INDIAN JOURNAL OF PRACTICAL PEDIATRICS

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Dr.K.Nedunchelian Editor-in-Chief	**Dr.S.Thangavelu** Executive Editor

CONTENTS

TOPIC OF INTEREST - "IAP-IJPP CME 2013"

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FOR YOUR KIND ATTENTION

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51ST ANNUAL NATIONAL CONFERENCE OF INDIAN ACADEMY OF PEDIATRICS

Venue: 5, Brilliant Convention Centre, Scheme 78, Part II, Indore (M.P.)

Achieving MDG-4: Strategies & Action

PEDICON 8–12 JANUARY 2014 INDORE

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1st May-31st July,13	9,500	9,500	12,000	12,000	7,000	7,000	850 USD	850 USD	12,500	50%
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16th Oct-15th Dec,13	13,000	13,000	15,000	15,000	10,000	10,000	1200 USD	1200 USD	15,500	NIL
On the spot Registration	16,000	16,000	18,000	18,000	13,000	13,000	1500 USD	1500 USD	18,500	NIL

1 USD = 55 INR

Payment Details

Conference Amount _____ **Workshop Amount** _____ (Please refer the back Page for Workshop Amount and Codes)

Workshop Preference 1. (Code No.) _____ **Workshop Preference 2. (Code No.)** _____ **Workshop Preference 3. (Code No.)** _____

Total amount _____ Amount in words _____

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IMPORTANT NOTES

- For PG: Separate certificate from HOD is mandatory.
- Senior IAP Member who completed 70 years of age before January 2013 are exempted from Registration till 30th September 2013. (Age proof necessary)
- Payment from Indian delegates and foreign delegates will be accepted in INR and USD respectively.
- This member's contribution includes 1000/- for Central Indian Academy of Paediatrics corpus fund.
- Kindly send us your dully filled registration form by Email/Fax along with your bank remittance copy.
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- Fill or Download the registration form and send the duly filled form to the secretariat with demand draft as per the applicable fee structure.
- The registration fee MUST reach the secretariat before or on the deadline date, after which the next slot shall be applicable.

Registration Fee Inclusions

- Admission to all Scientific Sessions, Inaugural Function and Exhibition area.
- Lunch on all days.
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- Cancellation must be notified to the secretariat in writing by email pedicon2014indore@gmail.com
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Add: OQ/1 CRP LINE, In Front Of Masjid
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51ST ANNUAL NATIONAL CONFERENCE OF INDIAN ACADEMY OF PEDIATRICS

Achieving MDG-4: Strategies & Action

PEDICON

8–12 JANUARY 2014 INDORE

Venue: 5, Brilliant Convention Centre, Scheme 78, Part II, Indore (M.P.)

Workshop Codes	WORKSHOP DETAILS	Academic Coordinator	Fee for Indian Delegates
1	Growth Monitoring	Dr. Vaman Khadilakar	1,000
2	Practical issues in Pediatric Epilepsy	Dr. Anaita Hegade	1,000
3	Asthma for Clinicians	Dr. Shishir Modak	1,000
4	Basic Paediatric Intensive care Course (Two days)	Dr. Praveen Khilnani	2,000
5	Advanced Mechanical Ventilation	Dr. Anil Sachdev / Anchor - Dr. Krishen Chug	1,000
6	Bed side Ultrasound in emergency & Critical Care	Dr. Dhireen Gupta	1,000
7	a) Art & Science of writing article in Journal b) Professional Social Responsibility*	Dr Harish Pemade Dr.Naveen Thacker	1,000
8	How to develop a successful Paediatric Practice	Dr. Pramod jog	1,000
9	Genetics in Paediatricians	Dr. Shubha Phadke	1,000
10	Comprehensive Adolescent Care	Dr. JS Tuteja	1,000
11	Fluid, Electrolytes, Acid base	Dr. Madhu Otiv	1,000
12	Computers & Information Technology in Padiatric Practice	Dr. Yatin Mehta	1,000
13	Infant & Young child feeding	Dr. Satish Tiwari	500
14	IAP-BLS Course for Health Care Providers	Dr LN Taneja / Dr.Sukhmeet Singh	1,000
15	Pediatric Gastroenterology workshop: What, When & How?	Dr. Neelam Mohan	1,000
16	Pediatric Hematology Oncology in office practice	Dr. Anupam Sachdev	1,000
17	Comprehensive Pediatric Infectious disease course (Two Days)	Dr. Abhay K Shah	2,000
18	Developmental Screening & Early stimulation	Dr. Navin Jain	1,000
19	Medico legal Issues in Paediatric Practice	Dr. Mukul Tiwari	1,000
20	Practice Cardiology in Paediatrics	Dr. Smita Mishra	1,000
21	Pals recertification course*	Dr. Arif Ahmad	2,000
22	Dialysis in Children	Dr Siddharth Sethi	1,000
23	Pediatric Emergency and acute care in office practice	Dr.Suresh Gupta / Dr. Indumati Sanathanam	1,000
24	Newborn Hearing Screening Programme	Dr. Abraham K Paul	500

*Special attraction for interested participants: 1.5 to 2 hours session on Professional Social Responsibility, this will cover How to write bestselling self-help health books for the use of general public and empowering patients to obtain optimal health care from health providers, Publication in newspapers, magazines and electronic media during Art & Science of writing article in Journal

Important Notes

- Registration is mandatory for attending the workshop.
- Workshop no. 4 & 17 are two days duration on 7th & 8th January 2014 while remaining is one day on 8th January.
- Details of the faculties shall be announced in due course of time.
- One can participate in one workshop only. However you can give up to 3 choices in order of preference.
- Maximum seats for all the delegates are 50 in each workshop. It is on first come first serve basis.
- For Indian delegates Fee structure of workshops is as per table.
- For SAARC/International Delegates workshop 4, 17 & 21 fee is 200 USD while remaining it is 100 USD.

Bird's eye view of CME programme

Thursday : 9th January

8.30 - 9.00 am	**Inaugural Function for CMEs**
09.00 - 10.00 am	Guest Lectures (1-2)
10.00 - 11.00 am	Panel discussion
11.00 - 12.00 Noon	Guest Lectures (3-4)
12.00 - 01.00 pm	Case based discussion
01.00 - 02.00 pm	Guest Lectures (5-6)
02.00 - 03.00 pm	Symposia
03.00 - 04.00 pm	Guest Lectures (7-8)
04.00 - 05.00 pm	Clinical quiz/Keypad
06.00 pm onwards	Inaugural function of Pedicon 2014

Scientific Programme - Bird's eye view

- Most sessions would run concurrently in six different halls
- Orations would be non concurrent & conducted in main hall

Time	Friday : 10th January	Saturday : 11th January	Sunday : 12th January
7.30 – 8.30 am	Meet the Expert	Meet the Expert	Meet the Expert
8.30 – 10.00 am	Sp. Chapters	Sp. Chapters	Sp. Chapters
10.00 – 10.30 am	Guest Lecture	Guest Lecture	Guest Lecture
10.30 – 11.30 am	S. C. Oration	Plenary I	Plenary II
11.30 – 12.30 pm	Dialogue session	Dialogue session	IAP Quiz/Awards
12.30 – 1.00 pm	Guest Lecture	Guest Lecture	Pictorial Quiz
1.00 – 1.30 pm	Free Papers	Free Papers	Free Papers
1.30 – 2.00 pm	Therapeutic Dilemma	Therapeutic Dilemma	Free Papers
2.00 – 2.30 pm	Diagnostic Difficulties	Diagnostic Difficulties	-
2.30 – 3.15 pm	Symposia	Symposia	-
3.15 – 3.45 pm	Challenges in Practice	Challenges in Practice	-
3.45 – 4.30 pm	Panel Discussion	Panel Discussion	-
4.30 – 5.00 pm	Hot topics	Clinical Quiz	-
5.00 – 6.00 pm	Sponsored Activity	Sponsored Activity	-

INSTRUCTIONS TO AUTHORS

General

Print the manuscript on one side of standard size A4, white bond paper, with margins of at least 2.5 cm (1") in double space typescript on each side. Use American English using Times New Roman font 12 size. Submit four complete sets of the manuscript.

They are considered for publication on the understanding that they are contributed to this journal solely.

All pages are numbered at the top of the right corner, beginning with the title page.

All manuscripts should be sent to: The Editor-in-Chief, Indian Journal of Practical Pediatrics

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1st Page –

Title

Name of the author and affiliation

Institution

Address for correspondence (Email, Phone, Fax if any)

Word count

No. of figures (colour / black and white)

No. of references

Authors contribution

2nd Page –

Abstract (unstructured, not exceeding 100 words) with key words (not exceeding 4)

3rd Page -

Acknowledgement

Points to remember (not more than 5 points)

Text

References

Tables

Legends

Figures – should be good quality, 4 copies black & white / colour,*

(4 x 6 inches – Maxi size) Glossy print

* Each colour image will be charged Rs. 1,000./- separately, with effect from January 2006 (Except for invited articles).

Text

Only generic names should be used

Measurements must be in metric units with System International (SI) Equivalents given in parentheses.

References

Recent and relevant references only

Strictly adhere to Vancouver style

Should be identified in the text by Arabic numerals as superscript.

Type double-space on separate sheets and number consecutively as they appear in the text.

Articles without references / defective references will entail rejection of article.

Tables

Numbered with Roman numerals and typed on separate sheets.

Title should be centered above the table and explanatory notes below the table.

Figures and legends

Unmounted and with figure number, first author's name and top location indicated on the back of each figure.

Legends typed double-space on separate sheet. No title on figure.

All manuscripts, which are rejected will not be returned to author. Those submitting articles should therefore ensure that they retain at least one copy and the illustration, if any.

Article Categories

Review article

Article should be informative covering the recent and practical aspects in that field. Main articles can be in 1500 – 2000 words with 12 – 15 recent references and abstract not exceeding 100 words.

Case report (covering practical importance)

250 – 600 words, 8 – 10 recent references

Clinical spotters section

150 – 200 words write up

With 1 or 2 images of clinically recognizable condition

(of which one could be in the form of clinical photograph / specimen photograph / investigation)

Letters to the Editor

200 – 250 words pertaining to the articles published in the journal or practical viewpoints with scientific backing and appropriate references in Vancouver style.

Check List

Covering letter by corresponding author

Declaration (as enclosed) signed by all authors **

Manuscript (4 copies)

Accompanied by a copy in CD / or submit as an email attachment in addition to hard copy.

Failing to comply with the requirement at the time of submission would lead to the rejection of the article.

Author's contribution / Authorship Criteria

All persons designated as authors should qualify for the authorship. Authorship credit should be based on substantial contributions to i) concept and design, or collection of data, or analysis and interpretation of data; ii) drafting the article or revising it critically for important intellectual content; and iii) final approval of the version to be published. All conditions 1, 2 and 3 must be met. Participation solely in the collection of data does not justify authorship and can be mentioned in the acknowledgement if wanted.

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Selection procedures

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IAP-IJPP CME 2013

THE WHO MULTICENTRE GROWTH CHARTS

Anuradha Bose

Abstract: *Growth charts were already in use and several countries, including India, had local growth charts. In the early 2000s, the WHO undertook the task of creating a multicountry growth reference standard. The aim was to create a set of standards, based on the growth of breast fed babies, as these babies were likely to represent how babies should grow. The WHO Multicentre Growth Reference Study (MGRS) was carried out between 1997 and 2003. A standard defines how children should grow, deviations from the pattern it describes are evidence of abnormal growth. A reference, on the other hand just provides and serves as a tool for comparison. The MGRS data provide a solid foundation for developing a standard because they are based on healthy children living under conditions likely to favour achievement of their full genetic growth potential. A cross-sectional design was adopted for children aged 18 to 71 months, as growth in this age range is more linear than for younger children. The WHO recommends the application of the MGRS charts for all children worldwide, regardless of ethnicity. Several countries have officially adopted the new standards and many others are in the process of doing so. Adopting and applying these standards will enable direct comparisons of the state of nutrition of under-5 children across nations, and provide comparable estimates of the levels of malnutrition. The 5 to 19 year charts can help in building up data on the creeping epidemic of childhood obesity in India.*

Keywords: *MGRS-Growth charts, Standard.*

The World Health Organization (WHO), in the year 2006, released a set of charts which are meant to set standards for growth. These charts combine data from 6 cities in countries across the globe: Davis, California, USA; Muscat, Oman; Oslo, Norway; Pelotas, Brazil; Accra, Ghana; and South Delhi, India. There is a striking omission here; there is no representation of the far Eastern countries, the reason for which is not documented in any of the WHO publications. This "WHO Multicentre Growth Reference Standards" were developed to replace the "National Center for Health Statistics (NCHS)"/ WHO international growth reference. The study combined a longitudinal follow-up from birth to 24 months with a cross-sectional component of children aged 18-71 months.

By including only children who were breast fed according to guidelines, born to mothers who were likely to provide an environment for attainment of full potential by the child, these MGRS charts reflect the standards for growth. Any deviation from these charts would therefore be considered to be deviations from the norm.

There were growth charts already in use, and several countries, including India, had local growth charts.

Why was the task of creating a multi country growth reference standard undertaken?

The most widely used charts earlier were the NCHS/ WHO reference. These charts had some limitations.[1] The data used to construct the reference covering birth to three years of age came from a longitudinal study of children of European ancestry from a single community in the USA. These children were measured every three months, an interval which is inadequate to describe the rapid and changing rate of growth in early infancy. Also, the statistical methods available at the time the NCHS growth curves were constructed were too limited to correctly model the pattern and variability of growth. As a result, the NCHS curves do not adequately or appropriately represent early childhood growth.

The fact that the NCHS reference does not adequately represent early childhood growth was further validated when a review was undertaken. This review was done by comparing children and infants who were predominantly breast fed, with the growth charts of the NCHS reference. The qualification that they had to be breast fed was included, as the growth of breast fed babies were likely to represent how babies should grow.

The data for comparison came from 2 subsets of data that were already available from other studies. The criteria

* Professor of Pediatrics,
Christian Medical College,
Vellore.

for selection of subjects for comparison were that they had to be exclusively or predominantly breast fed from birth to at least 4 months, and whose mothers continued breastfeeding for the first 12 months. In order to be comparable with the NCHS reference recruits, the children selected for inclusion in the comparison were predominantly of European background and of relatively high socioeconomic status. When the 2 breast fed groups were compared with each other, their Z scores were similar. When plotted on the NCHS curves, and thereby compared with the NCHS Reference, it was found that their weight-for-age and weight for length Z scores fell progressively from months 2 through 12, and those for length-for-age fell from 2 to 8 months. Breast fed babies represent how babies should grow. The fact that their growth did not correspond to the NCHS reference curves meant that the NCHS reference was not appropriate for breast fed babies.[2]

A logical outcome of this finding was to undertake the construction of a Standard, the WHO Multicentre Growth Reference Study (MGRS), which was implemented between 1997 and 2003.[3] The MGRS is unique in that it was purposely designed to produce a standard rather than a reference. Although" standards" and "references" both serve as a basis for comparison, each enables a different interpretation. Since a "standard" defines how children should grow, deviations from the pattern it describes are evidence of abnormal growth. A "reference" on the other hand just provides and serves as a tool for comparison and does not indicate that that is how babies should grow.

The MGRS data provide a solid foundation for developing a standard because they are based on healthy children living under conditions likely to favour achievement of their full genetic growth potential. Furthermore, the mothers of the children selected for the construction of the standards engaged in fundamental health-promoting practices, namely breastfeeding and not smoking (de Onis et al., 2004b). The details of how the study was standardized and their results are available.[4,5] The total sample size for the longitudinal and cross-sectional components from all six sites was 8440 children. A total of 1743 children were enrolled in the longitudinal sample, of which the mothers of 882 children were fully compliant and were included. The cross-sectional sample comprised 6669 children.

In India, the study was done in New Delhi, by a door-to-door survey conducted in 58 selected neighborhoods.[6] Pregnant women whose newborns were likely to be eligible for the longitudinal study were enrolled as were children between the ages of 18 to 71 months. A total of at least 17 years of education for the mother or father was used as a criterion. This was to facilitate selection of a subpopulation of infants who would have a greater chance of attainment of potential for physical growth. Neonates with evident congenital anomalies or illness necessitating more than 24 hours stay in a neonatal unit, were excluded. 21 visits were made in the first 2 years: at weeks 1, 2, 4, and 6; monthly from 2 to 12 months; and every two months in the second year. Anthropometric measurements were done by teams standardized in the procedures.

A cross-sectional design was adopted for children aged 18 to 71 months, as growth in this age range is more linear than for younger children. Using 18 months as the lower age limit and 71months as the upper age limit, there is an overlap of 6 months with the longitudinal study and a better estimate of growth at 60 months.

The following indicators are included in the MGRS charts: length/height-for-age; weight-for-age; weight-for-length ; weight-for-height ; body mass index-for-age (BMI-for-age); head circumference-for-age; arm circumference-for-age; subscapular skinfold-for-age; triceps skinfold-for-age; motor development milestones; weight velocity ; length velocity; head circumference velocity.

How frequently should the measurements be taken when monitoring the growth of a child?[7]

Ideally, weight should be taken at birth, then every 2 weeks until 2 months of age, monthly from 2 to 24 month, and 6 monthly from 24 to 60 months. During periods when clinically indicated, it can be taken monthly. Length/height should be taken at birth, repeated at 2 and 6 months and 6 monthly upto 60 months.

The development of a WHO growth reference for school-aged children and adolescents, ie, 5 to 19 years, was spurred by the release of the 2006 WHO MGRS growth reference standards for the 0 to 5 years group and recognition of the need for reference charts for the older age group. There were also increasing concerns, across many countries, of the rising prevalence of childhood obesity. The reference previously recommended i.e. the National Center for Health Statistics (NCHS)/WHO international growth reference, has some drawbacks. These are that the body mass index (BMI) -for-age reference, (1991) starts only at 9 years of age, has year-wise data and covers a limited percentile range.

After deliberations, it was decided that the conditions under which the 0 to 5 years charts were made, could not be recreated or applied to the older age group as their environment would be difficult to control, across countries. It was decided therefore to create a growth reference (note, and not a standard) for this age group, using historical data. The 1977 NCHS/WHO growth reference from 5 to 19 years, using the original sample would be merged

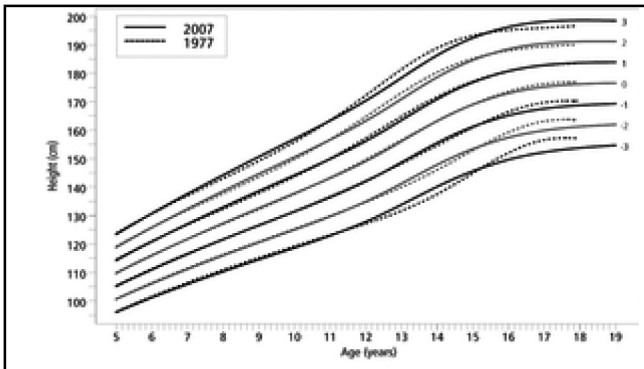

Fig.1. Comparison between the 1977 and 2007 height-for-age *z*-score curves – boys. who.int/bulletin/volumes/85/9/07

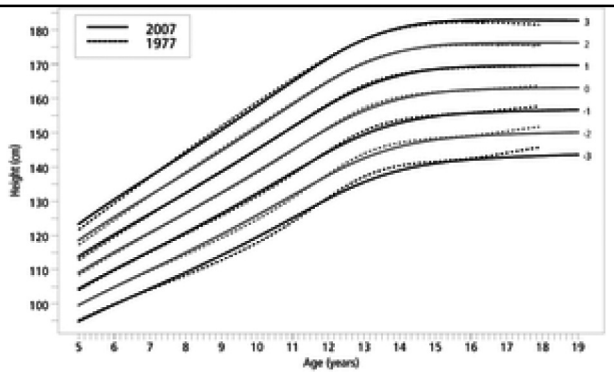

Fig.2. Comparison between the 1977 and 2007 height-for-age *z*-score curves – girls. who.int/bulletin/volumes/85/9/07

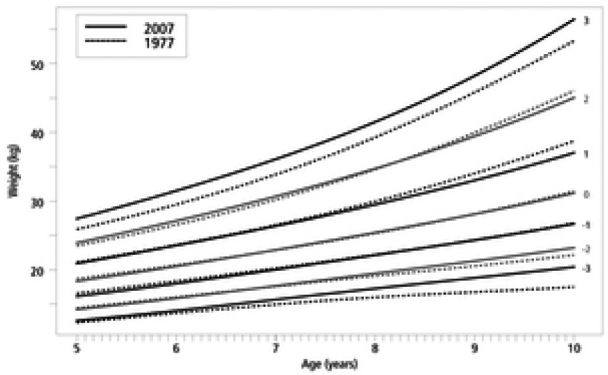

Fig.3. Comparison between the 1977 and 2007 weight-for-age *z*-score curves – boys

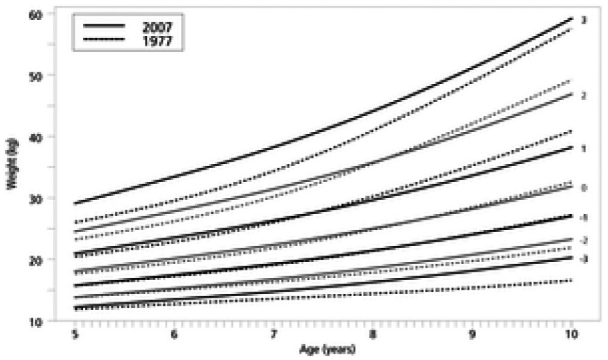

Fig.4. Comparison between the 1977 and 2007 weight-for-age *z*-score curves – girls

(Source: de Onis M, *Development of a WHO growth reference for school-aged children and adolescents in the WHO Bulletin (http//www.who.int/bulletin/volumes/85/9/07-043497/en)*

with the 0 to 5 WHO/MGRS data, using sophisticated statistical tools, namely the Box-Cox power exponential (BCPE) method. The details of these methods are beyond the scope of this review.[8] From 3 health or nutrition surveys, a total sample size of 22 917 (11,410 boys, 11,507 girls) were included. Approximately 320 outliers were excluded. The data from the MGRS was then merged with the NCHS data to create a smooth curve.

The two" height for age" curves closely follow each other with some differentials at the 15 year mark and above (Fig.1 & Fig.2.). There are, however, greater differences between the 2 curves for the "weight for age Z scores", as shown in the figures below (Fig.3 & Fig.4.).

How useful are the 5 to 19 year new growth charts?[9]

Some countries routinely monitor weight alone. They can use the weight for age charts up to age 10, using only weight. After that it would be necessary to use the body mass index (BMI) charts and the weight for height charts. Weight alone is not considered an adequate measure as it is unable to distinguish between relative height and body mass. BMI-for-age is a better measure for this purpose. This should be complemented by the height for age charts, which when used can identify those who are stunted.

The WHO recommends the application of the MGRS charts for all children worldwide, regardless of ethnicity. Several countries have officially adopted the new standards and many others are in the process of doing so. The growth charts and background information are available at www.who.int/childgrowth/en/. Free software is available for download, the WHO Anthro software, for use on personal computers and mobile handheld devices. (http://www.who.int/childgrowth/software/en/)

In conclusion, therefore, it is clear that there are growth reference standards that are applicable across countries, for the ages 0 to 5. Adopting and applying these standards will enable direct comparisons of the state of nutrition of under-5 children across nations, and provide comparable estimates of the levels of malnutrition. The 5 to 19 year charts have been constructed primarily to help detect the problems at the other end of the spectrum, that of excess weight. Use of the old charts may lead to an underestimate of the problem of overweight.[8] Obesity is not an inconsiderable problem in India, and uniform application of these charts can help in building up data on the creeping epidemic of childhood obesity in India.

Points to Remember

- ***Growth standard is a basis for comparison and deviations from the pattern it describes are evidence of abnormal growth.***
- ***To develop a growth chart or standard, children are selected from those living under favourable conditions. Mothers of these children should follow health promoting practices such as breast feeding.***
- ***MGRS chart is developed, based on data collected from cities across six countries, from children brought up in favourable environment.***
- ***New growth charts covering 5-19 year age group have been constructed to detect problems including excess weight.***
- ***For monitoring the growth of a child, weight should be estimated at birth, then every 2 weeks until 2 months of age, thereafter every month till 24 months. After that weight should be checked 6 monthly from 24 to 60 months. Length / height should be measured at birth, repeated at 2 and 6 months and 6 monthly upto 60 months.***

References

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CLIPPINGS

Guilliams K, Rosen M, Buttram S, Zempel J, Pineda J, Miller B, et al. Hypothermia for pediatric refractory status epilepticus. Epilepsia 08/02/2013.

Refractory status epilepticus (RSE) is a life–threatening emergency, demonstrating, by definition, significant pharmaco resistance. The authors describe five cases of pediatric RSE treated with mild hypothermia. This is the largest pediatric case series reporting treatment of RSE with mild hypothermia. Hypothermia decreased seizure burden during and after pediatric RSE and may prevent RSE relapse.

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MANAGEMENT OF LATE PRETERM INFANTS

***Giridhar S**

Abstract: *Late preterm infants range in gestational age from 34 0/7 to 36 6/7 weeks and are at greater risk of morbidity, such as respiratory complications, temperature instability, hypoglycemia, jaundice, feeding problems, neonatal intensive care unit admissions, mortality and adverse neurological sequelae when compared with term infants. They represent 75% of preterm birth and are the fastest growing subgroup of preterm infants. There is an urgent need to educate health care providers and parents about the vulnerability of late preterm infants, who are in need of diligent monitoring and care during the initial hospital stay and a comprehensive follow-up plan for post neonatal and long-term evaluations.*

Keywords: *Premature infant, Respiratory distress syndrome, Neonatal jaundice, hypoglycemia, Mortality.*

Births before 34 weeks of gestation are associated with higher morbidity and mortality which have been extensively studied. In contrast, there are relatively few published data focusing on outcomes of preterm infants born beyond 34 weeks of gestation, largely because of the presumption that these infants are 'almost term' and there is no cause for concern. Now, accumulating research is proving that prematurity by even a single week increases the risk for neonatal morbidity and mortality. Because the preterm birth rate is increasing and the late-preterm group (which constitutes approximately 75% of all preterm births), this class of newborns represents a large and fastest growing sub-set. Those who care for late-preterm infants need to recognize that such infants are physiologically immature even when they appear clinically "stable." All late-preterm infants need to be diligently evaluated, monitored, and followed up.

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Definition

Historically, these babies were classified as 'near-term' since their outcomes were not thought to differ substantially from those infants born at 37 weeks. "Near term" also implied that infants were physiologically "near enough" to term infants and that late preterm pregnancy and offspring could be managed similarly. In 2005, an expert panel, participating in the workshop on "Optimizing Care and Outcome of the Near-Term Pregnancy and the Near-Term Newborn Infant" sponsored by the National Institutes of Health suggested that the phrase "near term" be replaced with "late preterm".[1] The definition of "late preterm" birth, or birth at 34–0/7 through 36–6/7 weeks after the onset of the first day of the mother's last menstrual period, was developed to guide clinical care and research and emphasizes the premature nature of such infants (Fig.1).

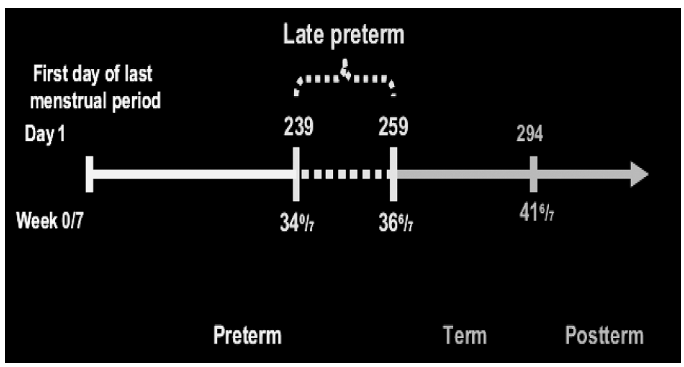

Fig.1. Definitions of various categories of gestational age

Epidemiology

After the 2005 NICHD workshop, there has been a surge in interest in this population of preterm infants. Indian population data is sparse, but there appears to be a concurrence with western studies, in terms of incidence and morbidities. It is generally accepted that late preterm births are increasing. The reason for the increase in late-preterm births is not well understood. One hypothesis is that it may be attributable, in part, to increased use of reproductive technologies and, as a result, an increase in multi fetal pregnancies.[2] Another hypothesis is that advances in obstetric practice have led to an increase in surveillance and medical interventions during pregnancy.

As a result, fetuses considered to be at risk of stillbirth, including those with intrauterine growth restriction, fetal anomalies and intrapartum asphyxia, may be identified earlier, which results in more deliveries at 34 to 36 weeks' gestation. Advanced maternal age and increased incidence of diabetes and hypertension complicating pregnancy also contribute to increased preterm birth rates. A very important and disturbing cause of late preterm birth is patient (and family) requests for deliveries during auspicious dates and deliveries planned as per convenience of the family and obstetrician. Concern about the practice of elective induction or caesarean delivery without medical indication prompted the March of Dimes to launch a national campaign in the USA, 'Healthy babies are worth the wait', to raise awareness among patients and providers on the importance of preventing non-indicated intervention.

Pathophysiology and clinical course

Late preterm infants are physiologically and metabolically immature. A complete understanding of the extent of immaturity in such infants is largely unstudied. Physiologic, anatomic and biochemical deficiencies in late preterm infants predispose to both short- and long-term complications. These include respiratory distress, apnea, hypothermia, hypoglycemia, hyperbilirubinemia, poor feeding, developmental behavioural difficulties and poor social outcomes.

Respiratory Distress: Many studies have confirmed that compared with term infants, all forms of pulmonary disorders occur at higher frequencies in moderate and late preterm infants. These include respiratory distress syndrome, transient tachypnea of the newborn, pneumonia, hypoxic respiratory failure, pulmonary hypertension and apnoeas. Late preterm infants are born during the transition from the terminal saccular period to the alveolar period of lung development. Functional deficiencies in surfactant and clearance of lung fluid, therefore occurs in many late preterm infants, predisposing late preterm infants to respiratory failure. Clearance of fetal lung fluid is controlled by the amiloride sensitive epithelial sodium channels (ENaC's). ENaC expression is developmentally regulated and peak expression in the alveolar epithelium is achieved only at term gestation, which leaves the late preterm infant with lower expression of these channels, thus reducing their ability to clear fetal lung fluid after birth. Thus the cardiopulmonary transition that is necessary immediately after birth for postnatal adaptation may be delayed in late preterm infants, which is reflected in higher rates of retained fetal lung liquid syndrome (transient tachypnoea) and respiratory distress syndrome than in term counterparts. Glucocorticoid surge that occurs during labour, up-regulates expression of ENaC's. Therefore respiratory morbidities are increased in case of preterm births after elective induction and caesarean section. Across studies, incidence of respiratory morbidities in late preterm infants varies from 8% to 30% compared to < 5% in term infants.[3-5] Whereas respiratory issues often tend to be transient in a vast majority of these neonates, some develop into PPHN or severe hypoxic respiratory failure requiring additional therapies such as nitric oxide, high frequency ventilation and extracorporeal membrane oxygenation. Studies have shown that pulmonary hypertension is more likely in late preterm infants who develop RDS than in similar infants born at 32 weeks' gestation. Such predisposition is attributed to a developmental increase in smooth muscle in the walls of pulmonary blood vessels. Keszler, et al reported that late preterm infants delivered by elective caesarean and managed as 'mild TTN' in small hospitals were at great risk for such complications.[6]

Apnoea: The incidence of apnoea in late preterm infants is 4% to 7%,[7] significantly lower than in extremely preterm infants, but significantly greater than in term infants (< 1% to 2%). The brains of such infants are significantly smaller, less myelinated, and contain fewer gyri and sulci than term infants. Therefore predisposition to apnoea occurs by the same mechanisms causing apnoea in early preterm infants. These include increased susceptibility to hypoxic respiratory depression, diminished central chemosensitivity to carbon dioxide, increased sensitivity to respiratory depression with laryngeal stimulation, immature pulmonary irritant receptors and decreased upper airway dilator muscle tone. Generally apnoeas are self-limiting with age and pharmacotherapy is generally not required.

Temperature regulation: Fat accumulation and associated hormone activity in the fetus peaks at term. In late preterm infants, thermoregulation is compromised by low amounts of brown and white fat, immature hypothalamic function and low concentrations of hormones responsible for brown-fat metabolism (such as prolactin, leptin, norepinephrine, triiodothyronine, and cortisol). During the cold stress that follows birth, hypothermia is experienced more often by late preterm infants than term infants.[3,8] The larger surface area-to-weight ratio and smaller size of the late preterm infant also contributes to the higher incidence of hypothermia.

Jaundice: Jaundice occurs more frequently in late preterm infants than term infants. The duration of jaundice is often more prolonged and peak concentrations of indirect bilirubin frequently are higher than found in term infants. The primary

factors causing physiologic indirect hyperbilirubinemia are delayed maturation and lower concentrations of uridine diphosphoglucuronate glucuronosyltransferase, the rate-limiting enzyme for conjugation of bilirubin. The enterohepatic circulation of bilirubin also contributes to bilirubinemia in late preterm infants, especially those infants whose feeding skills are insufficient or whose gastrointestinal motility is slow or impaired. Late preterm infants are twice as likely as term infants to have significantly elevated bilirubin values during the birth hospitalization. In addition, the peak in bilirubin concentration may occur 5 to 7 days after birth in late preterm infants, a time when many such infants are at home.[9] In addition, the neurological immaturity of late preterm infants make them vulnerable to bilirubin induced neurological damage and kernicterus.[10] Parental education and follow-up with a healthcare professional within 2 or 3 days of discharge for those late preterm infants discharged fewer than 3 days after birth are important care management priorities.

Glucose metabolism: Hypoglycemia can occur in newborns at all gestational ages because of insufficient metabolic compensation after the maternal source of glucose is lost following birth. The incidence of hypoglycemia is correlated inversely with gestational age, with late preterm infants having a greater risk than term infants.[11] The susceptibility for hypoglycemia decreases, usually within 12 to 24 hours after birth, because concentrations of enzymes responsible for gluconeogenesis and ketogenesis increase. Late preterm infants are predisposed to hypoglycemia because of immature hepatic glycogenolysis and adipose tissue lipolysis, hormonal dysregulation, deficient hepatic gluconeogenesis and ketogenesis, as well as low glucose reserves.

Feeding and gastrointestinal function: Feeding behaviour and gastrointestinal function are immature in late preterm infants. They can have feeding difficulties because of low oro-motor tone, in coordination of the suck-swallow-breathe sequence and gastrointestinal dysmotility.[12] This predisposes the infant to less calorie intake and dehydration. Importantly, breastfeeding late preterm infants, may be discharged before breast milk volume becomes sufficient and adequacy of breastfeeding, positioning and attachment has been assessed. These infants are often able to handle the low volume of colostrum, but are unable to latch onto the breast or co-ordinate the suck-swallow pattern once more abundant mature milk develops. This problem is compounded in primiparous mothers, who lack prior experience. Hence it is important to assess the late preterm infant for feeding success during the first days and weeks after birth, whether in the hospital or home. If the baby is not taking sufficient milk directly from the breast and supplementary feeds are necessary, expressed breast milk should be given by a spoon or cup. Inadequate feeding also predisposes to hypoglycemia and jaundice.

Immunological immaturity and infection: Immunological immaturity and lack of complete transfer of maternal antibiotics to the fetus prior to 37 weeks, tends to make the late preterm infant vulnerable to infection.[13] Increased incidence of caesarean deliveries, decreased exposure to the protective effects of colostrum due to mother-infant separation and alteration in the intestinal microbiota after hospitalization, all contribute to increased incidence of sepsis.

