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TOPIC OF INTEREST - "IAP - IJPP CME 2017"

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IAP - IJPP CME 2017

GROWTH CHARTS AND MONITORING

***Hemchand K Prasad**
****Vaman Khadilkar**

Abstract: *Growth is a measure of well being in a given child. All pediatricians should follow the growth monitoring guidelines released in 2007. Measured growth should be plotted on IAP modified WHO charts in children less than 5 years and on IAP 2015 charts above 5 years. Standard guidelines of WHO and IAP should be used to measure and plot growth measures. The redefined target range, short stature and overweight cut-offs must be used to diagnose growth problems early. The new charts allow a pediatrician to plot the growth in accurate months. It is also colour coded to diagnose and alert families of children with obesity. The definitions of stunting, wasting, overweight and obesity in different ages are presented. Early recognition of these growth abnormalities is crucial for the long term health of the child.*

Keywords: *Growth charts, Growth monitoring, Adult equivalent.*

Growth and development are complementary processes. Growth indicates the quantitative changes in the body-height and weight. The basic pre-requisites for optimal assessment of childhood growth are reliable growth parameter, reliable reference population, reliable cut-off and reliable calibrated instrument.

Importance of monitoring growth of a child

Growth is a measure of well being in a given child. Pediatric care consists of two important components: curative and preventive care. Vaccinations, growth monitoring and developmental assessments are important pillars of preventive care. Growth monitoring helps a pediatrician not only to reassure normalcy but also to identify growth disorders, nutritional disorders and systemic diseases, early. Normal growth occurs only if the child is healthy.

Growth standard and reference - Difference

A growth reference is a descriptive chart prepared from a population which is believed to be growing under optimal health and nutrition. A growth standard is a prescriptive standard prepared from a population where all possible environmental and nutritional variables are controlled. It is a sole independent instrument upon which decisions are made. The various growth charts that are available today are given in Box 1.

Box 1. Growth charts

National

i. Old IAP charts (K N Agarwal charts)[1,2] (descriptive)
ii. Charts from Khadilkar, et al (descriptive)
iii. Charts from Marwaha, et al[3] (descriptive)
iv. New IAP 2015 charts (descriptive for height and weight, prescriptive for BMI)[4]

International

i. Centre for disease control (CDC) charts (descriptive)
ii. WHO 2006 standards 0-5 years (prescriptive)
iii. WHO 2007 charts(descriptive)[5]
iv. International Obesity Task Force (IOTF) cut-offs for body mass index (BMI)[6] (prescriptive)

Interpretation of growth measures

Percentiles and Z–scores are the two ways of interpreting a growth measure. Percentiles and z-scores are interchangeable. On a practical note, it is suggested that pediatricians adhere to percentiles for common anthropometric measures and Z-scores be used in diseased children with severe growth abnormalities (e.g. severe chronic diarrhea and growth hormone deficiency). The equivalent percentiles and z-scores are shown in Table I.

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Table I. Anthropometric measures interpretation - Equivalent percentiles and Z-scores[7]

Z-score	Percentile
0	50
-1	15
-2	3
-3	1
+1	85
+2	97
+3	99

Need for using the same chart and cut-offs for interpretation

The usage of different growth charts and different cut-offs lead to different interpretation of the same anthropometric measure. Consider a 10 year old boy with a height of 124 cm and a BMI of 19.1 kg/m^2. If different growth charts are used he can be labelled as

a) Stunted – CDC or WHO 2007 charts

b) Normal stature – IAP 2015 chart (3rd percentile to diagnose short stature)

c) Overweight – IAP 2015 charts, IOTF charts and WHO 2006 charts

d) Normal BMI – CDC, Marwaha references or Khadilkar 2007 references

This ultimately results in ambiguity on the intervention warranted for the child and compromises care for the child.

Anthropometric measures to be recorded in office practice

The IAP has come out with growth monitoring guidelines for pediatricians to adopt in office practice in 2007.[8] The same guidelines are applicable today. The measurements should be made as per the recommendations of the WHO.[9] The recommendations depend on the age of the child:

a) 0-2 years - length, weight and head circumference at 0, 6, 10 and 14 weeks, 6, 9, 15 and 18 months (every vaccination visit),

b) 2-5 years – height, weight and head circumference every 6 months and

c) Beyond 5 years – height, weight, BMI (every 6 months till 9 years and annually there after); SMR every year.

Growth charts preferred in office practice

The growth charts committee of the IAP recommends that the following growth charts be used in pediatric office practice - IAP modified WHO charts in children less than 5 years and IAP 2015 5-18 years charts for children aged 5-18 years. Combined IAP - WHO charts from birth to 18 years (0-5 WHO and 5-18 IAP) are also made available for continuous growth monitoring. The similarities between the two charts are summarised in Table II.

Table II. Comparison of the similarities in the WHO 2007 standards and IAP 2015 references

	WHO 2006 standards[10]	IAP 2015 references
Nature	Prescriptive - described how children should grow	Prescriptive for BMI; descriptive for other aspects of growth (height and weight)
Norm	Breast feeding established as a biological norm	Good nutrition and health are a biological norm
Statistical methods	LMS (lambda-mu-sigma) method of statistics[11]	LMS method of statistics
Exclusion of obese subjects	Excluded weight for height more than +2 SD in cross sectional component and +3SD in longitudinal component; detects both malnutrition and overweight	Excluded weight for height more than +2 SD; detects both malnutrition and overweight
Study sample	Pooled sample from 6 countries (internationally representative sample)	Pooled sample from 13 studies (nationally representative sample)

BMI – Body mass index; LMS– Power in the Box - Cox transformation (L), Median (M), Coefficient of variation and skewness (S).

Method of plotting on growth chart

Using a growth chart begins with entering the name and date of birth on the chart. Growth is marked with a dot (not circle or cross) at point of intersection of measure (on the y-axis) and the chronological age (on x-axis). The target height is calculated as below and marked with a horizontal arrow at 18 years. Target range is marked as 6 cm above and below the target height.

$$\text{Target height in boys} = \frac{\text{Father's height + mother's height + 13}}{2}$$

$$\text{Target height in girls} = \frac{\text{Father's height + mother's height – 13}}{2}$$

When subsequent measurements are made on the same chart, the points are joined by a line. It is necessary to explain the findings to the parents, reassure them and remind them of the next growth measurement. The following common errors are to be avoided. All girls must have their growth plotted on pink chart and boys on the blue chart. Plotting growth on the chart for opposite sex is not acceptable. Weight should not be measured more than once in 15 days and 30 days - during and beyond infancy, respectively. This is to avoid unnecessary anxiety.

Changes in the current recommendations

The following aspects are new in the current recommendations (Table III). The target range has been lowered (from ±8 cm to ±6 cm) in line with the other international guidelines to facilitate early recognition of abnormal growth. The threshold for abnormality picked up by crossing of percentiles varies with age owing to the rate of growth being high in infancy and slower beyond infancy. One must remember that movement towards the 50th percentile is a good sign and away from the median should be viewed suspiciously. The new charts allow a pediatrician to plot at accurate months (i.e there are 12 divisions between two consecutive years) thus making the concept of decimal age obsolete.

New charts - More user friendly and parent friendly

The new charts are very user friendly. The IAP modified WHO charts allows one to plot - weight, height and head circumference on a single page and at convenient 15 day intervals. The weight, height (0-18) and BMI (5-18) measurements can be plotted at 6 monthly intervals on the 0-18 year charts (Fig.1). The BMI and weight for height charts in 0-18 charts and 0-5 charts respectively are colour coded - red colour indicating obesity. This is in principle with the idea of Prof David Morley - the founder of growth charts who intended that the charts should either reassure or alert the mother.

Figure 1(b) IAP 2015 charts for children 5-18 years and combined WHO IAP 2015 charts for 0-18 years.

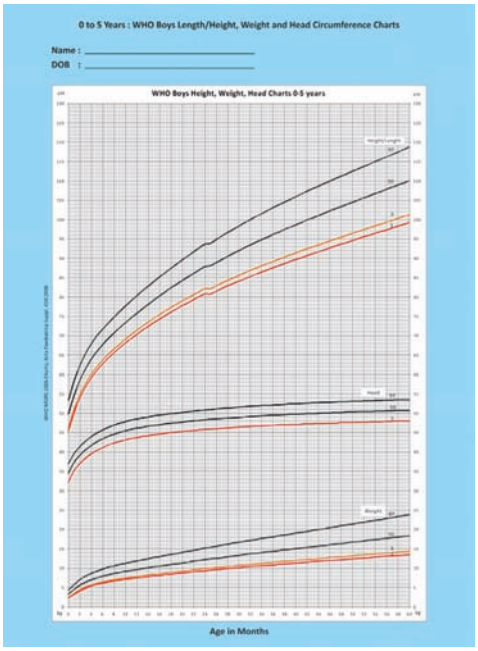
Recognition of abnormal growth using growth chart

The cut-offs and terminologies to be used are summarised in Table IV. One must note the following:

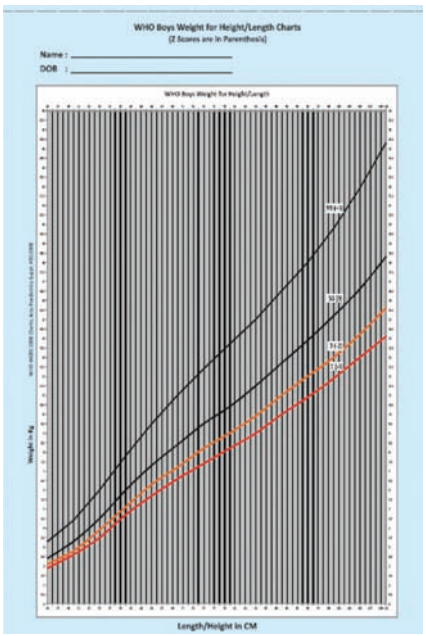
a) It is preferred to interpret weight in conjunction with height and not in an isolated perspective. This is on

Table III. Modifications in current recommendations of IAP growth charts

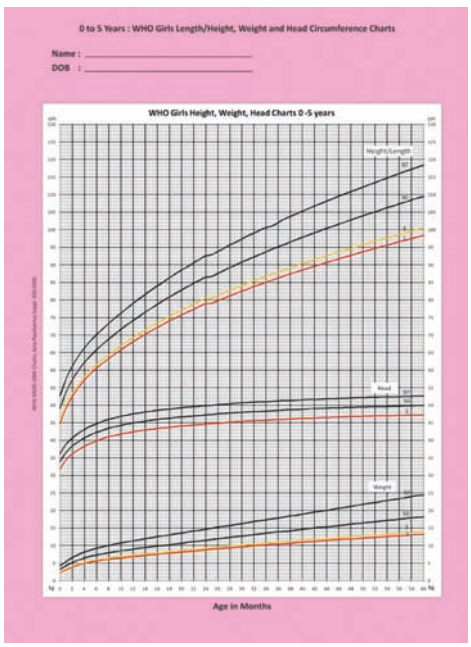
	Previous	Current
What chart should be used in a child <5 years	WHO 2007 standards	IAP modified WHO charts
What chart should be used in a child >5 years	Old IAP charts	Combined WHO-IAP2015 charts
Definition of short stature	<3rd percentile in IAP chart constructed for the growth monitoring guideline or <5th percentile on IAP charts	<3rd percentile on the new IAP chart
Overweight	>85th percentile	>23rd adult equivalent
Obesity	>95th percentile	>27th adult equivalent
Plotting age	Decimal age	On accurate months
Target range	8 cm above and below target height	6 cm above and below target height



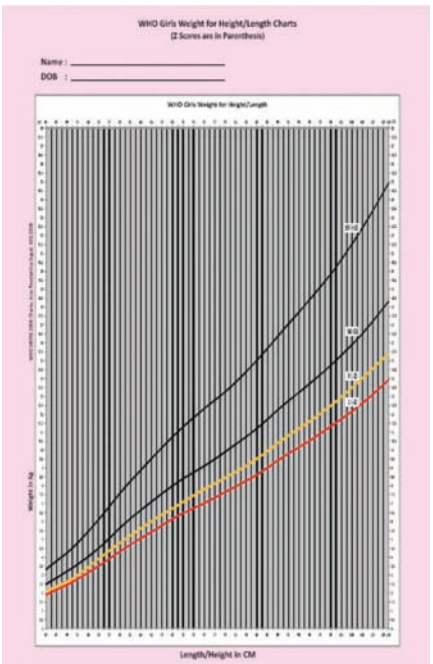
Height, weight and head circumference (boys)



Weight for height chart 0-5 years (boys)

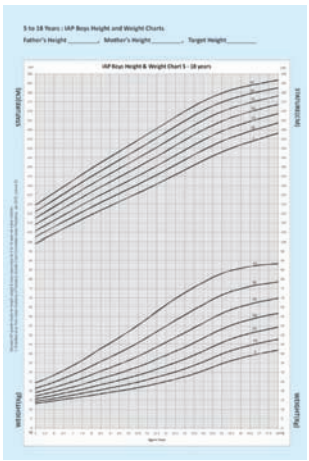


Height, weight and head circumference (girls)

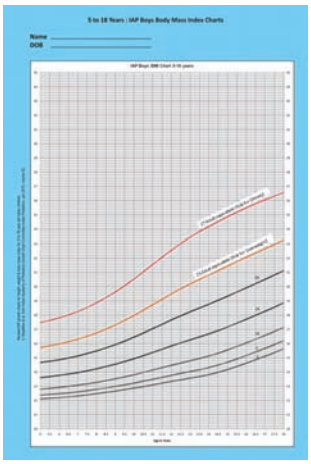


Weight for height chart 0-5 years (girls)

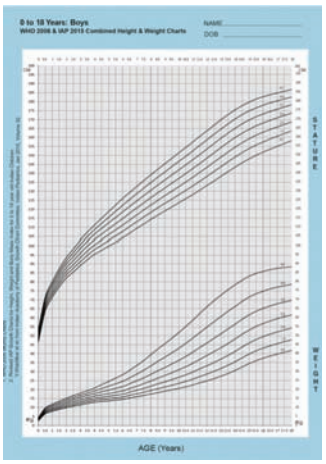
Fig.1(a) IAP modified WHO charts for children 0-5 years



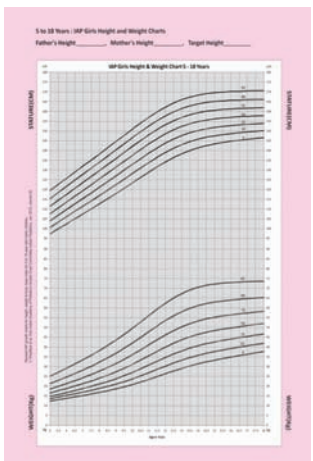
Weight and height 5-18 years (boys)



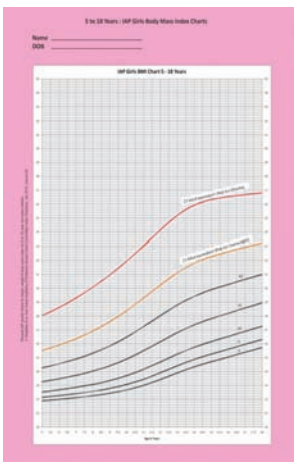
BMI 5-18 years (boys)



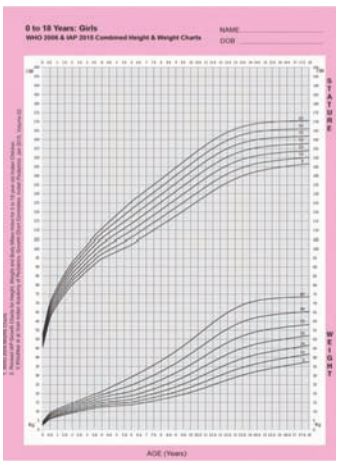
Weight and height 0-18 years (boys)



Weight and height 5-18 years (girls)



BMI 5-18 years (girls)



Weight and height 0-18 years (girls)

Fig.1(b) IAP 2015 charts for children 5-18 years and combined WHO IAP 2015 charts for 0-18 years

the lines of WHO recommendations. Hence, BMI or weight for height is more useful than isolated weight (especially in >5 years) in diagnosing wasting or obesity.

b) Crossing of percentile lines should be interpreted carefully. Although crossing of percentiles have to be viewed carefully, one must keep in mind certain clinical situations where crossing of percentiles may be physiological: SGA trying to catch up; a child with familial short stature catching down on height percentiles.

c) The growth charts committee recommends weight for height to diagnose wasting and obesity in the under 5 age group. This is purely for logistic reasons as errors are minimal on weight for height versus BMI. Weight for height charts and BMI for age assessments often complement each other.

The term 'Severe Acute Malnutrition (SAM)' was defined by WHO for health workers based on weight for height Z-score of less than -3 should be used beyond 6 months.

Recognition of abnormal growth - Actions

On recognition of abnormal growth, a pediatrician should check the accuracy of measurement/ plotting, look at the trend of deviation (a single cross sectional measure has limitations and growth does not always follow a smooth curve) and plan further evaluation. Once abnormal growth is recognised, a step wise analysis of anthropometry in the background of clinical scenario is necessary for further

Table IV. Abnormal growth parameters – Current acceptable terminologies

		<5 years	>5 years
Height	Stunting Severe stunting	<3rd percentile <0.1 percentile	<3rd percentile
Weight	Underweight Severe underweight	<3rd percentile <0.1 percentile	
BMI or weight for height	Wasting Severe wasting Overweight Obese	<3rd percentile <0.1 percentile >2SD from mean >3SD from mean	<3rd percentile >23rd adult equivalent >27th adult equivalent
Crossing of percentiles	Abnormal growth	2 major percentiles in 6 month period	2 major percentiles in a one year period

elucidation. The measures can be interpreted by either calculation of height age and weight age or by calculation of Z scores.

Calculation of height age and weight age

The appropriate anthropometric measure is plotted on the correct growth chart. A line is drawn from the plotted point to the 50th percentile and then vertically downwards from the 50th percentile to touch the X- axis which is the corresponding height age and weight age as depicted in Fig.2. A child who has:

a) CA = HA = WA : is a normal child

b) CA > HA > WA : has nutritional deprivation or systemic disease

c) CA > WA > HA : has endocrine disease

d) HA > WA > CA: has precocious puberty

e) WA > CA > HA : has endocrine obesity

f) WA > HA > CA : has nutritional obesity

It would be appropriate to remember that addition of accurately assessed bone age to the above equations in specialised units adds more value. But the above equations guide the pediatrician to rationalise and plan investigations (Fig.3).

Calculation of Z scores

Z score is a measure of deviation from the mean for the reference population. The Z score for a given anthropometric measure is calculated as follows:

$$\text{Z score} = \frac{\text{Measure – mean for age and sex}}{\text{SD for age and sex (reference population)}}$$

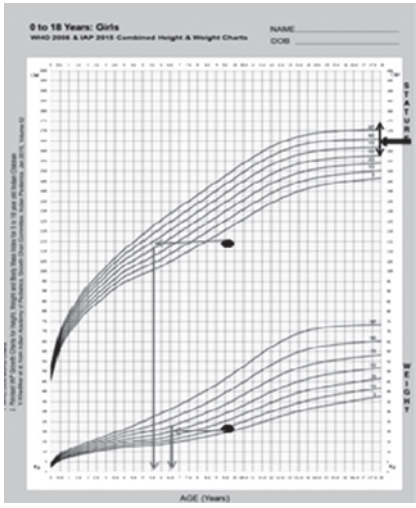


Fig.2. Calculation of height age and weight age

The same Z scores may be calculated by downloading the WHO anthro software (for WHO standards) or LMS data can be procured for the authors for usage on their personal computer. A child with:

a) Height Z score less than -2 is stunted

b) Height Z score more than +2 (5-18 years) and more than +3 (0-5 years) is tall stature

c) BMI or weight for height Z score less than -2 is wasted

d) Height Z score more than +1.32 for boys and more than +1.64 for girls on IAP 2015 charts and more than +3 (0-5 years) is obesity.

The etiology can be derived based on the child's growth (Table V).

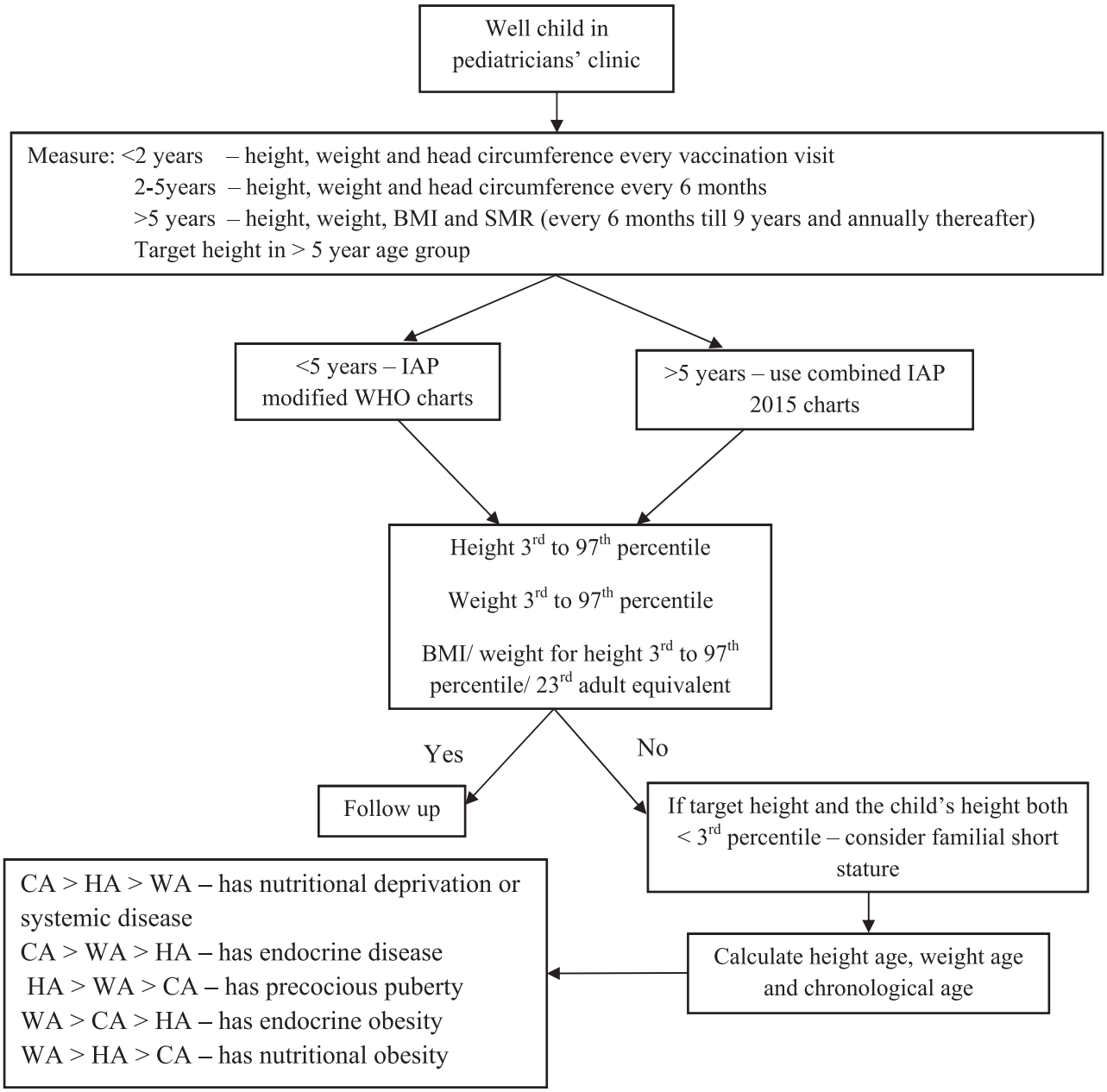


Fig.3. Summary of growth charts and growth monitoring in office practice

Growth of a LBW preterm baby – Assessment

The growth curves for preterm babies have been developed similar to the WHO 2007 standards - intergrowth 21st post natal standards.[12] These standards must be used in preterm babies till they reach term gestational age.

Specialised growth charts

The growth of certain genetic and skeletal pathologies are varied from normal children and these conditions have specialised growth charts. Special growth charts are available for: Down syndrome, Turner syndrome, achondroplasia, Russell Silver syndrome and hypochondroplasia. Such usage is mandatory to recognise co-morbidities to the pathologies e.g. usage of Down's syndrome chart will facilitate early recognition of hypothyroidism in these children.

Conclusion

Reliable growth markers, reliable references and cut-offs for usage have been summarised. It is of pivotal importance that all health professionals involved in the care of children equip their units with reliable calibrated

Table V. Growth parameters – Inference

Growth parameters		Etiology
Stunted and wasted	Stunting more than wasting	Endocrinopathy
	Wasting more than stunting	Systemic or nutritional disorder
Obese child	With tall stature	Nutritional obesity
	With short stature	Endocrine obesity
Tall child	With normal BMI	Precocious puberty

measurement devices and assess the growth of children as per laid down guidelines and contribute to preventive care of children.

Points to Remember

- ***A 'growth reference' is a comparison of the given child's growth to the local population. A 'growth standard' is a concept of "what should be the growth in optimal conditions".***
- ***Usage of different growth charts and different cut-offs lead to different interpretation of the same anthropometric measure.***
- ***All pediatricians should follow the IAP growth monitoring guidelines published in 2007[8].***
- ***IAP recommends - IAP modified WHO charts in children less than 5 years and IAP 2015 growth charts in children more than 5 years.***
- ***Key modifications in the new recommendations include - Lowered target range to ± 6 cm; new definition of overweight and obesity to 23rd and 27th adult BMI equivalent and definition of short stature to height < 3rd percentile.***
- ***The new charts are user friendly - Colour coding for obesity; plotting can be done in accurate months (not decimal age).***
- ***Once abnormal growth is recognised, calculating height and weight age or calculation of Z scores must be done for further evaluation.***
- ***Specialised growth charts - Intergrowth and syndrome specific charts must be used in preterms and syndromic children, as appropriate.***

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IAP - IJPP CME 2017

H1N1 REVISITED

***Vidya Krishna**

Abstract: *Seasonal influenza is an acute febrile respiratory illness caused predominantly by Influenza type A (H1N1). Influenza, traditionally an under recognised disease, gained notice after the 2009 pandemic. In children, it can cause asymptomatic infection to severe illness and even death, particularly in infants and those with co-morbid conditions. This article will focus on the epidemiology of influenza and clinical aspects including clinical features, complications, diagnosis, treatment and prevention including chemoprophylaxis, vaccination and infection control measures.*

Keywords: *Influenza, H1N1, Children.*

Influenza is an important cause of acute respiratory infections worldwide and is associated with more severe disease and mortality especially in children under 5 years of age. Influenza accounts for 10% of hospitalisation due to respiratory illness in children less than 18 years of age.[1] Various studies have shown that influenza accounts for 15% - 20% outpatient cases in children.[2,3]

Epidemiology

Classification

Influenza viruses belong to the Orthomyxoviridae and have 3 distinct types-A, B and C. The differences between the 3 viruses are given in Table I. They are enveloped single stranded RNA viruses. Influenza A viruses are further subtyped based on their hemagglutination (HA) and neuraminidase (NA) antigens (Fig.1).

The influenza viruses undergo continuous changes and hence, the vaccine strains must be updated every year. "Antigenic drift" refers to the minor antigenic changes in the HA or NA or both resulting in new strains of the virus and cause seasonal outbreaks or epidemics. "Antigenic shift" refers to abrupt and major antigenic changes in HA or NA due to genetic re-assortment with animal strains and is responsible for pandemics.

Seasonality

Influenza A (H1N1) and A (H3N2) are the circulating seasonal influenza A virus subtypes, with majority of infections caused by A (H1N1) this season. Influenza A (H1N1) virus is the same virus that caused the 2009 influenza pandemic. The two type B viruses also circulating as seasonal influenza viruses, are named after the areas where they were first identified, Victoria lineage and Yamagata lineage.[4] Influenza occurs year-round in the tropics and during winters in the temperate countries. Influenza peaks in India typically occur with the monsoons- North, northeast and southwest regions get their peaks in July-September whereas Southeast (Chennai and Vellore) has its peak in October-November. Srinagar (Northern most city) has its peak in January-March during the winter months.[5]

Disease burden and transmission

It is estimated that influenza results in approximately 374,000 hospitalizations in infants, (228,000 in children under 6 months of age) and 870,000 hospitalizations in children <5 years globally per year. Hospitalization rates are three times higher in the developing countries.[1] Significant morbidity and mortality can occur in children under 5 years of age (especially under 2 years), children with chronic cardio-pulmonary diseases including asthma, renal, hematologic, hepatic or metabolic conditions including diabetes mellitus, neuromuscular disorders, immunosuppressed and those on long term aspirin therapy who have a risk of Reye's syndrome. Other high risk groups include pregnant women, obese individuals and the elderly (>65 years of age).

