the baby in a clean dry warm cloth and cover with a blanket. Ensure baby's head, hands and feet are covered.
- Re-start Skin-to-skin contact as soon as mother and baby can be roomed-in

5. Prevention of infections: 'CLEAN CHAIN'

5.1. Clean delivery (WHO's six cleans)

- Clean attendant's hands (washed with soap)
- Clean delivery surface
- Clean cord- cutting instrument (i.e. razor, blade)
- Clean string to tie cord
- Clean cloth to cover the baby
- Clean cloth to cover the mother

5.2. After delivery

- All caregivers should wash hands before handling the baby
- Feed only breast milk
- Keep the cord clean and dry; do not apply anything
- Use a clean absorbent cloth as a diaper/napkin
- Wash your hands after changing diaper/napkin. Keep the baby clothed and wrapped with the head covered

5.3. Immediate Cord Care

- Clamp the cord after 1-3 min of delivery and cut with a sterile instrument
- Tie the cord between 2 to 3 cms from the base and cut the remaining cord.
- Observe for oozing blood. If blood oozes, place a second tie between the skin and first tie.
- Do not apply any medication/substance on the stump.
- Leave stump uncovered and dry.

5.4. Care of the eyes

- No routine eye care is required
- Do not instill any medicine in the eyes

6. Weighing the baby

- Weigh all babies before transfer from the delivery room
- Initiate breastfeeding within 1 hour
 - Support mother to initiate breast feeding within the first hour.
 - The baby's first feed of colostrum is very important because it helps to protect against infections.
 - The baby can feed from its mother whether she is lying down or sitting; baby and mother must be comfortable
- Do not give artificial teats or pre-lacteal feeds to the newborn e.g. sugar water or local foods or even water.

7. Examine the baby

7.1 A complete examination should be performed within about 60 minutes after birth

- Count the number of breaths during one minute.
- Observe the movement of the limbs when awake, their position when not moving and their tone.
- Observe the skin color.
- Inspect the following body areas for abnormalities: head, face, mouth and palate, chest, abdomen, genitalia, anus, limbs and skin

7.2 A Healthy baby should have

- Normal temperature, warm to touch, pink with Weight > 2.5 kg
- Breathe easily at 40-60 breathes/minute
- Move arms and legs equally when active and rest with limbs flexed
- Explain to mother the examination findings to allay her concern.
- Document in case record and ask her to inform you, in case any other concerns develop subsequently.

8. Give Vitamin K

- Vitamin K will protect babies from serious bleeding.
- Give Vitamin K by intramuscular (IM) injection 1.0 mg for every newborn (0.5 mg for newborn <1000 gm).
- Encourage mothers to breastfeed their baby during the injection for comfort.

9. Monitoring the Baby

Table-1: Monitoring the baby in the first hour after birth

Parameter	What to look for?
Breathing	Listen for grunting; Look for chest in-drawing and fast breathing
Warmth	Check to see if baby's feet are cold to touch (by using dorsum of your hands)
Color	Evaluate the color of the trunk and extremities

10. Special Situations

10.1 Difficult delivery:

- Encourage Skin-to-skin contact and breastfeeding in difficult deliveries (caesarean section, instrumental and breech delivery)
- Breastfeeding can begin as soon as the mother is comfortable and able to respond to her baby. It does not have to be delayed
- A mother who was given a general anaesthetic agent should begin skin-to-skin contact as soon as she is able to respond to her baby. This may be initiated within one hour of birth

- A mother who has had an epidural (spinal) anesthesia may be able to start skin-to- skin contact very soon after surgery
- These mothers will need additional assistance in positioning and attaching the baby comfortably. Breastfeeding in lying down position may be more comfortable in the first few days

10.2 HIV and newborn care at birth

- Care of a baby born to HIV +ve mother at delivery should be no different from the care already described.
- Standard safety precautions must be followed as with any other delivery.
- Baby can have immediate skin-to-skin contact as any other mother and baby.
- Exclusive breast feeding is the recommended feeding choice in their first 6 months, irrespective of the fact that the mother is on ART early (or) infant is provided with prophylaxis for 6 weeks.
- If mother chooses replacement feeding, prepare formula for the first few feeds. Ensure it is safe, affordable and sustainable for family.
- All other care (including cord care and eye care) remains the same.
- Give oral dose of Nevirapine for six weeks to the neonate as per national policy
- Mother should be counseled regarding the mode of feeding before delivery and dangers of mixed feeding.

11 Postnatal Care of Normal Baby

Ideally, all pregnant women should be counseled regarding the care of the baby during the antenatal period itself. This would help them to be mentally prepared to take care of their babies after birth.

11.1 Postnatal environment

- Ensure that the room is warm without air currents
- Keep mother and baby close together in same room and same bed
- Provide bed nets to sleep

11.2 The key areas of everyday care of a newborn baby include:

11.2.1 Breastfeeding

- Support exclusive breastfeeding on demand day and night.
- Ask the mother to get help if there is a breastfeeding difficulty.
- Assess breastfeeding in every baby before planning for discharge.
- If the mother reports a breastfeeding difficulty, assess breastfeeding and help her with attachment and positioning.
- DO NOT discharge the baby if breastfeeding is not established.

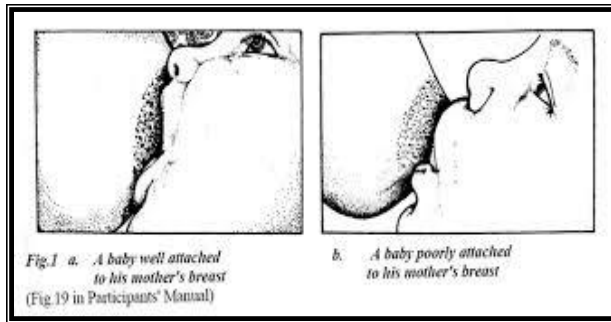


Figure 3.1 Attachment

11.2.2 Keeping the cord healthy

- Wash hands before and after cord care.
- Put NOTHING on the stump.
- Fold nappy (diaper) below the level of the stump.
- Keep cord stump loosely covered with clean clothes.
- If stump is soiled, wash it with clean water and soap.
- Dry it thoroughly with clean cloth.
- Look for signs of infection (daily)
 - Pus discharge from the cord stump
 - Redness around the cord especially if there is swelling
 - High temperature (more than 37.5°C) or other signs of infection
- Explain to the mother that she should seek care if the umbilicus is red or draining pus or blood.

It is important to teach the mothers that the umbilical stump should be left dry; they SHOULD NOT APPLY ANYTHING on the stump.

11.2.3 Ensuring hygiene

- Wash the face, neck, and underarms of the baby daily.
- Do not bathe the baby before 24 hours of age or postponed it till the cord falls to avoid infections
- In case of small babies, bathe only after the baby reaches a weight of 2000g.
- If bath is given ensure room is warm and there is no draught while changing clothes, washing and bathing
- Use warm water for bathing
- Thoroughly dry the baby, dress and cover after bath
- Take extra precautions if the baby is small
- Wash the buttocks when soiled. Dry thoroughly.
- Use cloth diaper on baby's bottom to collect stool. Dispose-off the stool as for woman's pads. Wash hands after disposing.
- Do not apply 'Kajal' on eyes

11.2.4 Looking for danger signs and giving treatment

- It is important that mothers, care givers and health workers are able to recognize the signs and symptoms which indicate that the baby is not well ('DANGER SIGNS').
- Early recognition of the danger signs will help in identifying those babies who need urgent care and treatment.

Danger Signs are -

- Not feeding well
- No movement
- Fast breathing (more than 60 breaths per minute)
- Moderate or severe chest in-drawing
- Jaundice on day 1 or palms or sole stained yellow (any age)
- Abnormal movements.
- Fever (temperature >37.5°C)
- Temperature <35.5°C or not rising after re-warming

12. Ensure immunization

All babies should receive the following vaccines immediately after birth before discharge from the health facility:

- BCG,
- OPV-0
- Hepatitis B (Hep B-0)

13. Criteria for discharge from a health facility

- Feeding well (suckling effectively) at least 8 times in 24 hours
- Baby has passed urine and stool.
- No danger signs
- Mother is confident to take care of baby
- Understands the need for follow up and danger signs when to report early
- For small baby below 2500gms: feeding well and gaining weight adequately

14. Advise on essential care for neonate at discharge

14.1. Feed breast milk

- Breast milk is the best and is the only food baby needs for first six months. Mother needs to breastfeed day and night, at least eight times in 24 hours. Mothers need to take nutritious meals and should drink lots of clean water.
- For a small baby who finds difficult to suckle, express breast milk and collect in a clean cup to feed the baby with a paladai, cup or spoon.

14.2. Keep clean

- Wash your hands with clean water and soap before every feed and after visiting toilet and handling baby's faeces / urine.
- Keep the surroundings clean
- Keep the cord stump clean, do not apply anything on cord

14.3. Keep warm

- Keep the baby well wrapped in a clean dry cloth or blanket (in cold season). Cover baby's head with part of cloth / blanket or put a cap on the

head. Keep the room warm, avoid direct draught of air.

- Keep next to mother for warmth; it promotes lactation and mother-baby bonding.
- Encourage Kangaroo Mother Care (KMC) for Low birth weight babies.

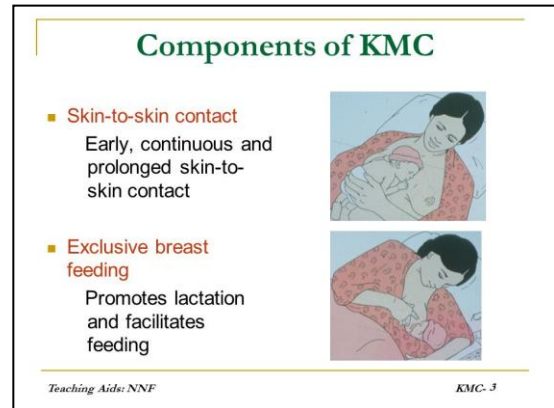


Figure 3.2 Kangaroo Mother Care (KMC)

14.4. Counsel and educate the mother and family

- Build confidence of the family in taking care of baby at home.
- Ensure that the family understands importance of administering prescribed medicines for the whole duration.
- Educate mother when to report for follow up after discharge.
- Educate mother when to report early if there is worsening of condition at any time after discharge.
- Educate mother for signs of well-baby feeds on breast, active behavior, pink extremities and trunk & extremities are warm to touch.
- Ensure baby is gaining weight on follow up.
- Advise for timely immunization.

15. Management of Common Clinical Conditions in Newborns

- There are several phenomena after birth that are normal and mothers only need reassurance. These include:

- Developmental variations & Physiological conditions
- Knowledge of developmental variations, physiological conditions and their evolution in newborns is important for advising and assuring the mother. Mothers observe their babies very carefully and are often worried by minor physical peculiarities, which may be of no consequence and do not warrant any therapy.

15.1. Mastitis neonatorum

- Engorgement of breasts occurs in term babies of both sexes on the third or fourth day and may last for days or even weeks which is due to persistence of maternal hormones for some time.
- Local massage, fomentation and expression of milk should not be done as it may lead to infection. Mother should be reassured that this regresses on its own.

15.2. Vaginal bleeding

- Vaginal bleeding may occur in female babies about three to five days after birth which is because of withdrawal of maternal hormones. The bleeding is mild and lasts for two to four days.
- Additional Vitamin K is unnecessary.

15.3. Muroid vaginal secretions

- Most female babies have a thin, grayish, muroid, vaginal secretion, which should not be mistaken for purulent discharge

15.4. Toxic erythema or Erythema neonatorum:

- This is an erythematous rash with a central pallor appearing on the second or third day in term neonates which begins on the face and spreads down to the trunk and extremities in about 24 hours. This should be differentiated from pustules which need treatment.
- It disappears spontaneously after two to three days without any specific treatment. The exact cause is not known.

- Weight loss of 6-8% (10-12% in preterm infants) in the first few days of life is normal and most infants regain their birth weight by 10-



14 days.

Figure 3.3: Erythema neonatorum

15.5. Bowel disorders.

- No medication should be prescribed for passage of stools after each feed (exaggerated gastrocolic reflex) as this is normal in some babies. From 3rd to 14 days many exclusively breastfeed babies pass loose stools (10-15 times/day) without illness/dehydration. These are transitional stools and require no medication.

15.6. Delayed passage of urine.

- Non-passage of urine by 48 hours after birth may suggest urinary tract anomalies. Such babies need to be investigated. Crying before passing urine is normal.
- Jitteriness is abnormal only when it is excessive or persists even during feeding and then it may suggest hypoglycemia or hypocalcaemia.

15.7. Dehydration fever.

- Transitory moderate fever (up to 38.50C) usually during the second or third day of life in summer months in an active baby, who sucks well, is normal and responds to lowering the environmental temperature.

15.8. Excessive crying.

- Most baby cry when either they are hungry or are having discomfort such as due to full bladder before passing urine, wet napkin, nose block, etc. Excessive inconsolable crying or high-pitched crying is indicative of meningitis or any other painful inflammatory conditions.

15.9. Umbilical granuloma.

- A red flesh-like nodule at the base of umbilical cord can be managed by cautery with Silver Nitrate or application of common salt for 3 to 4 days.

16. Normal phenomena in new born

- **Peeling skin:** Dry skin with peeling and exaggerated transverse sole creases is seen in all post-term and some term babies.
- **Milia:** Yellow –white spots on the nose or face due to retention of sebum, are present in practically all babies and disappear spontaneously.
- **Stork bites (Salmon patches or nevus simplex):** These are discrete, pinkish- gray, sparse, capillary hemangiomas commonly seen at the nape of neck, upper eyelids, forehead and root of the nose. They invariably disappear after a few months.
- **Mongolian blue spots:** In babies of Asiatic origin irregular blue areas of skin pigmentation are often present over the sacral area and buttocks, though extremities and rest of the trunk may also be affected. These spots disappear by the age of six months.



Figure 3.4: Mongolian blue spots

- **Subconjunctival hemorrhage:** Semilunar arcs of sub-conjunctival hemorrhage is a common finding in normal babies. The blood gets reabsorbed after a few days without leaving any pigmentation.

Figure 3.5: Subconjunctival hemorrhage



- **Epstein Pearls:** These are white spots, usually one on either side of the median raphe of the hard palate. Similar lesions may be seen on the prepuce. They are of no significance.
- **Sucking callosities:** The presence of these button like, cornified plaques over the centre of upper lip has no significance.
- **Tongue Tie:** It may be in the form of a fibrous frenulum with a notch at the tip of the tongue. This does not interfere with sucking or later speech development.
- **Non retractable prepuce:** The prepuce is normally non-retractable in all male newborn babies and should not be diagnosed as phimosis. The urethral opening is often pinpoint and is visualized with difficulty. The mother should be advised against forcibly retracting the foreskin.
- **Hymenal tags:** Mucosal tags at the margin of hymen are seen in two-third of female infants.
- **Umbilical hernia:** Umbilical hernia may manifest after the age of two weeks or later. Most of these disappear spontaneously by one or two years of age.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
3. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
4. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012

Further reading:

1. Vora S, Chandran S, Rajadurai VS, Hussain K. Hyperinsulinemic Hypoglycemia in Infancy: Current Concepts in Diagnosis and Management. Indian Pediatr. 2015 Dec; 52 (12): 1051-9.
2. Edmond KM et al. Delayed Breast feeding Initiation Increases Risk of Neonatal Mortality. Pediatrics. 2006 Mar; 117 (3): e380-6.
3. Government of India, Ministry of Health and Family Welfare. INAP: India Newborn Action Plan. September 2014. [Last Accessed on 9 October 2016].
Available from: http://www.newbornwhocc.org/INAP_Final.pdf



4. CARE OF AT-RISK NEONATES

1. An 'at-risk' neonate has one or more of the following features:

- Weight 1500-2499g
- Temperature (axillary) 36.0°C-36.4°C
- Babies with moderate or severe hypothermia who respond to warming
- Cried late (>1min) but within 5 minutes of birth
- Sucking poor, but not absent sucking reflex
- Depressed sensorium, but is arousable
- Respiratory rate of over 60 per minute, but no chest retractions
- Jaundice present, but no staining of palms/soles
- Presence of any one of the following:
 - Diarrhea or vomiting or abdominal distension
 - Umbilicus draining pus or pustules on skin
 - Fever

The care of 'at-risk' neonate should be initiated at the health facility itself under direct supervision. After

initial improvement, further care can be provided at home

2. The care of at-risk babies is outlined below:

2.1. Warmth:

Grading of hypothermia

- Normal temperature : 35.5-36.5°C
- Cold stress : 36.4-36.0°C
- Moderate hypothermia : 35.5-32°C
- Severe hypothermia : <32°C

Table 1: The steps are dependent up on the current temperature of the baby:

Temperature	Management
Normal temperature	<ul style="list-style-type: none"> - Prevent hypothermia - Wrap the baby in layers of clothing - Cover the head and limbs - Place the baby in direct contact with mother - In winter months, the room may have to be warmed with heater, etc
Cold stress (temperature 36.0°C and 36.4°C)	<ul style="list-style-type: none"> - Treat hypothermia - Wrap the baby with extra layers of clothing - Cover the head and limbs - Place the baby in close contact with the mother, preferably skin-to skin - In winter months, heat the room with a heater , etc.
Hypothermia (Temperature <36.0°C)	<ul style="list-style-type: none"> - Requires immediate exposure to a radiant heat source (such as radiant warmer) or heater - Other measures same as for cold stress

2.2. Stabilization

Most of these babies do not require stabilization other than prevention for hypothermia as above. If there is occasional apnea, physical stimulation may be provided.

2.3. Feeds

The baby is started on direct breast feeding. If not sucking well, she is provided expressed breast milk by spoon or paladai. Occasionally, expressed breast milk may have to be given by gavage feeding.

2.4. Specific therapy

Table 2: Some simple conditions can be readily treated at the health facility and later at home.

Condition	Treatment
Umbilical redness And Discharge	- Local application of 1% Gentian Violet - Syrup Amoxicillin 1.25ml TDS x7days
Skin pustules	Local application of 1% gentian violet. Syrup Amoxicillin 1.25ml TDS x7days
Pneumonia (Respiratory rate >60/min, no chest retractions)	Syrup Amoxicillin 1.25ml TDSx7days)

2.5. Monitoring

The following signs should be monitored every two hours:

- Signs to be monitored
- Temperature
- Convulsion
- Sucking
- Bleeding
- Sensorium
- Diarrhea
- Respiration
- Vomiting
- Apnea
- Abdominal distension
- Cyanosis

2.6. Re-evaluation

After stabilization and/or specific therapy, the baby has to be re-evaluated for improvement. The two cardinal signs of improvement are:

- The temperature will become normal (36.5°C - 37.5°C) and
- The baby will accept feeds well.

2.7. Communication

Communication with the family, especially the mother is very important during the management of at-risk and sick neonates.

Communication with the family

1. Reassure the mother and family.
2. Prepare a note regarding baby's condition and care.
3. If baby improves and is to be sent home, explain care of the baby at home.
4. If baby does not improve or worsens, explain the need for referral and care during transport.

3. FOLLOW - UP

- Advice about follow-up visits
- Keep the baby warm
- Provide exclusive breast milk feeding
- Continue the prescribed treatment
- Observe progress of baby
- Counsel and educate the mother and family
- Follow-up:
A home visit by the health worker (ASHA) one day after evaluation at hospital is desirable. Thereafter the baby should be seen within 24 hours and then on 3rd, 7th, 14th, 21st, 28th and 42nd day. Child has to visit every 3rd month up to 1yr.

Bibliography:

1. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
2. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012

Further reading:

1. Hashim, Muhammad. Et al. Common issues in the care of sick neonates. *American family physician*. 2002; 66 (9): p.1685.



5. CARE OF SICK NEONATES

1. A sick neonate is the one who has any of the following features:

- Weight <1500 g
- Temperature <36°C despite warming for one hour
- Cried after 5 minutes of birth
- Absent sucking
- Not arousable
- Respiratory rate more than 60/min with chest retractions
- Apnea or gasping respiration
- Central cyanosis
- Jaundice staining palms/soles
- Convulsions
- Bleeding
- Major malformation
- Presence of two of the following
 - Diarrhea or vomiting or abdominal distension
 - Umbilicus draining pus
 - Multiple skin pustules
 - Fever

Also remember that if an 'at-risk' neonate does not improve while being observed under your care, he is also considered a sick neonate.

2. Grading and management of hypothermia

Baby who is cold to touch both centrally and peripherally or temperature is less than 35.5°C

2.1. Management of hypothermia

- Record the actual body temperature
- Re-warm a hypothermic baby as quickly as possible:
- Severe hypothermia – Radiant warmer
- Mild to moderate hypothermia- Kangaroo mother care or Radiant warmer
- If hypothermia still persists despite taking above measures, infection should be suspected

2.2. Management of severe hypothermia

- Keep under radiant warmer
- Reduce further heat loss
- Infuse IV 10% Dextrose @ 60ml/kg/day
- Inject Vitamin K 1 mg and 0.5 mg for baby weighing less than 1000gm IM.
- Provide oxygen
- Consider and assess for sepsis

3. Prevent hypothermia: warm chain

Baby must be kept warm at all times right from birth. The "warm chain" is a set of 10 interlinked procedures carried out at birth and later

- Warm delivery room (>25°C)
- Warm resuscitation
- Immediate drying
- Skin-to-skin contact between baby and the mother
- Breast feeding
- Bathing and weighing postponed
- Appropriate clothing and bedding
- Mother and baby together
- Warm transportation
- Training/ awareness raising of health-care provider

4. Expression of breast milk

Breast milk expression is required for optimal feeding of newborns for preterm, low birth weight and sick newborns that cannot breastfeed but can tolerate assisted feeding.

4.1. Teach the mother to:

- Wash hands with soap and water before expression. Hold, handle or cuddle the baby
- Sit comfortably and hold the clean container near the breast
- Put thumb and index finger on the breast at the rim of the areola opposite each other. Support the breast with other three fingers.

- Press thumb and index finger slightly inward towards the chest wall
- Press the breast between the fore-finger and thumb. Press and release, press and release. This should not hurt
- Press the areola in the same way from the sides, this ensures that milk is expressed from all segments of the breast
- Avoid rubbing or sliding fingers along the skin
- Express one breast for at least 3-5 minutes until the flow slows; then express the other side; and then repeat on both sides
- To express breast milk adequately it may take 20-30 minutes

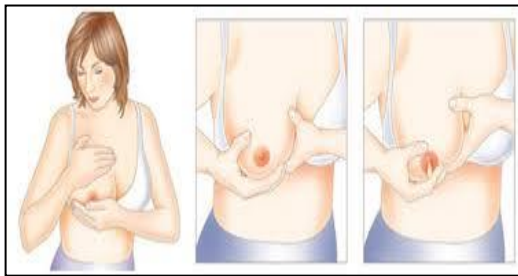


Figure 5.1: Expression of breast milk

- Cover the container of EBM with a clean cloth or a lid
- EBM can be kept at room temperature for 8 hours and in the refrigerator for 24 hours
- EBM stays in good condition longer than animal milk. Do not boil the EBM. For warming, place the container in a bowl of warm water
- Before feeding, gently shake the container or use a stirrer to recombine the separated fat globules with the rest of the milk
- Feed with cup or spoon or paladai, never feed with bottle

5. Assisted feeding of low birth weight neonates

5.1. Newborns that require assisted feeding:

- Preterm <34 weeks or birth weight <1800 g
- Babies having mild respiratory distress
- Babies with inability to feed at breast or by Katori-spoon/paladai
- Oro-facial defects/malformation (Cleft lip or palate)

4.2. Storing expressed breast milk (EBM)

Table 1: Guidelines for the modes of providing fluids and feeding

Birth weight (grams)	< 1200	1200-1800	>1800
Gestation (weeks)	< 30	30-34	>34
Initial feeding	Intravenous fluids try gavage feeds, if not sick	Gavage, try Katori-spoon if not sick	Breastfeeding, if unsatisfactory, give Katori-spoon feeds
After 1-3 days	Gavage	Katori-spoon	Breastfeeding
Later (1-3 weeks)	Katori-spoon	Breastfeeding	Breastfeeding
After some more time (4-6 weeks)	Breastfeeding	Breastfeeding	Breastfeeding

5.2. Mode for providing fluids and feeds

- Breast milk is the ideal feed for low birth weight babies.
- Those unable to feed directly on the breast can be fed Expressed Breast Milk (EBM) by gavage OR Katori-spoon or paladai.

