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- Editorial Board
Greetings from the Journal Committee of IJPP. In this issue we have focussed some more articles on “Pediatric Emergency Medicine”. The Journal Committee profusely thank Dr.P.Ramachandran and Dr. S. Shanthi who have vast experience and rich knowledge in Pediatric Intensive care, for formulating and editing the articles on this important subject.

The ‘Approach to a child with respiratory distress’ is well narrated by Dr. Mahesh Babu. He has stressed the need for proper history taking and a good quick clinical examination which will give clues to the cause of respiratory distress and anatomical localisation of the problem in most of the cases.

In his article on ‘Acute asthma’, Dr. Krishan Chugh has reported that many a times it occurs either due to non-compliance with treatment but sometimes it may be the first episode. This article deals with the management of acute episodes in detail once the child presents in the emergency department.

The “Endocrine emergencies in children” is discussed in detail by Dr.P.Raghupathy. He has stated that endocrine emergencies in childhood may not always be obvious in their clinical presentation. Hence, one should have a high index of suspicion for early clinical recognition and confirmation of diagnosis and any delay in diagnosis may lead to critically ill states with life threatening complications.

The clinical features following envenomation by the scorpion are dealt in detail by Dr.S.Mahadevan. He has given the various steps involved in managing children with scorpion sting, which we hope may be useful for practitioners, who are dealing with such cases.

A prior knowledge on the common poisonous snakes in India will be helpful in the management of children with snake bite. This topic is written by Dr. Kulandai Kasthuri who has been handling such cases in PICU of a tertiary care hospital. She has given a detailed account of various steps in the management of snake bite victims.

Dr. Gautam Ghosh, et al. have given the various steps in the approach and management of acute poisoning in children. He has also highlighted the various pharmacological antidotes for the ready reference for personnel involved in the emergency room.

Dr. Mahesh Baldwa, Chairperson-IAP Medico-legal group has given salient points on medico legal issues with the help of past case scenarios which will definitely guide all the health managers looking after the emergency room and intensive care.

In the practitioner’s column, Dr. Meharban Singh, a senior paediatrician with repute, has contributed an article on “Nutrition, health and wellbeing of children”. This article will be useful for all pediatric practitioners and also for family physicians.

The “Essentials of pediatric pulmonary function test” will be an eye opener for young paediatricians and health personnel who are dealing with pulmonary problems. The authors have done their best to make it simple for IJPP readers.

Under the radiologist column Dr.Vijayalakshmi, et al. have discussed the assessment of hepatomegaly and hepatic masses with the help of ultrasonogram. They have reiterated that ultrasonogram is a non-invasive primary screening technique and can be very informative in dealing with such cases.

We sincerely thank all the contributors for case study column. We welcome suggestions and guidance from our readers to improve the quality of the Journal and maintain the academic contents.
INSTRUCTIONS TO AUTHORS

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

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3rd Page -
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Unmounted and with figure number, first author’s name and top location indicated on the back of each figure. Legends typed double-space on separate sheet. No title on figure.

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

Case report (covering practical importance)

250 – 600 words, 8 – 10 recent references

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150 – 200 words write up
With 1 or 2 images of clinically recognizable condition
(of which one could be in the form of clinical photograph / specimen photograph / investigation)

Letters to the Editor

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

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All articles including invited articles will be peer reviewed by two masked reviewers. The decision of the Editorial Board based on the reviewers’ comments is final.

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APPROACH TO A CHILD WITH RESPIRATORY DISTRESS

*Mahesh Babu

Abstract: One of the common presentations to the emergency room is a child with respiratory distress. This could be due to a upper respiratory, lower respiratory or a non respiratory cause. A proper history and a good clinical examination, give the cause for respiratory distress in most of the cases.

Keywords: Respiratory distress, Causes

The basic function of the respiratory system is to provide oxygen and remove carbon dioxide from the body, in technical terms called oxygenation and ventilation respectively. Whenever there is a clinical situation causing compromise in either of these functions, the respiratory tract has to work harder to try and achieve its primary goal. This overactive respiratory system is what a clinician recognizes as respiratory distress.

Hence, a child with respiratory distress could:

1. Have normal gas exchange – i.e. the child is in a compensated phase of respiratory failure, when he is working harder and maintaining his blood gases. It is very important to recognize and treat this child before he gets into frank respiratory failure.

2. Have hypoxia and/or hypercarbia – i.e. the child is decompensated and is in frank respiratory failure. This child needs emergent medical care.

A child with respiratory distress can be recognised by tachypnea, increased work of breathing (chest indrawing), tachycardia and abnormal sounds like stridor, wheeze or grunt. A decompensated state (respiratory failure) can be clinically suspected when there is altered sensorium, cyanosis or ineffective breathing like see - saw movements (described later).

The history: Evaluation of key clinical symptoms

The clinician should ask focused questions based on the child’s chief complaints and significant findings, ensuring that one can elucidate the onset, duration, character, alleviating and exacerbating factors and treatment to date. The impact that the symptoms have on everyday activities, such as playing or exercise and the oral intake of liquids and food are very important. Measures of “thriving” and general “feeling” should be elicited. Though the majority of ill children have new and acute processes with short medical histories, always consider the possibility of an acute exacerbation of an indolent or more chronic process that has not been uncovered till now. As there exists a large number of children with special health care needs and underlying medical problems (ie, asthma, recurrent croup, cystic fibrosis, or bronchopulmonary dysplasia), it is important to take a detailed past medical history. Children with known complex heart disease and those with any chronic infectious or immunologic illnesses must be investigated with high suspicion for complications like infection. They may develop acute respiratory distress associated with their underlying condition. In children with their first significant episode of acute distress from

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bronchospasm secondary to asthma, a careful look at their medical history may reveal recurrent though mild symptomatology of reactive airway disease, such as mild persistent cough with exercise or with upper respiratory tract infections. Table 1 lists some of the symptoms that are important to consider when obtaining a focussed history.

**Table 1. Symptoms associated with respiratory distress**

<table>
<thead>
<tr>
<th>Breathing difficulty:</th>
<th>Rapid breathing, retractions (subcostal, intercostal, supraclavicular), abdominal wall muscle use and “see-saw” respiration, positional distress</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color change:</td>
<td>Pale or cyanotic</td>
</tr>
<tr>
<td>Noisy breathing:</td>
<td>Wheezing, stridor and grunting</td>
</tr>
<tr>
<td>Non-Localized Symptoms</td>
<td>Fever, poor feeding or drinking, Weight loss or failure to gain weight, pain, pallor, diaphoresis</td>
</tr>
</tbody>
</table>

The presence of important non-respiratory symptoms, such as fever, often helps to direct the evaluation to an inflammatory or infectious etiology or trigger. One should also elicit the history of other constitutional symptoms, such as the impact of illness on dietary intake, activity level, and weight loss to help judge the overall severity of the process.

**The physical exam: What are the important signs?**

It is useful to evaluate children for important signs of respiratory disease in a logical physiologic and anatomic order that assists in localizing the primary etiology.

The respiratory rate of the ill child is a key parameter that is often under-assessed. One should correlate the respiratory rate to the age of the child and to the sensorium of the child. Age specific respiratory rates as per ARI protocols (50 or more in 1-12 months and 40 or more in 1-5 years old) might be a good reference guide. It should also be appreciated that the respiratory rate is faster in children who are awake than in children who are asleep. A fast sleeping respiratory rate, therefore, has a greater significance.

The heart rate, temperature, state of perfusion and blood pressure give supporting evidence to the physiologic state of the child and may help the clinician to identify early cardiovascular compromise.

Sensorium is another important sign. Children with hypoxia can either be irritable or be drowsy, and sometimes slip into comatose state. Hypercarbia also produces drowsiness and hypersomnolescence.

When available, another useful measure in the evaluation of respiratory disease is the oxygen saturation, which can be obtained via pulse oximetry. This may be a useful measure to determine the presence of mild to moderate hypoxemia that is not evident on physical examination. It is useful to remember that pulse oximetry may not be accurate if the probe is not of the appropriate size for the child’s small fingers or toes, if the extremity is cold or has poor perfusion or when the hemoglobin molecule is saturated with something other than oxygen (such as carbon monoxide) or if there is significant methemoglobin.

It is useful to remember that in a child presenting with tachypnea without increased work of breathing (effortless tachypnea), metabolic acidosis should be suspected. It is also important to remember that in CNS disorders a child may present with respiratory failure without respiratory distress.
Clinical Pearls

There are some important clinical signs which can be considered as clinical pearls while assessing a child with respiratory distress.

Indrawing

Supra-ternal indrawing: When present suggests significant respiratory distress. It is a non localizing sign and is present in upper airway and lower airway pathologies. This sign suggests the use of accessory muscles of respiration.

Sub-costal indrawing: When present suggests much more significant distress. This is also a non localizing sign. This sign suggests that the diaphragm is working very hard. Normally, the outward recoil of the chest wall and the inward recoil of the subcostal area caused by the diaphragmatic action, cancel each other. However, when the diaphragmatic action is very strong then the sub costal recessions appear.

Inter-costal indrawing: This is a localizing sign. When present, intercostal indrawing suggests decreased compliance of lung and hence suggests parenchymal lung disease. Hence most children with pneumonia will have intercostal indrawing, as opposed to children with asthma or pleural effusions who will not have intercostal indrawing.

Sounds heard

(a) Stridor: Stridor is sine qua non of upper airway pathologies. Stridor is produced by passage of air through partially obstructed upper airway structures which are predominantly extra thoracic. These include structures comprising the pharynx, larynx, subglottis and the upper trachea till the thoracic inlet. Stridor is usually an inspiratory noise, occasionally having a biphasic component. The sound quality of stridor varies with the site of obstruction, with nasopharyngeal stridors having a low pitch quality and the sub-glottic stridor having a high pitched quality. Glottic problems usually cause biphasic stridor. Acute onset stridor is always to be considered as a medical emergency, since it suggests partial obstruction of upper airway. Therefore protection of airway is paramount before it gets worse.

(b) Wheeze: Wheeze is sine quo non of lower airway pathologies. Wheeze is produced by passage of air through partially obstructed lower airway structures which are predominantly intra thoracic. These include lower trachea, the major bronchi and further generations of bronchi till the respiratory bronchiole. Wheeze is usually an expiratory noise. Trachea is the only structure which has both an extra thoracic component and an intra thoracic component, hence tracheal pathologies can produce both stridor and wheeze.

Wheeze could be further classified as:

Focal: when it is heard over only one part of the chest. This suggests a local obstructive pathology and one should think about foreign body, or an extraluminal obstruction such as a lymph node or vascular compression.

Generalized: When it is heard all over the chest. This is further classified as

Monophasic wheeze: Where the quality of sound heard all over the chest is the same, as in a conducted sound. Monophasic wheeze suggests large airway pathologies such as tracheomalacia or broncho malacia or compression of main stem trachea.

Polyphonic wheeze: where the quality of sound heard is different in different parts of the chest. Polyphonic wheeze suggests small or distal airway disease such as asthma or bronchiolitis. This is because the lumen of the affected structures vary in different areas, and the pitch of the sound produced is inversely proportional to the diameter of the airway affected i.e smaller the airway, higher the pitch of the sound.
Table 2. Location and conditions producing respiratory distress

<table>
<thead>
<tr>
<th>Location of respiratory problem</th>
<th>Examples of conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper airway</td>
<td>Croup, epiglottitis, foreign body, tracheitis</td>
</tr>
<tr>
<td>Lower airway</td>
<td>Asthma, bronchiolitis, foreign body, pneumonia</td>
</tr>
<tr>
<td>Neuromuscular control</td>
<td>Seizure, CNS structural defects, acute encephalopathy of various causes, head trauma, acute paralysis, myopathy</td>
</tr>
<tr>
<td>Mechanics</td>
<td>Trauma, spinal or chest wall deformity</td>
</tr>
<tr>
<td>Pulmonary parenchyma</td>
<td>Pneumonia, interstitial lung disease, BPD, cystic fibrosis</td>
</tr>
<tr>
<td>Extra-pulmonary:</td>
<td>Heart failure, neurogenic hyperventilation, renal failure, drug overdose, Carbon monoxide (CO) poisoning, methemoglobinemia</td>
</tr>
<tr>
<td>Cardiac, CNS, renal, toxicologic, Oxy-hemoglobin delivery system</td>
<td></td>
</tr>
</tbody>
</table>

(c) Grunting: Grunting is sine qua non of parenchymal lung disease. It is produced in children with alveolar disease, where the child tries to produce greater intrinsic PEEP to keep their alveolus open. It is produced by a premature glottic closure at the end expiratory phase. Grunting suggests a parenchymal disease process like pneumonia and should be taken seriously.

Localisation in respiratory distress

Another way of approaching a child with respiratory disease is by considering the respiratory system as individual components like upper airway, lower airway or parenchymal disease, problems of neuromuscular control or mechanics and extra-pulmonary problems. Some of the common diseases affecting individual part are shown in Table 2.

First evaluate the upper airway, particularly for signs of potential obstruction. Is there noisy breathing on inspiration? Does the child’s posture have an important impact on the airway being opened maximally (eg, leaning in the sniffing position) and does it improve the condition? Is the noise seal-like or barking (subglottic obstruction), sonorous or harsh (nasal or pharyngeal) or high pitched (tracheal and intrathoracic large airways)? Upper airway conditions have the greatest risk of sudden deterioration and significant morbidity and mortality. Be careful to elicit enough history to discern the components of common illnesses while still looking for the clinical features that would cause one to search for the less common and potentially more risky condition. For example, focussing on the obvious assessment of infectious croup too early and missing the questions that might establish a risk for foreign body aspiration is a potential trap.

Within the chest, the function of the lower airways (central trachea to the small airways) can be impacted from a variety of disorders, both focal and diffuse. Clinical signs of lower airway pathology include hyperinflation of the lung and chest cavity, accentuation of the expiratory phase of respiration, and wheezing (higher pitched, continuous sounds on expiration). The presence of inspiratory wheezing in addition to expiratory wheezing is evidence of more severe lower airway obstruction, which as it progresses can lead to reduction of the tidal volume and the quality of the inspiratory breath sounds on
auscultation. All that wheezes is not asthma. Consider entities such as an aspirated foreign body, particularly with focal wheezing, or something compressing the intra-thoracic airways such as an enlarged lymph node or tumor, and rarely, a left atrial problem causing cardiac failure. In these instances, the physical exam may reveal focal problems or an abnormal cardiac examination. But more important is the history of choking spell or acute or recurrent focal wheezing at an age unusual for first asthma episode.

The next component to discuss is the pulmonary parenchyma. In general, one tends to think of pneumonia as the prime pulmonary parenchymal disease. When severe, one may expect tachypnea, grunting, and retractions. Focal findings may include splinting of the chest wall when there is pain, changes in breath sound quality, and crackles. The subtle alteration of breath sounds in cases of pneumonia may range from undetectable to harsh, bronchial, or the classic “tubular” breath sounds. As the pneumonia progresses, the physician may hear crackles from the opening of small distal airways closer to the alveoli. A complete physical examination of the child may reveal other important signs of chronic pulmonary parenchymal disease, such as failure to thrive or finger clubbing as seen in cystic fibrosis, or the signs of prematurity or other neonatal complications that may have accompanied bronchopulmonary dysplasia. More difficult to detect may be the subtle findings of pulmonary interstitial disease—such as tachypnea and end-inspiratory crackles—and constitutional findings—such as weight loss and fatigue—in the absence of fever. When the history does not seem to fit common processes such as pneumonia, one should dig deeper in the clinical assessment at the first encounter or do so when the patient does not respond to therapy as one would have anticipated.

Assessment of neuromuscular control is often delayed and forgotten. This system includes the feedback loop encompassing the brainstem respiratory centers, the connection to the muscles of respiration and the “internal” receptors that balance the measures of pH and PaO₂. Disorders that affect normal respiratory control are fairly common and often lead to respiratory failure without obvious respiratory distress like increased work of breathing. Signs of central nervous system-mediated respiratory problem may be ascertained through an assessment of the breathing pattern. Is the upper airway patent? Is the respiratory rate slow or absent? Is the pattern of respiration insufficient to move the chest wall? Is there an unconscious state or active seizure that may impair normal respiration? Is there evidence of abnormal peripheral muscle tone or lack of movement that would suggest metabolic or neuromuscular weakness? Examples of disorders of the central nervous system impacting on effective respiration include generalized seizures, an ingested poison or toxin, and a number of acquired disorders (eg, infections, trauma) and congenital nervous system problems and for peripheral neuromuscular disorders like muscle or nerve disorders.

The next location for the consideration of pathology that can contribute to respiratory distress is the effectiveness of the mechanics of respiration. The mechanics of respiration can be disrupted by the presence of upper airway obstruction, lower airway obstruction, chest wall or neuromuscular abnormality, and extrapulmonary problems. Any of these may lead to less than effective ventilation and respiratory failure. Examples include a reduced lung volume secondary to an intra-abdominal mass or large pleural fluid collection or the air trapping and large lung volume created by severe asthma. Primary causes of mechanical problems include congenital or acquired skeletal abnormalities and trauma (eg, flail chest). The assessment should
focus on the observation of the chest wall configuration and symmetry of the chest wall movement with respiration. Appropriate effort with ineffective respiration in the absence of an upper airway problem or serious intrapulmonary pathology is a sign of a mechanical problem. Non-specific features of mechanical problems include the presence and symmetry of retractions and abdominal muscle use. Generally the respiratory rate will be increased over normal in attempts to compensate, but it may prove ineffective.