These viruses are transmitted from person to person via the droplets generated by coughing and sneezing, generally within 3 feet distance. Transmission can also occur through contact with infected surfaces like tables, toys, door handles, etc. Viral shedding typically peaks at the onset of illness and lasts for 4-5 days. It is typically longer in infants (up to 7-10 days, maximum 21 days) and in the immunosuppressed (weeks-months).[6]

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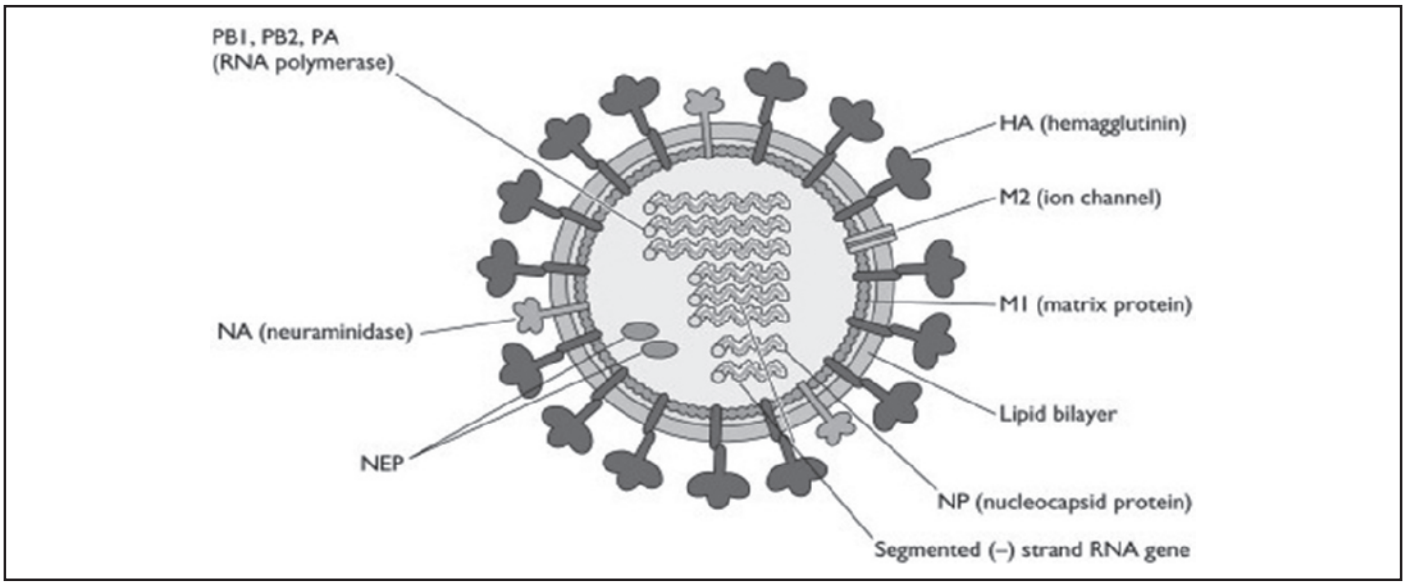
Table I. Influenza virus types

	Influenza A	Influenza B	Influenza C
Natural host	Humans, birds, swine, equine, marine mammals.	Humans only	Humans and swine
Epidemiology	Antigenic shift and drift	Antigenic drift only	Antigenic drift only
Clinical manifestations	Can cause large pandemics with significant mortality in young individuals	Severe disease in the elderly or patients with co-morbidities	Mild disease without seasonality.



NEP (nuclear export protein)

Fig.1.Structure of the influenza virus

(Source: http://www.virology.ws/2009/04/30/structure-of-influenza-virus)

Clinical features

Asymptomatic infections can occur in about 30%-60% of the children.[6] Symptomatic patients, after an incubation period of 1-4 days, typically develop an abrupt onset of high grade fever, usually with chills. They can also have non-productive cough, rhinorrhea, myalgia, headache and sore throat. Nausea, vomiting, diarrhea and abdominal pain can occur in about 30% especially with Influenza B.[6] Fever may sometimes be absent in young infants and neonates. Young infants can present as bronchiolitis, pneumonia, croup or sepsis like syndrome and neonates can have apnea or respiratory failure. Children with asthma may present with an acute exacerbation.

Complications

Complications are especially common in the high-risk groups mentioned above. The common respiratory complications include otitis media, sinusitis, viral and secondary bacterial pneumonia (typically with Streptococcus pneumoniae, Staphylococcus aureus -both MSSA and MRSA and Streptococcus pyogenes) and ARDS.

Non-respiratory complications include myocarditis, pericarditis, febrile seizures, status epilepticus, encephalopathy, encephalitis, myositis, rhabdomyolysis, mild-moderate transaminitis, Reye's syndrome with salicylate exposure and sudden death.[6] Influenza associated encephalopathy (IAE) is more common in children than adults and is typically associated with diffuse cerebral edema on neuroimaging.[7] Associated fever or respiratory symptoms may be absent in up to 40% of the children with IAE.[8] CSF is usually normal or has mild pleocytosis.[7] The pathogenesis is believed to be cytokine dysregulation and not direct viral invasion. Severe necrotising encephalopathy with bilateral thalamic necrosis is known to occur.[9] Milder forms of encephalitis with reversible

lesions (MERS) in the splenium of corpus callosum is also known.[10]

Categories

Ministry of Health and Family Welfare (MOHFW) of India has issued guidelines on clinical categorisation of patients for testing, treating and isolation puposes.[11]

Category A

Influenza like illness (ILI): Mild fever plus cough or sore throat with or without body ache, headache, diarrhea and vomiting. For patient in category A, no testing or treatment is required. Patients should confine themselves at home and avoid mixing up with public and high risk members in the family.

Category B

(i) Category A plus high grade fever and severe sore throat

(ii) Any mild ILI in

- Pregnant women
- Lung/ heart / liver/ kidney / neurological disease, blood disorders/ diabetes/ cancer / HIV-AIDS
- Patients on long term steroids/aspirin.
- Children with mild illness but with predisposing risk factors.
- Age greater than 65 years.

No testing for H1N1 is required for category-B (i) and (ii) and they can be managed at home. All patients of category-B (i) and (ii) should receive treatment with oseltamivir and should be followed up for improvement.

Category C

- Breathlessness, chest pain, drowsiness, fall in blood pressure, hemoptysis, cyanosis.
- Children with ILI (influenza like illness) with red flag signs: Somnolence, high/persistent fever, inability to feed well, convulsions, dyspnea /respiratory distress, etc.
- Worsening of underlying chronic conditions.

All these patients mentioned above in category - C require testing, immediate hospitalization and treatment.

Diagnosis

Nasopharyngeal (preferable) or throat swabs from older children and nasal or nasopharyngeal aspirates from infants are appropriate respiratory specimens for testing. Swabs should have a plastic shaft and dacron tips. Wooden shafts, cotton or calcium alginate tips must be avoided. In hospitalised and intubated children, lower respiratory tract should be tested if upper respiratory specimens are negative as the virus is shed longer in the lower respiratory tract.[12] Specimens should be transported in viral transport medium and should be kept at 4°C for no longer than 72 hours before testing and ideally should be tested within 24 hours of collection.

Molecular assays like RT-PCR or multiplex respiratory viral panels (like the Film Array respiratory panel, Biomerieux) are reliable for the diagnosis of influenza with a good sensitivity (86%-100%) and specificity. False negatives may occur especially later in the disease (> 3-4 days). Turnaround time (TAT) is around 6-8 hours.

Antigen based tests like rapid influenza diagnostic tests (RIDTs) and direct or indirect fluorescent antibody (DFA/IFA) staining have shorter TATs and good specificity (>95%) but have low sensitivity and cannot distinguish Influenza A and B. These tests are not recommended in the diagnosis of influenza by MOHFW.[12] Viral cultures have a longer TAT of 1-3 days for shell vial culture and 3-10 days for tissue culture and hence, may not be clinically relevant.

Treatment

Oseltamivir is approved by FDA for treatment in babies and children >14 days of life for treatment and children >1 year of age for chemoprophylaxis. Centers for Disease Control and Prevention (CDC) and the American Academy of Pediatrics (AAP) approve use of the drug for treatment in babies less than 14 days of life and for chemoprophylaxis above 3 months of age.[13] The dose of oseltamivir in the various weight bands is given in Table II. Dose in infancy is 3mg/kg BD. Duration of treatment is 5 days in uncomplicated cases and can be extended to 10 days.[14] Dosing adjustments need to be made for those patients with renal failure. Adverse events with oseltamivir include nausea, vomiting, serious skin reactions and sometimes transient neuro-psychiatric manifestations.

Chemoprophylaxis is recommended in high risk contacts at the above doses for 7 days.[13] Oseltamivir is safe in pregnancy and should be administered irrespective of the trimester. Severe complications are known to occur to mother in pregnancy though no vertical transmission is noted.[6] Resistance to oseltamivir is rare but can occur in the immunocompromised host.[13]

Other drugs: Inhaled zanamivir is approved but not for children with asthma / respiratory diseases. Dose for

Table II. Oseltamivir dose

Weight band	Treatment dose of oseltamivir	Chemoprophylaxis dose
<15 kg	30mg BD	30mg OD
15-23 kg	45mg BD	45mg OD
24-40 kg	60mg BD	60 mg OD
>40 kg	75mg BD	75 mg OD

treatment (>7 years) is 5 mg in each nostril twice a day for 5 days and for prophylaxis (>5 years) is 5 mg once a day for 7 days. Intravenous peramivir is not approved in age <18 years and is not found to be effective in severe disease. Both these drugs are not available in India. Amantadine and rimantadine have high resistance rates and are not useful in the treatment of influenza.

Vaccination

There are many types of influenza vaccines- trivalent split virion inactivated vaccine, live attenuated influenza vaccine, adjuvanted trivalent influenza vaccine, recombinant vaccine and quadrivalent influenza vaccine. The trivalent split virion vaccine is available and recommended in India. The vaccine is approved for age > 6 months and in pregnancy. Children under 5 years, high risk groups including pregnancy and all healthcare workers (HCWs) should be vaccinated annually, ideally in September-October in Tamil Nadu.[15] Protective antibody levels are known to drop to 50% by 6 months after vaccination and hence, it is mandatory to repeat vaccine annually. The vaccine is safe and the risk of Guillaine-Barre syndrome (GBS) is 1 in 1 million recipients and is the same as the risk of GBS in the general population. The vaccine should be administered with caution in patients with history of severe egg allergy only if expected benefits outweigh risks.[16]

Dosage and schedule

The dosing and schedule of influenza vaccines in children is given in Table III. The 2016-17 influenza vaccines contain HA derived from the following: (a) A/California/7/2009 (H1N1)-like virus, (b) A/Hong Kong/ 4801/2014 (H3N2) – like virus, and (c) a B/Brisbane/ 60/2008–like virus (Victoria lineage). The 2016–17 quadrivalent vaccines will contain the same three antigens and an additional influenza B virus HA, derived from a B/ Phuket/3073/2013–like virus (Yamagata lineage). The composition for 2016–17 represents a change in the influenza A (H3N2) virus and a switch in lineage for the influenza B viruses.

Table III. Vaccination dosage and schedule

Age	6-35 months	3-8 years	From 9 years
Dose	0.25 ml	0.5 ml	0.5 ml
No. of doses	2*	2*	1

**For previously vaccinated children, one dose should be sufficient.*

Infection control

Non-hospitalised patients: They should be advised to avoid crowded enclosed spaces and close contact with high risk groups. They should be encouraged to practise cough etiquette (maintain distance, cover coughs and sneezes with disposable tissues or clothing or cough in the crook of the elbow) and wash hands frequently.

Hospitalised patients: They should be placed in droplet and contact precautions. Healthcare workers (HCWs) should use hand hygiene, gloves and surgical mask and will require N-95 mask during suctioning and bronchoscopy. Isolation should be continued for the 7 days after the onset of illness or until 24 hours after the resolution of fever and respiratory symptoms, whichever is longer.[17] If resources are limited, patients can be cohorted and isolation rooms can be prioritized for those in the early phase of illness. A minimum distance of 3 feet between the beds with physical barriers like curtains or partitions will help to prevent the transmission in resource limited settings. Isolation precautions for longer periods should be considered in the case of young children or severely immunocompromised patients, who may shed influenza virus for longer periods of time and also, might be shedding antiviral resistant virus. Cough after influenza infection may be prolonged and may not be an indicator of viral shedding.

Points to Remember

- ***Influenza A (H1N1) is the major seasonal influenza virus.***

- ***High risk groups include children under 5 years, immunosuppressed, those with chronic medical conditions, obesity, pregnancy and those aged >65 years.***
- ***Testing for influenza is mandatory only for the hospitalized patients.***
- ***Treatment should be started without delay in the high-risk groups and the complicated cases.***
- ***Vaccination of the high-risk groups and healthcare workers should be done in September - October each year.***

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SEDATION AND ANALGESIA IN OFFICE PRACTICE

***Mullai Baalaaji AR**

Abstract: *The number of diagnostic and therapeutic procedures performed outside the operating room are increasing. Sedation and analgesia is required to control child's behaviour during the safe execution of an unpleasant procedure. The physiological characteristics of children make them vulnerable to the potential side effects of sedative agents. The key to provide safe procedural sedation involves choosing the right agent and anticipatory preparedness for any untoward event. Topical and locally acting agents along with non-pharmacologic interventions are useful adjuncts and help in reducing the sedative requirements.*

Keywords: *Pain management, Anesthesia, Analgesia.*

Children undergo a variety of diagnostic and minor therapeutic procedures outside the traditional operating theatres and it is imperative for the practicing pediatrician to have a knowledge of the unique needs of children, commonly used drugs, systematic pre-sedation assessment, intra-procedural monitoring and post procedural care.

Goals of sedation

Procedural sedation and analgesia (PSA) is the technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows safe execution of unpleasant procedures while maintaining cardiorespiratory function. The process is a continuous one, involving titrating the doses of agents to the desired effect while monitoring for the adverse effects resulting from drug overdosage. The goals of sedation are to control child's behaviour to allow safe completion of the procedure, minimise physical discomfort, anxiety and pain and return the child to a safe physiological state that allows for discharge/transfer. The ability of a child to co-operate for a particular procedure depends not only on the chronological age but also on cognitive and emotional development. The vulnerability of children's airway and protective reflexes makes them at risk for serious complications while administering sedative agents.

Levels of sedation

Sedation occurs as a continuous state from minimal sedation to general anesthesia depending on the drug, route, dosage used and patient characteristics. It is important to know the intended level of sedation to assess the level of care that is required (Table I).[1]

Pre-sedation assessment

A focussed history and physical examination are helpful to identify those who are 'at risk' for potential complications arising from sedative agents. SAMPLE is a mnemonic used for focussed history – S: signs and symptoms, A: allergy, M: medications used, P: past history, L: last meal, E: events. Pre-existing medical problems, prior adverse events to procedures/sedative agents and history of snoring also have to be detailed. Fasting recommendations before an elective sedation are shown in Table II.[2]

Assessment of airway is a key component of examination prior to PSA. Adequacy of visualisation of posterior oropharynx has been shown to correlate with visualisation of glottis during laryngoscopy. A modification of the original Mallampati score with child in supine position has been suggested in children. Other parameters to evaluate the child's airways include evaluation of mandible size, adequacy of mouth opening, size of tongue in relation to oral cavity, dentition, tonsillar enlargement, anatomical abnormalities of neck and neck mobility.[3]

Preparation for sedation

Preparation for procedural sedation involves identifying the right patient, informed written consent, arranging necessary equipments, medications and personnel. The acronym **SOAPME** is useful in planning for a procedure (Box 1). The procedure should be performed in the designated areas such as pediatric emergency department or intensive care unit.

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Table I. Comparison of different levels of sedation continuum

Physiologic function	Minimal sedation/ Anxiolysis	Moderate sedation/ analgesia	Deep sedation/ analgesia	General anesthesia
Responsiveness	Normal response to verbal stimulus	Purposeful response to verbal/tactile stimulus	Purposeful response following repeated/ noxious stimulus	Unarousable
Airway	Unaffected	No intervention needed	Intervention may be needed	Intervention often needed
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Often inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

Box 1. Preparation for sedation (SOAPME)

S - Suction: Size appropriate suction catheters and functioning suction apparatus

O - Oxygen: Supply/ delivery devices and flowmeter

A - Airway: Age appropriate bag valve mask, oropharyngeal airways, laryngoscope blades, endotracheal tubes, stylets

P - Pharmacology: Sedation, analgesic, antiemetic, resuscitation and reversal medications

M - Monitoring: SpO_2, ECG, blood pressure, end-tidal carbon-dioxide

E - Equipment/drugs for a particular case (e.g. defibrillator)

Table II. Fasting recommendations before elective sedation

Ingested food	Minimum fasting period (hours)
Clear liquids	2
Human milk	4
Infant formula	6
Nonhuman milk	6
Light meal*	6

**The amount and type of food must be considered to determine the fasting period*

Choice of agents

Knowledge about the pharmacokinetics, side effect profile of different classes of agents help in choosing the appropriate drug for a given clinical scenario. It is also important to adminster the right dosage at the right time, so that the effect peaks at the time of the unpleasant stimulus (Table III). The choice of drug depends upon the factors given in Box 2 and Table IV.

Box 2. Selection of PSA drugs

a) Intended purpose – Anxiolysis vs analgesia vs movement control

b) Desired route of administration

c) Pre-existing medical conditions

d) Familiarity of use

e) Availability/Cost

Benzodiazepines: They are one of the commonly used sedative agents in clinical practice. They are extremely potent amnestics, sedative hypnotics, but donot have analgesic properties. These agents act by augmenting γ-aminobutyric acid and glycine transmission. Midazolam is the prototype drug and can be administered transmucosal (buccal, rectal, nasal), intramuscular and intravenous routes. There is a dose-dependent depression of breathing that is potentiated with concomitant opioid administration. Significant hypotension can occur especially when administered as a rapid bolus injection in the setting of hypovolemia.

Opioids: Opioid analgesics are commonly used in combination with sedative agents during PSA. They act on mu (μ), kappa (κ) and delta (δ) opioid receptors and decrease the release of excitatory neurotransmitters from terminals carrying nociceptive stimulus. Morphine is the

Table III. Dose, onset and duration of action of commonly used sedative/analgesic agents

Drug	Dosage*	Onset of action	Duration of action
Midazolam	0.05-0.1 mg/kg	1-4 minutes	At least 30 minutes
Morphine	0.05-0.1 mg/kg	1-3 minutes	2-7 hours
Ketamine	0.5-2 mg/kg	1-2 minutes	30-60 minutes
Propofol	2-3 mg/kg	15-30 seconds	5-10 minutes
Dexmedetomidine	0.3-1 mcg/kg	10-15 minutes	At least 30 minutes
Etomidate	0.1-0.3 mg/kg	15-20 seconds	5-10 minutes
Triclofos	20-50 mg/kg (Oral)	30 minutes	8-10 hours

** Dosage is for intravenous route unless specified otherwise*

Table IV. Choice of agents for common procedures

Procedure type	Procedure	Goals of PSA	Choice of agents
Painless diagnostic	ECHO, USG, EEG, CT, MRI	Only sedation and motion control	Triclofos, midazolam
Painful diagnostic	FNAC, lumbar puncture, thoracentesis,paracentesis	Sedation, analgesia, anxiolysis and movement control	Topical / local anesthetics + midazolam / morphine / ketamine
Painful therapeutic	Incision and drainage, intercostal tube drainage, percutaneous nephrostomy	Sedation, analgesia, anxiolysis, movement control, amnesia and muscle relaxation	Topical / local anesthetics + midazolam and morphine / ketamine

prototype drug and has analgesic, sedative properties without producing amnesia. Fentanyl is about 100 times more potent than morphine and is devoid of sedative/ hypnotic action. All opioids produce side effects such as pruritus, nausea, vomiting, constipation, tolerance and dependence.

Ketamine: It is a N-methyl-D-aspartate (NMDA) antagonist that produces dissociative anesthesia, amnesia and analgesia. The minimal respiratory depressant effect and stable hemodynamic profile makes it an attractive agent in PSA. The side effects are hallucinations, myoclonic movements and excessive salivation. Other adverse effects that remain controversial include apnea in infants, laryngospasm and possible increase in intracranial pressure.

Propofol: It is an alkylphenol general anesthetic which acts by potentiating GABA-mediated synaptic inhibition. It has a rapid onset of action, dose-proportionate sedative effect without analgesia and rapid recovery time.[4] The side effects include pain on injection, negative inotropic effect and vasodilation. The risk of bacterial contamination of solution can be prevented by strict aseptic precautions and avoiding multiple use of opened vial. Prolonged infusions are not recommended due to the risk of fatal propofol infusion syndrome.

Dexmedetomidine: It is an α_2-adrenergic receptor agonist that acts on locus ceruleus, producing a state similar to natural sleep.[5] The sedative and analgesic property without significant respiratory depression has made it a popular agent for procedural sedation, nevertheless cost is a deterrent to its widespread use in our settings. The side effects include bradycardia and hypotension.

Etomidate: It is an imidazole derivative that acts by potentiating GABA inhibitory neurotransmission. It is generally devoid of adverse effects on cardiac and hemodynamic function, making it a promising agent in PSA. However, the concerns on suppression of endogenous corticosteroid production have led to widespread restriction on its use in pediatric clinical practice.[6]

Triclofos: It is an oral non-barbiturate sedative hypnotic that lacks analgesic properties. Oral usage and minimal respiratory depression have made it an ideal agent for outpatient use for non-painful procedures. Side effects include clumsiness, nausea, vomiting and skin rash.

Combination agents

A combination of drugs from different classes is commonly employed during PSA. While using more than one drug, titration of drug with lowest effective doses is of paramount importance but the potential for increased side effects have to be kept in mind. Midazolam and morphine, midazolam and ketamine are popular drug combinations during PSA.

Local anesthetics

Local anesthetics reversibly block conduction of impulses along peripheral and central neural pathways and are useful adjuncts to sedative/analgesics for painful procedures. Lignocaine is a commonly used local anesthetic, administered in a dose of 5-7 mg/kg. Vasoconstrictor, particularly epinephrine is frequently added to limit systemic absorption and lengthen the duration of sensory blockade. Care should be taken to avoid intravenous injection of local anesthetic agents. All local anesthetics are not only cardiac depressants but may also cause central nervous system excitation or depression.

Topical agents

Topical creams such as eutectic mixture of lidocaine and prilocaine produce effective skin anesthesia and can be applied under occlusive dressing one hour prior to procedures such as intravenous cannulation and lumbar puncture. Topical vapocoolant sprays delivered onto the skin just before needle insertion offers pain relief during intravenous cannulation.[7]

Non-pharmacologic interventions

Non-pharmocologic interventions such as distraction helps in reducing the procedure time as well as the number of staff required for a procedure.[8] The interventions that can be used to minimise pain are as follows:

a) Cognitive interventions: They are used in older children to direct attention away from the painful procedure. Examples include imagery, preparation and education of the child in an age-appropriate manner, coping statements, video games and television.

b) Behavioral interventions: The techniques include breathing exercises, positive reinforcement, coping behaviours, desensitisation and parental coaching.

Monitoring during sedation

The practitioner responsible for providing PSA should be skilled in resuscitation and trained in providing advanced pediatric airway support [certified in Pediatric Advanced Life Support (PALS)]. There should be supporting personnel trained in PALS to assist during resuscitation measures. The practitioners must be equipped with skills to rescue the child from a deeper level of sedation than that is intended. The name, dosage, route, time of administration of all drugs administered have to be documented by a designated person.

Baseline vital signs have to be recorded prior to administration of sedative medications. During the procedure, there should be a continuous monitoring of airway patency, respiratory rate, heart rate and oxygen saturation, which have to be documented at frequent intervals. Monitoring of ventilation using capnography is recommended especially in situations where purposeful verbal communication between the patient and provider is not possible. The child's head and neck position should be monitored to ensure airway patency. During restraint, care should be taken to avoid airway obstruction or chest restriction.[2,9]

Special situations

Neonates and former premature infants have immature hepatic and renal functions that alter drug metabolism, necessitating drug dosing modifications and extended post-procedure monitoring.[10] The increased risk of postanesthesia apnea in former preterm infants has to be borne in mind.

Children with anatomic malformations such as Pierre-Robin sequence, Treacher Collins syndrome, Apert syndrome, Crouzon syndrome, Down syndrome, micrognathia, macroglossia, tonsillar enlargement may need the help of advanced providers expertise in managing difficult airway.

Superior mediastinal syndrome needs special mention, least invasive diagnostic techniques need to be employed with avoidance of general anesthesia for diagnostic procedures. Personnel and equipment for emergency airway management including rigid bronchoscopy and tracheostomy should be available beforehand.

Post-procedure care

Children have to be monitored in a suitably equipped recovery area till they become awake. Continuous monitoring of vital signs is recommended till discharge

criteria are met. The goals during post-procedure period are stable vital signs, intact protective airway reflexes, ability to communicate at baseline, tolerate oral fluids and absence of pain.

Points to Remember

- ***Procedural sedation and analgesia (PSA) is a continuous process and involves titration of agents to the desired effect.***
- ***Pre-sedation assessment includes focussed history and physical examination of the child's airways.***
- ***The goals during PSA include sedation, anxiolysis, analgesia, motion control, amnesia and muscle relaxation and the choice of agents depends on the type of procedure and the intended need.***
- ***Careful monitoring is vital during and after sedation with appropriate documentation.***

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CLIPPINGS

Long-term effects of glucocorticoids on function, quality of life, and survival in patients with Duchenne muscular dystrophy: a prospective cohort study.

A 10 year follow up study of 440 subjects was conducted to determine the long-term impact of glucocorticoids on milestone-related disease progression across the lifespan and survival in patients with Duchenne muscular dystrophy.The results are as follows: In patients treated with glucocorticoids for 1 year or longer vs in patients treated for less than 1 month or never treated (log-rank $p<0{\cdot}0001$), time to all disease progression milestone events was significantly longer.Compared with treatment for less than 1 month, glucocorticoid treatment for 1 year or longer was associated with increased median age at loss of mobility milestones by 2.1-4.4 years and upper limb milestones by 2.8-8.0 years. In comparison with prednisone or prednisolone, deflazacort was related to increased median age at loss of three milestones by 2.1-2.7 years (log-rank $p<0{\cdot}012$).

McDonald CM, Henricson EK, Abresch RT, Duong T, Joyce NC, Hu F, et al. Long-term effects of glucocorticoids on function, quality of life, and survival in patients with Duchenne muscular dystrophy: a prospective cohort study. Lancet 2017 Nov 22. pii: S0140-6736(17)32160-8. doi: 10.1016/S0140-6736(17)32160-8.

IAP - IJPP CME 2017

FINANCIAL LITERACY FOR DOCTORS

Thirumalai Kolundu S

Doctors vulnerability

Due to our hectic work schedule, lack of time to read financial magazines and reluctance to discuss financial matters with peers, doctors seldom possess adequate financial knowledge.

Moreover we easily succumb to the pressure of the bank people and investment agents who are mostly our patients or relatives and take wrong financial calls resulting in huge loss.

In order to avoid this, we must take basic training to become financially literate and can also engage a financial adviser registered under Securities and Exchange Board of India (SEBI) to manage our finance.

Need for financial prudence

Due to extended period of education, we settle very late in our life and start to earn only at the age of 30 to 35 years. Marriage and having a child are also delayed because of this. Even then when we start our life, we go for loans to buy a car, house, to set up a clinic and to buy instruments leading to heavy debt trap which takes away most of our income as interest. To overcome all these things, we must have adequate financial knowledge to optimise our investments to get good returns.

Finance advice in a nutshell

1. Start financial planning and retirement planning at an early age as soon as we start earning and not very late in life.
2. Save with discipline and innovation.
3. Take term insurance up to 8 to 10 times of your annual income.
4. Take health insurance for Rs 5 lakhs immediately after starting to earn and increase it to minimum of 10 lakhs after getting married and make the policy as family floater.
5. Invest 6 months equivalent of your expenses in liquid funds as emergency money which can be withdrawn within 24 hours. This will earn double the interest when compared to SB account.
6. Percentage of saving equivalent to your age should be in fixed-income instruments like debt funds, PPF, tax free bonds and fixed deposits (last).
7. For income tax exemption under 80c, best options are Equity linked savings schemes (ELSS) and Public provident fund (PPF).
8. Only 5% of portfolio should be gold and that too only as gold exchange traded fund (ETF). Jewels will have only ornamental value and will not serve as good investment.
9. Buy first flat without any hesitation but think twice before buying second one. Most of the time good mutual fund earns more than the real estate.
10. Investment in mutual funds are very important and should make up 50% of our portfolio. Instead of investing in multiple mutual funds, choose 4 or 5 mutual funds and invest under systematic investment plan (SIP). Our spread in mutual funds can be like this - Large cap funds 2 numbers, diversified funds 2 numbers and one mid cap fund.
11. Investment in shares requires lot of knowledge. When we don't have time to read financial magazines, we can choose best 20 to 30 top performing companies in various sectors and buy 1 or 2 shares during every fall of SENSEX by 500 to 1000 points. Similarly we can buy Nifty BeES also during every fall of SENSEX. This will give a decent return of at least 15%. This will make us rich in a long time horizon.
12. Try to avoid high cost loans.
13. Try to use debit cards instead of credit cards. Credit cards favour uninhibited spending.