5.3. Techniques of assisted feeding:

5.3.1 Gavage feeding

- Place an oro-gastric feeding catheter of size 5-6 Fr after measuring the correct insertion length from ala of nose to tragus and from tragus to midway between xiphisternum and umbilicus

- Check correct placement by pushing in air with 10 ml syringe and listening with stethoscope over upper abdomen
- Attach 10 ml syringe (without plunger) at the outer end of the tube, pour measured amount of milk and allow milk to trickle by gravity. Close outer end of tube after feeding

Place baby in left lateral position for 15 to 20 minutes to avoid regurgitation

- Leave oro-gastric tube in situ
- Pinch the oro-gastric tube during withdrawal
- Measure pre-feed abdominal girth just above the umbilical stump. Do not attempt pre-feed aspirates
- Evaluate baby for ileus, if abdominal girth increases by >2cm from baseline
- Routine refeeding gastric aspirates are not recommended



Figure 5.2: Gavage Feeding

5.3.2 Katori-spoon/paladai feeds

- Place the baby in a semi-upright posture
- Place the milk filled spoon at the corner of mouth



Figure 5.3 Paladai feeding

- Allow milk flow into baby's mouth slowly, allowing him to actively swallow, avoiding the spill
- Repeat process till required amount has been fed
- Try gently stimulation if baby does not actively accept and swallow the feed
- If unsuccessful, switch back to gavage feeds.

6. Intravenous fluid therapy for newborn:

6.1 Criteria for starting intravenous fluids among newborns

- Neonates with lethargy and refusal to feed
- Moderate to severe breathing difficulty
- Babies with shock
- Babies with severe asphyxia
- Abdominal distension with bilious or blood stained vomiting

6.2. Choice of intravenous fluids

- Determine required volume of fluid as per birth weight and age (Table 2)
- Use 10% Dextrose for initial 48 hours of life
- After 48 hours, if baby is passing urine, use commercially available IV fluids such as Isolyte-P

Table 2: Fluid requirements of newborns

Day of life	Amount of fluids required (ml/kg/day)	
	Birth weight > 1500 g	Birth weight <1500 g
1	60	80
2	75	95
3	90	110
4	105	125
5	120	140
6	135	150
Day 7 onwards	150	150

- If the premixed solution is not available or baby requires higher GIR (Glucose infusion rates)
 - Take normal saline (NS) 20 ml/kg body weight

- Add remaining fluid volume as 10% Dextrose
- Add 1 ml KCL/100 ml of prepared fluid.

6.3. Administration of IV fluid

- Use micro-drip infusion set (where 1 ml = 60 micro drops)
- In this device, ml of fluid per hour is equal to number of micro-drops per minute
e.g. 6ml/hr = 6 micro-drops/minute
- Calculate rate of administration, monitor to ensure that micro-dropper delivers required rate
- Change the IV infusion set and fluid bag every 24 hours
- Before infusing IV fluid, carefully check
- Expiry date of the fluid
- Seal of the infusion bottle or bag
- Fluid is clear and free from any visible particles

6.4. Monitoring of babies receiving IV fluid:

- Inspect infusion site every hour for redness and swelling
- If redness and/or swelling is present, stop infusion, remove cannula, and establish a new IV line in a different vein
- Check the volume of fluid infused, compare to the prescribed volume and record all findings
- Measure blood glucose every nursing shift, i.e. 6-8 hours
- If the blood glucose is less than 45 mg/dl, treat for low blood glucose

- If the blood glucose is more than 150 mg/dl on two consecutive reading: Change to 5% Dextrose solution-measure blood glucose again in three hours
- Weigh the baby daily. If the daily weight loss is more than 5% increase the total volume of fluid by 10 ml/kg body weight for one day
- If there is no weight loss in the initial 3 days of life, do not give the daily increment
- If there is excessive weight gain (3-5%) decrease the fluid intake by 15-20 ml/kg/day
- Check urine output: Normally a baby passes urine 5-6 ml/kg/day

7. Management of hypoglycemia

- Hypoglycemia in newborns is defined as blood glucose levels less than 45 mg/dl
- Establish an IV line. Infuse a bolus of 2 ml/kg body weight of 10% dextrose slowly over 5 min
- If baby had convulsions, give bolus of 4-5 ml/kg of 10% dextrose
- If an IV line is not available, administer 2 ml/kg body weight of 10% dextrose by gastric tube
- Start infusion of dextrose at the daily maintenance volume to provide at the rate of 6 mg/kg/min
- Measure blood glucose after 30 min and then every four to 6 hrs
- If blood glucose < 25 mg/dl:
 - Repeat bolus of dextrose as above
 - Increase to infusion rate of 8 mg/kg/min

Bibliography:

1. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
3. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012

Further reading:

1. Rozance PJ, Hay WW. Hypoglycemia in newborn infants: Features associated with adverse outcomes. *Biol Neonate*. 2006;90(2):74-86.
2. Kumar V, Shearer JC, Kumar A, Darmstadt GL. Neonatal hypothermia in low resource settings: a review. *J Perinatol*. 2009 Jun;29(6):401-12.



6. Management of Low Birth Weight Babies

- Nearly 75 percent neonatal deaths and 50 percent infant deaths occur among the low birth weight neonates.
- Even after recovering from neonatal complications, some LBW babies may remain more prone to malnutrition, recurrent infections, and neurodevelopmental handicaps.
- Low birth weight, therefore, is a key risk factor of adverse outcome in early life.

1. Low Birth Weight

- Low birth weight (LBW) baby is the one who weighs less than 2500 g at birth.
- Low birth weight may result from either prematurity (gestational age <37 weeks) or intrauterine growth retardation (IUGR), which is also called small – for – date baby (SFD).

1.1 Preterm babies:

Preterm babies have distinct physical features that help in their recognition These are:

1.1.1 Skin: The skin of preterm neonate is thin, transparent and gelatinous whereas that of a term neonate is thick gelatinous and keratinized.

1.1.2 Hair: The back of the preterm babies has abundant growth of fine hair called lanugo.



Figure 6.1: Preterm vs term baby- Hair

1.1.3 Ear Cartilage: The external ear or the pinna is soft and devoid of cartilage in preterm neonates and hence, it does not recoil back promptly on being folded. In a term baby, there is instant recoil.

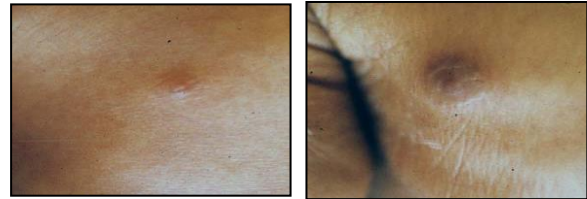


Figure 6.2: Preterm vs term baby- Breast nodule

1.1.4 Breast Nodule: Breast nodule measures less than 5mm in preterm neonates and 5 mm or more in term babies.



Figure 6.3: Preterm vs term baby- Sole Creases

1.1.5 Sole Creases: Anterior one third of the sole reveals a deep transverse skin crease in preterm neonates and in term neonates they are present over the anterior two-thirds.

1.1.6 External Genitalia: In males, the scrotum does not have rugae and testes are not descended into the scrotum. In female infants, the labia are widely separated, not covering the labia minora, resulting in the prominent appearance of the clitoris.

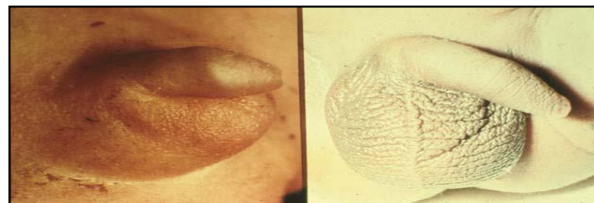
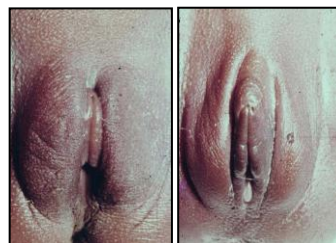


Figure 6.4: Preterm vs term baby- External Genitalia



1.2 Small for date babies/ Small for gestational age (SFD/SGA)

Small-for-dates neonates have an emaciated look and loose folds of skin because of lack of subcutaneous tissue. These are particularly prominent over the buttocks and the thighs. They look alert and often plethoric.

2. Problems of preterm neonates

The basic underlying feature of the preterm LBW infant is immaturity of its organ system. They are prone to develop

- Asphyxia necessitating resuscitation.
- Hypothermia
- Feeding problems - Preterm neonates less than 34 weeks of gestation cannot coordinate sucking and swallowing. Therefore, they are unable to feed from the breast.
- Respiratory distress syndrome (RDS): Preterm babies especially those less than 34 weeks have immature lungs, hence they develop RDS characterized by rapid and labored respiration, in-drawing of the chest, grunting and cyanosis.
- Apneic spells: Because of the immature respiratory control mechanisms these babies also have a tendency for apneic spells. In an apneic spell the baby stops breathing; develops slow heart rate and turns blue.
- Intra-Ventricular Hemorrhage (IVH): Preterm infants also have immature vascular bed around the brain ventricles. These delicate vessels may rupture and cause intra-ventricular hemorrhage.
- Hypoglycemia - Immature metabolic pathways of preterm infants predispose them to develop hypoglycemia.
- Hyperbilirubinemia
- Infection is another major problem among preterm babies and indeed an important killer because they are immuno-compromised hosts.
- Retinopathy of Prematurity (ROP): Preterm infants given excess oxygen may develop blindness because of damage to the immature retina.

3. Problems of SGA neonates

The basic underlying problem amongst them is in-utero undernutrition and hypoxia. They are prone to:

- Fetal distress, meconium passage in utero and birth asphyxia.
- Hypothermia.
- Hypoglycemia
- Congenital malformations.

4. Treatment:

Indication for hospitalization are:

- Birth weight of less than 1800 g;
- Gestational age of less than 34 weeks;
- Neonate who is not able to take feeds from the breast or by cup (Katori) and spoon (irrespective of birth weight and gestation);
- A sick neonate (irrespective of birth weight and gestation).

4.1. Keeping LBW Babies warm:

- Room temperature should be kept between 28-30°C.
- Baby should be provided skin to skin contact care (KMC) in the following ways:
 - Provide privacy to the mother. If mother is not available, skin to skin contact may be provided by the father or any other adult.
 - Request the mother to sit or recline comfortably.
 - Undress the baby gently, except for cap, nappy and socks.
 - Place the baby prone on mother's chest in an upright and extended posture, between her breasts, in Skin to Skin contact; turn baby's head to one side to keep airways clear.
 - Cover the baby with mother's blouse, 'pallu' or gown; wrap the baby-mother duo with an added blanket or shawl.
 - Breastfeed the baby frequently.
- If possible, warm the room (>25°C) with a heating device.
- Skin to Skin contact is the most practical, preferred method of warming a hypothermic infant in a primary health care facility. If not possible:

- Cloth the baby in 3-4 layers, cover head with a cap and body with a blanket or a shawl; hold baby close to caregiver's body, OR
- Place the baby under overhead radiant warmer, if available.
- Keep the young infant warm on the way to the hospital
 - By Skin to Skin contact OR
 - Clothe the baby in 3-4 layers, cover head with a cap and body with a blanket or a shawl; hold baby close to caregiver's body.

- In order to promote lactation and enable the baby to learn sucking, all babies on gavage or Katori-spoon feeds should be put on the breasts before each feed for 5 to 10 minutes. This will promote lactation and enable the baby to learn how to suck.
- When shifting a baby from one mode of feeding to another, be very careful. Introduce in new mode for only some of the feeds to begin with.
- The feeding of every baby should be individualized. The above recommendations should only serve as broad guidelines.
- Ensure use of expressed breast milk & start with small volume, and gradually build up.
- Most LBW babies weighing more than 1800 g are able to feed directly from the breast. In a stable, growing LBW baby daily intake of feeds should be gradually built up to 180-200ml/kg.
- LBW babies should be fed every 2-3 hours starting at 2 hours of age.

4.2. Nutrition & Fluids

- Neonates weighing less than 1200 g. or those having sickness should receive intravenous fluid initially.
- Enteral feeds should be introduced gradually by gavage as the baby's acute problem begins to settle.
- Infants weighing 1200-1800 g and not having significant illness should be put on gavage feeds initially.

Table 1: Guidelines for the modes of providing fluids and feeding			
Age	Categories of neonates		
Birth weight (gm)	< 1200	1200-1800	> 1800
Gestation (weeks)	< 30	30-34	> 34
Initial	IV fluids Triage Gavage feeds if not sick	Gavage feeds	Breast feeds If unsatisfactory, give cup-spoon feeds
After 1-3 days	Gavage feeds	Cup-spoon feeds	Breast feeds
Later (1-3 wks)	Cup-spoon feeds	Breast feeds	Breast feeds
After some time (4-6 wks)	Breast feeds	Breast feeds	Breast feeds

Note:

- i. On the first day the fluid requirements range from 60 to 80 ml/kg.
- ii. The daily increment in all the groups is around 15 ml per kg till 150 ml/kg is reached.
- iii. Adequacy of therapy is indicated by weight pattern in the expected range.

baby loses up to 1 to 2 percent weight every day amounting to 10 percent cumulative weight loss during the first week of life. Birth weight is regained by the 14th day.

4.2.1 Judging adequacy of nutrition

- The key measure of optimal feeding is the weight pattern of the baby. A preterm LBW

- SGA-LBW babies who are otherwise healthy should not have any appreciable weight loss at all and they should start gaining weight early.
- It is desirable to weigh all LBW babies at 2 weeks (to check regaining of the birth weight), 4 weeks (to ascertain a weight gain of at least 200-300g) and then every month. Hospitalized

LBW babies should be weighed every day on the same weighing machine.

- Excessive weight loss, or inadequate weight gain indicates inadequate feeding, cold stress, excessive insensible water loss or systemic illness (like anemia, sepsis, late metabolic acidosis etc).

4.3. LBW babies require close monitoring and follow up of

- Growth monitoring- head circumference and weight
- Developmental assessment and early stimulation
- Intraventricular haemorrhage screening by ultrasound cranium on day 1,3,7& at 4-6 weeks.
- Screening tests for hearing –at discharge
- Retinopathy of pre maturity screening at 1 month of age
- Screening for osteopenia of prematurity

4.4. Vitamin Supplements:

- All LBW Babies should receive intramuscular Vitamin K at birth. Every new born should receive Injection Vitamin K 1 mg and 0.5 mg, Intramuscular, as per the birth weight ≥ 1000 gm and < 1000 gm respectively.
- All pre-terms < 2000 gm should receive oral Vitamin and mineral supplement in doses shown below:
- Multivitamin preparation 0.3-0.6 ml (5-10 drops) / day (which usually provides vitamin A of 1000 IU/day and vitamin D 400 IU/day)
- Calcium 100-200 mg/kg/day
- Phosphorous 50-100 mg/kg/day.

All these supplements to be given till at least 6 months of age.

- Iron should be started at a dose of 1mg/kg/day at 4 weeks of age and provided till 12 months of age.
- Vaccination in LBW Babies
- If the LBW baby is not sick, the vaccination schedule is the same for as the normal babies. A

sick LBW babies however, should receive these vaccines only on recovery.

- Vitamin A 1000 IU orally daily – from 1-week age onwards

5. Immunization in special circumstances

5.1. Immunization in preterm infants:

In general, all vaccines may be administered as per schedule according to the chronological age irrespective of birth weight or period of gestation. Very low birth weight / preterm babies can be given immunization, if they are stable otherwise.

5.2 Children receiving corticosteroids:

- Children receiving IV immunoglobulin and oral corticosteroids in high doses (Prednisolone 1-2 mg/kg/day) for more than 14 days should not receive live virus vaccines until the steroid has been discontinued for at least one month.
- Killed vaccines are safe but may not be completely effective in such situations.
- Patients on topical or inhaled steroids should not be denied their age appropriate vaccine.

5.3 Children awaiting splenectomy:

Immunization with Pneumococcal, Hib, and Meningococcal vaccine should be initiated a few weeks prior to splenectomy.

5.4 Vaccination in children with HIV infection:

- Immune response may be suboptimal as it depends on the degree of immunodeficiency at that point of time.
- Re-administration of childhood immunization may be considered when their immune status has improved following anti-retroviral therapy.

5.5 Lapsed immunization:

There is no need to restart a vaccine series regardless of the time that has elapsed between individual doses. In case of unknown or uncertain immunization status,

however, it is appropriate to start the schedule as for an unimmunized child.

5.6. Minor illnesses

e.g. fever, diarrhea, respiratory infections and malnutrition should not be considered as contraindications to immunization.

- Mortality of LBW babies is inversely related to gestation and birth weight and directly to the severity of complication.
- In general, over 90% Low birth weight babies who survive the newborn period have no neurodevelopment handicaps.
- Therefore, essential care of the LBW neonates is a highly rewarding experience.

6. Prognosis

Bibliography:

1. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
3. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012

Further reading:

1. Saili A. Essential care of Low Birth Weight Neonates. Indian Pediatrics, 2008 Jan;45(1):13-5.



7 Assessment of Neonatal Sepsis

Neonatal sepsis is one of the three major causes of neonatal mortality. Sepsis is largely preventable.

1. Clinical manifestations of neonatal sepsis

1.1 Non-specific:

Lethargy, refusal to suckle, poor cry, not arousable, comatose

1.2 Gastro-intestinal:

Abdominal distension, diarrhea, vomiting, poor weight gain

1.3 Cardiovascular:

Hypothermia, poor perfusion, shock, bleeding and sclerema

1.4 Respiratory:

Cyanosis, tachypnea, chest retractions, grunt, apnea/gasping

1.5 CNS:

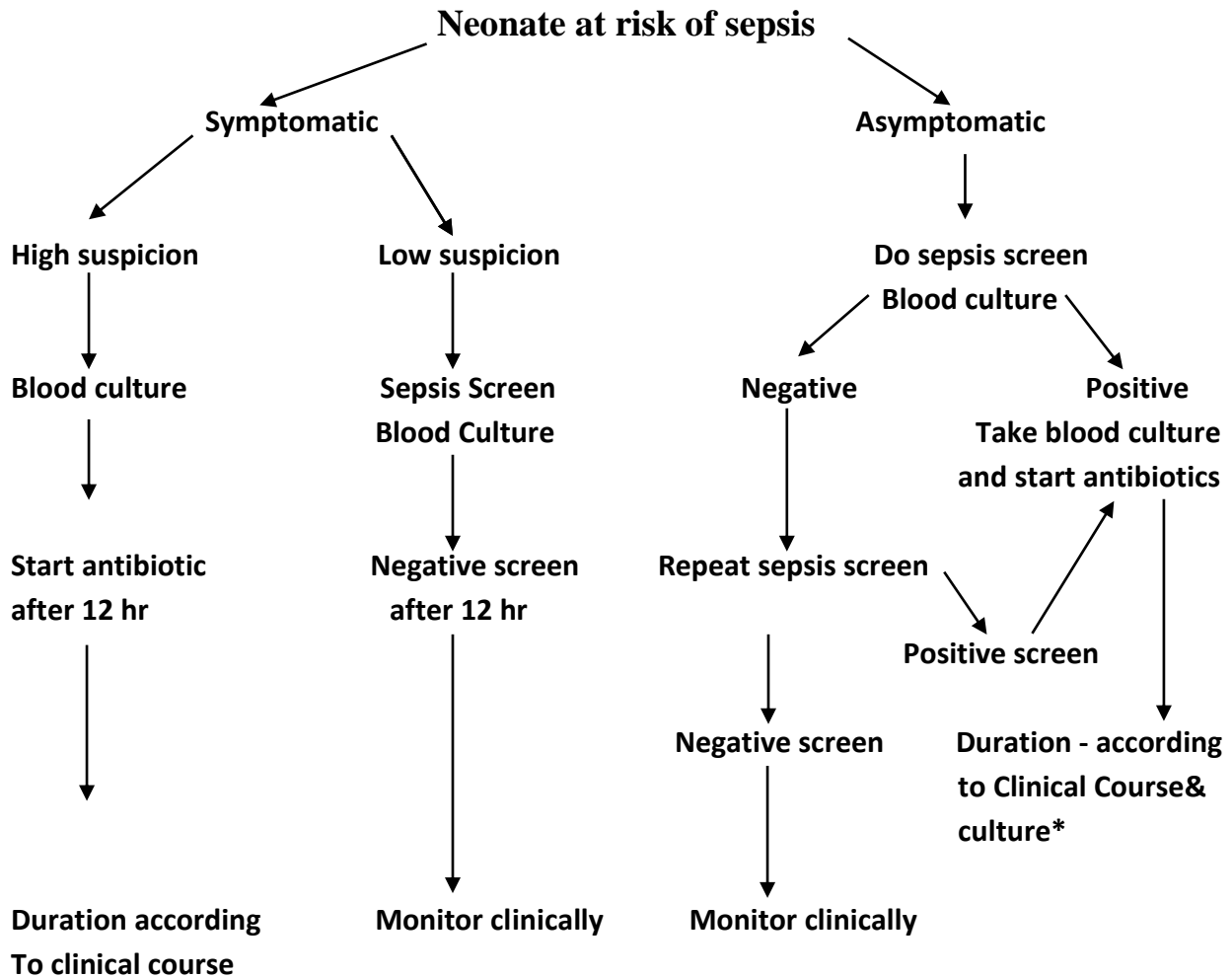
Fever, seizures, blank look, high pitched cry, excessive crying/irritability, neck retraction, bulging fontanel

2. Laboratory diagnosis of a newborn with sepsis

Sepsis Screening: Any of two tests that come positive out of the following five tests strongly indicate presence of sepsis:

1. Leukopenia (TLC/ Total leucocyte count <5000/cmm)
2. Neutropenia (ANC/ Absolute neutrophil count <1800/cmm)
3. Immature neutrophil to total neutrophil (I/T) ratio (>0.2)
4. Micro ESR/Erythrocyte sedimentation rate (>15mm 1st hour)
5. Positive CRP/ C-reactive protein

3. Approach to newborns at- risk of sepsis



Culture sterile - 7-10 days Culture Positive - 10-14 days

4. Antibiotic therapy for a newborn with sepsis

4.1. Choice of antibiotics

- Antibiotic therapy should cover the common causative bacteria, namely Escherichia coli, Staphylococcus aureus and Klebsiella pneumoniae
- A combination of Ampicillin and Gentamicin is recommended for treatment of sepsis and pneumonia