Last but not least are the non-respiratory problems that can lead to respiratory distress. The list is long and can involve any of a number of organ systems. Therefore, the physical examination of the child with respiratory distress should be complete. Is the cardiac exam specifically abnormal? Is there primary heart failure with secondary respiratory distress? Are there signs of pericardial effusion with narrow pulse pressure, pericardial rub, and distant heart sounds? Is there any suggestion of pulmonary embolus from history or from the “normal chest exam” with significant hypoxemia if a blood gas is measured? Is there evidence of acidosis (ie, hyperpnea) or other metabolic abnormality (ie, Kussmaul respiration with fruity breath) that may cause respiratory distress? Is there evidence of renal failure, liver disease, or a congenital problem associated with respiratory distress? Is there a suggestion of drug overdose or drug effect that is leading to respiratory distress? This may include miotic pupils with bradypnea from narcotics or the drunken stupor of alcohol abuse or small pupils, bronchorrhea, respiratory distress, and overactive bowel sounds from an insecticide exposure.

**Laboratory assessment**

No lab tests are required to make a diagnosis of respiratory distress. However, they may be required to quantitate the severity of respiratory distress and sometimes to localize the cause of distress. Some of the common tests done will include a pulse oximetry, ABG and chest x-ray. Pulse oximetry will give us the oxygenation status and if greater than 90% in room it correlates with a PaO2 of > 60mm/kg. Sometimes, children can be maintaining their saturations with minimum additional oxygen, but they could be having dangerously high carbon dioxide. To know the CO2 status, we will need to do arterial blood gas analysis or end tidal CO2 monitoring.

**Points to remember**

1. **Respiratory emergencies are quite common and can be potentially life-threatening.**

2. **Respiratory distress is characterised by normal sensorium and increased work of breathing.**

3. **Respiratory failure is present when there is altered sensorium and respiratory distress, except in CNS problems.**

4. **Respiratory failure is a clinical diagnosis and does not need an ABG.**

**Bibliography**


ACUTE ASTHMA

* Krishan Chugh  ** Gurinder Arora

Abstract: Acute exacerbation of asthma is one of the conditions which makes the parents rush their children to the emergency room. Many a times it occurs either due to non-compliance with treatment but sometimes it may be the first episode. All asthmatics should have a written action plan for home management. This article deals with the management of acute episode once the child presents in the emergency department.

Keywords: Acute asthma, Drugs, Management.

Bronchial asthma is a common problem with enormous medical and economic impacts. Despite improved understanding of the disease and pharmacological options, death and hospitalization still occur. Appropriate emergency management of acute asthma will have an impact on these statistics. Most of the acute attacks are either the first episode or due to non-compliance with the treatment; acute events occurring because of the treatment failure per se are uncommon. This article reviews home management of an acute attack, management in the Emergency Room (ER) and in the Pediatric Intensive Care Unit (PICU).

Home management i.e. prior to arrival in the emergency department

All asthmatics should have a written action plan that can help guide them in recognizing and assessing their overall asthma control and the severity of acute asthma exacerbations. Recognizing symptoms early and intensifying treatment soon after symptoms worsen can often prevent further worsening and can keep exacerbations from becoming severe.

The National Asthma Education and Prevention Program guidelines (NAEPP) recommend immediate treatment with rescue medication i.e. inhaled short acting β agonist up to 3 inhalations in 1 hour. A good response would be characterized by resolution of symptoms within an hour, no further symptoms over the next 4 hours, and improvement in PEF to 80% or more of the predicted or personal best.

If the child has an incomplete response to initial treatment with rescue medication (i.e. persistent symptoms and/or a PEF of 60-80% of predicted or personal best), an early arrival at the emergency department (ED) would prevent the attack progressing to severe stage.

At the emergency room the following signs should be recorded for assessment of severity of acute asthma:

1. Pulse: Increasing pulse rate generally denotes worsening asthma; bradycardia occurs in life threatening asthma as a pre-terminal event.
2. Respiratory rate and degree of breathlessness.
3. Use of accessory muscle of respiration: Best noted by palpation of neck muscles
4. Degree of agitation and conscious level.

(NB: Clinical signs may correlate poorly with the severity of airways obstruction. Some children
with acute asthma do not appear distressed. Special care should be given in assessment of adolescents).

**Evaluation of the severity of an acute episode of Asthma (BTS guidelines for management of acute attack of Asthma in children)**

**Assessment of acute asthma in children aged over 2 years**

**Acute severe**

1. Cannot complete sentences in one breath or too breathless to talk or feed.
2. Pulse rate: > 130 (2-5 years) or > 120 (beyond 5 years)
3. Respiratory rate: > 30 / min (beyond 5 years)
   > 50 / min (2-5 years)

**Life threatening**

1. Hypotension
2. Exhaustion
3. Confusion
4. Coma
5. Silent chest
6. Cyanosis
7. Poor respiratory effort

**Management upon arrival in the emergency department**

The key to managing acute episodes is to stabilize the patient as rapidly and as effectively as possible, ensure adequate oxygenation (children with life threatening asthma or SpO2 of < 92% should receive high flow oxygen via a tight fitting face mask or nasal cannula at sufficient flow rates to achieve normal saturations), and reverse bronchial narrowing with a minimum of side effects. Freedom from wheezing and normal pulmonary mechanics take a long time to achieve and need not be the primary goal of acute therapy. After the acute episode has ended, the residual deficits can be addressed with appropriate outpatient regimens. In this situation, weeks may be needed to completely stabilize all aspects of the disease.

**Care paths**

Care paths (practice guidelines) have been developed to improve the efficiency. In ED settings, the use of practice guidelines rapidly identifies individuals at risk for adverse outcomes, reduces admissions to both pediatric wards and PICU, lowers the length of stay, decreases the number of return visits in the next 48 hours, and lessens costs. On the inpatient side, there is a decrease in length of hospitalization and a better prognosis post discharge.

**First line therapy in the emergency department**

**Oxygen**

Profound hypoxemia is rare in uncomplicated acute asthma and few patients have oxygen saturations less than 90%. A child of acute asthma with SpO2 of < 92 % in the emergency department should be started on supplemental oxygen.

**Inhalation therapy with ß2 agonists**

Moderately short-acting ß2-adrenergic agonists such as salbutamol and terbutaline have rapid onset of action and provide three to four times more bronchodilatation than do methylxanthines and anticholinergics, making them the first-line treatment for acute illness. Long-acting agents such as salmeterol are not recommended in the acute setting but formeterol, is undergoing clinical trials to determine its efficacy.

**Metered Dose Inhaler (MDI) with spacer**

British Thoracic Society guidelines for management of acute asthma in children state that, a MDI and spacer are the preferred option in mild to moderate asthma.
5-10 puffs may be required depending on the severity. Each activation of MDI is followed by 8-10 normal breaths for the drug to be inhaled completely. Improvement in peak flow rate was as good\(^5\) or more\(^6\) with MDI and spacer compared to nebulizer.

**Nebulizer**

Salbutamol is nebulized in doses of 0.5ml (2.5mg) in children < 6 years and 1ml in > 6 years mixed with 2ml of NS. The dose can be repeated every 20 minutes three times.

Children with life threatening asthma may deteriorate during \(\beta_2\) agonist nebulization due to ventilation perfusion mismatch as salbutamol may cause pulmonary vasodilatation. Hence, during treatment always use \(\text{O}_2\) as a driving force to nebulize along with close monitoring of patient by a pediatrician.

Dose–response effects are found with the amounts commonly administered clinically. The degree of improvement is a function of how much medication is given, not of how it is delivered. There does not seem to be any advantage in giving larger quantities once pulmonary mechanics approach the lower limit of normal\(^7\).

**Continuous or intermittent nebulization**

Review of literature suggests that in cases of nonresponder asthma, continuous nebulization as an option can be tried with good monitoring of the patients for side effects\(^8,9\). The use of very high doses of inhaled salbutamol (as high as 40-80mg/hr), had the following side effects reported hypokalemia, hyperglycemia, which were thought to be because of the accentuation of these side effects during acute exacerbations of asthma\(^10\).

**Treatment for incomplete response**

- Individualize drug dosing according to severity and the patient’s response.
- The early addition of bolus dose of IV salbutamol (15ug/kg) can be an effective adjunct to treatment in severe cases.

- Systemic (intravenous or oral) corticosteroids should be used for all patients who do not favorably respond to the initial \(\beta\)-agonist therapy
- Addition of anticholinergic may increase lung function and may decrease hospital admission rate

**Corticosteroids**

Recommendations for use of steroids in case of acute asthma not responding to the initial inhalation therapy\(^2\):

To give prednisolone early in the treatment of acute asthma attacks. Use a dose of 20 mg prednisolone for children aged 2-5 years and a dose of 30-40 mg for children > 5 years. Those already receiving maintenance steroid tablets should receive 2 mg/kg prednisolone up to a maximum of 60 mg. Repeat the dose of prednisolone in children who vomit and consider IV steroids. Treatment up to three days is usually sufficient, but tailor length of course to the number of days necessary to bring about recovery. There is no role for nebulized steroids in acute asthma as per current guidelines.

**Anticholinergics**

If symptoms are refractory to initial \(\beta_2\) agonist treatment, add nebulized ipratropium bromide (250 mcg / dose mixed with \(\beta_2\) agonist solution). Repeated doses of ipratropium bromide should be given early to treat children poorly responsive to \(\beta_2\) agonists.

Ipratropium bromide or other anticholinergics may be used as an additional bronchodilator in conjunction with a \(\beta_2\) agonist in cases of acute moderate to severe asthma. It is nebulized in a dose of 250 mcg every 20 minutes along with salbutamol. Its most beneficial effects appear to be in multiple doses in more severe exacerbations. Literature has been inconsistent,
but indicates that anticholinergic therapy may increase FEV$_1$ or PEF, may decrease hospital admission rates slightly, may decrease the amount of β-agonist needed, and may prolong bronchodilator effect. There were no significant adverse reactions, however. In view of this, it is recommended to consider anticholinergic use in moderate to severe asthma exacerbations, along with β$_2$ agonist.

**Subcutaneous epinephrine**

In children with poor tidal volume as in life threatening asthma, epinephrine 1:10000 in a dose of 0.1ml subcutaneously should be administered and can be repeated every 20 minutes. After each dose child is reassessed and if improving, continued on nebulized salbutamol.

**Magnesium sulfate**

IV Magnesium sulphate is likely to be effective in avoiding hospitalization and improving bronchoconstriction and clinical symptoms of acute severe asthma in children when added to standard therapies of inhaled bronchodilators and steroids. The possible mechanism of action is by decreasing Ca$^{++}$ uptake leading to bronchodilation, inhibition of mast cell degranulation and release of mediators, inhibition of acetyl choline release and depression of muscle fibre excitability. The dose is 25-40mg/kg/day (maximum of 2 grams) dissolved in 30ml NS and administered over 30 minutes.

**Aminophylline**

Aminophylline is not recommended in children with mild to moderate acute asthma. Consider aminophylline in an High Dependancy Unit (HDU) or PICU with severe or life threatening bronchospasm unresponsive to maximal doses of other bronchodilators and systemic steroids with close and careful monitoring. Aminophylline is used in a loading dose of 5mg/kg as an infusion over 30 minutes followed by 1mg/kg/hr as continuous infusion. The loading dose is omitted if child is already on theophylline.

**High dose IV salbutamol in bolus form**

Statement from National Heart, Lung, Blood Institute (NHLBI) regarding high dose salbutamol given as a bolus sums up the current status of this mode of therapy of acute severe asthma:

Although inhaled β$_2$ agonists and corticosteroids have been the cornerstones of acute asthma management, there remains a need to develop new strategies to treat these patients more effectively. An intravenous bolus of salbutamol (15 µg / kg), given early in conjunction with conventional therapy (oxygen, inhaled β$_2$ agonist, and intravenously administered corticosteroids) results in more rapid recovery, as measured by clinical assessment scores and the need for inhaled β$_2$ agonists and oxygen. The only side effect was tremor. Intravenously administered β$_2$ agonists have been traditionally reserved for the patients with the most severe exacerbations and given by continuous infusion in an intensive care unit setting. Single dose of 15 mg /kg of I.V. salbutamol administered over 10 minutes in the initial treatment of children with acute severe asthma in the emergency department has been shown to shorten the duration of severe attacks and reduce overall requirements for inhaled salbutamol maintenance.

**Intravenous terbutaline infusion in acute severe asthma**

Terbutaline is recommended as a useful adjunct in asthma in those patients who fail to respond to standard initial therapy. Terbutaline was found to be effective and safe at doses of 1-5 µg /kg/ min. Side effects of the drug reported were increase in heart rate, significant fall in diastolic blood pressure which may also require inotropes and hypokalemia.
Ketamine in acute asthma\textsuperscript{15}

Ketamine promotes relaxation of airway smooth muscle fibers probably via an epithelial-independent mechanism.

The only randomized, double blind, placebo-controlled trial to assess the efficacy of ketamine in acute asthma carried out by Howton, et al, concluded that intravenous ketamine at doses low enough to avoid significant dysphoric reactions demonstrated no increased bronchodilatory effect over standard therapy.

Heliox\textsuperscript{16}

Heliox, a blend of helium and oxygen, reduces airway resistance and may be a therapeutic option for severe refractory asthma in intubated patients as there is a decrease in peak inspiratory pressure and PaCO\textsubscript{2}. The effects of heliox are transitory and disappear when air is once again inhaled. Its temporary use, however, may lower respiratory resistive work long enough to forestall muscle fatigue and/or improve ineffective mechanical ventilation until bronchodilators and steroids can take effect. The mixture may improve the distribution of inhaled agents and lead to a faster rate of resolution of obstruction. But there is insufficient evidence to establish the utility of heliox in routine emergency room treatment.

Anti-leukotriene agents\textsuperscript{17}

There are limited data on the effects of antileukotriene drugs in acute asthma. A clinical benefit of the type noticed with salbutamol definitely does not occur with antileukotrienes.

Ventilation in asthma\textsuperscript{18,19}

Ventilatory assistance can be lifesaving. Both noninvasive and invasive techniques are available. Noninvasive facemask ventilation may offer short-term support for some subjects with hypercapnic respiratory failure who can cooperate with their care and are able to protect their airways. Its applicability, however, is limited by its poor patient acceptance.

The generally accepted indications are progressive CO\textsubscript{2} retention, obtundation and impending cardiopulmonary collapse. Mere presence of hypercapnia is not sufficient. The goal of ventilatory support is to maintain adequate gas exchange until bronchodilators and corticosteroids relieve the airflow obstruction. This usually entails sedation, and possibly paralysis, as well as strategies to minimize dynamic hyperinflation. Ketamine may be necessary to supplement sedation with neuromuscular blockade with pancuronium, vecuronium, atracurium, or cisatracurium. All of the paralytic agents can be associated with myopathy, which is worsened by concomitant use of corticosteroids and aminoglycoside antibiotics.

Dynamic hyperinflation (auto-PEEP) has profound physiological effects. It rises directly with minute ventilation and can compromise cardiac output by reducing venous return. The institution of positive-pressure ventilation in an already hyperinflated thorax can markedly worsen hemodynamics and cause abrupt falls in blood pressure including cardiac collapse. Because the airways are heterogeneously narrowed, the less involved parts of the lungs may undergo regional overdistension when exposed to high inflation pressures and rupture. For these reasons, ventilatory strategies that provide the longest possible expiratory time are desired so that dynamic lung inflation is minimized. This goal is accomplished by combining the smallest tidal volume with the slowest ventilatory rate and fastest inspiratory time to keep a static end-inspiratory pressure (plateau pressure) of less than 30 cm H\textsubscript{2}O. Approaches designed to reduce auto-PEEP often result in hypoventilation. The resulting hypercapnia is well tolerated as long as it develops slowly and the PaCO\textsubscript{2} remains at 90 mm Hg or less. When necessary, the pH can be defended pharmacologically. Once the
bronchospasm is relieved, the patient can be weaned off rapidly.

Supportive treatment

Overall care of the child should also be given due consideration, with maintenance of good hydration status, control of temperature and strict maintenance of the fluid and electrolytes balance. Routine use of antibiotics in acute asthma is not indicated.

Prognosis

Despite concerns about increasing mortality, most patients survive acute episodes.

Points to remember

1. Parents should have a clear cut action plan to follow whenever their children with bronchial asthma worsen. This will minimise the severity of acute exacerbation.

2. In the emergency room, severity of acute asthma can be assessed based on clinical features and pulse oximetry on admission and during re-evaluation.

3. Treatment is tailored to the severity of acute episode.

4. In majority, administration of oxygen, aerosolised salbutamol and parenteral steroids brings about good improvement.

5. Only in non-responders or those presenting with life threatening episodes, subcutaneous epinephrine followed by IV magnesium sulphate or aminophylline infusion may be required. IV salbutamol in bolus form also may help in such severe cases.