It is ideal to attain financial independence by the age of 55, so that we can enjoy life in travel, reading, writing and spending time with our family and lead a peaceful life.

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IAP - IJPP CME 2017

THROMBOCYTOPENIA

***Janani Sankar**

Abstract: *In an infant or child thrombocytopenia can occur due to large spectrum of illness ranging from tropical infection to malignancy or bone marrow failure. it is important recognize signs and symptoms to identify the underlying illness which caused thrombocytopenia. Laboratory evaluation is essential to reach the diagnosis. Management is decided by the severity of thrombocytopenia, associated risk factors and underlying illness.*

Keywords: *Platelets, Bleeding.*

Thrombocytopenia, defined as a platelet count of less than 150,000/μL, is clinically suspected when there is a history of easy bruising or bleeding in a child. It may also present as an incidental finding during routine evaluation or during investigations performed for other reasons.

The etiology for thrombocytopenia can be broadly divided as decreased production and increased destruction (Table I).

Thorough history about the duration, severity and site of bleeds, associated systemic symptoms like fever, bone pains if any, previous history of transfusions, drug intake and family history are very vital to the etiological diagnosis. Clinical examination should focus on anthropometry, dysmorphic features and cutaneous markers which narrow down the etiology to bone marrow failure syndromes and findings such as lymphadenopathy and organomegaly points to an infiltrative disorder.

Lab investigations

In the complete blood count not only the platelet count and mean platelet volume (MPV) but also evidence of any other cytopenias (ie, anemia and leukopenia) should be noted. In a child with thrombocytopenia, a mildly elevated MPV is consistent with a destructive etiology, including ITP. A MPV that is considerably higher than normal suggests one of the macro thrombocytopenia syndromes (eg, Bernard-Soulier syndrome or MYH9-related disorders). A low MPV (3 to 5 fL) is typically seen in patients with Wiskott–Aldrich syndrome (WAS) and X-linked thrombocytopenia. These abnormalities should be further evaluated by visual examination of the peripheral blood smear as since both platelet counts and calculated MPV from automated cell counters can be inaccurate when platelet size is outside the reference range.

The peripheral blood smear must be carefully examined to estimate the platelet number, determine the platelet morphology presence or absence of platelet clumping and also to assess if there are associated white and red blood cell abnormalities.

Circulating blast cells suggest a leukemic process. Blast cells on the peripheral blood smear may be difficult to distinguish from atypical lymphocytes that are sometimes present in a postviral case of ITP.

Fragmented erythrocytes (schistocytes) suggest a microangiopathic process, such as disseminated intravascular coagulation (DIC), hemolytic-uremic syndrome, or thrombotic thrombocytopenic purpura. Spherocytes may suggest autoimmune hemolytic anemia coupled with autoimmune-mediated thrombocytopenia (Evans syndrome) In these patients, the direct antiglobulin test (DAT, also known as the Coombs test) will be positive. Less commonly, autoimmune-mediated neutropenia may also occur.

Bone marrow examination

In most cases of isolated unexplained thrombocytopenia in children, a bone marrow examination is not required in the initial evaluation unless there are clinical features that suggest bone marrow infiltration or failure.

A bone marrow examination is indicated if any of the following findings, which are suggestive of either marrow infiltration with abnormal cells or marrow hypocellularity are present -

(i) Evidence of involvement of other blood cell lines

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Table I. Thrombocytopenia in children - Etiology

Destructive Thrombocytopenias	Decreased platelet production
Immune mediated Immune thrombocytopenia (ITP)* Drug-induced thrombocytopenia Systemic lupus erythematosus (SLE) Infection such as malaria, scrub typhus, leptospirosis Neonatal immune thrombocytopenias Post-transplant thrombocytopenia	Infection: Epstein bar virus, cytomegalo virus, varicella, bacterial infection. Nutritional deficiencies (Folate, B 12, Iron) **Acquired bone marrow failure** Aplastic anemia, drugs, viral infections, Chemotherapy, radiation myelodysplastic syndromes
Non-immune Hemolytic-uremic syndrome Thrombotic thrombocytopenic purpura Extracorporeal therapies (eg, cardiopulmonary bypass) Congenital or acquired heart disease Disseminated intravascular coagulation Kasabach-Merritt syndrome Hypersplenism Hypothermia Type 2B Von Willebrand disease	**Infiltrative bone marrow diseases** Acute leukemias, lymphoma, metastatic cancers, infectious granulomas, storage diseases **Genetic causes of impaired thrombopoiesis** Wiskott-Aldrich syndrome/X-linked thrombocytopenia Inherited bone marrow failure syndrome Fanconi anemia Dyskeratosis congenita Swachmann Diamond syndrome Congenital amegakaryocytic thrombocytopenia (CAMT) Thrombocytopenia with absent radii (TAR) syndrome Congenital amegakaryocytic thrombocytopenia with radioulnar synostosis Familial platelet disorder with predisposition to myeloid malignancy Bernard-Soulier syndrome *MYH9*-related disorders Paris-Trousseau syndromeX-linked thrombocytopenia with dyserythropoiesis

Source : Donald LY, Donald HM, Leung LLK, Armsby C. Approach to the child with unexplained thrombocytopenia. UpToDate. Topic 5939 Version 19.0 last updated: Jul 10, 2015.

(e.g. anemia or leukopenia) in the absence of a likely alternative explanation.

(ii) Presence of blasts on peripheral blood smear.

(iii) Chronic, stable thrombocytopenia even when the presumed diagnosis is ITP as it is a diagnosis of exclusion.

(iv) Systemic symptoms (e.g. fever, weight loss, bone pain) that are consistent with an underlying malignancy

In patients with ITP, the bone marrow will be cellular, and the erythroid and myeloid precursors normal in number and appearance. The megakaryocytes are typically normal or increased in number, and may appear large and/or immature. By contrast, in patients with leukemia the normal cellular components of the bone marrow are partially or almost totally replaced by immature or undifferentiated cells. In bone marrow failure syndromes there is marrow hypocellularity, with decreased megakaryocytes.

Other tests

For children with evidence of hemolytic anemia on the peripheral blood smear (eg, spherocytes or schistocytes), the additional test are required. A direct antiglobulin (DAT, also known as a direct Coombs test) is done to detect an autoimmune hemolytic anemia. D-dimer and fibrinogen levels are useful for diagnosis of intravascular coagulation in children with schistocytes on the peripheral blood smear and/or vascular tumors on exam. Other tests to evaluate for a microangiopathy include serum lactic dehydrogenase, creatinine and ADAMTS13 activity.

Children with chronic thrombocytopenia or with features suggesting inherited bone marrow failure

syndromes (e.g. pancytopenia with short stature, café au lait spots) should undergo specific testing for these disorders in addition to a bone marrow biopsy. For example, the presence of increased chromosomal breakage in lymphocyte cultures with DNA cross-linking agents such as mitomycin C is diagnostic of Fanconi anemia.

Reticulated platelets - Although not yet widely available in clinical laboratories, many reports indicate that quantification of reticulated platelets (RP) may provide useful information for differentiating between disorders of platelet destruction and production. Analogous to red blood cell reticulocytes, RP consist of the larger, younger, more recently released platelets containing mRNA and reflect increased platelet turnover. RP measurem

End methods have not been standardized, but usually have involved flow cytometric techniques utilizing fluorescent dyes such as thiazole orange to stain mRNA. Successful validation and standardization of reliable, automated methods for measuring RP are anticipated as a future aid in the evaluation of thrombocytopenia.

Management

Bleeding risk and thrombocytopenia: Risk of spontaneous bleeding is high when the platelet count is < 10,000. For the same count risk is higher in thrombocytopenia due to decreased production as in bone marrow failure or leukemia. But the risk is less when it is caused by peripheral destruction because of the presence of younger healthy circulating platelets as in ITP.

Once the etiological diagnosis is made the treatment becomes individualised.

General measures to reduce bleeding: Restriction of activities, avoiding contact and collision sports, avoiding trauma, wearing helmets in ambulant children and avoiding drugs like NSAID are advocated. Antifibrinolytics and hormone therapy are used in adolescent girls to prevent blood loss due to menstrual bleed. Monitoring and educating the parents is an important component. The management of thrombocytopenia is dependent on etiology. Platelet concentrates are usually reserved for children with either severe thrombocytopenia or a bleeding sick child. The management of commonly encountered four disorders are discussed.

1. Dengue: Thrombocytopenia in dengue is not aggressively corrected. Platelet transfusion is often not decided on platelet count alone. It is indicated only in three situations. a) Adolescent girl with thrombocytopenia and menstrual bleed, b) Prior to procedures like central venous catheterization to maintain a safer count of 50,000. c) In an asymptomatic child when the platelet count is <10,000 where there is a risk of spontaneous bleed. Here again transfusion is not given if the child is hemodynamically stable and in the recovery phase.

2. In leukemia: Transfusion threshold is lower in a sick child with acute leukemia and there is a risk of spontaneous bleed.

3. Immune thrombocytopenic purpura: Acquired thrombocytopenias usually respond to intravenous gamma globulin and sometimes steroids in select situations. Clear protocols are available for the management of a child with ITP. It is decided by the severity of bleeding and degree of thrombocytopenia, individual patient characteristics and associated risk factors.

In the presence of life threatening bleeding such as cardiopulmonary compromise or intracranial bleed, resuscitation and immediate intervention is needed including one or the combination of the following - platelet transfusion, intravenous immunoglobulin or anti D immunoglobulin.

In non life threatening bleeding: There is no role of platelet transfusion. IVIG can be safely used in an emergency if bone marrow examination is not planned as it does not affect the bone marrow findings in leukemia. Anti D Immunoglobulin is used as an alternative in a child with rhesus positive blood group. Steroids, Dexamethasone or prednisolone is used when bone marrow examination is normal.

Low bleeding risk: Children with only skin bleed or no bleeding may not need any pharmacologic intervention and watchful waiting is the plan.

4. Inherited bone marrow failure syndromes are usually treated with stem cell transplantation.

Points to Remember

- ***Thrombocytopenia is defined as a platelet count of less than 150,000/microL. Spontaneous bleeding does not usually occur until the platelet count is less than 20,000/microL.***
- ***Clinical presentation with cutaneous (eg, petechiae, non-palpable purpura, ecchymoses) and/or mucosal (eg, epistaxis, gingival bleeding, bullous hemorrhage, menorrhagia) bleeding are common while intracranial hemorrhage (ICH) is rare.***

- ***Thrombocytopenia with systemic symptoms and/or the presence of lymphadenopathy or hepatosplenomegaly should raise suspicion for malignancy or and should be evaluated expeditiously.***
- ***Peripheral blood smear must be carefully examined for estimation of platelet number, morphology, presence or absence of platelet clumping and evaluation for associated white and red blood cell abnormalities.***
- ***Bone marrow examinations generally are not required for the initial evaluation in most cases of unexplained isolated thrombocytopenia in children.***

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NEWS AND NOTES

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and

32nd SOUTH ZONE CONFERENCE

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IAP - IJPP CME 2017

LATE TALKING TODDLER-WHEN TO WORRY?

Somasundaram A

Abstract: *Late talkers are children who exhibit delay in language onset with no other diagnosed disabilities or developmental delays in other cognitive or motor domains. The primary objective is to differentiate them from other causes of speech and language delay. They may present with expressive language delay only or mixed expressive and receptive delay. Family history, male gender, lack of communicative interaction between parents and children are the known risk factors for late talking. It is appropriate to adopt 'watchful waiting' strategy with late talkers who have good comprehension and without any family history of language problems.*

Keywords: *Late talkers, Expressive speech delay.*

'Late talkers' is a term used in the scientific field of atypical language development to describe toddlers who exhibit delay in expressive language skills, although they do have intact receptive skills with no other deficits, such as cognitive, neurological, socio-emotional or a sensory deficit.[1] Late talking does not define a clinical disorder, but may be a stage in the language development of a child or a symptom of an evolving disorder.

Late talking may be an early or secondary sign of disorders, such as specific language impairment, social communication disorder, autism spectrum disorder, learning disability, attention deficit hyperactivity disorder, intellectual disability or other developmental disorders. In order to make a differential diagnosis, it is critical to monitor the global development of a child in domains that include, but are not limited to, cognitive, communication, sensory and motor skills. The synonyms of 'late talkers' is given in Box 1. Some researchers distinguish a subset of children with LLE as "late bloomers". They infer that late bloomers catch up to their peers in language skills by 3 to 5 years of age.

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Box 1. Late talkers – Synonyms

Children with expressive language delay (It is delay not disorder)

Late language learners

Late language emergence (LLE)

Maturational speech delay

Einstein syndrome

Normal language development trajectory

All human languages have rules which determine their grammar, tune or music ("prosody") and the range of sounds used. Babies absorb these rules through a natural process of hearing and learning. How infants and children master the rules of language is one of the miracles of nature which is still not understood well till now. The normal language developmental milestones are summarized in the Table I.[2] The red flag signs suggesting a need for immediate evaluation are given in Table II.[3]

Risk factors

i) Male gender: A large-scale study by Zubrick, Taylor, Rice, and Slegers (2007) found that male children are three times more likely to have language delay compared with females. Horwitz et al. (2003) found that males between 12 and 17 months tend to have statistically significantly lower expressive language performance than girls.

ii) Heritability: Family history of language impairments appears to be one of the main risk factors for the presence of delayed language emergence in children.

iii) Communicative interaction between parents and late talkers: Parental language input to late talkers is quantitatively similar to input to children with typical language development. Parents of children with typical language development and parents of children with language delay produce similar language in terms of the number of utterances and the number of words. Nevertheless, in terms of quality of interaction, it seems that parents of late talkers respond less often to their children compared to parents of children with typical language development.

Table I. Speech and language - Developmental milestones

Age	Receptive	Expressive
0-2 months	Turns to sound Prefer voices Interested in faces	Cries
2-4 months	-	Coos
6 months	Responds to name	Babbles
9 months	Understands verbal routines	Points
12 months	Follows a verbal command	Jargon First words
15 months	Points to body parts by name	Learning new words slowly 10-20 words
18-24 months	Understands sentences	Learning new words quickly 50-100 words Uses two-word phrases
24-36 months	Answers questions Follows two-step commands	Uses three-words phrases Asks “what” questions 50% intelligible
36-48 months	Understands much of what issaid	Ask “why” questions 75% intelligible
48-60 months	Understands much of what issaid, commensurate withcognitive level	Creates well-formed sentences Tells stories 100% intelligible

Table II. Red flags suggesting need for immediate speech-language evaluation

Age	Receptive	Expressive
12 months	-	Does not babble, point, or gesture
15 months	Does not look at or point to 5 to 10 objects or persons when named by parents	Does not use at least three words
18 months	Does not follow one-step directions	Does not say “mama,” “dada,” or other names
2 years	Does not point to pictures or body parts when named	Does not use at least 25 words
2.5 years	Does not verbally respond or nod/shake head to questions	Does not use unique two-word phrases, including noun-verb combinations
3 years	Does not understand prepositions or action words	Does not use at least 200 words
-	Does not follow two-step directions	Does not ask for things by name
-	-	Repeats phrases in response to questions (echolalia)
At any age	-	Has regressed or lost previously acquired speech/ language milestones

iv) Parental stress: Children growing in an environment characterized by high levels of parental stress concerning a child's language difficulties are more likely to exhibit poor expressive language. It is worth noting here that mothers of late talkers in their reports evaluate their relationship with their late talking child as very stressful.

v) Socio-economic status of the family: Children who come from families characterized by a low educational level and poverty are more likely to experience delays and difficulties in expressive language. Highly educated mothers are more likely to use rich vocabulary and speak in longer utterances when interacting with their children. It is evident that factors such as mother's education, socioeconomic status, parental occupation, parenting style or even parental mental health issues (for example, maternal depression) have an impact on a child's likelihood of being a late talker.

Language profile of late talkers

Vocabulary: Late talkers appear to delay about 12 months in lexical acquisition compared to typically developing children. It has been known that late talkers actually use more communicative gestures than their typically developing peers. This is probably because late talkers cannot temporarily rely on their expressive language skills, and thus prefer using communicative gestures.

Linguistic outcome of late talkers

Some children recover and have comparable expressive language skills with their same-age typically developing peers. A significant number of children are expected to perform within the mean or above the 10th percentile on expressive language measures, whereas a smaller number of children continue to experience expressive language difficulties. They are at high risk for language disorders, learning disabilities, poor academic outcomes, social or behavioural problems and declining IQ as the child ages.[4]

Language delay-How to identify from other causes?[2]

The causes of speech and language delay can be primary or secondary. Primary speech and language delay include developmental speech and language delay, expressive language disorder and receptive language disorder. Secondary speech and language delay are attributable to other conditions such as hearing loss, intellectual disability, autism spectrum disorder, physical speech problems or selective mutism.

Primary

Developmental speech and language delay: Speech is delayed. Children have normal comprehension, intelligence, hearing, emotional relationships and articulation skills.

Expressive language disorder: Speech is delayed. Children have normal comprehension, intelligence, hearing, emotional relationships, and articulation skills. Expressive language disorder is difficult to distinguish at an early age from the more common developmental speech and language delay.

Receptive language disorder: Speech is delayed and also sparse, agrammatic and indistinct in articulation. Children may not look at or point to objects or persons named by parents (demonstrating a deficit in comprehension).Children have normal responses to nonverbal auditory stimuli.

Secondary

Autism spectrum disorder: Children have a variety of speech abnormalities, including speech delay (especially with concurrent intellectual disability), echolalia (repeating phrases) without generation of their own novel phrases, difficulty initiating and sustaining conversations, pronoun reversal and speech and language regression. Children have impaired communication, impaired social interaction, and repetitive behaviors/circumscribed interests.

Cerebral palsy: Speech delay in children with cerebral palsy may be due to difficulty with coordination or spasticity of tongue muscles, hearing loss, coexisting intellectual disability or a defect in the cerebral cortex.

Childhood apraxia of speech: Apraxia of speech is a physical problem in which children have difficulty making sounds in the right order, making it hard for their speech to be understood by others.Children communicate with gestures but have difficulty with speech (demonstrating motivation to communicate, but lack of speech ability).

Dysarthria: Dysarthria is a physical problem in which children can have speech difficulties ranging from mild, with slightly slurred articulation and low-pitched voice, to profound, with an inability to produce any recognizable words. Children communicate with gestures but have difficulty with speech (demonstrating motivation to communicate, but lack of speech ability).

Hearing loss after spoken language established: Speech and language are often gradually affected, with a decline

in the precision of speech articulation and a lack of progress in vocabulary acquisition. Parents may report that the child does not seem to be listening, or describe the child speaking better than listening.

Hearing loss before onset of speech: Speech is delayed. Children may have distortions of speech sounds and prosodic patterns (intonation, rate, rhythm, and loudness of speech). Children may not look at or point to objects or persons named by parents (demonstrating a deficit in comprehension). Children have normal visual communication skills.

Intellectual disability: Speech is delayed. Use of gestures is delayed, and there is a generalized delay in all aspects of developmental milestones. Children may not look at or point to objects or persons named by parents, demonstrating a deficit in comprehension.

Selective mutism: Children with selective mutism show a consistent failure to speak in specific social situations in which there is an expectation for speaking [e.g., at school] despite speaking in other situations.

Box 2. Interaction and information strategies

A) Interaction strategies

- Be face-to-face (Don't be an arm chair parent). Being at your child's level means he/she can see your face and mouth when you speak
- Establish eye contact
- Observe and listen
- Wait - to see the child's interest
- Allow your child to lead
- Take a turn

B) Information strategies

- Talk slowly
- Short sentences
- Repeat and/or emphasize key words
- Modelling
- Expansion - Use the **REPEAT + 1 WORD** rule (repeat the word the child has said and add another word to it)
- Asking questions

Box 3. 4S strategy

(i) Say less

Short sentences

Simplify messages

(ii) Stress

Emphasize key words

Vary and exaggerate tone of voice

Repeat

(iii) Go slow

Speak slowly

Use longer pauses between words

(iv) Show

Talk about familiar things / here-and-now

Use objects or point to the object

Screening and assessment

Parents of children with symptoms of delayed speech and language development usually approach a clinician when the child is 18-36 months old. In such cases any concern that something may be wrong with the child should be taken seriously, as parents' observations of abnormal behaviour in children at this age are quite accurate.

History should include information about consanguinity, family history of hearing loss, family history of late talking, birth history including prenatal (medications, radiation, TORCH), natal (gestation, weight, APGAR) and neonatal history (NICU stay, hyperbilirubinemia, seizures, meningitis, ototoxic drugs), environmental factors (e.g., amount of language stimulation), trauma to ear / ear discharge and developmental milestones history.

During examination the child should be observed while interacting with parents and also when the child plays. Dysmorphic features, growth abnormality, neurocutaneous markers should be noted. ENT examination along with neurological examination including cranial nerves, motor system and reflexes should be done.

Diagnostic workup includes audiometry, MRI/CT brain, karyotyping, IQ assessment and screening and subsequent assessment for language using standardised tools. Screening for speech and language delay can be done using early language milestone scale 0-3 years or Language Evaluation Scale Trivandrum (LEST). LEST 0-3 and 3-6

years is a simple tool developed by CDC Kerala. Receptive–Expressive Emergent Language Scale (REELS) is designed to identify receptive and expressive language problems in children up to 3 years.

Interventions for late talkers

Delay in speech and language development in children should be detected as early as possible to ensure optimal treatment and to prevent psychiatric problems later in life. This process of detection and treatment should be multidisciplinary, involving the parents, school teacher, pediatrician, pediatric neurologist, ear, nose, and throat specialist, child psychiatrist, child psychologist, linguist and speech pathologist.[5]

Interaction and information strategies help stimulate speech and language (Box 2) and to make the language easy to understand one needs to use the "4S" (Box 3).

Late talking toddlers can create anxiety to parents. In the majority of cases, there's no need for alarm. It is the duty of the paediatrician to examine the child in detail and ensure that there are no red flags needing immediate speech and language evaluation. Most children do develop language at their own pace.

Points to Remember

- ***Majority of late talkers do not have later language difficulties.***
- ***A family history of language/literacy problems is a risk factor for persisting problems.***
- ***A good clinical evaluation of the central nervous system, ear, nose and throat and audiometry are mandatory in all cases of suspected speech and language delay.***
- ***Almost all kids with autism are late talkers - but not all late talkers have autism.***
- ***'Watchful waiting' strategy can be adopted in late talkers who have good comprehension and without family history of language problems.***

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NEWS AND NOTES

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ELEVATED TRANSAMINASES IN A CHILD - APPROACH

Malathi Sathiyasekaran

Abstract: *Elevated transaminases or transferases implies that the levels of serum glutamic pyruvic transaminase (SGPT) or alanine aminotransferase (ALT) and serum glutamic oxaloacetic transaminase (SGOT) or aspartate aminotransferase (AST) are more than twice the upper limit of normal and indicate hepatocyte injury. This may be detected incidentally in an asymptomatic child or be present in a symptomatic child with liver disease. The level of transaminases may be elevated both in acute and chronic hepatitis but is not an index of prognosis. In acute hepatitis the level may indicate the etiology and recovery whereas in chronic hepatitis it is used as a surrogate marker to monitor therapy.*

Keywords: *Transaminases, Transferases, Hepatocyte injury.*

Transaminases or aminotransferases are catalyst enzymes which are detected normally in low levels in the serum (1-40U/L). The two transaminases are serum glutamic-pyruvic transaminase (SGPT) and serum glutamic oxaloacetic transaminase (SGOT) which are now designated as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) respectively. These enzymes help in the transfer of the α amino groups of alanine and aspartic acid to the α-keto group of ketoglutaric acid resulting in the formation of pyruvic and oxaloacetic acid (Box 1).

The transaminases or transferases are sensitive indicators of hepatocyte injury. Though they are grouped as liver function tests it is a misnomer since they do not detect functions of the liver.

Significance of SGOT and SGPT

SGPT is more liver specific and is present in the cytosol of the hepatocyte. SGOT is not liver specific and is present in several tissues such as liver, cardiac muscle, skeletal muscle, kidney, brain, pancreas, lung, leukocyte and erythrocytes. In the hepatocyte, SGOT is present mainly in the mitochondria (80%) and only 20% in the cytosol. When both enzymes are elevated and if SGPT is more than SGOT it would indicate cytosol injury as seen in viral hepatitis. Whereas if SGOT is more than SGPT, it may indicate mitochondrial injury as seen in drug induced or toxic hepatitis. Isolated elevation of SGOT may be due to muscle disorders or cardiac disease. Transaminases are considered as elevated when they are more than twice the upper limit of normal (ULN). In a child with jaundice the term hepatitis is used only when the transaminases are elevated.

Transaminase levels

The normal level of transaminases varies with age, gender, BMI, exercise and even the time of collection. The usual lab value ranges between 0-40 U/L and in day to day practice if the value is more than twice ULN, it is considered as elevated. However, several population based studies have documented that the upper limit of normal is much lower than 40 U/L. A landmark study done in 2002 by Prati et al., in 6835 adult blood donors have reported the 95th centile for SGPT in male donors as 30 U/L and for female donors as 19 U/L.[1] If this value is accepted as the upper limit of normal then even 40 U/L would be considered as elevated. In children and adolescents, studies have shown different values. England et al., have reported that in 1293 HCV uninfected children, the 95th centile in boys was 60 U/L and in girls 55U/L below 18 months of age.[2] In the above 18 months group the 95th centile in boys was 40 U/L and in girls 35U/L. The SAFETY (Screening ALT for Elevation in Today's Youth) study in 982 adolescents of 12-17 years by National Health and Nutrition Examination Survey (NHANES) was published in 2010 by Schwimmer et al where the 95th centile for SGPT in boys was 25.8 and in girls 22.1U/L.[3] Since the value differs in the various studies, the normal range in the laboratory where the blood is tested is taken to determine the cut off value.

Elevated transaminases - Approach

Children with elevated transaminases- SGPT more than twice ULN on two occasions 4 weeks apart should be subjected to detailed evaluation. Elevated SGPT may be

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Box 1. Transaminases and transfer of α amino group

Alanine + ketoglutaric acid $\xrightarrow[(ALT)]{SGPT}$ L–glutamic acid + pyruvic acid

L-aspartic acid + ketoglutaric acid $\xrightarrow[(AST)]{SGOT}$ L-glutamic acid + oxaloacetic acid

Box 2. Elevated transaminases in asymptomatic children - Common causes

- Chronic hepatitis - HBV, HCV
- Non alcoholic fatty liver disease (NAFLD)
- Wilson disease
- Glycogen storage disorder
- Drug induced liver disease (DILI)
- Autoimmune hepatitis

seen in an asymptomatic child (incidentally detected) or symptomatic child. It is essential that a detailed history, a complete clinical examination, careful perusal of available reports is done before subjecting asymptomatic or symptomatic child to further investigations.

I. Asymptomatic child with elevated transaminases[4]

Detailed history: A detailed history regarding jaundice in the past, blood transfusions, recurrent needle pricks, medications, family history of liver disease and dietary habits should be noted in detail.

Examination: The child's weight, height and BMI should be recorded. Acanthosis nigricans, if present, indicates insulin resistance and is seen in non-alcoholic fatty liver disease (NAFLD) whereas Kayser-Fleischer ring is an excellent clue for Wilson disease. The abdomen should be examined in detail and the size of the liver and its consistency should be noted. Splenomegaly is usually not a finding in NAFLD and GSD I but may be present in all the other conditions listed in Box 2.

Investigations: Careful perusal of available investigations is essential before performing new tests. Biochemical tests of the liver-serum bilirubin, alkaline phosphatase, gamma glutamyl transpeptidase and prothombin time are done. All children should have an ultrasound of the abdomen to check the liver size, echotexture and the grading of fatty liver. Specific investigations are shown in Table I. These conditions require definitive treatment even if the child is asymptomatic but presents with elevated transaminases. A simplified treatment protocol is shown in Table II.

II. Symptomatic child with elevated transaminases

The same approach to elevated transaminases as in an asymptomatic child should be done for a symptomatic child.

Detailed history: A detailed history should be elicited regarding fever, nausea, vomiting, jaundice, duration of illness, itching, stool and urine color, abdominal pain, fluid retention, urine output, loss of appetite, gastro intestinal bleed, altered sensorium, similar history in family or neighbourhood, blood transfusion, needle pricks, past history of jaundice, school performance, medications, rash and joint problem will give a clue to the type of jaundice as to whether it is hepatocellular or cholestatic. The duration of illness if more than 6 months is usually considered as chronic. But in children 3 months is accepted as cut off for chronicity.