- In suspected or confirmed meningitis, add Cefotaxime with an aminoglycoside

4.2. Antibiotic therapy of neonatal sepsis

Following table provides the antibiotics and dosages of antibiotics for newborn sepsis

4.2.1 Septicemia of Pneumonia

Table-1: Antibiotic Management for Septicemia of Pneumonia

Antibiotic	Each dose	Frequency		Route	Duration
		<7 days age	>7 days age		
Inj Ampicillin or	50 mg/kg /dose	12 hourly	8 hourly	IV	7-10 days
Inj Cloxacillin	50 mg/kg /dose	12 hourly	8 hourly	IV	7-10 days
and					
Inj Gentamicin or	5 mg/kg/dose	24 hourly	24 hourly	IV	7-10 days
Inj Amikacin	15 mg/kg/dose	24 hourly	24 hourly	IV	7-10 days

4.2.2 Meningitis

Table-2: Antibiotic management of Meningitis

Antibiotic	Each dose	Frequency		Route	Duration
		<7 days age	>7 days age		
Inj Ampicillin or	100 mg/kg/dose	12 hourly	8 hourly	IV	3 weeks
And					
Inj Gentamicin	2.5 mg/kg/dose	12 hourly	8 hourly	IV	3 weeks
OR					
Inj Gentamicin or	5 mg/kg/dose	12 hourly	8 hourly	IV	3 weeks
And					
Inj Amikacin	2.5 mg/kg/dose	12 hourly	8 hourly	IV	3 weeks

5. Supportive care of a Newborn with sepsis

- Provide warmth, ensure consistently normal temperature
- Start intravenous line
- If CFT >3 seconds, infuse normal saline 10 ml/kg over 20-30 minutes, repeat the same 1-2 times, if perfusion continues to be poor
- Infuse 10% dextrose 2 ml/kg stat
- Inject Vitamin K1 mg intramuscularly

- Start oxygen by hood or mask, if cyanosed or grunting
- Provide gentle physical stimulation, if apneic. Provide bag and mask ventilation with oxygen if breathing inadequate
- Avoid enteral feed if hemodynamically compromised, give maintenance IV fluids
- Consider use of Dopamine if perfusion is persistently poor
- Consider exchange transfusion if there is sclerema

6. Summary of commonly used dosage in Neonates

Drug	Dose	Route
Ampicillin	Age <7 days: 50 mg/kg/dose, q 12 hour Age >7 days: 50 mg/kg/dose, q 8 hour	IV
Gentamycin	Sepsis/ pneumonia 5 mg/kg/dose, q 24 hour Meningitis Age <7 days: 2.5 mg/kg/dose, q 12 hour Age >7 days: 2.5 mg/kg/dose, q 8 hour	IV
Amikacin	<7 days: 15 mg/kg/dose, q 24 hour	IV
Cefotaxime	<7 days: 50 mg/kg/dose, q 12 hour >7 days: 50 mg/kg/dose, q 8 hour	IV
Chloramphenicol	12 mg/kg/dose, q 12 hour	IV
Aminophylline	5 mg/kg loading, then 2 mg/kg/dose q 8-12 hour	IV
Vitamin K	1 mg	IM
Phenobarbitone	20 mg/kg loading over 10-15 minutes then 3-4 mg/kg q 24 hour	Loading IV Then IV, IM or oral
Phenytoin	15-20 mg/kg loading over 10-15 minutes then 5 mg/kg q 24 hour	IV
Dopamine/ Dobutamine	5-20 micro g/kg/minute	IV continuous

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
3. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
4. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012
5. Engorn B, Flerlage J. The Harriet Lane Handbook. 20th Edition. Saunders; 2014

Further Reading:

1. Isaacs D. Neonatal Sepsis: The Antibiotic crisis. Indian Pediatrics. 2005 Jan;42(1):9-13.



8. Treatment of Respiratory Distress in Newborn

1. Incidence:

Increases as Gestation age decreases
 28 weeks => 60 %
 28-34 weeks => 30%
 >34 weeks => 5-10%

Dexamethasone => 6mg IM 12 hourly for 2 Days

(NOTE: validity of this injection is for one week and do not repeat after that if more delay in delivery, because it affects brain of newborn).

2. Prevention:

Can be prevented by giving Antenatal steroid to mother

- Indicated for those who are at risk for preterm delivery in next 8 days
- Treatment for Prevention
 Betamethasone 12 mg IM once a day for 2 days
 OR

3. Assessment after delivery:

- Gestational age
- Temperature
- Heart rate
- SaO2
- Capillary refill time

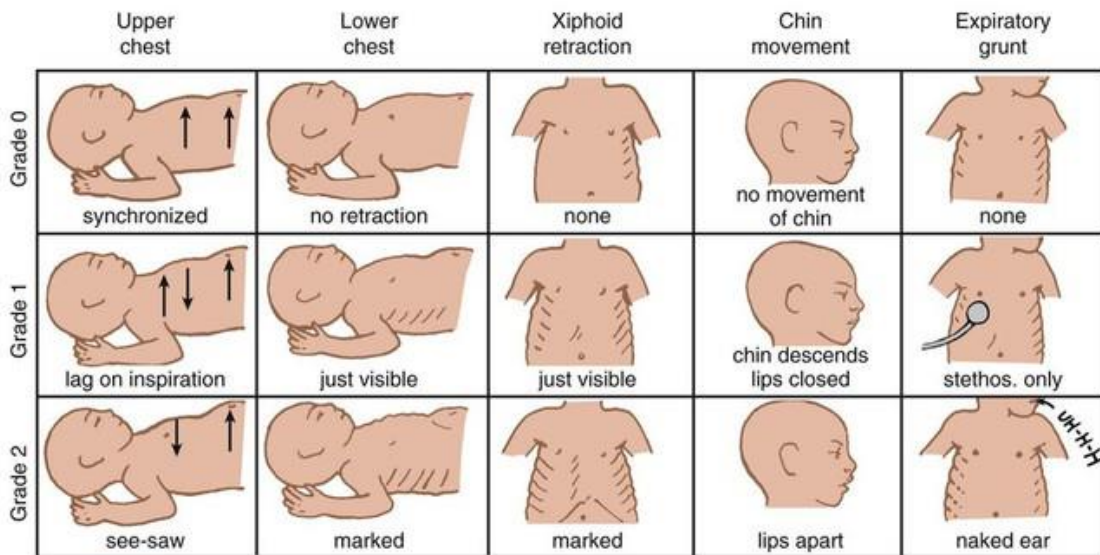


Figure 8.1 Silverman –Anderson score

4 Scoring of distress

FEATURE	SCORE 0	SCORE 1	SCORE 2
Chest Movement	Equal	Respiratory Lag	Seesaw Respiration
Intercostal Retraction	None	Minimal	Marked
Xiphoid Retraction	None	Minimal	Marked
Nasal Flaring	None	Minimal	Marked
Expiratory Grunt	None	Audible w/ stethoscope	Audible

Interpretation:

- SA score=> 3 Oxygen by hood
- 3 -7 => CPAP ventilation
- >7 Mechanical ventilation

4.2. Investigation:

- X ray chest: PA view
- Septic screening
- Blood culture
- Serum electrolyte
- ABG
- Blood glucose

5. Management:

In general:

- Maintain temperature =36.5 -37.5°C
- Restrict fluid to 2/3 maintenance
- Maintain SaO₂ 88-95%
- Antibiotics if septic screen +ve

Main management

- If FiO₂ requirement is < 30% & SA score <3
 - **Oxygen by hood**
 - Watch for diuresis
 - If improvement noticed – give maintenance fluid and start feed
- If FiO₂ Requirement is > 30% & SA score >3
 - **Start CPAP**
 - CPAP –Begin with 4-6 cm of water pressure and use FiO₂ 10 % more than required

- Monitor ABG
- W/F Apnea
 - Increase CPAP BY 1-2cm up to 8cm water pressure
 - And FiO₂ up to 100%
 - Start **Mechanical ventilator**: if with FiO₂ of 100% and CPAP of 8cm



- SA score is > 7
- OR
- Baby with persistent Apnea
 - Other
 - watch for diuresis
 - watch for clinical improvement
 - give maintenance of fluid

5.4 Fluid management

- If CRT is more than 3
 - Give 10ml/kg NS bolus
 - Monitor liver size
 - If require give Dopamine 5-10 micro gr/kg/min
- OR
- Dobutamine 10 micro gr/kg/min

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
3. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012

Further reading:

1. Prabhakaran P. Acute Respiratory Distress Syndrome. Indian Pediatrics. 2010 Oct;47(10):861-8.



9. MECONIUM ASPIRATION SYNDROME

1. Management in delivery room

1.1 If MAS, watch for –

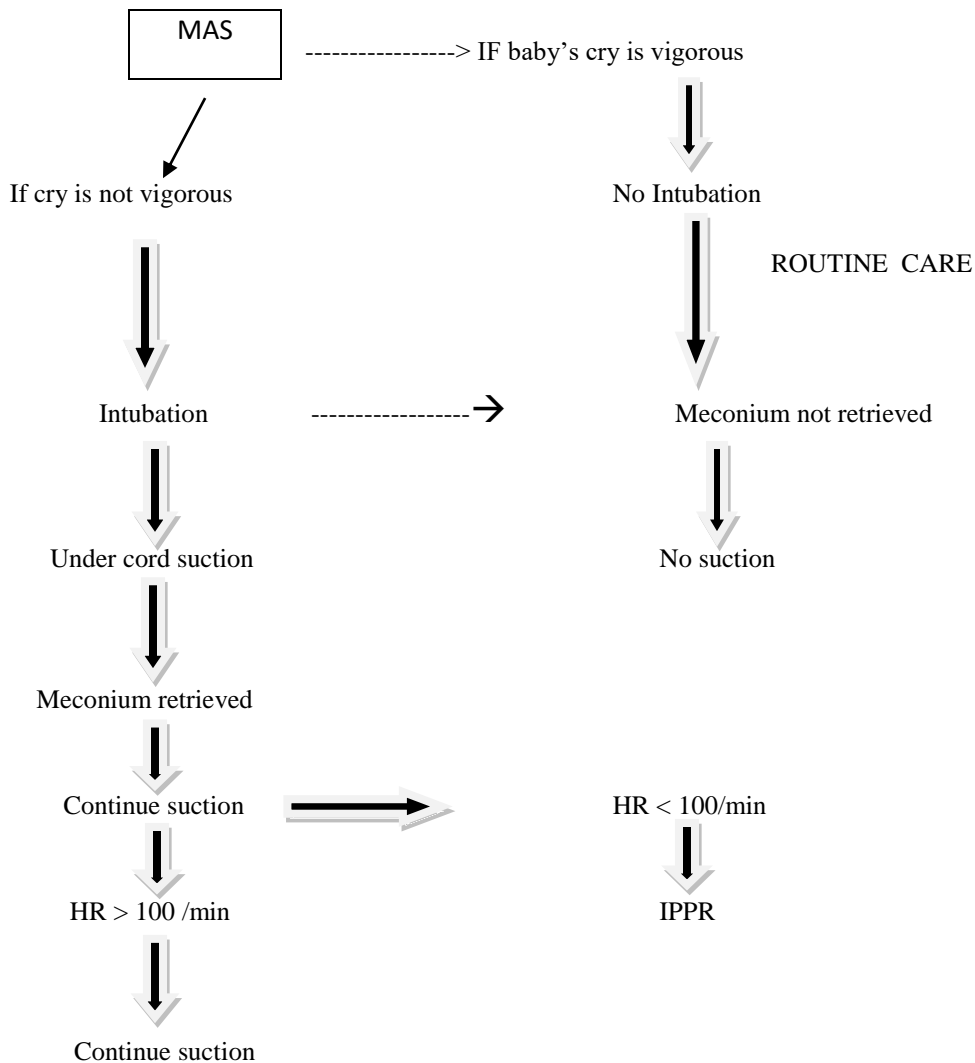
- Colour /consistency of meconium
- Gestational Age

- Vertex or breech presentation
- Birth Asphyxia

1.2 Perineal suction – With Dee Lee mucus

Aspirator at delivery of Head of baby

Figure-9.1: Indication of Endotracheal intubation



2. Management in NICU

2.1 Assessment of baby:

- Gestational age
- r/o IUGR
- r/o Umbilical cord /nail/skin staining

Downe score	0	1	2
Respiratory rate	<60	60-80	>80
Grunting	NO	Audible with Stethoscope	Audible without Stethoscope
Cyanosis	NO	In room air	With 40% oxygen
Air entry	good	decrease	Markedly decrease
Retraction	NO	mild	severe

2.2 Interpretation

score

1 -3 => mild respiratory distress => Oxygen by hood

4-6 => moderate respiratory distress => CPAP ventilation

>6 => severe respiratory distress => Ventilator

Bibliography:

1. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
2. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012

Further reading:

1. Wiedemann JR1, Saugstad AM, Barnes-Powell L, Duran K. Meconium Aspiration Syndrome. Neonatal Network. 2008 Mar-Apr;27(2):81-7.



3. Investigation:

- CBC
- ABG
- X-ray chest
- Blood culture

4. Supportive management

- Pass infant feeding tube=> gastric lavage
- Iv fluid initially if required
- Antibiotic if respiratory distress OR
- If septic screen is positive
- Treat complication
- Ventilator if required along with surfactant

5. Complication

- Atelectasis
- Hypoxic Ischaemic encephalopathy
- Pneumothorax
- PPHN (Persistent primary pulmonary hypertension) requiring nitrous oxide and sildenafil

10. BLEEDING NEONATE

1. Common causes

1.1. GI bleeding

- Swallowed maternal blood
- Hemorrhagic disease of New Born
- Sepsis
- Stress ulcer
- Platelet defect
- Clotting factor deficiency

1.2. Umbilical cord

- Slipped ligature
- Vitamin K deficiency

1.3. Skin:

Petechiae

- Sepsis
- Platelet disorder

Ecchymosis

- Sepsis
- Clotting factor deficiency

1.4 Occult blood

Cephalohematoma } Preterm
Intra Ventricular Hemorrhage } Vit..K deficiency
Subdural Hemorrhage } sick neonate

2. Approach

Maternal History INCREASE RISK OF

2.1.1 Antenatal infection

e.g. TORCH

} Thrombocytopenia

2.1.2 Drugs given to mother

Phenytoin

Phenobarbitone

Aspirin / Anticoagulant

} Hemorrhagic
disease of NB

2.1.3 Bleeding in mother =

Thrombocytopenia

e.g. ITP /SLE

Birth Asphyxia

Trauma

} DIC

2.2. Time of Bleeding

2.2.1 < 24 hrs = Swallowed maternal blood

hemorrhagic disease of NB

2.2.2 > 24 hrs = Classical hemorrhagic

disease of NB

- Sepsis /DIC
- Platelet abnormality
- Trauma
- Liver dysfunction

2.3. Other clinical features

- Petechiae
- Ecchymosis
- Pallor
- Jaundice
- Hepatosplenomegaly
- Bulging Anterior Fontanelle.

3. Investigation

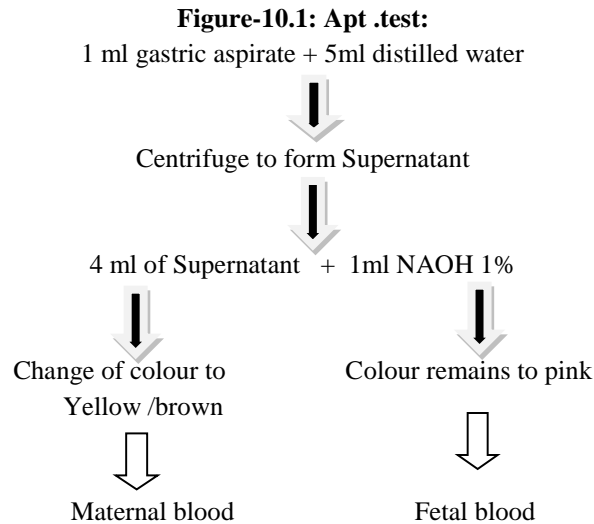


Table 1: Differential Diagnosis

	Platelet	PT	PTT
Thrombocytopenia	↓	N	N
Hemorrhagic disease of newborn	N	↑	↑
Clotting factor deficiency	N	N	↑
DIC	↓	↑	↑

4. Management

<p>A) If APT test shows –Maternal blood =>parental counseling</p> <p>B) If APT test shows –fetal blood</p> <p>a) Injection Vitamin K 1mg IV stat</p> <p>b) If PT/PTT increases OR in sick neonate 10-15ml /kg FFP (If required repeat after 12hrs)</p> <p>c) If Hb<10 gm% = Give fresh blood transfusion</p> <p>d) If platelet <50000 in sick Neonate OR <25000 in Healthy Neonate Platelet transfusion 10-20ml/kg</p>	<p>C) Treat for sepsis / DIC / primary disease</p> <p>Antibiotics:</p> <ul style="list-style-type: none"> • Ampicillin 50 to 100 mg/kg in divided doses IV. • Gentamycin 5 mg/kg in divided doses iv. • If not responding to these then start higher antibiotics such as Ceftriaxone, Meropenem etc. • Treatment of DIC. • Treatment of primary disease.
--	---

Bibliography:

1. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
2. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012



11. JAUNDICE IN THE NEWBORN

1. Introduction:

Hyperbilirubinemia is the commonest morbidity in the neonatal period and 5-10% of all newborns require intervention for pathological jaundice. Nearly 60% of term newborn becomes visibly jaundiced in the first week of life. In most cases, it is benign and no intervention is required.

2. Physiological jaundice:

Jaundice attributable to physiological immaturity of neonates to handle increased bilirubin production. Visible jaundice usually appears between 24-72 hours of age. Total serum bilirubin (TSB) level usually rises in full-term infants to a peak of 6 to 8 mg/dL by 3 days of age and then falls. A rise to 12mg/dL is in the physiologic range. In premature infants, the peak may be 10 to 12 mg/dL on the fifth day of life, possibly rising over 15 mg/dL without any specific abnormality of bilirubin metabolism.

3. Pathological jaundice:

- TSB concentrations exceeds 5 mg/dl on first day of life in term neonate, 10 mg/dL on second day, or 12-13 thereafter.
- Any TSB elevation exceeding 17 mg/dL.
- Appearance of jaundice within 24 hours, peak TSB levels above the expected normal range,
- Presence of clinical jaundice beyond 3 weeks and conjugated bilirubin (dark urine staining the clothes and light colored stool).

4. Signs & Symptoms:

4.1 Clinical examination of jaundice:

- The newborn should be examined in good daylight.
- The skin should be blanched with digital pressure and the underlying color of skin and subcutaneous tissue should be noted.
- Dermal staining in newborn progresses in a cephalo-caudal direction. Newborns detected to have yellow discoloration of the skin beyond the legs should have an urgent laboratory confirmation for levels of TSB.

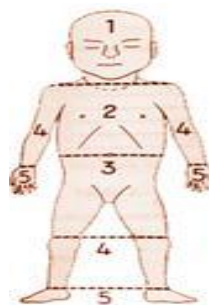


Figure 11.1: Levels of Jaundice

4.2 Clinical determination of jaundice by Kramer's criteria

Area of body	Range of serum bilirubin (mg%)
Head and neck	4-8
Upper trunk	5-12
Lower trunk and thigh	8-16
Arms and lower limbs	11-18
Palms and soles	>15

5. Investigations:

Following investigations must be done in a case of neonatal jaundice

- Serum bilirubin direct and indirect.
- Blood grouping of mother and child ABO and Rh.
- Direct Coomb's test in infant.
- Hematocrit and peripheral smear for RBC morphology and reticulocyte count.
- Indirect Coomb's test in mother if she is Rh negative

6. Complication:

6.1 Transient encephalopathy:

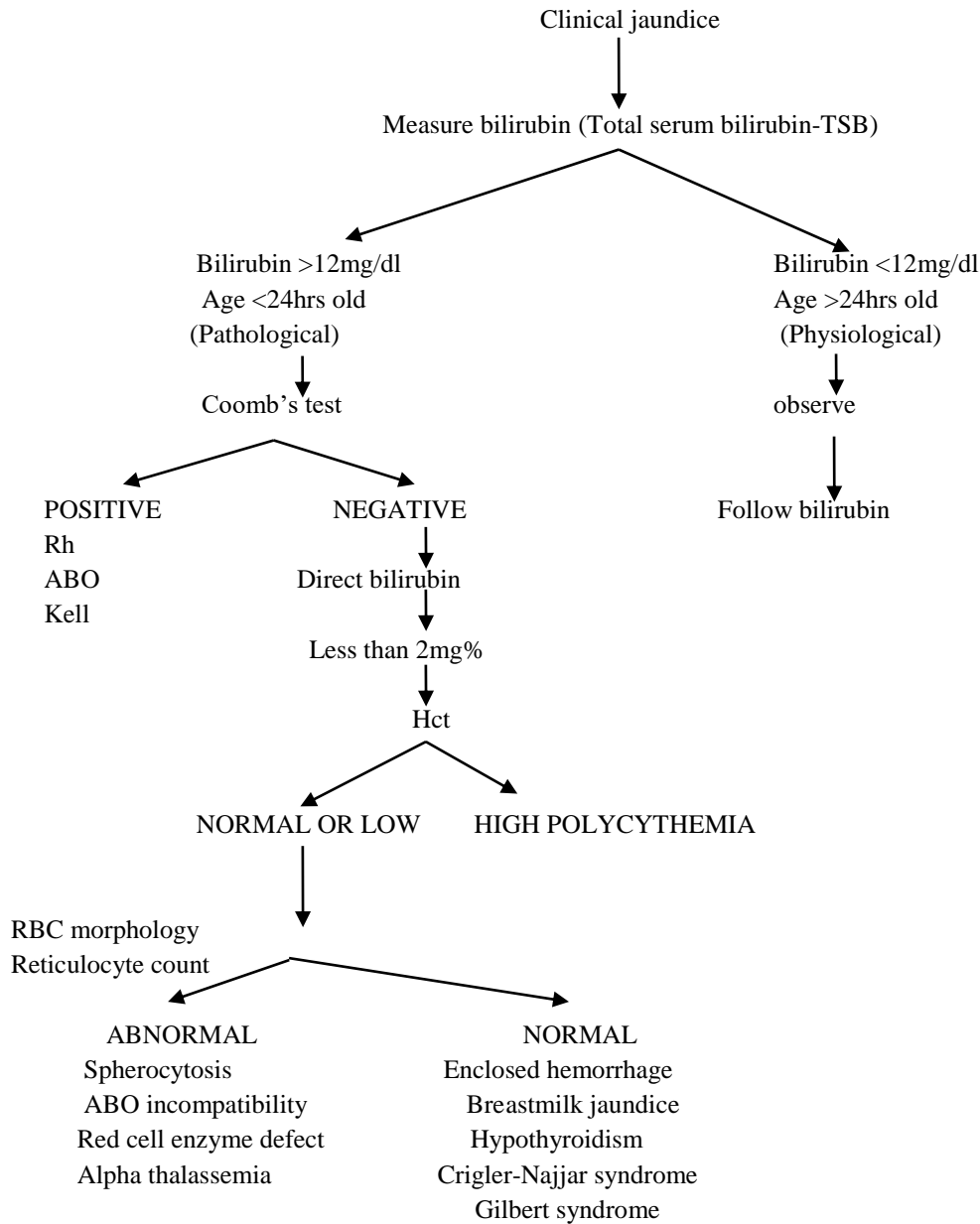
Early bilirubin induced neurologic dysfunction is transient and reversible. This is suspected by increasing lethargy with rising bilirubin levels but recovery following or prompt exchange transfusion.