References


5. Cochrane Database Syst Rev 2002; CD 000052


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**NEWS AND NOTES**

**FIRST NATIONAL CONFERENCE OF IAP COMMUNITY PEDIATRICS SUBCHAPTER COMMPEDICON 2006**

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ENDOCRINE EMERGENCIES IN CHILDREN

* Raghupathy P

Abstract: Endocrine emergencies in childhood may not always be obvious in their clinical presentation. Hence a high index of suspicion is required for clinical recognition and confirmation of diagnosis. Needless to say, any delay in diagnosis can lead to critically ill states with life-threatening complications. Some of these conditions may be the initial manifestation of a previously undiagnosed endocrine condition which makes it all the more difficult to think of the diagnosis. These emergencies may also arise in a known case as a result of stress situation, emotional crises, intercurrent infections, accidents etc. For example, diabetic ketoacidosis may be the presenting feature of type 1 diabetes mellitus. This complication may also arise in a child with established diabetics owing to non-compliance with the prescribed insulin therapy. In the enthusiasm to save the child during an emergency, one should not miss an important aspect of management, which is to collect the critical blood samples as these will help to confirm the diagnosis of a lifelong condition.

Keywords: Diabetic ketoacidosis, Cerebral oedema, Adrenal insufficiency, Syndrome of inappropriate ADH secretion.

Endocrine emergencies such as diabetic ketoacidosis, adrenal insufficiency and syndrome of inappropriate ADH secretion will be discussed in this article. Metabolic emergencies such as hypoglycemia, hyponatremia, hypernatremia, hypocalcemia, hypercalcemia, hypokalemia, hyperkalemia and metabolic acidosis may also have an endocrine aetiology.

Diabetic ketoacidosis

Among the endocrine emergencies in children, diabetic ketoacidosis (DKA) is relatively common. Severe insulin deficiency is the primary cause of DKA. Lack of insulin decreases the activity of GLUT4 glucose transporter and hence glucose cannot enter the cells (except in the brain which has a non-insulin regulated glucose transporter). As a result, intracellular low glucose level induces counter-regulatory hormone production and hyperglycemia raises serum osmolality causing osmotic diuresis. The combination of insulin deficiency and counter-regulatory hormone excess sets into motion the vicious cycle of excessive lipolysis and uncontrolled fatty acid oxidation with ketoacids (ketone bodies) production in the liver leading to metabolic acidosis, ketosis, dehydration, electrolyte depletion and ketonuria. Ketoacids often cause nausea and vomiting which increase the severity of dehydration. Dehydration in turn reduces renal clearance of glucose and ketoacids, further worsening the hyperglycaemia and acidosis. In effect, all these compensatory metabolic changes also contribute towards a significant risk of mortality making DKA a life-threatening emergency.

DKA generally occurs as a presenting manifestation at the time of initial diagnosis in
nearly 40% of children with type 1 diabetes mellitus (T1DM), especially in those under 5 years of age. This is mainly because the parents are too slow to recognize the child’s symptoms even when these develop over a few days or weeks. Such delayed diagnosis may be the cause of DKA even in the absence of a precipitating infection. DKA may also occur in an established case of T1DM during a stress situation, commonly a severe infection, or when insulin injections were omitted for various reasons, despite detailed parental education on home management of T1DM – parents seeking herbal and other indigenous therapies; omission of insulin during a severe infection, often influenced by poorly informed relatives with wrong concepts regarding chronic therapy; depressed or rebellious adolescents especially with lack of parental supervision.

Although DKA is potentially life-threatening, it is rarely fatal when the condition is promptly recognized and appropriate treatment is given. However, the best approach is its prevention through early recognition of symptoms such as polyuria, polydipsia, enuresis, lethargy, weight loss in a new case of T1DM and by education of families in confirmed cases regarding the importance of regular insulin therapy, the absolute need to provide increased insulin dosages during an intercurrent infection and the futility of the promises made by indigenous practitioners who do not have any effective alternative for insulin therapy.

Besides providing insulin, urgent treatment is required for the profound fluid and electrolyte deficits, hyperosmolarity and acidosis seen in DKA. This therapy should be carried out under close supervision and monitoring, as rapid treatment may by itself lead to complications such as cerebral oedema especially in infants and very young children.

The clinical features of DKA are polyuria, polydipsia, weight loss, lethargy, malaise, anorexia (or polyphagia in some), nausea, vomiting and abdominal discomfort or colicky pain which may even be mistaken for a surgical abdomen. Children may present with severe dehydration, hypotension and shock, drowsiness, unconsciousness or coma. Rapid deep breathing (Kussmaul’s respirations) suggestive of metabolic acidosis is a common presentation. There may be a fruity odour to the breath.

DKA is often precipitated by an infection and hence this should be actively searched for, evaluated and treated appropriately. Sinusitis, urinary tract infection, cutaneous infections or pneumonias are the common causative illnesses but may not be obvious on physical examination or x-rays. Even fever may be absent in the presence of dehydration. Interestingly, leucocytosis and a shift to the left observed on differential WBC count are observed consistently even if infection is not present. These changes are due to elevated levels of adrenaline and cortisol in DKA.

Emotional and psychosocial factors may also predispose to the onset of DKA in children with T1DM. However, in our country, where mostly family support is widely available for the children, psychological factors do not contribute to the evolution of DKA.

Diagnosis of DKA is made when the following are present.

1. Blood glucose 300 mg/dl,
2. Acidosis is present with arterial pH < 7.3 and serum bicarbonate < 15 mEq/L,
3. Serum bicarbonate < 18 mEq/L,
4. Marked glucosuria (++++ or ++++),
5. Severe ketonuria (++ to ++++).

Classification of DKA

Mild–pH > 7.2 and serum bicarbonate 10-15 mEq/L
Moderate – pH 7.1 - 7.2 and serum bicarbonate 5-10 mEq/L

Severe – pH < 7.1 and serum bicarbonate < 5 mEq/L

Treatment

Urgent and prompt treatment is essential but at the same time, it should be optimal and monitored closely to avoid the risk of cerebral oedema. This is an important complication of therapy, more often seen in infants and young children under the age of five. Children with DKA are best managed in an institutional setting with intensive care facilities. However, if a large centre is away at a considerable distance, these children can be managed quite effectively at even lower level hospitals. If the patient is referred to another hospital, it is always a wise practice to administer one dose of rapid acting insulin subcutaneously before sending away the child, to avert any further deterioration during the journey.

At admission, a blood sample is collected for blood glucose, urea, serum sodium, potassium, bicarbonate, venous blood gases and glycosylated hemoglobin. Urine is examined for the presence of glucose and ketones.

Fluid and electrolyte replacement (Table 1)

This is an important step in the treatment and is carried out after initial assessment of the patient. DKA is invariably associated with severe dehydration and should be treated so, even in the absence of all the signs of dehydration. Since hyperosmolarity is the rule in DKA, the initial hydrating fluid chosen is usually normal saline. Hypovolemic shock requires immediate commencement of rehydration over the next hour to expand peripheral circulation. An isotonic fluid such as normal saline or Hartmann Ringer Lactate solution may be administered at 15-20 ml per kg body weight. Moisture of mucous membranes and capillary refill time are useful in assessing hydration. When the hydration improves, glomerular filtration increases favouring glucose excretion. Hence, the blood sugar level keeps falling even prior to commencement of insulin infusion.

In a typical DKA case, the fluid deficit is calculated as 6% (60ml/kg) of body weight and 10% (100 ml/kg) for a child < 2 years of age. Using this volume, rehydration is done carefully over a period of 36 to 48 hours to avoid the complication of cerebral oedema. In a child presenting with clinical signs of severe dehydration, fluid deficit is calculated at 9% (90ml/kg) of body weight and 15% (150ml/kg) for a child < 2 years of age. It should be remembered that if the initial blood sugar value is 800 mg% or higher, or if the corrected serum sodium level is in the hypernatremic range, fluid deficit must be calculated for more severe dehydration. Measured hypernatremia will be another pointer to severe dehydration. The initial fluid chosen is normal saline and should be continued until the blood sugar level approaches 250 mg%, when a change to 5% dextrose saline solution may be done. Monitoring of all fluid intake and output during treatment and recording is essential. The temptation for overzealous treatment to correct the dehydration rapidly in the case of children who look very ill should be resisted.

A gradual decline in serum osmolality is desirable to avoid the complication of cerebral oedema. Hence 50-60% of the total calculated deficit is replaced within the initial 12 hours and the remainder over the next 24 hours. Maintenance fluids are calculated by carefully monitoring urine output during treatment.

Total body depletion of potassium occurs as a rule in DKA. Besides, during insulin therapy there is a risk of severe hypokalemia and requires careful potassium replacement. Serum potassium
level is estimated initially and monitored regularly. When serum potassium reaches the normal range, and urine output is good, potassium chloride is added to the intravenous fluids after the first hour of rehydration, usually at the rate of 10-20 mEq/L, but may need up to 40-60 mEq/L if there is protracted vomiting, hypokalemia or persistent acidosis. If the child is oliguric at the end of the first hour of rehydration, potassium chloride is added only if the serum potassium is < 4 mEq/L or if the ECG shows evidence of hypokalemia. Sodium deficit present in DKA is mainly due to osmotic diuresis. By its osmotic force, hyperglycemia draws intracellular water into the vascular space. The increased vascular volume dilutes the sodium content of the blood giving rise to “pseudohyponatremia”. Hence a correction is applied to arrive at the true level of serum sodium. For every 100 mg% rise in blood sugar, 1.6 mEq/L must be added to the actual value of serum sodium. The sodium deficit is taken care of by the normal saline solution used in initial rehydration and continued subsequently. Normal saline with added potassium is used as the hydrating fluid initially and later with 5% dextrose. Higher concentrations of sodium in the intravenous fluids will be needed in children who are at a high risk of developing cerebral oedema. Bicarbonate losses are huge in

Table 1. Guidelines for management of diabetic ketoacidosis

<table>
<thead>
<tr>
<th>Initial investigations:</th>
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<tbody>
<tr>
<td>Blood glucose, serum Na, K, Cl, HCO₃, blood gases, creatinine</td>
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<tr>
<td>Search for a precipitating infection – may need urine culture, blood culture, throat culture, chest radiograph</td>
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<tr>
<th>Indications for ICU care:</th>
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<tbody>
<tr>
<td>Unconscious child</td>
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<tr>
<td>Severe DKA</td>
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<tr>
<td>pH &lt; 7</td>
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<tr>
<td>Age &lt; 2 years</td>
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<tr>
<td>Blood glucose &gt; 1000 mg%</td>
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<th>Initial therapy:</th>
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<tr>
<td>20 ml/kg of 0.9% saline over 1 hour</td>
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<tr>
<td>Assess level of dehydration, calculate fluid replacement required, add maintenance fluids and administer intravenously over 36-48 hours Use normal saline solution as the initial intravenous fluid</td>
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<tr>
<td>Begin insulin infusion 0.1 unit/kg/hour after initial bolus of NS. Aim for fall of blood sugar at the rate of 100 mg%/hr and rise of blood pH by 0.03 / hr</td>
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<tr>
<td>Constantly monitor the patient’s vital signs, intake and output, 2-hourly blood sugars, pH, electrolytes, creatinine, urine ketones on all urine samples till negative on 2-3 consecutive samples</td>
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<tr>
<td>Continue insulin infusion till acidosis improves, even if the blood sugar level drops to 300 mg/dL</td>
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<tr>
<td>Change normal saline IV infusion to 5% dextrose saline when blood sugar is ~ 250mg/dL</td>
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<tr>
<td>Subsequently insulin infusion may be reduced by 0.05 units/kg/hr</td>
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<tr>
<td>Reassess the patient hourly at first, then every 2-3 hrs</td>
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<tr>
<th>Later therapy:</th>
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<tbody>
<tr>
<td>When the child has regained consciousness, feels hungry, is clinically more stable, offer clear fluids orally</td>
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<tr>
<td>If oral fluids are tolerated well, change to 6-hourly short acting regular insulin SC when the child’s condition is stable. When ketonuria has disappeared completely, mixed split insulin regime with intermediate and short acting insulins may be commenced</td>
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</table>
DKA, yet bicarbonate replacement is not usually required unless the pH is very low, (< 7.0). Correction of ketoacidosis can be achieved with initiation of insulin treatment and fluid replacement and bicarbonate infusion does not hasten this. On the other hand, bicarbonate infusion may increase the risk of cerebral oedema, worsen hypokalemia, and reduce tissue oxygenation. Phosphate losses are also high in DKA and to correct this, it is recommended that one half of potassium replacement may be given as potassium phosphate and the other half as potassium chloride. But in clinical practice, hypophosphatemia causing complications is extremely rare.

**Insulin therapy**

Continuous intravenous insulin infusion with regular insulin is recommended to ensure reversal of hyperglycemia to normal. The time to achieve this can be controlled by insulin infusion in such a way that it is not too slow or too rapid, as both can be risky. Insulin infusion is prepared by adding 50 units of short acting insulin (e.g. Actrapid, Humulin-R) to 500 ml of normal saline, so that 10 ml of the solution will contain one unit of insulin. This solution is prepared afresh every 24 hours and used in a pediatric microdrip set or infusion pump. The usual rate of insulin infusion is 0.1 unit/kg/hour and if acidosis is not corrected in the first few hours of insulin therapy, the rate of the drip may be increased by 0.05 unit/kg/hour, until acidosis improves. Generally, acidosis improves more slowly than hyperglycemia. If the rise of pH during therapy is slower than 0.03 per hour, higher rate of intravenous rehydration or insulin infusion may be tried. This drip is given separately from the other intravenous fluids used so that the insulin infusion can be regulated independently. Precaution is taken to prevent rapid fall of blood glucose to hypoglycemic levels by increasing the rate of glucose infusion rather than reducing the insulin administration. An important aim of therapy is to avoid cerebral oedema. Intermittent subcutaneous injections of regular insulin can be used for milder cases of DKA. It should be remembered that even in small towns, where there are no intensive care facilities or infusion pumps, children with DKA can be managed quite effectively with intermittent multiple intramuscular injections of insulin initially and later by subcutaneous doses of insulin with very good results. This may be particularly useful in a situation wherein the child presenting with life-threatening DKA may be stabilized first in this manner before referring the child to a large medical centre. Thus mortality during the travel can be successfully prevented.

Blood sugar monitoring must be carried out throughout the treatment to ensure a steady and gradual fall of blood sugar by 100 mg/dL per hour, to avoid hypoglycemia and to decide the switch over to a dextrose containing intravenous fluid. Insulin infusion is reduced when the acidosis is showing improvement and blood sugar continues to fall despite administration of dextrose infusion intravenously.

When the child’s consciousness also improves along with improvement of all the laboratory parameters, clear fluids are at first tried orally. If tolerated well, one can switch over to subcutaneous 6-hourly short acting insulin and solid food may be given. Every sample of urine is checked for ketones throughout this treatment and when ketones are consistently negative on 2-3 samples, short acting insulin may be replaced by a mixture of intermediate and short acting insulins, preferably before breakfast or dinner. **Cerebral oedema**

This is an unusual (in 1% of cases) but major complication of treatment for DKA, more often seen in infants and young children below the age of 5 years at the time of initial presentation. As it carries a high mortality, great care is taken to avoid this complication as best as possible.
Prompt clinical diagnosis, initiation of treatment with mannitol or other hyperosmolar agents, hyperventilation and respiratory support are essential to manage this life threatening complication. It often occurs after 4-6 hours after treatment has begun when biochemical abnormalities are improving. The clinical features are: recurrence of drowsiness following improved alertness with treatment, recurrence of vomiting, bradycardia, hypertension, headache, abnormal CNS findings, such as irritability, disorientation, unequal dilated or unreactive pupil, papilloedema, coma, and respiratory arrest with herniation of the brain stem. CT or MR scans done when the patient is sufficiently stable will detect the changes in the brain.

The head end of the bed is raised by 30 degrees, airway is secured, respiratory support is by endotracheal intubation with paralysis and sedation, and hyperventilation is commenced. The intravenous fluid rate is reduced to maintenance or less. Mannitol (0.5 to 1 gram/kg intravenously over 5 minutes) is an important aspect of the management and is repeated in half the dose once or twice till a response is obtained. Hyperventilation and deep sedation may be required for 24-48 hours.

The adverse prognostic factors in DKA are: young age, delay in diagnosis and treatment, severe dehydration, low PCO₂, elevated serum creatinine, treatment with bicarbonate, failure of rise of serum sodium with treatment, rate of fluid infusion > 4.0 L/M²/day.

**Adrenal insufficiency**

Adrenal insufficiency may occur in primary adrenal conditions which may be due to enzymatic block as in congenital adrenal hyperplasia (CAH), acquired causes (autoimmune or idiopathic cases, termed Addison’s disease) or due to pituitary causes such as ACTH deficiency. In the newborn, transient adrenal insufficiency may occur with maternal glucocorticoid therapy in antenatal period or may be due to adrenal haemorrhage following breech presentation, hypoxia or sepsis. Congenital adrenal hypoplasia is a rare cause of hypoadrenalism in the neonatal period primarily affecting boys but this condition may also present in later childhood. Autoimmune adrenalitis causing adrenal insufficiency is unusual in childhood.