Mortality: Early and late neonatal mortality in late preterm infants were, respectively, six and three times higher and infant mortality was three times higher than that of term infants.[14] This higher mortality is the indirect result of higher neonatal morbidities seen.

Brain development and long-term development: The late preterm brain weighs about two thirds that of a term infant, has significantly fewer gyri and sulci, and is less myelinated. Furthermore, preterm infants at term ages have relatively immature microstructural cerebral white matter than term infants, indicating that preterm birth has a negative impact on brain development.[15] In a large population of late preterm infants considered healthy at birth and assessed in prekindergarten and kindergarten, significantly more had developmental delay and were unsuccessful in school compared with healthy term infants. The incidence of attention-deficit/hyperactivity disorder is 1.7 times more frequent in late preterm than in term infants. Furthermore, in a large cohort of Swedish young adults, late preterm birth significantly contributed to the overall rates of disabilities in the Swedish population; more than 74% of disabilities occur in the 33 to 38 weeks' gestation cohort. Norwegian adults between 20 and 36 years of age who were born late preterm also were found to have higher rates of medical morbidities: cerebral palsy (2.7 times), intellectual disability (1.6 times), disorders of psychological development, behavior and emotion (1.5 times), schizophrenia (1.3 times). other major disabilities (1.5 times) and any medical disability severely affecting working capacity (1.4 times) than term infants. Immature nervous system structure and function, an adverse intrauterine milieu in pregnancies shortened for maternal or fetal conditions and complications of preterm birth likely contribute to the developmental, behavioral, educational and social disabilities described in late preterm infants.

Management

It should now be clear that late preterm infants are not term infants. It is of great importance that clinicians

treat these infants with the monitoring and care which they deserve and not club them with term infants. It is recommended that even stable late preterm infants be admitted to an area where they can be closely monitored for stability. They should have a physical exam on admission with determination of accurate gestational age. Vital signs and pulse oximeter check should be performed on admission, followed by vital signs every 3–4 hours in the first 24 hours and every shift thereafter. A feeding plan should be developed with formal evaluation and documentation of breast-feeding by caregivers trained in breast-feeding at least twice daily after birth. Serum glucose screening should be performed as per existing protocols for infants at high risk of hypoglycemia. They should be transferred to the mother's room or regular ''term'' nursery only if they demonstrate stability of temperature, vital signs, blood sugar and oral feeding.

Recommended minimum criteria for discharge of late-preterm infants (adapted from American Academy of Pediatrics guidelines)

1. Timing of discharge is individualized and based on feeding competency, thermoregulation and absence of medical illnesses. Late-preterm infants usually are not expected to meet the necessary competencies for discharge before 48 hours of birth.

2. Vital signs should be documented as being within reference ranges and stable for the 12 hours preceding discharge.

3. Infant has good urine output and at least 1 stool has been passed spontaneously.

4. Twenty-four hours of successful feeding and the ability to coordinate sucking, swallowing and breathing while feeding has been demonstrated. Any infant with a weight loss of more than 2% to 3% of birth weight per day or a maximum of 7% of birth weight during the birth hospitalization should be assessed for evidence of dehydration before discharge.

5. A pre-discharge bilirubin check has been done and plotted on a bilirubin normogram to determine the need for follow-up or treatment.

6. Physical examinations of the infant reveal no abnormalities that require continued hospitalization.

7. A follow-up visit should be arranged 48-72 hours post-discharge to assess for jaundice and adequacy of breast-feeding. Additional visits may be indicated until an established and maintained pattern of weight gain has been demonstrated.

8. The mother and caregivers have received information or training or have demonstrated competency in the following:

a. infant's hospital course and current condition

b. expected pattern of urine and stool frequency for the breastfeeding infant

c. umbilical cord, skin, and newborn genital care

d. hand hygiene, especially as a means to reduce the risk of infection

e. use of a thermometer to assess an infant's axillary temperature

f. assessment and provision of appropriate layers of clothing

g. identification of common signs and symptoms of illness, such as hyperbilirubinemia, sepsis, and dehydration; assessment for jaundice

Points to Remember

- ***Late-preterm infants are immature.***
- ***Infants born at 34 0/7 through 36 6/7 weeks gestation (239–259 days since the first day of the last menstrual period) should be referred to as "late preterm."***
- ***Late-preterm infants are physiologically immature and have limited compensatory responses to the extrauterine environment compared with term infants.***
- ***Late-preterm infants are at a greater risk of morbidity and mortality than are term infants.***
- ***During the birth hospitalization, late-preterm infants are more likely than are term infants to be diagnosed with temperature instability, hypoglycemia, respiratory distress, apnoea, jaundice, infections, feeding difficulties or mortality.***
- ***During the first month after birth, late-preterm infants are more likely than term infants to be rehospitalized for jaundice, feeding difficulties, dehydration, and suspected sepsis.***
- ***Collaborative counseling by both obstetric and neonatal clinicians about the outcomes of late-preterm births is warranted unless precluded by emergent conditions.***

- ***Appropriate discharge criteria and comprehensive follow-up plan needs to be implemented for this special population if infants.***

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CLIPPINGS

Fluoride varnishes for preventing dental caries in children and adolescents

Topically-applied fluoride varnishes have been used extensively as an operator-applied caries-preventive intervention for over three decades.

Objectives: To determine the effectiveness and safety of fluoride varnishes in preventing dental caries in children and adolescents, and to examine factors potentially modifying their effect.

Selection criteria: Randomised or quasi-randomised controlled trials with blind outcome assessment used or indicated, comparing topically-applied fluoride varnish with placebo or no treatment in children up to 16 years during at least one year. The main outcome was caries increment measured by the change in decayed, missing and filled tooth surfaces in both permanent (D(M)FS) and primary (d(e/m)fs) teeth.

Authors' conclusions: The conclusions of this updated review remain the same as those when it was first published. The review suggests a substantial caries-inhibiting effect of fluoride varnish in both permanent and primary teeth, however the quality of the evidence was assessed as moderate, as it included mainly high risk of bias studies, with considerable heterogeneity.

Marinho VCC, Worthington HV, Walsh T, Clarkson JE. Fluoride varnishes for preventing dental caries in children and adolescents. Cochrane Database of Systematic Reviews 2013, Issue 7. Art. No.: CD002279. DOI: 10.1002/14651858.CD002279.pub2.

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FEEDING DISORDERS IN INFANTS: 6 TO 24 MONTHS

Sathiyasekaran M

Abstract: *Pediatric feeding disorders are challenging problems encountered commonly in day to day practice. 25% of normal children present with a mild disorder which increases to 80% in children with developmental delay. The etiology is multifactorial comprising of medical, nutritional, behavioral, psychological and environmental causes. Feeding disorders should be conceptualized as a bio-behavioral problem, a continuum between psycho-social and organic factors. The clinical spectrum includes food selectivity, food refusal, excessive meal duration, dysphagia, choking, vomiting and inappropriate mealtime behaviors. Nutritional and cognitive impairment, growth failure, susceptibility to chronic illness and even death may occur as a result of this disorder. Assessment and treatment are best conducted by an interdisciplinary team including a pediatrician, gastroenterologist, nutritionist, behavioral psychologist and occupational and/or speech therapist.*

Keywords: *Feeding disorder, Bio-behavioral, Interdisciplinary team.*

Feeding disorder of infancy or early childhood is characterized by the failure (inability or refusal) of an infant or child to orally consume appropriate or adequate food for age that is required to gain weight and grow normally over a period of one month or more.[1]

Physiology of regulation of feeding and swallowing in infants

Feeding is a complex process and comprises of five phases: pre oral, oral, pharyngeal, esophageal and gastro intestinal.[2] Any condition which affects any one of the five phases results in a feeding disorder.

Pre oral phase: This phase involves appropriate food being provided and introduced into the oral cavity. This is initiated when the infant senses hunger and communicates the desire to eat. Normally the hypothalamus, sensory pathways, adipose tissue and endocrine organs control hunger and satiety. In addition, inflammatory cytokines, medications, emotional state of the child, painful or uncomfortable experience during a feeding episode, ability of the caretaker to understand and identify hunger cues in an infant play a very crucial role in feeding.

Oral phase: During this phase food bolus passes into the pharynx and involves mouthing, sucking and initiation of swallowing which appear inutero during different gestational age (Table I).[3] Exposure to amniotic fluid flavours which may change with maternal diet also occurs in utero. After birth the infant acquires feeding skills as shown in (Table II).[2] These critical or sensitive periods are important feeding mile stones in the infant and if not attained at the appropriate age may lead to a feeding disorder. Feeding and swallowing skill development parallels psychosocial milestones of homeostasis (0-3m), attachment (3-6m) and separation/individuation(6-36m).[4]

Table I. In utero development of swallowing function and gestational age[3]

Swallowing function	Gestational age (weeks)
Mouthing and sucking	13
Pharyngeal swallow	10 - 14
Mature taste receptors	12
True suckling	18 - 24
Tongue cupping	28
Sustain nutrition totally orally	34 - 37

Pharyngeal phase: This is an involuntary process and helps to propel the food into the esophagus without causing aspiration. Aspiration may occur if there is swallow-breathing, oro-pharyngeal or pharyngo-esophageal inco-ordination and occurs more often in preterm, those with cardio respiratory distress, congenital laryngeal abnormalities, myopathies and CNS abnormalities.

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Table II. Age and development of feeding skills[2]

Feeding pattern/skill	Age of child
Non-nutritive pattern of tongue movement extension and retraction	Birth to 2 months
Lateral movement which allows some bolus manipulation.	3 to 4 months
Soft food from a spoon	6 months
Mature form of mastication is seen(transition feeding).	6 to 8 months
Sucking is replaced by cup drinking and spoon feeding	12 months
Effective handling, crushing and grinding of food performed by rotatory chewing skills and lateral activity of the tongue	18 to 24 months
Eats regular food and drink from an open cup.	36 months
No new skill > 36 m	

Esophageal phase: The effective functioning of the upper and lower sphincters and propulsive peristalsis of the esophagus is essential for swallowing, transfer of food into the stomach and preventing aspiration. Anatomic and functional disorders and inflammation of the esophagus may present as feeding disorders.

Gastrointestinal phase: The receptive capacity of the stomach and its ability to empty the contents into the duodenum are essential for feeding and attaining satiety. Both delayed (gastroparesis) or rapid emptying(dumping) cause post prandial discomfort which in turn could lead to feeding disorder. Similarly motility disorders of the intestine and colon either neuropathic, myopathic or functional disorders could cause inappropriate fullness after feeds and feeding disorders.

Epidemiology

Incidence: Feeding disorders are common in early childhood with 25 % to 35 % of normal children presenting with mild feeding disorder and increases to 33-80 % in children with developmental delay.[5,6] Severe feeding problems occur in 3 to 10 % and are more common in preterm babies (10-49%),[1] those with physical disabilities (26-90%) and chronic illness. A relative increase may occur with improvement in preterm survival.[1,7] There is no data available from India.

Age: The mean age of presentation was 25+/- 4 months and 50 % of the children were less than 1 year in Rommel's series.[8] Medical and oral categories occurred more often in children less than 2 years of age and behavioral feeding problems in children above 2 years of age.[8]

Sex: Feeding disorder is seen in both sex equally.

Pre disposing factors: Some important predisposing factors are:

a) Prematurity: Infants born preterm and/or with a birth weight below the tenth percentile for their gestational age (GA) are at a greater risk of developing feeding disorder. A gestational age of 34 weeks is considered critical for development of efficient feeding and tolerance. There is a significant correlation between feeding disorders and ventilation, aspiration and nasogastric tube feeding indicating that medical intervention may influence oral feeding skills.[8]

b) Maternal /caretaker factors: Emotional disturbances such as depression, anxiety in a parent, inappropriate parent-child interactions, such as failure to read the child's hunger cues or forcing food when the child is not hungry may trigger the disorder.

c) Psychosocial factors: Child abuse, neglect, separated families, poverty and stress increase risk.

Classification of feeding disorders and diagnostic criteria

Over the past 20 years various classifications have been proposed. The division into organic (medical and nutritional) and non organic(psychological and behavioral) is not recommended.

The 5 common classifications of feeding disorders are shown in Table III.[9] A practical and acceptable categorization is oro pharyngeal, medical, behavioral and combination.[8]

Table III. Classification of feeding disorders[9]

Classification	Criteria /subgroups
I. Feeding disorders of infants and early childhood in "Diagnostic and Statistical Manual of Mental Disorders" (DSM-IV-TR)	a. Persistent failure to eat adequately as reflected by significant failure to gain weight or significant weight loss over at least one month. b. The disturbance is not due to gastro intestinal or other general medical condition. c. The disturbance is not better accounted for by another mental disorder (e.g., rumination disorder) or by lack of available food. d. The onset must be before the age of 6 years.
II.Feeding disorders in infancy and early childhood in"Diagnostic Classification of Mental Health and Developmental Disorder of Infancy and Early Childhood" (DC:0-3 R)	The 6 categories of "Feeding Behavior disorder" are: 1. Feeding disorder of state regulation.(0-2m) 2. Feeding disorder of care giver-infant reciprocity.(2-6m) 3. Infantile anorexia.(6m-3yrs) 4. Sensory food aversions. 5. Feeding disorder associated with concurrent medical condition. 6. Feeding disorder associated with insults to the GI tract or Post traumatic feeding disorder. The recent (DSM-V) 2013 includes 3 of Chatoor's categories 1.infantile anorexia, 2.sensory food aversion and 3.post traumatic feeding disorder
III. Complex Bio-Behavioral Pediatric Feeding Disorders (Burklow 1998)	(1) Structural abnormalities, (2) neurological conditions, (3) behavioral and psychosocial issues (4) cardio-respiratory problems, and (5) metabolic dysfunction
IV. Behavioral Pediatric Feeding Problems (Crist and Napier-Philips, 2001)	(1) Picky eaters, (2) toddler refusal-general, (3) toddler refusal-textured food, (4) older children refusal-general and (5) Stallers
V. Food refusal behaviors (Dovey et al, 2009)	(1) Learning dependent food refusal; (2) medical complications related food refusal; (3) selective food refusal; (4)fear based food refusal; (5) appetite awareness and autonomy based food refusal.

Aetiology

The etiology of feeding disorder is multifactorial comprising of nutritonal, structural abnormalities (nasopharynx, larynx, esophagus), neuro developmental disabilities, chronic illness behavioral and psychosocial (Table IV).[2] The proportion of medical, structural abnormalities or behavioral disorders depend on the centre which caters to these children. Burklow identified 80% with a significant behavioral component.[10] Romell, et al showed that medical disorder and behavioral problems could occur either alone or in combination and 86% had a medical disorder, 61% oropharyngeal dysfunction and 18% had a behavioral problem.[8]

Clinical presentation of feeding disorders: Children with feeding disorders may be brought with various symptoms such as difficulty in breast feeding, arching or stiffening of the body during feeds, food preferences, food refusal, food selectivity by type, texture or color, inappropriate meal time behaviors, excessive meal duration, storing food in mouth, gurgling, hoarse voice, vomiting, drooling of food, spitting, recurrent pneumonia, limited intake of food, food aversion, food phobia, less than normal weight gain or growth.

Natural history and potential consequences of feeding disorders: The majority of mild feeding difficulties especially those seen in normal children are transient and resolve without significant clinical disturbances. Dahl, et al reported that spontaneous resolution does not occur though growth and development are not compromised.[11] The outcome of feeding disorders secondary to a disease

depends on the management of the medical condition. Prolonged feeding disorders can result in cognitive impairment, emotional dysfunction, malnutrition, growth retardation and even death.

Evaluation of an infant with feeding disorder

History: A detailed history should be elicited including antenatal and perinatal history, family history of atopy or feeding problems; previous illnesses and hospitalizations and manipulation around the oropharynx, such as tube feeding. The chronology of feeding problems, diet since birth, changes of formulas, introduction of solids, current diet, textures, route and time of administration, and feeding posture should be recorded. Food aversions, quantities eaten, length of meals and associated routines, strategies already used, environment and behaviour around mealtimes need to be documented. In healthy children a meal time of more than 30 minutes may indicate a feeding disorder whereas in other children long meal times may indicate reflective or ineffective mechanics. History of recurrent pneumonia, stridor, snoring may point to upper airway disease that affect swallowing. Recurrent vomiting and abdominal pain could suggest gastroesophageal reflux disease or allergy to cow's milk. Specific food aversions may be a clue to food allergies, inborn errors of metabolism or sensory food aversions. Nutritional and psychological assessment should be done early during the period of evaluation.[2]

Observation of a feeding session: Observation is preferably done undisturbed using a video camera, both positive as well as negative interactions between the child and parent and the child's response during meals are observed.

Physical examination: The infant or child may appear normal or be irritable, difficult to console, apathetic and withdrawn: In severe forms growth retardation and development delay are obvious.

i. Anthropometric measurements, head circumference and growth chart documented since birth help to calculate the percentiles and assess severity

ii. Detailed evaluation during feeding such as muscle tone, posture, vomiting, gagging, choking is informative.

iii. Variation in articulation and voice quality should be identified since oropharyngeal disorders can affect both speech and swallowing.

iv. Craniofacial abnormalities, cleft lip, cleft palate, macroglossia should be documented.

v. Examination of the abdomen, respiratory, nervous and cardiovascular system is mandatory.

vi. Evaluation of psychomotor development. Only after a thorough physical examination and excluding any medical cause should a primary behavioral feeding disorder be considered.

Investigations: Children with normal physical examination, development and growth do not require any specialized investigation. Children with complex or severe form of feeding disorder with growth retardation or nutritional deficiency and those with underlying diseases need complete evaluation.[12,13]

A.Base line investigations: A complete blood count, ESR, urine and stool examination, serum albumin and protein, blood sugar, electrolytes, serum iron, iron-binding capacity, serum ferritin, creatinine, liver enzymes and prothrombin time are included. Xray chest should be done to identify structural, parenchymal diseases and cardiomegaly.

B.Investigations to confirm the underlying medical condition: These tests depend on the underlying medical disorder (Table IV)

i. Blood: eg. tissue transglutaminase antibody for celiac disease, screening for food allergies and metabolic disorders.

ii. Barium studies to identify esophageal stenosis, strictures, webs, achalasia, malrotation.

iii. Ultrasound of the abdomen for diseases of the pancreas, liver, kidney and peritoneum.

iv. Upper GI endoscopy with duodenal biopsy for mucosal lesions of the esophagus, stomach and duodenum.

v. Magnetic resonance imaging of the brain stem, skull base and spine when the swallowing problem is secondary to involvement of the lower cranial nerves.

vi. Computerised tomography of the chest for pulmonary or mediastinal lesions causing dysphagia and assess severity of chronic lung disease.

C. Investigations to assess oral, pharyngeal and esophageal swallowing disorders

These tests are done when there is risk of aspiration or incoordination of suck-swallow-breathing.[14]

Videofluroscopic swallowing study(VSS /VFSS): This is a relatively non invasive method of studying the oral and pharyngeal phases of swallow and helps in deciding the conditions and consistencies for a safe swallow.

Table IV. Causes of feeding disorders

I.Disorders that affect appetite, food seeking behavior and ingestion Depression, deprivation, poverty CNS disease: Diencephalic syndrome, maternal factors. **Metabolic disease** Hereditary fructose intolerance, urea cycle disorders, organic acidemia. **Sensory defects** Anosmia, blindness neuromuscular diseases, oral hypersensitivity, or aversion:due to lack of feeding during critical period. **Conditioned dysphagia** Aspiration, oral inflammation, gastro esophageal reflux, dumping syndrome post surgical.	**V.Disorders affecting suck/swallowing and breathing co ordination** Choanal atresia., cardiac disease, respiratory diseases with tachypnea.
II.Anatomical abnormalities of the oropharynx Cleft lip/palate, Pierre Robin syndrome macroglossia, tonsillar hypertrophy, caries teeth, retropharyngeal abscess.	**VI.Disorders affecting neuromuscular co ordination of swallowing** Cerebral palsy, brain stem glioma/myelomeningocele, Arnold Chiari malformation, postdiptheritic/polio paralysis, mysathenia gravis, myotonic dystrophy, muscular dystrophies and myopathies, cricopharyngeal achalasia, polymyositis/dermatomyositis, rheumatoid arthritis.
III.Anatomic/congenital abnormalities of the larynx /trachea Laryngeal cleft,laryngomalacia,laryngeal cyst, subglottic stenosis, tracheomalacia, tracheosophageal cleft, tracheo esophageal compression by vascular ring/sling.	**VII.Disorders affecting esophageal peristalsis.** Achalasia, pseudo intestinal obstruction, scleroderma, polymyositis/dermatomyositis, mixed connective tissue disease.
IV.Anatomic abnormalities of the esophagus Tracheo esophageal fistula, congenital esophageal atresia/stenosis., esophageal web, stenosis, stricture, foreign body/esophageal mass or tumor, dysphagia lusoria,vascular ring.	**VIII.Mucosal infections and inflammatory diseases causing dysphagia** Adeno-tonsillitis, esophagitis: CMV, herpes, candida, HIV.Crohn's disease, corrosive ingestion.
	IX.GI disorders causing decreased appetite and feeding disorders Short bowel syndrome, constipation, Hirschsprung's disease, chronic pancreatitis, chronic cholestasis.
	X.Miscellaneous conditions causing feeding and swallowing difficulties Chromosomal: Williams, Prader Willi, Cornelia de Lange, Trisomy 18, 21. Endocrine:Hypothyroidism, hyperparathyroidism, Epidermolysis Bullosa dystrophica.

Fibreoptic endoscopic evaluation of swallowing (FEES): A flexible scope is introduced through the nares into the oropharynx and the pharyngeal phase of swallow is evaluated.

Oesophageal manometry: This helps in evaluating motility disorders of the esophagus like achalasia.

D.Identifying behavioral feeding disorders: A novel tool IMFeDTM (Identification and Management of Feeding Difficulties) which helps to classify common feeding problems based on the parent's response to queries in a questionnaire is available. The kit includes parental guidance sheets and available therapeutic options

Management of feeding disorder: Management of the complex feeding disorder is best conducted by an interdisciplinary team[15] and should be comprehensive and include treatment of the medical condition, behavioral modification and parent education and training in appropriate parenting and feeding skills.[15]

A. **Reassurance:** When infants with mild feeding disorder are growing and developing normally no intervention is required and pediatricians can reassure parents.

B. Nutrition: i. Dietary intervention may be helpful in an infant with colic and vomiting. If cow's milk allergy is suspected exclusion of bovine milk in mother's diet and hypoallergenic formula for infants not breastfed may relieve symptoms.

ii. Dietary interventions play a definite role in inborn errors of metabolism such as hereditary fructose intolerance, urea cycle disorders and organic acidemia. Gluten free diet is a must for children diagnosed with celiac disease.

iii. Fortification of diet: Caloric intake should be increased if a child's growth appears to be stunted. Human milk and infant formula can be fortified with formula powder, carbohydrate, or fat. up to 3.4 to 4.2 kcal/mL. Solid food can be fortified with butter, vegetable oil, glucose polymers, and powdered milk. Calories may be improved with enteral formulas.

iv. Route of feeding: Oral route is always preferred and should be encouraged. Nasogastric feeding and gastrostomy (PEG) feeds are recommended as supplements for infants with chronic illness or neurodevelopmental disorder who are unable to ingest adequate calories. Feeding gastrostomy is also helpful when there is a possibility of aspiration or time per feed impedes with other nurturing activities. It is always preferable to continue safe oral feeding Parenteral alimentation is not an option except in a very sick infant when the gut is not functioning

C.Medications: In infants with GERD when symptoms persist in spite of thickening of feeds and hypoallergenic formula, oral proton pump inhibitors may be introduced. Prokinetics may be of benefit in children with pseudo intestinal obstruction or gastroparesis. Appropriate treatment of constipation is beneficial. Medical problems such as myasthenia gravis, CCF, should be treated.

D.Endotherapy: Endoscopic dilatation of esophageal stenosis, webs and strictures is very rewarding in children with feeding difficulties due to these disorders.

E.Surgery: Children with normal neurologic function generally do well after surgical correction of anatomic abnormalities of oropharynx, larynx, esophagus, etc. Neurologically disabled children, however, need nasogastric or gastrostomy feedings.

F.Behavioral interventions: Infants who are able to swallow but are unwilling to eat may benefit from behaviour modification programs. Behavioural interventions vary depending on the category of the disorder.

G.Occupational therapy or speech-language pathology interventions. Infants with cerebral palsy may benefit with this form of therapy. These interventions focus on oral motor skills (eg.increasing strength and range of motion of oral motor structures, preventing hypersensitivity, promoting proper positioning, bolus modification)

Prevention: Behavioral feeding disorders can be prevented by creating a pleasant environment and positive child parent interactions. Force feeding and punishing for not eating should be avoided. Prevention of sensory food aversions is by introducing various foods at 4 to 6 months of age. New foods should be introduced singly with patience. Prolonged nasogastric feeding and frequent change of tubes if minimized could prevent post traumatic feeding disorder.

Points to Remember

- ***Feeding disorders in young infants are common.***
- ***Etiology is multifactorial and may be a combination of medical and behavioral.***
- ***Symptoms range from food selectivity, vomiting to complete food refusal.***
- ***Majority resolve but some may persist resulting in in cognitive impairment, emotional dysfunction, malnutrition and growth retardation.***
- ***Assessment and management of complex disorder is best done by a multi disciplinary team.***

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CLIPPINGS

Hartling L, Newton AS, Liang Y, Jou H, Hewson K, Klassen TP, et al. Music to reduce pain and distress in the pediatric emergency department: A randomized clinical trial. JAMA Pediatrics 07/16/2013

Many medical procedures aimed at helping children cause them pain and distress, which can have long–lasting negative effects. Music is a form of distraction that may alleviate some of the pain and distress experienced by children while undergoing medical procedures. Music may have a positive impact on pain and distress for children undergoing intravenous placement. Benefits were also observed for the parents and health care providers. A randomized clinical trial conducted in a pediatric emergency department with appropriate sequence generation and adequate allocation concealment from January 1, 2009, to March 31, 2010. Individuals assessing the primary outcome were blind to treatment allocation. A total of 42 children aged 3 to 11 years undergoing intravenous placement were included. Music (recordings selected by a music therapist via ambient speakers) vs standard care.

The observations were as follows: With or without controlling for potential confounders, they found no significant difference in the change in behavioral distress from before the procedure to immediately after the procedure. When children who had no distress during the procedure were removed from the analysis, there was a significantly less increase in distress for the music group (standard care group=2.2 vs music group=1.1, P<.05). Pain scores among children in the standard care group increased by 2 points, while they remained the same in the music group (P=.04); the difference was considered clinically important. The pattern of parent satisfaction with the management of children's pain was different between groups, although not statistically significant (P=.07). Health care providers reported that it was easier to perform the procedure for children in the music group (76% very easy) vs the standard care group (38% very easy) (P=.03). Health care providers were more satisfied with the intravenous placement in the music group (86% very satisfied) compared with the standard care group (48%) (P=.02).

NEWS AND NOTES

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NEPHROTIC SYNDROME IN CHILDREN– AN UPDATE

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Abstract: *The commonest type of nephrotic syndrome seen in children is idiopathic nephrotic syndrome. Dysregulation of T cells was considered as the cause for proteinuria in earlier days. But better understanding of the molecular mechanisms lead us to think about various emerging new theories about proteinuria. Hypothesis about the mechanism of edema is also changing, with more focus on tubular epithelial sodium channels and others. Evaluation, management and complications are very specific in childhood nephrotic syndrome.*

Keywords: *Nephrotic syndrome, Diuretic resistance, Steroid sparing drugs, Rituximab, Stress therapy.*

The most common cause of nephrotic syndrome (NS) in children is idiopathic nephrotic syndrome (INS), also called as nephrosis.[1] Nephrotic syndrome is characterized by nephrotic range of proteinuria (>50 mg/kg/day), hypoalbuminemia (S.Albumin<2.5 g/dL), hyperlipidemia (S.Cholesterol>200mg/dL) and edema. Nephrotic children in relapse can present without edema as > 2+ proteinuria by dipstick alone, documents a relapse. Nephrotic range proteinuria is also documented with early morning urine protein of 3+/4+ (on dipstick or turbidometry method) or spot urine protein/creatinine ratio >2 mg/mg or urine albumin excretion >40 mg/m^2/hr on a timed-sample. As per Indian Society of Pediatric Nephrology (ISPN) guidelines, precise quantitative assessment of proteinuria, including 24-hour urine protein measurement is seldom necessary.[2,3]

Idiopathic NS is histologically characterized by minimal change disease, focal and segmental glomerular sclerosis (FSGS) and diffuse mesangial proliferation. Though INS is most common in children (90%), various other causes have also been found to be associated with NS. Infections like hepatitis B, hepatitis C, malaria and human immunodeficiency virus can cause NS. Among the systemic diseases, Henoch-Schönlein Purpura and systemic lupus erythematosus can manifest as NS. One of the presentations of hemato-oncologic diseases like leukemia and lymphoma is NS. Apart from these causes, drugs like non steroidal anti-inflammatory drugs (NSAIDs) and penicillamine, bee sting, food allergy and rarely metabolic disorders like glycogen storage disorders and Fabry disease can present as NS.[1] Common definitions used in nephrotic syndrome are given in Table 1.[2] These definitions are very useful to define the stage of the disease and to term the response to steroids in NS.

Pathogenesis of NS

For a long time NS in children was considered idiopathic and hence primary only. Initially allergic conditions and infections were considered as causes but later with better understanding of the disease process now the pathogenesis of NS in children is being discussed as due to immunological dysfunction.

Dysregulation of T cells was considered as the cause for proteinuria in earlier days. But better understanding of molecular mechanisms of glomerular filtration barrier has made us to think about various emerging new theories about proteinuria. The podocyte, one of the essential components of glomerular basement membrane plays a main role in the filtration mechanism. The multiprotein complex between adjacent podocyte foot processes, the slit diaphragm, is essential for the control of the actin cytoskeleton and cell morphology of podocytes.[4] Mutations in genes encoding podocytes and slit diaphragm proteins, including nephrin, podocin, transient receptor potential-6 channel, Wilms' tumor 1 gene and alpha-actinin-4 have been identified via genetic studies of inherited NS.[5] Circulating permeability factors like IL-13 and nuclear factor kappa B (NF-kB) which alters the glomerular filtration barrier can cause proteinuria. Finally the response to rituximab in NS, suggests a role for B cell involvement and/or aberrant B cell–T cell crosstalk in the causation of NS.[6]

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Table I. Commonly used definitions in NS

Remission	Urine albumin nil or trace (or proteinuria <4 mg/m^2/h) for 3 consecutive early morning specimens.
Relapse	Urine albumin 3+ or 4+ (or proteinuria >40 mg/m^2/h) for 3 consecutive early morning specimens, having been in remission previously.
Frequent relapsing nephrotic syndrome (FRNS)	Two or more relapses in initial six months or more than three relapses in any twelve months.
Steroid dependent nephrotic syndrome (SDNS)	Two consecutive relapses when on alternate day steroids or within 14 days of its discontinuation.
Steroid resistant nephrotic syndrome (SRNS)	Absence of remission despite therapy with prednisolone at a dose of 2 mg/kg/day for 4 weeks.

Edema in NS

Edema is the classic clinical presentation of nephrotic syndrome. The mechanisms of edema formation in NS have long been a subject of investigation. The 'under fill' hypothesis states that a decrement in oncotic pressure leads to excess filtration of fluid from the intravascular space to the interstitial space, causing hypovolemia, renal hypoperfusion, which in turn activates the renin–angiotensin-aldosterone system and secondary renal sodium retention. The 'overfill' hypothesis states that the nephrotic proteinuria causes primary renal sodium retention, leading to edema.[7]

Recently proposed mechanism of edema is sodium retention regardless of intravascular volume status and is due to the activation of epithelial sodium channel by serine proteases in the glomerular filtrate of nephrotic patients.[8] Plasminogen filtered by the nephrotic glomeruli is converted to plasmin by urokinase-type plasminogen activator in the cortical collecting duct cells. Plasmin then proteolytically removes the inhibitory domain from epithelial sodium channels (ENaC), resulting in near full activation of ENaC. Other proposed mechanisms of edema formation include a) increased angiotensin II-independent afferent and efferent arteriolar tone because of the increased efferent sympathetic nervous system activity, b) tubular resistance to atrial natriuretic peptide and c) increased number and activity of cortical collecting duct Na/K ATPase channels.[8]

Evaluation of NS

Initial evaluation should include measurement of height, weight, blood pressure and physical examination for documenting the severity of edema, focus of sepsis and signs of systemic illnesses like skin rashes, arthritis, hepatosplenomegaly etc. Baseline investigations should include urinalysis, complete blood count, renal function tests, serum albumin and cholesterol estimation, Mantoux testing, X-ray chest and ultrasonogram of the abdomen. Second line investigations like serum complement C3 and C4 levels, antinuclear antibody and hepatitis B surface antigen are needed in children presenting with systemic features as per need.

Management of NS

This includes both supportive and specific management.[3] General supportive measures include modulation in diet, salt and fluid intake. A normal salt containing balanced diet with adequate proteins (1.5-2.0 g/kg) and calories is recommended. Salt is restricted to 1-2 g per day in children with persistent edema. Fluid restriction is necessary in addition to the sodium restriction in these children. In severe edema, intake of fluid volume should be restricted to insensible water loss. In moderate edema, previous day's urine output and insensible water loss should be allowed. In mild edema, usually fluid is restricted minimally.[9] Children with NS can be allowed to have full activity unless they are sick.

Diuretics in edema management

In children with moderate to severe edema causing discomfort, diuretics should be added. Furosemide increases sodium delivery to the distal nephron. Under normal circumstances only a small fraction of the sodium is reabsorbed from the distal nephron and this leads to natriuretic effect. In NS, due to induction of Na, K-ATPase channels, there is an increase in sodium reabsorption thereby reducing natriuretic effect of furosemide. This can be overcome by co-administration of amiloride which inhibits the distal sodium reabsorption by blocking the ENaC channels and thereby overcomes furosemide resistance.[10] Hence, it is always advisable to use combination of diuretics

either in the form of loop/thiazide or loop/amiloride. Apart from this, few other causes should also be considered in resistant edema, like impaired absorption of oral diuretics due to bowel wall edema, which might reduce the drug's bioavailability and hence intravenous drugs are preferred in such situations. Furosemide resistance in NS may also be caused by binding of the diuretic to tubular fluid albumin. To prevent this, higher doses of furosemide can be tried to overcome binding of the drug to tubular fluid instead of giving as divided doses.[11] Continuous infusion of the loop diuretics has greater diuretic potency than the same total dose of drug given intermittently. In refractory edema, 20% albumin infusion (1gm/kg) can be tried. It is ideal to give furosemide at the end of infusion in order to prevent pulmonary edema. Thus plasma volume expansion with IV albumin might play an adjunctive role in the diuretic management. Albumin is an expensive medicine with only short-lasting benefit. Infused IV albumin is rapidly excreted into the urine. It should be reserved for the most resistant, severe and symptomatic edema. Head out water immersion is a cumbersome procedure tried in older days in children with nephrotic syndrome.[12] Dialysis in the form of peritoneal or hemodialysis can be performed in children with resistant edema to achieve ultrafiltration thereby to reduce edema, which of course is not routinely done.[9]

Specific management

The ultimate goal of our long-term management is to induce the remission; maintain it and at the same time minimize the side effects of the medications. Nephrotic syndrome should be treated adequately with corticosteroids, both in terms of dose and duration, which is an important determinant of the long-term course of the disease. The standard medication for treatment is prednisolone. Initial episode of nephrotic syndrome should be treated with prednisolone at a dose of 2 mg/kg per day (maximum 60 mg in single or divided doses) for 6 weeks, followed by 1.5 mg/kg(maximum 40 mg) as a single morning dose on alternate days for the next 6 weeks followed by tapering over 2 to 4 weeks.[2] By 4 weeks of daily steroid therapy if child does not go into remission they are considered to be" steroid resistant".