6.2 Kernicterus.:

This term has been traditionally used to describe the pathological findings of bilirubin toxicity within the brain. This includes staining and necrosis of neurons in the basal ganglia, hippocampal cortex, Sub-thalamic nuclei and cerebellum followed by gliosis of these areas, should the baby survive. The cerebral

cortex is generally spared, but 50 % of babies have extra-neuronal lesions with necrosis of renal tubular cells, intestinal mucosa and pancreatic cells. They may manifest as gastrointestinal hemorrhage or hematuria. Clinically, kernicterus is described in phases, which may progress over 24 hours to 7 days:

FIGURE-11.2: DIAGNOSTIC WORKUP FOR HYPERBILIRUBINEMIA IN NEWBORN



7. Treatment modalities of hyperbilirubinemia

- Hydration
- Phototherapy (Do not keep in sunlight).
- Exchange transfusion
- Drugs to increase conjugation.

7.1. HYDRATION

Continued and frequent breast feeding 8-10 times/day

7.2. Phototherapy

- Special blue lights to be used
- 45cm distance between baby and phototherapy unit, in conventional phototherapy unit and keep the baby as close as possible around 15-20 cm in LED phototherapy unit for intensive phototherapy.
- Eyes and genitalia should be covered
- Double surface phototherapy is preferred
- Watch for side effects (diarrhoea, skin rash, hyper/hypothermia)
- Healthy, term newborn (>37 weeks)

TABLE 1- Guidelines for Phototherapy according to AAP

Age (hours)	Consider Phototherapy TSB (mg/dl)	Phototherapy TSB (mg/dl)
< 24		
25-48	>12	>15
49-72	>15	>18
>72	>17	>20

NOTE: TSB-Total serum bilirubin

7.2.1. Based on birth weight and health of the newborn

TABLE 2- Phototherapy indications

Birth weight (grams)	Healthy Newborn TSB (mg/dl)	Sick newborn TSB (mg/dl)
<1000	5-7	4-6
1001-1500	7-10	6-8
1501-2000	10-12	8-10
2001-2500	12-15	10-12
	TERM	
>2500	15-18	12-15

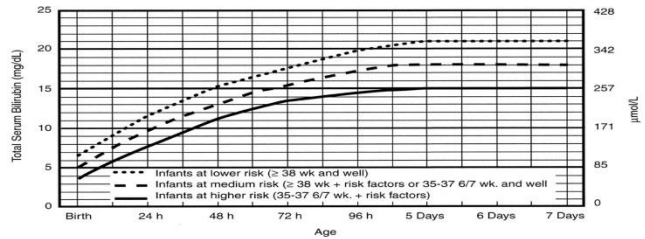


Figure 11.3 Phototherapy

7.3 Points to remember

- Try to establish diagnosis before instituting phototherapy by carrying out necessary investigations.
- Check blue light functioning; life of these light is 1500-2000hrs (approx. 3 months). Keep lights at distance of 18'' from the baby.
- When blue lights are not available four pairs of white tube light may be used instead.
- Change the position of baby after every 2 hrs.
- Babies can be taken out of phototherapy for breast feeding
- Monitor baby's temperature 2 hourly.
- Monitor fluid balance – daily weight and urine output. Increase fluid as necessary.
- Shield the eyes in both sexes to prevent the retinal damage and genitals in male to prevent mutation defect in adulthood.
- Monitor rise or fall of bilirubin every 12 hourly.
- Do not give phototherapy for direct hyperbilirubinemia.

7.4. Exchange transfusion.

7.4.1 Choice of blood

- If baby and mother is Rh-ve, use only Rh-ve blood.
- Always cross match donors blood with both mothers and baby's blood.

7.4.2 Criteria for Exchange Transfusion

- Cord bilirubin more than or equal to 4.5mg% and Hb < 11g%
- Rate of rise of bilirubin >1mg/dl despite phototherapy
- In LBW babies, indirect bilirubin > (weight in g)/100
- Exchange earlier at level of 2 mg% less for following Criteria- Sepsis, RDS, Asphyxia, Acidosis, Hypoglycemia.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
3. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
4. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012



12. MANAGEMENT OF SURGICAL NEONATE

1. Neonatal surgical problems

1.1. Major

- Tracheoesophageal fistula
- Diaphragmatic hernia
- Intestinal Obstruction
- Neural tube defect
- Anorectal malformation

1.2 Others

1.2.1 Orofacial

- Cleft lip
- Cleft palate
- Choanal Atresia
- Pierre robin sequel
- Laryngeal web

1.2.2 Gastrointestinal

- Esophageal Atresia
- Cong. Hypertrophic Pyloric Stenosis (CHPS)
- Gastrochiasis
- Abdominal wall defect
- Omphalocele
- Omphalitis

- Chest deformities
- Congenital Lobar emphysema
- Vascular Ring
- Orthopedic
- CTEV (Cong. Talipes Equino Varus)
- Developmental dysplasia of hip
- Other
- Clavicle fracture
- Humerus fracture
- Femur fracture

2. Transporting baby with surgical problems

- Write specific diagnosis
- Write, inform /Delivery details
- Give radiological, blood or other investigation report
- Give Inj. Vitamin K to all babies at birth
- Stabilize and Manage hypoglycemia / hypocalcemia / hypothermia or other metabolic problems

Figure 12.1 Gastrochiasis



Figure 12.2 Neural tube defect



Figure 12.3 Cleft lip



Bibliography:

1. Singh M. Care of Newborn. 8th Edition. CBS Publishers; 2015
2. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012



13. VITAMIN A DEFICIENCY

1. Introduction

Vitamin A Deficiency Symptoms –

- Delayed dark adaptation
- More prone for intestinal, respiratory and urogenital infections

2. Classification

XN-----Night Blindness

X1A----Conjunctival Xerosis

X1B----Bitot's Spots

X2-----Corneal Xerosis

X3A---Corneal Ulceration <1/3

X3B---Corneal Ulceration >1/3

XS----Corneal Scarring

XF---Xerophthalmic Fundus



Figure 13.1 Manifestation of Vit A Deficiency

3. Treatment immediately after diagnosis

3.1 Age wise doses of Vitamin A

<6 months-----50,000IU Vitamin A orally

6 to 12 months-----1,00,000IU Vitamin A orally

>12 months-----2,00,000IU Vitamin A orally

Next day-----Same age specific dose

At least 2 weeks later-----Same age specific dose

3.2 Important aspects of Vitamin A

- If the child more than 1 year of age and weighing less than 8 kg., then Vit. A dose is 1,00,000 IU to avoid Hypervitaminosis A
- Give oil based preparation
- Bottles have solution strength of 1, 00,000IU Vitamin A/ml
- Cold chain is not required.
- Shelf life of unopened opaque container is 2 years.
- Opened liquid is to be used in 6 to 8 weeks.
- Capsules are partially protected against loss of potency.

3.3 Treat

- Night blindness, conjunctival xerosis, Bitot spots, Corneal xerosis, Corneal ulceration, Keratomalacia in all except women of reproductive age group.
- In acute corneal lesion, patient should be referred to hospital on emergency basis.
- High dosages of Vit A can cause pseudo-tumour cerebri which will manifest as vomiting headache and bulging anterior fontanelle

3.4 Corneal Xerophthalmia

- i. Topical Antibiotic ointment (Tetracycline 1%, eye ointment, Tobramycin eye ointment 3% twice a day)
- ii. Eye protection with dark shades

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further Reading:

1. Arlappa N. et. al. Prevalence of Clinical and Sub-Clinical Vitamin A Deficiency Among Rural Preschool Children of West Bengal. Indian Pediatrics. 2011 Jan;48(1):47-9.



14. RICKETS

1. Introduction

Metabolic disease of childhood in which the osteoid, the organic matrix of bone fails to mineralize, due to interference with calcium metabolism.

2. Causes

- Vitamin D deficiency
- Malabsorption
- Renal disease
- Celiac disease
- Hepatic osteodystrophy
- Anti - epileptic drugs

10 important clinical features in Rickets

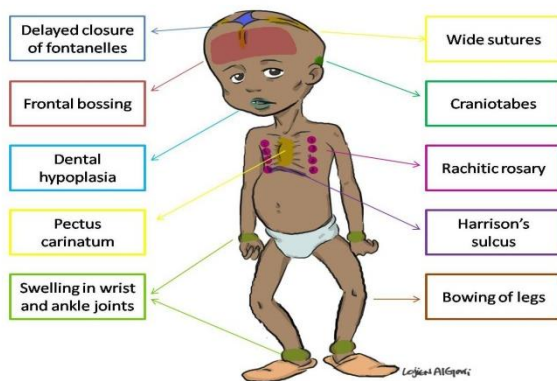


Figure 14.1: Clinical features in Rickets



Figure 14.2: Swelling in wrist due to Rickets

3. Investigations

3.1 Serology

- Serum calcium - normal or decreased,
- Serum phosphorus - decreased,
- Alkaline phosphatase - increased,

3.2 Urine Exam

Urinary calcium - decreased

3.3 X- ray:

- Generalized demineralization
- Loss of transverse trabeculae

Wrist x-rays in a normal child (A) and a child with rickets (B). Child with rickets has metaphyseal fraying and cupping of the distal radius and ulna.



- No sub-periosteal resorption of bone

4 Treatment

- Single oral dose of 6 lakh IU of Vitamin D
- 2nd dose after 3 to 4 weeks (if no sclerotic change is seen in x-ray)
- If the child responds to above treatment maintenance dose of 400 IU of vitamin D is given once Serum alkaline phosphatase is normal, consider corrective surgery, if any.
- Calcium - 0.5 to 3 gm/day for 3 months
- Vitamin D - 10,000 IU/day once a month
- High protein diet

It is important to note that Rickets is a disease of growing bones so it manifests in recovery phase of Severe Acute Malnutrition (SAM)

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Cashman KD. Vitamin D in childhood and adolescence. *Postgrad Med J.* 2007 Apr; 83(978): 230–235.
2. Pettifor JM. Rickets and vitamin D deficiency in children and adolescents. *Endocrinol Metab Clin North Am.* 2005;34(3): 537–553



15. MANAGEMENT OF CHILDREN WITH ANAEMIA

1. Introduction

Mild to Moderate anaemia is a common co-morbidity in children attending health facility for various conditions. Hence, anemia/pallor should be looked for in each patient attending the health facility.

Severe anemia in a child is suggested by the presence of severe palmar pallor and may be associated with a fast pulse rate, difficulty in breathing, or confusion or restlessness. Nutritional anaemia is the most common cause of anaemia in children.



2. Facility level management

Any child reporting to any facility (PHC level and above) with any illness will be assessed clinically by the attending Medical Officer for anaemia routinely and should undergo Hb estimation if found to be anaemic clinically

Nutritional anaemia results from deficiency of iron, folic acid and vitamin B12. Children having anaemia due to folic acid and/or B12 deficiency (megaloblastic anemia) may have hyper pigmentation of knuckles and occasionally bleeding manifestations due to thrombocytopenia.

The onset of anaemia in young children is generally after 6 months of age. Before this, iron in breast milk is sufficient to meet the needs of a breastfed child.

In India, diets for children in the age group 6–23 months are predominantly plant-based and provide insufficient amounts of micronutrients to meet the recommended nutrient intakes.

Table 1: Clinical assessment of anaemia in children less than 5 years

Findings on anaemia in children	
History	Examination
<ul style="list-style-type: none"> • Duration of symptoms • Usual diet (before the current illness) • Family circumstances (to understand the child's social background) • Prolonged fever • Worm infestation • Bleeding from any site • Lymph node enlargement • Previous blood transfusions • Similar illness in the family (siblings) 	<ul style="list-style-type: none"> • Severe palmar pallor • Skin bleeds (petechial and/or purpuric spots) • Lymphadenopathy • Hepato-Splenomegaly • Signs of heart failure (gallop rhythm, raised JVP, respiratory distress, basal crepitations)

All children referred from field to health facility due to palmar pallor will undergo Hb level estimation before initiating treatment

Children will be categorised as having mild, moderate and severe anaemia on the basis of Hb

levels and will be managed as per Table below.

Table 2: Treatment of Anaemia

Level of HB	Treatment	Follow-up	Referral
No Anaemia (>11 gm/dl)	20 mg of elemental iron and	100 mcg of folic acid in biweekly regimen	
Mild Anaemia (10–10.9 gm/dl) And Moderate Anaemia (7–9.9 gm/dl)	3 mg of iron/ Kg/ day for 2 months	Follow-up every 14 days by ANM Hb estimation after completing 2 months of treatment to document Hb>11 gm/dl	In case the child has not responded to the treatment of anaemia with daily dose of iron for 2 months, refer the child to the FRU/DH with F-IMNCI trained MO/ Paediatrician / Physician for further investigation
Severe Anaemia (<7 gm/dl)	Refer urgently to DH/FRU		

Note:

After completion of treatment of anaemia and attaining Hb level >11.5 gm/dl, the IFA supplementation to be resumed.

Treatment of anaemia with iron should be withheld in case of acute illness, severe acute malnutrition and in

a known case of haemoglobinopathy and anaemia in these cases should be treated as per the standard treatment guidelines, by the attending physician, as per the merit of the individual case.

2.1 Dose of IFA syrup

Table 3: Dose of IFA syrup for anaemic children 6 months–5 years

Age of Child	Dose and Frequency
6 months–12 months (6–10 kg)	1 ml of IFA syrup Once a day
1 year–3 years (10–14 kg)	1.5 ml of IFA syrup Once a day
3 years–5 years (14–19 kg)	2 ml of IFA syrup Once a day

2.2 Management of severe anemia at hospital level

2.2.1 History & examination

Table 4: Management of severe anaemia at FRU/DH (as per F-IMNCI) in children 6 months–5 years and 5-10 years

History to be taken for	Examination for
<ul style="list-style-type: none"> Duration of symptoms Usual diet (before the current illness) Family circumstances (to understand the child's social background) Prolonged fever Worm infestation Bleeding from any site Any lumps in the body Previous blood transfusions Similar illness in the family (siblings) 	<ul style="list-style-type: none"> Severe palmar pallor Skin bleeds (petechial and/or purpuric spots) Lymphadenopathy Hepato-splenomegaly Signs of heart failure (gallop rhythm, raised JVP, respiratory distress, basal crepitation)

2.2.2 Investigation & blood transfusion indication

Table 5: Investigation & Management

Investigations	Indication for Blood Transfusion	Blood Transfusion
<ul style="list-style-type: none">• Full blood count and• Examination of a thin film for cell morphology• Blood films for malaria parasites• Stool examination for ova, cyst and occult blood	<ul style="list-style-type: none">• All children with Hb \leq4 gm/dl• Children with Hb 4–6 gm/dl with any of the following:<ul style="list-style-type: none">• Dehydration• Shock• Impaired consciousness• Heart failure• Deep and laboured breathing• Very high parasitaemia (>10% of RBC)	If packed cells are available, give 10 ml/kg over 3–4 hours preferably. If not, give whole blood 20 ml/kg over 3–4 hours.

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Zlotkin S. Current Issues for Prevention and Treatment of Iron Deficiency Anemia. Indian pediatrics. 2002 Feb;39(2):125-9



16. MUMPS

1. Introduction:

Mumps is swelling of parotid glands due to infection of the parotids by mumps virus. Apart from parotids, it affects some other vital organs and systems in the body and can have serious complications with risk of morbidity and mortality. Therefore, it is important to know the course of illness, various complications, early detection for prompt management, and preventive measures.

2. Signs & Symptoms:

Un-immunized host gets infected from a case. Patient is infective 1-2 days before appearance to 2-3 days after disappearance of parotid swelling. Incubation period ranges between 2-3 weeks. 30-40% infections are sub clinical. After entry through air, the virus multiplies in respiratory tract and reaches all organs via blood. Prodromal symptoms are in the form of fever, muscle pains, neck pain, and malaise.



Figure 16.1: Mumps: Parotid Swelling

3. Investigation:

Diagnosis: is mainly clinical more suggestive with the history of contact. Typical painful tender bilateral parotid swelling with systemic symptoms is diagnostic.

4. Complication:

- CNS- Aseptic meningitis-meningoencephalitis along with parotitis or 10 days later. Meningitis is indistinguishable from any other meningitis. CSF study shows white blood cell pleocytosis predominance of lymphocytes. The CSF glucose content is normal in most patients, but a moderate hypoglycorrhachia (glucose content 20-40 mg/dl.) may be seen in 10-20% of the patients. The CSF protein content is normal or mildly elevated.

- Orchitis - Occurs in older patients. Occurs one week following parotitis with painful swelling of testes. Illness lasts for about 4 days. Testicular atrophy may follow in 1/3 patients with rare affection of fertility.
- Oophoritis in females may rarely occur.
- Pancreatitis - Epigastric pain, tenderness, vomiting, fever are suggestive. Laboratory evidence of raised amylase confirms the diagnosis.
- Thyroiditis - May follow after one week in adults with development of anti-thyroid antibodies.
- Myocarditis – Mild to moderate myocarditis with ST changes on ECG is found in some older patients. Chest pain, bradycardia and fatigue are presenting symptoms in a patient with mumps
- Deafness - transient or permanent unilateral or bilateral nerve deafness may follow mumps.
- Arthritis - Migratory poly arthralgia or even arthritis may be seen. These complications should be treated symptomatically.

5. Management:

Treatment:

- Mainly symptomatic with paracetamol(10-15mg/kg) and frequent gargling with warm saline.
- Orchitis is treated by bed rest and local support.
- Watch for evidence of complications. If arthritis occurs, prednisolone (1 to 2 mg/kg in divided doses for 5 to 7 days and then tapered).

6. Prevention:

MMR vaccine - combination of mumps, measles, and rubella offers above 95% protection. Primary immunization at the age of 15 months, followed by revaccination at 10 years.

Since it is a live viral vaccine, immuno-compromised hosts should not be vaccinated.

Isolation of the affected child is not useful to prevent spread to other children in usual contact. However,

fresh contacts like guest children can be protected if the affected child is isolated from them.

Children usually recover from mumps in about 10-12 days- First attack of mumps almost always gives

lifelong protection against another, therefore, such children do not benefit from any immunization later.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013



17. SEVERE ACUTE MALNUTRITION (SAM)

1. Introduction

Malnutrition remains one of the most common causes of morbidity and mortality among children. The high case fatality rates among severely malnourished children can be reduced by using standardized and easily implementable protocols.

2. Criteria for hospital admission

- Weight for height/length <-3 z score of median of WHO child growth standards or
- Mid-arm circumference < 11.5 cm or
- Presence of nutritional (bipedal) edema.

If weight-for-height or weight-for-length cannot be measured, use the clinical signs for visible severe wasting

3. Assessment of severely malnourished child

A good history and physical examination is required for deciding the treatment but always start the emergency treatment first. The details of history and examination can be recorded later.



Table 1: Severe Acute Malnutrition

History	Examination
<ul style="list-style-type: none"> • Recent intake of food and fluids • Usual diet (before the current illness) • Breastfeeding • Duration and frequency of diarrhoea and vomiting • Type of diarrhoea (watery/bloody) • Loss of appetite • Family circumstances (to understand the child's social background) • Chronic cough • Contact with tuberculosis • Recent contact with measles • Known or suspected HIV infection. • Immunizations 	<ul style="list-style-type: none"> • Anthropometry- weight, height/ length, mid arm circumference • Oedema • Pulse, respiratory rate • Signs of dehydration • Shock (cold hands, slow capillary refill, weak and rapid pulse) • Severe palmar pallor • Eye signs of vitamin A deficiency: <ul style="list-style-type: none"> - Dry conjunctiva or cornea, - Bitot's spots - Corneal ulceration - Keratomalacia • Localizing signs of infection, including ear and throat infections, skin infection or pneumonia- • Fever (temperature $\geq 37.5^{\circ} \text{C}$ or $\geq 99.5^{\circ} \text{F}$) or hypothermia (axillary temperature $< 35.0^{\circ} \text{C}$ or $< 95.0^{\circ} \text{F}$) • Mouth ulcers • Skin changes of kwashiorkor: <ul style="list-style-type: none"> - Hypo or hyperpigmentation - Desquamation - Ulceration (spreading over limbs, thighs, genitalia, groin, and behind the ears) - Exudative lesions (resembling severe burns) often with secondary infection (including Candida)

4. Laboratory Tests

- Hemoglobin or packed cell volume in children with severe palmar pallor.
- Blood sugar.
- Serum electrolytes (sodium, potassium)
- Screening for infections:
 - Total and differential leukocyte count, blood culture (If possible)
 - Urine routine examination
 - Urine culture
 - Chest x-ray

5. Organization of care

On admission, the child with severe malnutrition should be separated from infectious children and kept in a warm area (25–30°C, with no draughts), and constantly monitored. Washing should be kept to a minimum, after which the child should be dried immediately.

Facilities and sufficient staff should be available to ensure correct preparation of appropriate feeds, and to

carry out regular feeding during the day and night. Accurate weighing machines are needed, and records should be kept of the feeds given and the child's weight so that progress can be monitored.

6. Providing general treatment for malnutrition

There are 10 essential steps in two phases: an initial stabilization phase and a longer rehabilitation phase.

Table-2: Treatment for Malnutrition:

	Stabilization		Rehabilitation
	Days 1-2	Days 3-7	Weeks 2-6
1. Hypoglycemia	→		
2. Hypothermia	→		
3. Dehydration	→		
4. Electrolytes	→		
5. Infection	→		
6. Micronutrients	No iron		with iron
7. Initiate feeding	→		
8. Catch-up growth	→		
9. Sensory stimulation	→		
10. Prepare for follow-up	→		



6.1 Important things not to do and why?

- Do not give I/V fluids routinely. I/V fluids can easily cause fluid overload and heart failure. Only give I/V fluids to children with signs of shock.
- Do not give diuretics to treat oedema. The oedema will go away with proper feeding. Giving diuretics will worsen child's electrolyte imbalance and may cause death.
- Do not give high protein formula. Almost all severely malnourished children have infections, impaired liver and intestinal function. Because of these problems, they are unable to tolerate the usual amount of dietary protein.
- Do not give iron during the initial feeding phase. Add iron only after the child has been on catch-up formula for 2 days (usually during week 2).
- Giving iron early in treatment has been associated

with free radical generation and may interfere with the body's immune mechanisms against proliferating bacteria.

6.2. Treat hypoglycaemia

- If the child is lethargic, unconscious, or convulsing, give IV 10% glucose 5 ml/kg followed by 50 ml of 10% glucose or sucrose by NG tube. If IV dose cannot be given immediately, give the NG dose first. Give appropriate antibiotics and start feeding as soon as possible.
- If not lethargic, unconscious, or convulsing, give the first feed of starter formula (75 calories/100ml), if it is quickly available and then continue with 2 hourly feeds.

- If the first feed is not quickly available give 50 ml of 10% glucose or sugar solution (4 rounded teaspoon of sugar in 200 ml or one cup of water) orally or by nasogastric tube, followed by the first feed as soon as possible.
- Give 2-hourly feeds, day and night, at least for the first day.
- Give appropriate antibiotics.
- Keep the baby warm and check temperature.
- Prevent hypoglycaemia/Begin Starter Formula
- Feed 2 hourly, starting immediately or, if necessary, rehydrate first. Continue feeding throughout the night.