Adrenal insufficiency may also occur with adrenal haemorrhage in infectious conditions such as meningococcemia (Waterhouse-Fridericksen syndrome), in hypovolemic shock and hypoxia. In some children with severe hypothyroidism, adrenal insufficiency may be coexisting and requires caution during thyroxine therapy. Attaining euthyroid status may precipitate acute adrenal insufficiency with vascular collapse. Some rare conditions with adrenal insufficiency are: adrenoleucodystrophy, Smith-Lemli-Opitz syndrome (a disorder of cholesterol synthesis with abnormally low cholesterol, elevated 7-dehydrocholesterol and adrenal insufficiency) and Wolman disease (presenting with adrenal calcifications, intra lysosomal accumulation of cholesterol esters in many organs). Certain drugs such as the antifungal ketoconazole, rifampicin, phenytoin, phenobarbital and mitotane are also known to suppress the adrenal cortex.

**Clinical manifestations**

A high index of suspicion is necessary for diagnosis as otherwise vague symptoms such as fatigue and weakness may be missed. In severe cases, acute vascular collapse (shock) may occur but may still be mistaken for septic and other causes of shock. Neonates with CAH commonly due to salt losing form of 21-hydroxylase deficiency may present with hyperpigmentation, vomiting, polyuria (which is difficult to detect in a newborn), ambiguity of genitalia in a female infant, failure to gain weight, unexplained dehydration and acute onset of hypotension and
shock. Occasionally, 3-hydroxysteroid dehydrogenase deficiency also manifests with salt losing symptoms in the neonatal period. Older children and adolescents with adrenal insufficiency present with apathy, confusion, vomiting, dehydration, muscle weakness, abdominal pain, orthostatic hypotension and may need intravenous fluid resuscitation during a stress situation such as an asthmatic episode, a lung infection or even a minor infectious illness. Hypoglycemia is a prominent feature. When this induces breakdown of fat and mobilization of fatty acids as an alternate source of energy, ketosis results giving rise to anorexia, nausea and vomiting. The illness may be mistaken for an episode of gastroenteritis. Weight loss, growth failure and hyperpigmentation may be features of chronic primary adrenal insufficiency.

**Investigations**

Serum cortisol, ACTH, electrolytes, blood sugar, renin and aldosterone levels are measured at 0800 hours. Hyponatremia, hyperkalemia, hypoglycemia and ketosis are the findings that should suggest the possibility of adrenocortical insufficiency. ACTH level is high, urinary excretion of sodium and chloride is increased. Low basal cortisol with elevated plasma ACTH values indicates primary adrenocortical insufficiency as against low ACTH in secondary cases. Hyponatremia and hyperkalemia with elevated plasma renin concentration suggests mineralocorticoid deficiency. Elevated serum 17-hydroxyprogesterone will suggest CAH.

Short synacthen stimulation test is the confirmatory test for adrenal insufficiency. The basal value of serum cortisol is low and does not rise after a intravenous bolus of injection ACTH in a case of primary adrenal disorder. If the resting level is normal and there is a significant response to ACTH, secondary adrenal insufficiency should be suspected. Elevated very long chain fatty acid level in the plasma is diagnostic of adrenoleucodystrophy. Anti adrenal antibodies suggest an autoimmune pathogenesis.

**Treatment**

Blood samples must be collected for confirmation of diagnosis before any treatment is given. If the patient is in a stable condition, ACTH stimulation test may be carried out while the child is receiving fluid replacement therapy. Urgent replacement with water soluble hydrocortisone sodium succinate (100 mg/m² intravenously at 6-8 hour intervals) intravenously is essential in acute adrenal crisis along with fluids, electrolytes and glucose to replace fluid and electrolyte deficit, and to correct hypoglycemia. Normal saline solution with 5% dextrose is given intravenously to correct the situation and this will provide adequate fluid and sodium. If hyperkalemia is severe, treatment with IV calcium and/or bicarbonate or a cation exchange resin will be needed. Hydrocortisone (cortisol) replacement is continued orally in maintenance doses (10 mg/m²/24hours) after the acute manifestations of adrenal insufficiency are treated. Although thrice daily dosage is recommended, compliance is always a problem. A larger dose is given in the morning to match the physiological circadian rhythm of cortisol secretion. In the presence of mineralocorticoid deficiency, fludrocortisone is given orally in a dose of 50 to 300 micrograms daily in two divided doses. These maintenance doses are increased 2-4 folds temporarily during any acute stress situation such as surgery, infection or an accident. If oral cortisol is not tolerated during an emergency, intramuscular injection of hydrocortisone is essential and needed urgently. Monitoring the efficiency of the replacement therapy is done by periodic evaluation of serum cortisol levels and plasma renin activity. Overdose with glucocorticoid will produce all the manifestations of Cushing’s syndrome and with mineralocorticoid one can expect tachycardia, hypertension, fluid retention and occasionally hypokalemia.
Syndrome of Inappropriate Antidiuretic Hormone Secretion

The syndrome of inappropriate antidiuretic hormone (SIADH) secretion is the most common cause of euvolemic hyponatremia in pediatrics. The characteristic findings in this syndrome are: hyponatremia and hypo-osmolality resulting from inappropriate continued secretion and/or action of antidiuretic hormone (ADH) despite normal or increased plasma volume and low serum osmolality.

Arginine vasopressin (AVP), the naturally occurring ADH in humans, is synthesized in the cell bodies of neurons in the supraoptic and paraventricular nuclei of the anterior hypothalamus and carried along the supraopticohypophyseal tract into the posterior pituitary, where it is stored in association with a carrier protein, neurophysin. ADH is released from here directly into the circulation.

The release of ADH from the posterior pituitary is dependant on:

1. Osmoreceptors which detect changes in the extracellular fluid (ECF) osmolality. ADH release results from a 2% increase in the serum osmolality perfusing the supraoptic nuclei and ADH secretion diminishes with a 1.2% decrease in the serum osmolality, halting completely at plasma osmolality < 280 mOsm/kg.

2. Baroreceptors, located in the carotid sinus, aortic arch, and left atrium, induce a significant release of ADH with a 8-10% reduction in plasma volume.

These two sets of receptors normally act in close coordination to increase or decrease ADH release, although intravascular volume may be the major stimulus. ADH release is also affected by several drugs and stress situations such as pain or anxiety.

ADH mainly favours the reabsorption of water in the tubular fluid from the distal tubules and collecting ducts and has no effect on sodium reabsorption. ADH also exerts a pressor effect by causing arteriolar vasoconstriction and a rise in arterial blood pressure.

Pathophysiology

The basic defect in SIADH is uncontrolled excess of vasopressin secretion giving rise to water retention and volume expansion, presenting as puffiness of the face and increase in body weight. The clinical manifestations are often obvious when the patient continues to drink or has a fluid overload. Urinary sodium content will be high although the serum sodium and osmolality values are low. Increased extracellular fluid volume elicits decreased proximal tubular sodium absorption in an effort by the kidney to excrete sodium and decrease intravascular volume.

Hypervolemia suppresses the renin-angiotensin-aldosterone system during the water retention phase, but later, levels of renin and aldosterone rise again, perhaps in response to hyponatremia. The main mediator of the natriuresis in SIADH is probably the atrial natriuretic peptide (ANP), which may suppress proximal tubular reabsorption of sodium in response to expanded ECF volume. Sodium balance is maintained in SIADH, and the sodium output equals the intake.

Causes

CNS disorders: SIADH in children is most often observed in association with intracranial disease or injury (i.e., bacterial or tuberculous meningitis, brain abscess, encephalitis, head injury) and in postoperative patients.

Neoplasms: Malignancies producing excessive ADH secretion are uncommon in children.
Pulmonary disorders: Pneumonia and pulmonary tuberculosis causing SIADH are less common causes in children than in adults.

Excessive administration of vasopressin in the treatment of central diabetes insipidus.

Drugs: Vincristine, cyclophosphamide, carbamazepine

Clinical features

It is good to remember that in a vast majority of cases (~90%), it is a self-limiting condition, remitting spontaneously within 2 – 3 weeks of the initial event. Overt clinical manifestations of SIADH are largely related to the cellular swelling and cerebral oedema associated with hyponatremia. Most patients with SIADH are asymptomatic if the serum osmolarity remains above 240 mOsm/kg of water. The net result in SIADH is that the child is unable to excrete water. Hence the clinical manifestations of SIADH are those of water intoxication. Symptoms are more likely to develop in young children and elderly patients with hyponatremia.

• SIADH occurs most frequently in children with central nervous system infections, intrathoracic disease, and in postoperative patients.
• Among premature neonates, the syndrome most often accompanies brain injury and is closely associated with intracranial hemorrhage.
• Signs and symptoms of SIADH, as a rule, are those of hyponatremia and often are vague and nonspecific – nausea, vomiting, headaches, blurred vision, disorientation.
• The clinical manifestations of SIADH are usually related to the degree of the hyponatremia and to the rate at which hyponatremia develops.

SIADH is often first recognized on finding hypotonic hyponatremia in a child without other major symptoms and in the absence of dehydration.

Puffiness of face and weight gain of nearly 5% may seen. Skin turgor and blood pressure usually are normal. Obvious hypervolemia is absent.

Deep tendon reflexes are depressed and pathologic reflexes, such as positive Babinski reflexes, may be present. Altered sensorium with asymmetric pupils may be seen. Pseudobulbar palsy and seizures may occur. Cheyne-Stoke respirations may be present.

Diagnostic clues

Hyponatremia (serum sodium <135 mmol/L) with corresponding hypoosmolality (serum osmolality <280 mOsm/kg) is characteristic. There is continued renal excretion of sodium with urinary sodium level > 25 mmol/L. Urine is less than maximally dilute with urine osmolality > 100 mOsm/kg (more than plasma) and urine volume is low. Serum potassium remains unchanged. Other causes of hyponatremia such as adrenal insufficiency, congestive heart failure, pituitary deficiency, renal disease, hepatic disease and use of diuretic are absent.

Treatment

Treatment of hyponatremia in SIADH depends on the presence or absence of symptoms, the severity of hyponatremia, and its duration. Asymptomatic patients are usually treated in the immediate period with water restriction. Patients with CNS symptoms usually require more rapid correction of the hyponatremia and water restriction alone may not be sufficient.

Fluid restriction

Reduced water excretion by the kidneys is responsible for the physiological and biochemical
abnormalities in SIADH, such as hyponatremia, volume expansion, and sodium depletion. Therefore, water restriction corrects all these abnormalities and is the most important step in treatment of patients with SIADH. Fluid restriction to less than 75% of maintenance (i.e., 1000 mL/m²/d) usually allows for the slow excretion of retained excess fluid and results in a decrease in ECF volume with a concomitant fall in urinary sodium excretion.

If no improvement occurs in 4-6 hours, further fluid restriction to 50% of maintenance (i.e., 700-800 mL/m²/d) or lower is necessary. A few children may require more severe fluid restriction to as little as 10% of maintenance (i.e., 150-200 mL/m²/d). Sodium chloride intake is maintained during fluid restriction. 5% dextrose in 0.45 isotonic sodium chloride solution or 5% dextrose in lactated Ringer solution can be used, if intravenous fluids are indicated.

In most children with SIADH with mild to moderate symptoms, fluid restriction helps within 24 hours. Fluid intake can be increased as serum electrolytes and osmolality normalise.

**Hypertonic sodium chloride solution**

Use of hypertonic sodium chloride solution (3%) in children with SIADH is not often helpful and is indicated only when severe neurologic disease is present, viz., seizures or coma induced by hyponatremia (serum sodium <120 mmol/l). It may worsen the underlying condition by expanding the ECF volume further, resulting in greater decrease in sodium reabsorption by the proximal tubule and excretion of the administered sodium. A risk of heart failure exists in a patient who already is volume expanded.

Corticosteroids are of no direct benefit in SIADH. Vasopressin analogs with intrinsic antidiuretic antagonism are very promising but still experimental. Thiazides decrease free water excretion at the cortical diluting segment and can severely aggravate hyponatremia in patients with SIADH.

**Complications**

- Fluid overload
  - Pulmonary oedema
  - Hypertension
  - Anasarca
- Acute extracellular hypoosmolality
- Cerebral edema (may be observed at rates of plasma osmolality decrease faster than 10 mOsm/kg/hr)
- Permanent brain damage
- Cerebral herniation (has been observed in postmortem examination in both humans and experimental animals)

**Prognosis**

- Prompt recovery usually follows water restriction.
- Prognosis of SIADH is usually that of the underlying disease.

**Points to remember**

**Diabetic ketoacidosis**

*Although diabetic ketoacidosis is potentially life-threatening, it is rarely fatal when the condition is promptly recognized and appropriate treatment is given.*

Early recognition of symptoms such as polyuria, polydipsia, enuresis, lethargy, weight loss will certainly help in the diagnosis of type 1 diabetes mellitus which if treated promptly will avoid presentation with diabetic ketoacidosis.

In children with type 1 diabetes mellitus, the following points are to be emphasized to avoid precipitating diabetic ketoacidosis: the
importance of regular insulin therapy, the absolute need to provide increased insulin dosages during an intercurrent infection and the false promises made by indigenous practitioners.

Adrenal insufficiency

A high index of suspicion is necessary for diagnosis as otherwise vague symptoms such as hyperpigmentatiion, fatigue and weakness may be missed.

Older children and adolescents with adrenal insufficiency present with apathy, confusion, vomiting, dehydration, muscle weakness, abdominal pain, orthostatic hypotension and may need intravenous fluid resuscitation during a stress situation such as an asthmatic episode, a lung infection or even a minor infectious illness.

Syndrome of inappropriate ADH secretion

Signs and symptoms of syndrome of inappropriate ADH secretion, as a rule, are those of hyponatremia and often are vague and nonspecific - nausea, vomiting, headaches, blurred vision, disorientation.

The clinical manifestations of SIADH are usually related to the degree of the hyponatraemia and to the rate at which hyponatraemia develops.

Asymptomatic patients are usually treated with water restriction. patients with CNS symptoms usually require more rapid correction of the hyponatraemia and water restriction alone may not be sufficient.

Bibliography

SCORPION STING

* Mahadevan S

Abstract: The clinical features of scorpion sting are predominantly due to autonomic stimulation. Significant systemic manifestations are more commonly encountered in young children compared to adults in whom local effects predominate. Prazosin acts like a specific physiological antagonist and is indicated early in presence of systemic manifestations. In refractory cardiogenic shock and pulmonary oedema, ventilatory support with inotrope and vasodilator infusion in an intensive care setting may be life-saving.

Keywords: Scorpion sting, Child, Prazosin, Autonomic storm.

Scorpion sting is a common problem worldwide and often, children are victims of fatal stings. The clinical features following envenomation by the Indian red scorpion (Mesobuthus tamulus) are predominantly due to the effect of autonomic stimulation on the cardiovascular system. The steps involved in managing these children are outlined below.

Diagnosis

Step 1

Confirmation of the sting is done by history given by the eye witness or by observing the killed scorpion.

Step 2

Differentiation of a benign sting from potentially fatal envenomation. Severe local pain, local sweating and mildly raised blood pressure and no autonomic storm, indicate a non poisonous sting.

Step 3

Identification of autonomic storm which is evident soon after the sting (minutes to 4 hours). Vomiting, profuse sweating, cold extremities, excessive salivation, saliva as a rope, priapism in males and paresthesia all over and around the mouth are features of autonomic storm.

Cardiovascular signs include hypertension or hypotension, cardiac arrhythmias, sinus bradycardia or tachycardia, S3 gallop, transient non sustained ventricular tachycardia, transient systolic murmur and left ventricular failure. Oculogyric phenomenon, propped up eyes, puffy face and abdominal colic are seen in those with hypertension. It is observed that in children with scorpion sting profuse sweating may last for 7-20 hours, priapism / mydriasis for 6-18 hours, hypersalivation for 2-12 hours and tachycardia alone for 12-18 hours. Anuria, pulmonary edema presenting with pinkish froth and cyanosis, hypotension, convulsions and shock are late manifestations. Persistent tachypnea is an early sign of pulmonary edema in children.

The cause of hypotension in these children can be due to any one of the following factors.

Early short lasting: Hypovolemia (due to profuse sweating and vomiting), peripheral cholinergic or central vagal.
Delayed long lasting: Myocardial failure or decreased vascular resistance.

Asymptomatic (72-96 hours): Exhausted catecholamine stores.

Central nervous manifestations are infrequent. Intra cerebral hemorrhage is invariably fatal. Late presentations include, hemiplegia and choreoathetosis which may appear as late as 10 days after recovery from acute symptoms.

Management

Close monitoring of the following parameters is an essential part of management, viz cold peripheries, pulses, respiratory rate, heart rate, blood pressure and S3, S4 gallop. ECG and chest x-ray are needed in any child with significant features of systemic envenomation. Common ECG changes encountered include, premature ventricular contraction, bigeminy, tented T wave, acute myocardial infarction like pattern, ST depression, injury to conducting system i.e left anterior hemiblock, left or right bundle branch block and QT >500 msec.

For non poisonous sting: Pain relief is done by cooling of the affected part or local anesthetic agent; oral paracetamol and oral diazepam may be used.

Poisonous sting: Hospitalize for frequent monitoring and stabilisation of hemodynamics. Key clinical features determining the need for management in a High Dependency Unit or Intensive Care Unit are severe tachycardia, palmoplantar sweating, S3 gallop, hypotension, shock, pulmonary oedema and ECG changes.