In children presenting with relapse, always look for the precipitating factor, which most often is an infection. Adequate treatment of the infection may result in spontaneous remission. Relapse is treated with prednisolone 2mg/kg/day (single or divided doses) until urine protein is trace or nil for three consecutive days. Subsequently, prednisolone is given in a single morning dose of 1.5 mg/kg on alternate days for 4 weeks, and then discontinued.[2] Thus the usual duration of treatment for a relapse is 5-6 weeks. In children who do not attain remission despite two weeks of adequate treatment with prednisolone, it is extended for 2 more weeks. If they do not show remission despite 4 weeks of daily prednisolone, they are termed as" late steroid non-responders" and should be referred to pediatric nephrologist for further evaluation.

Role of steroid sparing drugs in NS

Levamisole: Children with frequently relapsing NS and steroid dependent NS in whom steroid threshold is high are the ideal candidates to be treated with levamisole. It is an immunomodulator and is given in a dose of 2.0-2.5 mg/kg on alternate days for 12-24 months along with steroids at a dose of 1.5 mg/kg on alternate days to start with, gradually tapering it to a maintenance dose of 0.25-0.5 mg/kg that is continued for 6-12 months. One should understand that levamisole is used to achieve a steroid sparing effect, ideally to be started after inducing remission. Adverse effects are leukopenia, flu-like symptoms, liver toxicity, convulsions and skin rashes. Total leukocyte count should be monitored every 12-16 weeks.[2]

Cyclophospamide: It is a cytotoxic alkylating agent which depletes cells of the immune system. Again the indications for use of cyclophosphamide are FRNS and SDNS. It has also been tried in children with SRNS with histopathology showing minimal change disease and minimal mesangial proliferation with variable results. Both oral and intravenous preparations are available with its advantages and disadvantages. It is given as 2.0-2.5 mg/kg/day for 12 weeks orally or intravenous monthly pulses of 6 doses, ensuring that the cumulative dosage does not exceed 168mg/kg/course. It should be instituted preferably following remission of proteinuria in FRNS and SDNS. Prednisolone is always co-administered for 6 to 12 months.[2]

Short term side effects are nausea, vomiting, hemorrhagic cystitis, leucopenia and alopecia. Total leukocyte counts are monitored every 2 weeks; drug should be temporarily discontinued if the blood count falls below 4000/mm.[3] An increased oral fluid intake and frequent voiding prevents the complication of hemorrhagic cystitis. Long-term side effects are gonadal toxicity, which is common if the recommended dose is exceeded and with use in post-pubertal males and rarely malignancy. Gonadal toxicity can be restricted with a single course of recommended dose only.

Mycophenolate mofetil (MMF): It is an anti-proliferative agent that inhibits T&B lymphocyte proliferation by inhibition of inosine monophosphate dehydrogenase

which is a key enzyme in purine biosynthesis. It is given at the dosage of 800-1200 mg/m^2/day in two divided doses along with tapering doses of prednisolone for 12-24 months. Leukopenia, gastrointestinal discomfort and diarrhea are the few potential adverse reactions with MMF use. Monitor leukocyte counts every 1-2 months. Like cyclophosphamide, treatment is usually withheld if count falls below 4000/mm.[3]

In SDNS/FRNS children who have failed to attain remission with steroids and levamisole or cyclophosphamide or MMF should undergo a diagnostic renal biopsy before instituting calcineurin inhibitors.

Calcineurin inhibitors (CNIs): This prevents T cell activation through inhibition of calcineurin-induced IL-2 gene expression. And also one of the postulated mechanisms of CNIs in inducing remission is, they stabilize the podocyte actin cytoskeleton.[4] Cyclosporin (CsA) is given at a dose of 4-5 mg/kg daily for 12-24 months. Dosage of tacrolimus is 0.1-0.2 mg/kg daily for 12-24 months. Prednisolone is co-administered and tapered to a low maintenance dose of 0.25-0.5 mg/kg which is continued for six or more months. Indications include SRNS and difficult SDNS who did not show satisfactory response to levamisole, cyclophosphamide and MMF. Adverse effects of tacrolimus are hyperglycemia, hypertension, nephrotoxicity, hyperkalemia, hypomagnesemia and rarely neurotoxicity like headache and seizures. It is advisable to keep the 12 hour blood trough level of 80 to 120ng/mL for cyclosporine and 5 to 8ng/mL for tacrolimus to reduce the CNI toxicity. Tacrolimus is preferred over cyclosporine inview of lack of cosmetic side effects like hirsutism and gingival hyperplasia which is seen with cyclosporin.

Rituximab: It is a novel genetically engineered anti-CD20 monoclonal antibody that selectively targets CD20-positive B cells. Proposed mechanisms of this drug in NS are restoration of T regulatory (T_{REG}) cell populations and/or up regulation of their functions, thereby decreasing the proteinuria.[13] Generally B cells activate T helper cells through antigen presentation. Depletion of the B cells might alter T cell function or subpopulation expansion. It also inhibits the production of cytokines, suppresses the circulating permeability factors. Rituximab is found to be very effective in difficult SDNS and has variable results with SRNS unresponsive to CNI's. Dosage is 375 mg/m^2 once a week for two or more doses to achieve CD19 levels below 1% of leukocytes. Adverse reactions in the form of infusion reactions like flu-like symptoms, tachycardia, hypotension or hypertension may be encountered. It is also associated with increased risk of infections, restrictive lung disease and progressive multifocal leukoencephalopathy.

Need for renal biopsy in children with NS

Renal biopsy is routinely not needed in children in every NS. But if there are unusual features in their presentation and the response to standard prednisolone therapy is not adequate definitely they may need biopsy. Children with NS, before and during therapy with drugs having nephrotoxic potential like CNIs, will need protocol biopsy. Common indications for renal biopsy in general in children with NS are given in Table II.

Immunization in NS

Live attenuated vaccines should not be given in immune compromised children. Children receiving prednisolone at a dose of >2mg/kg/day or total of >20 mg/day in those weighing above 10kg for more than 14 days are considered immune compromised. Inactivated or killed vaccines can be given. Live vaccines are administered once the child is off immunosuppressive medications for at least 4 weeks.[2] These vaccines may be given to patients receiving alternate day prednisolone at dose less than 0.5 mg/kg in case of need like planning for rituximab. All children with nephrotic syndrome should receive immunization against pneumococcal infections, as spontaneous bacterial peritonitis is common in them.

Stress dose of steroids in NS

Children who have received high-dose steroids for more than 2 weeks in the past one year are at risk of suppression of the hypothalamo pituitary-adrenal axis during the period of stress. These children should be supplemented with steroids during surgery, anesthesia or serious infections. Corticosteroids can be used as parenteral hydrocortisone at a dose of 2-4 mg/kg/day, or oral prednisolone at a dose of 0.3-1 mg/kg/day. This is given for the duration of stress and this can be tapered rapidly.

Daily dose of steroids in FRNS during infection

There are reports wherein children on alternate day maintenance dose of steroids for FRNS were given the same dosage on a daily basis during the period of infection management. This may help in reducing the relapse rates. Gulati,et al in a randomized controlled trial in children with frequently relapsing nephrotic syndrome have shown that daily administration of maintenance doses of prednisolone during inter-current infections, significantly reduces the relapse rates.[14]

Table II. Indications for renal biopsy in children with NS

At onset
• Age less than 1 year or more than 8 years
• Gross hematuria, persistent microscopic hematuria or low serum complement C3
• Sustained hypertension
• Renal failure not attributable to hypovolemia
• Suspected secondary causes of NS
After initiating treatment
• Proteinuria persisting despite 4 weeks of daily corticosteroid therapy
• Before treatment with cyclosporin A or tacrolimus
• After 2 year of calcineurin inhibitors to check for toxicity

Complications of NS

They can be divided as complications due to the disease per se and related to the drugs which are used to treat NS.[3]

Drug-related toxicity: Steroids can cause gastritis, hyperglycemia, cataract and cushingoid features. Hypertension and induction to diabetes mellitus should not be forgotten. As earlier discussed, steroid sparing drugs are also not devoid of adverse effects, hence both favourable and untoward effects should be balanced. Diuretics in general can cause dyselectrolytemia and hypovolemia. Loop diuretics and thiazide diuretics can cause hyperglycemia and dyslipidemia. Deafness and hypercalciuria are adverse effect of furosemide whereas hypercalcemia can be seen following long-term thiazide administration. Intravenous furosemide should always be given slowly with the rate not exceeding 1mg/minute to avoid ototoxicity.

Due to the disease: Loss of protein in NS may cause infections, biochemical hypothyroidism, hypocalcemic tetany, anemia and hypercoagulable states. This hypercoagulable state in children with risk of dehydration may present as renal, cerebral and peripheral venous thrombosis. Hypovolemia per se can cause acute renal failure and infections can aggravate it. One should not forget that rapidly progressive glomerulonephritis occurring on a pre-existing glomerular disease like NS.

Points to Remember

- ***Most common cause of nephrotic syndrome in children is idiopathic nephrotic syndrome.***
- ***Though it is called idiopathic, various pathogenetic mechanisms for proteinuria include dysregulation of T cells, genetic mutations, circulating permeability factors and aberrant cross talk between B and T cells.***
- ***Overfill hypothesis of edema formation is supported by activation of tubular ENaC.***
- ***Nephrotic edema should be treated cautiously with appropriate diuretics either alone or in combination with serial monitoring of electrolytes and other adverse effects.***
- ***Nephrotic syndrome should be treated adequately with corticosteroids both in terms of dosage and duration.***
- ***In case of relapse, adequate treatment of infection may result in spontaneous remission.***
- ***Low dose steroid is always co-administered with steroid sparing drugs in the initial period of treatment of FRNS and SDNS.***
- ***All steroid sparing drugs have their own benefits and adverse effects. Hence serial monitoring to look for adverse effects should be stressed.***
- ***Rituximab, a novel genetically engineered anti CD-20 monoclonal antibody which selectively targets CD20-positive B cells is useful in difficult SDNS and SRNS.***
- ***Parents of nephrotic syndrome children should be counselled regarding the need for vaccination, especially pneumococcal vaccination when the child is in remission.***
- ***In children with risk of suppression of hypothalamo pituitary adrenal axis should get stress dose of steroids during the period of stress if they have received steroids in the past one year.***

- ***Complication due to disease and drugs per se should be addressed as early as possible in children with nephrotic syndrome to prevent the adverse consequences.***

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NEWS AND NOTES

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IAP-IJPP CME 2013

NOISY BREATHING

Subramanyam L

Abstract: *Although the clinical utility of the respiratory noises is often assumed, unfortunately, distinguishing these noises from each other can be very difficult. Many children will have multiple noises, as the obstruction to airway is often extensive (eg,inflammation involving both upper and lower airways), the noise may vary from minute to minute, and some noises may not clearly fit into any one of these simple descriptors. This difficulty in categorizing the noise is worse when the noise is intermittent described by the child's parent and not confirmed by the clinician. Another problem is intra-observer reliability i.e whether the clinician will agree with himself when observing the same sign on two separate occasions. Further agreement between clinicians on the terminology of these noises is far from perfect. The purpose of discussion is to analyse the validity and reliability of these noises and emphasize their subsequent clinical relevance and diagnostic significance.*

Keywords: *Noisy Breathing, Stridor, Wheeze, Snore, Rattle.*

Noisy breathing can be explained using principles of basic mechanics. An analogy that one can use to relate to breathing is the efficiency and smoothness of a machine. A person who hears a machine make a grinding noise or rattle when it runs will most likely think that the machine does not run very well and that it has a problem. A part may be loose, a screw may need to be tightened, the machine might need lubrication, or it might even be ready to fall apart. A new car or appliance that runs smoothly and quietly also runs more efficiently. Noise can be thought of as a form of friction. Friction prevents machines from running smoothly and efficiently and is responsible for their wear and tear.

Noise is a signal which indicates that something is wrong. Friction is the main explanation for noise. Friction causes excessive wear and mechanics become unbalanced, overworked, inefficient, or out of synchrony and harmony. It is similar to the fact that pain is the body's way of stating that it has a problem. It might be justifiable for breathing to be noisy (such as gasping or heavy breathing) during an emergency, but overall, it should be as quiet as reasonably possible, even while breathing deeply. The importance of eliminating noisy breathing cannot be overstated.

Noisy breathing is generally caused when a blockage somewhere in the breathing passages creates abnormal airflow. The blockage can be anywhere from the nose to deep inside the lungs. Noisy breathing may be harmless or a life-threatening condition.

Respiratory sounds or lung sounds referred to normal breath sounds (inspiration/ expiration) are identified through auscultation of the respiratory system with stethoscope. "Adventitious sounds" (wheeze/crackles) are referred to those sounds heard apart from the normal breath sounds. Respiratory noises are abnormal noises which are heard in a child with respiratory disorders. They are recognized by parents as well as pediatricians without the help of stethoscope.

Respiratory noises are extremely common in infants and young children. These noises can be considered as either symptom (when reported on history by parents) or a sign (when confirmed on physical examination). When a patient presents with a history of wheezing, it is crucial to ask the patient or the parents to describe what they actually are experiencing or hearing. On many occasions, the word "wheezing" is used as a general term to describe noisy breathing, including stridor, snoring, rattling and snuffle noises.

Various types of sounds originating from respiratory system may be heard without the help of stethoscope. The intensity and pitch of these sounds alter depending on their site of origin within the respiratory tract. The general dictum is that the pitch of the sound keeps on increasing and the intensity keeps on decreasing as one goes down lower into the respiratory tract. For example, snoring is a highly intense but low pitched sound because it results from the region of oropharynx. On the other hand wheeze is a high pitched, less intense sound originating from lower airway. Thus identifying these noises correctly is of major clinical relevance, in terms of localizing the site of obstruction as listed in Table I.

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Basic knowledge of airway dynamics in health and diseases is essential to understand the patho-physiology of respiratory noises in pediatric practice. Factors influencing airway dynamics are the relation between volumes/ capacities, pressure/flow, compliance/resistance and ventilation/perfusion mismatch.

Volumes / Capacities

Tidal volume (V_T) is the amount of air moved in and out of the lungs during each breath. At rest, it is usually 6-7 mL/kg body weight. Functional residual capacity (FRC) is the amount of air left in the lungs after tidal expiration. FRC is abnormally increased in intra thoracic airway obstruction, which results in incomplete exhalation, and it abnormally decreased in alveolar-interstitial diseases. Pulmonary compliance is decreased at very low or high FRC. Pathophysiologic consequence of decreased FRC is hypoxemia. Nature has created a grunt to increase FRC which in turn improves hypoxemia.

Pressure / Flow

During normal respirations, intra thoracic airways expand in inspiration as intra pleural pressure becomes more negative and narrow in expiration as they return to their baseline at FRC. In diseases characterized by airway obstruction, much greater changes in intra pleural pressure are required to generate adequate airflow, resulting in greater changes in airway lumen. In extra thoracic airway obstruction, the increased negative pressure during inspiration is transmitted up to the site of obstruction. This results in collapse of the extra thoracic airway below the site of obstruction, making the obstruction worse during inspiration. This produces inspiratory difficulty, prolongation of inspiration and inspiratory stridor. In intra-thoracic airway obstruction, during expiration, the increased positive airway pressure rapidly dissipates above the obstruction. Consequently, there is a collapse of the intra thoracic airway above the obstruction as it is subjected to markedly increased positive intra pleural pressure, making the obstruction worse during expiration. This results in increasing difficulty during expiration, prolongation of expiration and expiratory wheezing.

Compliance / Resistance

Airway resistance is determined by diameter of the airway, its length, viscosity of gases and nature of airflow (laminar / turbulent). Compliance means distensibility or expansibility. The normal lung is an elastic organ with the property to expand like a rubber band and distend like a balloon, while the airway is like a hollow pipe which has no distensibility but has resistance. Increase in airway resistance or decrease in elasticity will reduce the compliance. Once compliance is decreased that means tidal volume decreases. In this situation, to maintain constant minute ventilation, respiratory rate must be increased to maintain normal minute ventilation with or without respiratory noises.

Ventilation / perfusion mismatch

Any insult to the pulmonary system may interrupt normal bronchial hygiene mechanisms and create a potential for retained secretions. Retained secretions lead to an inflammatory response of the pulmonary mucosa. The hyperemia (capillary congestion) and edema (swelling) resulting from this inflammatory reaction cause increased resistance to airflow. The narrowing of bronchial lumen caused by partial plugging with retained secretions also causes increased resistance to airflow. Thus, uneven areas of airflow resistance cause uneven distribution of ventilation, which leads to ventilation-perfusion mismatch (shunt effect) and in turn hypoxemia. Greater degrees of retained secretions will cause total plugging of bronchioles and absorption atelectasis of the distal lung parenchyma. Such atelectasis will result in a decreased lung compliance and increased shunting. Thus either minor or major degrees of retained secretions may lead to increased work of breathing and hypoxemia. These processes are reversible by mobilizing the retained secretions. Rattle is a result of retained secretions in the airways which are presumably moving with normal respiration. Whereas the snore and snuffle noises arises from an increase in the resistance to airflow through the upper airways predominantly in the region of oro-nasopharynx.

Noisy breathing is a common sign of airway disorder. When parents complain that a child has noisy breathing, the healthcare professional will want to know: is it constant or occurs only sometimes, is it inspiratory stridor, expiratory wheeze or biphasic noise like snore, snuffle, rattle (Table.I) whether intensity of noise is altered by change of position or feeding, whether respiratory noise goes away or aggravated during sleep.

Table I. Respiratory noises - with site of origin and phase of respiration

Noise	Site of origin	Phase of respiration
Stridor	Extra thoracic AW	Inspiration
Wheeze	Intra thoracic AW	Expiration
Grunt	Glottis	Expiration
Snore	Oro-pharyngeal AW	Insp. / Exp.
Rattle	Either intra or extrathoracic AW	Insp./exp.
Snuffle	Nasal passages	Insp. / Exp.

Abnormal sounds range from a high-pitched crowing during inhalation suggestive of extra thoracic lesion and continuous musical sounds during exhalation (wheezing), suggestive of intra thoracic lesion such as asthma. Repetitive loud snoring during sleep, interrupted by periods of silence in which there's no air flow, is a major sign of sleep apnea.

With this basic knowledge of pulmonary dynamics the next pragmatic approach is to define and describe assessment of each of these noises. For evaluation of respiratory noises patient's age and the course of onset (acute / persistent) and associated symptoms of infection such as fever, cough and running nose will give clue to clinical diagnosis.

There are vast underlying conditions both congenital and acquired, that can produce these noises. For practical purposes the most common acute and persistent causes of these noises are listed in Table II.

Table II. Common clinical causes of noisy breathing

Noise	Acute	Persistent
Stridor	Acute laryngotracheo bronchitis (viral"croup")	Laryngomalacia
Wheeze		
Infants	Acute broncholitis	Tracheobroncho malacia
Children	Viral-induced wheeze	Persistent asthma
Grunt	HMD / Pneumonia	COPD Pursed lips breathing
Snore	Acute enlarged T&A	OSAH
Rattle	Acute viral bronchitis	Retained secretions (neuromuscular disorder)
Snuffle	Coryza	Allergic rhinitis

Stridor

Stridor is predominantly inspiratory and indicates obstruction to airflow in the extra thoracic airways down to the level of the thoracic inlet. Because stridor is a sign of upper airway obstruction, it should never be ignored. At this point in the respiratory tract there is only a single air passage and severe obstruction may culminate in respiratory arrest.

If stridor is acute and associated with symptoms and signs of a viral respiratory tract infection plus a brassy (croupy) cough, then viral croup (larynotracheobronchitis) is most likely. Although acute epiglottis (caused by Haemophilius influenzae type B) is now rare because of improved vaccination coverage, the noise produced by this condition is typically a soft vibrating sound, rather than a harsh inspiratory stridor.

The upper airway is a complex anatomic region, which is best divided into supraglottic, glottic and subglottic areas. The supraglottic area includes the epiglottis, aryepiglottic folds and false vocal cords. Because this area is composed of soft tissue and muscle, it is prone to inward collapse, resulting in obstruction. This typically occurs in infants with laryngomalacia, the most common cause of stridor in infants and is a result of collapse of the supraglottic structures during inspiration. The glottic area includes the true vocal cords. Typically pathology at this level causes a soft, hoarse cry (voice) as well as inspiratory stridor. Examples include vocal card paralysis and laryngeal web. The subglottic area includes the extra thoracic trachea down to the level of the thoracic inlet. Persistent stridor in a prolonged ventilated child should make one to suspect subglottic stenosis.

For most infants, laryngomalacia is not a serious condition, as long as they are able to eat and grow. For these infants, laryngomalacia will resolve without surgery by the time they are 18 to 20 months old. However, a small percentage of babies with laryngomalacia do struggle with breathing, eating and gaining weight requirng prompt attention.

Wheeze

In evaluating a young child with parent-reported wheeze, the first step is to determine exactly what the parents mean by that term. In particular, whether wheeze is being used in generic sense to describe any noise or particularly a wheeze.

Wheezing refers to high pitched whistling sounds audible without auscultation by the stethoscope. Wheezing causes considerable anxiety to the parents. Partial obstruction of the bronchi and bronchioles leading to narrowing produces wheezing. Sufficient air must flow through the narrowed airway to produce the wheezing sound. This may be due to causes within the lumen or in the walls of the bronchi. Pressure from outside the bronchi may also be responsible in some cases.

In contrast to stridor, wheeze is predominantly expiratory and indicates obstruction to airflow in the intra thoracic airways. Wheeze can originate from airways of any size, from the large extra pulmonary airway or small

intrapulmonary airways. It can be high-pitched or low-pitched, consist of single or multiple notes and occur during expiration. Two important aspects of the medical history include the patient's age, the course of onset (acute / persistent) and associated symptoms, which will give a clue to the cause of wheeze.

In infants (younger than 12 months), a polyphonic wheeze on auscultation, particularly if accompanied by generalized inspiratory crackles, indicates probable acute viral bronchiolitis. Persistent wheezing presenting very early in life suggests a congenital or structural abnormality such as tracheobroncomalacia or vascular rings.

If a child presents with symptoms of airflow obstruction (recurrent cough, wheeze, breathlessness) with family history of atopy who on examination, have a combination of generalized, polyphonic wheeze, asthma is likely. Additional clinical information should be sought to rule this in or out, such as over inflation of the chest, prolonged expiration, or a trial of bronchodilator therapy.

In a young child, with either localized wheeze or monophonic wheeze, particularly if accompanied by asymmetric breath sounds (i.e,uneven air entry), a focal lesion must be ruled out. This could include an endobronchial obstruction (eg. foreign body), whereas the slowly progressive onset of wheezing in older child may be a sign of extraluminal bronchial compression by a growing mass or lymph node.

In a questionnaire based study to conclude whether early respiratory noises predict subsequent wheeze, it was found that children with a "whistle" at age 2 were more likely to wheeze and more likely to receive asthma treatment at age 5 years, compared with children with a "rattle" at 2 years.

Commonly used term "transient early wheeze" (TEW) describes otherwise healthy infants who wheeze in infancy but cease wheezing by age 3 years. Most studies of TEW indicate that these infants have abnormal lung function from birth (ie, smaller than normal calibre airways).

Available literature worldwide suggests three phenotypes of wheeze in early childhood, namely, transient early wheeze (TEW), persistent wheeze (PW) and late-onset wheeze (LOW). As discussed above, infants with TEW have abnormal lung function before the onset of their wheeze. Those with PW (ie, wheeze both in the first 3 years and beyond) appear to have definite atopic asthma. Those with LOW have no wheeze in the first 3 years, but after the age of 3 years. Children with LOW have been termed non-atopic wheezers (or viral-induced wheeze) and appear to have a very mild form of asthma.

Grunting

The presence of grunting indicates that the child is critically ill and is in impending respiratory failure. This sound is produced in expiration against a partially closed glottis. It is an attempt to maintain positive airway pressure during expiration for as long as possible. Such prolongation of positive pressure is most beneficial in small airway obstruction (such as bronchiolitis) and in alveolar diseases that produce widespread loss of functional residual capacity such as pneumonia and respiratory distress in newborn.

The expiratory grunt is a natural form of continuous positive airway pressure (CPAP) aimed at improving oxygenation and is mechanistically similar to "pursed lips breathing" in adults with COPD.

Snore

Although snoring is generally more obvious in inspiration, the noise is frequently audible throughout the respiratory cycle. The noise arises from an increase in the resistance to airflow through the upper airways, predominantly in the region of the nasopharynx and oropharynx. In young children the usual cause of snoring is enlarged tonsils and/or adenoids. Distinguishing primary snoring from pathologic obstructive sleep apnea-hypopnea (OSAH) is difficult clinically. Symptoms that suggest genuine OSAH include observed apnea, excessive daytime sleepiness, and behaviour or learning problems. If there are associated symptoms suggesting OSAH, a formal sleep study may be indicated. Over weight and obese children are at greater risk.

Rattle

A rattle is a result of excessive secretions in the large airways, which are presumably moving with normal respiration. Rattles may be heard in either, inspiration or expiration or both. Parents will often comment that as well as being able to hear the rattle, they can feel the rattle (as a vibration), when placing their hands over the child's chest wall.

Rattling is due to excessive secretions in the pharynx or tracheobronchial tree during breathing. It is present in asthma, bronchitis and tracheobronchial stenosis. Aspiration of gastrointestinal content into the tracheobronchial tree can also result in rattling. Some normal infants may have transient rattling but prolonged rattling is always pathological. If the child has an underlying chronic neurologic or neuromuscular condition, the most likely explanation is inability to cough and/or swallow normal secretions and appropriate investigation to rule out pulmonary aspirations and specific interventions are to be carried out.

Snuffles

Stuffy nose is common in newborn and infants. A newborn baby breathes 40-60 times a minute, in comparison to 12-20 times a minute for an adult. Newborns also have something called periodic breathing. This is when they breathe fast and shallow (almost like they are panting), followed by several second long pauses in their breathing. This is normal. One has to be concerned if baby is also limp, pale, or blue (which would be very unusual).

The terms "snuffles" are used to describe respiratory noises emanating from the nasal passages. These nasal noises are frequently audible in both inspiration and expiration, and often associated with visible secretions from the nares. Snuffle has also been used to describe any discharge from the nasal passages and is sometimes used to describe a minor viral upper respiratory tract infection.

In assessing young infants with persistent nasal obstruction, nasal patency needs to be assessed to rule out choanal atresia. Bilateral nasal obstruction in older children suggests either perennial allergic rhinitis or rarely nasal polyps (usually in association with cystic fibrosis). If there is persistent purulent nasal secretions chronic bacterial rhinosinusitis has to be considered.

Newborns also have tiny noses, breathe only through their noses and don't know how to clear nasal secretions. Many seem like they are always congested, even when they're not sick. If baby is sleeping and feeding comfortably, nothing needs to be done. If baby is uncomfortable, then a few saline drops may be put in the nose and then sucked with a bulb syringe if necessary. Newborns also sneeze, hiccup, sniffle and snort (like little piglets)! They also cough occasionally to help clear their secretions. One need not bother, as long as the baby is not bothered. If baby is struggling to breathe and is unable to feed or sleep because of difficulty in breathing even after suctioning the nose, it requires assessment.

Evaluation of respiratory noises

Respiratory noises in children require careful evaluation and close monitoring. When a child exhibits signs and symptoms of respiratory distress, careful interpretation and correlation with the disease is very valuable in the diagnosis and initiation of treatment. Normal breathing is effortless, while breathlessness, dyspnea and respiratory distress mean increased work of breathing, characterized by inter costal retraction, supra sternal hollowing and flaring of alae nasi. It is important to realize that increased respiratory rate can occur even in non respiratory conditions like metabolic acidosis, diabetic keto-acidosis and cardiac diseases. To differentiate respiratory from non respiratory conditions increased work of breathing like chest retraction is helpful which is marked in respiratory disease, minimal in cardiac diseases and absent in other conditions. Further evaluation of respiratory system depends on the presence of noises like stridor, wheeze and grunt which will help to differentiate different respiratory disorders.

In assessing infants with persistent stridor, but no significant increase in the work of breathing, no cough, normal cry, no apnea or cyanotic episodes and thriving, laryngomalacia is the most likely cause. No further investigation is warranted apart from follow-up review. In infants with recurrent or persistent polyphonic wheezing, not associated with significant cough, inspiratory crackles, or significant increase in the work of breathing, the most likely diagnosis is TEW. Unfortunately parents/physician commonly mislabel the rattle as a "wheeze", resulting in over diagnosis of asthma.

In most of the cases, the diagnosis is obvious to the healthcare professional from the history and physical exam. Occasionally, further tests are needed, depending on the suspected cause. A chest x-ray is commonly done to look for parenchymal infections and CT scan to look for mediastinal lesions. Flexible fiberoptic bronchoscopy is useful to detect structural anomalies such as laryngomalacia, vocal cord paralysis, subglottic stenosis and intraluminal airway obstruction (foreign body).

Acoustic analysis

Adult studies have highlighted problems with both accuracy (validity) and reliability of respiratory signs using a stethoscope. Given the difficulty of examining young, uncooperative children, errors could be substantially greater. In an attempt to improve the utility of respiratory noises, computerized acoustic analysis has been evaluated. Wheeze was characterized by a sinusoidal waveform with several distinct peaks and rattle by an irregular non sinusoidal waveform, with diffuse peaks in the power spectrum display.

In a study using vigorous methodology for evaluating a diagnostic test, the results for both validity and reliability of both the stethoscope and acoustic analysis were poor. Although future development of better acoustic analysis techniques could result in improved validity and reliability, at present it appears acoustic analysis has little to offer over the stethoscope in real life clinical situations.

An interesting attempt was made to assess the utility of video clips of children demonstrating noisy breathing. Although parents' reports of symptoms were in poor agreement with clinicians, the ability of parents to identify the origin (or location) of the noise was in agreement with the clinicians.

Finally it is obvious that quality evidence is needed to improve the utility of these reported and observed noises by the use of technology such as audio/video recording or computerized acoustic analysis. The conclusion drawn from the research evidence is that the use of computerised acoustic analysis to improve the utility of noises is disappointing but audio/video of typical noises can improve the accuracy of parents' descriptions of these noises.

How useful are they clinically?

Respiratory noise with associated signs will give clue to clinical diagnosis. In addition to tachypnoea and chest indrawing the presence of stridor (upper airway obstruction eg.croup), wheeze (small airway obstruction eg.asthma) and grunt (parenchymal lesion eg. pneumonia) will help to pin point the site of disease. At times snoring which originates from the flutter of the tissues of the oropharynx should be differentiated from other noisy breathing. By careful interpretation of these clinical signs, anatomical diagnosis is possible in a vast majority of infants presenting with respiratory distress. An error in recognizing these noises misleads diagnosis, unnecessary investigations and inappropriate treatment.

Points to Remember

- ***When the parents report about respiratory noises, it is the duty of pediatrician to confirm.***
- ***Many infants with parent reported wheeze, have a "rattle" rather than wheeze.***
- ***A major error is misclassifying stridor as a wheeze and missing significant upper airway obstruction.***
- ***Audio or video recording of respiratory noises is helpful to discuss with parents in day-to-day practice.***
- ***Time spent in eliciting the history is worth than ordering investigations without clinical clues.***

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CLIPPINGS

Comparing continuous electronic fetal monitoring in labour (cardiotocography, CTG) with intermittent listening (intermittent auscultation, IA)

Cardiotocography (known also as electronic fetal monitoring), records changes in the fetal heart rate and their temporal relationship to uterine contractions. The aim is to identify babies who may be short of oxygen (hypoxic), so additional assessments of fetal well-being may be used, or the baby delivered by caesarean section or instrumental vaginal birth.

Objectives was to evaluate the effectiveness of continuous cardiotocography during labour.

Selection criteria: Randomised and quasi-randomised controlled trials involving a comparison of continuous cardiotocography (with and without fetal blood sampling) with (a) no fetal monitoring, (b) intermittent auscultation (c) intermittent cardiotocography.

Authors' conclusions: Continuous cardiotocography during labour is associated with a reduction in neonatal seizures, but no significant differences in cerebral palsy, infant mortality or other standard measures of neonatal well-being. However, continuous cardiotocography was associated with an increase in caesarean sections and instrumental vaginal births. The challenge is how best to convey these results to women to enable them to make an informed choice without compromising the normality of labour.

Alfirevic Z, Devane D, Gyte GML. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. Cochrane Database of Systematic Reviews 2013, Issue 5. Art. No.: CD006066. DOI: 10.1002/14651858.CD006066.pub2. Assessed as up to date: January 31, 2013.

IAP-IJPP CME 2013

TROPICAL INFECTIONS IN THE PICU

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Abstract: *Tropical infections often cause life threatening illness requiring PICU admission. Diagnosis may be difficult in critically ill children. Severe dengue presents with third-spacing, shock, hemorrhage and organ impairment requiring fluid resuscitation and sometimes, blood transfusion. Malaria often causes complications (cerebral malaria, hypoglycemia, anemia, hyperparasitemia) having a high mortality if not treated promptly. Scrub typhus may cause shock, ARDS and renal failure and can mimic dengue. Outcome is good with specific therapy. Icteric leptospirosis causes jaundice, renal failure and hemorrhage, and residual renal and visual impairment may result. Multi-organ dysfunction is common in these infections needing ventilation, hemodynamic support and dialysis.*

Keywords: *Tropical infections, Dengue, Malaria, PICU*

Tropical infections are a common cause of morbidity and mortality in developing countries. These infections often cause life threatening complications that require care in the PICU. Notable among these, in the Indian scenario, are dengue, malaria, scrub typhus, leptospirosis and enteric fever.

Dengue

Dengue is a viral illness that has a wide clinical spectrum ranging from a mild nonspecific viral syndrome to a severe, occasionally fatal disease characterized by hemorrhage, shock and multi-organ dysfunction (Table I).

Clinical features

The clinical course of dengue is characterized by three well demarcated phases:

1. Febrile phase (2-7 days)
2. Critical phase (24-48 hours)
3. Recovery phase (48-72 hours)

Severe dengue

1. Shock: This is due to hypovolemia and may have distinctive features

- Initially compensated, with a normal systolic blood pressure, an elevated diastolic and narrow pulse pressure
- Cool mottled extremities, with delayed capillary refill
- Tachycardia may be absent
- In late stages: hypotension and unrecordable pulses

2. Bleeding: Common sites are gastrointestinal bleeds (hemetemesis and melena). Major life threatening bleeding is rare even in the presence of severe thrombocytopenia and coagulopathy.

Risk factors for bleeding are: a) prolonged shock, b) hypoxia and acidosis, c) hepatic or renal dysfunction, d) drugs (NSAIDs) and e) procedures (nasogastric tube insertion, arterial lines, intramuscular injections)

3. Organ impairment

a) Liver failure may be due to direct viral hepatitis or due to ischemic hepatic necrosis. This is associated with a high mortality.

b) CNS manifestations may be due to dengue encephalopathy or secondary mechanisms

c) Cardiac dysfunction: Both systolic and diastolic myocardial dysfunction are known

d) Renal failure

e) Other manifestations include hemolytic uremic syndrome, co-infections (malaria, leptospirosis and enteric fever), hemophagocytic lymphohistiocytosis.

Diagnosis

The diagnosis of dengue in children can be difficult as the initial manifestations may be non-specific.