6.3. Infection

6.3.1 Presume and treat infection

Assume all children with severe malnutrition admitted in a hospital have an infection and give broad spectrum antibiotics. If a specific infection is identified (such as Shigella), give specific appropriate antibiotics according to condition identified. Hypoglycaemia and hypothermia are often signs of severe infection.

6.3.2 Select antibiotics and prescribe regimen

Select antibiotics as shown in the chart below.

Table 3: Antibiotic Treatment

Status	Antibiotic
All admitted cases	<ul style="list-style-type: none"> • Inj. Ampicillin 50 mg/kg/dose 6 hrly and Inj. Gentamicin 7.5 mg/kg once a day for 7 days • Add Inj. Cloxacillin 50 mg/kg/dose 6 hrly if staphylococcal infection is suspected • Revise therapy based on sensitivity report
For septic shock or worsening/ no improvement in initial hours	<ul style="list-style-type: none"> • IV Cefotaxime 50 mg/kg/dose 6 hrly or Inj. Ceftriaxone 50 mg/kg/dose 12 hrly plus Inj. Amikacin 15 mg/kg/once a day
Meningitis	<ul style="list-style-type: none"> • IV Cefotaxime 50 mg/kg/dose 6 hrly or Inj. Ceftriaxone 50 mg/kg/dose 12 hrly plus Inj. Amikacin 15 mg/kg/once a day
Dysentery	<ul style="list-style-type: none"> • Inj. Ceftriaxone 100 mg/kg once a day for 5 days

6.3.3 Duration of antibiotic therapy depends on the diagnosis:

Suspicion of clinical sepsis: at least 7 days
Culture positive sepsis: 10-14 days
Meningitis: at least 14-21 days

Deep seated infections like arthritis and osteomyelitis: at least 4 weeks

6.4. Treat Associated Conditions

- Give Anti-malarials, if blood smear positive for malaria parasites.
- Start ATT if Tuberculosis is diagnosed or strongly suspected (Mantoux Test and X-ray chest).
- Suspect HIV if he/she has also other problems like persistent diarrhoea, oral thrush, pneumonia, parotid swelling or generalized lymphadenopathy
- Investigate and follow HIV guidelines.
- Severe anaemia: Give whole blood or packed cell transfusion if Hb is < 4g/dl or Hb is 4-6 g/dl and child has respiratory distress. Give 10 ml/kg slowly over 4-6 hours and give Inj. Frusemide 1 mg/kg at the start of the transfusion. Do not repeat blood transfusion within 4 days.
- If eye problems (Keratomalacia) due to vitamin A deficiency, in addition to vitamin A doses instill Ciprofloxacin eye drops 2-3 hourly and Atropine eye drops 3 times a day for 7-10 days. Also cover the eyes with pad and bandage.

- Skin lesions: Bathe or soak the affected areas for 10 min in 1% potassium permanganate solution and apply gentian violet or nystatin cream if available to skin sores and any barrier cream (zinc cream) to the raw areas.
- Persistent diarrhoea: Diarrhoea is common in severe malnutrition but with cautious refeeding, it should subside during the first week. In the rehabilitation phase, the poorly formed loose stools are not a cause for concern, provided the child's weight gain is satisfactory. If the child has persistent diarrhoea, screen for non-intestinal infections and treat appropriately. Continue breast feeding and try to give feeds with low lactose initially and subsequently change to lactose free options if diarrhoea persists.

6.5. Micronutrients

6.5.1 Give oral vitamin A in a single dose.

- Vitamin A orally in single dose as given below:
 - < 6 months: 50,000 IU (if clinical signs of deficiency are present)
 - 6-12 months: 1 lakh IU
 - Older children: 2 lakh IU
 - Children < 8 kg irrespective of age should receive 1 lakh IU orally.
- Give half of the above dose if injectable (intramuscular) vitamin A needs to be given.
- Give same dose on Day 0,1 and 14 if there is clinical evidence of Vitamin A deficiency.

6.5.2 Other micronutrients should be given daily for at least 2 weeks:

- Multivitamin supplement (should contain vitamin A, C, D, E and B12 & not just vitamin B-complex): Recommended Daily Allowance.
- Folic acid: 5mg on day 1, then 1 mg/day.
- Zinc: 2mg/kg/day.
- Copper: 0.3 mg/kg/day (if separate preparation not available use Commercial preparation containing copper).
- When weight gain commences and there is no diarrhoea add 3 mg of iron/kg/day.

6.6. Initiate feeding

Essential features of initial feeding are:

- Start feeding as early as possible.
- Feed the child if alert and drinking even during rehydration.
- Give frequent and small nutrient rich feeds of low osmolarity and low lactose.
- Offer 130 ml/kg/day of liquids (100 ml/kg/day if child has severe oedema), 80- 100 Kcal/kg/day and 1-1.5 g/kg/day of proteins.
- Use nasogastric feeding till child takes orally 75% of all feeds.
- If child breastfed, continue breastfeeding but give the feed first.
- Ensure night feeds.

6.6.1. Starter Formula

Starter formula is to be used during initial management. It is started as soon as possible and continued for 2-7 days until the child is stabilized. Severely malnourished children cannot tolerate usual amounts of proteins and sodium at this stage, or high amounts of fat. They may die if given too much protein or sodium. Starter formula is specially made to meet the child's needs without overwhelming the body's systems in the initial stage of treatment which provides 75 calories /100 ml and 0.9 gm of protein/100 ml.

6.6.2. Feed the child Starter formula orally, or by NG tube if necessary:

• Oral feeding

It is best to feed the child with a cup and spoon. Encourage the child to finish the feed. It takes skill to feed a very weak child, so nursing staff should do this task first and mother may help with feeding later when child becomes stronger. Encourage breastfeeding on demand between starter formula feeds.

• Nasogastric feeding

It may be necessary to use a NG tube if child is very weak. Use a NG tube if the child does not take 75% of the feed for 2-3 consecutive feeds.

Remove the NG tube when the child takes:

- 75% of the day's amount orally; or
- Two consecutive feeds fully by mouth.

6.6.3. Record intake and output on a 24-Hour food intake chart

Criteria for increasing volume/decreasing frequency of feeds

- If there is vomiting, significant diarrhoea, or poor appetite, continue 2-hrly feeds.
- If there is little or no vomiting, diarrhoea is less than before, and most feeds are consumed, change to 3-hrly feeds.

- After a day on 3-hrly feeds: If there is no vomiting, occasional diarrhoea, and most feeds are consumed, change to 4-hrly.

6.6.4. Monitoring

Monitor and record

- Amounts of feed offered and left over
- Stool frequency and consistency
- Vomiting
- Daily body weight

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Dasgupta R, Sinha D, Yumnam V. Rapid Survey of Wasting and Stunting in Children: What's New, What's Old and What's the Buzz? Indian Pediatrics. 2016 Jan;53(1):47-9.
2. De Onis M, Weise Prinzo Z. Managing Children with Severe Acute Malnutrition – What's New? A health policy perspective. Indian Pediatrics. 2014 Jan;51(1):17-8.



18. Fever with Rashes

1. Introduction:

Fevers associated with generalized skin eruptions/ rashes (exanthematous fevers) are common in childhood. These are often seen as epidemics in the periods of 'season change' like March-April and October-November when adenoviruses become active.

2. Differential Diagnosis of Exanthema

2.1 Macular and/or Papular rash

- a) Measles, Rubella, Erythema infectiosum, Roseola, Coxsackie virus, echovirus, CMV, Hepatitis B infections.
- b) Erythema multiforme due to Herpesvirus, Epstein Barr Virus, Adenovirus, Chlamydiae, Salmonella, Mycobacterium, Histoplasma and Coccidioides.

2.2. Nodular

- a) Fungal diseases, Atypical mycobacterial and pseudomonas infections.
- b) Erythema nodosum: Due to Streptococcus, Mycobacterium Tuberculosis and Lepae, Yersinia, Hepatitis C, Sarcoidosis, Drugs and inflammatory bowel diseases.
- c) Diffuse erythematous with peeling or desquamation: Scarlet fever, Toxic Shock Syndrome, Kawasaki Disease, Staphylococcal Scalded Skin Syndrome, Steven's Johnson Syndrome.

2.3. Vesiculo-bullous

Varicella, Herpesvirus, Coxsackie virus, Enterovirus, Meningococcal, Group A Streptococcal and Pseudomonas infections.

2.4. Petechial/purpuric

Epstein Barr virus, Echovirus 9, Cytomegalovirus, Rickettsia, malaria, Pneumococcal, Meningococcal and Listeria infections.

3. Measles

Measles is a communicable disease manifesting with fever, cough, coryza, lacrimation and Koplik spots in the pre-eruptive phase and a maculopapular rash starting on 4th or 5th day of illness. The rash heals leaving brawny pigmentation.

It is caused by a RNA virus classified as Morbillivirus, belonging to paramyxovirus family.

Clinical Features: Incubation period is 8-12 days.

Prodromal Phase: The onset is acute with moderate elevation of temperature, dry hacking cough, running of nose, sneezing, redness of the eyes and excessive lacrimation.

3.1. Signs & Symptoms:

Typical measly look in young babies is due to conjunctivitis, stomatitis and rhinitis due to viral rash. The first place for the rash to be seen is around opening of parotid ducts in the mouth/inner cheeks (Koplik's spots). Rash appears on face and progresses to appear on trunk and limbs over the following three to four days. Drying of rash and peeling of skin appears in the same sequence in next 4- 5 days.



Figure 18.1: Various types of Rashes

3.2. Investigation: HB, TLC, DLC, X-Ray Chest, Measles antibody test.

3.3. Complication:

Acute precipitation of Vitamin A deficiency in a child with measles can occur and –hence corneal ulceration is a complication to watch out for and to prevent. All children with measles should routinely receive vitamin A orally – 2 lakh units each on day 1, 2 and 14 (for infants the dose is half)

Due to depressed immunity because of measles, child can catch infections like diarrhoea, otitis media, pneumonia and TB. If the child continues to get fever even after the rash is dry, bacterial pneumonia should be suspected. Continued low fever and lack of appetite should make one suspect TB infection.

Extra-feeding during convalescence and when the child gets well is important to prevent malnutrition after measles.

3.4. Management:

Symptomatic treatment should be given Paracetamol 10 to 15 mg per kg for fever and Antihistaminic (Cetirizine)
Children 6-12 months: 2.5mg once a day,
Children 12-23 months 2.5mg twice a day,
Children above 6 years to adults: 5-10 mg/day, as a single dose or divided into 2 doses, for itching and rhinitis. Advice continued feeding. Keep baby clean by sponging or quick clean bath.

Treatment with antibiotics (Amoxicillin 30 mg per kg per day in 2 to 3 divided doses for 5 days) for superadded bacterial infections when detected.

3.5. Prevention:

Measles immunization as routine immunization should be promoted at 9 months and 2nd dose along with DPT Booster at 18-24 months.

Immunization given early to contact of measles case can prevent severe disease in the contact.

4. Chicken-pox:

Chicken pox is highly contagious disease, presenting with sudden onset of low fever, mild constitutional symptoms, a centripetal, pleomorphic rash appearing on the first day of the illness.

It is caused by Varicella Zoster virus belonging to Herpes Virus family.

Clinical Features: The incubation period is usually between 14 to 16 days (with a range of 11 to 21 days)

4.1. Signs & Symptoms:

Symptom of chicken-pox is characteristic rash. The rash appears in several "crops" of macules (red spots) quickly turning into watery vesicles and pustules is often more severe in older children and adolescents. over the next 2 to 3 days and then drying after scab formation over next 5 days.



Figure 18.2: Chicken Pox Rash

Due to multiple 'crops', appearing over a week or so, one can observe all types of lesions simultaneously on a patient's body. The rash is more on covered areas like trunk of body, and can appear inside mouth. Conjunctivae. Vagina leading to irritation symptoms. Chicken-pox patient is infective till all scabs are formed. Itching is prominent and scratching can lead to super added staphylococcal infection.



Figure 18.3: Vesicles & umbilication in chicken pox

4.2. Investigation: Clinically Diagnosed

4.3. Complication: Aspirin given to a child with chicken-pox infection may precipitate acute fatty infiltration of liver. Liver failure and encephalopathy (Reye's syndrome). Reyes' syndrome is to be suspected if there is disproportionate vomiting, rapid breathing (in the absence of chest signs suggestive of pneumonia) and altered sensorium. The patient should be referred for specialist care, where respirator is available; as this is a very serious condition with high mortality rate.

4.4. Management:

Symptomatic treatment with paracetamol 10 to 15 mg per kg per day for 5 days and anti-histaminic (Cetirizine) for 5 days is advised. Daily bath is also advised.

Acyclovir in the dose of 20mg/kg/dose that is 80 mg/kg/day in 4 divided doses for 5 days start treatment within the first 24 hours of rash onset can be given to immunocompetent patients.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Hay Jr. WW, Levin MJ, Deterding RR, Abzug MJ, Current Diagnosis and Treatment: Pediatrics, 22nd Edition, McGraw-Hill Education, 2014



19. Enteric Fever / Typhoid

1. Introduction: Although Enteric fever is still a common infection its presentation has changed partly due to vaccination and partly due to institution of antibiotics early in the course of illness.

Diagnosis is problematic since blood cultures may not reveal much and Widal test is positive only after 1 week and has many false positive and false negative results. Bone marrow culture is more informative, but not practical.

Hence possibility of enteric fever must be borne in mind while treating any pyrexia, which shows toxicity, and signs suggestive of enteric like confused sensorium, (no meningeal focal neurological signs).

2. Clinical Manifestations:

Classical presentations are becoming rare. Common specific manifestations are -

- Relative bradycardia compared to fever.
- Tumid tender abdomen (abdomen feels like balloon filled with water)
- Cloudy sensorium.
- Maculopapular erythematous rash on abdomen blanching on pressure (Rose spots)
- Centrally coated tongue.
- Soft spleen - 1-2 cm tender.
- Continuous fever, not touching the baseline. (If antipyretics are not given)
- Pea soup diarrhea, nausea, vomiting.
- Headache, myalgia, anorexia, malaise.

Enteric fever in children is more severe as compared to adults.



Fig. 19.1: Rose spot in typhoid

3. Investigation:

HB, TLC, DLC, Widal Test (Rising Titers), Blood Culture.

Complication: Shock, enteric perforation, haemorrhage meningitis, myocarditis. Can complicate the illness.

4. Management:

4.1 Drug Treatment

Chloramphenicol -75 mg/kg/day x 14 to 21 days

Or

Amoxicillin 100mg/kg/day (with clavulanic acid) x 14 days

Or

Trimethoprim – Sulphamethaxazole 10 & 50 mg/kg/day are also used with some success.

4.2 Multi-drug resistance:

Multi-drug resistant typhoid fever has emerged and is difficult to treat. Following drugs are found to be useful:

Third generation cephalosporins –

Cefixime 20 mg /kg 1 day in 2 divided doses

or Ceftriaxone 50 mg/Kg per day

or Ciprofloxacin 20 mg/Kg per day,

or Ofloxacin 15 mg/Kg 1 day.

} 10-14 days

The antibiotics should be continued at least 5-7 days after effervescence.

4.3. Early institution of steroids

Dexamethasone 3 mg / Kg stat dose followed by 1 mg/kg 6 hourly for 48 hours improves the survival of patients in shock, myocarditis' CNS complication.

4.4 Intestinal perforation requires

broad spectrum antibiotics, platelet transfusions for severe thrombocytopenia with hemorrhages. This treatment demands referral to higher level

5. Prevention:

2 types of vaccine are available in commercial market.

- An oral, live- attenuated preparation of the Ty21a strain of S. Typhi
- Vi capsular polysaccharide for 2 years and above.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Hay Jr. WW, Levin MJ, Deterding RR, Abzug MJ, Current Diagnosis and Treatment: Pediatrics, 22nd Edition, McGraw-Hill Education, 2014



20. ACUTE MENINGOENCEPHALITIS

1. Introduction

Acute meningoencephalitis is an acute inflammatory process involving meninges and brain tissue, due to infectious causes. The common etiological agents are viruses and bacteria. Children of any age may be affected.

2. Clinical Manifestations

Fever, headache, vomiting, irritability altered state of consciousness, signs of meningeal irritation and seizures.

3. Investigations

CSF examination differentiates the viral from bacterial cause of acute meningoencephalitis



Figure 20.1: Signs of meningitis

Table 1: CSF findings in meningoencephalitis

	Pressure (mmH ₂ O)	Leukocytosis (mm ³)	Protein (mg/dl)	Glucose (mg/dl)
Normal	50-80	<5, >75% lymphocytes	20-45	>50 or 75% serum glucose
Acute bacterial Meningitis	Usually Elevated (100-300)	100-10,000 PMN's* predominate	100-500	Decreased (<40)
Acute viral Meningoencephalitis	Normal or elevated	Rarely >1000 PMN's early but lymphocytes predominate in the most of the course	50-200	Normal rarely decreased
Tubercular Meningoencephalitis	Usually elevated	100-500 PMN's early but later lymphocytes predominate	100-3000	<50

*PMN's = Polymorphonuclear leucocytes

4. Treatment

4.1 Supportive treatment

Supportive treatment is the mainstay of therapy and is started immediately.

- Maintain airway, breathing and circulation.
- Control of seizures with IV injection of Diazepam 0.2 to 0.4 mg/kg stat followed by Inj. Phenytoin 10-20 mg/kg stat followed by 5 mg/kg/day in divided doses.

- Increased intracranial tension is treated by proper positioning of patient with head elevated at 15-30° position, fluid restriction to 2/3rd of maintenance, 20% Mannitol 5 ml/kg over 10-15 min followed by 3 ml/kg every 6 hourly for 48 hours and then SOS. Or Acetazolamide 50-75 mg/kg/day in 3 divided doses through feeding tube Or Glycerine 1 ml/kg/day through feeding tube may be added, if increased intracranial tension persists.

- d. Fever is controlled as given in section on fever. (Caution: Never give aspirin).
- e. The intravenous fluid at two-thirds of the maintenance requirement initially. The electrolyte concentration of the blood is monitored very closely. Any imbalance is treated promptly. Fluid restriction is not done, if patient is dehydrated or is in shock.
- f. Feeding: Initially the patient is kept nil orally for first 24-48 hours. Later on the feeding is guided by the level of sensorium. A tube feeding is helpful for feeding as well as for giving medicines.

4.2 Specific treatment

Until a bacterial cause is excluded, parenteral antibiotic therapy should be administered. The choice of antibiotics depends upon age of the patient and prevalence of organism in the area.

4.2.1 Age 0-3 months

- Inj. Cefotaxime 200 mg/kg/day IV in 4 divided doses for 14 days.
- Inj. Ampicillin 300 mg/kg/day IV in 4 divided doses for 14 days.

4.2.2 Age 3 months-12 years

- Inj. Ceftriaxone 100 mg/kg/day IV over 30-60 minutes in 2 divided doses for 10 days
Or Inj. Cefotaxime 200 mg/kg/day IV in 3 divided doses for 10 days
Or Inj. Ampicillin 300 mg/kg/day IV in 4 divided doses for 10 days

- Inj. Chloramphenicol 100 mg/kg/day in 4 divided doses for 10 days
- If Meningococci is suspected/isolated, Inj Penicillin G 300,000-400,000 IU/kg/day in 4 divided doses for 7-10 days.

4.2.3. Treatment Viral meningoencephalitis

- Herpes simplex virus (generally diagnosed by focal encephalitis or CT scan):
Inj. Acyclovir 30 mg/kg/day in 3 divided doses for 14-21 days.
- Non-HSV viral encephalitis is treated by supportive therapy only.
- Lumbar puncture is repeated at 48 hours to see the response. However, if the patient is improving well, a repeat lumbar puncture may not be necessary.

5. Advice at discharge

- Regular follow-up for neurological assessment including deafness is advised.
- Anticonvulsant therapy to be continued, if seizures are recurrent during course of meningitis. Children with sequelae would require assessment of handicap and multidisciplinary management.
- Occupational / physiotherapy may be taught during hospital stay itself.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. McInerney TK, Adam HM, Campbell D, Kamat DK, Kelleher KJ, eds. American Academy of Pediatrics (AAP) Textbook of Pediatric Primary Care. 5th Ed. Elk Grove Village; 2009



21. TUBERCULOUS MENINGITIS

1. Introduction

Tuberculous meningitis is the inflammation of meninges due to lymphohaematogenous spread of the primary infection of tuberculosis to the meninges, found in about 0.3% of untreated primary infection in children. It is the most dangerous form of extra-pulmonary tuberculosis. 70% of the cases are found in children less than 5 years of age.

2. Clinical Manifestations

The clinical progression of tubercular meningitis (TBM) may be rapid or gradual. The signs and symptoms progress slowly over several weeks and can be divided into three stages.

2.1 Stages:

- The **1st stage**, which typically lasts 1-2 weeks, is characterized by non-specific symptoms, such as fever, headache, irritability, drowsiness and malaise. Focal neurologic signs are absent.
- The **2nd stage** usually begins more abruptly. The most common features are lethargy, neck-rigidity, seizures, positive Kernig or Brudzinski signs, Hypertonia, vomiting, cranial nerve palsies and other focal neurologic signs.

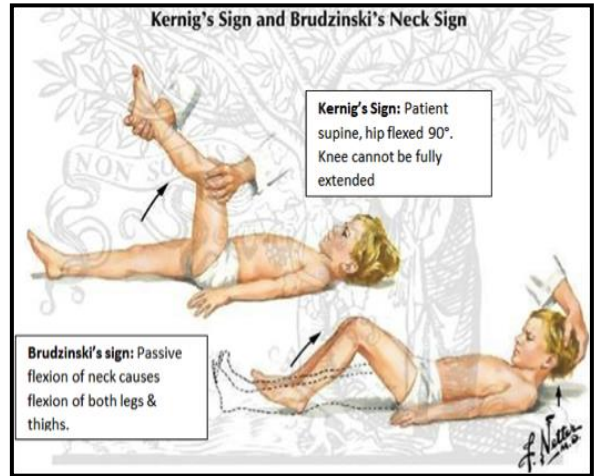


Figure 21.2: Kernig's sign & Brudzinski's Sign

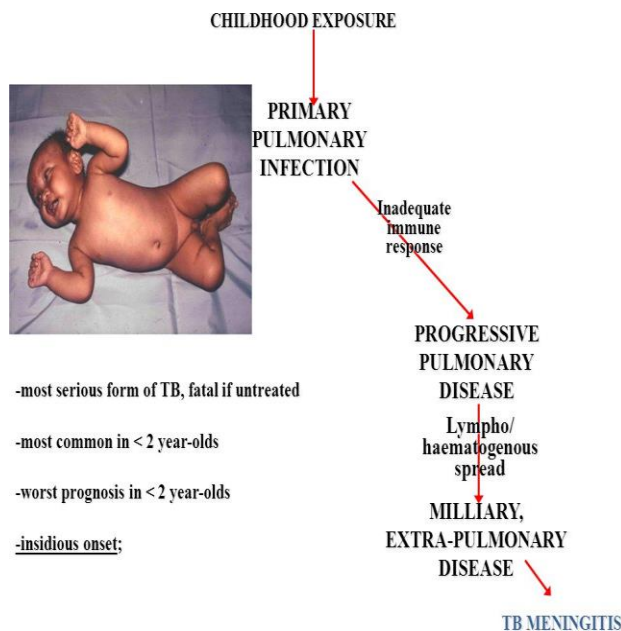
- The **3rd stage** is marked by coma, hemiplegia or paraplegia, hypertension, decerebrate posturing, deterioration of vital signs, and eventually, death.