Correction of dehydration is important. Vomiting, salivation and sweating contribute to dehydration. Confused agitated child can be given fluids by NG tube. Restriction of fluid due to fear of pulmonary edema is a common mistake. Hypovolemia correction is a priority. Oral rehydration or intravenous crystalloids to be given as dictated by the clinical picture.

Prazosin, a post synaptic α adrenergic receptor blocking agent, should be given in a dose of 30µg/kg in children which should be repeated three hourly until there are signs of clinical improvement in tissue perfusion such as warming of extremities, increase in urine output, appearance of severe local pain at the site of sting which was absent or tolerable on arrival, disappearance of paresthesias, reduction or improvement in heart rate and pulmonary edema, reduction in hypertension or improvement in blood pressure in case of hypotension without hypovolemia, reduction or disappearance of murmur and earliest most important subjective feeling of better or decreased restlessness in a small child. This is because the drug has 1000 times more affinity towards the activated alpha-1 receptors. Then dose is to be repeated six hourly till extremities become dry and warm. If the initial dose has been vomited (one should see the vomit carefully), it should be repeated. In a confused, agitated, non-cooperative child, prazosin should be administered by nasogastric tube. Prazosin is life saving drug hence attending doctor himself should administer the drug to the hospitalized patient and it should be clinically confirmed by noting the signs and symptoms that drug is absorbed in circulation and started acting. First dose phenomenon (fall in blood pressure following an intial dose of prazosin) is due to postural fall in blood pressure and is rare. This can be prevented by avoiding lifting the child and not allowing getting up from bed. Postural hypotension should be treated by giving head low position and intravenous fluid. Oral prazosin may be repeated every 3 hours till extremities are warm. After prazosin therapy, the following should be closely monitored to identify good response: Dilated peripheral veins, good volume pulse, warm extremities, reappearance of pain, adequate urine output, no paresthesia and natural sleep without sedation.
Pulmonary edema is a life threatening time limiting emergency, often fatal and needs rapid intervention. Patient should be in propped up position if there is no hypotension. Intravenous aminophylline 5mg/kg diluted in dextrose is given as a slow bolus to counter the associated bronchospasm. If available isosorbide buccal spray is useful or powder of nitroglycerine should be rubbed on gum and intravenous furosemide should be given to reduce the preload and pulmonary congestion. In cases of massive pulmonary edema (blood stained froth from nostrils and mouth), intravenous sodium nitroprusside (SNP) drip 0.5 microgram per kg per minute is started and dose raised continuously according to patient’s response and blood pressure up to 8 \( \mu \)g/kg/min. Blood pressure should be closely monitored and maintained at 80-90 mm Hg of systolic blood pressure. SNP has to be prepared from fresh powder every four hours; the bottle and saline set should be protected from light. At times a severe case may require 15-36 hours of SNP drip to clear pulmonary edema. Patient should be given oral or injectable cynacobalamine to avoid cyanide toxicity whenever SNP is given for long time. Before starting SNP, IV furosemide is given to avoid sudden fall of intra-ocular pressure and ocular bleed due to SNP drip. IV nitroglycerine can be used in a dose of 0.5-5 microgram per kg per minute if SNP is not available. In case of shock or hypotension, early administration of dobutamine 5-15 microgram per kg per minute along with SNP drip may be life saving. In children, after 20-24 hours of sting, marked tachycardia (130 and above), warm extremities, pulmonary edema or air hunger respond to IV dobutamine drip, which may be required for 48 hours.

Presence of pulmonary edema has no relationship to intravascular volume. One should not assume such patients to be fluid overloaded. Diuretics may be harmful. Cardiac output can be improved with dobutamine. Morphine is contraindicated.

In occasional victims with myocardial dysfunction, ventricular premature contraction or R on T phenomenon and ventricular tachycardia respond to intravenous lidocaine.

Triaging, categorizing victims of scorpion sting for appropriate management are summarized in Table 1.

Key instructions for the ICU staff handling scorpion envenomation are: (a) Propped up position, oxygen, sublingual nitroglycerin or isosorbide spray in patients with pinkish froth. (b) Prepare SNP from fresh powder every 4 hours. 

### Table 1 - Management of scorpion sting

<table>
<thead>
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<th>GROUP I</th>
<th>Local symptoms only: Analgesics.</th>
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<td>GROUP II</td>
<td>Systemic manifestations but hemodynamically stable: Prazosin and oral fluids</td>
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<td>Systemic manifestations and stable at admission with subsequent destabilization: Prazosin+dobutamine +/- Sodium Nitroprusside</td>
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<td>GROUP IV</td>
<td>Life threatening complications and hemodynamic compromise at admission: ICU protocol (Fig 1)</td>
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</table>
1. Oxygen By Mask / Nasal Prongs

2. Maintenance Intravenous Fluids after careful initial boluses of crystalloid

3. Intravenous Dobutamine Infusion (5 – 15 µg/Kg/min)

4. \(\alpha\)-Adrenergic Blocker (oral or through NG tube): Tab Prazosin (30 µg/Kg/dose)

No improvement in 4 – 6 Hours/ Deterioration

Steps 1, 2 & 3 as above

5. Intravenous infusion of Nitroglycerine (0.5 – 5 µg/Kg/min) if hypotensive (or) Sodium Nitroprusside (0.5 – 8 µg/Kg/min) if normotensive/ hypertensive ±

6. Mechanical Ventilation CPAP/ IMV

Improvement

Taper and stop Dobutamine infusion

Taper and stop Nitroprusside/ Nitroglycerine infusion (1 hour before stopping vasodilator infusion start oral prazosin. To be given Q6h for the next 24 hours)

Fig 1. Intensive care unit protocol for children with scorpion envenomation with hemodynamic compromise and / or pulmonary edema
hours. (Dose: SNP – 0.5 – 8 μg / kg/ min).
(c) Protect bottle and IV line from light.
(d) Nitroglycerine, another alternative. (NTG: 0.5-5 μg/kg/min) upto 12 - 36 hours.
(e) Ventilatory support can be life saving.
(f) Dobutamine 5-15 μg/kg/min as infusion.

Atropine, steroids, antihistamines, beta-blockers, calcium channel blockers, excessive diuretics, adrenaline and narcotics should be avoided. They do more harm than good in scorpion envenomation. Newer reports of carnitine for myocardial dysfunction in scorpion sting victims tend to shift the focus towards costly therapy for this rural emergency. Such uncontrolled observations could lead to neglect of life-saving cheaper alternatives like prazosin.

Indian experience is limited in the use of scorpion antivenin, though benefits are reported from USA, Mexico, Saudi Arabia and Brazil (Centruroides species)\(^4,5,6\). Trials in Tunisia (RCT) found no useful role for antivenin in severe envenomation\(^7\).

**Conclusion**

Alpha blockade effect of Prazosin prevents the evolution of serious cardiovascular morbidity in victims of scorpion sting. The interval between sting to administration of prazosin is an important prognostic factor. A standard intensive care protocol with dobutamine, sodium nitro prusside/nitroglycerin has been successfully adopted for this emergency\(^8\).

**Points to remember**

1. **Significant systemic effects of scorpion envenomation in children are due to outpouring of catecholamines and predominently involve the cardiovascular system.**

2. **Prazosin acts like a physiological antagonist and its early administration when systemic manifestations appear, ensures a better outcome.**

3. **Escalated therapy in the form of ventilatory support, inotropes and vasodilators in an intensive care unit may be required in cardiogenic shock or massive pulmonary oedema.**

**Reference**

Abstract: Snake bite, a predominantly rural problem, mainly occurs in older children with increasing outdoor activity. Out-of-hospital management is limited to immobilizing the limb and transporting to a center where assessment and if necessary, ASV administration can be done. In the hospital, initial stabilization and supportive measures are a priority. ASV is required only if there is systemic manifestation and significant local reaction. The dose is based on grading of clinical manifestations and children require as much dose as adults.

Keywords: Snake bite, Snake envenomation, Anti-snake venom.

There are about 3500 known species of snakes seen worldwide, of which about 500 species are poisonous. Among the 330 species of snakes found in India, 70 species are poisonous (40 land snakes and 30 sea snakes). Annually India records about 10,000-15,000 deaths due to snake bite. The case fatality rate being 2-10%. The mortality rate is higher in children.

The most common Indian venomous snakes referred to as the “Big Four” are the common krait, common cobra, saw-scaled viper and Russell’s viper. The clinical manifestations are mainly due to the venom having hemotoxic, neurotoxic and myotoxic components (Table 1).

Pathophysiology of venomous snake bite

Among the various species, the average yield per bite in terms of dry weight of lyophilized venom is 60mg for cobras, 63 mg for Russell’s viper, 20mg for krait and 13mg for saw scaled viper. The respective “fatal doses” are much smaller viz. 12mg, 15mg, 6mg and 8mg respectively. However, clinical features and outcome are not that simple to predict because every bite does not result in complete envenomation. Between 20% and 80% of venomous snake bites, even with puncture marks may not result in signs of envenomation.

Snake venom is a complex mixture of enzymatic and non-enzymatic compounds, nontoxic protein, carbohydrates and metals. There are 20 different enzymes like phospholipases A2, D-hydrolases, proteases, hyaluronidase, nucleotidase and ATP ase. The non enzymes are neuro-toxins and haemorrhagins. The pathogenesis and effects are given in Table 2.
Hemotoxic features are predominantly due to viper bites and neurotoxic features are due to cobra and krait bites. Local effects are seen both in viper and cobra bites.

**Approach to an individual allegedly bitten by a snake**

Determine whether the patient is actually bitten by the poisonous snake: Elicit focussed history. Look for fang marks which may vary from a few millimeters to as much as 4 cm, depending upon the species. The depth of the bite varies anywhere from 1-8 mm. In some cases of bites, fang marks may not be visible at all. Time of onset of poisoning may be as early as 5 minutes in cobra bites or as late as 10 hours in krait bites. In viper bites the mean duration of onset of symptoms may be 20 minutes. In sea snake bites the myotoxic features occur within 2 hours.

**Factors determining the severity of envenomation**

1. Age: Younger the child, more severe the features
2. Location: Bites on face and neck and directly into the blood stream are more dangerous
3. Activity: Running or active movement of the limb after the bite increases the risk of venom absorption.

**Clinical manifestations**

Apart from non-specific symptoms like severe vomiting, headache, myalgia, vertigo, tingling and numbness over tongue, mouth and scalp and hypersalivation, there may be specific local and systemic manifestations. When the child is brought to the hospital, a rapid clinical examination of vitals (airway, breathing and circulation) and features of local and systemic envenomation is done.

**Local**

- Local-pain, tenderness, oedema within 6 to 8 minutes up to 30 minutes.
- Local bleeding including petechial and purpuric rash and blistering are common in viper bites.
• Wet gangrenous lesions, blistering and compartment syndrome can occur in cobra bites
• Regional lymphadenopathy has been reported as an early and reliable sign of systemic poisoning.
• Local effects are minimal in krait bite.

Systemic Manifestations

• Neurological symptoms: Mostly seen in bites by cobra and krait. Ptosis is the earliest followed by external ophthalmoplegia, hyperacusis, weakness of muscles of palate, jaw, tongue, larynx, neck and muscles of deglutition. Generally cranial nerves are involved earlier, followed by drowsiness, coma and finally respiratory muscle paralysis. Diaphragm is affected terminally followed by respiratory failure.
• Bleeding manifestations are seen in bites by vipers characterized by prolonged clotting time, bleeding at the site of bite, skin bleeds, bleeding from gum, GIT, urinary tract and cerebral haemorrhages
• Cardiotoxic features like tachycardia, hypotension and hyperkalemic cardiac arrest can occur in viper bites. Myocardial infarction and sudden cardiac arrest may be seen in cobra and krait bites. ECG changes of hyperkale-mia can occur in renal failure due to bites by Russell’s viper and sea snakes.
• Acute renal failure may occur in Russell’s viper bite due to various reasons (prolonged hypotension, intravascular hemolysis or DIC with clinical picture like Hemolytic Uremic Syndrome). Muscle necrosis and myoglobinuria occur in sea snake bite which may also lead on to acute tubular necrosis.
• Transient severe abdominal pain may be seen in krait bite.

Recurrent manifestations of poisoning may occur due to ongoing action of the venom which has a half life of 26 to 96 hours. So, daily evaluation of the patient is essential for 3 to 4 days. Delayed manifestations in an initially stabilized patient can occur even after 3 weeks, the venom being released from local blebs which act as venom depots not accessible to anti-venom.

Investigations

Laboratory tests are useful for monitoring, prognosticating and determining type of intervention. ELISA studies are now available to identify the species involved based on the antigens but these tests are expensive and not freely available.

1. CBC—May show anemia, leucocytosis and thrombocytopenia
2. Peripheral smear—May show evidence of hemolysis (particularly in viperine bites) and DIC
3. Coagulation profile—Prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen, fibrin degragation products (FDP) and clotting time may be defective. Clot lysis which indicates the quality of clot formed may be a better indicator of coagulation capability than the actual time required for formation, since clot lysis has been observed in several patients who have normal clotting time. Prothrombin time (PT) is more sensitive for assessing coagulopathy. Clotting time by Lee White method (6–8 hourly till values normalize) is useful for periodic assessment of state of coagulation.

20 minute whole blood clotting time: Incoagulable blood is a cardinal sign of systemic envenomation by most of the viper bites. A simple bedside test is adequate for clinical purpose. This test is useful to monitor the effectiveness of ASV therapy when more sensitive tests of coagulation are not easily available. 2 to 3 ml of blood is taken and kept in a new, clean, dry, test tube undisturbed for 20 minutes. At the end of 20 minutes the tube
is tilted once to check if clotting has occurred. If blood is clotted it rules out significant coagulation disturbance.

4. BUN, creatinine, electrolytes-azotemia, hyperkalemia may be present

5. Urine analysis for hematuria, proteinuria, hemoglobinuria, or myoglobinuria

6. ECG: Changes are usually nonspecific and include bradycardia, AV block with ST segment elevation or depression, features of hyperkalemia if present

**Management**

i. Out-of-Hospital care (first aid)

ii. Supportive therapy

iii. Specific therapy

iv. Local wound management

**Out-of-Hospital care (First Aid)**

Out-of-Hospital care should focus on stabilization and rapid transport of victim to a health care facility with the capability of anti venom administration\(^8\). This includes:

1. Immobilization of the bitten extremity in a neutral position in every case with the help of a splint and not allowing the victim to walk or run is important. The patient should maintain strict rest, as movement of even the unbitten extremities increases the lymphatic absorption from the bitten side. He should be transported in a vehicle or on a stretcher in a lying down position. Constriction bands or pressure-immobilization method are recommended during transport of the victims.

Constriction bands: These are broad, flat bands applied proximal to the bite site to exert a pressure great enough to occlude superficial veins and lymphatics (>20 mm Hg) but, with enough space between the band and limb to admit one finger. It should not be so tight to obliterate the peripheral arterial pulse\(^8\).

Pressure-immobilization method: Firmly wrap the bitten extremity with an elastic bandage. The tightness of the wrap is described as approximating an elastic wrap for an ankle sprain. This technique slows the systemic absorption of the venom by trapping it at the bite site\(^8\).

2. Avoid incision and excision over the bite, chemical application, cauterisation, suction, cryotherapy, tourniquet and electric shock as these may cause more tissue injury leading on to gangrene and uncontrolled bleeding.

3. Avoid giving the patient food or drink, as there is a potential danger of vomiting due to envenomation

4. If the snake has been killed, it should be brought carefully to the hospital for species identification. It should not be touched with bare hands as some snakes may sham death and even severed head can inject venom\(^4\).

**Supportive therapy**

This is the most important aspect of the therapy.

1. Reassure the patient

2. Take care of airway, breathing, circulation (ABCs)

3. Monitor vitals, urine for hematuria and clotting time; monitor heart rate, respiratory rate, chest expansion and sensorium periodically. Close observation for early neurotoxic effects such as ptosis, ophthalmoplegia, speech and swallowing difficulty is periodically carried out.

4. Ventilatory support may be needed for respiratory failure or unstable airway.

5. Vascular access should be obtained in the unbitten limb. Treat shock with IV fluid boluses (NS or RL).

6. Fluid management and inotropes as needed to maintain normal perfusion.
7. Avoid IM injections
8. Anti-convulsants for seizures
9. Keep the pressure immobilization/constriction band in place till the antivenom is administered. When child is brought to hospital without constriction band or pressure immobilization, a sphygmomanometer cuff inflated 25-35mm Hg placed at least 2-4 inches proximal to the bite site or leading edge of swelling may be applied and kept during intra-hospital transport.
10. Sedation and analgesics for pain; avoid NSAIDs.
11. Send blood for grouping, typing and cross matching before administering anti-venom.
12. Neostigmine may be tried in Indian cobra and krait bite presenting with neurological manifestations - dose 50 -100 µg/kg IV every 4 hours with atropine 0.02 mg/kg IV five minutes prior to neostigmine

Specific therapy (Anti-venom)

Anti snake venom (ASV) ingredients: Polyvalent snake anti-venom manufactured by Haffkine Bio-pharmaceutical Corporation Ltd is lyophilized powder. It is of equine origin and is effective against the Big Four (common cobra, common krait, Russell’s viper and saw scaled viper). King Cobra bite will not respond to the commonly available ASV. So, specific monovalent ASV should be used. Each ml of the reconstituted snake anti-venom neutralizes 0.6 mg each of the Indian cobra and Russell’s viper venom, 0.45mg of krait and saw scaled viper’s venom. ASV is also available in liquid form (10ml/vial).