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Table.I. WHO classification of Dengue

Probable dengue	Dengue with warning signs	Severe dengue
Lives in/ Travel to endemic area	Intense abdominal pain or tenderness	Severe plasma leakage with rising hematocrit leading to: 1.Shock 2.Fluid accumulation (pleural, ascitic) 3.Respiratory distress
Fever + 2 or more of the following:-	Persistent vomiting	
- Nausea and vomiting - Rash - Aches and pains - Positive tourniquet test - Leukopenia - Any warning sign	Clinical fluid accumulation Mucosal bleeding Lethargy, restlessness Liver enlargement > 2 cm Increase in hematocrit with concurrent rapid decrease in platelet count	Severe bleeding Severe organ impairment Liver: Elevated transaminases (AST or ALT $\geq$ 1000) CNS: Impaired consciousness, seizures
Laboratory confirmed dengue		

Differential diagnoses are bacterial septic shock, malaria, leptospirosis, enteric fever and meningococcal septic shock.

Laboratory confirmation

1. Serology is the most reliable diagnostic method. IgM and IgG by ELISA on acute (1-5 days) and convalescent (15-21 days) sera. Alternatively, a single test may be done on serum after 5 days of fever.

Management

Principles of management

Identify and determine whether the child has dengue, the clinical phase and severity

Indications for PICU admission

1. Shock
2. Respiratory distress
3. Abnormal bleeding
4. Organ impairment

Priorities of management

1. Replacement of fluid losses
2. Early recognition and treatment of hemorrhage
3. Prevention and management of fluid overload
4. Management of organ impairment

Initial management

1. Stabilize airway and breathing
2. Establish IV access
3. Obtain baseline hematocrit, platelet count, electrolytes, renal and liver function, blood grouping and cross matching.
4. Insert a urinary catheter
5. If an alternative infection is suspected, start appropriate antimicrobial agents after taking suitable samples for diagnosis (blood cultures, smear for malarial parasite)
6. Determine the degree of shock.

Fluid resuscitation in compensated shock

- Start a fluid bolus with isotonic crystalloid solution (normal saline or Ringer's lactate) at 10-20 mL/kg/hr over one hour and reassess the condition.
- If the condition improves, reduce the fluids to 10 mL kg/hr for 1-2 hours; then 7 mL/kg/hr for 2 hours; 5ml/kg/hr for 4 hours and then 3 mL/kg/hour which can be maintained for 24-48 hours.
- Target an urine output of in 1ml/kg/hr in infants and 0.5ml/kg/hr in older children. Reduce fluids earlier if the urine output is higher (1.5-2 ml/kg/hr) and if oral intake improves.

- If the condition does not improve after a bolus, repeat hematocrit
  - If the hematocrit is still high or increases, administer a colloid bolus at 10-20mL/kg over 1 hour; then reduce to 10mL/kg/hr for 1 hour and later to 7mL/kg/hr. Change to crystalloids when condition improves.
  - If the hematocrit decreases compared to the baseline and child is still hemodynamically unstable, consider bleeding, in which case, a whole blood or packed red cell transfusion needs to be given.
  - If there is no bleeding, give a colloid bolus at 1-2ml/kg/over 1 hour and reassess clinical status and hematocrit thereafter. Consider a blood transfusion if there is no clinical improvement.

Fluid resuscitation in hypotensive shock

- Crystalloid or colloid bolus of 20mL/kg over 15-30 minutes and reassess
- If the child improves, a colloid infusion of 10mL/kg/hour for 2 hours and then reduce the infusion rate as mentioned before. Fluids may be tailored to urine output and oral intake.
- If the child does not improve, repeat hematocrit
  - If the hematocrit is low (<30-35% in infants, <35-40% in children), consider bleeding. If there are signs of severe bleeding, a fresh whole blood or packed red cell transfusion should be given.
  - If there is no bleeding, administer second colloid bolus of 10-20 mL/kg. Repeat clinical assessment and hematocrit should be done and consider blood transfusion.
  - If hematocrit is still high after the first bolus, give second bolus with colloids at 10-20mL/kg over 30-60 minutes and reassess. If there is improvement the infusion rate may be reduced as described previously. If not, repeat hematocrit and decide whether to give further fluid or blood transfusion.
- Do not use hypotonic and glucose containing fluids

Monitoring

1. Monitor vital signs, level of consciousness, respiratory distress, abdominal girth and urine output frequently.
2. Check hematocrit initially 2-4 hourly and less frequently as the child improves and hematocrit falls.
3. Platelets, coagulation, electrolytes and blood glucose.

Duration of intravenous fluid therapy

Continue fluid therapy till the capillary leak phase lasts, typically 24-48 hours.

Management of hemorrhage

Consider hemorrhage in the following situations:

1. A hematocrit that is falling or lower than expected, in a child who is hemodynamically unstable
2. No improvement in the hemodynamic status despite fluid boluses of 40-60 mL/kg with crystalloid or colloid

The most important intervention here is transfusion with fresh whole blood or packed red cells.

Transfusion with platelets, FFP or cryoprecipitate has not been found to be beneficial and may add to fluid overload. Prophylactic use of platelets does not prevent bleeding. These blood products may be considered if the patient continues to bleed despite 2-3 aliquots of blood transfusion.

Anti-D immunoglobulin, IV immunoglobulin and activated factor VII are currently not recommended as they are not proven to be of clinical benefit.

Management of fluid overload

Fluid overload may lead to massive pleural effusion and ascites. The best treatment is prevention, by avoiding prophylactic blood products, restricting fluid intake based on perfusion and urine output. In established fluid overload, diuretics (frusemide infusion) may be used once the capillary leak phase is over and the child is hemodynamically stable. Peritoneal dialysis is rarely required, in the absence of renal failure.

Respiratory support

This may be required in patients with hypoxia, increasing work of breathing or altered sensorium. NIV may be attempted in selected children. Only experienced personnel should perform intubation and ventilation.

Inotropes and vasopressors

Inotropes are used only if there is myocardial dysfunction. Vasopressors should be avoided since they can worsen end organ perfusion.

Acute liver failure

Hepatic failure should be monitored and treated as per existing guidelines (high dextrose infusion rates, N- acetyl cysteine, vitamin K, lactulose and gut sterilizing agents, mechanical ventilation).

Metabolic derangements

Hyperglycemia, hypoglycemia, hyponatremia, hyperkalemia and metabolic acidosis may occur.

Mortality

Although the mortality in dengue is low (<1-5%), in severe dengue shock it may be as high as 26% especially when the shock is profound and treatment delayed.

MALARIA

Malaria is endemic in several states in India. Although most cases of severe malaria are due to Plasmodium falciparum, P. vivax is also reported to occasionally present with a complicated course.

Clinical features

The main symptoms are fever, headache, nausea, vomiting, myalgia and arthralgia. Physical examination may show pallor and hepatosplenomegaly.

Diagnosis

Microscopy by thick and thin smear is the gold standard. Rapid diagnostic test kits detect the presence of parasitic antigens. They have more than 90% sensitivity and specificity.

Complications

P. falciparum is the commonest cause of severe malaria and can be fatal if not treated promptly. There may be altered consciousness, prostration, seizures, or shock.

Differential diagnosis

The other conditions to consider include meningitis, pneumonia, septic shock, dengue, leptospirosis and scrub typhus.

Management of severe malaria

Severe malaria is a medical emergency and the child should ideally be admitted to a PICU.

The airway, breathing, hemodynamic status, level of consciousness and sugar level should be checked rapidly. If the diagnosis is in doubt, appropriate antibiotics as per guidelines for sepsis / meningitis should be given after obtaining blood cultures.

Initial investigations

Complete blood count, electrolytes, renal and liver function tests and coagulation should be checked along with blood grouping. If indicated, obtain chest X-ray and urine examination (including urine hemoglobin)

Specific antimalarial therapy

Parenteral therapy should be used for the first 24 hours and monotherapy avoided. Either artemisinin or quinine based combination therapy should be used regardless of chloroquine sensitivity. In children, artemisinin based therapy is preferred

1. Artesunate: 2.4 mg/kg body weight IV or IM at admission and at 12 hr and 24 hr followed by once daily. Total duration of therapy - 7 days.

Along with artesunate one of the following should be given:

- Clindamycin 10 mg/kg 12 hourly for 7 days
- Doxycycline 3mg/kg daily for 7 days (> 8 years of age)
- Amodiaquine: target dose 10mg/kg (range 7.5-15 mg kg) for 3 days
- Sulfadoxine/pyrimethamine: dose range is 25-70/1.25-3.5mg/kg of Sulfadoxine/pyrimethamine as a single dose.
- Mefloquine is contraindicated in cerebral malaria

2. Quinine: 20 mg/kg in (5% dextrose) IV over 4 hours, at admission, followed by 10 mg/kg every 8 hourly

If the child has received quinine already, do not give the loading dose. If parenteral quinine needs to be given for more than 48 hours, the dose should be reduced to 7mg/kg. Quinine can cause arrhythmias and hypoglycemia. Continuous cardiac monitoring and frequent glucose monitoring are essential. Total duration of therapy should be 7 days. Add clindamycin or doxycycline to the quinine regime, for 7 days.

3. Artemether: 3.2 mg/kg IM at admission followed by 1.6mg/kg daily. This should be used only if none of the aforementioned alternatives are available.

Artemether/lumefantrine is available as a fixed-dose formulation. Lumefantrine has a dose range of 10-16mg/kg twice a day for 3 days.

Primaquine: 0.75mg/kg as a single dose on day 2 (for gametocidal action). It is contraindicated in infants < 4 months.

Supportive therapy

Monitoring

Monitor vital signs, level of consciousness and urine output closely. Check blood glucose every 4 hours.

Airway protection: Intubate if level of consciousness is low and child is unable to protect the airway

Fluid therapy: Fluids should be tailored on an individual basis depending on the clinical status. Both under and over hydration are detrimental. Examination of the perfusion, urine output, skin turgor and vital signs, assisted by echocardiography if available can help guide fluid therapy.

Hyperpyrexia: Control fever by tepid sponging, fanning, cooling blankets or antipyretics (Paracetamol is preferred over NSAIDs)

Seizures: Use benzodiazepines to terminate seizures. Phenobarbitone should be used only when respiratory support is available. There is no role for prophylactic anticonvulsants.

Hypoglycemia: Blood glucose should be checked periodically and treat hypoglycemia.

Severe anemia: The threshold for transfusion is generally a Hb < 5 g/dL, but the decision should be individualized to the patient.

Pulmonary edema: Fluid restriction and diuretics may be tried in hemodynamically stable children, but positive pressure (CPAP or ventilation) may be required.

Acute kidney injury: Pre-renal causes may be treated with additional fluids. Intrinsic renal failure may need peritoneal or hemodialysis.

Disseminated intravascular coagulation and abnormal bleeding: FFP, cryoprecipitate and platelet transfusion may be necessary. If bleeding is severe, PRBC may be needed.

Metabolic acidosis: The underlying cause needs to be treated (hypoglycemia, hypovolemia, sepsis, shock). Bicarbonate infusion is rarely indicated unless there is hemodynamic instability.

Shock: Fluid resuscitation, inotropes and vasopressors may be needed. Bacterial sepsis should be considered and broad spectrum antimicrobials started after taking blood cultures.

Nutrition: Patients may be allowed to feed orally once child regains consciousness. Nasogastric feeds may be started in intubated patients who are hemodynamically stable.

Exchange blood transfusion: Exchange transfusion has been recommended in the past for severe parasitemia, but data to support its utility are limited.

SCRUB TYPHUS

This is a rickettsial illness caused by Orientia tsutsugamushi that is transmitted through the bite of the larval forms of the trombiculid mite. In India it is seen in the drier parts of several states especially the central and southern regions.

Clinical features

Manifestations can range from a mild febrile illness to multisystem involvement with shock and organ dysfunction. Children usually present with moderate to high grade fever that may last upto 10 days. There may be no localizing signs. Regional or generalized lymphadenopathy, hepatomegaly or splenomegaly may be seen frequently. About 30% children may have a maculopapular rash. Abdominal pain, vomiting and diarrhea may also occur at presentation.

The diagnostic feature is a single painless eschar with an erythematous rim seen at the site of the larval bite. It is often present in the groin, around the genitalia or behind the ear. The reported incidence of an eschar is highly variable (7-68%) but its presence almost clinches the diagnosis.

Leukocytosis and thrombocytopenia are common. In severe illness, there may be coagulopathy, elevated liver enzymes and azotemia.

Complications

Pneumonia with or without ARDS, meningoencephalitis, acute renal failure, myocarditis and a septic shock-like syndrome, may be seen.

Diagnosis

Serology: IgM antibodies by Indirect Immunofluorescence Assay has a sensitivity of >90% after 11 days of fever.

Differential diagnoses

Enteric fever, dengue, other rickettsioses, leptospirosis, malaria and infectious mononucleosis. In an extremely sick child, pneumonia, meningitis and bacterial septic shock should be considered. But the features that may help in diagnosis are: (a) Presence of an eschar, (b) Hepatosplenomegaly and lymphadenopathy, (c) Absence of hemoconcentration and (d) Abnormal coagulation profile but unlike in dengue, the PT here is more deranged compared to a PTT.

Initial management

This is no different from other patients with shock – the airway, breathing and circulatory systems are stabilized. Shock is treated with fluid boluses initially, followed by inotropic support if required. Anticonvulsants may be needed for seizures.

Definitive therapy

Doxycycline (4mg/kg/day oral or IV in 2 divided doses) is the recommended regimen. But alternatives are: (a) Tetracycline (25-50 mg/kg/day PO divided 6 hourly) or (b)Chloramphenicol (50-100 mg/kg/day divided 6 hourly) or (c) Azithromycin (10mg/kg/day oral or IV). Therapy should be continued for at least 5 days and till the patient is afebrile for $\geq$ 3 days.

LEPTOSPIROSIS

This is a spirochetal zoonotic infection having a variable presentation ranging from a mild flu-like illness to fulminant multiorgan involvement causing hemorrhage, renal failure and death. The severe form (Weil's disease) is less common in children.

Clinical features

The disease usually has a biphasic course with septicemic and immune phases separated by an asymptomatic period. It may have one of the following presentations:

1. Anicteric leptospirosis: Common symptoms during the septicemic phase are fever, chills, headache, conjunctival suffusion, myalgia, nausea and vomiting. Mild jaundice and mental status changes may be present.

The immune phase is characterized by aseptic meningitis and anterior uveitis.

2. Icteric form (Weil's disease): The septicemic phase is similar to the anicteric type. The immune phase is characterized by jaundice, renal failure and hemorrhage. There may be dyspnea and cough progressing to ARDS and pulmonary hemorrhage. Hemorrhagic manifestations include epistaxis, purpura, petechiae, mild hemoptysis and subconjuctival hemorrhage which is pathognomonic.

Serum bilirubin is markedly elevated with mild elevation of hepatic transaminases. Liver failure is uncommon. Lymphocytosis and thrombocytopenia are common. Muscle CPK will be elevated. Chest X ray shows patchy infiltrates.

Differential diagnoses

1. Severe dengue and other viral hemorrhagic fever, Hantavirus.
2. Malaria, enteric fever, other rickettsial infections.
3. Toxic shock syndrome.

Diagnosis

1. Microscopic agglutination test (MAT) on patient serum. A four-fold rise in titers in paired sera or a titer more than1:100 is diagnostic.
2. Real time PCR is a rapid and reliable test but has limited availability.

Management

PICU admission may be required in the following situations:

1. Hemorrhage
2. Acute renal failure
3. Respiratory distress and ARDS

Initial management should include stabilization of airway and breathing, evaluation of hemodynamic status and management of shock. If bacterial sepsis is considered, appropriate antibiotics should be started after taking blood cultures.

Initial investigations

1. Blood counts
2. Electrolytes, renal and liver function tests
3. Creatine kinase (specifically CPK-MM)
4. Coagulation
5. Blood grouping and cross matching
6. Chest X ray

Antibiotic therapy

All severe cases should be treated with antibiotics. Penicillin, doxycycline, cefotaxime and ceftriaxone may be used. In areas where co-infection with rickettsiosis is common doxycycline is preferred.

Supportive care

Appropriate supportive care will be required for shock, ARDS, renal failure and hemorrhage.

Kala-azar (Leishmaniasis)

This is common in the eastern and north eastern parts of India. Although, it is usually a chronic disease, acute deterioration requiring PICU care may be seen in visceral leishmaniasis in the following situations:

1. Severe anemia causing congestive cardiac failure
2. Severe malnutrition with secondary bacterial sepsis and septic shock

Definitive management involves chemotherapy with antimony compounds (sodium stibogluconate) or Amphotericin B. Supportive care in the form of blood transfusion, antibiotics for bacterial sepsis and hemodynamic support for septic shock may be necessary.

Points to Remember

- ***The specific diagnosis of tropical infections can be challenging especially in the PICU***
- ***Severe dengue is a potentially fatal illness but can be treated effectively with early fluid resuscitation. Early recognition of warning signs, shock, hemorrhage and fluid overload are important.***
- ***Severe malaria may be difficult to differentiate from dengue, scrub typhus, bacterial sepsis, pneumonia and meningitis. Repeated microscopy and rapid antigen testing may aid diagnosis. Prompt antimalarial therapy is essential to prevent mortality.***
- ***Scrub typhus may clinically resemble severe dengue. Absence of hemoconcentration, prolonged prothrombin time (compared to partial thromboplastin time) and the presence of an eschar demarcating the chigger bite are distinguishing features.***
- ***The icteric form of leptospirosis (Weil's disease) can cause jaundice, renal failure and pulmonary hemorrhage. Hemodialysis and mechanical ventilation are often necessary.***

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NEWS AND NOTES

International Scientific Conference on Probiotics and Prebiotics – IPC 2014, Hungary

Date: 24 – 26th June, 2014

Contact:

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Organising Committee of IPC 2014

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ABDOMINAL PAIN – MEDICAL OR SURGICAL?

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Abstract: *The first step in evaluation of abdominal pain in children is to differentiate surgical and medical conditions. Prompt identification of the etiology is important in the successful management of the case. An insight into the approach to the child with abdominal pain is discussed here.*

Keywords: *Acute abdomen, Surgical causes.*

Abdominal pain is a common complaint for which children seek medical attention. Diagnosing the cause of abdominal pain can be difficult, because many diseases can cause this symptom. The most frequent cause is medical, but surgical causes are more serious which may require urgent intervention. Sometimes a medical disease can progress to a surgical complication which should be recognised and intervened.

Though the abdomen is a Pandora's Box throwing surprises, the enigma that is associated with abdominal pain can be understood by adopting a systematic approach in evaluation. Abdominal pain may be due to organic or functional causes. Organic pain has physical, biochemical and pathologic basis.

Neurological basis of abdominal pain

Pain receptors in the abdomen respond to mechanical and chemical stimuli.[1] Stretch is the principal mechanical stimulus involved in visceral nociception, although distention, contraction, traction, compression and torsion are also perceived. Visceral receptors responsible for these sensations are located on serosal surfaces, within the mesentery and within the walls of hollow viscera, where they exist between muscularis mucosa and sub mucosa.

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Abdominal pain of functional aetiology

Abdominal pain affects 20% of school children and results in school delinquency, disruption in family routine, creating anxiety and depression for the parents.[2] Functional abdominal pain (FAP) is a genuine abdominal pain, but has no objective evidence of underlying organic disease.

The vast majority of medical causes of functional abdominal pain falls under the ambit of "Functional Gastrointestinal disorder ((FGID)".This group of disorders are well recognised and classified as per Rome criteria.[3] They are an important cause of recurrent abdominal pain and include functional dyspepsia, irritable bowel syndrome and abdominal migraine. The primary role of the pediatrician is to identify 10-15% of these children in this FGID group who in due course will have underlying organic disorder and will exhibit change in symptomatology or develop focal findings on abdominal examination.

Abdominal pain of organic etiology

The central focus in evaluating children with organic abdominal pain (OAP) is to determine if pain requires medical or surgical intervention. OAP can be acute or chronic in onset. A detailed history, complete physical examination and appropriate investigations are the most important elements in the evaluation of organic abdominal pain. History eliciting should be obtained directly from the patient regarding pertinent aspects of pain like location, timing, frequency, radiation and its relation to food. Physical examination should be done in a congenial environment in the presence of the parents. Findings of jaundice, rashes, swelling, bruising, injuries, signs of abuse, pain on limb movements, costovertebral angle tenderness suggest OAP. Perianal area examination combined with gentle digital examination should be done in select cases to detect bleed per anus, mass within rectal lumen or outside. The site of pain can point to the etiology (Table I).

Red flag signs of surgical abdomen

Red flag signs and symptoms of organic causes of pain abdomen are most likely due to surgical conditions.[4] Bilious emesis, colicky pain progressing to constant pain, localised pain and tenderness, guarding, visible intestinal peristalsis and mass on palpation point to surgical causes of abdominal pain.

Table I. Site of abdominal pain vs etiology

Site of abdominal pain	Condition
Epigastrium	Acid peptic disease, pancreatitis, appendicitis
Upper right abdomen	Hepatitis, cholecystitis, liver abscess, choledochal cyst
Middle and right lower abdomen	Appendicitis, Meckels diverticultis, malrotation, FGID
Loin	Renal diseases, ureteric colic
Suprapubic	Cystitis, ovarian torsion
Left upper and lower abdomen	Constipation, Hirschsprungs disease

Investigations

The laboratory and imaging evaluation should be driven by an index of suspicion based on the history, signs and symptoms elicited during the course of examination.

Laboratory panel should include a complete blood count including platelet count, blood sugar, renal function test, electrolytes and urine routine examination. Additional investigations will be case specific.

Imaging: A plethora of imaging modalities are available today. Appropriate and focussed investigations should supplement the provisional diagnosis. Priority should be given to basic investigations like X-ray. This is especially important in evaluation of acute pain abdomen and should neither be skipped nor replaced before the next level of imaging is planned.

Plain films of the abdomen are diagnostic in acute abdominal pain due to surgical causes especially in bowel obstruction and hollow viscous perforation. Findings such as free air (pneumoperitoneum), dilated small bowel loops of jejunum and ileum give an immense clue to the surgical cause. Skiagram will also reveal supra diaphragmatic diseases such as pneumonia, pleural effusion which will be referred as upper abdominal pain.

Ultrasonogram abdomen (USG) is the most useful noninvasive investigation and it should not be omitted. Positive USG helps to plan the next level of investigations.

Contrast upper and lower GI studies will aid in diagnosing conditions causing chronic abdominal pain such as bands, malrotation, strictures, polyps and inflammatory bowel disease.

Contrast enhanced CT abdomen (CECT) with both intravenous and oral contrast is a good diagnostic tool in selected cases of gastrointestinal, urinary tract and retroperitoneal diseases. The risk of ionising radiation should be weighed against the reward of diagnosis.

Magnetic resonance imaging technique (MRI) in conjunction with techniques for vascular imaging is an excellent tool for diagnosing vascular anatomical problems. Mesenteric vascular disorders, malignant mass lesions, intestinal malrotation can be diagnosed by this noninvasive method. Magnetic resonance cholangio pancreatography (MRCP) is a good non invasive imaging modality for pancreatic - biliary disorders.

Direct visualisation techniques like upper GI endoscopy, colonoscopy aids in diagnosis of specific conditions causing abdominal pain, like acid peptic disease, Crohns disease, ulcerative colitis, polyps etc.,

Minimal invasive surgery (laparoscopy) can be diagnostic as well as therapeutic in many conditions causing chronic abdominal pain.

Acute medical abdominal pain masquerading as surgical abdomen

There is an increased incidence of certain infectious diseases in certain seasons. The symptomatology of these medical diseases will catch even an astute clinician unaware, who will consider them as a surgical cause for abdominal pain. A common and important condition is dengue fever. Serositis of the peritoneal surface and capillary leak in this condition will produce generalised tenderness over abdomen. Contemplating surgical intervention for abdominal pain in dengue fever will spell disaster. Clinical suspicion and appropriate investigations will clinch the diagnosis.

The other medical conditions which merit mention are leptospirosis and diabetic ketoacidosis. The former, which is another seasonal disease presenting with fever and

jaundice can produce severe right upper quadrant pain due to acalculous cholecystitis. But this can be managed by medical treatment and cholecystitis resolves.

Juvenile diabetic ketoacidosis is an important metabolic condition which can mimic acute surgical abdomen. Severe dehydration and tachypnoea due to acidosis along with biochemical parameters should alert the clinician of this possibility.

Acute medical abdominal pain transiting to surgical intervention

Initial presentation of dysentery which does not resolve with established medical treatment should alert the clinician of the possibility of segmental enteritis. This is a form of mesenteric vasculitis.[5] Abdominal pain, prostration and development of bilious emesis differentiate dysentery from segmental enteritis. Skiagram of abdomen will reveal jejunal loops which are a characteristic finding of this disease. Prompt surgical consultation and further management by the pediatric surgeon is most appropriate for this condition.

Female children may present with high grade fever, abdominal pain and sometimes in septic shock. Re-evaluation after medical management should give rise to strong suspicion of acute primary peritonitis. Laparoscopy or laparotomy should be performed.

Chronic medical abdominal pain transiting to surgical intervention

Children who are diagnosed to have pancreatitis are on medical management for a considerable period of time. Medical management is empirical and directed in providing symptomatic relief to pain. Chldren's addiction to parenteral narcotics is now becoming common. The best indication for surgical intervention is based on MRCP findings of the pancreatic duct size. In children, if the pancreatic duct size is 8 mm or more, surgical drainage of the duct should be done which gives good relief from pain and eliminates the need for narcotics.

Another condition in this group is Crohns disease which is usually managed medically. But evolution of obstructive symptoms like strictures, fistulas and increasing frequency of abdominal pain are indications for surgical intervention (Fig.1).

Acute surgical abdominal pain always managed surgically

The red flag signs previously described along with appropriate investigations should enable recognition of the following surgical conditions such as appendicitis, Meckels diverticulitis, intussusception, intestinal malrotation,

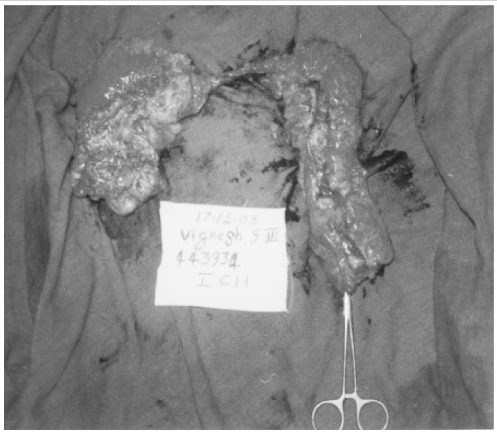

Fig.1. Crohn's disease

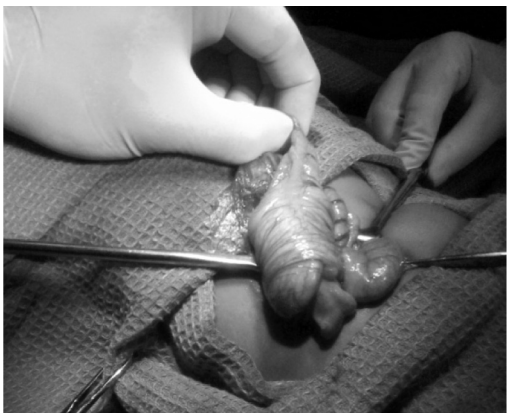

Fig.2. Intussusception

choledochal cyst, ovarian torsion, strangulated inguinal hernia, perforating and blunt abdominal trauma, splenic injury, iliopsoas abscess and suppurative mesenteric adenitis. These common conditions are to be considered and managed surgically (Fig.2).

Acute surgical abdominal pain, with initial medical management

The following conditions with severe acute abdominal pain such as ureteric colic, cystitis, acute pyelonephritis, acute pancreatitis and acute cholecystitis are initially managed conservatively. Surgical intervention is required on a case to case basis depending on the disease progression which is assessed clinically and by investigations.

Conclusion

Abdomen is a temple of surprises. The myriad of causes underlying the abdominal pain can be assessed by eliciting the patient's history, physical examination and performing appropriate investigations. The diagnosis can be established in a majority of cases and triage of the abdominal pain as medical or surgical will become easy if a systematic approach is adopted.

Points to Remember

- ***Bilious emesis ,focal tenderness, guarding and X ray revealing small bowel dilatation indicate surgical cause of abdominal pain.***
- ***Change of pattern and persistence of symptoms warrant further evaluation for surgical causes.***
- ***Laparoscopy has an important role in the evaluation and management of abdominal pain.***

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CLIPPINGS

Drug treatment for myotonia (delayed muscle relaxation after contraction) in muscle diseases such as myotonic dystrophy and myotonia congenita

Abnormal delayed relaxation of skeletal muscles, known as myotonia, can cause disability in myotonic disorders. Sodium channel blockers, tricyclic antidepressive drugs, benzodiazepines, calcium-antagonists, taurine and prednisone may be of use in reducing myotonia.

Objectives was to consider the evidence from randomised controlled trials on the efficacy and tolerability of drug treatment in myotonia .

Selection criteria: We considered all (including quasi) randomised trials of participants with myotonia treated with any drug treatment versus no therapy, placebo or any other active drug treatment.

Authors' conclusions: Due to insufficient good quality data and lack of randomised studies, it is impossible to determine whether drug treatment is safe and effective in the treatment of myotonia. Larger, well-designed randomised controlled trials are needed to assess the efficacy and tolerability of drug treatment for myotonia.

Trip J, Drost GG, van Engelen BGM, Faber CG. Drug treatment for myotonia. Cochrane Database of Systematic Reviews 2006, Issue 1. Art. No.: CD004762. DOI: 10.1002/14651858.CD004762.pub2. Assessed as up to date: July 30, 2009.

Renal doppler evaluation in the child with hypertension

The purpose of this study was to determine the utility of Doppler US in children as a screening tool and to better define clinical features of children in whom arteriography should be performed. Doppler US is a useful screening examination when evaluating children with hypertension, detecting renal artery stenosis in most affected children. The clinical risk classifications are helpful in guiding which children should proceed with arteriography regardless of the Doppler US results.

Chhadia S, Cohn RA, Vural G, Donaldson JS. A reasonable screening discriminator. Pediatr Radiol 07/24/2013.

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ANTIBIOTIC RESISTANCE – PREVENTIVE STRATEGIES

***Suresh Kumar D**

Abstract: *Antimicrobial resistance is recognized as one of the great threats to human health worldwide. The discovery of antibiotics seven decades earlier fundamentally transformed the way physicians care for patients, shifting their approach from a focus on diagnoses without treatment to a treatment-focused approach that saves lives. Seven decades of medical advances achieved by antibiotics are now seriously threatened by the convergence of relentlessly rising antibiotic resistance and the alarming and ongoing withdrawal of pharmaceutical companies from the antibiotic market with dry antibiotic pipeline. Without antibiotics, diverse fields of medicine will be severely hampered, including surgery, the care of premature infants, cancer chemotherapy, care of the critically ill and transplantation medicine, all of which are feasible only in the context of effective antibiotic therapy. The optimum solution to tackle the problem of antibiotic resistance remains investment in the infrastructure required to reduce the burden of infectious diseases. However, in the short term the best approaches rely on increasing awareness about antibiotic misuse, developing standard treatment guidelines for practitioners in different settings, restricting the choice of antibiotics, and providing feedback to practitioners on local patterns of resistance. In this article we are exploring how to tackle this global crisis locally.*

Keywords: *Antibiotic resistance, Problem tackling.*

Antibiotic resistance (AR) is a global problem, however it is particularly worrying in India, where infectious diseases are much more common.[1,2] Appropriate microbiological and diagnostic facilities are rarely available or affordable, hospital standards are inconsistent, antibiotics are readily available over the counter at pharmacies, poor drug quality owing to use beyond expiry data, improper manufacture or storage conditions may also contribute to resistance. Antibiotic misuse by unskilled practitioners is prevalent and there is complete lack of awareness among physicians[3] and public regarding the problem. Nearly 70% of India's 1.2 billion populations live in rural areas where, despite government efforts, hospitals are lacking basic diagnostics, medications, physicians and treatment guidelines. Rather than relying on physicians, many rural patients turn to local pharmacists and whatever drugs they have in stock.

Microbes do not need our help in creating antibiotic resistance.[4] On the other hand, what our antibiotic abuse can do is to lead to early onset of resistance and increase the rate of spread of bacterial resistance by applying selective pressure via exposure to the thousands of metric tons of antibiotics we have used in patients and livestock over the past half century. Ultimately, we must concede that we will never truly defeat microbial resistance; we can only keep pace with it. The only viable, long-term solution to the problem of microbial resistance is to have in place in perpetuity a continuing, steady development of new antibiotics, better sanitation infrastructure, antibiotic sparing strategies including immunotherapeutics and vaccines, diagnostics, antibiotic stewardship programs to improve targeted therapy, and well coordinated infection control programs to respond to new drug-resistant threats.

Role of (IAP) Indian Academy of Pediatrics in tackling antibiotic resistance

1. Lack of awareness and knowledge among physicians

The major reason that antibiotics are prescribed inappropriately is that there is a lack of awareness about antibiotic resistance and knowledge about infectious diseases and antimicrobial therapy.[3] Physicians are afraid not to prescribe antibiotics. IAP should take leadership role in educating its members by organizing antibiotic week to create awareness about antibiotic resistance (similar to breastfeeding week), dedicated workshops, short term courses, standard treatment guidelines, case based discussions, newsletters, journal articles, separate sessions during annual conferences regarding antimicrobial resistance and antibiotic stewardship. These educational efforts must be expanded to undergraduate and postgraduate students, pharmacists and nurses.

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2. Patient, public and media engagement and information

One of the challenges with the prescribing of antimicrobials in primary care is managing patient expectations. Substantial increases in public education about bacteria and anti-biotic resistance is very important. The general public must be made aware of the facts concerning the important roles that bacteria have in their lives and well-being, the precious nature of antibiotics and the concomitant importance of using them prudently.

There are a number of resources that can assist practices and patients including leaflets, posters and information sheets and some practitioners already use information leaflets during a consultation as an alternative to prescribing antibiotics, when this is appropriate. Greater involvement of the community, voluntary sectors and proactive engagement with media should be encouraged to disseminate the message about appropriate usage of antibiotics to the wider public.

3. Monitoring of antimicrobial usage and surveillance of resistance

Effective antimicrobial resistance surveillance systems are required (similar to acute flaccid paralysis surveillance) to rapidly identify existing and emerging resistant organisms; measure the prevalence of these organisms; identify any associations between antimicrobial resistance and antimicrobial prescribing patterns; and devise strategies to limit spread. The role of social medias in the surveillance should be explored.

4. Research and development

Research undertaken locally or elsewhere may highlight aspects of antimicrobial prescribing or resistance which may require changes in healthcare practice. Young researchers and postgraduates should be encouraged and supported with funds to do research in infectious diseases, antibiotic resistance and journals should fast track their publications.

5. Antimicrobial stewardship in all primary care settings

Antimicrobial stewardship in primary care is one of the responsibilities of primary care providers and is an integral part of prescribing practice. Approximately 80% of antimicrobial prescribing is estimated to take place in the community, therefore if an antibiotic is required it is important that primary care prescribers use an appropriate antibiotic at an appropriate dose, given the increasing problems with resistance and the need to preserve the usefulness of more specialized antibiotics. Primary care prescribers also need to manage the pressures that come from patients' expectations for an antibiotic.

Role of Individual physicians

Antimicrobial resistance is a world-wide threat but it needs action at local level as well as globally. The cornerstone of any plan to reduce antimicrobial resistance is the prudent and appropriate use of existing antimicrobials. It is critically important that members of the broader pediatric community become advocates in this campaign and work with the Indian Academy of Pediatrics (IAP) to put a "human face" on the problem of drug resistant infections. In the meantime, physicians must take care to prescribe antibiotics appropriately, to minimize the rate of spread of drug resistance. Most importantly, we must educate each other, our patients, the media, and politicians about this problem. Only the medical community can provide an accurate perspective and rational balance to this issue.