3. Complications:

Survivors may have motor deficits, cranial nerve deficits, mental retardation, learning disabilities, seizures, hydrocephalus, blindness, deafness and diabetes insipidus.

4. Investigations

- The diagnosis is made by analysis of CSF on lumbar puncture, which shows lymphocytic leukocytosis with elevated protein and a low sugar (for details see Table 1 of Chapter 20. Acute Meningoencephalitis).
- Demonstration of AFB in CSF confirms the diagnosis, but the yield is very poor. Culture of CSF shows growth of *M. tuberculosis*, takes too much time.
- Positive tuberculin skin test corroborates the diagnosis but may be negative in severely malnourished/disseminated disease.
- 20-50% of children have a normal chest radiograph others may show primary disease.
- CT scan or MRI of brain may be normal during early stages of the disease. Later, it can show exudates in the basal cisterns of brain, periventricular ooze and hydrocephalus. Some may show tuberculomas.



5. Treatment

Treatment consists of proper supportive care, including nonpharmacological treatment, specific anti-tubercular therapy, treatment of increased intracranial tension and, if required, surgical treatment.

5.1 Nonpharmacological

- Nutrition: After initial stabilization, nutritional rehabilitation should be done as given in section on protein energy malnutrition.
- Skin care and prevention of bedsores.
- Care of bowel and bladder.
- Physiotherapy and occupational therapy should be instituted early to prevent deformities and contractures.

5.2 Pharmacological

- Appropriate fluid therapy to correct dehydration due to frequent vomiting and decreased oral intake.
- Fluid restriction upto 3/4th or 2/3rd of maintenance.
- Treatment of raised intracranial tension
 - Inj. Dexamethasone: 0.15 mg/kg IV 6 hourly for 2 weeks followed by Tab.
 - Prednisolone 1.5 mg/kg/day orally through feeding tube for 4 weeks. This should be tapered over another 2 weeks. A total of 6 to 8 weeks of therapy with steroid is recommended.
 - Mannitol (20% solution) 1.5 to 2 g/kg or 8-10 ml/kg over 30-60 minutes. Repeated every 6-8 hours for 7 days. Lower doses (0.25 g/kg/dose) can also be tried. Or Glycerol 1 ml/kg/dose every 6-8 hours, diluted in orange juice or water, given through feeding tube. Or Tab. Acetazolamide 50 mg/kg/day in 3 divided doses for 2-3 weeks.

- Presence of seizures necessitates treatment with phenytoin or carbamazepine in appropriate doses (for details see section on Epilepsy in Chapter).
- Specific anti-tubercular therapy—as given in management of tuberculosis (see section on Tuberculosis).

5.3. Surgical treatment

Ventriculo-peritoneal shunt (VP shunt): TBM shows some degree of hydrocephalus by 4 weeks. Obstructive hydrocephalus should be shunted immediately. Non-obstructive hydrocephalus with increased intracranial pressure as shown by ventricular tap or CT scan will also be benefited by VP shunt. An early shunt is preferable.

5.4. Follow-up

- Patient should be kept under follow-up after discharge from the hospital and assessed for neurological deficit and features of increased intracranial pressure (ICP). One of the common causes of increased ICP is untreated hydrocephalus or blocked shunt.
- Check compliance to drugs and ensure that occupational therapy/physiotherapy is being continued.
- Assess physical, mental, visual and auditory handicap and take expert opinion for rehabilitation from other specialists.
- **Patient/parent education**
 - Seriousness of disease must be explained.
 - Contact survey should be done and any other member in the family found to have active TB should be counselled to attend TB clinic for therapy.
 - Need for compliance should be emphasized.
 - Drug toxicity and side effects must be explained.
 - Neurological deficits may appear even in a patient on therapy.

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013.



22. ACUTE RESPIRATORY INFECTION

1. Introduction

Acute Respiratory Infection (ARI) is an important cause of infant and child mortality and also the commonest cause of morbidity among children below five years. ARI is the infection of any part of respiratory tract, which includes cough, common cold, pharyngitis, pneumonia, laryngitis and ear infection.

Acute respiratory infections in children can involve upper respiratory tract (nose, throat) or lower respiratory tract (bronchi, lung). Lower respiratory tract infections (broadly termed as pneumonias) are a major cause of death of infants and children in India, accounting for about 30% under-five deaths. Timely treatment, based on well-researched algorithms can save most children with ARI. Majority of the cases of ARI have non-severe

disease and can be managed in the community with oral cotrimoxazole. Severe ARI cases require urgent referral to a facility where injectable antibiotic therapy and supportive care are available.

2. Diagnosis & management of ARI

Classification and management of ARI is based upon three important factors. These are age of the child, respiratory rate and danger sign like chest in-drawing.

2.1 Age of the child and respiratory rate

Children are classified into three age groups for ARI management. These age groups and criteria for fast breathing are given in table below:

Table 1: Cut off points for fast breathing

Sr.	Age group	Criteria for fast breathing
1	0 - 2 months	Respiratory rate more than 60 / minute
2	2 months to 1 year	Respiratory rate more than 50 / minute
3	1 year to 5 years	Respiratory rate more than 40 / minute

2.2 Chest in-drawing

In normal child, during respiration, chest expands when the child breathes in and compresses when the child breathes out. In children with severe pneumonia, chest moves in when the child breathes in. This is called chest in-drawing.

2.3 Pneumonia in age group 0-2 months

Important aspects about pneumonia in age group 0-2 months are as below:

- In these children, there are no usual features of pneumonia like fever, cough, etc.
- Child may have only fast breathing and/or chest in-drawing.
- Pneumonia in child less than 2 months of age is always severe & mortality is high

Such cases should be treated in the facility where specialist & ICU are available.

Classification

- No pneumonia
- Severe pneumonia/Very severe illness

2.3.1 No pneumonia

If respiratory rate of child is less than 60/minute, then the child is classified as 'No Pneumonia'.

- Give Paracetamol ¼ tablet (500mg) if there is fever and demonstrate to the mother how to clean nose with normal saline drops.
- Advise mother to continue breast-feeding.
- Inform mother about danger signs of pneumonia, e.g. breathlessness (rapid movement of abdomen), inability to drink, excessive drowsiness, hypothermia, high fever etc.
- Ask mother to immediately bring the child back to health facility, if she observes any of the signs mentioned above.

2.3.2 Severe pneumonia/Very severe illness

If child below 2 months presents with fast breathing (RR > 60/minute) and/or chest in-drawing

Plus

One or more of the following signs

- Inability to drink
- Excessive drowsiness
- Stridor, wheeze
- Cyanosis

- Hypothermia
- Convulsions

Such child should be referred to specialist.

In case referral cannot be executed, treat the child as below

Table 2: Treatment of Pneumonia

Antibiotic	Dosage	Frequency		Route
		Age < 7 days	Age > 7 days- 2 months	
Inj. Benzyl penicillin OR	50,000 IU/kg/dose	12 hourly	6 hourly	IV/IM
Inj. Ampicillin AND	50 mg/kg/dose	12 hourly	8 hourly	IV/IM
Inj. Gentamycin	2.5 mg/kg/dose	12 hourly	8 hourly	IV/IM

2.4. Pneumonia in age group 2 months to 5 years

Children with ARI of 2 months to 5 years of age are classified into four groups as follows -

- No pneumonia
- Pneumonia
- Severe pneumonia
- Very severe illness

2.4.1 No pneumonia

When respiratory rate is normal and no sign of chest in-drawing is seen child is diagnosed as 'no pneumonia'. These children should be treated at home & observed for appearance of danger signs (increase in RR and chest in-drawing) by mother. Following points should be advised to mother -

- Continue breast-feeding if child is breast-fed.
- Give plenty of oral fluids including water or ORS to child.
- If fever is present, give Tab. Paracetamol(5 to 10 mg/kg).
- Teach mother how to clean nose with saline water. This will clear airway of child and improve breast-feeding.
- In community, many home remedies are used for ARI. One of the best among them is honey & ginger. Advise mother to give honey and ginger to child.
- Inform mother about danger signs of pneumonia, e.g. breathlessness, inability to drink, excessive drowsiness, hypothermia, high fever etc.
- Advise her to bring child back to health facility if she observes any of the danger sign.

2.4.2 Pneumonia

If child has fast breathing (RR: 2-12 months > 50/ min. & 1-5 years > 40/ min.) and no chest in-drawing, diagnose child as Pneumonia **Treatment of pneumonia**

- Give child Cotrimoxazole for 48 hrs.
Treatment schedule for pediatric cotrimoxazole is -
2-12 months: 2 tablets twice daily or syrup 5 ml. twice daily
1-5 years: 3 tablets twice daily or syrup 7.5 ml. twice daily.
Pediatric tablet contains Sulfamethoxazole 100 mg & Trimethoprim 20 mg.
Syrup 5 ml. contains Sulfamethoxazole 200 mg and Trimethoprim 40 mg.
- If fever is present, give Tab. Paracetamol. (5 to 10 mg./kg)
- Teach mother how to clean nose with normal saline. This will clear airway of child and improve breast-feeding.
- Keep baby warm by covering with warm clothes and keeping in lap.
- Assess child after 48 hrs:
 - If improvement - Continue CTZ for 3 Days
 - Child has Chest in-drawing and RR may be fast - complete 5-day course
 - If no improvement or child gets deteriorated refer child to specialist.

2.4.3 Severe Pneumonia

Child with chest in-drawing & having RR normal or fast is diagnosed as severe pneumonia and should be treated by specialist. Give oxygen if child has cyanosis till the child is referred.

Treatment

- a. Treat for first 48 hours by

Table 3: Treatment of Severe Pneumonia

Antibiotic	Dosage	Interval	Route
Inj. Benzyl penicillin OR	50,000 IU/kg/dose	6 hourly	IM
Inj. Ampicillin OR	50 mg/kg/dose	6 hourly	IM
Inj. Chloramphenicol	25 mg/kg/dose	6 hourly	IM

b. Assess after 48 hrs

i. If improvement, continue for next 3 days as below

Table 4: Further management of Severe pneumonia

Antibiotic	Dosage	Interval	Route
Inj. Procaine penicillin OR	50,000 IU/kg (Max 4 lac units)	Once	IM
Tab Ampicillin OR	50 mg/kg/dose	6 hourly	Oral
Tab Chloramphenicol	25 mg/kg/dose	6 hourly	Oral

ii. If no improvement, change antibiotic as below

- If Ampicillin was given earlier then change to Inj. Chloramphenicol
- If Chloramphenicol was given earlier change to Inj. Cloxacillin 25 mg/kg/dose 6 hourly IM + Gentamycin 2.5 mg/kg/dose 8 hourly IM.

iii. Important aspects of antibiotic treatment

- Treatment with antibiotics should be continued for at least five days.
- Continue treatment for at least 3 days after child recovers.
- If Cloxacillin & Gentamycin are started continue for three weeks.

iv. In addition to this give following

- Administer Oxygen if required
- Continue breast feeding during illness
- If fever, give tablet or syrup Paracetamol
- If wheeze, treat by using bronchodilator
- Give plenty of oral fluids.

2.4.4 Very severe illness

a) Child has:

Chest in-drawing and RR may be fast or normal

Plus, One or more of following signs -

- Inability to drink
- Excessive drowsiness
- Stridor, wheeze
- Cyanosis
- Hypothermia
- Convulsions

Diagnose the child as suffering from very severe illness & refer the child to specialist having facility of intensive care unit.

3. Treatment of childhood community-acquired pneumonia

The management of pneumonia is guided by the severity of the disease as listed in Table below:

3.1 Classification:

Table 5: Signs & symptoms

Sign or symptom	Classification	Treatment
<ul style="list-style-type: none"> • Central cyanosis • Severe respiratory distress • Not able to drink due to respiratory distress 	Very severe pneumonia	<ul style="list-style-type: none"> • Admit to hospital • Manage the airway • Give oxygen • Give recommended antibiotic • Treat high fever if present
<ul style="list-style-type: none"> • Chest in drawing 	Severe pneumonia	<ul style="list-style-type: none"> • Admit to hospital • Give recommended antibiotic • Treat high fever if present
<ul style="list-style-type: none"> • Fast breathing 2 months to 12 months: ≥ 50 breaths/min 12 months to 5 years: ≥ 40 breaths/min • Definite crackles on auscultation 	Pneumonia	<ul style="list-style-type: none"> • Give appropriate antibiotic for 5 days • Soothe the throat and relieve cough with a safe remedy • Treat high fever if present

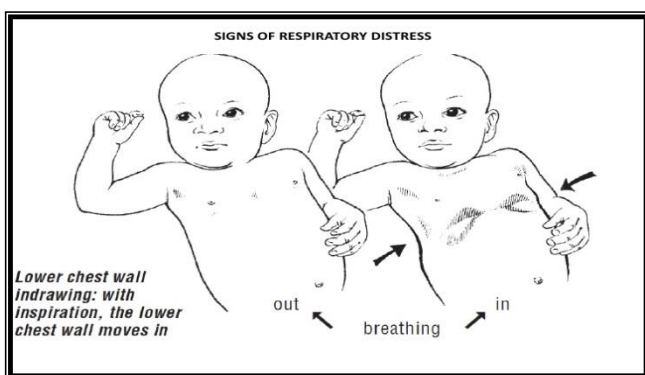


Figure 22.1 Signs of Respiratory distress

3.2 Treatment of very severe illness

3.2.1 Admit the child in hospital

3.2.2 Obtain a radiograph of the chest if facilities are available for the same. Radiography in very severe pneumonia is required at admission to assess the extent of disease and to rule out presence of pneumothorax or effusion. In case of severe distress, stabilize and oxygenate the child before sending for radiograph.

3.2.3 Give antibiotics

a) Give Injectable Ampicillin (50 mg/kg IM/IV every 6 hours) and Gentamicin (7.5 mg/kg IM/IV once a day). If the child responds well, discharge after 5 days to continue treatment

at home with oral Amoxicillin (15 mg/kg per dose three times a day) plus IM Gentamicin (7.5 mg/kg IM/IV once a day) daily for a further 5 days.

b) Alternatively, give Injectable Chloramphenicol (25 mg/kg IM or IV every 8 hours) until the child has improved. Then continue the same drug orally in the same dose for 3 times a day for a total course of 10 days.

c) If the child does not improve by 48 hours to any one of these treatments, reassess for complications and switch to Injection Ceftriaxone (80 mg/kg IM or I/V once daily) for 10 days.

Staphylococcal pneumonia is suspected if:

- There is a rapid progression of the disease, or
- There is pneumatocele, or pneumothorax, or effusion on chest X-ray, or
- Child has large skin boils or abscess or infected scabies, or
- Post-measles pneumonia which is not responding within 48 hours to the initial therapy.

If staphylococcal pneumonia is suspected, add Inj Cloxacillin (50mg/kg/dose, every 6 hourly) to any of the above choice of antibiotics.

When the child improves, continue Cloxacillin orally 4 times a day for a total course of 3 weeks at least. Children with complicated pneumonia (Empyema) need longer therapy for 4-6 weeks.

3.2.4 Give Oxygen

- Where pulse oximeter is available, use oxygen saturation of the blood (SaO₂) to guide oxygen therapy. Maintain SaO₂ ≥ 92%. Continue with oxygen until the signs of hypoxia (such as severe lower chest wall in-drawing or breathing rate of ≥ 70/min) are no longer present.

3.2.5 Give supportive care

- Ensure that the child receives daily maintenance fluids appropriate to child's age. Encourage breastfeeding and oral fluids once the distress settles and the child is able to feed.
- If the child has fever (≥38.5°C) which appears to be causing distress, give oral Paracetamol (15mg/kg/dose).
- If wheeze is present, give a rapid-acting bronchodilator (as described in the next section).
- Remove any thick secretions in the nose/throat, which the child cannot clear, by gentle suction.

3.2.6 Monitor the child

The child should be checked by nurses at least every 3 hours and by a doctor at least twice a day. Monitor for signs of improvement. A patient who is improving on treatment should have:

- An improvement in the respiratory rate.
- Less in-drawing of the lower chest wall.
- Less fever, and /or
- Improved ability to eat and drink.

3.2.7 Watch for complications

If the child has not improved after two days, or if the child's condition has worsened, look for complications or other diagnoses. If possible, obtain a repeat chest X-ray. Consider transfer to a higher facility in case of poor response or deterioration despite second-line therapy.

3.2.8 Monitoring

The child should be checked by nurses at least every 6 hours and by a doctor at least once a day. Monitor for signs of improvement as discussed above.

Table 6: Summary of management of ARI cases.

No.	Type	Who can manage	Where can be managed
Age group 2 months to five years			
1	No pneumonia	ASHA Anganwadi Worker, MPW	Home management
2	Pneumonia	MPW/HA, MO	Management at home or PHC
3	Severe pneumonia	PHC MO / specialist	PHC/ referral center
4	Very severe illness	Specialist	Specialist with ICU facility
Age group below 2 months			
<ul style="list-style-type: none"> • Child should be referred immediately after giving one dose of CTZ 			

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013.



23. BRONCHIAL ASTHAMA

1. Introduction:

Bronchial asthma is a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli. It manifests by widespread narrowing of the airways causing paroxysmal dyspnea, wheezing or cough. The diffuse obstruction to the airflow is reversible in a large majority of cases, either spontaneously or in response to treatment. Bronchial reactivity is a necessary component of asthma. Asthma is a result of multifactorial inheritance.

2. Signs & Symptoms:

- Recurrent cough
- Breathlessness
- Wheezing

3. Clinical classification

- Mild Intermittent
- Persistent
- Mild
- Moderate
- Severe

3.1. Mild Intermittent

- Symptoms < 2times a week and asymptomatic in between
- Nocturnal symptoms < 2 times /month

3.1.1 Treatment

Salbutamol inhaled (100 MCG/puff) 1-2 puffs as per requirement for children less than 2 years use face mask with spacer

OR

Salbutamol syrup/tablet

0.1-0.2 mg/kg/dose three times a day till symptoms subside

3.2. Mild Persistent

- Symptoms > 2times/week but ,1time/day
- Nocturnal symptoms > 2 times / month
- Asymptomatic in between exacerbations peak flow rate (PEFR) > 80%

3.2.1 Preference for Treatment

Preferred treatment: Inhaled steroids (low dose) 2 puffs twice / day

Less Preferred treatment: Montelukast, Theophylline, Cromoglycate and treatment of mild intermittent asthma

3.3. Moderate persistent

- Symptoms > 2 times/ week
- Nocturnal symptoms >1/week
- PEFR 60-80%

3.3.1 Treatment

- Inhaled steroid Medium dose
- If response not satisfactory
- Add inhaled LABA (Salmeterol/ Formoterol)/ Montelukast/ long acting theophylline

3.4. Severe Persistent

- Daily symptoms often severe
- Activity limited
- Growth affected
- Frequent Nocturnal symptoms
- Frequent hospitalization
- PEFR<60%

3.4.1 Treatment

- Inhaled steroids high dose
- Inhaled LABA /Montelukast / Long acting Theophylline
- If response not satisfactory
- Oral Prednisolone 2mg/kg/day in three divided doses & inhaled Salbutamol as required

3.4 Treatment Guidelines for acute exacerbation of Asthma

- O2 by mask (4-6 liters /min)
- Salbutamol MDI (100MCG/puff) with spacer and mask for < 2 years 4-8 puffs every 20 min x 3 OR Salbutamol nebulization 0.15mg/kg (min 2-5mg) diluted in 3 ml saline can be repeated 3 times every 20 min
- Inj. Adrenaline (1:1000) or Terbutaline (0.01mg/kg) SC may be repeated thrice every20 min if both above are not available AND
- Inj. Hydrocortisone hemisuccinate 10mg/kg/dose 4 times a day OR Inj. Dexamethasone0.2 mg /kg/dose
- This must be converted to oral prednisolone once patient is stable

- If response not satisfactory refer to higher center stat

IMP: Evaluate after 1 hour

- Good response

Send home: Salbutamol MDI+ Tab

Prednisolone 2mg/kg/day x 5-7 days

4. Investigation:

- Pulmonary function test
- Absolute Eosinophil count
- Chest X-ray
- Skin test & Allergy test



Figure 23.1 Metered dose inhaler

Table 1. Drugs used in the long-term control of asthma in children

Medication	Dosage form	Dosage
Formoterol	DPI: 12 µg per single-use capsule	1. Capsule every 12 years
Salmeterol	MDI: 25 µg per puff	1-2 puff every 12 hours
Fluticasone/salmeterol	DPI: 100, 250, or 500 µg of fluticasone with 50 µg of salmeterol	1 inhalation twice daily; dosage depends on severity of asthma
Cromolyn	MDI: 1 mg per puff Nebulizer solution: 20 mg per ampule	1-2 puffs 3 to 4 times daily 1 ampule 3 to 4 times daily
Nedocromil	MDI: 1.75 mg per puff	1-2 puffs 2 to 4 times daily
Montelukast	4 or 5 mg chewable tablets, 4 mg packet of oral granules, 10 mg tablets	Age 12-23 months: 4 mg oral granules at bedtime Age 2-5 years: 4 mg at bedtime
Zafirlukast	10 & 20 mg tablets	Age 7-11 years: 20 mg twice daily, 1 hour before or 2 hours after meals

Medication	Dosage form	Dosage
Theophylline	Liquids, sustained- release Tablets and capsules.	Loading dosage is 5 mg /kg per day Maintenance dose < 1 years: [(0.2 × age in weeks) + 5 mg] per kg day > 1 years: 16 mg per kg per day

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson’s Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Hay Jr. WW, Levin MJ, Deterding RR, Abzug MJ. Current Diagnosis and Treatment: Pediatrics. 22nd Edition. McGraw-Hill Education; 2014
3. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
4. Cloherty JP. Manual of Neonatal Care (SAE). 7th Edition. Wolter Kluwer; 2012

Further reading:

1. McInerny TK, Adam HM, Campbell D, Kamat DK, Kelleher KJ, eds. American Academy of Pediatrics (AAP) Textbook of Pediatric Primary Care. 5th Ed. Elk Grove Village; 2009



24. Breath holding spell

1. Introduction:

- A baby with crying followed by loss of consciousness, tonic posturing (features of cerebral anoxia).
- Cyanotic Breath holding spell more common form
- Usual age of onset is 6 months, peak by 2 years, abate by 5 years.
- After a shrill cry, forced expiration followed by apnea and cyanosis leading to loss of consciousness associated with clinical jerks or posturing.

2. Signs & Symptoms:

- Baby may be cyanosed (blue)\ Baby may be pale
- History of a predictable stubborn behavior, precipitating event like upsetting the infant
- History of painful stimulus or hit on head cyanosis leading to loss of consciousness associated with clinical jerks or posturing.

- Less common, Apnea followed by pallor, hypotonia and tonic seizure.

3. Complication:

- Local trauma
- Aspiration pneumonia.