Indications: Every snake bite, even by poisonous species does not warrant snake anti-venom. The empirical use of anti-venom should be avoided due the risk of hyper sensitivity reactions. So ASV is indicated only if there are signs of local/systemic envenomation. When indicated, antivenom should be administered without delay.

Dose: Depends on the severity of the envenomation. The ideal dose is not known and there is no universally accepted standard regarding optimum dose of ASV. The recommended dose of ASV based on severity of clinical features is given in the Table 3. It should be remembered that the envenomation grade is only for the initial guidance for ASV therapy, as the severity can change over time. ASV doses may have to be repeated based on reassessment.

<table>
<thead>
<tr>
<th>Severity of envenomation</th>
<th>Clinical features</th>
<th>Amount of anti-venom</th>
</tr>
</thead>
<tbody>
<tr>
<td>No envenomation</td>
<td>No fang marks, no local/systemic reactions</td>
<td>NIL</td>
</tr>
<tr>
<td>Mild envenomation</td>
<td>Fang marks, local swelling and pain with/without lymphadenitis and local ecchymoses/purpura, no systemic signs</td>
<td>5 vials</td>
</tr>
<tr>
<td>Moderate envenomation</td>
<td>Above features + swelling progressing beyond the site of the bite, mild systemic symptoms like nausea, vomiting and paresthesia, mild coagulation defect (clotting time more than 10 mins, clot size &lt;50% of whole blood in the tube, a small speck)</td>
<td>10 vials</td>
</tr>
<tr>
<td>Severe envenomation</td>
<td>Marked swelling of extremity, subcutaneous ecchymoses, severe systemic signs and symptoms, DIVC, proteinuria, hematuria, encephalopathy, shock, paralysis incoagulable blood</td>
<td>15-20 vials</td>
</tr>
</tbody>
</table>
Preparation and administration of anti-venom

Mix 1 vial of anti-venom (lyophilised powder) with 10ml of distilled water and rotate it between the palms of the hands till the antivenom is fully dissolved and appears clear. Don’t shake vigorously. If foam appears or if the solution is turbid or milky, it indicates denatured protein and there is a greater risk of anaphylaxis if this is used. Before administering anti-venom, ask for history of previous administration of antiserum, allergy, bronchial asthma and urticaria.

Sensitivity test is done only if ASV administration is planned. Inject subcutaneously 0.1ml of the antivenom diluted 1:10. Observe the patient for 20-30 minutes for local/general reactions of hypersensitivity. If no reaction is seen, dilute the content of 1 vial (10 ml) with 40 ml NS(1:4 dilution). Administer this slowly over 1 hour. Watch for any reaction; if no reaction occurs, then the required dose is administered over 3-4 hours.

Continued administration of anti-venom: After initial bolus, depending on clinical response, 2 vials of anti-venom are infused in 100ml of fluid every 4-6 hours till all signs of envenomation disappear and clotting time is less than 10 minutes.

Reaction: Hyper sensitivity reactions may occur in 3-4% of cases usually within 10 minutes to 3 hours after starting the infusion. Reactions range from urticaria, hypotension to acute life threatening anaphylaxis. Drugs and equipment for cardiopulmonary resuscitation and for anaphylaxis should be kept ready. Serum sickness occurs after 1-4 weeks.

Treatment of anaphylaxis:
1. Give 0.01 ml/kg of 1:1000 IM adrenaline (maximum 0.5 ml). Repeat doses may be needed.
2. Volume replacement for shock-NS
3. Chlorpheniramine maleate 0.2 mg/kg/dose IV
4. Hydrocortisone 6mg/kg/dose IV
5. Oxygen and intubation facility should be ready

If patient found sensitive to the equine ASV, desensitization may be necessary by administering graded dose of anti-venom at regular and adequate intervals.

Timing of anti-venom: Best effects are observed within 4 hours of bite. But it is never too late to start ASV in viper bites as some times neutralizable venom is found even after 3 weeks.

Local wound management:
- Clean the wound
- Leave the wound open
- If swelling or tenderness is apparent, mark the proximal edge and time it so that progression can be easily monitored.
- Measure the circumference of the injured extremity at the level of oedema and record progression of oedema hourly.
- Wound debridement may be required after 3-5 days.
- Fasciotomy may be done for massive oedema due to compartment syndrome after careful assessment. It is ideal to substantiate elevated intracompartmental pressure by compartmental pressure monitoring.

Compartment syndrome:
Swelling of muscles within the tight fascial compartment of the limb may raise the intracompartmental pressure leading on to ischemic damage and neurological dysfunction. The features are severe pain, weakness of the limb, distal anesthesia, pallor and tenseness of the limb on palpation. Fasciotomy is done to relieve the intracompartmental pressure after coagulation is corrected with adequate ASV.
Other supportive measures

1. A booster of Tetanus toxoid if appropriate.
2. Broad spectrum antibiotics with anaerobic coverage
3. Blood transfusion-whole blood or plasma as needed in DIC.
4. Management of ARF if present is usually conservative.
5. Peritoneal dialysis may occasionally be necessary.

Points to remember

1. Most of the snake bites are due to non-poisonous snakes.
2. Some bites even by poisonous snakes may not cause symptoms. Reassurance, allaying of anxiety and hospitalization for 24-48 hours are all that is required in such cases.
3. The main clinical features of systemic envenomation will be bleeding manifestation (hemotoxic) due to viper bites and bulbar or respiratory paralysis (neurotoxic) due to cobra or krait bites.
4. If signs of envenomation are present the emphasis is on stabilization of ABCs, adequate antivenom administration with continuous and repeated monitoring for 3 to 4 days will be life saving.

References


NEWS AND NOTES

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ACUTE POISONING IN CHILDREN

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** Arun Kumar Manglik

Abstract: A poison has been defined as a substance (including medicines) which when introduced into or absorbed by living organisms, causes injury or death. Majority of the accidental poisoning occurs below 5 years and at home. A poisoned child may present as an emergency with or without multi-system involvement. Toxidromes (constellation of signs and symptoms seen commonly with a particular poison) help in diagnosis of unknown poisons. Management constitutes of resuscitation, evaluation, detoxification and removal phases. Special emphasis is given to the two common poisons namely organophosphates and kerosene.

Key words: Poisons, Diagnosis, Management, Organophosphate, Kerosene.

The peak incidence of accidental poisoning is in the second year of life as the child’s instincts lead him to explore everything around, as he/she acquires the ability to do so. This tendency starts decreasing after 4-5 years of age as he/she tends to be selective in choosing things for the purpose of ingestion, putting to good use his/her experience. Male children due to greater activity and children from poor socio-economic class due to less supervision and more access to common and carelessly stored chemicals and drugs, are more prone to poisoning. The second group is adolescents who fall prey to suicidal poisoning due to emotional stress. The exact incidence of acute poisoning is not known in India but it is quite common and often unreported in children. According to WHO the mortality due to poisoning ranges from 0.3%- 0.7% worldwide. There are six basic methods of exposure in children: oral, inhalational, dermal, ocular, envenomation, and transplacental. Most poisonings are acute. Poisoning should be viewed as a multiple chemical trauma. Animal bites e.g. scorpion and snake bites are excluded from the present discussion.

Approach to poisoning

I. Primary survey and stabilization

The general approach to evaluation and support of cardio-respiratory functions is the same as practised in PALS (Pediatric Advance Life Support) courses. Assessment and resuscitation are carried out simultaneously in the primary survey.

II. Secondary survey

Evaluation

i) History: It is necessary to know that only less than 10% of poisoning may be symptomatic at presentation. History of acute onset, prior normal status, child left unsupervised, drugs consumed by other family members, spillage of drugs and chemicals and emotional stress of the child (usually adolescent) must be considered. Suspicion is most important for diagnosis. A history of poison ingestion has to be searched for. Considering the general presentations of common poisoning in children, one or more of the following should alert towards possible poisoning:
• Disturbed consciousness without a cause
• Abnormal behavior of sudden onset
• Arrhythmias without a heart disease
• Respiratory distress without pneumonitis
• Shock, mostly third space loss
• Unusual odor from mouth.
• Cyanosis, without heart disease or distress
• Severe vomiting and/or diarrhea without gastroenteritis
• Seizures without a reason
• Metabolic derangements, often acidosis

Identification of the toxin, its form and dose, time lapsed after exposure, route of exposure, any underlying medical condition and home management given before arrival are important histories one should take. Appearance of symptoms chronologically and similarity of symptoms in all involved children are to be recorded.

ii) Physical examination: Some findings on clinical examination may give a clue to the identity of the poison.

a) Odour: Smell of kerosene (kerosene, petroleum products), garlic (organophosphates, arsenic) and acetone (salicylate, methanol) may be suggestive.

b) Skin and mucous membrane: Cyanosis may indicate methemoglobinemia due to dapsone or nitrates and carboxy hemoglobinemia due to carbon monoxide (CO) poisoning. Sweating and lacrimation (organophosphates, amphetamines, cocaine and barbiturates), dry and hot skin (dhatura, anticholinergics, tricyclic antidepressants) and pallor (sympathomimetics, insulin) may be the other clues.

c) Temperature: Hypothermia may suggest poisoning due to sedatives, phenothiazines, barbiturates, carbamazepine or nimesulide.

Hyperthermia may indicate anti-cholinergics, tricyclic antidepressants (TCA’s), salicylates or theophylline.

d) Pulse rate: Unexplained bradycardia (digitalis, sedatives, organophosphates, β-blockers, calcium channel blockers), tachycardia (anti-cholinergics, TCA’s) or arrhythmia (anti-cholinergics, TCA’s organophosphates, digitalis) may be suggestive of poisoning.

e) Respiration: Bradypnea or depressed respiration due to barbiturates or narcotics, tachypnea due to salicylates or amphetamines, Kussmaul breathing due to salicylates or methanol and pulmonary edema because of aspiration, narcotics and TCA’s may be useful findings.

f) Central nervous system: Seizures may occur in ingestion of organophosphates (OP’s), sympathomimetics, camphor, INH, theophylline and anti-cholinergics. Fasciculation (OP’s), nystagmus (phenytoin, carbamazepine), hypertonia and myoclonus (anticholinergics), weakness and paralysis (OP’s, botulism, heavy metals), ataxia (barbiturates, phenytoin, carbamazepine, sedatives), delirium (anticholinergics, alcohol, cocaine), and coma (anticholinergics, alcohol, OP’s, anticonvulsants, CO and salicylates) are other neurological findings. Eye signs like miosis (narcotics, OP’s, mushrooms, barbiturates), and mydriasis (anticholinergics, cocaine, TCA’s) should be looked for.

g) GIT: Vomiting and diarrhea may occur in iron, mushroom and OP poisoning.

h) BP: Hypertension may be seen in sympathomimetics, OP, TCA, cocaine andamphetamine poisoning.

iii) Toxidromes: The characteristic clinical manifestations of a specific drug or toxin or a group of drugs is called toxidrome. Table 1 lists some common toxidromes.
Anticholinergic toxidrome (Atropine, TCA, Antihistaminics, Mushrooms)  
Agitation, hallucination, coma, extrapyramidal symptoms, mydriasis, flushed warm dry skin and mucus membrane, tachycardia, arrhythmia, hypo/hypertension, paralytic ileus (Hot as a hare, Blind as a bat, Dry as a bone, Mad as a hatter).

Cholinergic toxidrome (insecticides like organophosphates, carbamates): These are discussed separately

Opiates / Narcotics / Clonidine  
Bradycardia, hypotension, pulmonary odema, bradypnea, hypothermia, coma, miosis,

Sedatives / Hypnotics / Barbiturates  
Hypothermia, bradypnea, nystagmus, ataxia, coma, hypotension, miosis / mydriasis, vesicles, bullae

Salicylates  
Vomiting, hyperpnea, fever, lethargy, coma, acidosis

Phenothiazines  
Hypotension, tachycardia, tachypnea, hypothermia, lethargy or coma, tremor, miosis, extra pyramidal symptoms (torsion of head and neck, oculogyric crisis, trismus, ataxia, back arching, tongue protrusion)

Sympathomimetics (Amphetamines, Ephedrine, Cocaine, Aminophylline)  
Tachycardia, arrhythmia, psychosis, hallucination, delirium, nausea, vomiting, abdominal pain, piloerection.

iv) Laboratory tests that can suggest poisoning

1) Hypoglycemia: Insulin, Ethanol, Acetaminophen, Salicylates
2) Hyperglycemia: Salicylates, Organophosphates, Iron.
3) Hypocalcemia: Methanol, Ethylene glycol
4) Elevated anion gap metabolic acidosis: Methyl alcohol, Ethanol, Salicylates, CO, INH
5) Urinalysis: Oxalic acid crystals (Ethylene glycol), Ketonuria (Ethanol, Salicylates).

The clinical management is based on history and clinical findings. Toxicology screening (from urine and blood) is rarely needed for immediate management. It may be useful for confirmation of poisoning, on-going management and medico-legal purposes.

Management

More than 75% of the poisoning may be managed at home as majority of poisons are non toxic (Table 2) and amount of ingestion is insufficient.

Indications for admission: The presence of disturbed consciousness, seizures, shock, severe vomiting and/or diarrhea, cyanosis, respiratory distress mandate admission, preferably in intensive care unit.
1. Resuscitation and stabilization

The classical PALS protocol (ABC) is followed with special emphasis on keeping the airway open and intubating children with loss of protective airway reflexes. Oxygen, glucose and naloxone may be used as a therapeutic trial.\(^7\)

The initial gastric aspirate is kept for chemical examination.

2. Symptomatic and supportive therapy

Specific management may be needed in the following situations: shock and electrolyte abnormalities, cardiac arrhythmia, seizures, hypothermia, pulmonary edema, rhabdomyolysis.

3. Detoxification\(^1\)

i. Removal of unabsorbed poison

Skin or eyes, if they are source of exposure need to be thoroughly washed and clothes removed. Remove the person from the site if that environment can lead onto further exposure.

Gastrointestinal detoxification\(^8\)

1. Emesis: Syrup of Ipecac: 10ml (6-12 months), 15ml (1-12yrs) followed by water. It is no more used in a hospital setting. Even for use in out of hospital setting it is not available freely. Emesis is contra indicated in young infants < 6 months of age, in corrosive and hydrocarbon ingestion and in children with altered sensorium.

2. Gastric lavage: Orogastric lavage allows direct irrigation and removal of unabsorbed poison. If gag reflex is absent the airway must be protected by endotracheal intubation. Size of tube used is 30F between 2-5yrs (biggest tube which can be safely inserted is selected). It is done with warm 0.9% saline in the dose of 15ml/kg cycles until the return lavage fluid gets clear. The lavage return should approximate the amount of fluid given to avoid fluid retention. It is useful in removing highly toxic substances e.g. organophosphate. It is contraindicated in unconscious and unstable patients and hydrocarbon and corrosive poisoning. Side effects may be fluid retention and aspiration. Gastric lavage is found to be useful if it is done within one hour of ingestion of the toxin (32% of ingested drug removed).

3. Reduction of absorption (activated charcoal)\(^9\): Activated charcoal is produced from wood, petroleum which is heated to 900°C with steam and CO\(_2\). The net result is marked increase in total absorptive surface as high as 1600m\(^2\)/gm. The dose is 1-2 gm/kg, total average 30-60 gms.

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<table>
<thead>
<tr>
<th>Category</th>
<th>Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nontoxic substances</td>
<td>Inks, mosquito repellants, incense, saccharine, calamine lotion, candle, chalk, pencil lead (graphite)</td>
</tr>
<tr>
<td>Potentially toxic substances</td>
<td>After shave lotions (ethanol), aspartame (sweetener), sindoor (lead, mercury salts), kajal (lead salt), jewellery cleaners (caustics), bleach / detergent (alkali), disinfectant (phenol, lysol) toilet cleaners (acid), varnish, nail polish cleaner (acetone), rat poisons (aluminium phosphide, zine phosphide, warfarin like compounds, arsenic or thalium), button batteries (acid/alkali/lithium), hair bleach (hydrogen peroxide)</td>
</tr>
</tbody>
</table>

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Table 2. Common household items: Are they poisonous?
given as a suspension of 20% activated charcoal in 70% sorbitol base. If only tablets are available, they can be crushed and mixed with water or fresh juice in the form of a thick soup. It acts within 30 minutes. It is useful in poisoning with many toxins like TCA, INH, digoxin, barbiturates, dapsone and anticonvulsants. If is not useful in iron and OP poisoning. Multiple doses may be required when the drug is excreted into gut after initial absorption (phenobarb, carbamazepine, theophylline, dapsone). The use of activated charcoal is considered an important method of gastrointestinal decontamination. The main constraint is that it is not freely available. It is contraindicated in intestinal perforation or ileus or when oral antidote like N-acetyl cystine is used.

4. Whole bowel irrigation (WBI)\textsuperscript{10}: It refers to mechanical cleansing of the entire bowel by large volumes of colonic lavage solution. Polyethylene glycol (EZLAX, PEGLEC), a non absorbable vehicle mixed in a balanced electrolyte solution is run at 25-40ml/kg/hr by naso-gastric or rectal route for 4-6hrs, keeping the child in a semi-fowler position until the rectal effluent gets clear. A dose of anti-emetic may help. It is indicated where activated charcoal is not useful. It is a recent armamentarium in poisoning and may be used in iron, lithium, theophylline, cocaine, heroin and sustained release drug poisoning. It is contra-indicated in ilues, intestinal obstruction, perforation and GI bleeding.