Conclusion

Antibiotic resistance is life-threatening in the same sense as cancer both in the numbers and the likely outcome; it must be taken as a matter of extreme urgency, and tackled immediately. The cost of the undertaking above recommendations will be infinitesmally small in comparison to the economic and human cost of doing nothing. The late Joshua Lederberg observed: "barring geno-suicide, human domination is challenged only by pathogenic microbes, for which we are the prey, they are the predators.[5] In natural evolutionary competition there is no guarantee that we will find ourselves the survivors." "The future of humanity and microbes will likely evolve as episodes of our wits versus theirgenes".

Points to Remember

- ***Educate pediatricians, students and public regarding antibiotics and antibiotic resistance.***
- ***Establish national and regional surveillance system to monitor antibiotic resistance patterns.***
- ***Before writing antibiotic, write diagnosis and if you want to use antibiotic use maximum dose for minimum period.***
- ***Involve specialists in the management of difficult to treat/multidrug resistant infections.***

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BOOK REVIEW

MALARIA (Third Edition)

Authors: Gupta BD, Maheswari RK

Publishers: United India Periodical Pvt. Limited, New Delhi.

Pages: 342

Price: Rs.295/-

This book reviews the current knowledge regarding the unique infection, malaria. This book is entirely devoted to all aspects of malaria. This book is divided in to various sections on history, vectors, life cycle, immunology, prevention of malaria, etc.The highlight of the book is laboratory diagnosis of malaria; many of the controversies are well discussed. The book is printed on a quality paper using large print which makes reading an enjoyable experience. Overall, it is a very well written book on malaria with special chapters on malaria in children. The book will be of great boon to the post graduate students, faculty members and practicing physicians.

Reviewed by:

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NEWS AND NOTES

NECON 2013 (XXXIII Annual Convention of National Neonatology Forum)

Hyderabad, December 13-15, 2013

Enquiries to:

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IAP-IJPP CME 2013

NEUROIMAGING OF THE PEDIATRIC BRAIN – A PICTORIAL REVIEW OF MR IMAGING STRATEGIES

Gopinathan K

Abstract: *Magnetic resonance imaging of pediatric brain is nowadays routinely used as imaging method of choice for the detection of morphological and functional changes of the brain . The advent of fast sequences, which allow high signal-to-noise, high-resolution datasets has facilitated standardized and reliable protocols for MRI to be acknowledged as a valuable tool in many paediatric centers. This review covers technical requisites, sequence details, and provides a practical approach for routine diagnosis of normal myelination and its imaging pitfalls. The characteristic imaging findings of various pediatric brain disorders which includes congenital malformations,arteriovenous malformations, paediatric stroke , tumours and tumour related issues and neuro infections are briefly discussed. Finally a brief note of MR spectroscopy in normal pediatric brain and its pivotal role in various clinical challenges is discussed.*

Keywords: *MRI, Spectroscopy, MR Angiography, MR Venography, Pediatric brain.*

Magnetic resonance (MR) imaging of the pediatric brain has shown large insight into the maturational processes that take place after birth. The MRI technique and its inherent tissue contrast have made possible to see in minute detail changes in cortical folding, involution of the germinal layer, premyelination changes within white matter, myelination, iron deposition and the various aspect of the disease process that is not possible with computed tomography (CT) or ultrasound (USG). Advanced techniques like diffusion, spectroscopy, perfusion, CSF flow imaging and functional bold MRI technique are extremely useful in study of the cellularity, cell injury, metabolic changes, tumor vascularity and cortical mapping.

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In other imaging modalities, CT shows poor soft tissue differentiation and contrast enhancement and lacks functional details when compared to MRI . But CT is more useful for the bony cortex integrity than MRI. It uses ionizing radiation and potentially adds radiation burden to the young children.

Regarding the ultrasonogram, even though it has an advantage of bed side availability, because of fontanella closure it is useful only in the neonates and young infants before the closure of fontanel and also its high observer variation limits the usage. So MRI plays major role in the identification and characterisation of the pediatric brain pathology.

Basic requirements

Atleast 1.5 T MRI unit is required. Basic T1, T2 and FLAIR sequence can be acquired in low tesla unit but rest of the advanced MRI studies (i.e DWI) can be done only on at least 1.5T magnets. Child friendly atmosphere like maintaining body temperature with insulate covering / MR compatible incubator for preterm infants are the other requisites. Child has to be stand still in the MR gantry for at least 12 min for meaningful MRI exam, to prevent movement related artifacts. Hence sedation is needed in most of the studies. Regarding sedation, most babies tolerate oral triclofos well only about 5% need IV sedation. Choice of IV medication (midazolam / propofol / ketamine) should be decided by the duty physician / anesthetist depending upon the child physical condition. MRI compatible monitors and anesthetic circuits are essential.

Indications: MRI is the primary imaging modalities in most of the brain pathologies except head injury where CT is more useful to find out bony fracture and subarachnoid hemorrhages quickly.

Contraindications: Absolute contraindications include, electronically, magnetically and mechanically activated implants (Cardiac pacemakers, ferromagnetic haemostatic clips in the central nervous system. Relative contraindications include cochlear implants, other pacemakers, e.g. insulin pumps and nerve stimulators, prosthetic heart valves haemostatic clips and non-ferromagnetic stapedial implants.

MRI signals, normal appearance and pathologies: Gross difference in the signal intensity of the brain parenchyma of the newborn and older children is mainly due to myelination. T1 hyper and T2 hypointensities of the newborn cortex becomes of intermediate SI by 4–6 months. By that time the white matter (WM) has myelinated and become of high SI on T1W and later low SI on T2W images. In myelination T1 effects precede than T2 changes, so hypo / dys / demyelinated white matter appear as T2 hyperintense and T1 hypointense areas.

Myelination of brain takes place in an orderly manner. Myelination progresses from caudal to cranial, dorsal to ventral, and from central to peripheral. Sometimes peritrigonal white matter appears hyperintense on T2 weighted with preserved rim of myelination consistent with terminal zone of myelination –seen upto 18-24 months. It should be differentiated from the periventricular leucomalacia which show loss of normal periventricular T2 hypointensity. At term, myelination is visualised in the thalamus, posterior limb of internal capsule (PLIC), part of the globus pallidus, optic and acoustic radiations as well as in the pre- and postcentral subcortical WM. Absent T1 bright PLIC is usually seen in the hypomyelination and hypoxic ischemic changes. Freely moving clear fluids like CSF / arachnoid cyst appear bright on T2 W and completely suppressed on FLAIR sequence, where as epidermoid cysts appear bright on T2W and not suppressed on FLAIR sequence. Prominent Virchow-Robin (VR) spaces noted normally in the anterior commissural region, subcortical region show central tiny vessel and surrounding fluid accumulation. It should not be misreported as lacunar infarct.

Fat appears hyperintense on T1W sequence and mild hypointense on T2W sequence. Bony cortex and calcium appear dark in both T1 and T2W sequence. Table I summarizes MRI signal intensity in different pathologies.

Table I. MRI signal intensity in different pathologies

	T1 WI	T2 WI	FLAIR
Solid mass	Gray	Bright	Bright
Cyst(fluid)	Dark	Bright	Dark
Fat	Bright	Gray	Gray
Acute blood	Dark	Bright	Bright
Sub acute blood	Bright	Dark	Dark
Chronic blood	Bright	Bright	Bright

A pictorial description of various MRI images from normal to pathological is given (Figs.1 to 24).

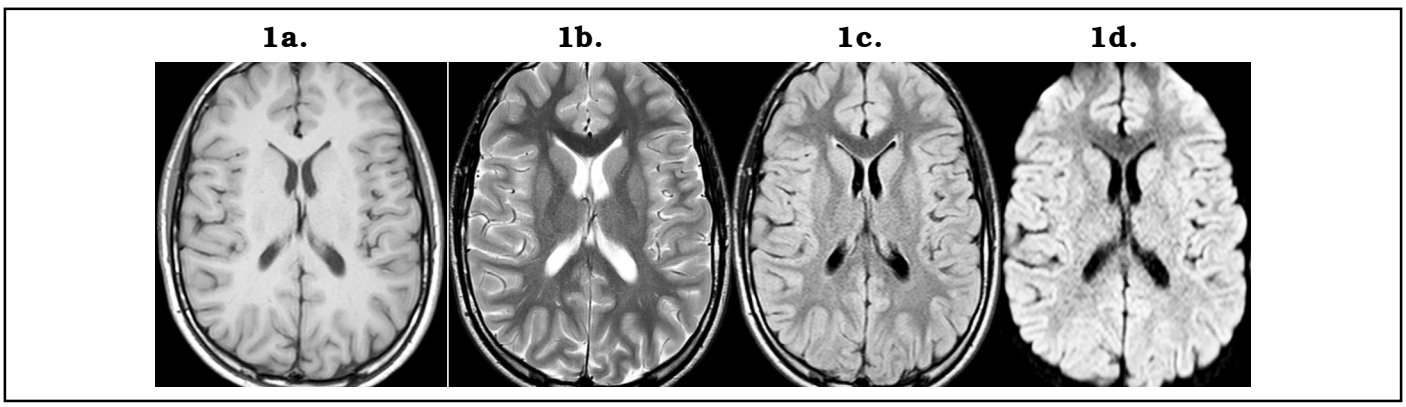


Fig.1. (1a to 1d): Normal MR imaging pattern in 2 yr old children

Fig.1a. T1 W images show hyperintense white matter and mild hypointense cerebral cortex. Fig.1b.T2 W images show hyperintense grey matter and hypointense white matter and bright CSF . Fig.1c FLAIR show same as T2 W images but dark CSF. Fig 1d. DWI show diffusion images but grainy appearance.

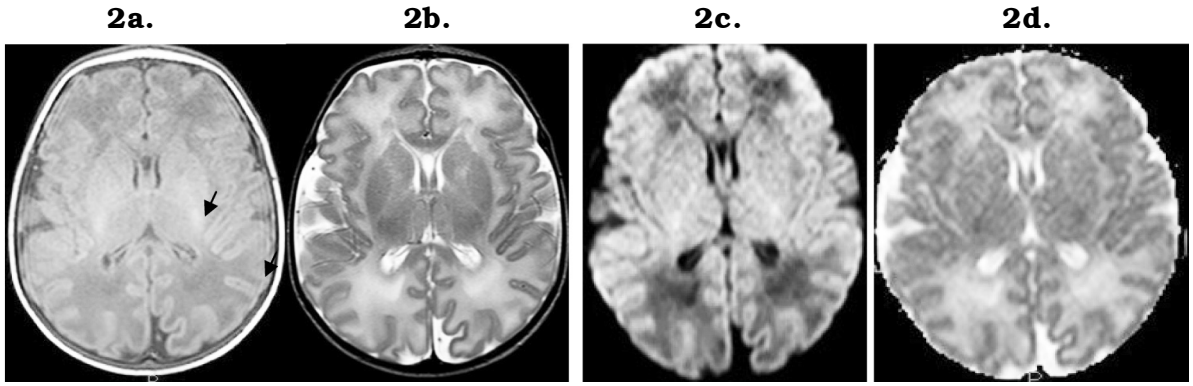


Fig.2. (2a to 2d) Term infant MRI brain

Fig 2a. T1W sequence- Hyperintense cerebral cortex and hypointense cerebral white matter. Fig 2b.T2W sequence –hyperintense white matter and hypointense cerebral cortex. Fig2c.DWI show mild diffusion restriction along the posterior limb of the internal capsule s/o myelinated fibres.Fig 2d. Apparent diffusion co-efficient (ADC) shows mild hypointensity of the posterior limb of internal capsule and facilitated diffusion along the rest of the white matter and corresponds to the non myelination.

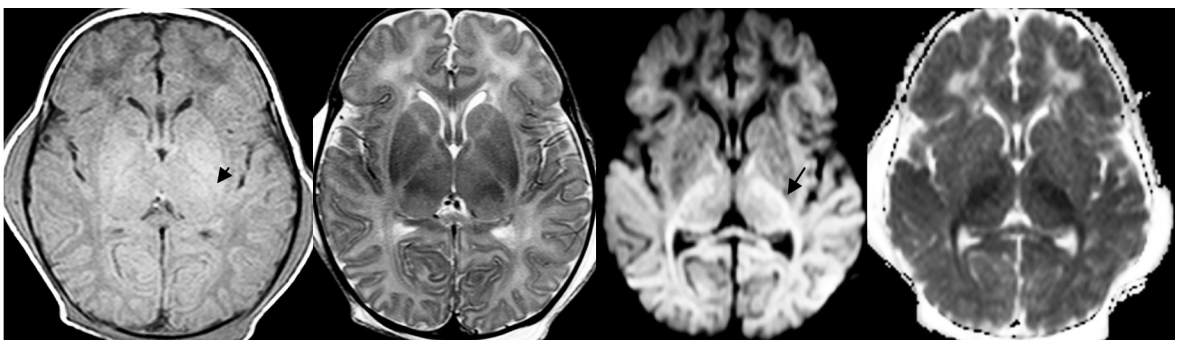


Fig.3. T1,T2,DWI and ADC show hypoxic ischemic encephalopathy changes - loss of the T1 hyperintense myelinated posterior limb of internal capsule with diffusion restriction.

MR signal intensities depend on oxygenation status of hematoma. It varies from acute, subacute and chronic hematomas (Fig. 4-8).

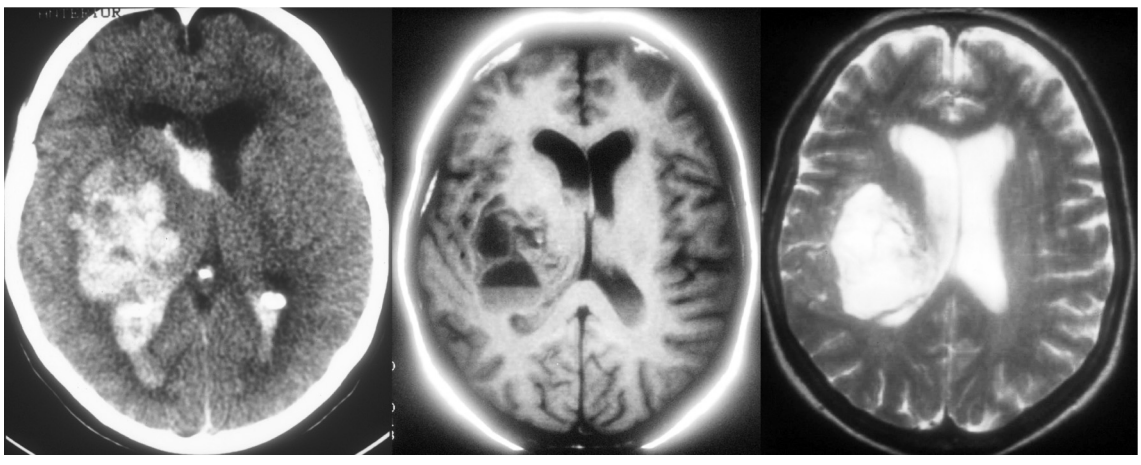
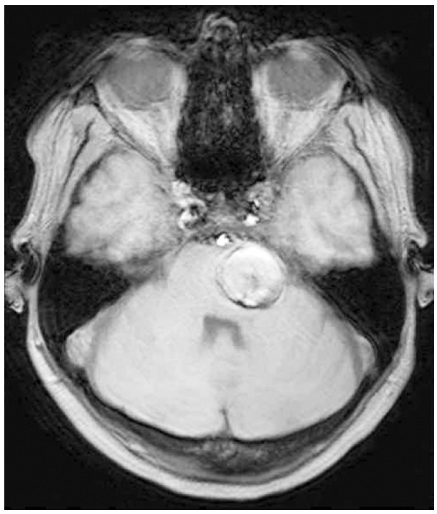


Fig.4. (4a to 4c) CT, T1 and T2 show acute intracranial hemorrhages in the right putamen and thalamus, which appear T1 hypo and T2 hyperintense lesions due to oxyhemoglobin in the hematoma.



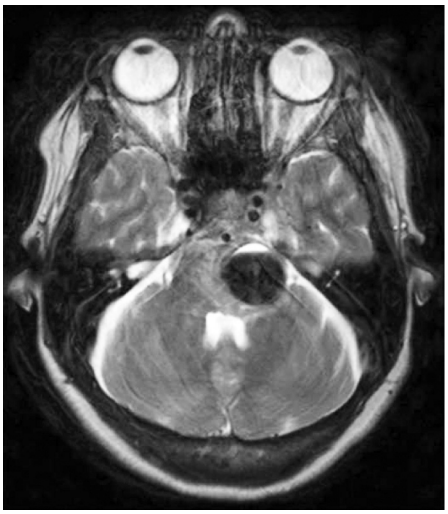
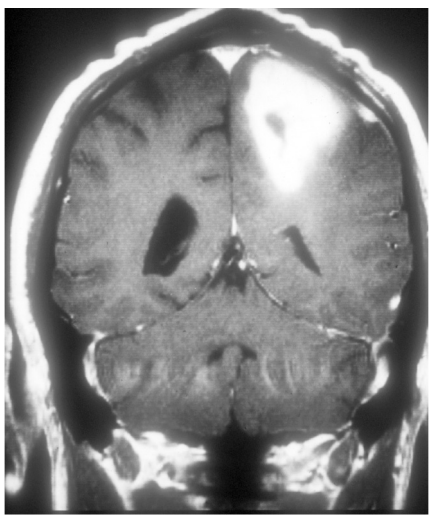


Fig.5. (5a & 5b). Early subacute hemorrhage in the left side of pons which appears T1 hyper and T2 hypointensity due to intracellular methemoglobin



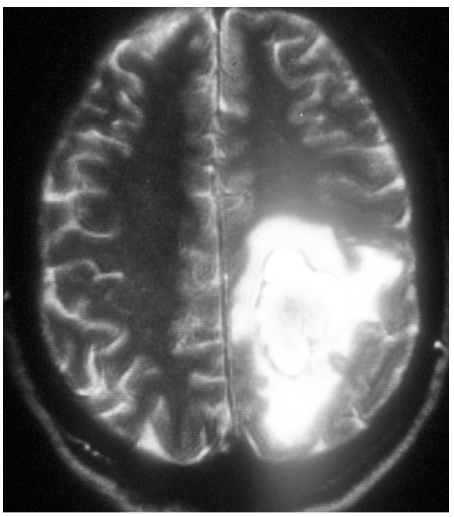


Fig.6. (6a & 6b). Left high parietal late sub acute hemorrhages which appear hyper intense on both T1 and T2 weighted images.

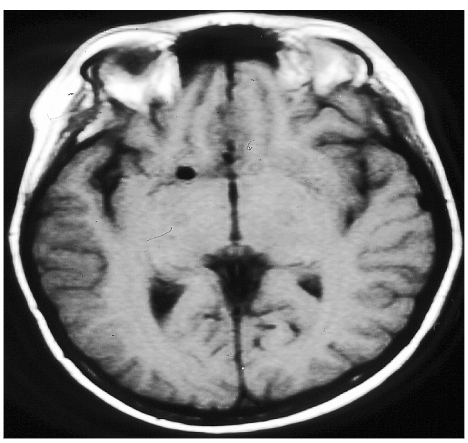


Fig.7. Flow void in the right MCA aneurysm

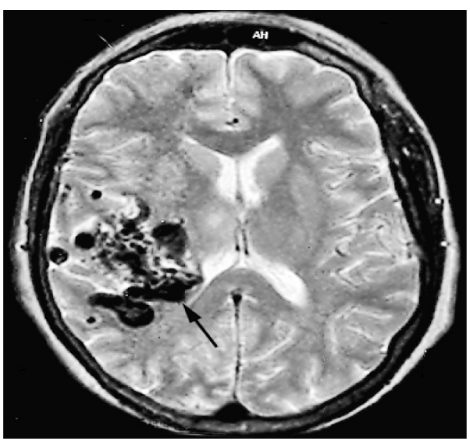
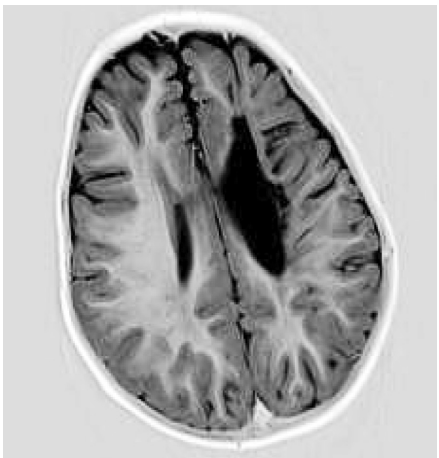


Fig 8. Serpigenous flow voids in AVM

Susceptibility weighted imaging / Gradients echo imaging is Useful for detection of the hemorrhages, calcium and iron deposition, mainly due to paramagnetic effect. Even remote hemorrhages can be picked using this sequence (Fig.9).



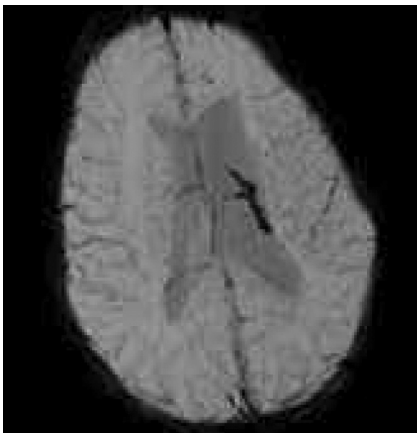


Fig.9. (9a & 9b). MRI brain 2 year old child SWI show chronic hemorrhages in the left ependymal margins, a case of perinatal hypoxia.

Congenital malformations are typically diagnosed with routine T1 and T2 imaging (Fig. 10-15).

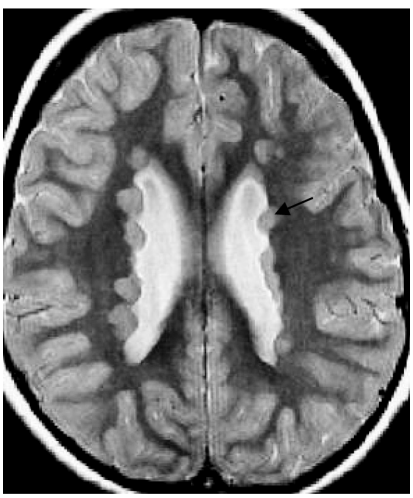


Fig.10. Nodular heterotopia - heterotopic nodule along the cortex with perilateral ventricular region

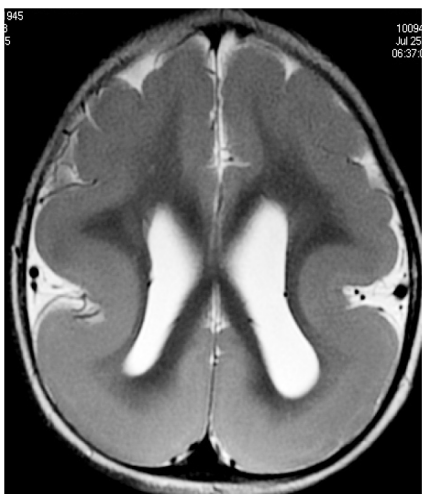


Fig.11. Lissencephaly - smooth thickend figure of 8 appearance

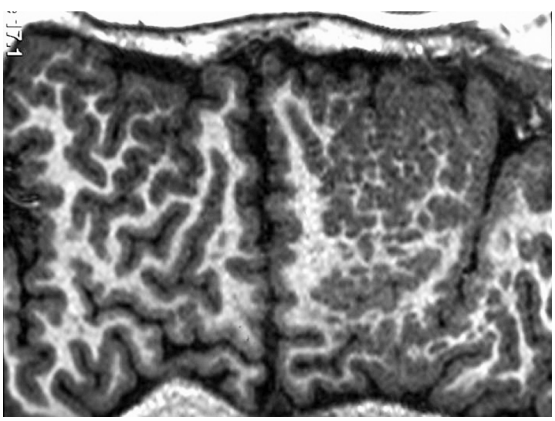


Fig.12. Polymicrogyria: Toomany malformed cortex in the left frontal region

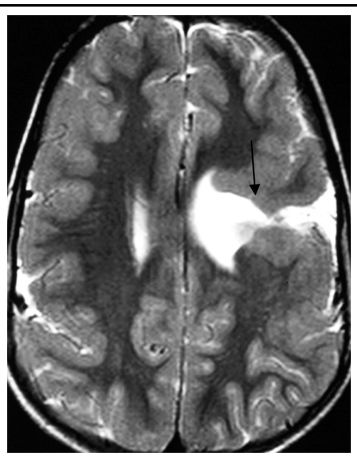


Fig.13. Scizhencephaly: Grey matter lined cleft in the left parietal region

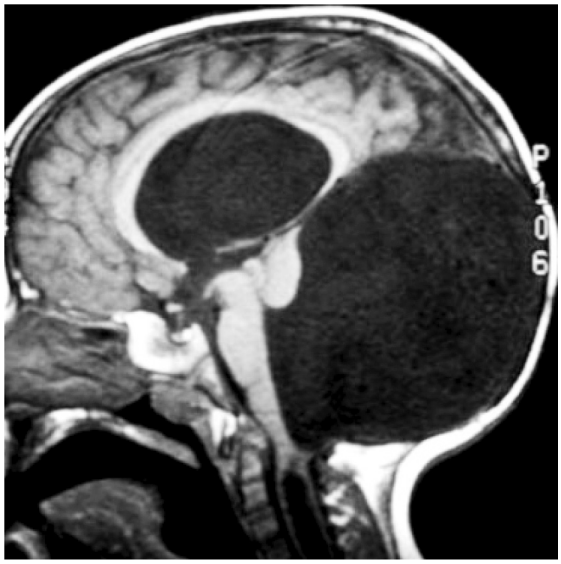


Fig.14. Dandy walker malformation Post fossa cyst with vermian agenisis

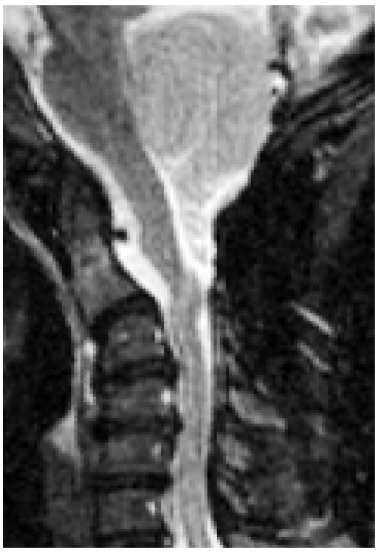


Fig.15. Arnold chiari malformation type 2 herniation of the brain stem and cerebellar tonsil

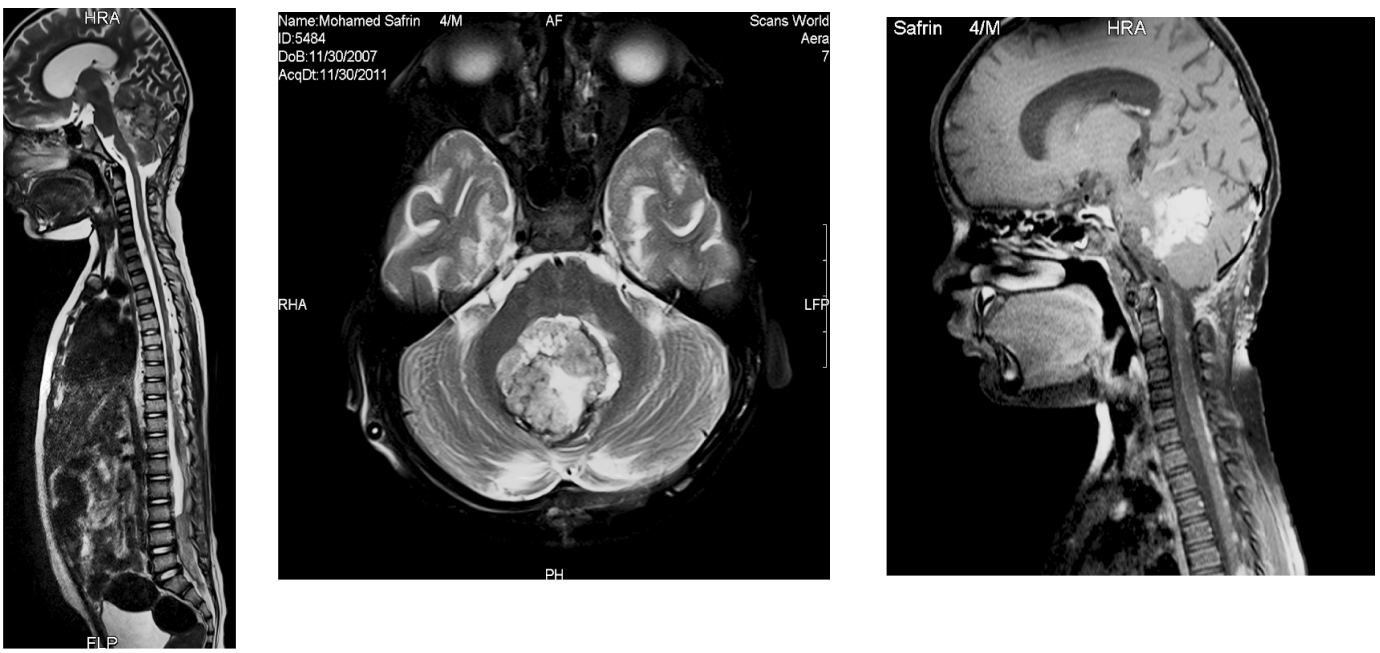


Fig.16. Medulloblastoma - Mass lesion of the vermis with drop metastasis in the lower spinal canal

When imaging the congential malformation, infection and tumor, entire neuroaxis (screening of brain and spine) is essential. Infection and drop metastassis can extend upto the caudal spinal canal (Fig.16). Also in brain development, varying steps (i.e proliferation and migration) taking place at overlapping time intervals, will result in spectrum of anomalies than single anomoly. The rule is if you find one anomaly you have to search for other one. Since brain and spinal cord are formed from the neural tube it is common to see the anomolies in all the levels.

MR angiogram: Both arteriogram & venogram can be taken without giving IV contrast (Fig. 17 & 18) .

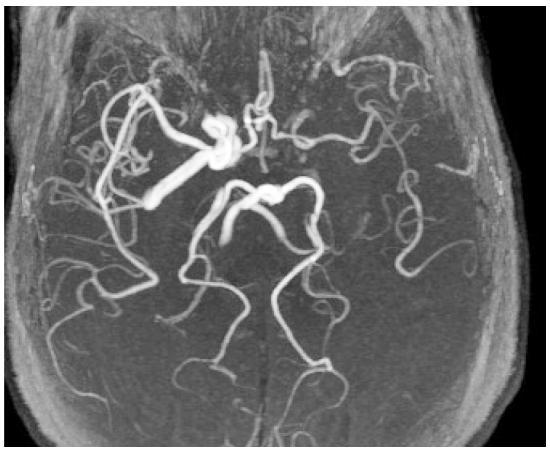


Fig.17. MR arteriography: Complete occlusion of left ICA

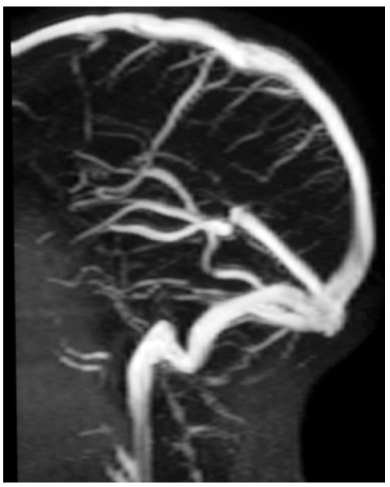
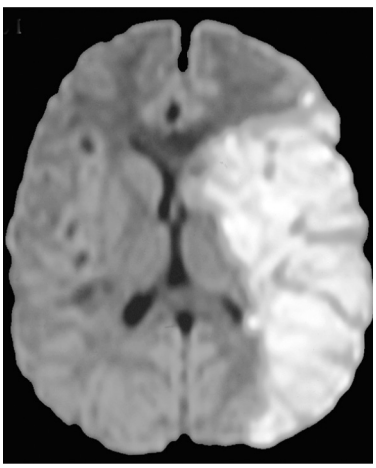


Fig.18. Normal MRVenography

DWI : Diffusion weighted images (DWI) reflect the brownian motion of water molecules within the brain. This diffusional motion is limited by tissue structure particularly in WM tracts where diffusion of water is free along the length of axons than across them. Diffusion weighted imaging is more useful in detecting cytotoxic edema, brain abscess (Fig. 19 to 21).



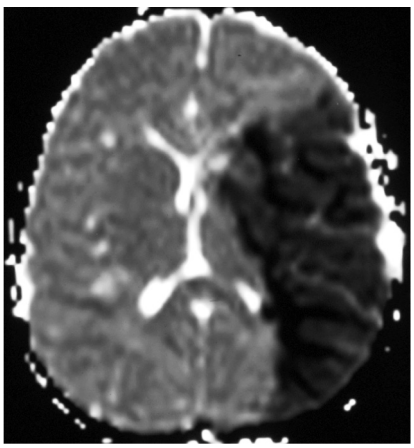


Fig.19. DWI & ADC show diffusion bright and corresponding low apparent diffusion co-efficient (ADC) value suggestive of acute infarct in the left middle cerebral artery territory

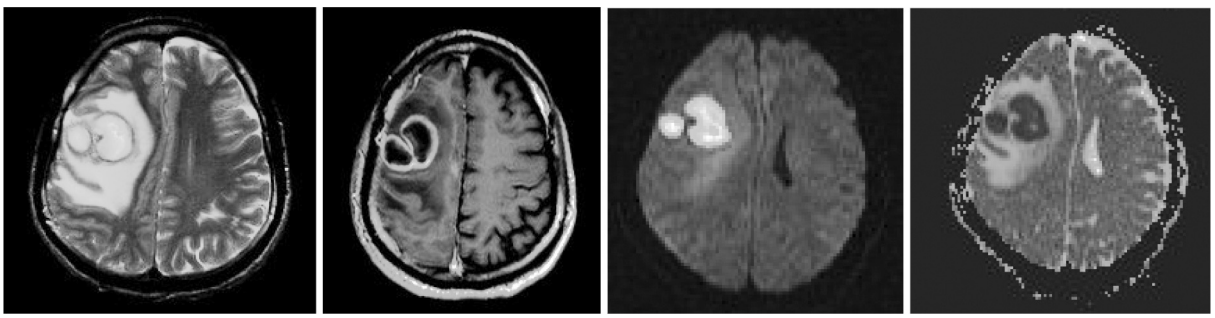


Fig.20. Brain abscess: Peripherally enhancing, T2 bright lesions with diffusion restriction

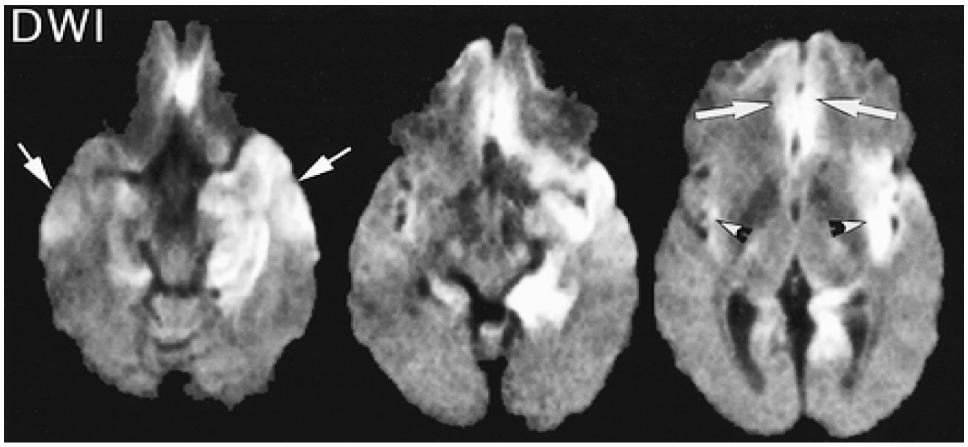


Fig.21. Diffusion restricting lesion in both temporal and frontal lobe, classical of herpes encephalitis.