Clinical examination: Icterus, scratch marks, liver size and consistency, presence of free fluid and splenomegaly should be looked for. Eyes should be examined for KF ring. Presence of firm liver with or without splenomegaly is a marker for chronic liver disease.

Investigations: All available investigations should be carefully scrutinized. A low hemoglobin may indicate blood loss or nutritional deficiency. A low platelet count may be a feature of cirrhosis. Bicytopenia or pancytopenia may give a clue to hemophagocytic lymphohistiocytosis (HLH). Serum bilirubin with fractionation, alkaline phosphatase, albumin, gamma glutamyl transpeptidase and prothrombin time should be studied. A low albumin and prolonged prothrombin time may indicate chronic liver disease with impaired function. All symptomatic children should have an ultrasound of abdomen and the details of liver echotexture and presence of ascites should be noted.

Specific investigations: Viral serology-HBsAg should be done and if positive anti HBc IgM has to be done in those with acute hepatitis. If the history and clinical findings are suggestive of chronic hepatitis HBsAg, HBeAg, anti HBe and HBV DNA, anti HCV and HCV RNA should be done. If there are features of infective non viral hepatitis, work up for typhoid, rickettsia and leptospirosis should be done.

Table I. Investigations in asymptomatic children with elevated transaminases

Disease	Investigations
Chronic HBV infection	HBsAg, HBeAg, anti HBe, HBV DNA (viral load), liver biopsy
Chronic HCV infection	Anti HCV, HCV RNA (Viral load)
NAFLD	Blood sugar, lipid profile, fibroscan, liver biopsy
Wilson disease	Serum ceruloplasmin, 24 hour urine copper and liver biopsy
Glycogen storage disease	Fasting blood sugar, triglycerides, lactate, uric acid, liver biopsy
Autoimmune liver disease	ANA, ASMA, globulin profile, IgG, liver biopsy

ANA – Antinuclear antibody; ASMA – Anti smooth muscle antibody

Table II. Treatment in asymptomatic children with elevated transaminases

Disease	Treatment
Chronic HBV infection	Peg IFN and antivirals entecavir, tenofovir
Chronic HCV infection	Peg IFN + ribavirin or oral direct acting antivirals (DAAs)
NAFLD	Regular exercise, weight reduction, vitamin E
Wilson disease	D pencillamine
Glycogen storage disease	Uncooked corn starch
Autoimmune liver disease	Steroids / azathioprine

Peg IFN – Pegylated interferon

Table III. Elevated trasaminases in symptomatic children - Etiology

Acute	Chronic
I. Infective hepatitis: viral (HAV, HEV, HBV, HCV, EBV, CMV) bacterial (typhoid), spirochetal (leptospirosis), protozoal (malaria)	I. Infections: HBV, HCV
II. Non infective	II. Non infective
Medications	Metabolic: Wilson's
Metabolic: Wilson's, glycogen storage disease	Autoimmune hepatitis
Autoimmune: Type I, 2	Medications
Vascular	Vascular
Malignancy	Malignancy
Hemophagocytic lymphohistiocytosis	

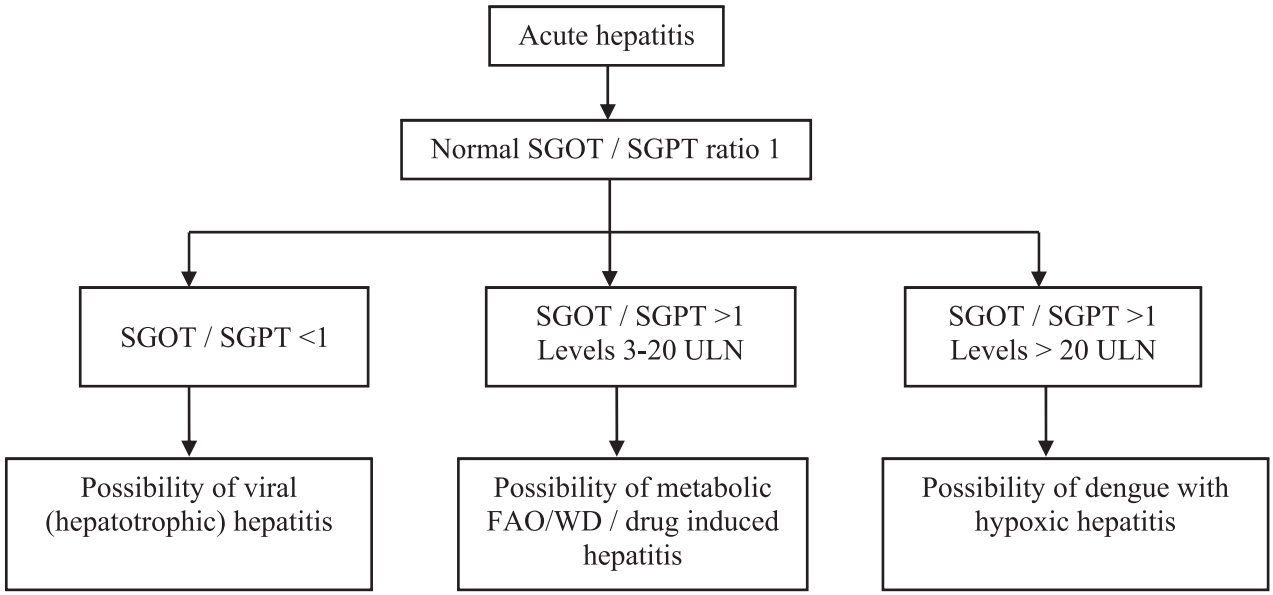

Fig.1. Approach to a symptomatic child with acute hepatitis

Table IV. Etiology according to level of transaminases

Mild (2-3 elevation)	Moderate (>3-20 elevation)	Marked (>20 elevation)
Viral infection	Bacterial hepatitis(typhoid)	Viral (hepatotrophic virus)
Sepsis	Spirochetal (leptospirosis)	Hypoxic (DSS, CCF)*
Cholestasis	Metabolic (Wilson,GSD)*	Drug induced hepatitis*
Liver abscess	Biliary obstruction	Autoimmune hepatitis
Non alcoholic steato hepatitis	Autoimmune hepatitis	

GSD: Glycogen storage disease, DSS: Dengue shock syndrome, CCF: Congestive cardiac failure

** Mitochondrial injury: SGOT > SGPT*

Autoantibodies, ceruloplasmin etc., may be necessary to exclude autoimmune hepatitis (AIH) and Wilson disease (WD). Upper GI endoscopy should be done in a child with features of chronic liver disease to exclude portal hypertension. If there is ascites a diagnostic tap and analysis is necessary.

The hepatic causes of elevated transaminases in a symptomatic child which may be due to acute hepatitis or chronic hepatitis as shown in Table III. The important non hepatic causes of elevated transaminases in a child are celiac disease and muscular dystrophy.

a) Acute hepatitis

Level of transaminases: According to the level of enzymes elevated transaminases can be divided as mild, moderate or marked as mild 2-3 ULN, moderate >3-20 ULN, marked >20ULN. The various causes of hepatitis can be arbitrarily categorized into the three groups as shown in Table IV. Marked elevation of transaminases is seen in viral hepatitis due to the hepatotrophic viruses, toxic hepatitis as seen in drug induced liver injury (DILI) and hypoxic hepatitis as seen in dengue shock syndrome. In the latter there is a simultaneous increase in LDH and the transaminases which will decrease as soon as the child is stabilized which is not a feature in viral hepatitis.

Ratio of SGOT /SGPT: The normal SGOT /SGPT ratio is 1 and if it is more than 2, a mitochondrial injury should be suspected as seen in dengue shock, metabolic liver disease e.g. Wilson disease and drug induced hepatitis (paracetamol). This change in ratio is not a feature in the

Table V. Usefulness of elevated transaminases in acute and chronic liver disease

	Acute liver disease	Chronic liver disease
Diagnosis	Confirms hepatitis Levels and SGOT / SGPT ratio may give clue in etiological diagnosis	Confirms hepatitis Levels and SGOT / SGPT ratio do not help in etiological diagnosis
Monitoring	Decreases with recovery; beware may also indicate massive necrosis	Helps in monitoring therapy
Prognosis	No	No
Therapy	Specific treatment for underlying etiology. Fall in levels supports response to therapy.	Specific treatment for underlying etiology. Fall in levels supports response to therapy

classical viral hepatitis where the SGPT is markedly elevated because the injury is more in the cytosol. The level of transaminases and the SGOT / SGPT ratio may give some clue to the etiology which can then be confirmed with definitive tests as shown in Fig.1 and Table IV.

Serial transaminases in acute hepatitis: In a child with acute viral hepatitis a fall in the transaminases may indicate recovery if the serum bilirubin is also decreasing and the prothrombin time is normal. However, a rapid fall in transaminases with rising serum bilirubin and prolonged prothrombin time may indicate hepatocellular necrosis and acute liver failure. Therefore the level of transaminases is not a useful prognostic index in acute hepatitis and is not included in the prognostic indices of acute liver failure.

b) Chronic hepatitis

The causes of chronic hepatitis are shown in Table III.

Level of transaminases: In chronic hepatitis there is a mild or moderate elevation of transaminases. In the majority they are usually 3 to 5 times the ULN. A value more than 5 times ULN with elevated bilirubin more than 5 mg /dL may indicate acute on chronic liver failure. In cirrhosis the transaminases may be normal indicating that the level of transaminases does not help in prognostication in acute as well as in chronic liver disease. In chronic HBV infection the elevated transaminases helps in classifying the phase of chronic infection. The transaminases are normal in immune tolerance and inactive HBsAg carrier phase.

Level of transaminases and therapy: In chronic HBV infection at present only those with elevated transaminases more than twice ULN should be treated. In chronic HCV, the transaminases may be normal and child is treated based on the HCV RNA and the fibrosis score on fibroscan. The transaminases therefore are good surrogate marker for response to therapy in chronic HBV infection and autoimmune hepatitis. In end stage liver disease the pediatric end-stage liver disease (PELD) score is used for registering for liver transplant which does not include these enzymes as indices.

In conclusion, the role of elevated transaminases in acute or chronic liver disease in relation to four parameters-diagnosis, monitoring, prognosis and therapy are shown in Table V.

Points to Remember

- ***Elevated transaminases are markers of hepatocyte injury and do not indicate liver function.***
- ***A child with elevated transaminases whether asymptomatic or symptomatic needs to be evaluated in detail.***
- ***In acute hepatitis elevated transaminases may give some clue to etiology and indicate recovery when the level decreases.***
- ***Transaminases are not a marker for prognosis in either acute or chronic liver disease.***
- ***In chronic hepatitis B elevated transaminases helps in initiating therapy.***
- ***Transaminase levels are used as a surrogate marker for monitoring therapy in both chronic hepatitis B and autoimmune hepatitis.***

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CLIPPINGS

Positive and Negative Themes Found in Superhero Films.

Superhero films have become incredibly popular. The objective of this study was to determine the positive and negative themes found in a select number of superhero films. A total of 30 superhero films were analyzed. The average numbers of positive and negative themes were 19.4 and 29.5 mean events per hour for all included films, respectively. The most common positive themes were "assisting others/protecting the public," "positive relationships with family/friends," and "teamwork/collaboration." The most common negative themes were "acts of violence/fighting," "use of guns/knives/lethal weapons," and "bullying/intimidation/torture." Based on the superhero films included in our study, the number of negative themes, especially acts of violence, outweighs positive themes. Although an exposure to positive themes found in superhero films may be beneficial to the development of children, pediatric health care providers should counsel children and their families in an attempt to limit their exposure to violence.

Bauer M, Georgeson A, McNamara C, Wakefield BH, King TS, Olympia RP. Positive and Negative Themes Found in Superhero Films. Clinical Pediatrics 2017; 56(14): 1293-1300.

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HYPOPIGMENTED SKIN LESIONS – WHAT A PEDIATRICIAN SHOULD KNOW?

***Madhu R**

Abstract: *Hypopigmentation in a child, is of deep concern to the parents mainly owing to the social stigmatization and the belief that all hypopigmented lesions are either due to leprosy or vitiligo. Hypopigmentation may be congenital or acquired, localized or generalized, circumscribed or diffuse and may be due to infectious or non-infectious causes, with or without scales. It becomes important for the pediatrician to be well versed with the clinical presentation of the various common conditions associated with hypopigmentation. Four congenital causes of hypomelanosis and few acquired causes of hypopigmentation such as pityriasis alba, pityriasis versicolor, polymorphic light eruption, vitiligo, post inflammatory hypopigmentation and leprosy will be discussed in this article.*

Keywords: *Hypopigmentation, Congenital, Acquired.*

Hypopigmented skin lesion in a child is a cause of mighty concern to the parents who always think that it is either vitiligo or leprosy, both of which carry a strong social stigma. It becomes a difficult task for the pediatrician, many a times to arrive at the correct diagnosis due to the close resemblance of these lesions and to address the anxious parents and at times the children too. Hypopigmentation may be congenital or acquired, localized or generalized, circumscribed or diffuse and may be due to infectious or non-infectious causes, with or without scales. A well elicited history and an astute clinical examination will aid in clinical diagnosis. Dermatological examination of a hypopigmented skin lesion is incomplete if one does not use a magnifying lens to look for the border (well defined or well to ill-defined), surface, shape, presence of scales, atrophy and distribution of hair. Raghavendra,et al observed nevus depigmentosus, nevus anemicus, halo nevus, tuberous sclerosis complex, piebaldism and Hypomelanosis of Ito in the decreasing order of frequency to be the common congenital disorders of pigmentation in a study done at Rajasthan.[1] A south Indian study observed the frequency of hypopigmentary disorders among children to be 3.28 per 1000 children attending the dermatology outpatient department. Pityriasis alba, vitiligo, leprosy, nevus depigmentosus and pityriasis versicolor were the most common hypopigmentary disorders observed in this study.[2]

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Approach to a hypopigmented lesion

First and the foremost factor to be considered is whether the lesion is congenital or acquired. Congenital hypopigmented lesion may be localized, circumscribed hypomelanosis or associated with extracutaneous, systemic manifestations. Congenital, localized hypomelanosis without systemic involvement include nevus achromicus or nevus depigmentosus and nevus anemicus. Those with systemic involvement are Hypomelanosis of Ito, tuberous sclerosis and nevoid hypomelanosis. Common acquired hypopigmented conditions in children may be classified as those associated with the presence of scales such as pityriasis versicolor, pityriasis alba and polymorphic light eruption and those without scales such as vitiligo, Hansen disease, lichen striatus and post inflammatory hypopigmentation. Other less common conditions in office practice include kwashiorkor, lichen sclerosus, onchocerciasis and post kala azar dermal leishmaniasis. Approach to hypopigmented skin lesions is depicted in the Fig.1.

Congenital hypomelanosis

Nevus achromicus / Nevus depigmentosus

Nevus achromicus also known as nevus depigmentosus (ND)[3] occurs in 1.6% to 4.7% of children.[4] It may be present at birth or may occur during the early infanthood.[3] Clinical diagnostic criteria proposed by Coupe in 1976 include the presence of leukoderma at birth or during early childhood with no alteration in the distribution, texture or sensation over the lesion throughout life and absence of hyperpigmented border around the achromic lesion. Nevus achromicus, a non-familial well circumscribed hypomelanosis is said to occur probably due to a developmental defect in the fetal melanocyte.

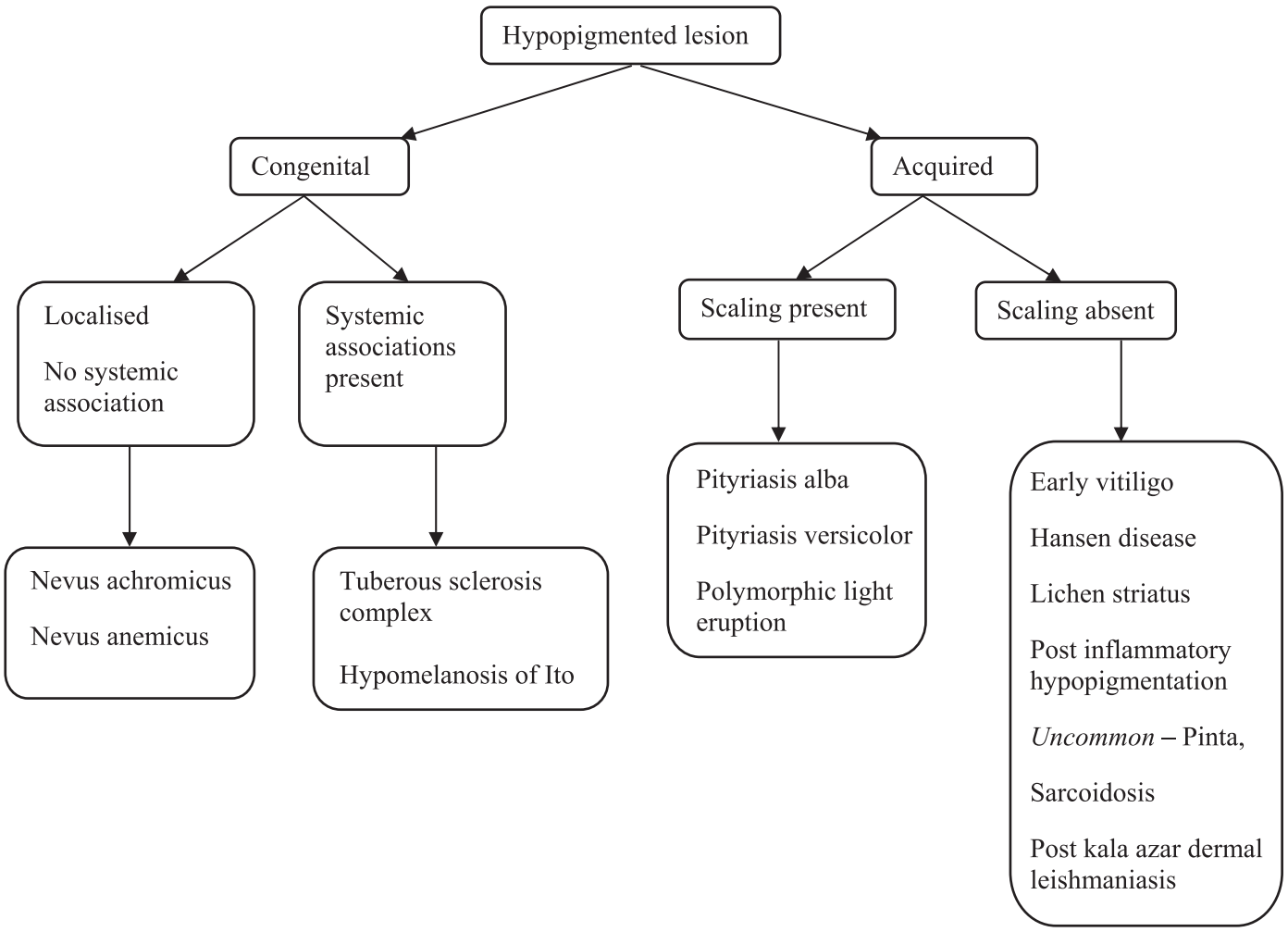

Fig.1. Approach to hypopigmented skin lesions

Melanocytes are normal in number, while the melanosomes are found to be abnormally small, reduced in content and scattered.[5] There are autophagosomes and aggregation of melanosomes within the stubby dentrites of melanocytes along with a defective transfer of melanosomes to the keratinocytes. ND usually occurs as a solitary lesion but may be focal, segmental or multiple following the Blaschko's lines.[6]Multiple nevus depigmentosus along the Blaschko's lines associated with systemic involvement is referred to as Hypomelanosis of Ito.[3] ND is typically characterized by the presence of hypopigmented rather than depigmented macules or patches of variable sizes with regular or serrated borders. Lesions usually do not cross the midline and are seen on the trunk, neck, face and proximal part of the extremities. However, ND may occur in any site of the body.

Treatment: Reassurance should be given as there is no effective treatment for ND. Camouflage could be suggested for children and adolescents. Subsequently, cosmetic concern could be addressed by procedures such as blister roof grafting or cultured epidermal grafting.[5]

Nevus anemicus

Nevus anemicus (NA) is a vascular developmental anomaly, often present since birth, but may present in infancy or early childhood. NA occurs as a result of neurally mediated vasoconstriction. There is a localized vascular hypersensitivity response to catecholamines which implies that there is only a functional abnormality while the number of melanocytes and melanosomes are normal. It is characterized by the presence of well demarcated, hypopigmented patches with serrated margins most commonly seen on the trunk. Diascopy of the adjacent skin (pressing the skin with a glass slide) results in blanching and merging with the nevus anemicus lesion). On firm stroking of the lesion, wheals appear without erythema. Nevus anemicus may be associated with capillary vascular malformation, phakomatosis pigmento vascularis and neurofibromatosis.[6,7,8]

Treatment: Camouflage may be helpful to address the cosmetic concern of the individual and that of the parents.

Hypomelanosis of Ito

Hypomelanosis of Ito (HI) is characterized by the presence of multiple nevus depigmentosus following Blaschko linear pattern in association with extra-cutaneous involvement. HI has been reported to occur in 1in 8000 to 10,000 cases. Familial tendency with autosomal dominant or autosomal recessive pattern of inheritance has been reported. Though both ND and HI are due to mosaicism, type of mutation and the timing of mutation during the development of embryo determines the pattern, number and the association of extracutaneous involvement. Mosaicism is considered to be most likely due to chromosomal non-dysjunction during embryo development. Short arm of the X-chromosome, the short arm of chromosome 12 and chromosome 18 are more commonly involved, although many chromosomes that cause disruption of either the expression or function of pigmentary genes can be affected.[3]

Clinical features: HI is characterized by the presence of well demarcated, hypopigmented patches with regular or irregular border occurring in multiple segments along the Blaschko linear pattern or block pattern, which may become systematized. Systemic associations are seen as mainly neurological, ocular and musculoskeletal involvement. Neurological manifestations include developmental delay, mental retardation, seizures, microcephaly, deafness, hypotonia, ataxia and hyperkinesia. Ophthalmic involvement may present as micropthalmia, corneal opacity, cataract, nystagmus, strabismus, myopia, ambylopia and retinal degeneration. Skeletal abnormalities that may occur are facial and limb asymmetry, short stature, thoracic deformity, syndactyly, brachydactyly and polydactyly.[3, 6,9]

Treatment: There is no specific treatment available for HI, though the use of 308-nm Excimer laser has been reported in literature.[6]

Tuberous sclerosis complex

Tuberous sclerosis complex also known as Bourneville's disease is a hereditary disorder with autosomal dominant inheritance with variable expressivity. This disorder which occurs in 1 in 6000 to 1 in 10,000 population results due to mutation of either TSC 1 gene encoding hamartin or TSC 2 gene encoding tuberin. Multiple system involvement is seen in the form of hamartomas of the skin, eye, brain, heart, lungs, kidneys and the skeletal system. Cutaneous manifestations of tuberous sclerosis include hypopigmented macules, angiofibroma, fibrotic plaques or nodules, fibrous tumours, Shagreen patch, ungual or periungual fibroma, gingival fibroma and Confetti skin lesions. Presence of three or more hypopigmented macules of size more than 5 mm is considered as one of the major features in the diagnostic criteria for tuberous sclerosis complex. These lesions may be present in about 97% of patients at birth or a little later in life and are found to be the most common clinical feature in these children. They are most commonly seen on the trunk with the size ranging from few millimeters to centimeters. Shape of these hypopigmented macules may be oval, linear, round (thumb-print), confetti-like or the most characteristic lance- ovate shape, commonly referred to as the ash leaf spots or ash leaf macules . Wood lamp examination will aid in better visualization of the hypopigmented macules and differentiation from vitiligo. Histopathological examination of the hypopigmented lesions reveals a normal number of melanocytes with a reduction in the number, size and melanization of melanosomes.[4.10, 11]

Treatment: Reassurance and camouflage are the options for hypopigmented lesions. Usually patients with tuberous sclerosis complex tend to more concerned about the disfigurement due to the facial angiofibromas than about the hypopigmented macules which are more often present in the trunk.

Acquired hypopigmented skin lesions

Acquired hypopigmented skin lesions, although may be classified based on the infective etiology, is best approached on the basis of the presence or absence of scales. Among those disorders associated with the presence of scales, common conditions such as pityriasis versicolor, pityriasis alba and polymorphic light eruption would be discussed.

Acquired hypopigmented disorders associated with scaling

Pityriasis versicolor: Tinea versicolor (Misnomer),Tinea flavea, Dermatomycosi furfuracea, Liver spots, Chromophytosis.[12]

Pityriasis versicolor (PV) is a chronic, recurrent, asymptomatic superficial fungal infection caused by Malassezia which are lipophilic yeasts. The word, "Pityriasis" is derived from a Greek word which means "bran like" and "versicolor" means varied colour to denote the achromic (hypopigmented) and chromic

(hyperpigmented) forms of PV. Wilan first described the lesions of pityriasis versicolor in 1801.[13] Malassezia forms a part of the normal flora of the skin in more than 90% of normal adults. In infants, colonization has been observed by 40 days of life.[14] It affects about 30%-40% of population in temperate climate. In a study conducted in Rajasthan, this infection was reported in 21% of the study population comprising of children and adolescents.[1]Study by Reddy VS, et al done at Kerala observed pityriasis versicolor to be the second common fungal infection (39.4%) next to dermatophytosis (48.5%).[15] Pityriasis versicolor is more common in adolescents and young adults compared to younger children.[16] A positive family history has been reported, especially in first degree relatives. Infection results due to the shift from the commensal yeast phase to the mycelia phase under certain predisposing conditions such as hot and humid tropical climate, individual host susceptibility, malnutrition, hyperhidrosis, occlusive clothing, increased sebum production, corticosteroid therapy, immunosuppression and Cushing's disease.[17] Among the 13 species of genus, pityriasis versicolor is usually caused by Malassezia globosa and less often by M sympodialis and M furfur. The causative organism does not affect the nails, mucous membrane or hair shaft, but may cause pulmonary and systemic infections in infants on intravenous lipid therapy.[12] Depressed or altered cell mediated immunity may be considered as a factor in the development of this infection.[14]

There have been varied mechanisms postulated for the hypopigmented and hyperpigmented lesions of pityriasis versicolor. Malassezia species produce azalaic acid which competitively inhibits tyrosinase, resulting in hypopigmentation. Scales produce a sunscreen effect. Azelaic acid is also said to have a direct cytotoxic effect on the hyperactive melanocytes. Electron microscopic studies have revealed that there were smaller than normal melanosomes in achromic pityriasis versicolor (hypopigmented type) while, there were abnormally large melanosomes in chromic (hyperpigmented) type. Stratum corneum is found to be thicker in the hyperpigmented lesions.[12,14,17]

Clinical features: Characteristic lesions are hypopigmented or pigmented macules and patches with well defined border and fine branny or furfuraceous scales over the sebum rich areas. When the scales are not visible, scratching with a finger nail causes loosening of the scales making it perceptible, which is termed as, "Scratch sign or Coup d'Ongle sign or Besnier's sign" and this is considered as an important diagnostic feature of pityriasis versicolor.[18] Scratch sign would be negative if a patient has taken bath or has undergone treatment. Scaling can also be observed by stretching the skin and this is termed as, "Zireli's sign".[17] Common sites of involvement include chest, upper back and arms. Lesions may be less often seen on the face, neck, forearms, thighs, scalp and rarely over the palms and dorsum of fingers. Face is the most frequently affected site in infants and children, with the forehead, temple, medial canthi of eyes and paranasal area being involved.[2,14] Extensive lesions and recurrent episodes are usually seen in the adolescents. Wood's lamp examination of the scaly lesions reveals a pale yellow fluorescence. Diagnosis is usually clinical, in the presence of the characteristic clinical features. When there is a clinical suspicion, scraping of the scales could be done using a blunt scalpel and examined in 10% potassium hydroxide for hyaline, short, aseptate, angulated hyphae, spherical yeast cells either as isolated or in groups and blastospores (budding yeast cells). This characteristic appearance of the hyphae and the yeast cells in clusters has been referred to as "Spaghetti and meat ball" or "banana and grapes appearance".

Box 1. Therapy of Pityriasis versicolor[14,17]

Localised lesions

Clotrimazole, miconazole, ketoconazole, sertaconazole, eberconazole creams twice daily Terbinafine cream twice daily

Extensive lesions

Ketoconazole lotion / shampoo – short contact for 10 minutes before bath x 10 days (to be applied over the neck, trunk, arms and forearms)

Selenium sulfide shampoo daily – short contact for 10 minutes before bath (Cosmetically unacceptable)

Systemic therapy - Tab Fluconazole 400 mg stat dose
Tab Fluconazole 300 mg once a week x 2 weeks
Itraconazole 200 mg x 5 days

Recurrence

Itraconazole 400 mg od monthly once x 6 months

Treatment: Localised lesions may be treated with topical azole antifungal agents or topical terbinafine cream for a period of 4 to 6 weeks. Extensive lesions over the trunk have to be treated with short contact therapy for 10 minutes before bath with azole lotions or shampoo once a day for 10 days. Details of treatment is given in Box1.