Gastric lavage, use of activated charcoal and whole bowel irrigation are the only currently recommended methods for GIT decontamination.

ii. Elimination of absorbed poison

This may be achieved by

(a) Forced Diuresis\textsuperscript{4}: may be alkaline (sod.bicarbonate) for salicylate / phenobarbitone / isoniazid poisoning or acid (ammonium chloride 1.5gm in 5%dextrose.) for amphetamine / quinidine poisoning. But it is of limited clinical value. It is not recommended because of potential volume overload and electrolyte abnormalities. Just alkalinisation of urine (urine pH maintained at 7.5 or slightly more) with IV sodabicarb may enhance urinary excretion of weak acids like salicylates and phenobarbitone. It is also indicated in TCA poisoning. Sodabicarb is administered in a dose of 2-3 ml/kg by slow infusion 4 to 6 hourly.

(b) Dialysis: indicated in severe apnea / a critical toxin level / failure of other measures / prolonged coma / delayed drug toxicity\textsuperscript{4} due to Salicylates, Lithium, INH, Methanol, Ethylene glycol and Phenobarbitone (SLIME-P). Hemodialysis is more effective.

(c) Hemoperfusion: Blood flows through charcoal or ion exchange resins. It is used in theophylline, phenobarbitone, phenytoin and carbamazepine poisoning. This may damage the platelets.

iii. Antidotes\textsuperscript{1}

Physical: Demulcents like oil and egg prevent absorption by forming a coating on mucous membrane of stomach and may be used in heavy metal poisoning. Currently they are not used. Activated charcoal is the only physical antidote recommended.

Specific antidotes: These are available only for a few poisonings (Table 3).

**Hydrocarbon poisoning**

There are four groups of hydrocarbons according to viscosity e.g. Very low (furniture polish), low (benzene, toluene), moderate (kerosene, gasoline) and high (petroleum, paraffin).

**Kerosene poisoning\textsuperscript{4}**

The toxicity of these substances is primarily due to pulmonary aspiration and not due to systemic absorption. The risk of aspiration is
<table>
<thead>
<tr>
<th>Poison</th>
<th>Antidote</th>
<th>Indication</th>
<th>Dose of antidote</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen (Paracetamol)</td>
<td>N-Acetyl cysteine (Mucomyst)</td>
<td>Serum level &gt; 200mcg/ml within 4hrs.</td>
<td>140mg/kg PO, then 70mg/kg every 4hrs (17 doses in 3-4days)</td>
</tr>
<tr>
<td>Anticoagulant (Warfarin)</td>
<td>Phytomenadione (VitK&lt;sub&gt;1&lt;/sub&gt;)</td>
<td>Bleeding</td>
<td>5-10mg IM / IV</td>
</tr>
<tr>
<td>Beta-blocker, Calcium channel blockers (CCB)</td>
<td>Glucagon</td>
<td>Bradycardia</td>
<td>0.05-0.1 mg/kg bolus, then 0.05mg/kg/hr Titration accordingly 0.1 to 2.0 mcg/kg/min – Isoprotenenol 0.1 to 1 mcg/kg/min – Epinephrine 0.5 ml/kg/bolus-calcium for CCB poisoning</td>
</tr>
<tr>
<td>Calcium blockers (CCB)</td>
<td>Isoproterenol / Epinephrine, IV calcium</td>
<td>Bradycardia</td>
<td>0.05-0.1 mg/kg bolus, then 0.05mg/kg/hr Titration accordingly 0.1 to 2.0 mcg/kg/min – Isoprotenenol 0.1 to 1 mcg/kg/min – Epinephrine 0.5 ml/kg/bolus-calcium for CCB poisoning</td>
</tr>
<tr>
<td>Benzodiazepine</td>
<td>Flumazenil</td>
<td>Sedation</td>
<td>0.2mg over 30 sec. repeat q 1min (1 mg max.)</td>
</tr>
<tr>
<td>Ethylene Glycol Or Methanol</td>
<td>Ethanol</td>
<td>Serum level &gt; 20mg/dl; osmolar gap with metabolic acidosis</td>
<td>750mg/kg as 10% Ethanol in 5% GDW (loading) 80-150mg/kg/hr(maintenance) to maintain blood ethanol level 100-150 mg/dl</td>
</tr>
<tr>
<td></td>
<td>Fomepizole</td>
<td>It inhibits alcohol dehydrogenase</td>
<td>15mg/kg (load), 10mg/kg q12hr 4doses for methanol and until level &lt; 20 mg/dl for ethylene glycol.</td>
</tr>
<tr>
<td>Heavy metals</td>
<td>Calcium EDTA BAL Penicillamine DMSA</td>
<td>Pb/Mn/Ni/Hg/Cu Pb/As / Cu /Au / Hg Cu / Pb /Au / Hg Pb / Hg / As</td>
<td>EDTA – 1-1.5g/m&lt;sup&gt;2&lt;/sup&gt;/day IV(2dd) x 5 days BAL - 12-24mg/kg/day deep IM (6dd) x 3 days Penicillamine - 20-40mg/kg/day PO DMSA - 10mg/kg/dose q8hrly PO for 5days, q 12 hr 14 days</td>
</tr>
<tr>
<td>Isoniazid</td>
<td>Pyridoxine</td>
<td>Seizures</td>
<td>Same dose as INH ingested or if amount not known, 25-50 mg/kg IV every 30 minutes till seizures controlled</td>
</tr>
<tr>
<td>Iron salts</td>
<td>Desferoxamine</td>
<td>Serum level&gt; 350mcg/ml /</td>
<td>15mg/kg/hr IV infusion or 50mg/kg/IM repeat every 4-8 hr until urine color normal (no more vin rose color), serum iron and clinical condition normal</td>
</tr>
<tr>
<td>Methemoglobinemia (Dapsone / nitrites/ nitrates / sulphonamides)</td>
<td>Methylene Blue</td>
<td>Symptomatic / level &gt; 30-40%</td>
<td>1-2 mg/kg of 1% soln. in 5-10min repeat q 30-60min. Maximum total dose 7 mg/kg</td>
</tr>
<tr>
<td>Narcotics</td>
<td>Naloxone</td>
<td>Symptomatic</td>
<td>0.01mg/kg /dose; if no response repeat 0.1 mg/kg every five minutes till response.</td>
</tr>
<tr>
<td>Organophosphates, carbamates</td>
<td>Atropine</td>
<td>Muscarinic effects</td>
<td>0.05 mg/kg IV; repeat every 5-10 mins to reverse muscarinic effects; maintain atropinisation for 24 to 48 hours</td>
</tr>
<tr>
<td>Organophosphates</td>
<td>Pralidoxime</td>
<td>Nicotinic effects</td>
<td>25-50 mg/kg in NS over 30 minutures; repeat after 30-60 minutes; then q 12 hr if symptoms persist</td>
</tr>
<tr>
<td>Phenothiazine</td>
<td>Diphenhydramine</td>
<td>Oculogyric crisis</td>
<td>5mg/kg/day 3 dd (max. 300mg/ day) oral or IV</td>
</tr>
<tr>
<td>Sulphonylureas (Antidiabetics)</td>
<td>Octreotide</td>
<td>Hypoglycemia</td>
<td>25 mcg SC 8&lt;sup&gt;th&lt;/sup&gt; hourly as required</td>
</tr>
</tbody>
</table>

As = Arsenic, Au = Gold, Cu = Copper, Hg =- Mercury, Mn = Magnese, Ni = Nickel, Pb = Lead, dd = divided doses
higher with moderate viscosity, highly volatile compounds (gasoline, kerosene) than with low viscosity less volatile ones (petroleum, paraffin).

Clinical features

1) Respiratory: Cough, respiratory distress, fever, dyspnea, wheeze, cyanosis and rarely hemoptysis. This occurs within 6-24hrs of aspiraton. One gets the smell of kerosene from the child’s breath.

2) CNS: Somnolence, depression which may be secondary to hypoxia or due to additives (blue pigment in commercial kerosene!).

3) Gastrointestinal: Nausea, vomiting, abdominal pain and diarrhea.

4) Hematological: Hemolysis, hemoglobinuria. It occurs rarely with gasoline ingestion due to red cell damage.

Laboratory findings: Chest X-ray within 24 hours may confirm chemical pneumonia. Complete blood count and ABG are rarely needed.

Treatment: Is usually supportive and includes resuscitation and stabilization. Emesis, activated charcoal and lavage are contraindicated for kerosene poisoning. The child with respiratory distress requires oxygen, IV fluids and close monitoring. Respiratory failure is a rare complication and requires ventilatory support. The outcome is usually good.

Organophosphate poisoning\(^{11}\) (OP):

OP’s include malathion, parathion and fenitrothion (Tik 20). Absorption occurs through skin, eyes, inhalation and ingestion. Stronger concentrations (40-50%) are present in industrial formulations than in domestic (1-2%) ones.

OP’s act by phosphorylating the active or esteric site of acetyl cholinesterase leading to irreversible inhibition of the enzyme and excessive accumulation of acetylcholine. Carbamates like propoxur (Baygon) cause reversible inhibition of cholinesterase and cause less CNS effects. The clinical signs are due to muscarinic, nicotinic and central nervous system effects.

a) Muscarinic (post-ganglionic parasympathomimetic): D(diarrhea), U(urinary incontinence), M(miosis), B(bradycardia), B(bronchorrea), E(emesis), L(lacrimation), S(salivation) – “DUMBBELS”

b) Nicotinic (sympathetic and skeletal muscle): Muscle twitching, fasciculation, paralysis, tachycardia, hypertension (most dangerous), hyperglycemia

c) Central nervous system effects: Confusion, slurred speech, ataxia, seizures, periodic breathing, coma etc.

RBC cholinesterase < 50% of value is diagnostic (rarely done)

Treatment

Stabilization

Rapid cardiopulmonary assessment and support is the priority. Treat seizures, if present.

Decontamination

Removal of contaminated clothes and cleaning the skin and eyes (by irrigation) are carried out wherever appropriate. Gastric lavage is done as soon as possible protecting the airway.

Specific Treatment

Atropine (in doses 5-10 times greater than usual dose): It blocks muscarinic action only. Initial dose: 1-2mg IV (>12yrs) and 0.05mg/kg IV every 5-10min (<12yrs). It may be doubled every 5 min until response. End point is drying of mucosa and and respiratory secretions (not pupil size or heart rate!). Maintenance dose is required for 24-48 hrs depending on symptoms to prevent rebound phenomenon.
Pralidoxime : Counteracts nicotinic symptoms only and hence should be added to atropine. It has to be given within 48 hrs (preferably within 12 hours) as it has poor action against aged acetyl cholinesterase. Dose is 25-50mg/kg in N.saline IV in 30 mins. It may be repeated after 30-60 minutes and then 12-24 hrly if symptoms reappear. Pralidoxime does not have effect on CNS manifestations. Atropine is used for CNS effects of OP. Pralidoxime is not indicated in known carbamate poisoning.

Drugs contraindicated in OP poisoning are morphine, phenobarbitone, frusemide, and theophylline.

Sequelae :
a) Prolonged memory loss, peripheral neuropathy, personality changes
b) Intermediate syndrome: Respiratory failure, bulbar or nuchal or proximal limb weakness 24-96 hrs after resolution
c) Delayed neurotoxicity or neuropathy : Sensorimotor polyneuropathy occurring 1-3 weeks after poisoning. Flaccid paresis and wasting may ensue after 12-15 months.

Medicolegal issues\textsuperscript{13}: Doctor, Poisoning and Law:
a) A doctor should give information about his patient to police or legal authority especially in
i) severe toxicity or death ii) strong suspicion of homicidal poisoning (Sec 39 IPC).
b) He should supply all records to the police or magistrate if asked for under Sec. 175 Cr. IPC. Noncompliance of such obligation (Sec. 202 IPC.) or deliberate concealing of facts or lying (Sec. 193 IPC) will make him liable to prosecution.
c) He should always suspect homicidal causes in each case (though extremely rare in childhood in
our country) and seek a second opinion if needed. He should keep all the records for future references.\textsuperscript{6}

Prevention\textsuperscript{1}

Childhood poisoning in our country is mostly accidental. This makes this hazard preventable by good social education. A large proportion of accidental poisoning is trivial. A few hours of hospital observation is only needed. These children can be sent home after providing anticipatory poison prevention guidelines.

Key Messages:

1) Strongly suspect poisoning in acute but unexplained symptoms suddenly appearing in an otherwise healthy child.

2) Clinical features are the key to diagnosis as confirmatory tests are usually not available in most of the cases.

3) Stabilization of vital parameters in the initial phase, evaluation of the poison (identification and severity assessment) and detoxification are the key points in management.

4) Activated charcoal is the mainstay of non-specific gastrointestinal decontamination. Whole bowel irrigation is a new armamentarium. Specific antidotes are available for a few poisons only. Hence supportive therapy remains the mainstay in management.

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**NEWS AND NOTES**

November 11-12, 2006.
Pre-congress Live Operative Workshop on Anorectal Malformation
(led by Prof Alberto Pena), AIIMS, New Delhi.

November 12, 2006,
Pediatric intensive nursing care, including neonatal resuscitation and safe transport
Conference Hall, AIIMS, New Delhi

20th CONGRESS OF ASIAN ASSOCIATION OF PEDIATRIC SURGEONS with Executive Meeting of WOFAPS,
Taj Hotel and Convention center, New Delhi.

November 16-17, 2006
Post Congress Live Operative Workshop on Pediatric Urology ( VUR, Intersex and Hypospadias)
( Prof Prem Puri, Prof Snodgrass, Mr A.Bracka, Prof. John Hutson, Prof Asopa), AIIMS,
New Delhi.
MEDICO - LEGAL ISSUES IN EMERGENCY ROOM

* Mahesh Baldwa

Abstract: Enactment of Consumer Act along with inclusion of medical professional in its ambit has put cases of alleged medical negligence under fast tract of judicial remedy. A number of tertiary care centers in the form of intensive neonatal care units and intensive pediatric care units with huge investment in infrastructure have come up. State of art infrastructure costs a fortune and escalates cost of quality care in pediatric treatment. Heavy cost of emergency and critical care treatment has made this emerging pediatric subspecialty, a hotspot of litigations. In today’s scenario doctors are health risk managers for their critically sick patients who need vigilant monitoring and timely treatment to avert further crisis and complications that are common and foreseeable. So, the corollary is that doctors are expected to chart the course of the health of their critically ill patients with minimal health hazards by use of state of art and costly monitoring equipment. Any action or inaction (act of omission or commission) of the doctor that accelerates or increases the health risks of a critically ill patient may result in an allegation of breach of duty of doctor. Establishment of consumer courts has put the cases of medical negligence on fast tract remedy. There is no limit for asking of compensation for alleged medical negligence. The amount of money quoted is mind-boggling. Patients may sue a doctor for compensation by asking usually lakhs of rupees and some times in crores. Medical indemnity insurance policy is the only way out to practice emergency and critical care pediatrics peacefully in such an odious scenario by which these risks of litigations could be managed and any claim if arises could be paid.

Key words: Emergency room care, duty of care in emergency, omission of duty, death, disability

Introduction

The monitoring facilities and advancement in maintaining vital parameters in a critically ill child till the basic pathology is taken care of by appropriate treatment, are significant advances in the field of pediatrics. This has ushered in a new era of critical care and emergency in pediatrics.

In today’s scenario doctors are health risk managers of their critically ill patients. Any action or inaction (act of omission or commission) of the doctor that accelerates or increases the health risks of a critically ill patient may result in an allegation of breach of duty of doctor. Establishment of consumer courts has put the cases of medical negligence on fast tract remedy. There is no limit for asking of compensation for alleged medical negligence. The amount of money quoted is mind-boggling. Patients may sue a doctor for compensation by asking usually lakhs of rupees and some times in crores. Medical indemnity insurance policy is the only way out to practice emergency and critical care pediatrics peacefully in such an odious scenario by which these risks of litigations could be managed and any claim if arises could be paid.

Likely situations of medico-legal importance

There are situations where it is mandatory to inform the law enforcers and/or legal authorities (usually it is local police station) regarding patients seen in emergency room. Because doctors duty is to treat the patient and duty of police is to find out whether any crime was committed on victim/patient for making him/her suffer from problems listed below:

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A) Tetanus, gas gangrene, significant burns, head injuries, significant violence needing inpatient treatment, motor vehicular and other accidental fractures, accidental falls needing inpatient treatment, attempted suicides, attempted poisoning, attempted homicide, human or animal or snakebite, rape, minor’s pregnancy and MTP, battered baby. In case of death doctors should insist for post mortem in all of the above situations.

B) In case of attempted poisoning or poisoning, doctor is duty bound to collect a specimen of stomach wash (usually 100ml or more in a clean glass bottle), blood samples in EDTA and plain bulbs (usually 2ml each), as applicable and feasible and hand it over to the police with proper labeling of name, sex, age, time of collection, brief history and treatment given. In case of death due to poisoning always insist for post mortem.