Post contrast study

Intravenous gadolinium is useful in detecting blood brain barrier breakdown in infection / active demyelinative foci, neoplasm and meningeal enhancement in meningitis (Fig.22 to 24). Ring enhancing lesions are classically seen in tuberculomas/cysticercosis. But tumors, subacute hematomas, demyelination also show ring enhancement. Correlation of other sequences are essential for the diagnosis. Renal function status is essential because gadolinium can cause nephrogenic systemic fibrosis in renal function impaired patients.

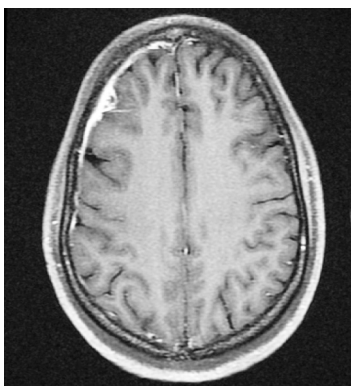


Fig.22. Pachymeningitis in the right frontal region

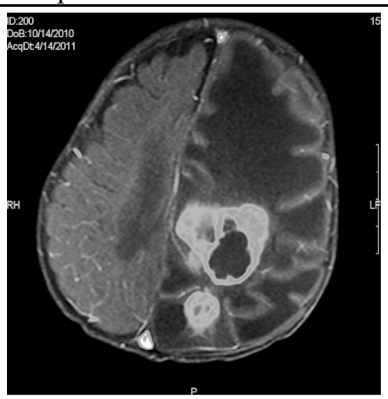


Fig.23. left parietal multiple ring enhancing abscess formation

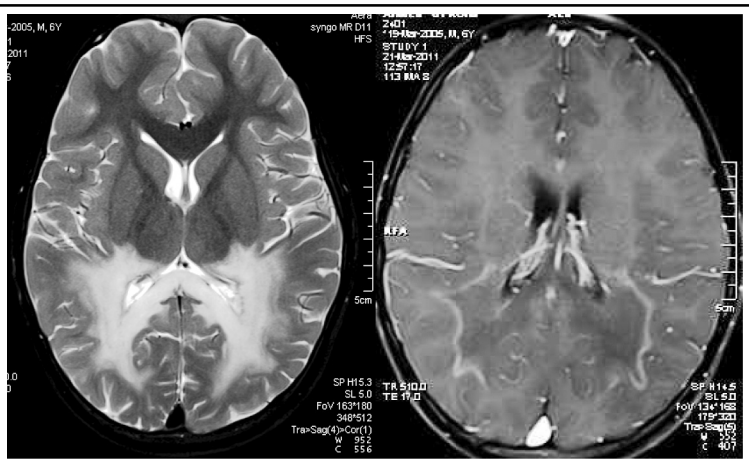


Fig.24. Adrenoleucodystrophy: Peripherally enhancing active demyelinative rim around the peri trigonal region

MR Spectroscopy (MRS)

MRS tells metabolites changes in the given area. Usually multivoxel spectroscopy technique is employed for simulataneous comparision of the abnormal and normal areas. Normally N acetyl aspartate (NAA) falls at 2ppm,choline at 3.2ppm, Creatine at 3 ppm and lipid and lactates at 0.9-1.3 ppm. NAA accounts for neuronal integrity, choline for cell turnover and myelination status, creatine for cerebral metabolism marker (internal reference) and lactate indicates anerobic metabolism.

Usually lactate peaks are not seen in normal brain parenchyma and are elevated in centre of the demyelination/ infarcts/mitochondrial disorders. Peaks also vary according to the myelination age. Largest peak in normal newborn brain is Cho, whereas largest peak in the 1 yr old normal child is NAA (Fig. 25 and 26). Prominent lactate peaks are seen in hypoxic ischemic encephalopathy (Fig. 27) and absent creatine peak in creatine deficiency (Fig. 28).

Sometimes spectroscopy is diagnostic in disease like Canavan's disease (Elevated NAA)(Fig. 29), non ketotic hyperglycenimia (at 3.56pmms) and congenital creatine deficiency syndrome (absent creatine peak) (Fig. 28) and phenylketonuria. Spectroscopy may be useful in differentiating the tuberculomas from the parasitic lesion (neurocysticercosis/hydatid cyst) by looking lipid peak in tuberculoma, where as parasitic cysts show prominent succinate (2.4ppm) and lactate (1.3) peak (Fig. 30 and 31). Aminoacid peak at 0.9 and 1.9ppms is seen in abscess.

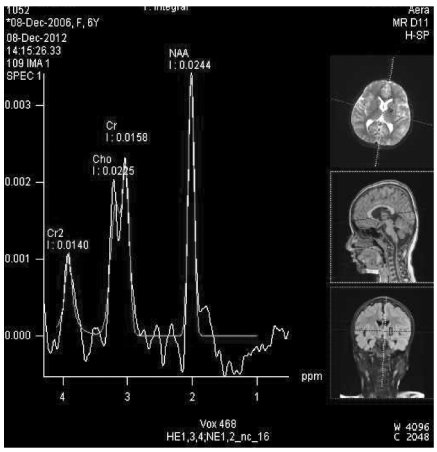


Fig.25. Spectroscopy of 6yr old child, show prominent NAA peak

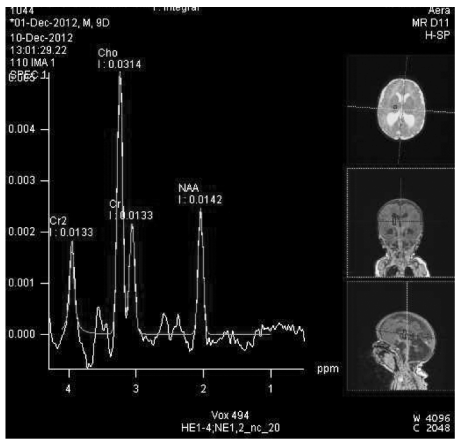


Fig.26. Prominent choline peak in 10 days old normal child

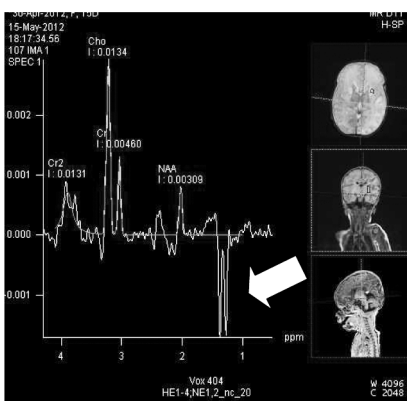


Fig.27. Prominent lactate peaks at 1.3 ppm congenital hypoxic ischemic encephalopathy

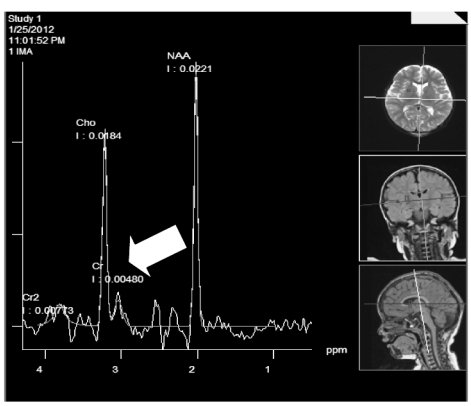
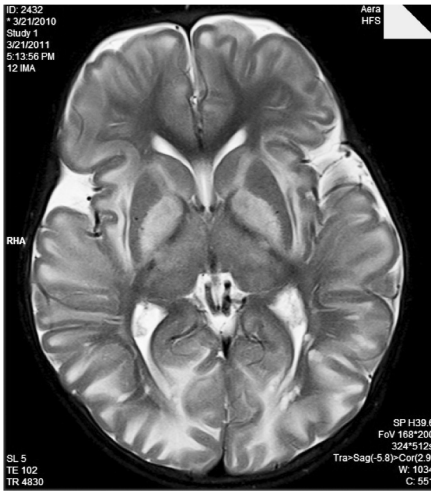


Fig.28. Absent creatine peak at 3 pmm in creatine deficiency



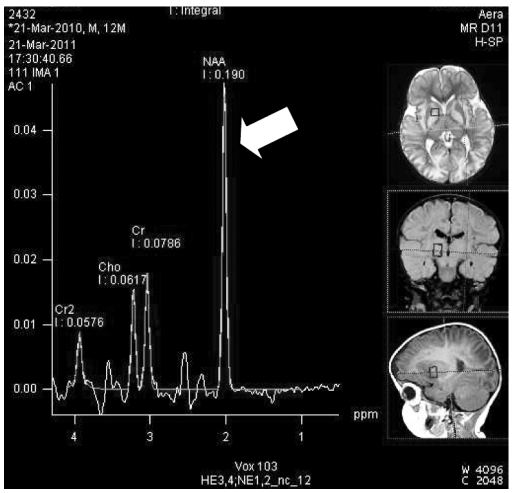
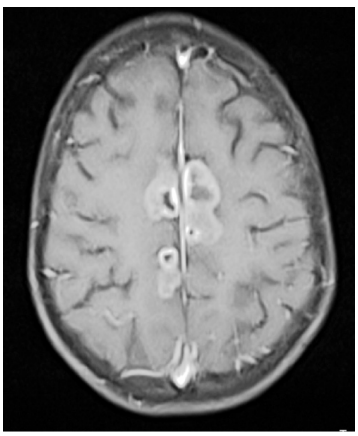
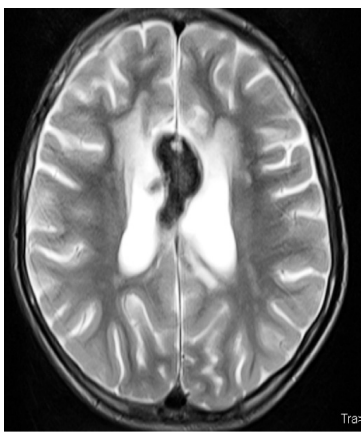


Fig.29. Elevated NAA peaks at 2.0ppm in Canavan disease





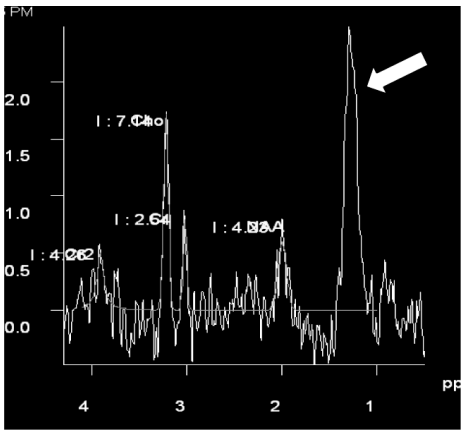
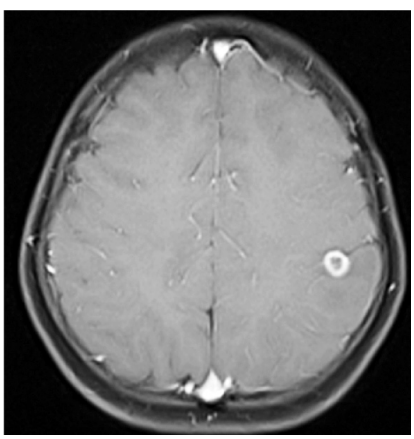
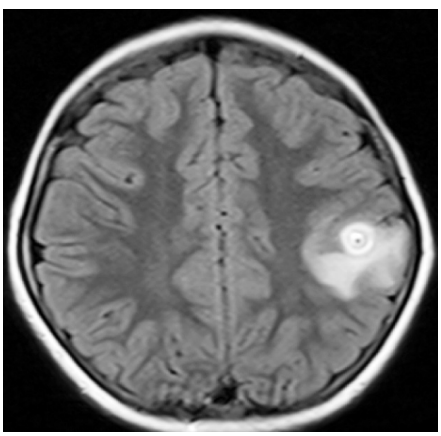


Fig.30. Tuberculomas: Ring enhancing T2 hypointense lesions with prominent lipid peak at 1.2 ppm





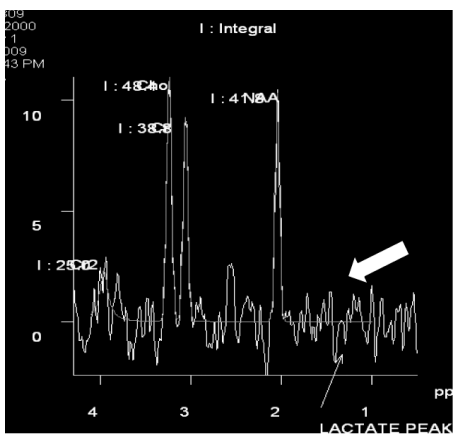


Fig.31. Neurocysticercosis, showing ring enhancing lesion with central dot and surrounding vasogenic edema and prominent lactate peak at 1.33 ppms

Conclusion

Interpretation and clinical appilication of pediatric neuroimaging requires, clear knowledge of various basic MRI sequences and the physics behind them. Vast knowledge about normal evolution of human brain and its varying imaging characteristics helps us to solve clinical queries.

The goal of this review was to summarize some of the best papers in the field of pediatric neuroradiology. However, the article highlights the imaging capability of MRI to noninvasively monitor physiologic changes in the normal and abnormal pediatric brain. The reader is encouraged to read the books and articles in their entirety to better understand the essentials contained in them.

Points to Remember

MRI plays vital role in

- ***Charecterisation of congenital anomalies accurately.***
- ***Prognostication of hypoxic ischemic encephalopathy using DWI & Spectroscopy***
- ***Prompt diagnosis of pediatric stroke and exclusion of stroke like lesions with the help of DWI.***
- ***Differentiation of various infections so that targeted therapy could be instituted.***
- ***Make evaluation of various pediatric leucodystrophies simple.***

For further reading

1. Pediatric Neuroimaging. Barkovich AJ. 3rd edn, Philadelphia, Pa: Lippincott Williams & Wilkins, 2012.
2. MRI of the Neonatal Brain, Mary A Rutherford. (ed).
3. Heinz ER, Provenzale JM. Imaging Findings in Neonatal Hypoxia: A Practical Review Am J Roentgenol 2009; 192: 41-47.
4. MR Imaging for Diagnostic Evaluation of Encephalopathy in the Newborn**.** *Radio Graphics* 2010; 30:763-780.

CLIPPINGS

Intrapartum antibiotics for known maternal Group B streptococcal colonization

Maternal colonization with group B streptococcus (GBS) during pregnancy increases the risk of neonatal infection by vertical transmission. Administration of intrapartum antibiotic prophylaxis (IAP) during labor has been associated with a reduction in early onset GBS disease (EOGBSD). However, treating all colonized women during labor exposes a large number of women and infants to possible adverse effects without benefit.

Objectives: To assess the effect of IAP for maternal GBS colonization on neonatal: 1) all cause mortality and 2) morbidity from proven and probable EOGBSD, late onset GBS disease (LOD), maternal infectious outcomes and allergic reactions to antibiotics.

Selection criteria: Randomized trials assessing the impact of maternal IAP on neonatal GBS infections were included.

Authors' conclusions: Intrapartum antibiotic prophylaxis appeared to reduce EOGBSD, but this result may well be a result of bias as we found a high risk of bias for one or more key domains in the study methodology and execution. There is lack of evidence from well designed and conducted trials to recommend IAP to reduce neonatal EOGBSD. Ideally the effectiveness of IAP to reduce neonatal GBS infections should be studied in adequately sized double-blind controlled trials. The opportunity to conduct such trials has likely been lost, as practice guidelines (albeit without good evidence) have been introduced in many jurisdictions.

Ohlsson A, Shah VS. Intrapartum antibiotics for known maternal Group B streptococcal colonization. Cochrane Database of Systematic Reviews 2013, Issue 1. Art. No.: CD007467. DOI: 10.1002/ 14651858.CD007467.pub3. Assessed as up to date: November 10, 2012.

IAP-IJPP CME 2013

LITERATURE SEARCH USING PUBMED

***Naresh P Shanmugam**
****Subashini P**

Abstract: *Literature search is an essential tool for evidence-based practice. It is difficult to get the right answer unless the right question is asked. While performing literature search unless the right question is asked, the search will not bring out appropriate articles. This article deals with basic methods involved in performing literature search using PubMed.*

Keywords: *PubMed, MEDLINE, MeSH*

Reading published literature is an important way of gaining knowledge in any field. The literature that a textbook provides is usually an overview, generic and mainly based on theories and not up to date. For evidence-based practice, knowledge about current practice could be obtained only through reading journal articles. Index Medicus is a catalogue that contains abstracts of all the indexed journals that would be found in a library. Couple of decades ago, any one who would like to look at published scientific articles about a specific topic had to go through hard copy of Index Medicus to collect abstracts of published articles on selected topic and if found useful, to retrieve the full article from published journal. This was considered to be time consuming and laborious process, but with no other option researchers who wanted to write papers or post graduates who would like to do thesis had to undergo this process. With the advent of internet, paper printed Index Medicus became obsolete and the last printed version was stopped in 2004.

All the material that was available in printed media in Index Medicus was made available in its online counter part MEDLINE. MEDLINE is the electronic database that contain references and abstracts on life sciences and biomedical topics. The United States National Library of Medicine (NLM) at the National Institutes of Health maintains the database.

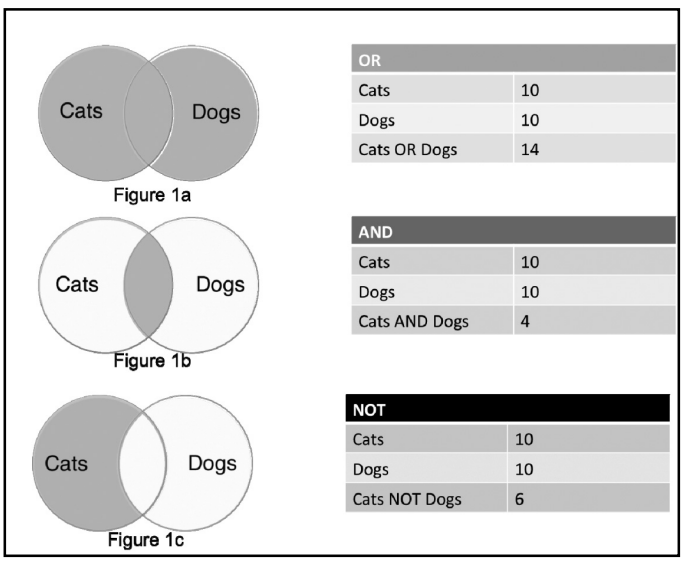
The internet interface through which the search could be performed is called PubMed. PubMed is the gateway to MEDLINE, the National Library of Medicine's bibliographic database. MEDLINE contains over 14 million records from over 4,600 current biomedical journals. Similar to MEDLINE there are other medical online databases such as Embase and The Science Citation Index.

Literature search

Performing a literature search starts with asking the right question, unless right question is asked the search engine will not give the articles you would be looking for.

Boolean logic

Most of the search engines (e.g. PubMed) was built on principles of Boolean logic. This refers to the logical relationship among search term. It has three logical operators: **AND**, **OR, NOT**. When several key words are used to search, these three operators are used as link words to help in bringing out the most appropriate search items. For example if we are looking for publications about cats and dogs, there would be always overlap publications which would include both cats and dogs. (Fig.1).



OR	
Cats	10
Dogs	10
Cats OR Dogs	14

AND	
Cats	10
Dogs	10
Cats AND Dogs	4

NOT	
Cats	10
Dogs	10
Cats NOT Dogs	6

Fig.1.

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Savitha Medical College, Chennai.

Using boolean logic operator if we use the link words OR, it will bring all the publications about cats, dogs and cats & dogs (Fig.1a), using link word AND it will bring up publications which has both cats & dogs (Fig.1b), and finally using the word NOT it would exclude one of them along with publications that has both cats & dogs (Fig.1c).

PubMed search

Entering key aspects of a subject into PubMed's search window can carry out simple searches on PubMed. PubMed translates this initial search formulation and automatically adds field names, relevant MeSH (Medical Subject Headings) terms, synonyms, Boolean operators, and the resulting terms appropriately, enhancing the search formulation significantly.

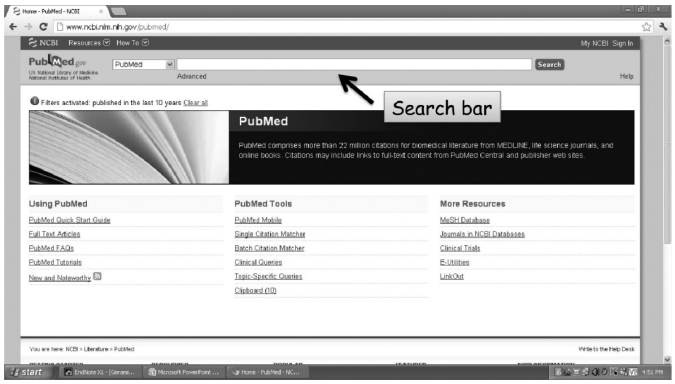

Fig.2a.

When www.pubmed.com is accessed, the interface screen where we would be working on opens up (Fig.2a).

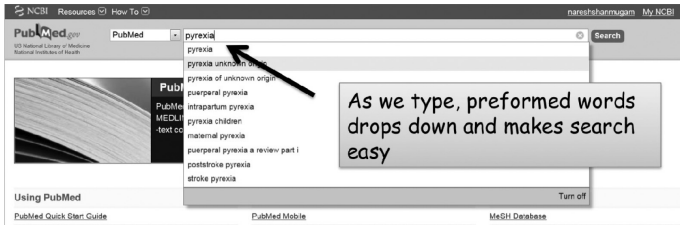

Fig.2b.

In the search bar we could type our search of interest, for example "pyrexia of unknown origin". When we start typing initial few alphabets a preformed menu drops down (Fig.2b), if we have the search question we could click on it or if intended search question is not there, free text typing could be done. When search button is clicked it will bring all the published articles where "pyrexia of unknown origin" is used as keyword (Fig.2c).

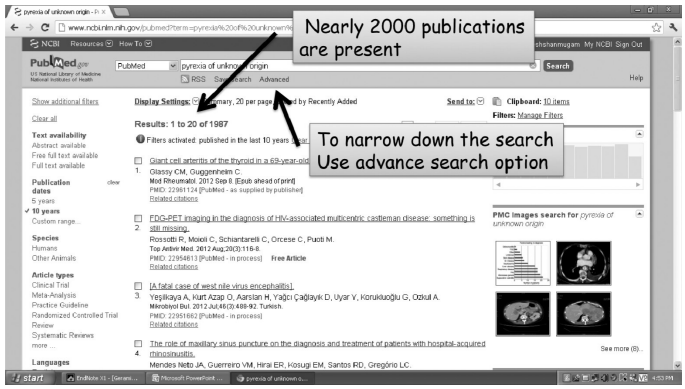

Fig.2c.

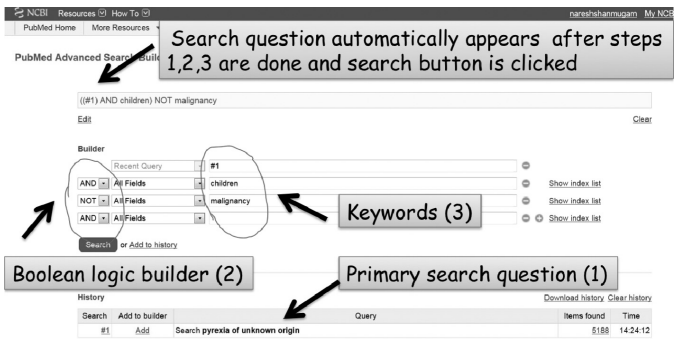

Fig.2d.

But if we want to be very specific such as "pyrexia of unknown origin in children excluding due to malignancy", then advanced option has to be used (Fig.2d). The advanced window has a Boolean logic builder, where the question would be "pyrexia of unknown origin AND children NOT malignancy". This will bring all the articles on the above mentioned question, which could be read through and interested articles could be saved (Fig.2e).

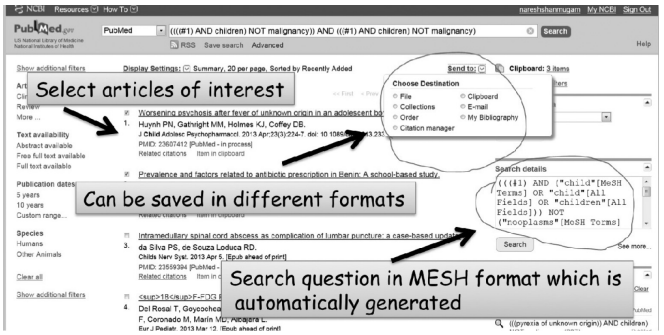

Fig.2e.

Medical Subject Headings (MeSH)

As there is quiet a lot of variability in individual search for same concept, the published articles have to have a consistent way to retrieve information. **MeSH** is the controlled vocabulary used for indexing articles for the

MEDLINE® subset of PubMed. MEDLINE/PubMed expert team reviews every journal article and index it with about 10-15 appropriate subject headings, subheadings and supplementary concept records, so that when the specific terminology is used in search, it brings out the article. This hierarchical categorization of headings, subheadings and supplementary is called "MeSH tree structures" and are updated annually.

MeSH contains nearly 27,000 descriptors (keywords), when typed and requested search, the window will provide a short definition, year of introduction and several subheading.

Subheadings are used to help describe more completely a particular aspect of a subject. In the MeSH database, subheadings logically paired with the main heading are presented. For example if the descriptor is "pyrexia of unknown origin", the subheading helps in more refined focused search such as classification, complications, management etc. If there are certain things which need exclusion it could be typed in the search builder e.g not malignancy.

In addition to the descriptors, MeSH also contains some 139,000 Supplementary Concept Records. These do not belong to the controlled vocabulary as such; instead they enlarge the thesaurus and contain links to the closest fitting descriptor to be used in a MEDLINE search. Many of these records describe chemical substances. MeSH terms apply only to Indexed for MEDLINE citations. A strong feature of PubMed is its ability to automatically link to MeSH terms and subheadings. Sample of MeSH search window is shown in Fig.3.

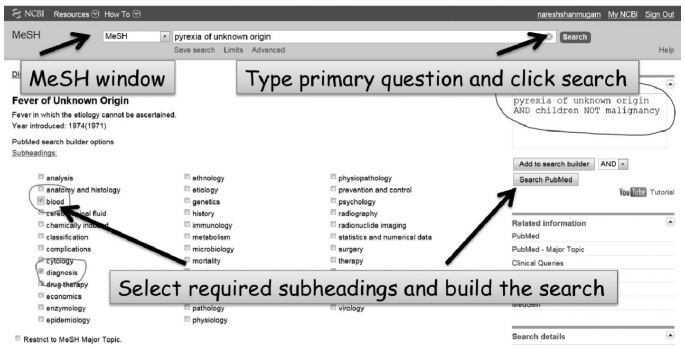

Fig.3.

Saving and retrieving the data

The PubMed has optional facility to register ("My NCBI"), which is free of cost (Fig.4a).

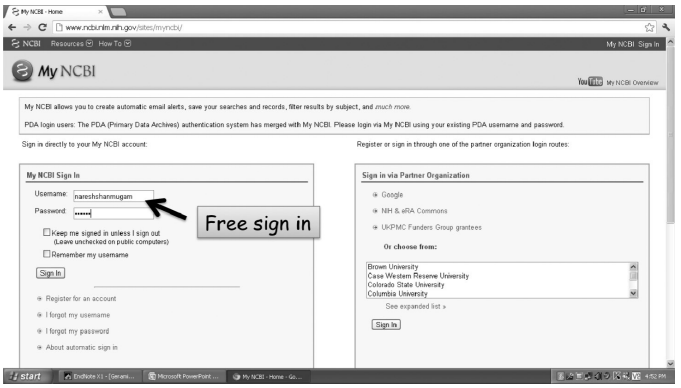

Fig.4a.

Once registered, it provides personal log-in facility which enables to save the search items which could be retrieved latter. It also enables to build search data base (Fig.4b) on different topics so that it becomes handy while writing up a paper.

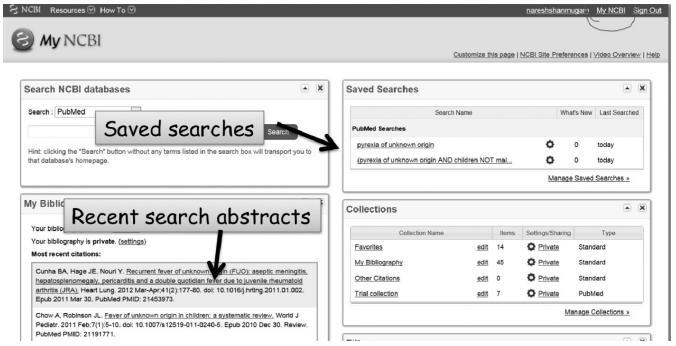

Fig.4b.

The "My NCBI" area can be accessed from any computer worldwide and so its easy for researches to store and retrieve any where.

Conclusion

PubMed is a user-friendly and free Internet facility which has made medical database within easy reach. The above-mentioned article is only a brief over view about PubMed literature search. For further reading and web-based tutorials please access the following web link http://www.ncbi.nlm.nih.gov/pubmed.

Points to Remember

- ***Unless right question is asked, web search will only yield incomplete search results.***
- ***Need to understand Boolean logic operators and use them appropriately during literature search.***
- ***While writing paper/ thesis it is essential to quote exactly how the search was performed so that it could be reproduced by a different operator.***

For further reading

1. http://www.ncbi.nlm.nih.gov/pubmed.

GENERAL ARTICLES

MEDICO LEGAL APPROACH TOWARDS VICTIMS OF SEXUAL OFFENCE

***Garudadhri GV**

Abstract: *Sexual offences against children are increasing enormously in recent years. A pediatrician/ medical practitioner would often come across such cases. It may be cumbersome for the practitioner to deal with such cases, as there may be confusions regarding medico-legal management, right from informing the police and testifying as expert witness in the Court of Law. For this, basic knowledge about the sexual offences and its approach is essential. A simple approach is given when one encounters a child victim of sexual offence.*

Keywords: *Sexual offences, Medical approach.*

India, a nation of a billion has the tradition of respecting women and caring for children since ages. Indian constitution also provides to make special provisions for them. But in recent years, the sexual offences against children are increasing enormously. When a pediatrician comes across such situation, it may be difficult for him to deal with such cases, as there may be uncertainities regarding medico-legal management, right from informing the police and testifying as expert witness in the court of law. For this, basic knowledge about the sexual offences and its examination procedure is essential. Here one such simple approach to the victim of sexual offence of child is presented.

As the laws against sexual offences are quite different in children and adults, the sexual offences in children will be considered first before going to examination approach.

Classification of offences[1]

A. Penetrative sexual assault: It includes the following acts of commission by any person on a child:

a) Penetrating the penis or any object or a part of the body into the vagina, anus or urethra. (In case of penis, penetration even into the mouth).

b) Making the child to do penetration by penis or any object or a part of the body.

c) Manipulating any part of child to cause penetration

d) Applying mouth to penis, vagina, anus or urethra.

B. Aggravated penetrative sexual assault: It includes commission of offence mentioned in (A) by a person of having trust or confidence on or by a child, and also a person on duty, be it a public or private servant. (eg. police, member of armed force, management or staff of hospital/jail/ orphanage/place of custody/educational institution/public servant, etc.).

C. Sexual assault: Any person who does following acts with sexual intent is said to cause sexual assault:

a) Touches the vagina, penis, anus or breast of the child

b) Makes the child touch the vagina, penis, anus or breast of such person or any other person, or

c) Does any other act with sexual intent which involves physical contact without penetration

D. Aggravated sexual assault: It includes commission of offence mentioned in (C) by a person of trust and confidence, on duty be it public or private.(eg. police, member of armed forces, management or staff of hospital/ jail/orphanage/place of custody/educational institution/public servant, etc.).

E. Sexual harassment: It includes following acts with the child by any person with sexual intent:

a) Uttering any word or making any sound or gesture or exhibiting any object or part of body with the intention that such word or sound shall be heard, or such gesture or object or part of body shall be seen by the child.

b) Making the child exhibit his body or any part of his body, such that, it is seen by such person or any other person.

c) Showing any object to a child in any form or media for pornographic purposes.

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d) Repeatedly or constantly following or watching or contacting a child either directly or through any means.

e) Threatening to use, in any form of media, a real or fabricated depiction through electronic, film or digital or any other mode, of any part of the body of the child or the involvement of the child in a sexual act.

The punishment for all the offences varies and includes imprisonment with or without fine and is shown in Table I.

Pediatricians often come across cases relating to A, B, C & D of above classification. The aggravated offence, i.e., B & D often has to be determined by the investigating officer. But, any evidence obtained by the pediatrician while taking history or doing examination should be noted and if possible preserved.

Table I. Punishment for sexual offences

Offence	Imprisonment	
	Not less than	Up to
A	7 yrs	Life
B	10 yrs	Life
C	3 yrs	5 yrs
D	5 yrs	7 yrs
E	-	3 yrs

Medical approach

As per the existing law, the pediatrician or the medical practitioner whenever, he/she comes across any case of child who is a victim of sexual offence, has to do medical examination without waiting for FIR or a registered complaint in accordance with appropriate procedural section of the criminal code. A physician is a person professed in art of healing and relieving pain. When a victim comes, the practitioner has to calm down the victim, be empathetic and take relevant history, conduct appropriate medical examination and preserve necessary evidence. If victim is a girl, medical examination shall be conducted by a woman registered medical practitioner. Medical examination shall be conducted in the presence of either parent or any person in whom the child reposes trust or confidence. In case the parent or any other person is not present, it shall be conducted in the presence of a woman nominated by the Head of Medical Institution. It is to be remembered that, provisions of sections of any offence will not apply in case of medical examination or treatment of a child undertaken with the consent of parents/ guardian. In addition to history and examination findings, following things should be noted as per Sec 164A CrPC-[2]

a) Name and address of the victim.

b) Name and address of the person who has brought the child.

c) Consent of the victim: Here, for medical examination of child above 12 years, consent of the child should be taken. And for medical examination of child below 12 years and/or if the child is with mental illness, consent of the parent or guardian or by the person on whom child reposes trust and confidence should be taken.

d) Date and time of commencement of examination.

e) General mental condition of the child.

f) Any injuries over the body- be it over the genitalia and/or over rest of the body.

g) In all cases, sample for the DNA should be collected.

h) In opinion, the grounds on which such an opinion is obtained should be mentioned.

i) Date and time of completion of examination.

j) In addition, any other relevant material details may be noted / collected.

Information to police

As any pediatrician or medical practitioner comes across such a case, he has to report to either special juvenile police unit or local police. The police will ascribe an entry number for every case. Then they will record the information in writing, read it over to the informant and enter it in a book to be kept by them. Failure to report such case is punishable with imprisonment for up to 6 months and/or fine as per "Protection of Children from Sexual Offences Act, 2012".

Expert may be appointed who is a person trained in mental health, medicine and child development, required to facilitate communication with a child whose ability to communicate has been affected by trauma, disability or any other vulnerability. He may also appoint a "Person familiar with the manner of communication of the child" or a "Special educator" who is a person trained in communication with children with special needs in a way that addresses the child's individual differences and needs, which include challenges with learning and communication, emotional and behavioral disorders, physical disabilities, and developmental disorders.[3]

The registered medical practitioner rendering emergency medical care shall attend to the needs of the child, including:

1. Treatment for injuries including genital injuries, if any.
2. Treatment for exposure to sexually transmitted diseases (STDs) including prophylaxis for identified STDs.
3. Treatment for exposure to Human Immunodeficiency Virus (HIV), including prophylaxis for HIV after necessary consultation with infectious disease experts.
4. Possible pregnancy and emergency contraceptives should be discussed with the pubertal child and her parent or any other person in whom the child has trust and confidence.
5. Wherever necessary, a referral or consultation for mental or psychological health or other counseling should be made.