4. Management:

- Examination to rule out other seizure mimicking conditions.
- Reassurance of parent that these are not seizures, EEG not required, inter-ictal EEG is normal.
- Should not reinforce this behavior, put the child in safe place, avoid cuddling.
- Iron supplements if anemia, delayed weaning etc. (3 mg/kg for 3 months.)
- Syrup Piracetam (40-100 mg /kg twice a day) may be used for few months if the breath holding spells are very frequent,
- Parental counseling to prevent reinforcement of this behavior.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Singh P, Seth A. Breath Holding Spells – A Tale of 50 Years. Indian pediatrics. 2015 Aug; 52(8): 695-6.
2. Sawires H, Botrous O. Placebo-controlled trial on the effect of piracetam on breath-holding spells. *Eur J Pediatr.* 2012 Jul;171(7):1063-7.



25. Bronchiolitis

1. Introduction:

- Viral in etiology (Respiratory syncytial virus)
- Most frequently in children < 12 months of age

2. Signs & Symptoms:

Initial URI symptoms followed by Increasing cough, Respiratory distress, Wheeze and feeding difficulty

3. Investigations:

CXR: Hyper inflated lungs with patchy infiltrates

4. Management:

4.1 Treatment

- Treatment of bronchiolitis is essentially symptomatic.

- Child should be treated in a humid atmosphere preferably in sitting position with head and neck elevated.
- Supportive measures such as oxygen by hood (10 litre/ minute) or by mask (5 litre / minute).
- IV fluids if child is not able to feed orally.
- Antibiotics have no role.
- Monitor - Respiratory Rate, Respiratory distress, pulse oximetry
- A trial dose of Nebulized salbutamol / epinephrine if wheezing is marked.
- As child improves wean off oxygen and increase oral feeds.
- If child develops severe respiratory distress, increasing hypoxemia, cyanosis or fatigue – ventilatory support may be required.

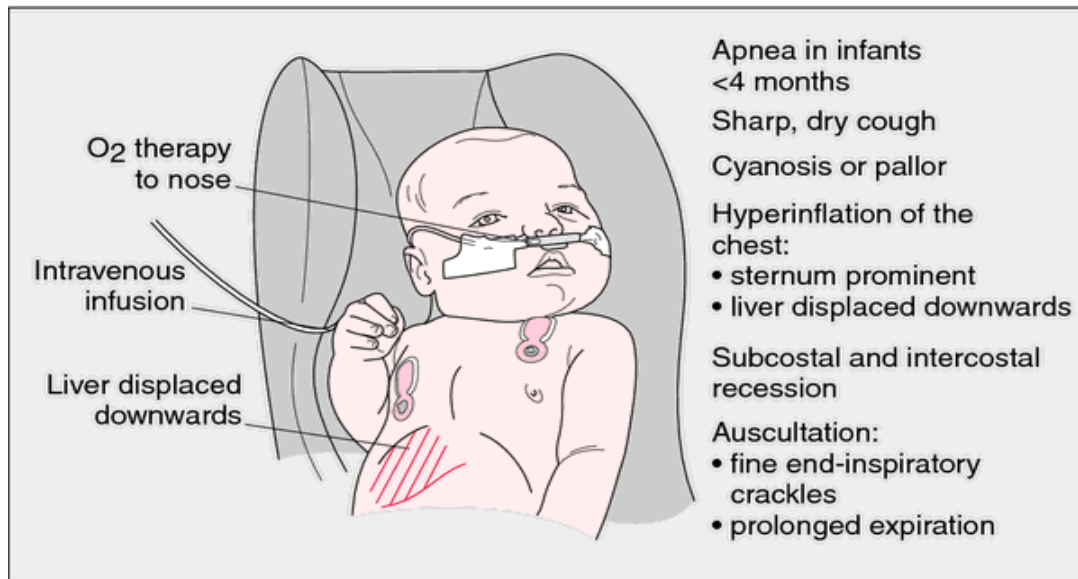


Figure 25.1: Clinical features of severe bronchiolitis in an infant. From Lissauer and Graham, 2002.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. McInerny TK, Adam HM, Campbell D, Kamat DK, Kelleher KJ, eds. American Academy of Pediatrics (AAP) Textbook of Pediatric Primary Care. 5th Ed. Elk Grove Village; 2009
2. Sharma S. Management of Bronchiolitis. *Indian Pediatrics*. 2014 Mar;51(3):235-6



26. Empyema

1. Introduction:

- Characterized by presence of pus or microorganisms in the pleural fluid
- Occurs as a complication of Pneumonia
- Influenza, Streptococcus Pyogenes, Staph aureus, Streptococcus Pneumoniae, Haemophilus are the common organisms.

2. Signs & Symptoms:

Common symptoms are

- Fever
- Chills
- Toxaemia
- Respiratory distress
- Grunt and
- Chest pain (pleuritic pain)

3. On examination

- Decreased chest expansion
- Diminished breath sounds
- Dullness on percussion on affected side and
- Mediastinal shift to opposite side

4. Investigations:

- Chest X-Ray: Obliteration of costo-phrenic angle; diffuse homogenous opacity.
- USG chest: size, site of effusion, adhesions or loculations can be made out.



Figure 26.1: Chest X-Ray in empyema

- Diagnostic thoracocentesis: usually in fifth intercostal space over mid-axillary line using a large bore needle.
- Pleural fluid for Gram stain, culture and sensitivity
- Pleural fluid pH, sugar is reduced and protein is elevated.

5. Management:

5.1 Treatment comprises of chest drain and antibiotics.

- Chest drainage using an intercostal drainage tube inserted in the region of maximal dullness (usually V or VI intercostal space in axillary region) and connecting to a sterile under water drainage bottle.
- Chest drainage is kept till the drainage decreases to < 25 ml/day and there is good lung expansion.
- If there is no chest expansion by clinical or radiological methods, surgical opinion is sought

5.2. Antibiotics

- Cloxacillin with Cefotaxime or Ceftriaxone is the first line antibiotic; switch over to oral antibiotics after child becomes afebrile and chest tube is removed.
- Total duration of 4 - 6 weeks of antibiotic therapy
 - Cloxacillin: 100 - 200 mg/kg/day in 4 div. doses
 - Cefotaxime: 150 - 200 mg/kg/day in 3 or 4 div. doses

5.3. Supportive care: Oxygen, good nutrition

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Ranjini EK, Agarwal I, Kirubakaran C. Pneumococcal subdural empyema in young infants. Indian Pediatrics. 2004 May;41(5):505-8



27. Approach to Fever

1. History:

- Type of fever
- Associated symptoms – chills / rigor, cough, sore throat, ear pain, urinary symptoms, bleeds etc.
- Previous illness and treatment.
- If any Feeding difficulty, respiratory distress.

2. Clinical Examination

- Check Temperature, Blood pressure, Pulse, Perfusion
- Skin: Rashes, Bleed, Cyanosis
- Eyes: Pallor, Icterus
- Mouth: Ulcer, Thrush
- Ear: Discharge, Redness, Tenderness
- Throat: Congestion, Tonsillitis
- CNS: Meningeal irritation, altered sensorium
- Abdominal examination: Hepatomegaly, Splenomegaly
- Respiratory System: Tachypnea, Retraction, Crepitation, Wheeze

3. Basic investigations in high risk group and fever beyond 5 days in low risk:

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013.
3. Engorn B, Flerlage J. The Harriet Lane Handbook. 20th Edition. Saunders; May 2014.

- Total count, differential count, peripheral smear, Platelet count
- Urine analysis, urine culture and sensitivity
- Blood culture and sensitivity
- Chest x-ray
- C-reactive protein
- Mantoux test
- CSF analysis if required
- Other investigations
 - Liver function test
 - Renal function test
 - USG abdomen
 - Blood for leptospirosis
 - Serology for dengue
 - Widal test
 - Bone marrow

4. Refer if:

- Fever with unconsciousness
- Fever with shock
- Severe respiratory diseases
- Bleeding diathesis
- Refractory seizures



28. ACUTE FLACCID PARALYSIS (AFP)

1. Definition:

- Acute flaccid paralysis is defined as sudden onset of weakness and floppiness in any part of the body in a child < 15 years of age or paralysis in a person of any age in whom polio is suspected [without any obvious cause (e.g. severe trauma or electrolyte imbalance like hypokalemia)].
- AFP is a notifiable disease and all cases must be reported immediately to Nodal Officer and District Surveillance Officer, NPSP Unit, Directorate of Family Welfare.
- India has shifted to the Virological system of case classification, i.e. within 90 days of paralysis onset; all cases should undergo final classification as confirmed polio, non-polio AFP or compatible with poliomyelitis.

2. Salient Features

- The paralysis is of acute onset (<4 weeks) and the affected limb(s) are flaccid (floppy or limp).
- If the AFP is due to polio, then sensation is never affected. Other important differentials to be considered in cases with AFP are detailed in Table-1.
- This includes possible illness due to Guillain-Barre syndrome, Transverse Myelitis, Traumatic Neuritis, viral infections caused by other Enteroviruses, toxins and Tumours.
- Isolated facial paralysis is also included.
- Pseudo paralysis due to pain in Congenital Syphilis, Osteomyelitis, Abscess, Scurvy, unrecognized trauma leading to contusions, slipped epiphysis or fractures, etc. can also mimic AFP.

3. Differential Diagnosis:

Table 1- Important differential diagnosis of AFP (adapted from Field Guide, MOHFW, GOI)

Signs and symptoms	Polio	GBS	Transverse myelitis	Traumatic or injection neuritis
Age	Most cases occur under 3 years of age	Usually above 2 years of age	Mostly above 4 years of age	No age limit
Progression of paralysis	24-48 h onset to full paralysis	Hours to days	Hours to 4 days	Hours to 4 days
Fever onset	High always present at onset of flaccid paralysis disappears the following day	Not common	Rare	Commonly present before, during and after paralysis
Flaccidity	Acute, asymmetrical, Proximal	Acute, symmetrical, Distal	Acute lower limbs symmetrical	Acute, asymmetrical Limb
Muscle tone	Diminished	Diminished	Diminished in lower limbs	Diminished in affected limb
Deep tendon reflexes	Decreased or absent	Absent	Absent in lower extremities, later hyper-reflexia	Decreased or absent
Sensation	Severe myalgia but no sensory deficit	Cramps, tingling hypoanaesthesia of palms and soles	Anaesthesia of the lower limbs with sensory loss	Pain in gluteal region

Signs and symptoms	Polio	GBS	Transverse myelitis	Traumatic or injection neuritis
Cranial nerve	Only in bulbar or bulbospinal cases. Loss of gag reflex most common Only in bulbar or bulbospinal cases. Loss of gag reflex most common	Often present affecting VII, IX, X, XI, XII	Absent	Absent
Respiratory insufficiency	Only when bulbar and bulbospinal involving respiratory muscles	In severe cases	Sometimes	Absent
CSF WBCs proteins	High WBCs. Normal or slightly Increased	<10 High	Normal Normal or slightly elevated	Normal Normal
Bladder dysfunction	Absent	Transient	Present	Never
Nerve conduction velocity in 3rd	Abnormal, anterior horn cell disease	Abnormal, demyelination	Normal of abnormal has no diagnostic value	Abnormal in sciatic nerve
EMG 3rd week	Abnormal	Normal	Normal	Normal
Sequelae at 3 months and up to a year	Severe asymmetrical atrophy, skeletal deformities may develop later	Symmetrical atrophy of distal muscles, diplegia, atrophy after years	Flaccid	Moderate atrophy only in affected lower limb

4. Confirmation

- An AFP case is “confirmed” as Polio only by the isolation of wild poliovirus from any stool specimen.
- An AFP case is classified as “non-polio AFP” if wild poliovirus is not isolated from adequate stool specimens.
- If stool specimens are inadequate, final classification of the AFP case as either non-polio AFP or compatible with Polio will depend on the results of 60-days follow-up examination.
- If the 60-days follow-up examination shows no residual weakness, the case is classified as non-polio AFP.
- The final classification of the case as “compatible “or discarded as “non-polio AFP” is determined by the National Expert Review Committee (ERC) which meets every month in New Delhi to review all such cases.
- **Adequate stool:** Two specimens collected within 14 days of paralysis onset and at least 24 hours apart; each specimen must be of adequate volume (8-10 grams) and arrive at a WHO-accredited laboratory in good condition (i.e., no desiccation, no leakage, with adequate documentation and evidence that the cold chain was maintained.)

5. Treatment for Acute Polio like illness

All cases should be treated as below except patients with isolated single lower limb involvement and reporting after 4 days of onset of paralysis and currently not progressing for more than 48 hours.

5.1. Nonpharmacological

- Complete bed rest and correct positioning of the affected limbs in the optimal position as follows:
 - Hip—slight flexion, knee—5° flexion, foot—90° with support against the soles.
 - Both legs should be supported from the lateral sides with pillows or rolled towels or salt/sand packs to prevent rotation.
- When pain subsides, passive movements of the joints for about 10 minutes, 2-3 times a day.
- Warm water fomentation using hot packs with soaked towels wrapped around the affected parts for about 10 minutes, 2-3 times a day help in relieving pain.
- If transient urinary retention occurs, alternate hot and cold compresses over the suprapubic region.

Caution: No massage or intramuscular injections as it may further precipitate paralysis. Watch for progression, particularly for the involvement of the respiratory muscles.

5.2. Pharmacological

- There is no specific drug therapy for polio. For fever and pain, use Paracetamol or Ibuprofen
- Referral to a tertiary care center with a ventilatory support facility, if there is



Figure 28.1: A child with a deformity of her right leg due to polio

progression of paralysis, respiratory distress, bulbar involvement, paralysis of upper limbs which is <3 days old (there is higher risk of diaphragmatic involvement in such cases), marked drowsiness or any other complication.

5.3. Patient/parent education

- No dietary restrictions, however, continue breastfeeding or other regular feeding.
- Paralysis progresses usually for about 4-7 days after onset. Recovery may start thereafter over days to weeks with little recovery of strength after 6 months of illness. A regular physiotherapy facilitates recovery of muscles.

Note: Post-polio residual paralysis should be referred for rehabilitative services to an appropriate centre.

6. Action expected on admission of suspected AFP case:

- Report to higher authority immediately
- Take adequate stool sample
- Send the stool sample maintaining cold chain to district headquarter

Protocol for AFP surveillance

Step	Timing	Description
Case Detection	at diagnosis	Follow case definition for AFP
Case Reporting	≤ 48 hours of report	Tel:
Timing of stool specimens	within 2 weeks of onset of paralysis	2 stool specimens collected no less than 24 hours apart .
Collection of specimens		Fresh stool, or rectal swabs containing fecal material (at least 8g – size of an adult thumb). Place in a sterile glass bottle
Transport of Stools	as soon as able. Specimens arriving at national laboratory ≤ 3 days of being sent	Maintain a cold chain of 2 - 8 °C. Transport in dry ice if transportation will take > 24 hours Caution: avoid desiccation, leakage; ensure adequate documentation
Follow up of patients	60 days from	To determine whether there is residual paralysis on follow up

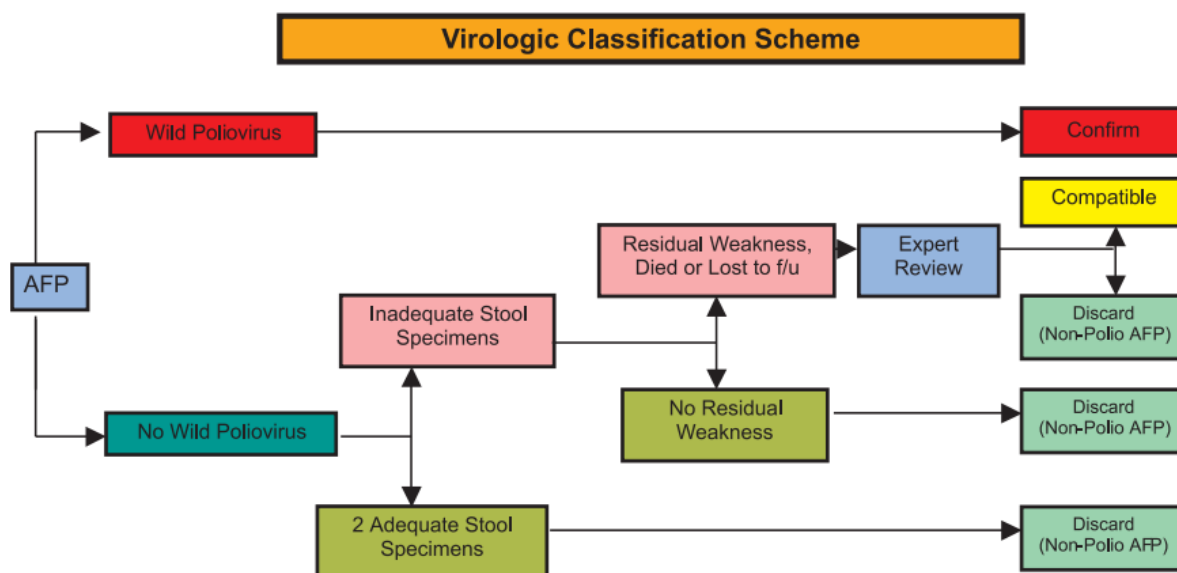


Figure 28.2 Virological Classification

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Government of India, Ministry of Health and Family Welfare. Field Guide - Surveillance of Acute Flaccid Paralysis. 3rd edition. 2005: New Delhi, MOHFW-NPSP; pg-42.
2. Francis PT. Surveillance of acute flaccid paralysis in India. The Lancet. 2007 Apr 21; 369(9570): 1322-3
3. Francis PT. Non-Polio AFP rate and Polio Eradication. *Indian Pediatrics*. 2008 May;45(5):422-3.



29. FEBRILE SEIZURES

1. Introduction

- Febrile seizures are brief (2-5 min), generalized tonic-clonic and self-limited seizures followed by a brief post-ictal period of drowsiness, in an otherwise healthy, febrile child of 6 months to 5 years of age, without any evidence of underlying neurological disease.
- They are the most common seizure disorder during childhood, with a uniformly excellent prognosis.
- They occur rarely before 6 months and after 5 years of age. The peak age of onset is approximately 14-18 months of age, found in 3-4% of young children.
- There is a strong family history of febrile convulsions in siblings and parents, suggesting a genetic predisposition.
- Except for the cases at high risk, simple febrile seizures rarely develop into epilepsy.

2. SALIENT FEATURES

- Febrile seizures usually occur when the temperature is rising rapidly, to generally 39°C (102°F) or more of core temperature. They are of two types:
 - (i) Typical (simple) febrile seizure occurs on day 1 of fever, does not last for more than 10 minutes; generalized tonic-clonic; generally, not more than one episode within 24 hours.
 - (ii) Atypical or complex febrile seizure may persist for more than 15 minutes; it could be focal in nature; more than one episode of seizure in 24 hours; associated with abnormal neurological findings or deficits. An organic cause such as an infectious or toxic process should be considered and investigated.
- Late onset febrile seizures, persistent febrile seizures, generalized epilepsy and Febrile seizure plus (GEFS+) and febrile status epilepticus (FSE) are part of the Spectrum of febrile seizures

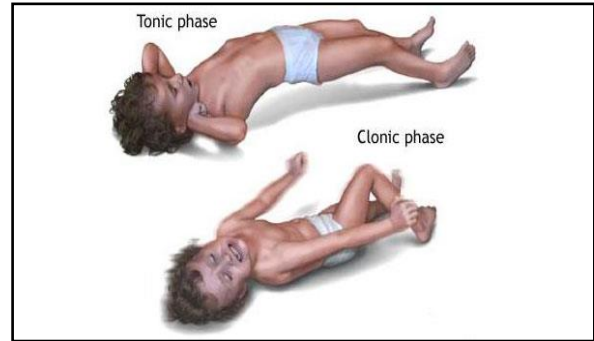


Figure 29.1 Tonic-Clonic Seizure

3. Investigations

- Lumbar puncture: A lumbar puncture with examination of CSF is essential to rule out possibility of meningitis in cases with first episode of febrile seizures.
- EEG has no role in case of simple febrile seizures. However, in cases with atypical febrile seizure or in a child with high risk for developing epilepsy, it may be helpful.

4. High risk for developing Epilepsy

It includes a positive family history of Epilepsy, Initial febrile convulsion prior to 9 months of age, a prolonged or atypical febrile seizure, delayed developmental milestones and an abnormal neurological examination.

5. Treatment

Most febrile seizures are brief and would be over by the time a child is brought to the doctor or health facility. Management includes definitive diagnosis, restraint in investigations, treatment of an acute episode, prophylaxis for future episodes and family counselling. Role of defervescence in preventing febrile seizures is questionable.

5.1. Nonpharmacological

Clear the airway, semi-prone lateral position and Oxygen therapy.

5.2. Pharmacological

- In cases presenting with seizures, the mainstay of management is prompt administration of anticonvulsants.
- The best drug is Diazepam / Midazolam/ Lorazepam in a dose of 0.3 mg/kg by slow intravenous or rectal route. It can be repeated, if seizures do not subside (per rectal dose may be given up to 0.5 mg/kg/dose).
- Intermittent prophylaxis (during febrile illness)
 - It is a safe and effective method of prophylaxis but does not reduce the risk of future Epilepsy.
 - Tab Clobazam 0.75 mg/kg for 2-3 days in 2 divided doses during fever or Tab/Syr. Diazepam 0.3 mg/kg/dose every 8 hours (1 mg/kg/day) for 2-3 days of febrile illness, started on the day of onset of fever. Dose can be adjusted, if over sedation or ataxia noted.
- Continuous prophylaxis
 - Febrile status, complex and recurrent febrile seizures (>6/year in spite of intermittent prophylaxis) may need EEG, neuroimaging and continuous prophylaxis with AED.

Phenobarbitone and Valproate may be used in infants and older children, respectively, for 1-2 years. Carbamazepine and Phenytoin are not useful.

6. Patient/parent education

- The parents and caretaker should be assured of the benign nature of the disease and should be told that no neurological deficit or mental retardation occurs as a result of simple febrile seizure.
- They should be taught about control of fever at home.
- They can be taught to give Diazepam per rectally at home.
- Routine immunization as per schedule should be followed.
- After DPT vaccination, oral Paracetamol 15 mg/kg/dose every 6 h for 2 or 3 days and similarly, after measles vaccination, oral Paracetamol in the same dose started from the day of vaccination and given for 3 to 4 days to avoid precipitation of febrile seizures.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Pratibha D. Singhi, M. Srinivas. Febrile Seizures. Indian Pediatrics. 2001; 38: 733-740.



30. Acute Nephritis

1. Introduction:

- It follows streptococcal infection of throat or skin by 1-2 weeks. Glomerular injury will clinically present as acute nephritis.
- In majority of pediatric patients, it is Post Streptococcal glomerulonephritis.
- Age group 3-12 years.
- Disease is self-limiting and generally resolves in one month; however, microscopic urinary changes may persist up to one year.

2. Signs & Symptoms:

Hematuria, Oedema, Hypertension, Varying degree of oliguria or anuria. May present with CCF. Many primary or secondary Glomerular diseases will present as acute Nephritis.

3. Lab Investigations:

- Urine exam: RBCs, RBC casts, significant proteinuria^{2+/3+}
- ASO titre increases
- Chest X-ray: Cardiomegaly, Pulmonary vascular congestion, Effusion
- Haemogram: Normocytic normochromic anaemia
- BUN, Serum Creatinine raised

4. Complication:

Congestive heart failure, encephalopathy may occur in a few patients.