Pityriasis alba

Pityriasis alba (PA) is an asymptomatic, non-specific eczematous dermatitis of unknown etiology, commonly seen in children in the age group between 3-16 years with equal sex predisposition. It is said to affect 1.9% to 5.25% of preadolescent children.[19] PA results in post inflammatory hypopigmentation and is said to be more common in children with darker skin types. Though it is often associated with atopic dermatitis, PA can occur in the unaffected children. Dry skin, exposure to sunlight or wind and soap can affect the outcome of pityriasis alba. There is no definite association between bacterial, fungal or parasitic infections and PA. Electron microscopy revealed a normal number of melanocytes with a reduction in the number and size of the melanosomes.[20]

Clinical features: PA is characterized by the presence of round or irregular hypopigmented patches with ill-defined borders and fine scales. Initial phase of erythema is most often not made out and children are brought for consultation when the hypopigmentation and scaling have set in. Usually PA gets noticed in children after prolonged sun exposure during sports activities when the surrounding skin gets sun tanned but not the PA affected skin. Lesions range in size from 0.5cms to 2 cms in size and are most commonly seen on the face, especially the cheeks, perioral area and chin. They may also occur on the neck, upper arms and upper trunk. Two uncommon variants namely extensive and pigmenting pityriasis alba have been reported. PA runs a chronic and recurrent course in children, sometimes extending up to 1 year duration.[21, 22]

Treatment: Parents need to be counseled about the post inflammatory hypopigmentation that takes a long time to repigment. General measures such as reduction of sun exposure and regular use of sunscreens should be advocated. Topical application of emollients is the first line of management. Mild topical corticosteroids such as hydrocortisone cream or desonide cream are useful. Topical tacrolimus and pimecrolimus may be effective in the persistent lesions.[20]

Polymorphic light eruption

Polymorphic light eruption (PMLE), an idiopathic immune mediated photosensitive disorder, is the most common photosensitive dermatoses seen in children. It is characterized by the occurrence of pruritic, polymorphic lesions in the sun-exposed sites of the body, on exposure to ultraviolet radiation. PMLE is more common in children with Fitzpatrick's skin types I – IV, especially in the school going children who are more vulnerable due to prolonged hours of playing in the sun. Episodes of PMLE occur during spring (March) and early summer. Sharma, et al reported a high incidence of PMLE during March and September in a study done at Varanasi. PMLE may also occur due to penetration of ultraviolet radiation through window glasses or even during winter at high altitudes. Lesions are of acute nature and may develop within hours to days after sun exposure and last for one to two weeks after cessation of sun exposure. However, there may be persistence of lesions for weeks in the presence of continued exposure to sun, after which lesions may improve with reduction in frequency and severity due to the phenomena of hardening which occurs as the summer progresses. Hardening refers to the increased tolerance of skin with continued exposure to sun. PMLE has been postulated to be due to delayed hypersensitivity reaction to endogenous cutaneous photoantigen of unknown nature induced by exposure to ultraviolet (UV) radiation. In normal individuals, UV light induced immunosuppression prevents the autoimmune reactivity that may result due to UV radiation. There is resistance to this UV induced immunosuppression in those with PMLE. Hereditary form of PMLE with autosomal dominant inheritance has been reported in native Americans.[23-29]

Clinical features: PMLE is seen more often on the face and ears in children, when compared to adults. Usually affected sites are the malar areas of the cheek, bridge of the nose, forehead, chin, 'V' area and nape of the neck, extensor aspect of the forearms and arms, dorsum of the hands and upper back. Pruritic skin lesions develop on the sun exposed sites, within minutes to hours to days after sun exposure depending on the individual susceptibility. Though the lesions are polymorphic, in any single individual, lesions are always monomorphic and even during the recurrent episodes, lesions are of the same morphology. Various lesions of PMLE are erythematous to skin coloured grouped papules, plaques, vesicles, papulovesicles, hypopigmented or eczematous patches and nodules, of which the papular type is the most common. Hyperpigmentation is often observed within the hypopigmented patches. Lesions tend to be symmetrical in distribution. A pinpoint papular variant with lesions resembling lichen nitidus has been described in individuals with dark skin colour. Rarely erythema multiforme like or urticarial lesions may occur. Hypopigmented patch of PMLE could be differentiated from pityriasis alba and pityriasis versicolor by the presence of itching after sun exposure and symmetrical distribution when present.[23,25, 26]

Treatment: Children with PMLE should be advised to limit exposure to sunlight from 9 am to 4 pm. General preventive measures such as use of long sleeved shirt, broad brimmed hat and umbrella should be strictly followed. Broad spectrum sunscreen that will offer protection against UVA and UVB is preferable. Sunscreen is to be applied 20 minutes before sun exposure and is to be reapplied every 3-4 hours. In addition, some children may require the application of low potent topical steroid such as hydrocortisone cream on the face or mid potent steroids such as fluticasone or mometasone for lesions on the forearms or back.

Common hypopigmented disorders without scaling

Among those hypopigmented disorders that are not associated with scales, leprosy, vitiligo and post inflammatory hypopigmentation would be discussed.

Leprosy (Hansen's disease)

Childhood leprosy is considered to reflect the burden of leprosy in the community. Studies have shown that boys are affected more than the girls. Children in the age group of 10-12 years are most commonly affected. Index case is most often a close family member or a neighbor with multibacillary disease. Hypopigmented macules and patches are the most common skin lesions seen in children with leprosy. In a clinic based epidemiological study of pediatric leprosy done at Kerala, Nair observed hypopigmented macule to be the most common primary skin lesion in 71.66% of patients.[30] Hypopigmented macule or patch is usually a feature of indeterminate leprosy, tuberculoid or borderline tuberculoid leprosy. Borderline tuberculoid leprosy is the most common type of childhood leprosy, followed by indeterminate leprosy and tuberculoid leprosy. Borderline lepromatous leprosy and lepromatous leprosy are rare in children.

Clinical features: Indeterminate leprosy, considered as the first sign of leprosy is the most common presentation in children. At this stage of early manifestation of infection, lesion heals without leaving any sequel if the host immunity is good. Children with indeterminate leprosy present with one or few asymptomatic, ill-defined hypopigmented macules with normal sensation. Lesions are commonly seen on the face, buttocks and extremities. Peripheral nerve examination is normal. It is very difficult to make a diagnosis at this stage as biopsy may not be a feasible option in children, especially if the lesion is on the face and hence, may require observation over a period of time with regular follow up. Lesions in indeterminate leprosy may undergo spontaneous resolution or may evolve into a more definite spectrum of leprosy.

True tuberculoid (TT) leprosy: TT leprosy is characterized by the presence of 1 to 3 well defined, anesthetic macules or plaques with dry surface. Trophic changes such as loss of sweating and loss of hair are present. Asymmetrical thickening of the peripheral nerves in the region may be present. Sites of predilection in TT leprosy are similar to indeterminate leprosy.

Borderlne tuberculoid (BT) leprosy: Patients with BT leprosy may present with 3-10 well to ill defined hypopigmented, hypoanestheic patches with trophic changes. Satellite lesions are present in the periphery of the lesion. There is asymmetric thickening of the peripheral nerves.

Borderline lepromatous leprosy: Multiple hypopigmented patches and plaques with a tendency towards symmetrical distribution are seen. Sensation may vary. Peripheral nerve thickening may also show tendency towards symmetry.

Lepromatous leprosy: Lepromatous leprosy is rare in children. Characteristic skin lesions include multiple macules, infiltrated plaques and nodules in a symmetrical distribution.

Treatment: Treatment is based on the classification into paucibacillary and multibacillary leprosy. Paucibacillary

Table I.Treatment schedule in children 10 – 14 years[31]

Drug	Paucibacillary - 6 months		Multibacillary - 1 year	
	Monthly supervised dose	Daily dose 2- 28 days	Monthly supervised dose	Daily dose 2- 28 days
Rifampicin	450 mg	—	450 mg	—
Dapsone	50 mg	50 mg	50 mg	50 mg
Clofazamine	—	—	150 mg	50 mg alternate days

leprosy refers to the presence of 1 to 5 lesions including single nerve involvement with negative slit skin smear (SSS). Multibacillary leprosy is inclusive of six and more skin lesions or more than one nerve involvement irrespective of the number of skin lesions and a positive SSS. Treatment schedule for children between the age group of 10-14 years is given in Table I.

In children younger than 10 years, drug dose may be calculated based on the body weight [31]

i) Rifampicin - 10 mg /kg body weight once a month,

ii) Dapsone - 2mg/ kg body weight / day

iii) Clofazamine - 2-3 mg/kg body weight once a month and 1 mg/kg body weight on alternate day.

Early vitiligo

Vitiligo is an acquired, autoimmune disorder characterized by the presence of well demarcated depigmented macules and patches that occur due to progressive loss of melanocytes. This is considered to be a result of cytotoxic activity of autoreactive T cells against the melanocytes. Studies have detected autoantibodies against melanocyte specific antigens that destroy the melanocytes. Vitiligo occurs in 1% of the world population and can affect any age group. Among these patients, 50% of them have their onset before 20 years of age, while in 25% of patients, vitiligo develops before 8 years of age. Vitiligo is said to have a polygenic inheritance. Monozygotic twins have been reported to have a prevalence of 23%, siblings 6% and first–degree relatives 7%-12%. Classical vitiligo lesions are seen as well demarcated, depigmented ivory white macules and patches, which pose no difficulty in diagnosis. However, an early vitiligo lesion may appear hypopigmented due to the partial loss of pigmentation and hence may be confused with pityriasis alba or pityriasis versicolor. In this scenario, it is noteworthy that an early vitiligo lesion appears as a well defined patch without scales, whereas pityriasis alba is characterized by ill-defined borders and pityriasis versicolor by well defined macules and patches with fine branny scales. Wood's lamp examination provides a good delineation between the normal skin and the vitiligenous lesion.[4, 32]

Treatment: Early vitiligo could be treated by application of topical corticosteroids such as fluticasone, mometasone or betamethasone valerate once daily for a period of 3-4 weeks, followed by intermittent application. Combination of topical corticosteroid with topical calcineurin inhibitor such as tacrolimus and pimecrolimus, has been found to effective in producing repigmentation.

Post-inflammatory hypopigmentation

Many inflammatory disorders such as infections, burns, bullous disorders, psoriasis, pityriasis rosea, pityriasis alba, pinta, sarcoidosis etc are associated with post-inflammatory hypopigmentation. Exact mechanism of hypopigmentation in these disorders is not clear. It has been postulated that the keratinocytes that are injured due to inflammation are not in a position to accept the melanosomes that are being transferred from the dendrites of the melanocytes temporarily. Post-inflammatory hypopigmentation takes months to years to repigment.[4]

Conclusion

Hypopigmented skin lesion in a child even if single, is worrisome to the entire family, which makes it difficult for the attending physician, be it a pediatrician or a dermatologist, as one has to not only make a correct diagnosis and start treatment, but in addition has to offer proper counseling and reassurance to the parents. Proper elicitation of history and astute clinical examination will aid in correct diagnosis of these lesions.

Points to Remember

- ***Hypopigmentation may be due to infections or non-infectious causes, with or without scales. It can be congenital or acquired, localized or generalized, circumscribed or diffuse.***
- ***Reassurance, camouflage and masterly inactivity are the options in children with localised congenital hypomelanotic conditions - Nevus depigmentosus and nevus anemicus.***
- ***Children with Hypomelanosis of Ito and tuberous sclerosis complex require evaluation for systemic associations.***
- ***Common acquired hypopigmented conditions with scaling includes pityriasis versicolor, pityriasis alba and polymorphic light eruption.***
- ***Hypopigmented patch of PMLE could be differentiated from pityriasis alba and pityriasis versicolor by the presence of itching after sun exposure and symmetrical distribution when present***
- ***Common acquired hypopigmented conditions without scaling includes leprosy, vitiligo and post-inflammatory hypopigmentation.***

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EARLY CHILDHOOD CARIES - CAUSES AND MANAGEMENT

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Abstract: *Early childhood caries is a disease affecting the primary dentition of children below six years of age. Previously, termed as 'nursing caries' or 'baby bottle caries', this disease has become endemic in all developed as well as developing countries. Although, there are numerous factors associated with this disease, night time feeding practices are strongly associated. This disease not only affects the child's teeth but also affects the overall health. Children with early childhood caries have been associated with poor quality of life. This disease is completely preventable when intervened by the first year of life.*

Keywords: ***C**aries, Children, Full mouth rehabilitation, Prevention.*

The disease of early childhood caries (ECC) is defined by the American Academy of Pediatric Dentistry (AAPD) as the presence of one or more decayed (non cavitated or cavitated lesions), missing (due to caries) or filled tooth surfaces in any primary tooth in a child 71 months of age or younger. In children younger than 3 years of age, any sign of smooth-surface caries is indicative of severe early childhood caries (S-ECC). From ages 3 through 5, one or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth or a decayed, missing, or filled score of ≥4 (age 3), ≥5 (age 4), or ≥6 (age 5) surfaces is diagnosed as S-ECC.[1] In simple words, the presence of any decayed tooth or teeth in children below 6 years of age is diagnosed as early childhood caries. The presence of any decay in the front teeth in children less than 3 years is diagnosed as severe early childhood caries.

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Consequences of ECC

ECC is a serious health problem in very young children. Though it is not life threatening, if left untreated it may lead to pain, swelling, compromised chewing ability and bacteremia. It may be followed by malocclusion in permanent dentition, phonetic problems, suboptimal health, sleep disturbances, lower self-esteem and failure to thrive.[2,3] Consequences of ECC include a higher risk of new carious lesions in both the primary and permanent dentitions,[4,5] hospitalizations and emergency room visits,[6,7] increased treatment costs,[8] risk for delayed physical growth and development,[9,10] loss of school days and days with restricted activity,[11,12] diminished ability to learn[13] and diminished oral health-related quality of life. It has been demonstrated that dental caries can gradually reduce a child's ability to gain weight, which may get reversed after complete oral rehabilitation.[14]

Etiology and risk factors

The etiology of ECC is multi-factorial. Risk factors associated with ECC include early colonization of mutans streptococci (MS) through vertical and horizontal transmission, low salivary flow at night, the presence of plaque, poor oral hygiene, frequency and timing of consumption of sugar-containing drinks.[15–18] Other reported associated factors are nocturnal breastfeeding[19] and prolonged duration of breastfeeding,[20] presence of enamel hypoplasia,[21] molar-incisor hypomineralisation[22] and the child's socioeconomic status.[23] There are controversies regarding breast milk and bottle milk as etiological factors for dental caries.[24] A large randomized trial in the area of human lactation provides no evidence of beneficial or harmful effects of prolonged and exclusive breast-feeding on dental caries at early school age.Though it was a large scale RCT, there were few limitations on these trials in terms of measuring the outcome at 6 years of age, lack of pediatric dentists involvement in the study (public health dentists recorded the caries status), not scoring or documenting non cavitated lesions. To understand the role of night time feeding practices, further research is needed in the age group of 1-3 years, as during later stage of development (after 2.5- 3 years) multiple other factors play a greater role and make the etiology of the caries process complex and difficult to understand. Many maternal factors

Table I. Factors found to be significantly related to the prevalence and/or incidence of deciduous caries in children age 6 years and under

Socio- demographic factors	Dietary factors	Oral hygiene	Factors related to breast/bottle feeding	Oral bacterial flora
Gender of child (male), public rather than private school, family income, father unemployed, low parental education, low maternal education, single mother, occupation of head of household, high number children per family, 3+ adults in household, mother with non full-time jobs, birth order, immigrant background, mother's young age, 2+ children living household,cohabitation of parents, ethnicity, parental occupation	High frequency, high sugar foods/day, high number of between meals sugary food/drink, no set time for snacks, cariostat, score, high pocket money for sweets, delayed of weaning, not eating fruit as a snack, high sugar / fat snacks, 5xdaily sweet snacks, candy >1x/week, low magnesium intake, high iron intake, high cariogenicity score, high daily frequency of sugar intake at nursery, high daily weight of sugar intake at nursery, >6 eatings / drinkings per day, food before sleeping, fruit juice at bed-time, sugary bed-time drink, carbonated drinks at bed-time, daily sucrose intake, night-time meals/drinks, night-time juice, frequency of consumption of diluted syrup, milk intake score, dates eaten daily, frequent consumption of sugary drinks, frequent consumption of carbonated drinks, amount and frequency of sweet consumption	Daily tooth-brushing, frequency of tooth-brushing: more often & less often, age at which brushing started, visible plaque, combined frequency brushing and parental supervision, adults involved in brushing, lack of use of fluoride toothpaste, not having teeth cleaned at bed-time, high gingival score	Bottle as opposed to breast fed, duration of breast feeding, nocturnal breast feeding, night-time bottle use, use of sugar/cereal in the bottle, frequency of breast feeding, bottle with sugary drink at bed-time, bottle/breast fed to stop baby crying at night, duration of bottle feeding with fruit juice, breast fed or plain milk in bottle at night, still bottle / breast fed at 18 months, bottle at night >24mths, duration of breast or bottle feeding, if child slept with bottle or breast at 12 months, bottle carried around during the day	Presence of strep. mutans, presence of lactobacilli, strep. mutans count, rare transfer of maternal saliva to baby

that may predispose children to ECC are bad infant-feeding practices, poor maternal knowledge of oral hygiene practices, maternal nutrition and maternal stress (Table I).[25-31] Additionally, newly-erupted teeth, because of immature enamel and teeth with enamel hypoplasia may be at higher risk of developing caries[32]. In 2004, Rebecca Harris conducted a systematic review and concluded that there are 106 risk factors associated with ECC.[33]

Microbial risk markers

Microbial risk markers for ECC include mutans streptococci (MS) and lactobacillus species.[34] However, new tools for bacterial identification (e.g., polymerase chain reaction (PCR) techniques, 16s rRNA gene sequencing) are revealing the complexity of the oral microbiome and other bacterial species that may be associated with ECC.[35]

ECC and quality of life

Several studies have addressed the effects that ECC has on a child's and their family's quality of life (QoL). Quality of life factors frequently associated with poor oral health were tooth aches, having trouble eating certain foods, missing school, difficulty to brush the teeth, frequent episodes of fever, cough and cold and poor self-image (Fig.1). Studies have also investigated the potential positive effect of treatment intervention on QoL for children with severe caries. These studies demonstrate improved oral health-related QoL following treatment under general anesthesia. One to four weeks following treatment under general anesthesia, children were reported to have better perceived quality of life. Thus, ECC has a tremendous, but almost invisible, impact on society and the health care system.[36]

Management

Due to the aggressive nature of ECC, cavities can develop quickly and if untreated, can infect the tooth's pulpal tissue. Such infections may result in a medical emergency that could require hospitalization and the extraction of the offending tooth.[37] Managing ECC is challenging task for even a trained pediatric dentist. In ECC management, treating dental pain is very important. Pain is difficult to measure due to its subjectivity. Children may not have the language skills to communicate the level of pain they are feeling and assessing pain levels often depends on the report of parents or pain scale indicators.[38] As a result, it's possible to undertreat or overtreat pain, each of which carries its own set of health risks.[36] Since, ECC mostly affects very young age-group comprising children lacking cooperative ability, the treatment is done mostly under sedation, general anesthesia, etc. Whenever, a need for multiple dental procedures like pulp therapy, stainless steel crowns and deep caries management are required, full mouth rehabilitation under general anesthesia is the best way as it allows the pediatric dentist to provide the highest quality of dental care (Fig.2 & 3). However, the benefits and the risks involved should be weighed for each patient and a decision should be taken accordingly. For children with mild ECC (children with white spot

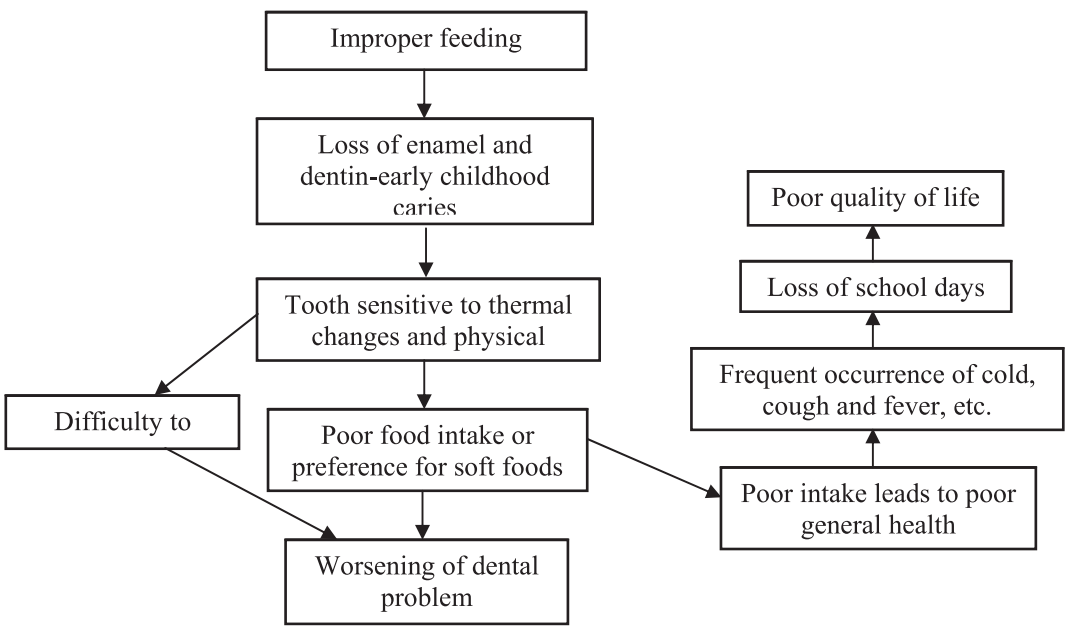

Fig.1. Early childhood caries – Progression

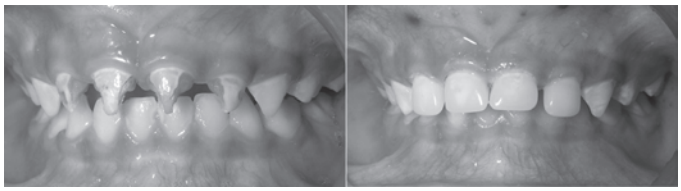

Fig.2.Full mouth rehabilitation. (Pre and Post-operative)

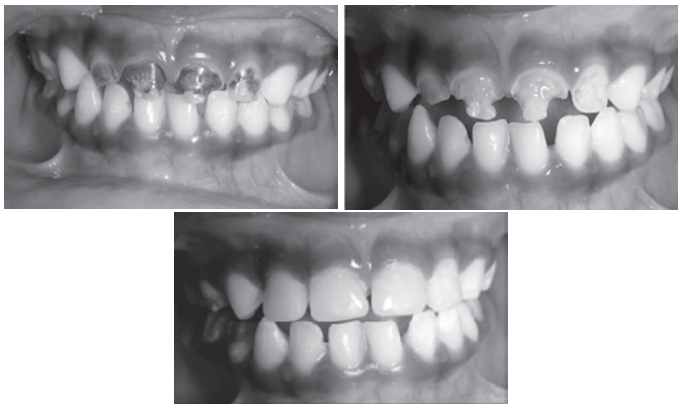

Fig.3.Full mouth rehabilitation - Restoration of maxillary teeth using strip crowns (Pre operative, after caries removal and post operative)

lesions or one or two small cavities), chairside treatment is attempted. Caries removal is done by a slow-speed (micromotor) followed by restoration.[39]

Prevention

Preventive measures can be categorized into community based measures, professional measures and home care methods. Community-based measures are community water fluoridation and education programs organized at the community level, professional measures are steps taken at the dental office by a dentist to prevent ECC. These measures include teaching appropriate oral hygiene measures, regular fluoride application, recall visits, etc. Home care methods are efforts taken by the parents in relation to dietary habits, feeding habits and oral hygiene measures. Prevention of transmission of mutans streptococci from mothers with high counts of mutans streptococci can be achieved by use of xylitol containing chewing gum after? delivery. Studies have shown significantly lower def index in children whose mothers have used xylitol chewing gums from 3-24 months after delivery. Dental education to improve the habits of parents or caregivers through anticipatory guidance and infant oral health care measures for ECC prevention can be promoted. If the oral hygiene practices are established early this disease can be prevented. This can be achieved by promoting first dental visit at the age of one or after the eruption of the first milk tooth whichever is earlier.[40]

Points to Remember

- ***Presence of caries in children below 6 years of age can be diagnosed as early childhood caries.***
- ***First dental visit of a child must be as soon as the first milk tooth erupts or on their first birthday, whichever is the earliest.***
- ***Early childhood caries can result in poor quality of life for the child. This can be reversed by full mouth rehabilitation. Consequences of not treating can lead to damage to permanent teeth as well as increased caries risk on permanent teeth.***
- ***Full mouth rehabilitation under general anesthesia is one of the best method for rehabilitation of multiple caries in children of pre cooperative age.***
- ***Measures like good oral hygiene practices, periodic fluoride application and periodic dental check up every 6 months can possibly prevent early childhood caries.***

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HEAD INJURY IN CHILDREN - TRIAGING AND IMAGING

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****Viveka Saravanan**
****Ravi LA**

Abstract: *Head injury in infancy and childhood differ significantly from adults in the modes of injury, mechanisms of damage and the management of specific problems. Fall from height forms the most important cause of pediatric head injury. The overall outcome for children with head injury is better than that of adults with the same injury score but recovery takes a longer time.*

Keywords: *Head injury, Assessment, Management.*

Traumatic brain injury (TBI) is a major cause of disability and death in children and young adults worldwide. It is considered a "silent epidemic" because the general public are mostly unaware of the scale of the problem.[1] Head injury is one of the most common injuries in childhood comprising about 84.3% of all injuries of which isolated head injury constitutes (78.9%) and polytrauma including head injury about 5.4%. It is one of the most frequent reasons for a visit to the emergency department.[2] No age is exempt from head trauma with nearly 25%-27% of all victims being less than 16 years of age.

Fall from height such as from unprotected roof tops or balconies while playing (56.5%) remains the most common cause of head trauma followed by road traffic accidents (21%), simple fall from chair or bed (17.5%) and fall of heavy objects on the head.[3] Boys are more prone for head trauma than girls with ratio being 1.6:1.[4]

The common modes of injury in different age groups vary - non accidental trauma in infants, falls in toddlers and injuries during sports and games in school aged children and road traffic accidents in adolescents.

Significance

It takes a second to injure the brain but it takes weeks, months to years to recover. There has been a general view that young children can bounce back from any insult. There is a common belief that a younger brain is more plastic and is better able to find new ways of doing things. It is true that children fare better than their adult counterparts in the immediate post traumatic period. However, the neuro-cognitive impairments they face in the domains of attention, concentration, social skills, executive skills, communication and learning are often ignored. Hence, it is very important to prevent head injury in children and treat aggressively if it happens.

Pediatric uniqueness

Children have a larger head to body surface area - 18% versus 7% in adults which makes them prone for head injury.[5] The compliant skull of a child is easily deformed and so impacts on the brain produce coup injury in contrast to adults in whom the brain is forced against bony prominences to produce countercoup injury. Pediatric brain has a higher water content 88% versus 77% in adults which makes the brain softer and prone for acceleration-deceleration injury. The water content is inversely proportional to myelination and the unmyelinated brain is more susceptible to shear injuries.

Children have a soft cervical vertebra and supple neck which increase their susceptibility for injuries of upper cervical spine. They also have a smaller airway compromised by large tonsils, adenoid and anteriorly placed larynx which is more compromised when neck is flexed or extended. Infants, in general, are more vulnerable to abusive trauma due to their size, dependency on caretakers and their inability to report abuse. Premature or disabled children are also more likely than other children to suffer this type of trauma, perhaps because their caretakers may feel additionally burdened.[6] Children are dependent on the caregiver for supervision and safety and ironically often the care givers are the perpetrators of child abuse.

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Types of injury

There are two types of injury namely the primary injury which is the direct consequence of the initial physical insult. It comprises irreversible cell damage that is the main determinant of clinical outcome. In the secondary injury, inflammatory and neurotoxic responses triggered by the primary injury induce edema, hypoperfusion, hypoxia and ischemia.[7] These changes often lead to raised intracranial pressure (ICP), temperature dysregulation, loss of autoregulation and seizures. Much of these secondary injuries may be amenable to intervention and left untreated can significantly increase morbidity and mortality associated with TBI.

Assessment

Assessment begins with primary survey which is a focused physical examination to identify and treat life threatening injuries and prevent or minimize secondary brain injury.[8]

Airway: Airway should be stable to provide oxygenation and ventilation. Head and neck should be kept in neutral position. Airway is cleared by a jaw thrust maneuver instead of head tilt chin lift. Cervical spine should be protected by a cervical collar of appropriate size in older children and by sand bags and towel rolls in younger children as up to 10% of children can have associated cervical spine injury. TBI patients should be considered to have a full stomach. In children with suspected basilar skull fracture, orogastric and not nasogastric tube should be passed, as there is a possibility that the tube may enter the cranial cavity through the fractured site.