Any forensic evidence collected in the course of rendering emergency medical care must be packed, labelled, sealed and handed over to the investigating officer for scientific analysis or examination by forensic science laboratory or any other laboratory like pathology, biochemistry, etc. respectively.

Financial assistance for rehabilitation or relief

The special court, in appropriate cases, on its own or on an application filed by or on behalf of the child, passes an order for interim compensation to meet the immediate needs of the child for relief or rehabilitation at any stage after registration of the first information report (FIR). Such interim compensation paid to the child may be used for the management of victims.[3,4]

Conclusion

The knowledge for pediatricians about medico-legal approach towards victims of sexual offences would help them to manage without much anxiety. This could provide appropriate therapeutic, legal and financial aid or relief for the victim.

Points to Remember

- ***Medical examination should be carried out as per the laid down procedure, necessary evidence has to be preserved and important details should be documented.***
- ***The doctor coming across such a case, has to report to the special juvenile police unit or local police.***
- ***Necessary treatment and counselling has to be provided taking the help of other specialists.***

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CLIPPINGS

Continuous support for women during childbirth

Historically, women have been attended and supported by other women during labour. However, in hospitals worldwide, continuous support during labour has become the exception rather than the routine.

Objectives: To assess the effects of continuous, one-to-one intrapartum support compared with usual care. Secondary: to determine whether the effects of continuous support are influenced by: (1) routine practices and policies; (2) the provider's relationship to the hospital and to the woman; and (3) timing of onset.

Selection criteria: All published and unpublished randomised controlled trials comparing continuous support during labour with usual care.

Authors' conclusions: Continuous support during labour has clinically meaningful benefits for women and infants and no known harm. All women should have support throughout labour and birth.

Hodnett ED, Gates S, Hofmeyr G, Sakala C. Continuous support for women during childbirth. Cochrane Database of Systematic Reviews 2013, Issue 7. Art. No.: CD003766. DOI: 10.1002/14651858.CD003766.pub5

GENERAL ARTICLES

HOW TO CARE FOR LOW BIRTH WEIGHT BABY AT HOME?

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Abstract: *Care of the low birth weight baby at home is a challenge. With proper training and supervision, low birth weight newborn care can be done well in home settings leading to improvement in survival and health seeking behavior. Fundamentals of such a care include early recognition, prevention and treatment of common neonatal problems. The components of home based care are health education, provision of essential newborn care - breast-feeding, thermal care, hygiene, monitoring for any infection, early recognition of illness, provision of emergency care and early referral. Home based care is complementary to facility based care, a must in "chain of survival" and a continuum of care from home to hospital .*

Keywords: *Low birth weight, Home based care, Essential newborn care*

Low birth weight (birth weight <2500 g) is indisputably a very important indirect cause of death in neonates the world over. LBW babies are usually born in poor families and remain the most vulnerable, unable to reach, afford or seek hospital based care. If they cannot reach health services, the health services must reach out to them. Nearly all such basic essential newborn care can be provided safely, effectively and at low cost at household level by trained health workers with a modest training duration and ensuring high program coverage.[1,2] Babies < 32 weeks or < 1500 grams are not included as they need facility based care. The article describes the components of LBW care at home with special emphasis on practical issues.

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Components of LBW care at home[3,4,5]

1. Thermal care

Thermoregulation is a fundamental right and all newborns need adequate warmth for growth. LBW babies are vulnerable to temperature fluctuations. The assessment for thermal well being is made by observing the color of the sole and touching with dorsum of hand the abdomen and feet. In a baby with normal temperature both abdomen and feet are warm to touch. When feet are cold and abdomen is warm, it indicates that the baby is in cold stress. In hypothermia, both feet and abdomen are cold to touch. The health provider should be aware of additional efforts needed to keep baby warm (Table.I).

Table I. Good practices for thermal care

- Postpone baby bath till cord falls.
- Keep the newborn close to the mother in a clean, dry and warm room.
- Dress the baby with adequate clothing (1-2 layers in summer and 3-4 layers in winter).
- Always cover the head of the baby with a cap/cloth.
- Provide skin-to skin contact, as long as possible, day and night, by placing the baby on the mother's chest, between the breasts.
- Cover the baby and the mother with additional blanket or shawl in cold weather.
- Use gloves and socks as and when required.
- Warm the room with a room heater or use a 200 W bulb placed at a distance of 50 cm, as needed.
- Prevent air currents in the room.

Rooming in: The mother and the baby are nursed together. This helps in bonding and initiation of breastfeeding. Separation of baby from the mother even for a couple of hours has a significant adverse impact on successful breastfeeding.

Kangaroo Care: The naked baby with only a nappy and a cap is placed between mother's breasts in direct skin-to-skin contact, in an upright position with the head turned to

one side and in slightly extended position, for as long as possible. This results in increased breastfeeding rate as well as increased duration of breastfeeding, effective thermal control with a reduced risk of hypothermia and better weight gain pattern. Such care can be provided by mother, father or any family member and extended as long as mother is comfortable.

If the baby appears cold, re-warming is done by skin-to-skin contact, covering and wrapping the baby, taking efforts to raise room temperature, providing warmth by a heater and offering additional breastfeed. Use of hot water bottle for (re) warming the baby is avoided. If the baby appears warm, the baby is unclothed, efforts to lower the room temperature are taken (open the windows, turn on the fan), the baby kept away from sources of heat, direct sunlight and breast feeding is ensured. Both hypothermia and hyperthermia can be signs of sepsis and one should seek evaluation at the earliest.

2. Breast feeding

LBW babies need to be given adequate feeds frequently to prevent hypoglycemia and to ensure adequate growth. The preferred choice for feeding LBW babies is mother's milk due to its innumerable advantages. Every effort should be made to advocate, support and promote breast feeding every 2-4 hours. Adequacy of feeding is judged by urine color and frequency, baby behavior, weight gain and maternal perception. All mothers should be made aware about proper positioning and attachment for successful breastfeeding. Expression of milk should be taught to mothers where baby cannot breast feed. If baby is not sucking well consider alternative methods of feeding using expressed breast milk directly into baby mouth, using cup/spoon or a syringe feeding (Table.II).

Table II. Good practices for breast feeding

- Assist, supervise, start breastfeeding within the first one hour of delivery.
- Ensure colostrum feeds
- Give breastfeeding whenever the baby demands, day and night.
- Give exclusive breastfeeding for six months.
- Do not give any liquid (honey, sugar water, jaggery, ghutti, gripe water, etc) or feed along with or after breastfeeding.
- Continue breastfeeding even if the mother or the baby is ill.

3. Hygiene and prevention of infection

Many of the infections and diseases can be prevented by following some simple clean practices.

Cleans: Clean hands, clean bed, clean room can prevent many infections in the baby and mother. Hand washing with soap is one of the most effective ways of preventing infection. Mother should wash hands before breastfeeding, cooking food, eating, after cleaning the urine/stool of the baby or changing nappies and after using toilet. The baby's room should be kept clean. Ensure clean clothes, blanket/ sheets for the baby and mother. All family members must follow clean practices.

Bathing: Routine bathing should be avoided in view of risk of hypothermia. Minimize exposure during baths. The infant can be sponged, as required. Infant can be bathed once every few days. There should not be an attempt to remove vernix from the body by any means, as it can result in trauma to skin and increase chance of infections.

Eye care: Eyes of the infant must be cleaned with a clean swab soaked in normal saline or sterile water. Cleaning is from inner to outer canthus using a separate swab for each eye. There is no role for routine antibiotic prophylaxis for prevention of ophthalmia neonatorum.

Cord care: The umbilical cord must be kept open and dry with no local applications.

Nail care: Keep nails clean and trimmed regularly of both-mother and baby.

Oil massage: Oil massage is a low cost traditional practice well ingrained in Indian culture. However, there is paucity of data as to what oil should be used for this purpose

4. Identifying unwell baby

LBW babies are at increased risk of becoming sick. The following danger signs must be explained to the mothers: i) difficulty in feeding ii) convulsions iii) lethargy (movement only when stimulated) iv) fast breathing (respiratory rate of >=60)/mt), severe chest in-drawing vi) temperature of 37.5^{o} degrees C or more or below 35.5^{o}. The mother should be made aware to contact the nearest health care provider for the sickness and guidance.

Evaluation for jaundice: All LBW infants must be examined for the development and severity of jaundice during few days of life. Visual assessment in daylight is the preferred method. Babies with visible jaundice in first 3 days should be systematically evaluated.

5.Care of the sick newborn

A baby with pustules (>10), discharge from umbilicus needs local cleaning, application of antiseptics and oral antibiotic. Presence of purulent discharge from the eye is treated with cotrimoxazole or tetracycline eye drops. Any danger sign should be referred to a health facility immediately. Studies [2,4] show antibiotics (co-trimoxazole or gentamicin) can be given at home by appropriately trained and well-supervised community health workers for management of sepsis and pneumonia when health facility is not reachable. Urgent arrangements for referral should be made. If the baby is able to feed, then breast feeding should be continued on the way. Skin to skin contact helps keep the baby warm. If this is not possible, the baby is wrapped and kept close with the mother.

6.Nutritional supplements

All LBW infants (preterms) should receive drops of a multivitamin preparation, and iron every day. Iron is started in a dose of 2 mg /kg/ day from 4 weeks of age. Multivitamin and iron are continued at least for one year of age.

7.Stimulating development

If the baby is provided ways to see, hear, move arms and legs freely and the baby is touched, gently stroked and held, it helps in baby's development. Keeping the small baby in skin-to-skin contact is particularly useful to stimulate the baby. If the mother and other family members look into the baby's eyes and talk to the baby, it also helps in the baby's development**.** They should try to do this as often as possible.

8.Traditional practices

A variety of traditional practices are common in India. Some of these can be beneficial such as oil massage, inconsequential such as putting black mark on forehead. However some practices such as applying kajal/surma in eye, instillation of oil in ear, instillation of boric acid in nostrils or applying substances such as cow dung on cord should be actively discouraged.

9.Follow up

LBW babies should be followed up weekly for weighing, assessment of feeding, early diagnosis and management of illnesses, health education of parent and general health until they have reached 2.5 kilogram. All LBW babies should receive vaccines as per schedule irrespective of weight. Mother should be provided ample opportunity to ask questions and clarify all her doubts.

10.Maternal counseling

Mother needs to eat extra for herself and to make breast milk for the baby also. Healthy mother can produce better quality and higher quantity of milk. Mother must have all types of food with no restrictions. Good diet and regular intake of IFA tablets helps in adequate breast milk and growth of the baby. Mother should bathe regularly and use clean clothes to prevent infection. She should not do heavy work till six weeks after delivery. The fastest possible modes of transport to hospital (either planned or in emergency) must be explored in advance. Support from some relatives/ friends/neighbours after the delivery may be very useful. If the mother is seriously ill, seek help from the doctor and follow advice. Birth spacing choices should be advised to avoid unwanted or frequent pregnancies.

Role of home based newborn care (HBNC)[5,6]

HBNC is a complementary strategy to facility based care provided through skilled health workers (ASHA or ANM/VHW) where access to health facility is limited in settings with high neonatal mortality. All newborns are seen on day 1, day 2, day 3, day 7 and day 14. Studies show that the home visits lead to improved coverage of key newborn care practices such as early initiation of breastfeeding, exclusive breastfeeding, skin-to-skin contact, delayed bathing, attention to hygiene, such as hand washing with soap and water, and clean umbilical cord care. This strategy has shown positive results in high mortality settings by reducing deaths by 30 to 61%.[7]

Points to Remember

- ***Nearly all essential newborn care can be provided safely, effectively and at low cost at household level.***
- ***Fundamentals of LBW care include recognition, prevention and treatment of common problems.***
- ***The basic components of LBW care at home are a) Increased attention to keeping the newborn warm, including skin-to-skin contact with the mother; b) assistance with initiation of breastfeeding, expressing breast milk if the baby is not strong enough to suckle, c) extra attention to hygiene, especially hand washing; d) extra attention to danger signs and the need for early care seeking and referral; and e) periodic home visits for wellbeing surveillance and monitoring growth.***
- ***Providing basic care to LBW newborn at home has been identified as a critical intervention that helps in preventing newborn deaths.***

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CLIPPINGS

Revised Diagnostic Criteria for Pseudotumor Cerebri Syndrome in Adults and Children

This article offers criteria for diagnosing pseudotumor cerebri syndrome in patients ranging from ages 3 to 60 years old, revised from criteria created in 2002. The authors advocate for using the inclusive diagnostic term "pseudotumor cerebri syndrome" rather than "benign intracranial hypertension" or "idiopathic intracranial hypertension," as this vision-threatening condition is by no means benign and, in some patients, the precipitating cause is known rather than idiopathic. The syndrome can be subdivided into primary (i.e., idiopathic) and secondary forms. Secondary causes range from medication-induced to cerebral venous sinus thrombosis.

The authors offer diagnostic criteria for three clinical scenarios:

- Pseudotumor cerebri syndrome with papilledema: Neurological examination is normal except for cranial nerve abnormalities; neuroimaging is normal except for findings suggestive of high pressure; cerebrospinal fluid (CSF) composition is normal; and the opening pressure of a properly performed lumbar puncture is elevated ($\geq$250mm CSF in adults and unsedated children and $\geq$280mm CSF in sedated children).
- Pseudotumor cerebri syndrome without papilledema: Criteria are the same as above, along with unilateral or bilateral sixth nerve palsies.
- Suggested pseudotumor cerebri syndrome: Criteria for this scenario are fulfilled if there is no papilledema or sixth nerve palsy, but the other criteria are met and neuroimaging findings suggest high pressure.

Friedman DI et al. Neurology 2013 Aug 21.

ERRATUM

We regret that clipping on page 205 in Vol.15 (3) Jul-Sep 2013 have been erroneously repeated on page 244 also.

DRUG PROFILE

USE OF ANTI-INFLAMMATORY DRUGS

Jeeson C Unni

Abstract: *Nonsteroidal anti-inflammatory drugs (NSAIDs) possess antipyretic, analgesic and anti-inflammatory effects. They are frequently used in children and have numerous therapeutic indications, the most common ones being fever, postoperative pain and inflammatory disorders, such as juvenile idiopathic arthritis (JIA) and Kawasaki disease. This article deliberates on the development of NSAIDs over the years, their indications in children, adverse effects and guidelines for choosing one NSAID over another.*

Key words:*Anti inflammatory drugs,NSAIDS,COX-1 inhibitor, COX-2 inhibitor*

Anti-inflammatory drugs were discovered by accident while analyzing content of plants and their extracts that were being used for the relief of pain, fever and inflammation. The leaves and bark of the willow tree, used as a remedy for aches and fever [1] contains salicin which gets metabolized into salicylic acid in the human body.[2] The non-steroidal anti-inflammatory drugs (NSAIDS) were developed from organic acids – the pre-prostaglandin period before the 1970's and thereafter when effects on prostaglandin production formed part of the screening in the drug-discovery process aspirin, indomethacin and phenylbutazone and were chosen from the anti-inflammatory drugs in animal studies because they produced the least gastro-intestinal (GI) side effects. In the 1990's the two cyclo-oxygenase (COX) enzyme systems controlling the production of prostanoids [prostaglandins (PGs) and thromboxane (TxA2)] were discovered that changed the approach to dealing with inflammatory processes; COX-1 produced PGs and TxA2 that regulate gastrointestinal, renal, vascular and other physiological functions and COX-2 regulated production of PGs involved in inflammation, pain and fever. Drugs that selectively controlled COX-2 and spared COX-1 - that was responsible for the adverse effects – were then sought. Research resulted in discovery of the highly selective COX-2 inhibitors the coxibs (celecoxib and rofecoxib) - which had negligible GI side effects. Alarm bells rang when in late 2004 rofecoxib was withdrawn worldwide because of serious cardiovascular side-effects causing concerns and initiating research into the CVS and CNS (stroke) effects of the coxibs. The importance of COX isoforms in the pathogenesis of non-arthritic or non-pain states such as cancer and other neurodegenerative diseases and the applications of NSAIDs and the coxibs in the prevention and treatment of these conditions as well as aspirin and other analogues in the prevention of thrombo-embolic diseases now constitute one of the major therapeutic developments of this century. Though steroids are the most potent anti-inflammatory agents in children and adults alike, this article will focus on use of NSAIDs in children.

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Choice of anti-inflammatory drugs in children

There is very little difference in the anti-inflammatory effect of various NSAIDs but response to the drugs and their tolerance varies from child to child. Most children respond to any NSAID and some who do not respond to one would respond to another. Though analgesic effect starts almost immediately after the first dose and full effect occurs within a week, anti-inflammatory effect may not manifest/ not be clinically demonstrable even after 3 weeks (effect in JIA takes 4-12 weeks).[3] If adequate effect is not seen within the duration mentioned another NSAID needs to be started. Further, it is not possible to predict which child would respond to which NSAID. The child friendly formulations of various NSAIDs need to be used.

NSAIDs reduce the production of prostaglandins by inhibiting the enzyme COX-2. Since GI side-effects are rare in children taking NSAIDs for short periods, highly selective COX-2 inhibitors are not mandatory and their role in children is undetermined. Ibuprofen and naproxen are propionic acid derivatives used in children. Ibuprofen has anti-inflammatory, analgesic and antipyretic properties. It has fewer side-effects than other NSAIDs but its anti-inflammatory properties are weak. Most studies highlight its antipyretic and analgesic effect. Naproxen combines good efficacy with a low incidence of side-effects.[4]

Diclofenac, indomethacin, mefenamic acid and piroxicam have properties similar to those of propionic acid derivatives: Diclofenac is similar in efficacy to naproxen.[5] Indomethacin has an action equal to or superior to that of naproxen, but with a high incidence of side-effects including headache, dizziness and gastro-intestinal disturbances. It is rarely used in children and should be reserved for situations when other NSAIDs have been unsuccessful. Mefenamic acid has minor anti-inflammatory properties. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment and must be used with extreme caution in children less than 14 years of age.[6]

Piroxicam is as effective as naproxen and its half life of 30 hours in children permits a once daily dosing of this drug.[7] GI side effects and skin reactions limit its use. Meloxicam and Etoricoxib are the only 2 selective inhibitors of cyclo-oxygenase-2 that have been licensed for use in adolescents[8] when they are intolerant to other NSAIDS. They are not to be used in children.

Aspirin has limited use in children because it has been associated with Reye's syndrome. Aspirin-containing preparations should not be given to children and adolescents under 16 years, unless specifically indicated, such as for Kawasaki syndrome, rheumatic fever, Henoch Schonlein purpura, prophylaxis of clot formation after cardiac surgery, or for prophylaxis of stroke in children at high risk. If aspirin causes dyspepsia, or if the child is at a high risk of gastro-intestinal bleeding, a proton pump inhibitor or a H_2-receptor antagonist can be added. Dosage of individual anti-inflammatory drugs is given in Table I.

Table.I. Dosage schedule of anti-inflammatory drugs

Drug	Dosage
Aspirin	Fever: 10-15 mg/kg/dose 4-6 times daily (maximum 4gm/day). Antiinflammatory, analgesic dose: 80-100 mg/kg/day. Kawasaki disease: 25mg/kg/dose 4 times daily for 14 days followed by 5mg/kg once daily for 6-8 weeks. If no evidence of coronary lesions - discontinue
Ibuprofen	Pain/fever - 5-10mg/kg/dose 3-4 times daily (max 20mg/kg/day upto 2.4gm/day). Juvenile idiopathic arthritis and other rheumatic disorders - 30-50mg/kg/24hr in 3-4 divided doses - higher doses in systemic JIA. Closure of PDA in newborn (IV preparation) - Dose as ibuprofen base and given at 24 hr interval - First dose 10mg/ kg, 2nd and 3rd dose 5mg/kg. If PDA does not close 48 hr after last dose or reopens, a 2nd course of 3 doses may be given.
Naproxen	Oral 10-20mg/kg in 2 divided doses (max 1gm/ day). In severe cases upto 15mg/kg may be used for a few weeks.
Diclofenac	Oral/rectal > 6 months age 300 microgm - 1mg/kg/ dose 3 times daily (max 150 mg/day). IM/IV > 6 months age 300 microgm - 1mg/kg/dose 1-2 times daily (max 150 mg/day for max 2 days). Topical 2-18 yr small amt 3-4 times daily.
Indomethacin	Pain/ inflammation in rheumatic disease and nephrogenic diabetes insipidus - Oral 1- 2mg/kg in 2 divided doses. Closure of PDA in newborn - IV over 20-30min 100-200mcg/kg as a single dose followed by 2 doses 100mcg/kg at 24hr intervals; if residual patency is noted, 100mcg/kg x 3 more doses 24hrly. Monitor for adequate urine output.
Piroxicam	Oral < 15kg 5mg, 16-25kg 10mg, 26-45kg 15mg and >46kg 20mg once daily.
Meloxicam	Oral/rectal 12-18 yr < 50kg 7.5mg and > 50kg 15mg once daily. In renal failure, dose of 7.5mg not to be exceeded.
Etoricoxib	Oral – Osteoarthritis – 16-18yrs – 30mg OD, increased if indicated to 60mg; JIA and ankylosing spondylitis - 16-18yrs – 90mg OD; Acute gout - 16-18yrs – 120mg OD for a max of 8 days.

Cautions and contra-indications

NSAIDs should be used with caution in children with a history of hypersensitivity - attacks of asthma, angioedema, urticaria or rhinitis - to any NSAID. NSAIDs should also be used with caution in coagulation defects.[9] Acute pain in hemophiliacs may be treated with paracetamol and chronic pain with either paracetamol or NSAID.[10] Caution may also be required in children with allergic disorders like bronchial asthma and atopic dermatitis.[11,12]

In children with cardiac impairment, caution is required since NSAIDs may impair renal function. All NSAIDs are contra-indicated in severe heart failure.[13] Diclofenac and the selective inhibitor of cyclo-oxygenase-2, etoricoxib, are contra-indicated in cerebrovascular disease, peripheral arterial disease and mild to severe heart failure and hypertension.[14]

All NSAIDs (including COX-2 selective inhibitors) are contra-indicated in children with active gastro-intestinal ulceration or bleeding.[13] Piroxicam and ketorolac are contra-indicated in children with a history of gastro-intestinal bleeding, ulceration or perforation.[7] Other non-selective NSAIDs are contra-indicated in similar situations and if there is a past history of GI bleed attributed to any NSAID treatment. NSAIDs should also be used with caution in Crohn's disease or ulcerative colitis, as these conditions may be exacerbated[15] though the mechanism of this effect remains inadequately defined.

Hepatic impairment: Children have a large reservoir of hepatic function and hence drug induced hepatotoxity is rare in this age group. However, since hepatotoxicity is considered a class characteristic of NSAIDs they should be used with caution in children with hepatic impairment.[16,17] Hepatic failure predisposes the child to GI bleeds and fluid retention and hence NSAIDs are an absolute contraindication. Most NSAID reactions are hepatocellular and idiosyncratic.

Renal impairment: NSAID users had a 3-fold greater risk for developing a first-ever diagnosis of clinical ARF compared with non-NSAID users in the general population.[18] These drugs should therefore be avoided if possible or used with caution in children with renal impairment.[19] The smallest effective dose for shortest possible duration with regular monitoring of renal function should be the dictum as sodium and water retention could further hamper renal function; deterioration in renal function has also been reported after topical use.[20]

Pregnancy: NSAIDs may be avoided during pregnancy or given only if the potential benefit of the drug outweighs risk to the pregnancy and/or the fetus. Though there are studies that suggest an association of the risk of closure of fetal ductus arteriosus in utero and possibly persistent pulmonary hypertension of the newborn in offspring of mothers who have taken NSAIDs in the 3rd trimester[21,22], there are other prospective studies that do not confirm this association.[23,24] Reports of delay in the onset and increase in the duration of labour have also raised concerns regarding use in pregnancy.

Breast-feeding: NSAIDs should be used with caution during breast-feeding.

Side-effects

The side-effects of NSAIDs vary in severity and frequency. Gastro-intestinal disturbances including discomfort, nausea, diarrhea, and occasionally bleeding and ulceration may occur.

Conclusion

NSAIDs are effective in reducing fever, pain and inflammation in children, with a good tolerance profile. Pharmacokinetic studies are needed to characterise the disposition of NSAIDs in very young infants. More studies in children on pharmacokinetics, side-effects and efficacy of specific COX-2 inhibitors are required before they can be recommended with confidence in this age group.

Points to Remember

- ***There is very little difference in the anti-inflammatory effect of various NSAIDs.***
- ***Anti-inflammatory effect may not manifest/ not be clinically demonstrable even after 3 weeks.***
- ***Ibuprofen combines anti-inflammatory, analgesic, and antipyretic properties. It has fewer side-effects than other NSAIDs but its anti-inflammatory properties are weak.***
- ***Naproxen combines good efficacy with a low incidence of side-effects.***
- ***Diclofenac is similar in efficacy to naproxen.***
- ***Indomethacin has an action equal to or superior to that of naproxen, but with a high incidence of side-effects.***
- ***Meloxicam and Etoricoxib are the only 2 selective inhibitors of cyclo-oxygenase-2 that have been licensed for use in adolescents.***

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DERMATOLOGY

TOPICAL STEROIDS

*Vijayabhaskar C

Abstract: *Topical steroid is the most commonly prescribed medicine in dermatological conditions by virtue of its anti inflammatory and anti proliferative properties. Sometimes topical steroids are abused by practitioners and patients but most often they are underutilized. Potent steroids should not be used in children as the larger surface area results in increased absorption. Appropriate potency of steroids in appropriate concentration should be used in the appropriate areas of the body to enhance the efficacy and to minimize the adverse effects. Quantity of steroids that is to be applied is measured by finger tip units. Frequency of use and duration of application of topical steroids play a major role in determination of the adverse effects. FDA guidelines should be followed in using the topical steroids so that maximum benefit could be achieved.*

Key words: *Topical steroids, Potency, Finger tip unit.*

Topical steroid is the most commonly prescribed medicine in many dermatological conditions. It has got anti-inflammatory and anti- proliferative properties. Appropriate usage of topical steroids helps in the treatment of various dermatological conditions. On one hand there is apprehension in the usage of topical steroids by the practitioner and the parents and on the other hand they tend to be abused by both the physicians and the parents. Many a times this drug is reused by the parents for the same condition when it recurs or is used for similar looking new conditions without consultation. Topical steroids are to be carefully used in infants and children in view of increased skin permeability and larger surface area.

Classification of topical steroids (Table I)

The potency of the topical steroids is measured by the vasoconstrictive properties, which means the ability of the steroids to produce cutaneous vasoconstriction in a normal individual.

The potency of the molecule depends upon the structure of the corticosteroid, vehicle, concentration of the molecule and the nature of the skin on which it is used.[1]

Structure

As seen in the Table I, the structure differs in each class of the steroid. Fluorinated and halogenated corticosteroids are more potent and equally have more side effects than the non halogenated corticosteroids. Hydrocortisone butyrate and hydrocortisone valerate are mid potent steroids when compared to hydrocortisone acetate which is least potent steroid.

Vehicle

Any topical preparation will have an ingredient molecule and a vehicle to carry the molecule. They are available as ointments, lotions, creams, gels etc. 'Ointments' are to be used for dry and thick lesions as they have occlusive effect and result in good hydration. Potency is higher in the ointment form than for the cream form of the same molecule. It is cosmetically not acceptable and also not recommended over the hairy region and intertrigenous areas. 'Creams' disappear when applied to the skin and are cosmetically acceptable. They are used more on the exudative lesions and in the intertrigenous areas. They are less potent when compared to the same molecule in the ointment form. Sometimes preservatives added in the creams may cause burning and stinging sensation.[2] Lotions and gels are not greasy and disappear immediately on application but have occlusive property. They are preferred in the hairy region and over the intertrigenous areas.

Concentration of the molecule

The potency varies at different concentration of the same molecule. The higher the percentage the higher the potency.

Nature of the skin

High potent steroids are to be avoided in areas like face, groin, axillae and areas of occlusion. In diseased skin, potent steroids and ointment based steroids are better

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avoided for the risk of absorption. In palms and soles which have thick skin potent topical steroids could be used.

Finger tip unit

A finger tip unit (FTU)[3] is the amount of ointment expressed from a tube of 0.5cm diameter nozzle applied from the distal skin crease to the tip of the index finger. One FTU is equivalent to 0.5gram. One finger tip unit is used to treat an area of skin of a child equivalent to twice the size of the flat of an adult hand with the fingers together. It would be better if the practitioner could calculate the fingertip units required and explain to the parents in a way they understand like peanut size or pepper size. Requirement of topical steroids in FTU in different areas is given in Table II.[4]

Frequency of use

Most of the drugs are used as twice daily application. Mometasone and Methylprednisolone can be used as once a day application. Many studies have shown that even fluticasone could be used as once a day application and hence side effects could be minimized.

Duration

Most of topical preparations are advised for 2 weeks and a few steroids like desonide, fluticasone and hydrocortisone could be used for 4 weeks without any adverse effects. In case the skin lesions are not getting under control, one has to rethink about the diagnosis or the potency of the steroids has to be increased.

Table I. Classification of topical steroids based on potency

Classification and potency	Molecule	
Class 1 Very high potency	0.05% 0.05% 0.05%	Clobetasol propionate cream/ointment Halobetasol propionate cream/ointment Betamethasone dipropionate ointment
Class 2 High potency	0.25% 0.1%	Desoximetasone cream/ointment Mometasone furoate ointment
Class 3 Medium potency	0.05% 0.05% 0.05% 0.1% 0.005%	Betamethasone dipropionate cream Fluocinonide cream Desoximetasone cream Betamethasone valerate ointment Fluticasone ointment ointment
Class 4 Medium potency	0.1% 0.1% 0.1% 0.025%	Mometasone furoate cream Triamcinolone acetonide cream/ointment Betamethasone valerate cream Fluocinolone acetonide ointment
Class 5 Lower medium potency	0.05% 0.1% 0.025% 0.1%	Fluticasone propionate cream/lotion Triamcinolone acetonide cream/lotion Fluocinolone cream Hydrocortisone butyrate cream/ointment
Class 6 Low potency	0.05% 0.1% 0.025% 0.01% 0.1%	Desonide cream Hydrocortisone butyrate lotion Triamcinolone acetonide cream/lotion Fluocinolone acetonide lotion Betamethasone valerate lotion
Class 7 Lowes potency	Hydrocortisone acetate Dexamethasone Methylprednisolone	

Withdrawal of steroids

Sometimes abrupt withdrawal of steroids may result in appearance of similar lesions. Hence once the dermatoses is under control, topical steroids may be replaced with emollients and topical steroids may be used twice a week or on weekends until complete resolution. In children with atopic dermatitis, topical steroids may be substituted with calcineurin inhibitors.

Steroid responsive dermatoses

Topical steroids are used in atopic dermatitis, seborrhoeic dermatitis, eczemas, papular urticaria, lichen planus, psoriasis, vitiligo, bullous dermatoses, alopecia areata, granuloma annulare, lupus erythematosis, etc.

FDA approval

According to FDA, topical steroids are not recommended in infants below 3 months of age. Use of desonide 0.05% and hydrocortisone butyrate 0.1% cream is approved above 3 months of age.[5] Of late, many guidelines recommend fluticasone 0.05% cream above 3 months of age. mometasone 0.1% cream/ointment and prednicarbate 0.1% cream/ointment are advised above 2 years of age. Super potent steroids like clobetasol propionate and halobetasol propionate are advised above 12 years of age.

Adverse effects

Various factors such as young age, amount of corticosteroid applied, extent of the skin disease, duration of the treatment, potency of topical steroid, occlusion and hydration of the skin[1] determine the adverse effects of topical steroids.

Local side effects

Long term use may lead to side effects such as atrophy, telangiectasia, discoloration, hair growth, acneiform eruption and secondary infection. When used near the eye for a prolonged period of time glaucoma, cataracts and susceptibility to bacterial and fungal infection could occur. Tachyphylaxis can occur with superpotent steroids. Superpotent steroids are best avoided in children . Allergic contact dermatitis to topical steroids has been reported. This may occur due to the vehicle used or the topical steroid molecule itself.[6] This can be clinically evident when corticosteroid sensitive dermatitis worsens during therapy.

Systemic side effects

Topical steroids can be absorbed through skin and could result in suppression of the hypothalamo-pituitary-adrenal axis, Cushing's syndrome and growth retardation in infants and children.[7] In infants and children, catch up growth is expected when topical steroids are discontinued except during puberty as it may cause premature epiphyseal closure before catch up growth can occur.

Tips on using topical steroids

Appropriate topical steroids which will clear the lesion in the shortest time are to be chosen. One has to be kind to the skin, but never should be a coward in choosing the appropriate topical steroid. There are conditions which

Table II. Finger tip units and amount in grams for topical steroids

Guidelines for children				
Anatomic area	**FTU required**	**Amount needed for twice daily regimen in grams**		
	3 – 6 mos of age	**1 – 2 yrs of age**	**3 – 5 yrs of age**	**6 – 10 yrs of age**
Face and neck	1/1	1.5/1.5	1.5/1.5	2/2g
Arm and hand	1/1	1.5/1.5	2/2	2.5/2.5
Leg and foot	1.5/1.5	2/2	3/3	4.5/4.5
Anterior trunk	1/1	2/2	3/3	3.5/3.5
Posterior trunk and Buttocks	1.5/1.5	3/3	3.5/3.5	5/5

Courtesy * Adapted from Long CC, Mills CM, Finaly AY. Br J Dermatol 1998;138:293-6.

involve the thick areas like palms and soles where high potent steroids may need to be used in the initial period. After a period of one week less potent steroid may be substituted. High potent topical steroid should never be withdrawn abruptly.

In areas like face, diaper areas and skin folds with thin skin, only class 5, 6 and 7 topical steroids are to be used. Over the eyelids only class 7 steroids are to be used for not more than 4 weeks of continuous therapy.

On the hair bearing areas, moderate to low potency steroids in gel or lotion forms are to be used.

In steroid responsive dermatoses with secondary infection, infection is to be treated with oral/topical antibiotics for 5 days which is to be followed by topical steroids.[8]

Hand outs about the usage of topical steroids to the parents may be given.

Conclusion

Topical steroids are good weapons to fight dermatological diseases in children, provided the right steroid is chosen in terms of appropriate potency and vehicle. Appropriate use with regard to frequency, duration and site of application will result in good therapeutic efficacy and minimal adverse effects. As far as possible, mid potent to least potent topical steroids are to be used. Counseling the parents regarding the proper use of the topical steroids with emphasis on regular follow up, advise against self medication and use of over the counter products are very important. Prescription sharing should be discouraged as it always results in adverse effects.

Points to Remember

- ***Always choose mid potent to low potent steroid to control the steroid responsive dermatoses in children.***
- ***Use the least potent steroids to control the disease.***
- ***Use a particular steroid for 2 weeks and if necessary for a period not exceeding 4 weeks.***
- ***Select a steroid which is approved for that age group.***
- ***As far as possible try to calculate the dose of topical steroid in finger tip units and explain to the parents the equivalent of it in their understandable language.***
- ***Educate the parents about good effects of topical steroids if used properly. If a steroid responsive dermatosis is infected, first control the infection with topical/ oral antibiotics and start on topical steroids.***

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NEWS AND NOTES

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RADIOLOGIST TALKS TO YOU

IMAGING THE NECK

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****Natarajan B**
****Jeya Rajiah**
****Kasivisalakshi KP**
****Balan MP**

Children often present with infective or inflammatory mass lesions in the neck; the most common being lymph nodes. Since lesions in the neck are superficial they are best evaluated with ultrasound. High frequency probes give an excellent view of salivary glands, thyroid gland, lymph nodes and neck vessels. Apart from lesions arising from these organs there are a group of congenital lesions seen in the neck like hemangiomas, lymphangiomas, dermoid cysts and branchial cysts.