5. Treatment

5.1 Diet-Proteins, sodium, potassium should be restricted. Urine output should be measured accurately.

5.2 Daily monitoring of weight.

Protein restriction up to 0.6mg/kg/day

5.3 Drugs:

- Antibiotics

- Oral Penicillin 25000-50000u/kg/Day
OR
- Inj. Penicillin 25000-50000 U/kg/Day IM/IV in 4 divided doses x 10 days
OR
- Erythromycin 40mg/kg/day in 4 divided doses x 10 days

Although a 10-day course of systemic antibiotic therapy with penicillin is recommended to limit the spread of the nephritogenic organisms to other children's, antibiotic therapy does not affect the natural history of acute nephritis.

- **Diuretics:** Frusemide 1-2mg/kg/day in 1-2 divided doses
- **Antihypertensive:**
 - (a) Nifedipine: Usual Pediatric Dose of nifedipine for Hypertensive Emergency
 - Immediate release capsules: 0.25 to 0.5 mg/kg/dose (maximum 10 mg/dose) repeated every 4 to 6 hours if necessary. Maximum dose: 1 to 2 mg/kg/day.
 - Initial doses less than or equal to 0.25 mg/kg/dose may result in a less dramatic decrease in blood pressure and be safer than larger initial doses. Some centers use initial doses of 0.1 mg/kg/dose.
 - (b) Enalapril: Usual Pediatric Dose for Hypertension
 - Oral tablets or solution: Children 1 month to 17 years: Initial dose: 0.08 mg/kg/day (up to 5 mg) in 1 to 2 divided doses. Adjust dosage based on patient response.
 - Maximum dose: Doses greater than 0.58 mg/kg (40 mg) have not been evaluated in pediatric patients.

5.4 Rest

6. Refer patient to higher center in case of following condition

- CCF
- Uncontrolled HTN or its complications

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
3. Hay Jr. WW, Levin MJ, Detering RR, Abzug MJ. Current Diagnosis and Treatment: Pediatrics. 22nd Edition. McGraw-Hill Education; 2014



31. Nephrotic Syndrome

1. Introduction:

- Nephrotic syndrome is characterized by massive proteinuria, hypoalbuminemia and edema. Hyperlipidemia is usually associated; hematuria, hypertension and impaired renal function are occasionally seen.
- More than 90% of childhood nephritic syndrome is primary (idiopathic). Other causes are Amyloidosis, Vasculitis, SLE, Post-infectious GN and Hepatitis B Nephropathy.
- Nephrotic syndrome in children can be divided into two groups based on renal histological characteristics:
 - i) Minimal change nephritic syndrome (MCNS):** This is usually sensitive to steroids with a satisfactory long-term outcome.
 - ii) Nephrotic syndrome with significant lesions:** It is usually associated with less satisfactory course, tends to be steroid resistant and a significant proportion progress to chronic renal failure.

2. Clinical Features:

- The onset is insidious with edema first noticed around the eyes and subsequently around the legs.
- Gradually edema may become generalized, with ascites, hydrothorax and hydrocele. Severe muscles wasting are seen.
- It is a triad of hypoproteinemia, hypercholesterolemia and proteinuria.



Fig 31.1 Edema Around Eyes & Generalized Edema

3. Investigations:

- Urine >3+ proteinuria, No RBCs/ Casts
- Azotemia not significant
- Serum proteins < 3.2gm/dL
- Serum cholesterol raised

4. Complication

Spontaneous bacterial peritonitis is the most common complication which should be treated adequately before starting steroids

5. Management

5.1. 1st Episode

- The child should receive a high protein diet no extra salt is given.
- Prednisolone (2mg/kg/day) in 2-3 divided doses along with antacid given till patient goes into remission (Urine Albumin 1+/absent for 5 days) OR up to 8 weeks and then tapered to 2mg/kg/alternate day x3-6 months and then stopped
- Ideal calculation 60mg/m² BSA

5.2. Relapse:

- Urine Albumin 2+ >5 days / oedema. Sometimes resolves in a week if precipitated by infection, then infection is treated.
- If not resolved continue Prednisolone 2mg/kg/day in 2-3 doses till urine is protein free for 5 days, then taper alternate day x 4 week and then stop.
- General care
- Ca⁺⁺ & K⁺ supplement if on Frusemide
- Frusemide: 1-2mg/kg/day for edema with expert advice.

5.3. Treatment of frequent relapses:

- Long term alternate day Prednisolone: Following completion of treatment for relapse, alternate day Prednisolone is slowly tapered to minimum maintenance dose (0.3 – 0.7mg/kg). The dose is maintained for 9-12 months.
- Levamisole: After inducing a remission, Levamisole is administered at a dose of 2-2.5 mg/kg on alternate days. Co-treatment with Alternate day Prednisolone is given in decreasing doses, until a dose of 0.3-0.5mg/kg is reached, for 2-3 months.
- Cyclophosphamide and Cyclosporine A are used in some cases.

6. Refer to higher center

- For confirmation of diagnosis
- Systemic features: Arthritis/ Rash/ Hepatosplenomegaly
- Persistence of Hypertension / Azotemia/ Hematuria
- Steroid dependent or resistant or frequent relapse

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
3. Hay Jr. WW, Levin MJ, Detering RR, Abzug MJ. Current Diagnosis and Treatment: Pediatrics. 22nd Edition. McGraw-Hill Education; 2014

Further reading:

1. Bagga A. Revised guidelines for management of steroid-sensitive nephrotic syndrome. Indian Journal of Nephrology. 2008;18(1):31-39.
2. Indian Pediatric Nephrology Group, Indian Academy of Pediatrics. Consensus Statement on Management of Steroid Sensitive Nephrotic Syndrome. Indian Pediatrics. 2001 Sep; 38(9):975-86.



32. Congestive Heart Failure (CHF)

1. Introduction:

Every Cardiac patient has potential to develop CHF. It is not a diagnosis. It is a clinical syndrome due to an underlying anatomical or pathological cause which is the primary diagnosis.

2. Definition: Congestive cardiac failure is defined as “inability of the heart to maintain an output, at rest or during stress, necessary for the metabolic needs of the body (systolic failure) and inability to receive blood into the ventricular cavities at low pressure during diastolic (diastolic failure)”.

3. Etiology

The causes of diastolic failure are given below:

- i) Mitral or tricuspid stenosis.
- ii) Constrictive pericarditis
- iii) Restrictive cardiomyopathy
- iv) Acute volume overload (acute aortic or mitral regurgitation)
- v) Myocardial ischemia
- vi) Marked ventricular hypertrophy
- vii) Dilated cardiomyopathy

The causes of systolic failure or mixed systolic diastolic failure can be divided into two groups according to age.

- The commonest cause of CCF in infants is Congenital heart disease, whereas in the older children it is Rheumatic fever and Rheumatic heart disease.
- Causes of Congestive Cardiac Failure

Infants:

- Congenital heart disease
- Myocarditis and primary myocardial disease
- Paroxysmal tachycardia
- Anemia

Children:

- Rheumatic fever and RHD
- CHD complicated by anemia infection
- Hypertension
- Myocarditis
- Upper respiratory obstruction.

4. Symptoms:

Slow weight gain, easy fatigability, persistent horse crying, wheezing, excessive perspiration, puffiness of face, edema, not able to suck at mother's breast due to breathlessness.

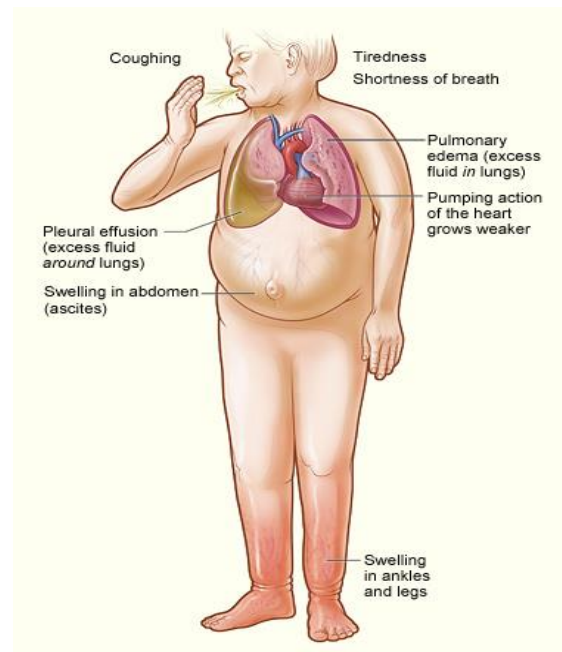


Figure 32.1: Symptoms of CHF

5. Signs:

- Left sided failure is indicated by tachypnea and tachycardia.
- Persistent cough, wheezing, crepts in the chest.
- Right sided failure signs are hepatomegaly, facial edema, edema on feet.



Figure 32.2: Edema on feet

6. Treatment:

It is based on following principles:

- i. Reducing cardiac work.
- ii. Augmenting myocardial contractility.
- iii. Improving cardiac performance by reducing the heart size.
- iv. Correcting the underlying cause.

- Control of excessive salt and water retention with diuretics.
- Improve cardiac contractility with digoxin.
- Prevent and reverse neuro-hormonal changes that lead to progressive worsening of cardiac status with beta blockers, ACE inhibitors.

6.1 Digoxin

- Drug of choice in chronic CHF with atrial fibrillation.
- In CHF with sinus rhythm it gives symptomatic benefit
- Dose
 - Total digitalizing dose in children: 30 - 40 mcg/kg. ½ the total dose stat: ¼ after 8 hrs; ¼ after 16 hrs.
 - Daily maintenance dose: ¼ of total digitalizing dose. Once daily or 2 divided doses.
- Hypokalemia may aggravate digitalis toxicity especially with concomitant diuretic administration. Use oral KCl supplement or use potassium sparing diuretics such as spironolactone.

6.2 Oral Diuretics

- Frusemide: 1 - 2 mg/kg/day or
- Hydrochlorothiazide: 1 - 1.5 mg/kg/dose every 12 - 24 hours or
- Spironolactone: 1 - 2 mg/kg/day

6.3 ACE inhibitors

- Indicated for all patients with congestive heart failure.
- Captopril: 0.1 to 0.5 mg/kg/dose oral every 8 to 12 hourly upto 4 mg/kg/day or
- Enalapril: 0.1 mg/kg/dose oral every 12 - 24 hourly upto 0.5 mg/kg/day

6.4 β-blockers

- β-blockers is an integral part of congestive heart failure therapy nowadays.
- Metoprolol or carvedilol is also used

6.5 Diet

Calories - Recommended daily dietary allowance plus 20 - 30% in shunt lesions Avoid salty foods and additional salt in cooking Iron supplementation

7. When to refer

- Severe respiratory distress
- Acute pulmonary oedema
- Refractory CCF
- Cardiogenic shock

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
3. Hay Jr. WW, Levin MJ, Deterding RR, Abzug MJ. Current Diagnosis and Treatment: Pediatrics. 22nd Edition. McGraw-Hill Education; 2014
4. McNerny TK, Adam HM, Campbell D, Kamat DK, Kelleher KJ, eds. American Academy of Pediatrics (AAP) Textbook of Pediatric Primary Care. 5th Ed. Elk Grove Village; 2009



33. PICA

1. Introduction:

- Pica involves repeated and chronic ingestion of non-nutrient substances including mud, plaster, paint, earth, clay, etc.
- Children with PICA usually have history of neonatal insults.
- Most of the time, it is self-limiting and represents manifestations of family disorganization, poor supervision, and affectional neglect.
- Testing or mouthing of strange objects is normal in infant and children up to age of 2 years.
- Common in children from lower socioeconomic strata and at times in the malnourished and mentally subnormal children.

2. Signs & Symptoms:

Pallor and chronic abdominal pain are main complaints.

3. Treatment

- Pica below two years does not need any intervention.
- Children with pica are at increased risk of lead poisoning, iron deficiency, and parasitic infections. They should be investigated for these problems and if present, treated suitably.
- Education, guidance and counselling of the family.
- The child has to be kept occupied in other tasks and provided with the environmental stimulation.

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013



34. NOCTURNAL ENURESIS

1. Introduction:

Enuresis is defined as normal nearly complete evacuation of the bladder at a wrong place and time at least twice a month after the fifth year of life. Bedwetting at night is known as nocturnal enuresis. Enuresis may be primary or secondary:

2. Primary enuresis:

Repeated passage of urine into clothes/bed during night in a child more than 5 years of age. Most common cause in primary enuresis is inappropriate toilet training. Other causes could be genetic, sleep disorder, reduced Anti Diuretic Hormone (ADH) at night. In some cases, there may be organic etiology such as obstructive uropathy or UTI. There may be associated with mental retardation or spinal cord abnormalities.

3. Secondary enuresis:

The child has been dry for several months and again starts bed wetting. Too enthusiastic and immature toilet training, emotional stress, parent child maladjustment, urinary tract infections, diabetes mellitus or diabetes insipidus. can cause secondary enuresis.

4. Signs & Symptoms:

Involuntary discharge of urine after the age at which bladder control should have been established (5 years).

In primary nocturnal enuresis, child has never been dry at night while in secondary, child has been continent for at least 6 months before the child begins to wet again.

5. Investigations:

- Full medical history
- Genital and neurological examination
- Urine for albumin, sugar, microscopy, specific gravity and culture.
- USG voiding cystourethrogram and urodynamic studies.

6. Treatment

6.1. Nonpharmacological (effective in 30% cases)

- Rule out organic causes.
- Restrict fluid intake in the evening.
- Bladder exercises:
 - Hold urine as long as possible during the day.
 - Practice repeated starting and stopping the stream at the toilet bowl.
- Emotional support to child.
- Behaviour modification- Child should not be given liquids after meal in the evening. Practice getting up from bed and going to the bathroom at bed time before sleep.
- Alarm device- alarm device is used to elicit a conditioned response of awakening to the sensation of a full bladder.

6.2. Pharmacological

Indicated only in children > 6 years where sufficient trial of non-pharmacological management has failed with following:

- Tab. Imipramine: (0.9-1.5 mg/kg/day/PO) 6-8 year (25 mg), 9-12 year (50 mg), >12 year (75 mg) once a day at bedtime. Success rate 30-60%, relapse rate 90%. or
- Tab. Desmopressin: 0.1-0.5 mg at bedtime.
Or
- Desmopressin acetate (nasal spray, 10 mcg per spray): Start with 10 mcg given at bedtime daily and increase gradually by 10 mcg/per week to a maximum of 40 mcg per day. If effective, it should be used for 3-6 months. Success rate is 40-60%; relapse rate is 90%.

(Caution: Not effectively absorbed in rhinorrhea. If not used properly may cause hyponatremia)

7. Referral

Refer the patient to a higher centre, if organic cause is suspected or when diagnosis is in doubt.

8. Parent education

- Reassure the parents that condition is self-limiting.
- Ask the parents to maintain a diary record of dry nights; reward the child for such nights. Avoid punitive measures.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Mathew JL. Evidence-Based Management of Nocturnal Enuresis: An Overview of Systematic Reviews. Indian Pediatrics. 2010 Sep;47(9):777-80.



35. Thrush / Candidiasis

1. Introduction:

- Candida fungal infection in the oral cavity is common and may be seen as early as 7 to 10 days of age (peak 4th week of life).
- After infancy it is usually secondary to treatment with broad-spectrum antibiotic.
- Chronic or recurrent oral candidiasis is seen in children having immuno-deficiency, HIV/AIDS, undergoing cancer therapy and in severe malnutrition.
- Hypoparathyroidism, Addison's disease, Autoimmune disorders are other rare causes.

2. Salient Diagnostic Features:

- Thick white patches on an angry red base in the mouth may spread to involve the lips, buccal mucosa, tongue and palate.
- Asymptomatic or may cause pain in the mouth, discomfort, anorexia and feeding difficulty.
- Diagnosis is confirmed by the fact that on removing the plaques, spots of bleeding are seen on the mucosal surface.
- Faulty sterilization of bottle and nipple causes persistent or recurrent infections / thrush.



Figure 35.1: Candidiasis / oral thrush

3. Treatment: Drugs

- In case of breast fed baby, the medicine has to be applied to mother's nipples also to prevent cross / re-infection.
- Clotrimazole 1% cream, gel or lotion, oral application 3-4 times/day after feeding for 5-7 days (or 1-2 days beyond recovery).
Or
- Gentian violet 1% aqueous solution, 1-2 times a day, for 5-7 days (can stain tissues and clothes).

- In resistant/ chronic cases (patients with major underlying disease)
Tab. Fluconazole 3-6 mg/kg once daily for 5-7 days.

4. Prevention:

- Emphasize on avoiding bottle feeding /bottle hygiene, care/ hygiene of the nipple and treatment of vaginal candidiasis in expectant mother.

Bibliography:

1. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013



36. Congenital Hypothyroidism

1. Introduction:

- It may be familial or sporadic, goitrous or non-goitrous.
- In about 85% cases the etiology is dysgenesis.
- All new born babies should be screened.

2. Signs & Symptoms:

At birth –

- Lethargy
- Prolonged Unconjugated Hyperbilirubinemia
- Constipation
- Hypothermia
- Bradycardia
- Large Anterior fontanelle
- Posterior fontanelle more than 0.5 cm which is normal in only 3% neonate
- Somnolence and Choking spells during nursing are present during first month of life
- Affected infants cry little, sleep much

Develop with passage of time

- Large tongue
- Hoarse cry
- Facial Puffiness
- Umbilical Hernia
- Hypotonia
- Mottling
- Cold hands and feet

3. Investigation:

- TSH level should be estimated after 72 hours of life if TSH is more than 20 mU/L then Hypothyroidism is considered and Eltroxin should be started.
- Second screening at 2 to 6 weeks (routinely in VLBW and NICU graduates)

4. Treatment of Congenital Hypothyroidism

As soon as possible diagnosis.

4.1 Counseling of Parents

4.2 Eltroxin

- 10-15 microgram/kg---start higher dose
- Do NOT use liquid preparation
- On above dose, T4 Normalises in 1 week and TSH in one month
- Aim: to normalize serum T4, avoid hyperthyroidism, promote normal growth and development

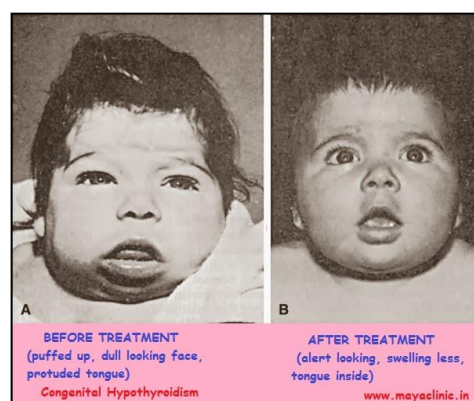


Fig 36.1: Congenital Hypothyroidism: Before & after treatment

4.3 Repeat T4 and TSH at 2 and 4 weeks after initiation of therapy.

- After one year tests to be done 1-2 monthly
- Between 1 and 3 years do tests 2 to 3 monthly
- Over 3 years do tests yearly till growth is complete.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013
3. Hay Jr. WW, Levin MJ, Deterding RR, Abzug MJ. Current Diagnosis and Treatment: Pediatrics. 22nd Edition. McGraw-Hill Education; 2014

Further reading:

1. Rose SR et. al. Update on newborn screening and therapy for congenital hypothyroidism. Pediatrics. 2006 Jun;117(6):2290-303.
2. Nair PS, Sobhakumar S, Kailas L. Diagnostic re-evaluation of children with congenital hypothyroidism. Indian Pediatrics. 2010 Sep;47(9):757-60.



37. Urinary Tract Infection (UTI)

1. Introduction:

Approximately 8% of girls and 1 - 2% of boys are likely to get UTI during childhood. A significant proportion of children less than 2 years developing UTI have underlying urinary tract anomalies, most often vesico-ureteric reflux (VUR). UTI in a setting of VUR may lead to renal scarring, an important cause of chronic renal disease. Early recognition and treatment of UTI and urinary anomalies is essential to prevent such complications.

2. Signs & Symptoms

2.1 Neonates

Sepsis like features with fever or hypothermia, lethargy, poor feeding, poor weight gain, jaundice and shock; urinary symptoms may be absent.

2.2 Infants and children below 2 years

Unexplained fever; urinary symptoms minimal or absent.

2.3 Adolescents

Mostly related to lower urinary tract such as dysuria, frequency, urgency and suprapubic pain. Renal parenchymal involvement is indicated by high fever, chills, rigors and flank pain.

3. Investigations:

3.1 Urine analysis

May suggest UTI in the form of increased leukocytes in urine. Gram stain of centrifuged urine specimen may show bacteria. Dipstick for nitrite reduction and leukocyte esterase may help in rapid diagnosis.

3.2 Urine culture

- This is the only confirmatory test for UTI. Every effort must be made to properly collect and send a urine sample before antibiotic is started. In infants and young children UTI should be suspected if there is unexplained fever.
- A midstream clean catch specimen is ideal. Soap or antiseptic solution should not be used before collection. In infants, urine can be obtained by suprapubic aspiration.

3.3 USG Abdomen and Pelvis, Voiding Cystourethrogram

These will identify upper and lower urinary tract abnormalities, including vesicoureteral reflux in complicated UTI

4. Common organisms responsible for UTI:

- E. coli, Occasionally Klebsiella, Staph epidermidis or Strep fecalis may be responsible.
- A colony count of > 10⁵ colony forming units (CFU) / ml of single species in a clean catch specimen indicate significant bacteriuria.
- Presence of any bacteriuria in suprapubic specimen is significant.

5. Treatment

For the purpose of management UTI is divided into complicated and uncomplicated UTI.

5.1 Complicated UTI

- Temperature > 39°C, persistent vomiting, renal angle tenderness and systemic toxicity are features of complicated UTI
- Infants below 3 months of age and those with complicated UTI should receive parenteral antibiotics initially.
- In young infants (< 3 months) entire treatment is parenteral
 - Cefotaxime 100 - 150 mg/kg/day in 3 div doses, or
 - Ceftriaxone 75 mg/kg/day in 1-2 doses, or
 - Gentamycin 5 - 7.5 mg/kg/day single dose, or
 - Amikacin 15 - 20 mg/kg/day single dose, or
- For older children, after first 2 - 3 days, oral antibiotics may be started based on antimicrobial sensitivity. Total duration of treatment is 10 - 14 days.
- Oral antibiotics
 - Amoxicillin 20 - 40 mg/kg/day in 2 - 3 doses, or
 - Cefadroxil 30 mg/kg/day in 2 doses, or
 - Cephalexin 50 mg/kg/day in 3 doses, or
 - Cefixime 8 mg/kg/day in 2 doses, or
 - Ciprofloxacin 10 - 20 mg/kg/day in 2 doses

5.2 Uncomplicated UTI

- Children > 3 months of age and those who do not have features of complicated UTI can be treated with oral Amoxicillin (10 to 15 mg per kg) or Cefadroxil (5 to 10 mg per kg) for 7 to 10 days (based on sensitivity).
- Though fluoroquinolones are effective and safe for UTI, they are not the first-line antibiotics.

Bibliography:

1. Kliegman R, Stanton B, Geme JS, Schor N. Nelson's Textbook of Pediatrics. 20th Edition. Elsevier; April 2015
2. Paul VK, Bagga A. Ghai Essential Pediatrics. 8th Edition. CBS Publishers; 2013

Further reading:

1. Lin CW, Chiou YH, Chen YY, et. al. Urinary tract Infection in neonates. Clinical Neonatology. 1999; 6 (2). [cited 18 July 2016]
Available from: <http://www.son.org.tw/upload/Jour/2/199912/1.pdf>