Spinal immobilization: Spinal immobilization should be maintained during intubation since cervical injury cannot be excluded. Full spinal immobilization is indicated in children whose GCS<15 (Table I) with neck pain and tenderness, focal neurological deficit and paresthesia in extremities.

Breathing: When pediatric GCS is less than or equal to 8, endotracheal intubation with rapid sequence induction (RSI) with cervical spine protection should be done. Oro tracheal intubation is indicated in suspected basilar skull fracture. Rapid sequence intubation (RSI) technique involves pretreatment with lidocaine which minimizes increase in ICP that can be associated with airway manipulation and sedation which can be provided by etomidate and thiopental which are neuroprotective. Paralysis can be induced by non depolarising agents such as vecuronium or rocuronium. Succinyl choline is

Table I. Glasgow coma scale (GCS)

Activity	Best Response		
	Adults/Older Children	Infants (modified GCS)	Score
Eye opening(E)	Spontaneous	Spontaneous	4
	To speech	To speech	3
	To pain	To pain	2
	None	None	1
Verbal(V)	Appropriate speech	Coos, babbles	5
	Confused speech	Irritable, cries but consolable	4
	Inappropriate words	Cries, Inconsolable	3
	Incomprehensible or	Moans to pain	2
	none specific sounds	None	1
	None		
Motor(M)	Obeys	Normal spontaneous movement	6
	Localizes pain	Withdraws to touch	5
	Withdraws to pain	Withdraws to pain	4
	Decorticate to pain	Decorticate to pain	3
	Decerebrate to pain	Decerebrate to pain	2
	None	None	1

contraindicated in neuromuscular disorders as it can cause malignant hyperthermia. Ketamine is contraindicated in patients with significant eye injuries as it can increase both ICP and intra ocular pressure.[9] Oro-tracheal route is the preferred one for intubation. It is ideal to ventilate with 100% oxygen to maintain normocapnia.

Circulation: Hemodynamic instability is unlikely to be caused by an isolated traumatic brain injury except in case of large scalp laceration which can cause severe bleeding in an infant. Hence, if the child presents with shock, other sources of blood loss such as intra abdominal injuries should be ruled out. There is no single best fluid for children with traumatic brain injury, but isotonic crystalloids are widely used and have good scientific basis. Normal saline or lactated ringer's solution should be the standard resuscitation fluid. Fluid restriction is no longer recommended.[10] Dextrose containing fluids should be avoided in the correction of shock as the resulting hyperglycemia is associated with a poor outcome.[11] Similarly even brief periods of hypotension is associated with poor outcome and hence should be treated aggressively. Since children have a large body surface area, hypothermia poses a real danger. Under stress, hypothermia causes acidosis and worsening of metabolic status.

Disability: Levels of consciousness is assessed by AVPU (alert, responds to verbal stimuli, responds to painful stimuli, unresponsive) scale in the emergency department and by modified Glasgow Coma Scale further (Table I).

Secondary survey: Secondary survey aims at detailed examination and assessment of individual systems, identifying all injuries and directing further treatment.

Table II. Head injury site and signs

Site	Signs
Head	Bruising, laceration, swelling, depression,
Neck	Deformity, tenderness, muscle spasm
Eyes	Raccoon eyes, pupil - size, equality, reactivity, retinal hemorrhage
Ears	CSF otorrhea, Battle sign (ecchymosis over mastoid)
Nose	CSF rhinorrhea, deformity, swelling, bleeding
Mouth	Soft tissue injuries, dental trauma
Motor functions	Lateralizing signs

Head, neck, eyes, ears, nose, mouth and motor function should be examined methodically (Table II and Fig.1a, 1b, 1c).

History

A proper history is crucial in the management of head trauma. Historical features regarding the mechanism of injury (witnessed / unwitnessed), time of injury, circumstances of injury for e.g. accident, non accidental trauma, unexplained fall (seizure or arrhythmia to be considered) and state in which child was found (LOC, seizures) are important. Presenting symptoms such as loss of consciousness, repeated vomiting, worsening headache, ear, nose, throat bleed and their duration, condition prior to consultation (stable, deteriorating, improving), past h/o bleeding diathesis and medication use such as anticoagulants should be sought for.

Parents provide the most reliable and trust worthy information. However if the history is inconsistent and does not match with the physical findings, then the possibility of inflicted injury (child abuse) should be thought of. Unless a high index of suspicion is maintained, much of these can go unrecognized. Presence of fundal hemorrhages, fractures of various ages on skeletal survey,

Fig.1a.Raccoon eyes

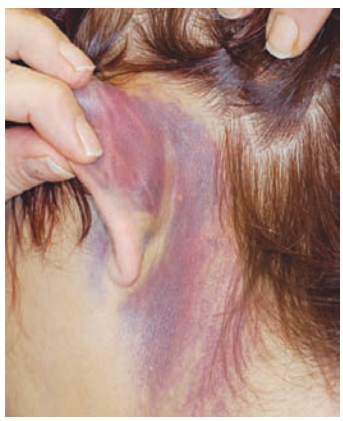

Fig.1b.Battle sign

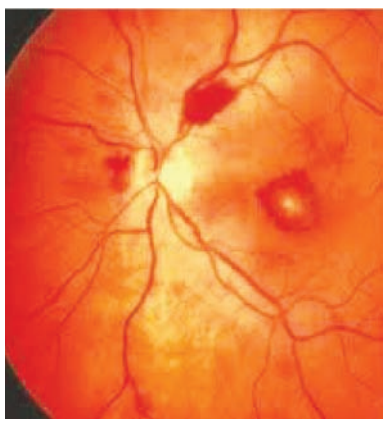

Fig.1c.Retinal hemorrhages

Table III. Various grades of head injury- Clinical features

Mild head injury	Moderate head injury	Severe head injury
PGCSs 13-15	PGCSs 9-12	PGCSs 3-8
No LOC	Brief LOC at injury, drowsy/alert currently	Responds to pain
Up to one episode of vomiting	Two / more episodes of vomiting	Unresponsive
Alert, stable	Persistent headache	Localizing signs
Scalp bruising/laceration present	Up to a single brief seizure	Signs of increased ICP
Normal examination	Large scalp hematoma/laceration	Penetrating head injury
		CSF leak from nose / ear

subdural hematoma in neuro imaging are clinical clues to suspect non accidental trauma.

Classification

Head injury in children can be classified into mild (score 13-15), moderate (score 9-12) and severe (score 3-8) head injury based on Pediatric Glasgow Coma Scale (PGCS) scores and their clinical features is given in Table III.

Investigations

Blood investigations such as complete blood count, blood sugar, grouping and typing, coagulation profile should be carried out. Non contrast CT brain with bone window is the most useful imaging study in patients with head trauma.[7] Head CT is the imaging modality of choice in children with severe traumatic head injury. By definition, all children with moderate to severe head injury have an abnormal neurologic evaluation and should have a head CT. In mild head injury whether to do CT or not is a dilemma. There are decision rules that can assist the clinician to decide about neuro imaging in children with minor head trauma. The three of the largest derived rules include Canadian Assessment of Tomography for Childhood Head rule (CATCH).[12] Childrens Head Injury Algorithm for prediction of Important Clinical Events (CHALICE)[13] and Pediatric Emergency Care Applied Research Network (PECARN) rules.[14] Only PECARN has been derived and validated among the three (Fig.2).

A period of observation may allow the physician to make a more informed decision on neuroimaging in a majority of the head-injured children. Studies have shown that a delayed presentation of severe head injury is very uncommon in children who present with only mild symptoms from minor head injury.[15, 16] There is also growing evidence demonstrating that observation before CT is safe in a large majority of patients, especially among those who present with mild symptoms.[17,18] The optimal duration of observation after head injury is however yet to be well-defined and more study is needed in this area. The indications for neuro imaging are given in Box 1 and the common findings in CT is given in Fig.3a, 3b, 3c & 3d.

Indications for skull X-ray

Skull X- rays do not give any direct information about intracranial injury, hence the indications are limited. Whenever there is a suspicion of non accidental trauma, skull radiograph is done as a part of skeletal survey. Rarely it is done to screen for fractures in selected asymptomatic patients of 3 to 24 months age with scalp hematomas (Fig.4a & 4b).

Box 1. Neuro imaging indications

- PGCS < 14
- Suspicion of open or depressed skull fracture
- Signs of basilar skull fracture
- Dangerous mechanism of injury (fall from height > 3feet or 5 steps, RTA)
- Large, boggy scalp hematoma especially non frontal
- Persistent vomiting (3 or more discrete episodes)
- Worsening headache
- Irritability at the time of examination
- Focal neurological deficit
- Suspicion of abusive head trauma
- Bleeding diathesis or on anticoagulants

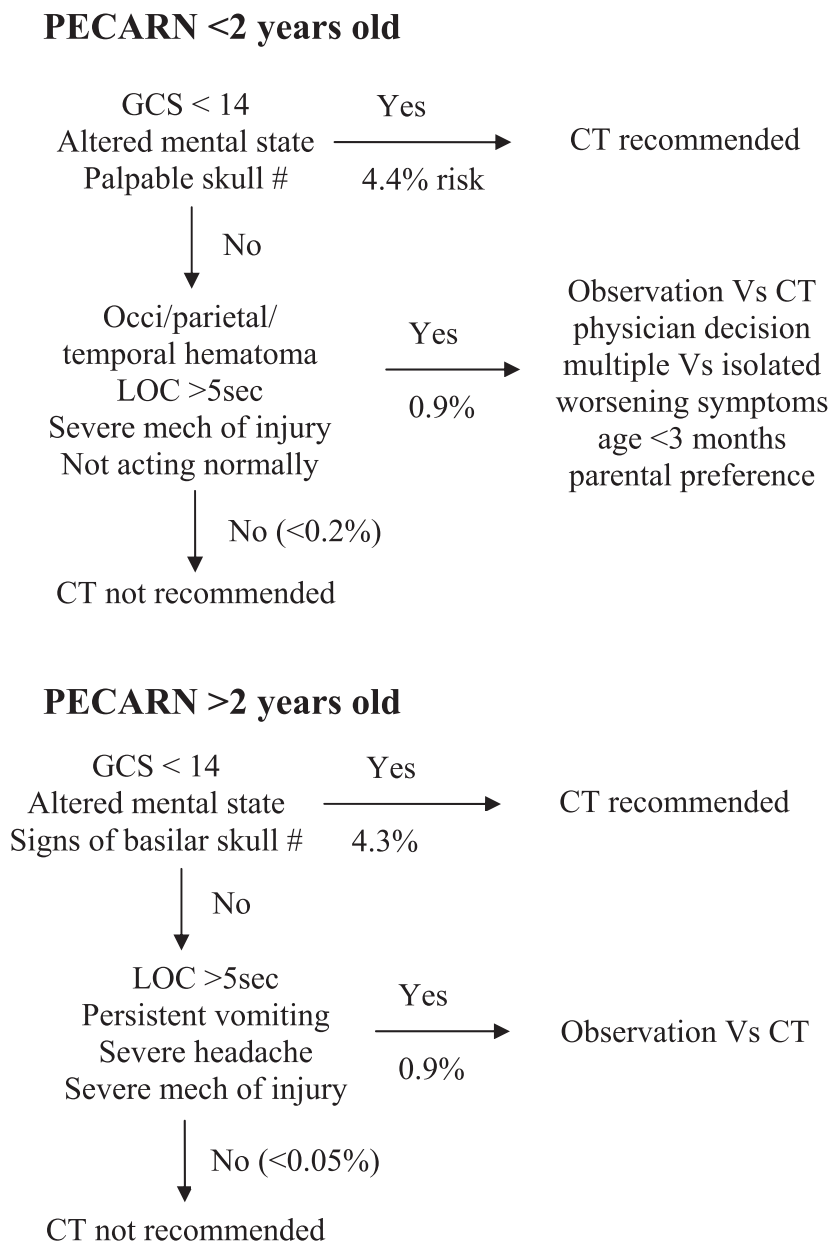

Fig.2.Decision on ordering CT based on Pediatric Emergency Care Applied Research Network (PECARN) rules.

Cervical spine X-ray - indications: X-ray cervical spine is taken whenever there is neck pain or tenderness, dangerous mechanism of injury such as fall from a height of > 3 feet or 5 stairs and high speed motor vehicle accidents.

CT cervical spine - indications: CT cervical spine is indicated in children with GCS less than 13, focal neurological signs, paresthesia in upper or lower limbs, significant bony injury in plain X rays, strong clinical suspicion of cervical injury despite normal X-rays.

Management

Increased intracranial pressure

Neuro observation: Level of consciousness, pupil size and reactivity, vitals such as heart rate, respiratory rate, blood pressure, temperature and oxygen saturation should be monitored in every child with traumatic brain injury. In children whose GCS score is less than 15, it has to be done on half hourly basis till GCS becomes 15. In children whose GCS is equal to 15, monitoring has to be done on half hourly basis for 2 hours, hourly for 4 hours and 2 hourly thereafter.

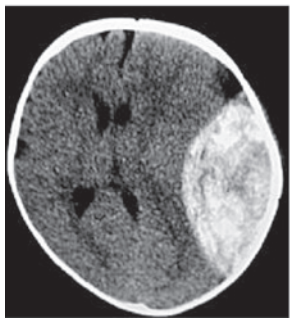

Fig.3a.Extradural hemorrhage

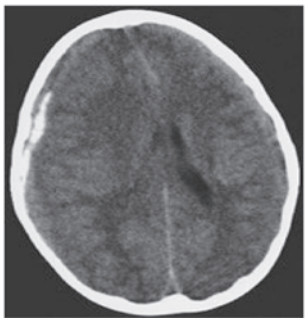

Fig.3b.Subdural hemorrhage

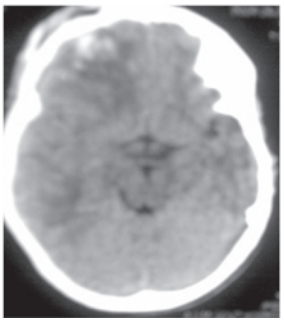

Fig.3c.Contusion

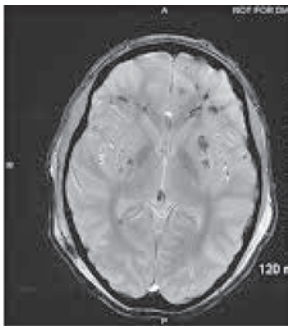

Fig.3d. Diffuse axonal injury

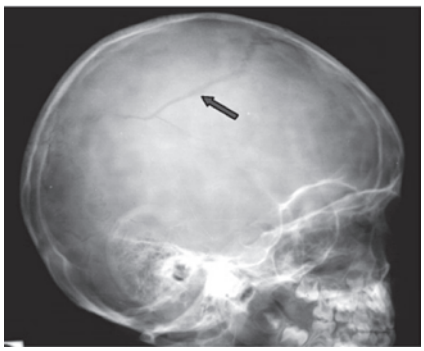

Fig.4a. Linear skull fracture

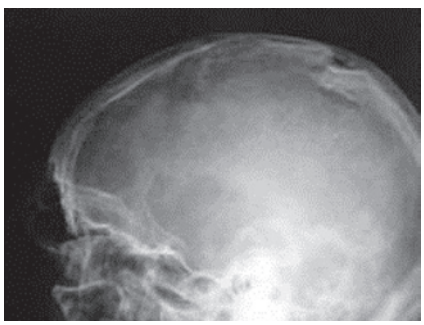

Fig.4b.Depressed fracture

Indications for neurosurgical consultation: Significant abnormality in neuro imaging, persistent coma, deterioration in GCS score, progressive neurological signs, seizures, penetrating injury and CSF leak are the indications for neurosurgical referral in children with head injury.

Maintaining the head and neck in midline to avoid the kinking and compression of the jugular vein and elevating the head end to 30 degrees to improve the venous return are useful. Hyperosmolar therapy with intravenous hypertonic saline or mannitol are useful to bring down the increased intracranial pressure. Hyperventilation as an anti edema measure should be used carefully as it decreases the ICP by vasoconstriction which can worsen already existing cerebral ischemia. Hence, it is recommended only in emergency situations such as herniation. Steroids have no effect and are not recommended in children with head injury.[7] However, in the presence of spinal cord injury, prompt use of intravenous methyl prednisolone is indicated. Barbiturates decrease ICP, provide cerebral protection and decrease the metabolic demand, but can cause severe hemodynamic compromise. Hence barbiturates are used as rescue therapy in refractory cases. Decompressive craniectomy with duraplasty is life saving in refractory cases.[7] Hypothermia decreases ICP, metabolic demand and seizures. However, benefits are not confirmed in multicentric trials. Moreover it causes rebound rise in ICP during rewarming. Careful monitoring of ICP during nursing care, clustering the nursing activities and limited handling of the patient are essential. As suctioning can increase ICP, it is better to suction only when needed. Pre-oxygenation before suctioning and use of lidocaine intravenously or intratracheally are simple measures to prevent increase in ICP when suctioning is done.

Seizures: Post traumatic seizures occur in about 10% of children with head trauma. They affect the outcome by increasing the ICP, metabolic demand, leading to hypoxia and hypoventilation. Short acting benzodiazepines are used for immediate control of seizures followed by phenytoin or phenobarbitone for maintenance. The former is preferred as it does not produce CNS depression. Though not indicated in all patients, prophylactic treatment with phenytoin or fosphenytoin may be given in children with severe TBI, intracranial hemorrhage, depressed skull fracture to prevent early post traumatic seizures.[7]

Disseminated intra vascular coagulation (DIVC): Thromboplastin from the injured brain can initiate the coagulation cascade and cause DIVC as early as 1-2 hours after surgery. Profuse bleeding can occur which is difficult

to control. One third of children with severe TBI can have DIVC.

Neurogenic pulmonary edema: Neurogenic pulmonary edema can occur 2-12 hours after head injury characterized clinically by hypoxia, hypoventilation, hypercarbia, bilateral fluffy infiltrates in X-ray chest. Medullary ischemia causes sudden massive surge in the sympathetic activity which increase the pulmonary venous pressures and shifts the blood from systemic circulation into pulmonary circulation leading onto transduction of fluid into the alveoli. High levels of PEEP may be needed to overcome pulmonary odema. However it can interfere with cerebral venous drainage and can cause increased ICP.

In cases of skull fractures with CSF leakage, pneumococcal vaccination in unvaccinated children and prophylactic antibiotics should be administered.[19]

Outcome

The overall outcome for children with head injury is better than that of adults with the same injury score.[20] Recovery in children takes longer- months to sometimes years whereas adults reach maximum recovery by about 6 months following the injury. Poor prognosticating factors include low GCS, prolonged altered level of consciousness, persistent hyperglycemia, presence of subarachnoid hemorrhage, diffuse axonal injury. Evidence of parenchymal damage is an independent poor prognosticating factor. Early sequelae include transient cortical blindness, seizures, cranial nerve palsy, diabetes insipidus, syndrome of inappropriate ADH secretion, cerebral venous sinus occlusion and hemiparesis. Late sequelae include post traumatic epilepsy, head ache, aneurysm, meningitis, memory loss and behavioral problems.

Points to remember

- ***Head injury remains the leading cause of death and disability in pediatric trauma victims.***
- ***Fall from height is the most common cause of head trauma in children.***
- ***Presence of fundal hemorrhages, fractures of various ages on skeletal survey, subdural hematoma in neuro imaging are clinical clues to suspect non accidental trauma.***
- ***It is very important to prevent head injury in children and if it occurs, should be treated aggressively.***
- ***Non contrast CT brain with bone window is the most useful imaging study in patients with head trauma.***
- ***Maintenance of adequate airway, breathing and circulation would minimize the secondary brain injury.***
- ***Prompt management of increased intracranial pressure, hypothermia, seizures and neurogenic pulmo nary odema would significantly reduce the morbidity.***
- ***The overall outcome for children with head injury is better than that of adults with the same injury scores.***

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NEWS AND NOTES

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TEN PITFALLS IN MANAGEMENT OF URINARY TRACT INFECTION

***Sudha Ekambaram**
****Vaishnavi Raman**

Abstract: *Urinary tract infection (UTI) is a common disease in infants and young children. Infants present with fever. Children have associated urinary symptoms. Immediate diagnosis and appropriate therapy prevents renal scarring, hypertension and reduced renal function. This presentation will assist the treating pediatrician to identify top clinical pitfalls in managing UTI.*

Keywords: *UTI, pyuria, uroprophylaxis, investigations, antibiotics.*

Urinary tract infection (UTI) is relatively common in children, with 8.4% of girls and 1.7% of boys having at least one episode by 7 years of age.[1] The most common pathogen is Escherichia coli, accounting for approximately 79% of UTIs in children.[2] UTI if missed or untreated can lead to renal damage/scars that can progress later to hypertension, preeclampsia, proteinuria and renal insufficiency.[3] It is the duty of primary care pediatrician to diagnose and treat UTI appropriately in children. The 10 pitfalls outlined below are common fallacies related to the management of UTI seen in day to day practice.

Are the symptoms of UTI similar in all age groups?

Symptoms of childhood UTI differ depending on the age. Fever is a common feature. Newborns can present with non-specific systemic symptoms such as hypo/hyperthermia, vomiting, refusal of feeds, jaundice, lethargy, poor weight gain and abdominal distension while infants can additionally have vomiting, poor feeding and lethargy.[4] Children usually present with typical urinary symptoms like dysuria, increased frequency, urgency, cloudy urine, hematuria, renal angle and suprapubic tenderness[5] Note that fever associated with symptoms of rhinitis, cough, wheezing, diarrhea or skin rash suggestive of a viral infection need not be investigated for UTI.

Lower urinary tract infection can present with or without low grade fever. Cystitis occurs mainly in post-pubertal girls presenting as dysuria and increased urinary frequency. Fortunately for cystitis a 2-4 day course of oral antibiotic based on local community-acquired E.coli susceptibility is likely to be effective.[6]

Is bag sample the ideal method of collecting urine in children?

A sample collected from urinary bag can be used for routine urinalysis but should not be used for urine culture. The only utility of this method is that a negative culture in bag urine sample rules out UTI. A midstream clean catch urine sample should be collected for culture in toilet-trained children; while others should have urine collected by catheter or suprapubic aspirate.[7]

Does a cloudy or smelly urine indicate UTI?

Urine colour, clarity or odour alone should not be used to diagnose or start antibiotic therapy. Foley et al., in their study concluded that visual inspection of urine was not accurate to rule out UTI after testing 100 urine samples for clarity by determining if newsprint could be legibly read through them.[8] Foul-smelling urine is an unreliable indicator of infection and is usually dependent on patient's hydration status and concentration of urea in the urine.[9]

Can absence of pyuria exclude urinary tract infection in febrile infant with urine culture positivity?

The absence of pyuria in urine sediments does not rule out UTI. One needs to look for other evidences. A small percentage (9%) of children with acute pyelonephritis (APN) can have absence of pyuria and is more common in neutropenic febrile children [10] (Reference needed). Conditions in which urine contains a significant amount of pus cells but without bacterial growth (sterile pyuria) are glomerulonephritis, renal stone disease, renal tuberculosis, dehydration and vaginits/urethritis.[11]

Does bacteria in the urine culture always indicate UTI?

The presence of bacteria on microscopic examination in the urine or a positive urine culture without UTI

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symptoms is not an indication of UTI. It could be due to contamination or asymptomatic bacteriuria which needs no treatment.[7] Common reasons for false positive urine cultures are inappropriate urine collection method in a female child, infant with diarrhoea, child with tight prepuce and poor hygiene, and adolescent girl during post menstruation days. When in doubt these children must be followed up for a few months.

What is the significant number of bacteria per ml of urine that indicates UTI?

Infection of the urinary tract is identified by growth of a significant number (10^5 CFU/mL) of organism of a single species in the midstream clean catch urine, in the presence of symptom(s).

According to American Academy of Pediatrics (AAP) guideline, significant bacteriuria in infants and children is the presence of at least 5 x 10^4 CFU/mL of a single urinary pathogen in midstream clean catch urine.[12] According to Indian Society of Pediatric Nephrology (ISPN) guideline urine culture is considered positive even if it demonstrates growth of a single bacterium with the following colony counts: (i) any growth by suprapubic aspiration (ii) >5 x 10^4 CFU/mL by urethral catheterization or (iii) >10^5 CFU/mL by midstream clean catch.[13] But a count of even 10^4 CFU/mL with symptoms is considered significant especially if the child has had antibiotics prior to culture.[14]

Should we delay treatment in UTI until urine culture report is available?

Fever with no apparent source with two or more positive rapid urine tests (positive nitrite test, positive leukocyte esterase test, pyuria (>5 pus cells/HPF) and even one bacteria in a Gram's stain) is a strong indicator of UTI and antibiotics can be introduced after sending urine culture. As E. coli is sensitive to 3rd generation cephalosporins (ceftriaxone, cefixime) or amikacin, these would be the ideal empirical drugs of choice. Antibiotics should be changed based on subsequent culture sensitivity pattern. A normal renal function needs to be documented when on amikacin.[7]

Oral antibiotics is indicated in stable children above 3 months of age. Parenteral therapy is indicated in urosepsis, bacteremia, newborns and infants less than 3 months of age, noncompliance, associated congenital anomalies of kidney and urinary tract (CAKUT) and acute kidney injury. Duration of treatment is 7–14 days depending on clinical condition.[13]

What is the greatest risk factor for recurrent UTI in children?

Bowel bladder dysfunction (BBD) is the most common risk factor for UTI seen in children nowadays. BBD presents with a broad spectrum of manifestation which includes lower urinary tract symptoms like frequency, urgency and dysuria associated with bowel dysfunction in the form of constipation / encopresis. Correction of voiding dysfunction and constipation leads to a decrease in UTI recurrence.[13]

When is circumcision indicated in children with UTI?

According to AAP Task Force, circumcision is indicated in children with recurrent UTI or vesicoureteric reflux (VUR).[15]

Young infant with grade IV reflux. Is prophylaxis indicated? What is the drug of choice?

In view of the Randomized Intervention for Children with Vesicoureteral Reflux (RIVUR) trial results, the debate on antimicrobial prophylaxis has moved from "no prophylaxis" to "selective prophylaxis" in children with VUR. [16] The decision to give antibiotic prophylaxis in children with reflux still remains a clinical dilemma. Emerging evidence indicates that not all children diagnosed with VUR require antibiotic prophylaxis. Neonates with antenatally diagnosed hydronephrosis, infants with all grades of VUR, low-grade (I or II) VUR with recurrent febrile UTIs, children with high-grade (III–V) VUR, BBD, or renal cortical abnormalities can be given antibiotic prophylaxis (Table I). Afebrile grade I or II VUR can be observed for spontaneous resolution without prophylaxis.

Drugs used for prophylaxis in recurrent UTI should have low serum and high urinary level, wide spectrum activity, least effect on fecal flora, minimal side effects and low bacterial resistance. Ampicillin, amoxicillin and cephalexin are appropriate prophylactic drugs in children less than 3 months. The typical dose is one fourth of the therapeutic dose given once daily in the evening to

Table I. VUR prophylaxis–A practical approach as per ISPN guidelines

VUR - Grades	Uroprophylaxis
I, II	6 months – 1 year
III, IV, V	Till 5 years of age
Associated BBD	Till BBD normalizes irrespective of grade

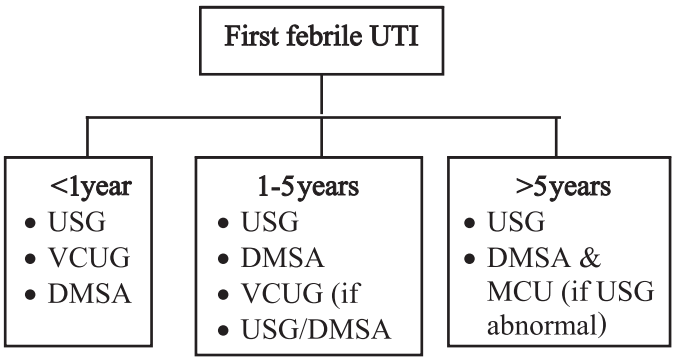

Fig.1. Evaluation following initial UTI as per ISPN guidelines

maximize overnight drug levels in the bladder. Nitrofurantoin (NFT), cotrimoxazole and cephalexin are appropriate drugs in children older than 4 months. The discontinuation of NFT by parents is a possibility in view of its gastrointestinal complications. Ampicillin and amoxicillin, because of increasing antimicrobial resistance, are not recommended as the first choice and beyond 2 months of age.