Thyroxine masses are not very commonly seen in children. Newborn goitre may be due to maternal thyroxine ingestion. Diffuse enlargement of the thyroid gland with normal echo texture is often seen in adolescents due to increased hormone requirement. Hormone studies are mandatory. Thyroid nodules are rare in children but have to be immediately worked up as they have a greater tendency to harbour malignancy than nodules in adults. Papillary thyroid cancer is the commonest thyroid malignancy in children and carries a better prognosis than in adults. Biopsy is essential for diagnosis. Thyroid gland is usually resistant to bacterial infection, but rarely an abscess may form in the thyroid. It is often seen in the left lobe and is associated with persistent pyriform sinus between the thyroid capsule and the pharynx. Fig.1 shows a thyroid abscess in the left lobe seen as a hypo echoic lesion in the anterior part.

Another important condition pertaining to the thyroid is the ectopic thyroid gland. This may be seen anywhere along the line of thyroid descent from the base of the tongue to thyroid location in the midline. It is seen as an echogenic round lesion like a dermoid (Fig.2). It does not have a normal bi-lobed structure. It is essential to identify this as an ectopic thyroid and the only existing thyroid tissue so that it is not removed inadvertently. To avoid missing this diagnosis one has to look for the thyroid gland in its normal location. Fig.3 shows normal thyroid gland with two lobes linked across the midline by the isthmus. A cystic lesion along the line of descent of the thyroid gland is a thyroglossal cyst. Most of them are located at or below the hyoid bone.

The salivary glands may also enlarge with inflammation. Mumps causes parotid gland enlargement. Recurrent parotitis can cause sialectasis that may be seen as multiple small black or cystic areas. Calculi are common in the submandibular glands. These are seen as bright echogenic lesions with posterior acoustic shadowing (Fig.4). There may be associated sialadenitis causing enlarged glands. Another lesion seen in the sublingual gland is a cyst or ranula that arises from rupture of a salivary duct or acini with extravasation of saliva. It is actually a pseudocyst. If it is confined to the floor of the mouth it is called a ranula and if it escapes into the neck below the mylohyoid muscle it is called a "plunging ranula". Fig.5 shows a plunging ranula which is seen as a hypo dense cystic lesion in the neck just above the hyoid bone.

A cystic lesion seen along the anterior border of the sternomastoid muscle is a congenital branchial cyst (Fig.6). This occurs due to failure of obliteration of the second branchial cleft. Sometimes it may open to the skin or communicate internally to the pharynx through a fistula.

The sternomastoid tumor is seen as a spindle shaped enlargement of the sternomastoid muscle in the perinatal period. It is due to birth injury. Ultrasound will confirm the diagnosis. The swelling subsides later with fibrosis causing torticollis.

Cystic hygromas are seen as multiseptated cystic lesions. They can occur anywhere but are common in the neck and in the neck they are common in the posterior triangle. They can steadily increase in size over the first few months of life. MRI is done to study the extent and shows a septated cystic mass with high signal on

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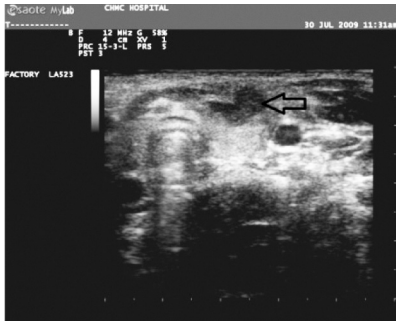

Fig.1. Abscess in left lobe of thyroid(arrow)

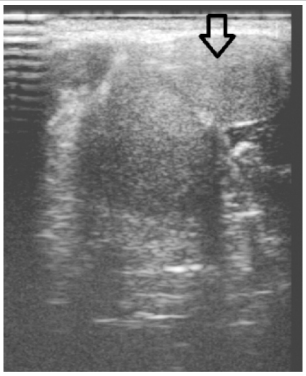

Fig.2. Echogenic dermoid (arrow)

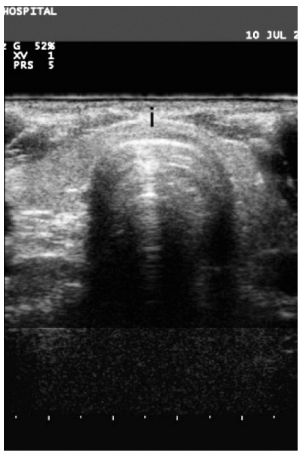

Fig.3. Normal thyroid (i is isthmus)

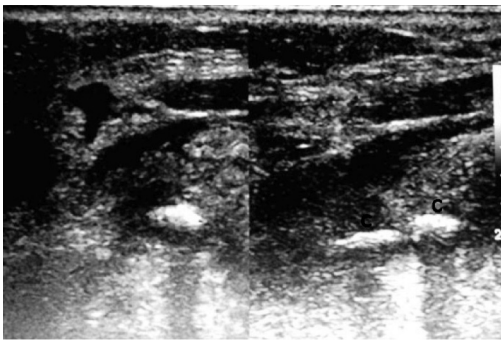

Fig.4. Salivary gland calculi(c)

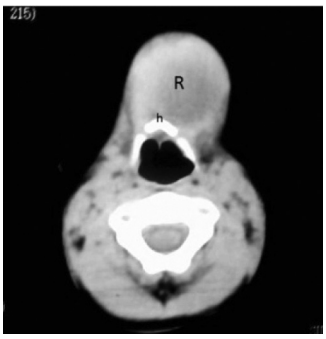

Fig.5. Plunging ranula. (h- hyoid bone)

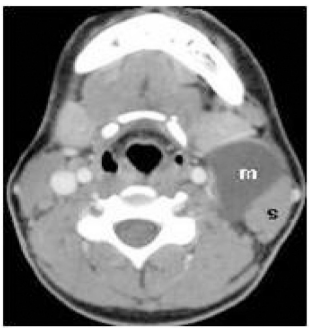

Fig.6. Branchial cyst(m). S is sternomastoid

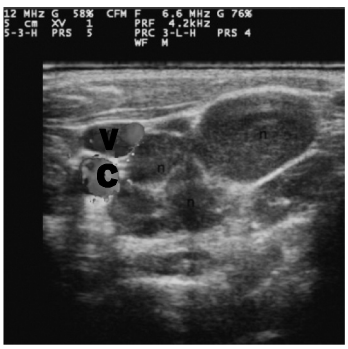

Fig.7. Lymph nodes (c- common carotid artery & v- jugular vein)

T2 images and low signal on T1 images. Hemorrhage in the lesion can cause high signal in T1 images and hemorrhage-fluid levels. Hemangiomas are well defined hypo echoic lesions with vascularity. After the initial diagnosis with ultrasound, MRI is done to delineate the extent of the lesion. Deep hemangiomas and venous malformations demonstrate intermediate signal in T1-weighted images, heterogeneous high signal on T2-weighted images and prominent enhancement. Involuting hemangiomas show focal areas of high signal intensity on T1-weighted images due to fatty replacement. Serpigenous flow voids are features of high flow lesions.

The most common lesion is lymphadenopathy and enlarged nodes are seen as multiple round lesions. Doppler will show the hilum with a small vessel entering the gland. They are commonly infective but biopsy is essential to rule out malignancy or confirm tuberculosis. Some other malignant masses that may occur in the neck like rhabdomyosarcoma and neuroblastoma are seen as solid masses and require biopsy for correct diagnosis.

CLIPPINGS

'Scared straight' and other juvenile awareness programs for preventing juvenile delinquency

'Scared Straight' and other similar programs involve organized visits to prison by juvenile delinquents or children at risk for criminal behavior. Programs are designed to deter participants from future offending through firsthand observation of prison life and interaction with adult inmates. These programs remain in use despite research questioning their effectiveness. This is an update of a 2002 review.

Objectives: To assess the effects of programs comprising organized visits to prisons by juvenile delinquents (officially adjudicated, that is, convicted by a juvenile court) or pre-delinquents (children in trouble but not officially adjudicated as delinquents), aimed at deterring them from delinquency.

Selection criteria: We included studies that tested programs involving the organized visits of delinquents or children at risk for delinquency to penal institutions such as prisons or reformatives. Studies that had overlapping samples of juvenile and young adults (for example, ages 14 to 20 years) were included. We only considered studies that assigned participants to conditions randomly or quasi-randomly (that is, by odd/even assignment to conditions). Each study had to have a no-treatment control condition and at least one outcome measure of 'post-visit' criminal behavior.

Authors' conclusions: Programs such as 'Scared Straight' increase delinquency relative to doing nothing at all to similar youths. Given these results, we cannot recommend this program as a crime prevention strategy. Agencies that permit such programs, therefore, must rigorously evaluate them, to ensure that they do not cause more harm than good to the very citizens they pledge to protect.

Petrosino A, Turpin-Petrosino C, Hollis-Peel ME, Lavenberg JG. 'Scared Straight' and other juvenile awareness programs for preventing juvenile delinquency. Cochrane Database of Systematic Reviews 2013, Issue 4. Art. No.: CD002796. DOI: 10.1002/14651858.CD002796.pub2. Assessed as up to date: June 30, 2012.

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CASE STUDY

RIGHT VENTRICULAR OUTFLOW TRACT ECTOPICS IN COUPLETS IN A 6-YEAR-OLD CHILD

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****Saminathan D**
*****Balasubramanian T**

Abstract: *Ventricular arrhythmias can occur in children with normal heart. Idiopathic monomorphic ventricular arrhythmias commonly arise from the right ventricular outflow tract. One form of such arrhythmias is the frequent ventricular premature complexes. The prognosis of outflow tract arrhythmias is generally favourable, but there is a potential to develop tachycardia induced cardiomyopathy.*

Keywords: *Cardiac arrhythmias, Ventricular premature complexes, Right ventricular outflow tract.*

Ventricular arrhythmias in structurally normal hearts can be broadly considered under 1) non life-threatening monomorphic and 2) life-threatening polymorphic rhythms. Monomorphic ventricular arrhythmias are classified on the basis of origin in the heart;[1] most common site being the outflow tract (OT).[2] 70-80% of the OT arrhythmias arise from the right ventricular outflow tract (RVOT).[3] We report a 6-year-old child who came for routine immunization and was incidentally found to have RVOT arrhythmia.

Case Report

A 6-year-old female child was brought for immunization - 2nd booster DPT/OPV. A general and system examination was done. Cardiovascular system examination revealed irregular rhythm of heart sounds. There was no history of fever, chest pain, palpitations, dyspnea on exertion or syncopial attacks in the past. There was no history of recent ingestion of drugs, cough syrups or previous hospitalization. There was no family history of sudden cardiac death.

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A 12 lead ECG was taken (Fig.1). The findings were ventricular premature contractions (VPCS) in couplets with a left bundle branch block (LBBB) morphology and inferior axis. Echo and colour doppler studies showed a structurally normal heart with normal left ventricular function.

With the above clinical and ECG findings, a diagnosis of idiopathic RVOT ectopics in couplets was considered. The child was started on beta blockers - oral propranolol 10mg twice daily. A repeat 12 lead ECG was taken after 3 weeks (Fig.2). The rhythm had restored to sinus rhythm. The child was advised to continue oral propranolol and is on regular follow up.

Discussion

Idiopathic ventricular OT arrhythmias manifest clinically in 3 forms. According to index clinical arrhythmia on presentation, the 3 forms are sustained ventricular tachycardia, non sustained ventricular tachycardia (NSVT) and repetive VPCS.[4] VPC is a premature, wide QRS complex that has a distinct configuration and is not preceded by a P wave. They may appear in pattern of two consecutive VPCS (couplets), bigeminy or trigeminy. The occurrence of 3 or more consecutive VPCS is considered VT.[5] Among patients with frequent VPCS, 50% had only isolated VPCS and/or couplets, whereas 50% also had short runs of NSVT.[6]

OT arrhythmia is due to triggered activity secondary to CAMP mediated delayed after depolarizations. The tachycardia can be terminated with adenosine, beta blockers or calcium channel blockers.[1]

RVOT arrhythmias manifest at the age group 30 to 50 years; range being 6 to 80 years.[7] Females are more frequently affected.[3,8] Patients may be asymptomatic, but often present with palpitations, chest pain, dyspnea, even syncope.[1] RVOT arrhythmias can often be provoked by exercise, caffeine, emotional stress and hormonal flux as during pregnancy. Isoproterenol infusion, pseudoephedrine and rarely aminophylline, calcium infusion or atropine may facilitate arrhythmia induction.[3,5]

On 12 lead ECG, there is a LBBB pattern in precordial leads. Also consistent with the OT site is the inferior ECG axis.[1] RVOT arrhythmias should be distinguished from the

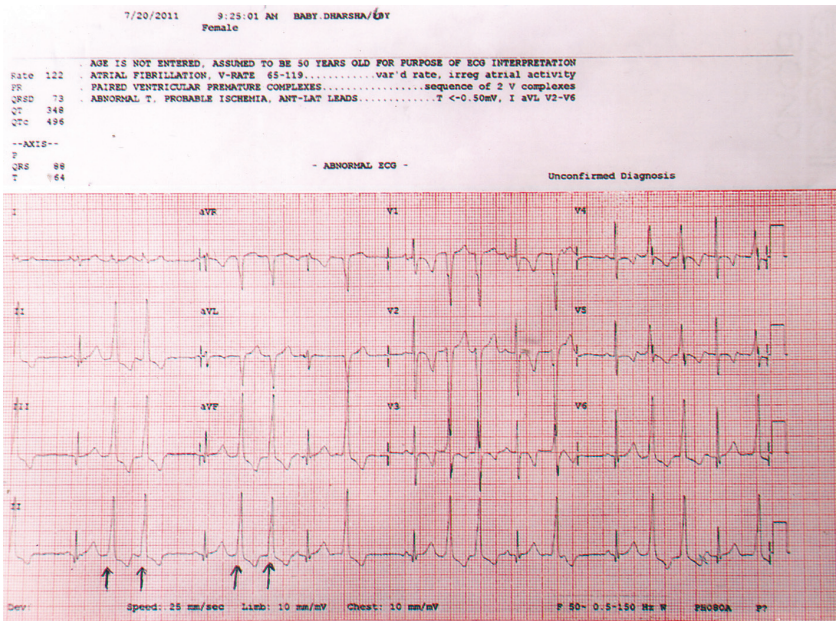

Fig.1. Ventricular premature complexes in couplets in all leads

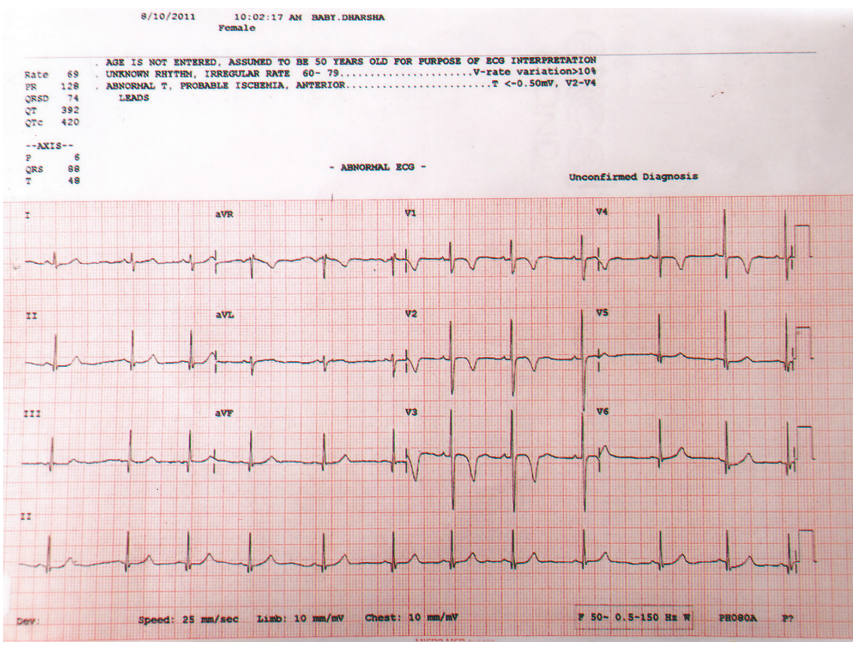

Fig.2. Sinus rhythm restored

inapparent cardiac disease, arrhythmogenic right ventricular dysplasia.[8,9]

In general, RVOT tachycardia shows a benign course.[10] Frequent VPCS can cause left ventricular dysfunction.[3] There is a potential to develop VPC related cardiomyopathy.[4,11]

Long term oral therapy with beta blockers may control the arrhythmias.[1] When they are highly symptomatic and refractory to oral antiarrhythmic therapy or when they cause ventricular dysfunction, radiofrequency ablation is a recommended treatment with high success rate and low risk of complications.[3]

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CLIPPINGS

Dressings or topical agents for preventing pressure ulcers

Pressure ulcers, which are localised injury to the skin, or underlying tissue or both, occur when people are unable to reposition themselves to relieve pressure on bony prominences. Pressure ulcers are often difficult to heal, painful and impact negatively on the individual's quality of life. The cost implications of pressure ulcer treatment are considerable, compounding the challenges in providing cost effective, efficient health services. Efforts to prevent the development of pressure ulcers have focused on nutritional support, pressure redistributing devices, turning regimes and the application of various topical agents and dressings designed to maintain healthy skin, relieve pressure and prevent shearing forces. Although products aimed at preventing pressure ulcers are widely used, it remains unclear which, if any, of these approaches are effective in preventing the development of pressure ulcers.

Objective was to evaluate the effects of dressings and topical agents on the prevention of pressure ulcers, in people of any age without existing pressure ulcers, but considered to be at risk of developing a pressure ulcer, in any healthcare setting.

Selection criteria: RCTs evaluating the use of dressings, topical agents, or topical agents with dressings, compared with a different dressing, topical agent, or combined topical agent and dressing, or no intervention or standard care, with the aim of preventing the development of a pressure ulcer.

Authors' conclusions: There is insufficient evidence from RCTs to support or refute the use of topical agents applied over bony prominences to prevent pressure ulcers. Although the incidence of pressure ulcers was reduced when dressings were used to protect the skin, results were compromised by the low quality of the included trials. These trials contained substantial risk of bias and clinical heterogeneity (variations in populations and interventions); consequently, results should be interpreted as inconclusive. Further well designed trials addressing important clinical, quality of life and economic outcomes are justified, based on the incidence of the problem and the high costs associated with pressure ulcer management.

Moore ZEH, Webster J. Dressings and topical agents for preventing pressure ulcers. Cochrane Database of Systematic Reviews 2013, Issue 8. Art. No.: CD009362. DOI: 10.1002/14651858.CD009362.pub2. Assessed as up to date: February 1, 2013.

CASE STUDY

SPONTANEOUS PERFORATION OF THE BILE DUCT IN AN ADOLESCENT - AN UNUSUAL COMPLICATION OF CHRONIC CALCIFIC PANCREATITIS

*** Sumathi B**
**** Venkatachalam A**
***** Nandhini G**
****** Sathiyasekaran M**
******* Ramakrishnan R**
******** Jayanthi V**

Abstract: *Spontaneous perforation of common bile duct (SPBD),an unusual cause of acute abdomen presenting as biliary ascites is very rare. The aetiology of SPBD is multifactorial and include congenital mural weakness of the common bile duct, ischemia, distal biliary obstruction, pancreaticobiliary malunion, infection, trauma and rarely acute or chronic pancreatitis. Diagnostic ascitic tap helps in diagnosis. We report an adolescent with chronic calcific pancreatitis who had spontaneous perforation of bile duct and was managed by therapeutic endoscopic retrograde cholangio pancreaticography (ERCP).*

Key words: *CCP, Adolescent, SPBD, Biliary ascites.*

Spontaneous perforation of the bile duct (SPBD) presenting as biliary peritonitis is a rare entity in adolescents and adults, more often reported in neonates and young infants.[1] The term "spontaneous" is used when there is no underlying trauma or iatrogenic injury.[2] This terminology may seem to be a misnomer as the spontaneity of the perforation is secondary to several etiologies.[3] The possible causes for spontaneous perforation are ischemia, infection, choledochal cyst, distal bile duct obstruction, pancreatico biliary malformations or idiopathic.[4,5] Congenital malformation or mural weakness of the bile duct at the junction of common bile duct (CBD) and cystic duct during embryogenesis is a possible explanation for the common site of perforation.[6] The transient increase in intra biliary pressure due to sludge, stones, stricture may cause a "blow out" at this weak point. Biliary ascites due to bile duct perforation is a rare complication of either acute[7] or chronic pancreatitis[8,9] and may pose a diagnostic challenge since it may mimic pancreatic ascites. Surgical intervention is the accepted modality of treatment of SPBD especially in neonates.[4,10] Endoscopic retrograde cholangio pancreatico graphy offers an excellent non surgical alternative technique for diagnosis and therapy for SPBD.[3,7]

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Case Report

A 12 years old boy presented to the emergency department with severe upper abdominal pain and generalised abdominal distension for 3 days. He was diagnosed at the age of 10 years to have "tropical calcific pancreatitis (TCP)". Since diagnosis he had three to four episodes of abdominal pain requiring analgesics. There was no past history of jaundice.

Magnetico Resonance Cholangio Pancreaticography (MRCP) showed a dilated and irregular pancreatic duct measuring 8mm with intra ductal pancreatic stones, largest one measuring 9mm in the region of the head close to ampulla. A stricture was seen at the terminal end of the pancreatic and bile duct (Fig.1). The CBD was mildly dilated. There was no choledocholithiasis, cholelithiasis or pancreatico biliary malformation. On admission. the child was febrile, toxic, tachypnoeic with mild icterus. The pulse rate was 162 per min and blood pressure was 90/60 mm of Hg. Abdomen was distended, tense with diffuse tenderness and sluggish bowel sounds. A diagnosis of acute on chronic pancreatitis with cholangitis, pancreatic ascites and peritonitis was considered.

Investigations: Hemogram: Hb 10 gm/dL, WBC 18,000 cells/cu mm with 85% neutrophils, platelet count 2100 00 cell/cu mm, CRP 124 mg/L. Liver function tests: S bilirubin 2.1 mg/dL, direct 1.4 mg/dL, AST 160 U/L, ALT

89I U/L, ALP 630 U/L, GGT 193 U/L, blood sugar 82 mg/dL, S amylase 686 IU/dL, lipase 1220 U/L and S. creatinine 0.8 mg/dL. Arterial blood gas analysis revealed pH 7.44, PCO_2 26.6, PO_2 138.3, HCO3 17.9.

Methicillin resistant Staphylococcus aureus (MRSA) grown in blood was sensitive to teicoplanin. Urine culture was negative.

X-ray chest showed bilateral pleural effusion. Ultrasound of the abdomen showed a dilated pancreatic duct, fluid collection in the peri pancreatic bed, distended small bowel loops and ascites.

Management: Child was managed with intravenous fluids and analgesics. Broad spectrum antibiotic cefaperazone sulbactam was initiated and later switched over to teicoplanin. Therapeutic paracentesis was done to relieve the progressive painful abdominal distension causing respiratory distress. The ascitic fluid was greenish in color. Analysis of the fluid showed 580 WBC /cu mm, amylase 540 IU/L, a high bilirubin of 8 .2 mg/dL, LDH 354 IU/L, and SAAG < 1.1, suggestive of biliary ascites .Contrast enhanced computed tomography (CECT) confirmed chronic pancreatitis. CBD was not well seen. In addition, there was a loculated right subdiaphragmatic fluid collection with internal septae.

Final diagosis was bile ascites secondary to spontaneous bile duct rupture. ERCP showed a leak in the CBD at the junction of CBD and cystic duct (Fig.2) with an impacted calculus at the ampulla. The stone was extracted after a precut papillotomy (Fig.3) and a 7 Fr 10

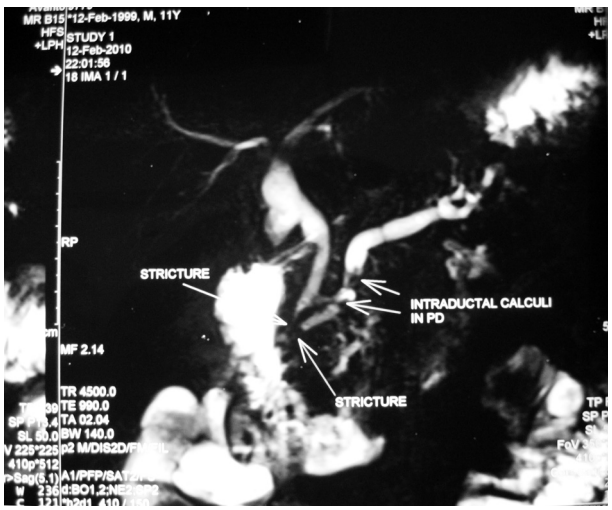

Fig.1. MRCP showing a dilated and irregular pancreatic duct with intra ductal pancreatic stones, stricture at the terminal end of the pancreatic and bile duct

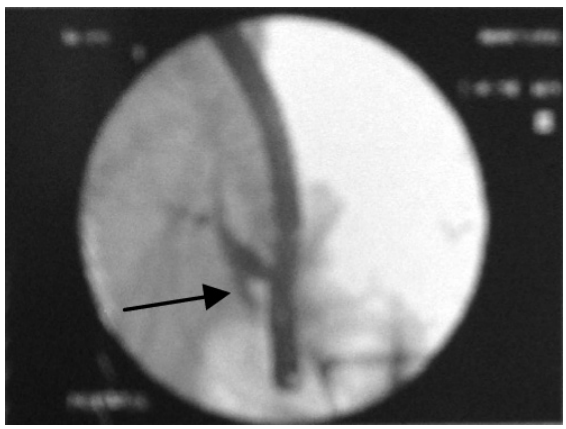

Fig.2. ERCP showing leakage of contrast at junction of CBD and cystic duct

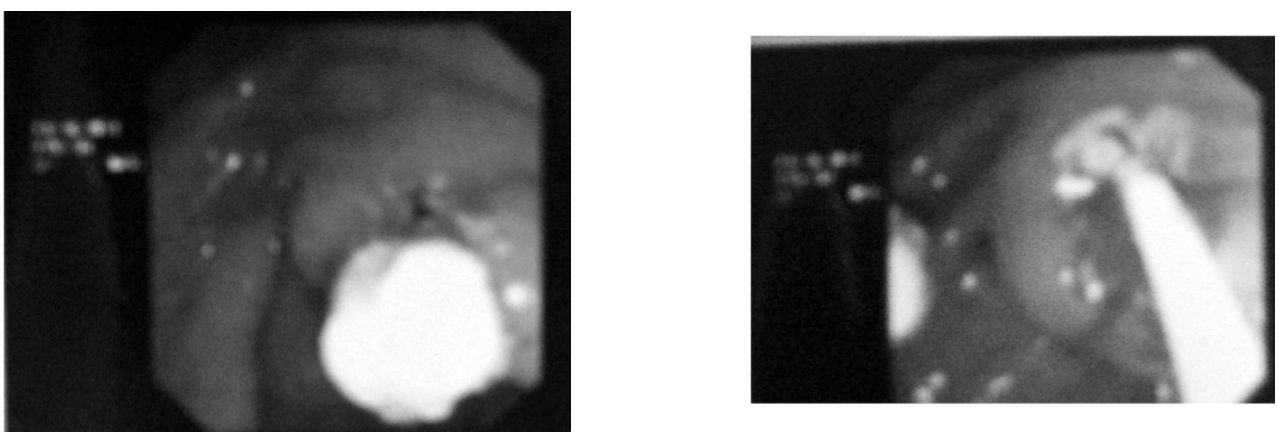

Fig. 3a & 3b: Duodenoscopy showing pancreatic stone at ampulla and stent in situ

cm pig tail stent deployed across the leak. Post procedure, abdominal pain and distension decreased with resolution of ascites. The biliary stent was removed after 6 months.

Discussion

Spontaneous perforation of the bile duct, though uncommon in infants and children, is frequently cited as the second most common indication for jaundice requiring surgical intervention in infancy.[11] Dijkstra in 1932 reported the first case and since then about 150 cases have been described in the literature.[12] A review of 20 years data (1988 to 2008) revealed 60 cases of SPBD in infants and children. SPBD usually occurs within the first year of life with the age at presentation ranging from 25 weeks' gestation to 15 years with a median of 4 months. In two-thirds of children the most common presentation is abdominal distension and/or jaundice.[1] Other symptoms include fever, vomiting, abdominal mass and pale-colored stools. Acute onset may occur in 10 -20 %.[10] In majority of neonates and young infants the presentation is subacute.[13]

Serum bilirubin and liver enzymes are often normal or only mildly elevated. When elevated, this suggests obstruction to the biliary tree and in the presence of co-existing fever would indicate cholangitis. The triad of peritonitis, absence of free air in the abdomen and high bilirubin in ascitic fluid (more than serum bilirubin) are diagnostic of biliary ascites.[1, 10]

Ultrasound is an ideal screening procedure to detect gall stones, choledochal cyst, dilated biliary or pancreatic duct, pancreatic calculi, ascites, loculated fluid collections and pneumoperitinoeum. Radionuclide studies demonstrating the tracer in the peritoneal cavity and not in the intestine is diagnostic but was not performed in this child due to non availability of the investigation.[14] MRCP helps to delineate the anatomy of the biliary and pancreatic system.[15]

The congenital mural weakness of the anterior wall at the junction of the common bile duct and cystic duct during embryogenesis in addition to an increase in the intra ductal pressure are the proposed theories for the common site and trigger for perforation.[6] The site of leak could also occur anywhere along the CBD, cystic duct or hepatic duct. Various etiologies for SPBD have been reported in children including ischemia, choledochal cyst, viral infections such as HIV,[16] anomalous pancreatico biliary malformation (PBM), Ivemark syndrome[17] and distal biliary obstruction due to stenosis, stones or sludge. Ng WT, et al reported that virtually all Asian patients with SPBD had a radiographically demonstrable long common pancreatico biliary channel.[18] In PBM the explanation for the perforation is the reflux of the pancreatic juice into the bile duct which gets activated and becomes potentially destructive when it mixes with the bile. Chronic calcific pancreatitis as a cause of distal biliary obstruction leading to bile duct perforation has been eluded in literature. During the last 20 years only 2 cases both [8, 9] from India of chronic calcific pancreatitis with bile duct perforation have been reported. Pancreatico biliary malformation with a long common channel was present in one case[8] but was not identified in this child who had a distal CBD stricture secondary to CCP. Distal common bile duct strictures have been reported to occur in 2.7% to 45.6% of patients with CP. These strictures can occur from inflammation, fibrosis or compression from a pseudocyst or pancreatic intra ductal stone.[19] CBDS occurs as a consequence of recurrent acute inflammatory episodes which may ultimately result in a peri ductal fibrotic stricture. In this patient a combination of an increase in intra ductal pressure caused by the distal CBD stricture and the impacted pancreatic stone along with bile stasis and cholangitis would explain the cause of perforation.

Surgical intervention has been the universal recommendation for SPBD. An operative cholangiogram helps to identify obstructive from non obstructive perforations. Conservative management with either peritoneal or T tube drainage suffices in the majority. If there is obstruction or PBM then biliary enteric anastomosis or biliary reconstruction (BR) may be necessary. Depending on the child's condition and surgical expertise biliary reconstruction can be done either at the same sitting or as a second laparotomy. Laparoscopic surgery is now a definite alternative for diagnosis and percutaneous drainage. Broad spectrum antibiotic is recommended for all children with SPBD even though the bile may be sterile. Portal vein thrombosis has been reported as a complication of SPBD usually in posterior perforations.[20]

Therapeutic ERCP in SPBD as the initial cholangiographic technique and the primary therapy spares the need for exploratory surgery as in this case. In the literature since 2006 there have been 3 cases[3,7,16] of SPBD managed by ERCP of which one[16] required surgery. To our knowledge this is the first report in the pediatric age group of SPBD in CCP where ERCP has helped in diagnosis and therapy. ERCP therefore plays a definite role in SPBD in older children especially if there is an underlying problem such as pancreatic calculi, choledocholithiasis or distal bile duct stricture which can also be managed endoscopically. The rarity of SPBD in CCP, a simple test such as paracentesis which helps in diagnosis and the successful endoscopic management are the highlights of this report.

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NEWS AND NOTES

'CME on Pediatric Dermatology'

DERMPED CME 2013

Organized by IAP Dermatology Chapter

Venue: Savera Hotel, Chennai Date: 24th November 2013

For Details, please contact:

Dr.V.Anandan & Dr.C.Vijayabhaskar,
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223, L-Block, 21st Street, Anna Nagar East, Chennai - 600 102.
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LETTER TO EDITOR

With reference to the clipping on 2013;15(3);221 the authors observation "Electroencephalogram should be done in all patients with complex febrile fits especially those who had multiple or prolonged seizures" has to be considered against existing guidelines and text book statements. "EEG is generally not indicated because it has limited prognostic value and it cannot be used as a means for selecting candidates for prophylactic therapy. I. that EEG changes are more common in children with febrile seizures with complex febrile seizures, focal seizures and pre existing neurological abnormalities the association was not as close as to that of age and number of previuos convulsions."(AICARDI). This is also the same view by American Academy of Pediatrics. In status epilepsy of all causes including febrile status it is useful (febstat study). Hence the pediatricians should take a balanced view of the article until future studies confirm this findings.

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REVIEWER INDEX

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Aruna Mohan
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Balameena S
Baldev S. Prajapati
Bhaskar Raju B
Dignant shastri
Gnanasambandam S
Jayanth K. Iyengear
John Matthai
Kabra SK
KalpanaGowrisankar
Kulkarni ML
Kumaresan G
Kundan Mittal
Leema Pauline
Madhu R
Madhulika Kabra
Mamta Manglani
Mamta Muranjan
Meena Jayashankar
Nagabhushana S
Panchapakesa Rajendran
Rajiv Kumar Bansal
Rasik Shah
Revathy Raj
Ritabrata Kundu
Riyaz A
Sujata Sawhney
Sujatha Jagdeesh
Sundararaman PG
Surekha Rajashyaksha
Velmurugan R
Vijayakumar M
Vijayasekaran D
Vijyalakshmi G
Viswanathan V

AUTHOR INDEX

SUBJECT INDEX

15th Annual Conference of IAP Intensive Care Chapter

National Conference of Paediatric Critical Care

NCPCC 2013

WITH

Mahabale shwar

1st Annual Conference of Maharashtra State IAP Intensive Care Chapter

(MSICC 2013)

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CME (Advance)	2000	2000	2000	2000
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3. Bronchiolitis – update
4. Dr.L.Subramaniam Oration
5. Non resolving pneumonia
6. Asthma medication delivery: Mist and myths
7. Newer investigations for M.Tb diagnosis
8. What is new in acute asthma management
9. Infantile wheeze
10. Sinusitis - bacterial or viral
11. Pneumococcal vaccines –current stand
12. Modes of NIV
13. ABG - simplified
14. Neonatal bronchoscopy
15. Cystic lung lesions – medical or surgical?
16. Interpretation of CT / MRI chest
17. Management of empyema: antibiotics, fibrinolytics or VATS?
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*Registration Fee	Up to 30th Nov 2013	After 30th Nov 2013
Post Graduates	Rs.800	Rs.900
Delegates	Rs.900	Rs.1000
Spot		Rs.1200

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Siddharth Memorial Oration

"Opportunities and challenges of therapeutic approaches for Duchenne Muscular Dystrophy"

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What is the need for changing maintenance IV fluid therapy	Dr.Bala Ramachandran
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