Yet another clinical dilemma is about imaging protocol. Renal ultrasonogram evaluation is the standard tool for evaluating children with a first febrile UTI as per ISPN guidelines (Fig 1). It could identify obstructive uropathy, hydronephrosis, high grade VUR, dilated collecting systems and anomalies of the kidneys. Second is the voiding cystourethrogram (VCUG) for diagnosing VUR and for assessing the degree of VUR and posterior urethral valve in male children. Third is dimercatosuccinic acid (DMSA) scan which can diagnose acute pyelonephritis during acute illness and renal scars when performed 3 months following the acute illness. Recently there are suggestions to do DMSA earlier than VCUG. A VCUG can be avoided if the DMSA shows no nucelopenic areas indicating pyelonephritis or scars. This can avoid unnecessary exposure to radiation as in VCUG. In recurrent UTI at any age we need to evaluate with USG, DMSA and VCUG.

Conclusion

Pediatricians are the primary care physicians involved in the diagnosis and management of children with UTI. There are many pitfalls in the interpretation of patient's symptom, lab values and treatment that leads to either over or under treatment of UTI. Not only a good clinical history and supportive laboratory data but also clear understanding about the common pitfalls will help the treating doctor to manage UTI in children better and prevent the complications associated with it.

Points to Remember

- ***Early diagnosis and prompt treatment of UTI will reduce renal damage.***
- ***Bag sample is not the method of choice for urine culture.***
- ***Diagnosis of UTI is not based on a single factor but collective factors like symptoms, pyuria and urine culture.***
- ***BBD is the common risk factor noted for breakthrough UTI.***
- ***Uroprohylaxsis is not a universal therapeutic choice.***

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CLIPPINGS

Impact of the US Maternal Tetanus, Diphtheria, and Acellular Pertussis Vaccination Program on Preventing Pertussis in Infants <2 Months of Age: A Case-Control Evaluation.

A total of 240 cases and 535 controls were included; 17 (7.1%) case mothers and 90 (16.8%) control mothers received Tdap during the third trimester of pregnancy. The multivariable Vaccine effectiveness (VE) estimate for Tdap administered during the third trimester of pregnancy was 77.7% (95% confidence interval [CI], 48.3%-90.4%); VE increased to 90.5% (95% CI, 65.2%–97.4%) against hospitalized cases.Vaccination during pregnancy is an effective way to protect infants during the early months of life. With a continuing resurgence in pertussis, efforts should focus on maximizing Tdap uptake among pregnant women.

Skoff TH, Blain AE, Watt J, Scherzinger K, McMahon M, Zansky SM, et al. Impact of the US Maternal Tetanus, Diphtheria, and Acellular Pertussis Vaccination Program on Preventing Pertussis in Infants <2 Months of Age: A Case-Control Evaluation. Clin Infect Dis. 2017 Nov 29;65(12):1977-1983. doi: 10.1093/cid/cix724.

NEWS AND NOTES

PEDIATRIC PULMONOLOGY UPDATE - 2018

Pediatric Pulmonology Foundation Chennai & Indian Academy of Pediatrics National Respiratory Chapter

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DRUG PROFILE

ANTIARRHYTHMIC AGENTS

***Jeeson C Unni**
****Ranjit Baby Joseph**
***** Sajana TM**

Abstract: *Symptomatic cardiac arrhythmias are relatively uncommon in childhood, accounting for about 5% of the emergency hospital admissions. Atrial tachyarrhythmias are the most common rate and rhythm disturbance in this population. With the advent of effective ablation therapy, management of arrhythmia has undergone dramatic change. A clear understanding of the mechanisms that initiate the rhythm disturbances and the various pharmacological agents that are used would enable optimal management of arrhythmias. Some of the agents that are licensed for use in children are discussed in this article.*

Keywords: *Arrhythmias, Action potential, Supraventricular tachycardia, Ventricular tachycardia.*

Cardiac rhythm is maintained with the help of cardiac action potential. The resting membrane potential of cardiac cells is maintained mainly by potassium ions. Complex interplay of various cations like sodium, potassium and calcium results in generation of action potential.[1] Any deviation in the normal pattern of cardiac rhythm results in arrhythmia. The various drugs used in the management of cardiac arrhythmia focus on stabilisation of cardiac action potential by acting at different levels. In general, antiarrhythmic drugs are classified into four categories based on their mechanism of action (Vaughan Williams classification) but this classification is not of great clinical relevance (Table I).[2]

Antiarrhythmic drugs can also be classified clinically as those acting on i) supraventricular arrhythmias, ii) supraventricular and ventricular arrhythmias and iii) ventricular arrhythmias (Table II). The negative inotropic effect of these agents tends to be additive. Hence, special care should be taken if two or more drugs are prescribed especially if myocardial function is impaired. Most of the antiarrhythmic drugs can provoke arrhythmias especially when there is an underlying hypokalemia.[3]

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Class I A agents

This group of drugs impair the entry of sodium into the cells and exert a membrane stabilizing effect which ultimately slows the rate of depolarization causing reduction in the excitability of atrial and ventricular tissue, allowing the SA node to regain dominance of the cardiac rhythm. It also prolongs the effective refractory period and abolishes the impulse re-entry by blocking the cardiac potassium channels and thus delaying repolarization. The resultant QT prolongation can be pro-arrhythmic and can sometimes precipitate Torsade de Pointes. The negative inotropic effects make it unsafe for use in patients with structural heart disease. Quinidine, procainamide and disopyramide are the drugs which belong to this class.[4]

Quinidine

It is derived from cinchona. It delays depolarization and repolarization by Na^+ channel and inward rectifier potassium (IKi) channel blocking effects respectively. It is effective in atrial arrhythmias such as atrial flutter and fibrillation, re-entry SVTs and ventricular arrhythmias such as VT. Oral and IV formulations are available and ideally needs a test dose to assess for idiosyncratic reaction.

Dosage[5]: IV- As quinidine gluconate: 2-10mg/kg/dose 3-6 hourly. PO- As quinidine sulphate: 15-60mg/kg/day in divided doses 6^{th} hourly. Toxicity is indicated by increase in QRS interval by>0.02 sec.Therapeutic levels: 3-7mg/L.

Side effects include GI symptoms, hypotension, tinnitus, TTP, rash, heart block and blood dyscrasias. When used alone, it may cause 1:1 conduction in atrial flutter leading to ventricular fibrillation. It can also cause idiosyncratic ventricular tachycardia with low levels, especially when initiating therapy. Its use has declined because of the potent side effects mainly thrombocytopenia, hemolytic anemia, abdominal cramps, diarrhea, decreased hearing and tinnitus, QRS widening and proarrhythmia.[6]

Table I. Anti-arrhythmic drugs - Vaughan Williams classification

Class	Mechanism of action	Drugs
I	Fast sodium channel blockers	
Ia	Prolong repolarization	Quinidine, procainamide, disopyramide
Ib	Shorten repolarization	Lidocaine, mexiletine, phenytoin, tocainide
Ic	Little or no effect on repolarization	Flecainide, encainide, moricizine, propafenone
II	Beta blockers	Propranolol, esmolol, metoprolol
III	Potassium channel blockers	Amiodarone, sotalol, dofetilide, ibutilide
IV	Slow calcium channel blockers	Verapamil, diltiazem
V	Miscellaneous drugs	Digoxin, adenosine, magnesium

Table II. Anti-arrhythmic drugs - Clinical classification

Type of arrhythmia	Drugs preferred
Supraventricular	Adenosine, digoxin, verapamil
Supraventricular and ventricular	Amiodarone, beta blockers, flecainide, procainamide
Ventricular	Lidocaine

Procainamide

Procainamide has both Na^+ and K^+ channel blocking effects. The onset of action is between 10 to 30 minutes.[7] It is usually given as several small slow intravenous boluses, with close monitoring for hypotension and cardiac depression. It is the drug of choice in the treatment of post-operative junctional ectopic tachycardia (JET).[8,9] It is reported to be more effective than amiodarone in a pediatric cohort with a variety of SVT including orthodromic reciprocating tachycardia, intra-atrial re-entrant tachycardia (IART) and ectopic atrial tachycardia (EAT).[10] Other indications include atrial flutter, atrial fibrillation and AV nodal re-entrant tachycardia (AVNRT).[11-13]

Dosage: PO: 20–100 mg/kg/day in 6 divided doses. Maximum: 4 g/24 hour. IV: 3–6 mg/kg/dose IV over 5 min and can be repeated every 5 min to a maximum of 15 mg/kg. Maintenance: 20–80 mcg/kg/min as IV infusion.

Side effects include hypotension, arrhythmias (including QT prolongation and Torsades des Pointes), lupus-like syndrome (usually reversible upon discontinuation) and blood dyscrasias (e.g. agranulocytosis).[11,14,15]

Disopyramide

This has similar action as quinidine without the adrenergic effects. It is effective in refractory vasodepressor syncope.[16] The negative inotropic effect inhibits the ventricle from becoming hypercontractile which is the trigger for the vasodepressor reflex. The dose used is 20-30 mg/kg/day in 3-4 divided doses. Side effects include anticholinergic effects such as urinary retention, constipation, blurred vision and dry mouth. Renal clearance may be affected by betablockers.

Class 1B

These drugs shorten action potential duration and repolarization by blocking the fast Na channel activity. It includes lidocaine, mexilitine, phenytoin, tocainide and moricizine.

Lidocaine

It may be used in cardiopulmonary resuscitation in children with ventricular fibrillation especially in Torsade de Pointes or pulseless ventricular tachycardia unresponsive to DC. shock, but only if amiodarone is not available. Dose needs to be reduced in children with persistently poor cardiac output and hepatic or renal failure.[17]

Dosage: [18] IV/IO: Neonates and children 1 month-12 years: 0.5-1mg/kg as bolus followed by infusion of 0.6-3mg/kg/hour.

Side effects include CNS effects like dizziness, confusion, paresthesia, altered sensorium, respiratory depression, convulsions, hypotension and bradycardia.

Contraindications include all sinoatrial disorders, all grades of atrio ventricular block, severe myocardial depression and acute porphyria.

Mexiletine

This drug, the oral form of lidocaine with similar Na channel effects, is effective in chronic therapy of some VTs and also long QT syndrome (LQTS [type III]).[19] The safety and efficacy in children are not well documented. The most common side effects associated with mexiletine therapy have been gastrointestinal and nervous system effects but is usually well tolerated.

Dosage: 1.4 to 5 mg/kg/dose given every 8 hours. It is ideal to begin with lower initial dosages and titrated to desired effects and serum concentrations.[20]

Mexiletene is an useful drug for the long term control of ventricular arrhythmias in children with congenital heart disease; much less effective in the settling of cardiomyopathy and least effective in those with normal heart. Contraindications to its use are congestive heart failure (CHF), hypotension and a history of seizures. Its use is not recommended in less severe arrhythmias like asymptomatic ventricular premature contractions or conduction disturbances. Side effects are minimal and mild.[21]

Phenytoin

It has similar effects as lidocaine on the Na channel but also has Ca channel blocking and sinus node and AV node effects in higher concentrations. Its effect of depressing phase 4 depolarization is used in treatment of digoxin toxicity. It is used in some forms of refractory ventricular arrhythmias. It crosses the placental barrier and is known to have a potent teratogenic effect on fetus.[22]

Dosage[23]: Loading dose (all ages): 1.25mg/kg IV every 5 minutes up to a total of 15mg/kg. Maintenance dose: Child (IV/PO): 5-10mg/kg/day in 2-3 divided doses.

Side effects include hypotension, nystagmus, ataxia, gingival hyperplasia (on prolonged use) and skin rash (may progress to Steven Johnson's syndrome). It is contraindicated in patients with heart block or sinus bradycardia. Sudden death late after surgery for congenital heart disease is usually attributed to ventricular dysrhythmias, which may be difficult to suppress. Phenytoin orally was found to be effective in suppressing ventricular dysrhythmias especially VPCs after surgery for congenital heart disease.[24]

Class I C

These agents are potent in blocking Na and affecting repolarization. They also have an effect on inward K channel and the slow inward Ca current. They include flecanide, propafenone and encanide.

Flecainide

It prolongs the QRS duration by acting on the bundle of His, purkinje system and ventricular myocardium. It has a negative inotropic action and may induce ventricular arrhythmia in patients with significant myocardial dysfunction.

Flecainide can be used for termination of acute SVT as well as can be used for prophylaxis of SVT in cases refractory to conventional drugs like digoxin, betablockers and calcium channel blockers. It is particularly useful for treatment of automatic atrial tachycardia, JET, atrial flutter, postoperative intra-atrial re-entrant tachycardia and VT.[1]

Dosage[3]: In cases of resistant re entry supraventricular tachycardia, ventricular ectopics or ventricular tachycardia, arrhythmias associated with accessory conduction pathways (WPW syndrome), paroxysmal atrial fibrillation- Oral: Neonate- 2 mg/kg 2-3 times daily. Dose to be adjusted according to response and plasma flecainide levels. Child 1month to 12 years: 2 mg/kg 2-3 times daily. Dose to be adjusted according to response and plasma flecainide levels (maximum 8 mg/kg/day or 300 mg/day). Child 12-18 years: initially 50-100 mg twice daily; maximum 300 mg/day.

By slow IV injection/ infusion in cases of acute episode of SVT: Neonate- 1-2 mg/kg over 10-30 minutes. If necessary followed by continuous infusion at the rate of 100-250 mcg/kg/hour until arrhythmia is controlled. Child 1month- 12 years: 2 mg/kg over 10-30 minutes. If necessary can be followed by continuous infusion at the rate of 100-250 mcg/kg/hour until arrhythmia is controlled. Child 12-18 years- 2 mg/kg (maximum 150 mg) over 10-30 minutes. If necessary it has to be followed by continuous infusion at a rate of 1.5 mg/kg/hour for 1 hour, then reduced to 100-250 mcg/kg/hour until arrhythmia is controlled.

Flecainide is safe and effective in children with supraventricular tachycardia. The drug was found to be very effective for treatment of fetal tachyarrhythmias. It may not be safe for children who have structurally abnormal heart and atrial flutter or ventricular arrhythmias. The safety of flecainide for patients with ventricular arrhythmias and normal heart requires further investigation.[25]

Side effects include body ache, asthenia, tremors, headache, fatigue, agitation and gastrointestinal upset. The most dreaded side effect is proarrhythmia, seen in 7%-8% of cases which is more likely if there is myocardial ischemia or ventricular dysfunction.[1] In children it has also been reported to cause incessant supraventricular tachycardia[26,27,28] cardiac arrest or sudden death[27,28] and non-sustained ventricular tachycardia.[29] The incidence of arrhythmogenesis is said to be lower in children than in adults.[30] Though the arrhythmogenic effects of flecainide (and encainide) in children is to be reported in 5%-7%[28] and found to be equally common in those with and those without structural heart disease,[29] in further review it has been suggested that flecainide is effective and probably safe in children with supraventricular tachycardia and structurally normal hearts.

Contraindications include heart failure, abnormal LV function, long standing atrial fibrillation where conversion to sinus rhythm is not attempted, hemodynamically significant valvular heart disease, sinus node dysfunction and atrial conduction defects.

Monitoring: Flecainide should be started in a hospital setting. The QRS duration needs to be monitored meticulously. A 10% increase is expected. An increase in QRS duration of more than 25% during flecainide therapy is a sign of potential risk of proarrhythmia and the drug should be stopped or the dose reduced. Plasma levels of flecainide should be monitored ideally, especially in hepatic or renal impairment, but this facility is not yet available in India.

Propafenone

In addition to blocking Na^+ channels, propafenone also has β adrenergic blocking and weak Ca^{2+} channel blocking activities resulting in prolongation of the refractory periods in the atrium and ventricle as well as slowing conduction at the AV node. It is effective in the treatment of JET, EAT, AVNRT, atrial flutter, AV reciprocating tachycardia (AVRT) and chaotic atrial tachycardia, but is generally used as a second line agent after other therapies have failed.[31]

Dosage: Oral: 200 mg/M^2 /day or 7-10 mg/kg/day in 3 divided doses. The dose is increased every 3 days by 20%–30% if needed to a maximum of 500 mg/M^2/day or 18-20 mg/kg/day. IV: 0.2 mg/kg/dose every 10 minutes to a maximum of 2 mg/kg followed by infusion at 4–7 mcg/kg/min.

Side effects include hypotension with intravenous administration, vomiting and unpleasant taste with oral administration, transient blurred vision, elevations in liver enzymes, edema, and rarely agranulocytosis. Electrophysiologic adverse effects include QRS and PR widening, sinus node dysfunction, AV block, bradycardia, arrhythmias and QT prolongation.

Class II agents

Class II drugs reduce sympathetic activity which are known to be pro-arrhythmic and also propagate re-entry mechanisms. Beta blockade reduces the spontaneous firing rate of the SA node and ectopic pacemakers, prolongs intra nodal conduction and the refractory period of AV node. This results in a negative chronotropic effect reducing cardiac work. Beta blockers are effective in prophylaxis of SVT by inhibiting the initiating atrial ectopic beat. Class II drugs include beta-1 cardiac selective (atenelol, metoprolol), non selective (propranolol, nadalol) and drugs that have intrinsic sympathomimetic activity (Pindalol).

Propranolol

Propranolol is the drug of choice in LQTS and also the most common beta blocker used for chronic prophylaxis of SVT, the first line drug in re-entry SVT of all ages (including newborn), some ventricular arrhythmias (especially catecholamine sensitive).[32] It has membrane effects on Na^+ channel and weak Ca^{++} channel effects with higher doses. Though there is little effect on the action potential duration, it prolongs intranodal conduction (increases AH interval and Wenckebach block).

Dosage[3]: Oral: Neonates- 250-500mcg/kg 3 times daily, adjusted according to response. Child 1month - 18years: 250-500 mcg/kg 3-4 times daily, adjusted according to response to a maximum of 1 mg/kg 4 times a day, total daily dose not exceeding 160 mg daily.

IV: By slow injection, with ECG monitoring: Neonate: 20-50 mcg/kg repeated if necessary every 6-8 hours. Child 1 month - 18 years: 25-50 mcg/kg repeated every 6-8 hours if necessary.

Side effects include GI disturbances, bradycardia, heart failure, hypotension, conduction disorders, peripheral vasoconstriction and bronchospasm.

It is contraindicated in children with asthma, uncontrolled heart failure, marked bradycardia, hypotension, sick sinus syndrome, second or third degree AV block, cardiogenic shock, metabolic acidosis, severe peripheral arterial disease and pheochromocytoma.

Esmolol

Esmolol is a relatively cardioselective beta blocker

with a very short duration of action, used intravenously for short term treatment of supraventricular arrhythmias and sinus tachycardias particulary in the perioperative period. Contraindications and the side effects are similar to that of propranolol.

Dosage[3]: IV: Child 1 month – 18 years: 500 mcg/kg through a central venous catheter over 1 minute followed by infusion of 50 mcg/kg/min for 4 minutes. If inadequate response, the loading dose is repeated, maintenance infusion increased by increments of 50 mcg/kg/min until effective or maximum infusion of 200 mcg/kg/min is reached.

Metoprolol

Though Metoprolol is not considered as a first line beta blocker, it can also be used in selected patients.

Dosage: Oral: Child 1 month- 12 years: 1 mg/kg twice daily, increase if necessary to 8 mg/kg/day (maximum 400 mg) in 2-4 divided doses. Child 12-18 years: initially 50-100 mg daily increased if necessary to 200 mg daily in 1-2 divided doses; maximum 400 mg daily.[3]

Class III agents

Class III agents prolong the repolarisation phase by reducing the K+ efflux out of the cells. The time interval required for re-excitation is prolonged and hence arrhythmias are suppressed. The predominant advantage is that it can be used even in the presence of left ventricular dysfunction. Significant QT prolongation and attendant risk of Torsades des Pointes is an adverse effect to be watched for.[1] Amiodarone, sotalol, ibutalide and dofetalide belong to this class.

Amiodarone

Amiodarone has been described as close to being the ideal antiarrhythmic agent in SVT and VT in children. Even though it is primarily a class III anti-arrhythmic drug, it has other class effects also. The predominant effect after IV administration is due to its betablocking and calcium channel blocking actions. The onset of action depends on the route of administration. As interactions with numerous drugs are known, careful monitoring is required.[1]

It is commonly used as first line therapy to manage JET and other supra ventricular arrhythmias, particularly in patients with structural cardiac abnormalities or decreased cardiac output. It is also indicated for difficult to control SVT, which may be due to AVRT, AVNRT, atrial flutter or AF, automatic EATs and life threatening ventricular arrhythmias. It has also been successfully used in combination with flecainide[33] and propranolol[34] for refractory tachyarrhythmias in infants and children.

Dosage[1]: IV - Loading dose is 5 mg /kg given over 20- 30 min, followed by 5-15 mcg/kg/min infusion. In rare cases, a higher loading dose, up to a maximum of 15 mg/kg, can be given. Oral - Loading dose is 5 mg/kg given 2-3 times a day (maximum 200 mg/dose) for 5 days followed by 5 mg/kg/day as a single dose.

Side effects: IV amiodarone may result in hypotension, nausea, sweating and hot flushes. Chronic use of oral amiodarone can cause cardiac effects like bradycardia, prolongation of QT interval, myocardial depression, endocrine effects like hypothyroidism or hyperthyroidism, pulmonary complications like pulmonary alveolitis, pneumonitis and fibrosis, neurological effects like peripheral neuropathy, vertigo, headache, insomnia and dermatological effects like rashes, photosensitivity etc., Other side effects seen occasionally are ocular changes like corneal deposits, optic neuritis and hepatic dysfunction. Amiodarone decreases the clearance of digoxin, flecainide, procainamide and warfarin. There is increased risk of ventricular arrhythmias when given with erythromycin.

Monitoring: BP monitoring is mandatory when using IV amiodarone, especially in patients with ventricular dysfunction. Periodic ECGs must be done to look for QT interval changes. Testing for thyroid functions (before starting amiodarone, after loading dose and 6 monthly), liver functions (before, after loading and 6 monthly), pulmonary functions, chest X-ray (before and 3-6 monthly later) and slit lamp examination of the eyes should be carried out periodically. Amiodarone, though a very effective antiarrhythmic agent, is not really recommended especially for chronic use. Unfortunately due to difficulties in procuring other, safer antiarrhythmics, amiodarone is being widely used in India. It should ideally be reserved for refractory arrhythmias which are not responding to simpler medications.[1]

Sotalol

It is a non- cardioselective betablocker with additional class III anti-arrhythmic activity. Thus it is a K+ channel blocker as well as non-selective â adrenergic blocker. At lower doses the β adrenergic effects predominate whereas the K+ channel effects predominate at higher doses.[31] It can suppress ventricular ectopic beats and non-sustained ventricular tachycardia. It prolongs action potential duration and results in lengthening of QTc interval. It also exerts a negative inotropic and chronotropic effect and reduces AV nodal conduction.

The pro-arrhythmic effects of sotaolol may prolong the QT interval and induce Torsade de Pointes especially in children with sick sinus syndrome.[1, 3]

Sotalol is indicated for refractory atrial tachyarrhythmia and arrhythmias in postoperative patients. It is superior to class I agents for ventricular arrhythmias and preferred over amiodarone due to less serious side effects. It is also safe to use in fetal arrhythmias.[1]

Dosage: Sotalol is given orally in a dose of 2-4 mg/ kg/day in two divided doses. Doses up to 8 mg/kg/ day have been used. Body surface area is reported to be a better predictor for sotalol dosing; the recommended dose is 30-70 mg/ M^2/day.[1]

Side effects: Sotalol is a relatively safe drug apart from its proarrhythmic effect seen in 3%-5%, due to prolongation of QTc interval. Bronchospasm may occur in predisposed patients, due to its beta blocking effect. Sotalol has also been associated with proarrhythmia, including bradycardia, QT prolongation, and Torsade de Pointes.[35]

Class IV agents

These agents block the cardiac Ca^{2+} channels and decreases cardiac conduction velocity by prolonging repolarization at the AV node. They are contraindicated when preexcitation is manifest, as they can facilitate antegrade conduction during atrial fibrillation leading to ventricular fibrillation and arrest. Verapamil and diltiazem are drugs in this group.

Verapamil

Verapamil is the most selective agent for the myocardial Ca^{2+} channels. It is used to treat atrial flutter, atrial fibrillation and AVNRT in children. It can also be used as a second line agent for SVT unresponsive to first line therapies. Verapamil is contraindicated in infants less than one year old because it has been associated with increased apnea, bradycardia, hypotension and cardiac arrest in this age group. It should not be used in patients with decreased cardiac function or in those receiving β-blockers because of the risk of bradycardia and AV block.[3, 29]

Dosage: In Supra ventricular arrhythmias: IV over 2-3 minutes: Child 1-18 years: 100-300 mcg/kg (maximum 5 mg) as a single dose, repeated after 30 minutes if necessary.

Side effects include flushing, rash, worsening heart failure, seizures, increased liver enzymes, tinnitus, vertigo, dyspnea and bronchospasm. Diltiazem blocks the inward Ca channel activity and has predominant effect on sinus and AV node. It has been used predominantly in the control of hypertension and acute treatment of SVT. Its use is similar to verapamil and dosing recommendations are 0.5-2 mg/kg/day in 2-3 divided doses. Side effects include bradycardia and postural hypotension.

Class V agents (Miscellaneous)

These include miscellaneous drugs like digoxin, adenosine and magnesium which act by various mechanisms as anti arrhythmics.

Digoxin

Digoxin has both cholinergic and anti adrenergic effects which serve to slow the sinus rate and AV node conduction by prolonging the effective refractory period. It also blocks Na^+/K^+ adenosine triphosphatase and increases intracellular Ca^{2+}, which likely is responsible for its inotropic properties but may also contribute to its proarrhythmic effects.[36]

Oral administration of digoxin slows the ventricular rate in atrial fibrillation and in atrial flutter. However, intravenous infusion is rarely effective for rapid control of ventricular rate. Now a days it is not commonly used in SVT where adenosine is used more frequently.

Adenosine

Adenosine is the drug of choice for terminating supra ventricular tachycardias including those associated with accessory conducting pathways like WPW syndrome. It does not cause significant hypotension. It can be used safely in children with impaired cardiac function or post-operative arrhythmias. The injection should be rapidly administered into a central or a large peripheral vein.[1]

It is effective in the termination of sinus node re-entry tachycardia and in re-entrant tachycardias that use the AV node as a part of the re-entrant mechanism. Tachycardias originating in the atrium (e.g. atrial fibrillation, atrial flutter, EAT, IART) do not rely on the SA or AV node and thus are not typically terminated by adenosine. EAT has a variable response to the administration of adenosine and may produce transient termination or rate slowing, which is thought to be secondary to adenosine's anti adrenergic effect.[37]

Dosage[3] : IV/IO dose: First dose 0.1 mg/kg rapid bolus (max: 6 mg), second dose: 0.2 mg/kg rapid bolus (max: 12 mg).[38]

Beyond neotatal period : 150 mcg/kg, if necessary repeat injection every 1-2 minutes increasing dose by 50-100 mcg/kg until tachycardia is terminated or maximum single dose of 300 mcg/kg given.

Contraindications include second or third degree AV block and sick sinus syndrome, severe hypotension, decompensated heart failure and asthma. Side effects though rare but include nausea, AV block, flushing, angina, dizziness, dyspnoea, headache and palpitations.

Magnesium

Magnesium sulphate injection is recommended for the emergency treatment of serious arrhythmias, especially in the presence of hypokalemia and when the ventricular tachycardia shows the characteristic twisting wave front known as Torsade de Pointes.[3]

Dosage: IV-Over 10-15 minutes. Child 1 month- 18 years: 0.1-0.2 mmol/kg (25-50 mg/kg magnesium sulphate heptahydrate); maximum 8 mmol (2 gm magnesium sulphate heptahydrate); dose repeated once if necessary.

Conclusion

Pharmacotherapy of arrhythmias in children is very effective in situations where ablation is not preferred. Side-effects of different drugs and combinations, drug interactions and patient compliance need to be taken into consideration and monitored during treatment. Initiation of the drug should always be done in a hospital setting, preferably by a pediatric cardiologist who is well acquainted with these drugs.

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CLIPPINGS

Randomized Double-blind Trial of Ringer Lactate Versus Normal Saline in Pediatric Acute Severe Diarrheal Dehydration.

The aim of this study was to compare the effectiveness of Ringer Lactate (RL) versus normal Saline (NS) in the correction of pediatric acute severe diarrheal dehydration, as measured by improvement in clinical status and pH (≥ 7.35).The primary outcome was an improvement in clinical status and pH (≥ 7.35) at the end of 6 hrs. Secondary outcome measures were changes in serum electrolytes , renal and blood gas parameters, the volume of fluid used for rehydration excluding the first cycle, time to start oral feeding, hospital stay and cost effectiveness analysis.Primary outcome was achieved in 38% (relative risk=1.63, 95% confidence Interval 0.80-3.40) in RL and NS groups, respectively. No significant difference were observed in secondary outcomes in electrolytes , renal and blood gas parameters.Study concluded that in pediatric acute severe dehydration , resuscitation with RL and NS was associated with similar clinical improvement and biochemical resolution.Hence .NS to be considered as the fluid of choice because of the clinical improvement, cost and availability.

Kartha GB, Rameshkumar R, Mahadevan S. Randomized Double-blind Trial of Ringer Lactate Versus Normal Saline in Pediatric Acute Severe Diarrheal Dehydration. J Pediatr Gastroenterol Nutr 2017 Dec; 65(6):621-626. doi:10.1097/MPG.0000000000001609.

SURGERY

MEDIASTINAL TUMOURS IN CHILDREN - AN INSIGHT

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Abstract: *Common complaints for which children seek pediatric consultation are recurrent lower respiratory tract infection, wheeze, respiratory distress with or without fever and pneumonia. Pediatricians should be diligent to unmask a mediastinal tumour that may underlie a persistent or recurrent pneumonia. Chest X-ray in these children may give a clue for further evaluation. The earlier the diagnosis of mediastinal tumour is made, better the long term outcome. This review article highlights the common presenting features of mediastinal tumours and approach to their management.*

Keywords: *Bronchopneumonia, Mediastinal tumour, Thoracic neuroblastoma.*

Mediastinal tumours in children include a wide spectrum of histopathological lesions, of which 65%-72% are malignant.[1,2] Mediastinal lesions can be asymptomatic or present with common respiratory symptoms like cough, wheeze, chest pain, hemoptysis, respiratory distress with or without fever. Mediastinal lesions due to lymphoproliferative diseases can present acutely with fever, weight loss or night sweats in addition to the respiratory symptoms.

Though pediatricians encounter these common respiratory symptoms in day to day practice, it is the persistence of these symptoms that should alert the treating physician to evaluate further with the help of imaging facilities.

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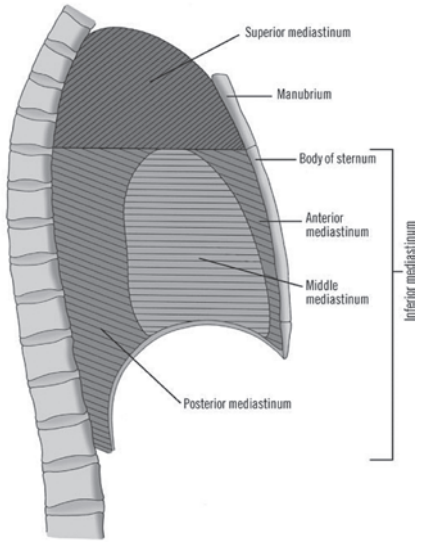


Fig.1.Subdivisions of mediastinum

Mediastinal anatomy

Mediastinum is an anatomical space situated between the thoracic inlet superiorly, the diaphragm inferiorly, hila of the lungs laterally, sternum anteriorly and vertebral bodies posteriorly. Mediastinum can be divided into superior mediastinum and inferior mediastinum by an imaginary line extending from the sterno-manubrial joint anteriorly to the lower border of fourth thoracic vertebra posteriorly (Fig.1).

Superior mediastinum extends from thoracic inlet up to on imaginary line between angle of Louis and fourth thoracic vertebra. Inferior mediastinum which is below the superior mediastinum can be further divided into anterior, middle and posterior mediastinum. Anterior mediastinum extends from sternum up to the pericardium. Middle mediastinum includes pericardium, heart and hilar structures of both lungs. Posterior mediastinum extends between pericardium and pre vertebral fascia. The anatomical structures located in the mediastinum classically give rise to the lesions, which when imaged and evaluated provide the type of lesion and tumour. This helps in the diagnosis and management (Table I).

In children, 44% of lesions arise in the antero superior mediastinum as reported by Grosfeld et al.,[1] and they include lymphoma, teratoma, germ cell tumours and cystic

Table I. Common mass lesions in mediastinum

Anterior	Middle	Posterior
Thymus	Pericardial teratoma	Neurogenic tumours
Hyperplasia	Pericardial cyst	Neurofibroma
Teratoma		Neurilemmoma
Mature		Neurofibrosarcoma
Immature		Sympathetic origin tumours
Malignant		Ganglioma
Germ cell tumours		Ganglioneuroblastoma
Yolk sac tumours		Neuroblastoma
Embryonal tumours		Parasympathetic origin tumours
Mixed tumours		Paraganglioma (pheochromocytoma)
Lymphoma		Enteric cyst (foregut duplication cyst)
Hemangioma		

hygroma. In the middle mediastinum, 20% of lesions occur which predominantly include lymphoma and also the rare pericardial cyst and cardiac tumours while 36% of lesions occur in the posterior mediastinum where tumours arising from the neurogenic structures in para vetebral sulcus - ganglioneuroma, neuroblastoma and neurofibroma occur.[3]

Diagnosis

Mediastinal tumours are occasionally diagnosed before birth. Most cases are diagnosed following evaluation for respiratory symptoms like cough, wheeze, stridor, chest pain, respiratory distress with or without fever in infancy or early childhood period. Unusual manifestations of mediastinal tumours include enlarged neck veins in a crying child, sternal bulge due to anterior mediastinal mass, symptoms due to recurrent laryngeal nerve or phrenic nerve palsy, Horner syndrome due to compression of nerve trunks, opsoclonus-myoclonus syndrome and sudden weakness of lower limbs or paraplegia. Phantom hernia can also be diligently observed in the child. Hemoptysis or trichoptysis due to erosion of tumour into bronchi, rupture into pleural cavity, or heart failure are rare presentations.[4, 5]

Constant chest pain in the parasternal region should not be ignored as it can be a manifestation of anterior mediastinal tumours. Posterior mediastinal tumours cause chest pain due to the direct compressive effect on the inter costal nerves or rib erosion.

Persistent cough, wheeze, recurrent or non-resolving pneumonia can be due to the direct compression on the bronchi by evolving posterior mediastinal tumour.[6] Neuroblastomas presenting with opsoclonus-myoclonus syndrome due to an autoimmune mechanism are said to have better prognosis indicating a mature phenotype and are more common in thoracic neuroblastomas.[7] Other signs like Horner's syndrome by compressive effect and paraparesis and paraplegia by dumb bell tumours extending into spinal cord can occur in superior mediastinal and posterior mediastinal tumours respectively. Germ cell tumours in mediastinum are more common in adolescent boys. These tumours can produce hormonally active substances like beta HCG leading to precocious puberty in some cases.

The evaluation of a mediastinal lesion should start with a simple chest X-ray. Lateral and oblique views should be included in the digital X-ray to localize an opacity seen on a frontal projection.

Contrast enhanced computerised tomography (CECT) of chest is the investigation of choice for evaluation and staging of mediastinal tumours and its efficacy increases when combined with angiography. CECT can be done rapidly without the need for anesthesia in children.

Magnetic resonance imaging helps in characterisation of lesion but the prolonged duration of study and the noise requires careful procedural sedation as the child may already have respiratory symptoms including respiratory distress. Posterior mediastinal tumour with an intraspinal extension always needs evaluation with MRI spine.

Tumour markers like serum alpha fetoprotein (AFP), beta-human chorionic gonadotropin (β-hCG), urine vanillyl mandelic acid and lactate dehydrogenase are necessary for

Table II. Approach to common mediastinal masses

General investigations	Germ cell tumour	Neuroblastoma	Lymphoma
Chest-xray AP/lateral views	**X ray chest**: lesion in anterior mediastinum +/- coarse calcifications	**X-ray chest**: lesion in posterior mediastinum +/- stippled calcifications.	**X-ray chest** Mediastinal widening. Lesion can occur anywhere in mediastinum.
Complete blood hemogram.	**CECT chest**: Heterogenous lesion with mixed fat and bone densities.	**CECT Chest**:Heterogenous lesion in posterior mediastinum with or without spinal involvement.	**CECT chest and abdomen**: Multiple mediastinal nodes with or without compression of vital structures. Hepatosplenomegaly may be present. LDH Bone marrow aspiration. Peripheral node biopsy with immunochemistry and staining.
Peripheral smear.	Alpha feto protein Beta-Hcg	**MRI Spine:** if involvement present. Urine VMA, Serum ferritin, LDH Bone marrow aspiration. USG / CT abdomen. Bone scan.	
Renal function test (before contrast CT)			
CECT chest +/- angiogram.			

diagnosis as well as prognostication. Other investigation includes USG abdomen, bone marrow aspiration or bone scan as a part of metastatic work up. PET scan though does not have much of a role in diagnosis, may be useful for assessing therapeutic response and recurrence in lymphomas. A well structured, clinically oriented approach increases the efficiency of the investigations and eliminates unnecessary studies (Table II).

Management

The operability and resectability of tumours can be assessed with the help of investigations. Mediastinal tumours can be approached by conventional surgical approach like thoracotomy or by video assisted thoracoscopic surgery (VATS). Any lesion less than 6 cm in size can be approached by VATS. VATS can itself be a diagnostic tool for tissue biopsy especially in a large tumour. Preoperative anesthetic work up should include the assessment of tracheal compression by tumour and the mode of anesthesia should be well planned to avoid any intra operative catastrophe.

In general, thoracic neuroblastoma are said to have good prognosis[8] compared to abdominal neuroblastoma if tumour excision is complete and followed by adjuvant chemotherapy (Fig.2a, 2b, 2c and 2d). Dumb bell tumours in view of intraspinal extension will require laminectomy. Germ cell tumour responds well to three drug chemotherapy (PEB regimen – cisplatin, etoposide and bleomycin - four to six cycles). Total excision of germ cell tumour with adjuvant chemotherapy makes them eminently curable.

Role of surgery in mediastinal lymphoma is primarily in establishing diagnosis by biopsy. Peripheral lymphnode biopsy is preferable and if indicated mediastinal biopsy can be taken thoracoscopically. Chemotherapy is the mainstay of management in these tumours.

Tumours or tumour-like lesions arising from the thymus include thymic cysts, thymic hyperplasia, thymomas and thymolipomas. Thymic hyperplasia is commonly seen in antero-superior mediastinum and is often

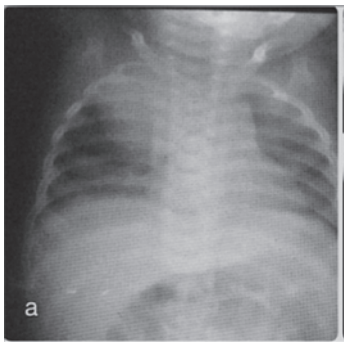
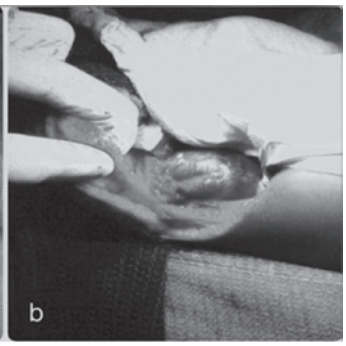


Fig.2a. X-ray chest - Homogenous opacity in right upper lobe



2b.Intra operative photograph thoracic neuroblastoma

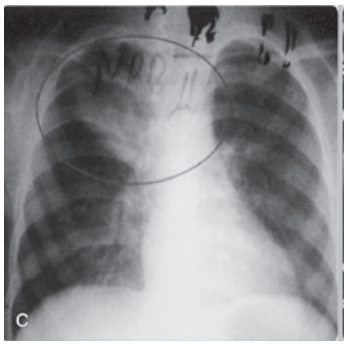


Fig.2c. X-ray chest - Non homogenous opacity in right upper lobe

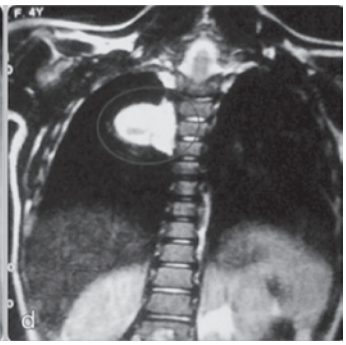


Fig.2d. MRI - Hyper-intense mass posterior mediastinum right side with fine calcifications (HPE neuroblastoma)

detected as 'Sail sign' in chest X-ray when evaluated for pneumonia.[9] Thymic hyperplasia regresses spontaneously and may need steroids for remission if symptomatic.

Primitive neuroectodermal tumour known as Askin's tumour is a rapidly growing tumour of the chest wall and in a short time can invade the posterior mediastinum. Diagnosis can be made with VATS and follow up treatment with chemotherapy.

Conclusion

Persistent or recurrent respiratory symptoms in a child calls for a proactive approach with further investigation if the chest skiagram shows a non resolving opacity. If detected early and managed appropriately, mediastinal tumours have a good outcome.

Points to Remember

- ***Persistent pneumonia in a child should be thoroughly investigated to unmask a mediastinal lesion.***
- ***CT chest is an important first line investigation to evaluate a non-resolving chest opacity.***
- ***Early diagnosis and total excision of mediastinal tumours will have a favourable outcome.***
- ***Video assisted thoracic surgery will play a crucial role in evaluation and management of thoracic lesions.***

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RADIOLOGY

TORTICOLLIS

***Vijayalakshmi G**
****Natarajan B**
****Abirami K**
****Thangalakshmi A**
****Raveendran J**

Torticollis is a symptom with many causes. Congenital vertebral anomalies, strabismus and nystagmus, trauma, inflammations and tumors near the cervical spine can cause torticollis. The simplest and least alarming is the congenital muscular torticollis that is evident from history and onset or duration of the posture. It is the commonest cause of torticollis in the infant. There may be a history of birth trauma. In utero crowding is another etiological factor accounting for torticollis being associated with developmental dysplasia of the hip. It usually presents by the age of two months. The ear on the side of the contracture is tilted towards the ipsilateral shoulder while the chin is rotated towards the contralateral side. Plagiocephaly with occipital flattening on the same side is seen. This asymmetry is not seen when torticollis is acquired later in life. Hence, the diagnosis of congenital muscular torticollis is essentially clinical. However, ultrasound is a useful investigation in the presence of a palpable mass. The normal muscle is hypoechoic with short echogenic lines running within, representing the perimysium in between bundles of muscle fibers. In Fig.1 there is a focal widening of the sternomastoid with loss of normal muscle echogenicity (arrow) with normal thickness of the sternomastoid seen superiorly (arrowhead).

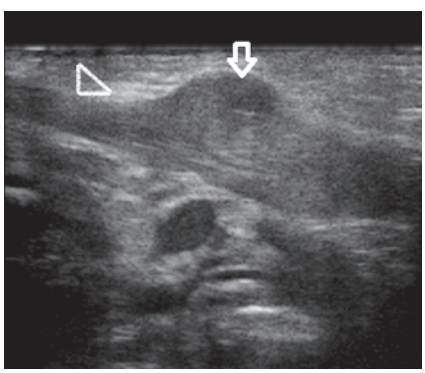

Fig.1.Ultrasound neck- Sternomastoid tumor (arrow) with normal muscle thickness (arrow-head)

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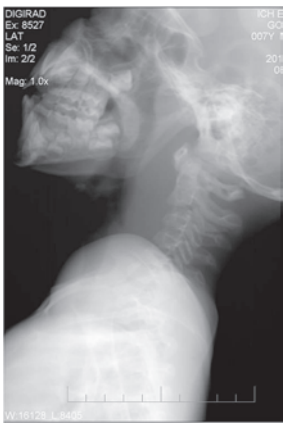

Fig.2.X-ray neck (lateral) - Retropharyngeal abscess

The child in Fig.2 presented with acute painful torticollis. The child had fever and pain on swallowing. Lateral X-ray of neck shows the space between the air column and the spine at the level of C2 and C3 is clearly widened to more than the width of the vertebrae. Retropharyngeal abscess is common in children and not in older children mainly because the space involutes as they grow older. The retropharyngeal space is the area between the buccopharyngeal fascia (which closely invests the pharyngeal constrictors) and the prevertebral fascia (which borders the cervical spine). It contains loose connective tissue and is rich in lymph nodes that can trap infective organisms leading on to inflammation and suppuration. Torticollis is due to irritation of inflammed, edematous neck muscles that go into spasm. Mild subluxation of C1 over C2 can also be seen in Fig.2.

The prevertebral fascia usually prevents the spread of inflammation into the prevertebral space from the retropharyngeal space. Fig.3 is an X-ray of neck lateral view. The retropharyngeal soft tissue thickness is normal. However, C3 vertebral body is porotic implying destruction

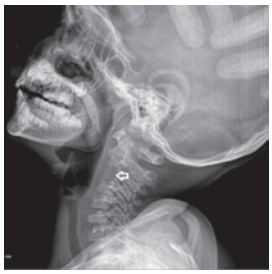

Fig.3. X-ray neck lateral - (arrow points to porotic C3)

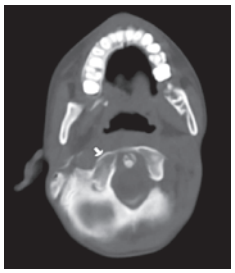

Fig.4.CT neck - Lysis of anterior arch of C1 (arrow)

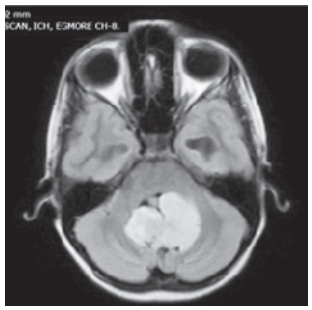

Fig.5.CT brain - Hyperintense midline

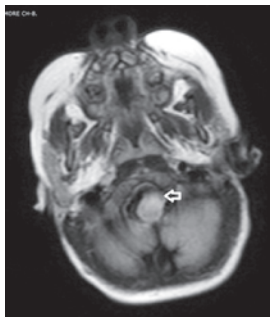

Fig.6. CT brain - Cervical cord

of bone. This is spinal caries affecting the third cervical vertebra. There is mild narrowing of C2-3 inter space. The torticollis in this child is due to irritation of the longus colli and the longus capitis muscles. CT neck of the child (Fig.4) shows the right side of the anterior arch of C1 is porotic compared to the left alone with rotation of C1 to the right. The lysis is again due to tuberculous infection of the vertebra. One can also see that there is no pus collection as yet. Inflammation and irritation of the muscles is causing torticollis which also serves to splint and protectively immobilise the neck.

Rarely torticollis may be the only presenting symptom of a posterior fossa tumor even without headache, vomiting or ataxia (Fig.5). This child, who came with torticollis had a normal neurological examination. Her neurological examination was normal. Power in all limbs, reflexes and gait were normal. The head and neck MRI revealed a midline, round, lobulated, hyperintense medulloblastoma in the cerebellum just behind the fourth ventricle. Many reasons have been offered for torticollis in patients with posterior fossa tumors. These include compensation for diplopia, herniation of cerebellar tonsils, irritation of the spinal accessory nerve because of descent and impaction of the cerebellar tonsils in the foramen magnum or just to maintain a fixed posture to avoid stretching and irritation of the dura.

Fig.6 is that of one year old child whose mother complained of child having torticollis for an uncertain period of time. She was sure that the child was normal at birth. MRI of the cervical spine was done keeping in mind congenital vertebral abnormalities. But the vertebral canal showed an exophytic mass from the cervical cord with poorly demarcated margins. Intramedullary spinal cord tumors are rare but should be borne in mind. For these reasons, torticollis should always be subjected to a full neurological and radiological examination.

CLIPPINGS

Randomized trial of dexamethasone vs prednisone for children with acute asthma exacerbations.

The authors intended to analyze if 2 doses of dexamethasone were as effective as 5 days of prednisolone/ prednisone therapy in improving symptoms and quality of life of children with asthma exacerbations, admitted to the emergency department (ED). A randomized, noninferiority trial conducted including patients aged 1-14 years who presented to the ED with acute asthma to compare the efficacy of 2 doses of dexamethasone (0.6mg/kg/dose, experimental treatment) vs a 5-day course of prednisolone/prednisone (1.5 mg/kg/d, followed by 1mg/kg/d on days 2-5, conventional treatment revealed the following.. 2 doses of dexamethasone could possibly serve as an effective alternative to a 5-day course of prednisone/prednisolone for asthma exacerbations, as estimated through the persistence of symptoms and quality of life at day 7.

Paniagua N, Lopez R, Murioz N, Tarmes M, Mojica E, Mojica E, Arana-Arri E, et al. Randomized trial of dexamethasone vs prednisone for children with acute asthma exacerbations. J Pediatr 2017 Dec;191:190-196.e1.

CASE REPORT

RECURRENT VAGINAL FOREIGN BODY - TWO MUCH PRANK

***Udayakumar N**
****Abhinayaa J**
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******Priyadharshini R**

Abstract :*Vaginal foreign body is a common cause of vaginal bleeding in prepubertal girls. Recurrence in the absence of abuse is rare. Examination and ultrasound-abdomen of a six-year, eight month girl with history of two-week vaginal-bleeding and five-day fever revealed the presence of vaginal foreign-body. Child reported insertion of hairpin, diagnosed and removed. Treatment with gynecological, psychological evaluation and follow-up was successful.*

Keywords: Recurrent vaginal foreign body, Children, Abuse, Vaginal bleeding

Vaginal foreign bodies are the cause of genital complaint in 4% of prepubertal girls. In prepubertal children, it is the cause of the vaginal discharge in 18% and upto 50%with vaginal bleeding.[1] The majority of patients are between the ages of three and nine.[2] Children may be exploring their body out of curiosity, pruritis or behavior related to sexual abuse.[3] The recurrence of vaginal foreign body in the absence of child abuse makes this case unique.

Case report

A 6 years 8 months old girl, who lives with her parents, presented with a history of vaginal bleeding for 2 weeks and fever for 5 days. Detailed history was taken to rule out various organic conditions. Clinical examination did not reveal signs of precocious puberty or overt signs of sepsis. Child's developmental milestones were normal, scholastic performance was good. She had prepubertal Tanner staging. Sexual abuse was ruled out by detailed history. Investigations including complete hemogram, bleeding time, urine routine and urine culture were normal. Ultrasound abdomen revealed two linear streaks in the vagina, probably foreign body (Fig.1).

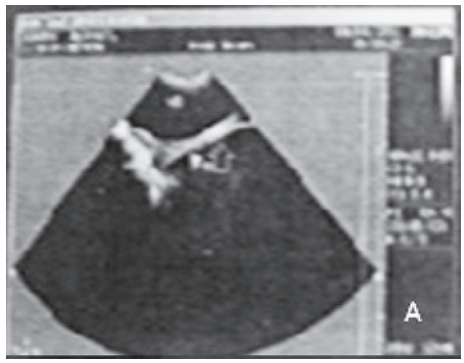

Fig.1. Ultrasound abdomen showing two linear streaks in vaginal area, probable foreign body

Examination by gynecologist revealed mild labial adhesion and intact hymen. Per rectal examination revealed two vertical ridges in the anterior wall. On vaginoscopy under general anesthesia, two linear foreign bodies, a pencil measuring 5 cms (Fig.2) and a crayon measuring 4 cms (Fig.3) in length, were detected and were removed. Vaginal irrigation was done with povidine iodine solution. The child's symptoms settled. Before discharge both the child and her parents were counseled.

Six months later, the child informed her mother that she had put a hair pin into the vaginal orifice. Revealed a hair pin like structure in the pelvic area (Fig.4) and was removed.

A detailed psychological assessment of the family was done. There has been no apparent emotional or psychological disturbance. The parents were advised to provide avenues for the child to play outdoors and also supervise the child when she handles certain objects. Follow-up till 12 months showed no recurrence of similar instances.

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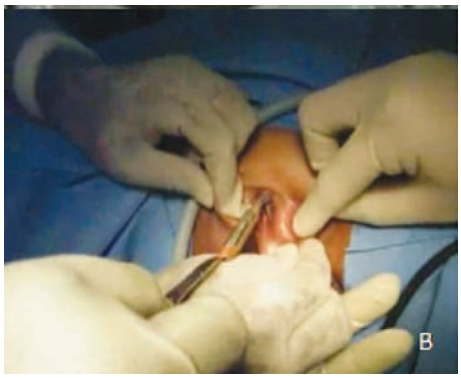

Fig.2. Pencil being removed

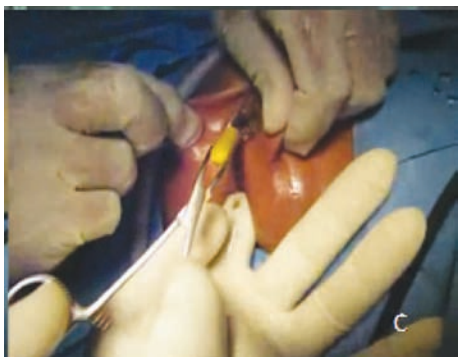

Fig.2. Crayon being removed

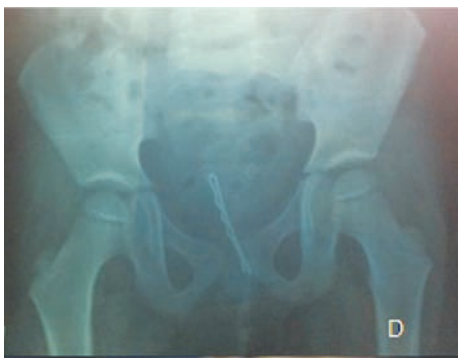

Fig.4. X-ray pelvis - Hairpin in pelvic area

Discussion

The causes for vaginal bleeding in prepubertal girls include accidental trauma, sexual abuse, vulvovaginitis, endocrine abnormalities, lichen sclerosus, urethral prolapse, blood dyscrasias, hemangioma and tumours. The foreign bodies that may be found in the vagina includes safety pins, hair grips, pencils and wad up pieces of toilet paper.[2] Depending on the nature and shape of the object, it can classically result in local vaginitis, associated with purulent, foul smelling bloody vaginal discharge or an abrasion and rarely urinary and fecal fistulae.[3]

The time from insertion to symptoms and subsequent extraction of vaginal foreign bodies varies greatly. Stricker et al studied 35 girls with vaginal foreign bodies and found that symptoms exhibited for less than 1 month in 60% and more than 1 year in 11%, prior to evaluation and treatment.[4] Unfortunately, many girls may tolerate symptoms for a long time before seeking treatment. Reasons for the delay in diagnosis are multifactorial. It includes a poor or unclear history, anxiety, denial, fear or embarrassment about vaginal complaints, along with difficulty in performing a thorough physical examination, by the pediatrician requiring multiple evaluations before the foreign body is found. History of self reporting of foreign body is present in 54%.[4]

A careful history taking, high index of suspicion and local examination, bimanual recto-abdominal palpation can diagnose 91% of the vaginal foreign bodies. Pelvic ultrasound, plain X-ray of pelvis and Magnetic Resonance Imaging (MRI) are recommended to diagnose, but vaginoscopy is confirmatory. X-ray abdomen, which can detect radiopaque foreign bodies and pelvic ultrasound are the initial investigations.[5] MRI is the best technique in children for evaluating nonmetallic vaginal foreign bodies which are missed by X-ray pelvis and ultrasound.[6]

Self insertion of foreign body is common (75%). Normal masturbation or curiosity, modelling behavior or sexual abuse believed to cause vaginal insertion of foreign bodies.[4] Children exposed to sexual abuse exhibit a number of sexualized behaviors, including inserting objects into the vagina or anus.[4] The child and the family should be assessed psychologically to rule out underlying emotional and behavioral problems affecting social competence and adaptiveness. Treatment includes timely removal and vaginal irrigation with normal saline, or povidone-iodine solution. Generally in older children and distally located foreign bodies, removal is easy.[7] One should be aware of hymenal injury if the child is uncooperative or very young.

The presence of any vaginal foreign body in a pre-pubertal girl should elicit concern about sexual abuse and thorough probing into the family dynamics and counseling will be needed. In a developmentally normal child without abuse, the need to stress on outdoor and recreational activities are also important.

Acknowledgement: We would like to acknowledge the help from department of Obstetrics and Gynecology and department of Psychology, Sri Ramachandra Medical College and Research Institute for helping us in the management of this child.

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CLIPPINGS

Chronic cough postacute respiratory illness in children: a cohort study.

A Prospective study was undertaken among the children attending the ED with ARI with cough.Children were followed weekly for 28 days; those with a persistent cough at day 28 were reviewed by a paediatric pulmonologist.. main outcome was cough persistence at day 28 and pulmonologist diagnosis.839 children (median age= 2.3 years, range=0.5 months to 14.7 years, 60% male) were enrolled over 2 years. Most children (n=627, 74.8%) had cough duration of <7 days at enrolment. At day 28, 171/839 (20.4%, 95% CI 17.7 to 23.1) children had persistent cough irrespective of cough duration at enrolment.When chronic cough develops post-ARI, clinical review is warranted, particularly if parents report a history of prolonged or recurrent cough. Parents of children presenting acutely to ED with cough should be counseled about the development of chronic cough, as an underlying respiratory condition is not uncommon

O'Grady KF, Drescher BJ, Goyal V, Phillips N, Acworth J, Jason Acworth JM, et al. Chronic cough postacute respiratory illness in children: a cohort study. Archives of Disease in Childhood 2017; 102:1044-1048.

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