C) There are situations apart from this that may require information to police or post mortem depending upon facts and circumstances of a case. Doctor should continue to treat with meticulous history recording, examination, investigations needed and treatment as per reasonable norms of medical practice.

1. Indoor admitted child falling from cot or in the bathroom and getting significant injury and needing inpatient treatment.

2. Operation table deaths or post operative critically ill child dying

3. Child developing gas gangrene and gangrene due to infused fluids or intravenous lines

4. Almost instant intra muscular nerve palsy

5. Deaths resulting from anaphylaxis or Steven Johnson syndrome due to a drug

6. Post procedure death like for example after lumbar puncture, liver biopsy and other biopsies.

7. Deaths due to bleeding and disseminated intra vascular coagulation, reason not obvious.


What is the procedure if a child is brought dead by a mob (or lot of relatives):

1. If there is a mob (or lot of relatives) immediately divide doctor’s working team to two parts. One team shall explain to the parents that child is dead yet if you permit we may give some treatment but situation may not change. Second team tackles the mob that wants the child to be treated anyhow. Tell them he is already dead but the team of doctors is doing their best.

2. Declaration of death in a child brought dead by mob should be essentially preceded by rapid assessment and an attempt at resuscitation after talking to the groups as mentioned above. This will buy time and wisdom for medical team to declare death at the terms and conditions desired by medical team rather than be swept away with unruly behaviour of mob.

3. When a child is brought by mob, who are not amenable for explanation then one should surely inform police for protection and then only declare final death. The situation will be considered all the more serious, if the child is brought dead. Avoid loose talk which may spark problems and may lead to physical abuse of medical team.

What is the procedure if the child is brought dead by parents:

1. Declare death, inform police and ask for postmortem.

2. If mob gathers later on, then inform police, hold talk with mob leader, be empathetic, sympathetic, humanistic and soft-spoken. Let some one senior handle the situation.

What to do if child is brought dead who was under your treatment for a serious disease?
In such a case one may have to issue death certificate. In United kingdom (U.K. rule of 21) if patient is under treatment for 21 days then one may have to issue death certificate.

How should we transport the sick and serious child?

1. Doctor and nurse team should accompany the patient.  
2. Ambulance should be equipped with adequate doses of emergency medicines, injections, intravenous fluids and oxygen.  
3. Monitoring equipments like stethoscope, blood pressure instrument, cardiac monitor machine with preferably a defibrillator and a ventilator.

Standard of medical care in emergency room should be high because emergency room care claims giving state of art services, as mentioned:  
1. Duty of care in emergency room (which means actively avoiding all kinds of dangers i.e. health risks from all sources i.e. from disease, drugs and surgery, all the time) to your patients by continuous monitoring of all relevant vital parameters and investigations.  
2. Law requires proportionate degree of care in emergency room. Higher the risk undertaken, higher is the standard demanded by law in caring for critically ill.  
3. If there is any lack of care, on the part of medical practitioner in monitoring or treatment of a critically ill, doctor’s actions which cause acceleration of disease process leading to death or disability is actionable under law.  
4. Under law for actionable negligence, such an acceleration should be caused by breach of duty of a doctor (lack of due care of critically ill and lack of caution in monitoring critically or delaying or omitting to give treatment to critically ill) which should result in actual (proved) physical or mental damage to patient.

5. There should be close nexus between such acceleration of disease process caused by negligence of doctor and not because of inherent nature of disease. Such acceleration should cause disability or death due to breech of duty (lack of due care and caution) resulting in damage.  
6. Legally if there is no resultant damage due to lack of care, then no compensation can be given to patient.

Consent, Dissent, Assent, Counselling, Forewarning  
1. In emergency rooms standards for informed consent are lower than usual cold situations. In dire emergency, courts waive off consent in favour of giving lifesaving treatment, even though nature of treatment may amount to adventure. In a case of a road traffic accident, victim’s vitals were stabilized by giving emergency treatment before shifting to higher center, where one limb had to be amputated because of delay in referral; court did not hold doctor negligent in causing delay in referring because stabilization of vitals was crucial before transfer of patient otherwise patient would have been dead during transit. In another case of vehicular accident, a reasonable delay in preparing for operation and arranging for 19 bottles of blood was permitted by court even though patient died postoperatively.  
2. Some times in emergency, omission to perform operation for want of consent may amount to negligence. An emergency appendicectomy was not done, for want of written consent or dissent from patient. The doctor was held liable on this as patient died of burst appendix. Remember written dissent is more important than consent for invasive procedure, surgery, investigation, transfer and referral in emergency situations.
Prevention or detection of complications, monitoring, treating or transferring serious patients

Monitoring and record keeping

1. Monitoring serious patients by keeping record and using available gadgets and investigations or referral by providing ambulance to transfer\textsuperscript{18,26}. Basic minimum for monitoring is recording pulse, respiration, temperature, blood pressure, intake and output chart.

2. Remember proper record is a valid defence in medical negligence case as the law asks for a show of care rather than cure\textsuperscript{27}.

Critically ill patients where “known complications” which cannot be prevented is present

A boy was bitten by a cat and was given anti-rabies vaccine. He developed neuro-paralytic reaction\textsuperscript{28}, for which he was hospitalized and died in ICU. In this case there is no negligence as standard textbooks and WHO report of 1984 mention neuro-paralytic reactions as a well known complication of ARV. Here a proper ICU treatment was given with care.

Cases related to anaphylaxis\textsuperscript{29}

- If a doctor neither does penicillin test dose nor have emergency medicines for treating anaphylaxis and fails to treat complications resulting in the patient, the doctor is held negligent.

Cases related to improper drug administration resulting in complication

a) A practitioner inadverdently administered a medication intra arterially instead of intravenously. The patient developed gangrene of the thumb, index and middle fingers. The local court held the doctor as negligent and the patient was entitled to receive adequate compensation\textsuperscript{30}.

b) Analgin was given intravenously to a 16 year old boy resulting in gangrene of the leg resulting in amputation; the doctor had to pay a compensation\textsuperscript{31}.

c) Emergency drugs such as pentazocine, diazepam and atropine were given by an ayurvedic physician to a patient with abdominal pain; the patient developed severe pain in fingers and subsequent gangrene, requiring amputation of three fingers. The ayurvedic physician not qualified to administer allopathic drugs had to pay compensation.

d) In another case though the patient developed gangrene of right hand requiring amputation following IV pentazocine and promethazine given in the right arm, the doctor was not considered negligent\textsuperscript{32}.

Emergency blood transfusion:

Some times emergency blood transfusion of a wrong blood group may cause mismatch\textsuperscript{33} transfusion reactions; rarely AB positive child may be given B positive blood\textsuperscript{34}. Blood transfusion in emergency rooms have been the source of transmitting hepatitis B and HIV infections\textsuperscript{35,36}. It is better to be safe than be sorry later by following proper blood checking norms.

Summary: This article is intended to provide the emergency care provider and those who deal with critically ill patients with the required knowledge and wisdom regarding the relevant laws applicable to practicing critical care. It will also help to prevent, solve and understand the day-to-day legal problems related to emergency care. All of us are aware and have experienced that ignorance breeds and feeds uncertainty. Uncertainty breeds and feeds unfounded fears. We also know that unfounded fears usually never become true, but only make life stressful and unlivable. In the light of basic legal knowledge, let us dispel these unfounded legal fears and do the right deeds in the
right direction. Let us not give up and practice defensive medicine for fear of legal wrangles.

**Points to remember**

1. **Do not forget to take medical indemnity policy and keep renewing yearly and then only keep on handling emergencies.**

2. **Document parameters monitored with respective time and date on indoor case paper.**

3. **Do not forget to xerox the thermal paper printout readings of ECG, ABG etc. as the readings fade by the time they are viewed in court of law.**

4. **Do not forget to inform local police station regarding cases which necessitate this procedure.**

5. **Keep ready formats of consent, dissent, assent, counseling, forewarning and get it signed, dated and timed.**

6. **If you need to transport sick and serious patients in ambulance do in well-equipped ambulance with qualified doctor and nurse to handle emergency situations.**

7. **In case of death in emergency room, declare death only when you have completed indoor case records and talked empathetically to relatives.**

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BASICS IN PEDIATRIC PULMONARY FUNCTION TEST

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Abstract: Pediatricians utilise a wide variety of laboratory tests including radiography in day-to-day practice to facilitate diagnosis and treatment of pulmonary disease. However pulmonary function tests (PFT) are not commonly used despite the high incidence of asthma and chronic cough seen in office practice. The underutilization is probably due to decreased awareness and lack of understanding of PFT. This article discusses the basic principles involved in the interpretation of PFT.

Keywords: Spirometry, Peak flowmetry, Pulmonary function test, PEFR, Children

Pulmonary function testing is one of the basic tools for evaluating a patient’s respiratory status. A basic knowledge of the normal lung volumes and capacities is essential for proper understanding of the pulmonary function tests (Fig.1). In day-to-day practice, PFT includes spirometry and peak expiratory flowmetry (PEFM). These are usually performed in children above 5-6 years of age.

Spirometry

The instruments used for spirometry tests are called “spirometers”. The tracing generated by the spirometer is called a spirogram. Spirometry is the timed measurement of dynamic lung volumes during forced expiration and inspiration. Four indices are mainly used to interpret spirometry^{1,2}.

a) Forced vital capacity (FVC): This is the total amount of air expelled after a maximal inspiration. The normal value is 100 ± 20%.

b) Forced expiratory volume (FEV₁): This is the amount of air that can be expelled in one second after a maximal inspiration i.e. the fraction of forced vital capacity expired during the first second. The normal value is 100 ± 20%.

c) Forced expiratory ratio: This is the ratio of FEV₁ with FVC (FEV₁/FVC). The normal value is more than 80%.

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Fig 1: Normal lung volumes and capacities

IRV- inspiratory reserve volume, TV- tidal volume, ERV- expiratory reserve volume, RV- residual volume, TLC- total lung capacity, FRC- functional residual capacity, IC- inspiratory capacity, VC- vital capacity
d) Forced expiratory flow (FEF\textsubscript{25%-75%}): This is the percentage of FVC calculated between the first 25% and the last 25% of FVC and is also called maximal mid expiratory flow (MMF 25-75%). It is useful to evaluate small airways. The normal value is 100 ± 35%.

Spirometry is reported as both absolute values and as predicted percentage of normal. Predicted spirometric values depend on various factors like race, sex, age and height. There is no single standard reference value and the predicted values has to be in accordance to the target population.

The spirometer cannot measure static lung volumes and capacities like residual volume (RV), functional residual capacity (FRC) or total lung capacity (TLC), in which airflow does not play a role. These parameters can be measured using gas dilution and body plethysmography techniques, but they are not required in routine assessment of common lung problems.

**Spirometry Test**

The success of spirometry is in producing an acceptable with/without reproducible FVC curve. With adequate training and encouragement children above 5 to 6 years of age can produce an acceptable FVC curve. The environment for testing should be child friendly. Before attempting spirometry, it is important to make the child and attenders familiar with the laboratory, instruments and persons.

Criteria for acceptability are: a) There should be no artifact due to cough, glottic closure or equipment leak, b) The start should be without hesitation and c) There should be at least six seconds of smooth continuous exhalation and/or a plateau in the volume time curve of at least one second, or a reasonable duration of exhalation with a plateau\textsuperscript{3}.

Criteria for reproducibility are\textsuperscript{3}: From the acceptable spiograms the difference between the 2 largest FVC and FEV\textsubscript{1} should not be more than 0.2L.

**Procedure\textsuperscript{2}**

- The child’s torso and head should be erect during the procedure. The study may be done either in sitting or standing position. Occasionally few may experience syncope or dizziness while performing the forced expiratory maneuver. Thus, sitting position is preferred.
- The mouthpiece is held in between the lips to form an airtight seal. The use of nose clip for all spirometric maneuvers is strongly encouraged.
- The child should take a slow, full and deep inspiration followed by a brief hold of the breath and then a sustained forceful expiration (with maximum effort) without coughing or quitting during the procedure. The child should be taught and encouraged during expiration to achieve a complete forced vital capacity \textit{i.e.}, “blowing” as long as possible or at least 6 seconds. FVC maneuver in spirometry is similar to blowing of a balloon. Application of nose clips may yield better results.

The child is allowed at least three but not more than eight attempts to meet acceptability and reproducibility criteria, as fatigue might play a role in the results. An acceptable spirogram should not be discarded even if it is not reproducible, as FEV\textsubscript{1} from such spirogram may be valid and the volume expired may be an estimate of the true FVC. However FEV\textsubscript{1}/FVC and FEF\textsubscript{25-75%} may be overestimated\textsuperscript{3}. The highest FEV\textsubscript{1} and FVC can be reported from 2 different spiograms. But FEF\textsubscript{25-75%} and instantaneous expiratory flow rates, such as FEF\textsubscript{max} (i.e. PEFR) should be obtained from the same best acceptable spirogram. The best accepted curve is the one that has the largest sum of FEV\textsubscript{1} and FVC. Test
acceptability is best determined by examining the flow volume loop and volume time curve. Variable effort, cough and early glottic closure can be seen on the graphs but may not be apparent by simply looking at values for FEV\textsubscript{1} and FVC. Reproducibility criteria helps to ensure that adequate effort has been made as maximal patient effort leads to reproducible spiromgrams. The same person, preferably a medical personal should perform and interpret spirometry, as the spirogram is effort dependent.

Types of spirogram

The spirogram produced depends on the machine used but the measurement technique, principle and interpretation of results remains the same. There are 3 types of spirograms\textsuperscript{2}:

a) The standard spirometric curve is a volume time curve i.e. plot of volume Vs time. This records only the expiratory phase. (Fig2)

b) A flow volume curve plots flow Vs volume. This also records only the expiratory phase.

c) A flow volume loop includes the forced expiratory and inspiratory effort. At the beginning of the procedure the subject exhales as forcibly as possible, the result is an FVC curve. At the end of FVC maneuver the subject inhales, as rapidly and forcefully as possible, the result is an inspiratory curve. The expiratory and inspiratory curves together describe a flow volume loop (Fig 3).

Most of the commercially available spirometers have both volume time curve and flow volume loop tracings.

Interpretation of spirometry

Spirometry for lung is akin to ECG for heart. In most cases it does not give us the etiological diagnosis but give us the physiological status with clues for diagnosis. Ideally spirometry should be interpreted with the flow volume loop, volume

![Fig 2: Volume time curve](image)

![Fig 3: Flow volume loop](image)
time curves and absolute values for flows and volumes. As clinicians we must be able to identify an abnormal spirogram and should be able to diagnose whether it is obstructive or probable restrictive pattern depending on the values and shape. The primary abnormality detected by spirometry is airway obstruction while restrictive lung diseases cannot be diagnosed by spirometry alone. To diagnose restrictive lung disease the lung volumes (including RV/ FRC/ TLC) have to be confirmed. This could be done by closed circuit helium dilution/ gas dilution techniques, open circuit nitrogen washout method, body plethysmography and radiographic planimetry. In obstructive pattern it is necessary to differentiate extrathoracic (upper airway) and intrathoracic (lower airway) obstruction.

**Interpretation of spirometry depending on spirogram values**: The spirometry values of the child are compared against the nomograms available for the population. In restrictive lung disease the child is not able to inhale the expected amount of air as compared to his or her peers, but is able to exhale the inhaled air normally. In obstructive lung disease (e.g. asthma) the child is not able to breath out as expected like his or her peers (i.e. air trapping).

Disproportionate reduction in the FEV₁ as compared to the FVC and therefore reduced FEV₁-to-FVC ratio (due to slow rate of airflow) is the hallmark of obstructive lung diseases. Reduction in the FVC, FEV₁ that is a fraction of the FVC, is also proportionally reduced, with FEV₁/FVC being normal (proportionally reduced) or elevated (as there is no impedence in expiration) in restrictive lung diseases.

In early or mild asthmatics because of air trapping the TLC will increase and the FEV₁ and FEV₁/FVC ratio is deceptively normal. In such conditions, the measurement of FEF₂₅-₇₅% may be diagnostic. If the child quits before the end of the FVC manoeuvre, the FVC is underestimated and in the early stages of obstruction, the FEV₁/FVC ratio may be normal resulting in a wrong interpretation as restrictive lung disease instead of obstructive disease.

**Interpretation of spirometry depending on spirogram shape**:

In restrictive lung disease, all lung volumes and flows (inspiratory and expiratory) are reduced resulting in a small loop without any change in the shape i.e shape maintained but size is altered. In obstructive lung disease, the shape of the loop is altered. In intrathoracic (lower airway) obstruction the shape of the expiratory limb (upper loop) is altered. Whereas in extrathoracic (upper airway) obstruction the shape of the inspiratory limb (lower loop) is altered without changing the shape of the upper loop. For easy interpretation of spirometry results the algorithm in fig 4 may be followed⁴. The overall validity of spirometry depends on equipment calibration, patient’s cooperation and effort to produce an acceptable and reproducible spirogram.

**Peak Expiratory Flow Metry (PEFM)**

Peak flow meter is a handy, portable device used for recording peak expiratory flow rate (PEFR). The Peak flow value is measured in liters per minute. In children with asthma, peak flow can be monitored and recorded at home. It may be useful in the diagnosis and monitoring of asthma patients on follow-up.

**Technique for measuring a peak flow**

- The pointer on the meter is moved to zero
- The meter is held horizontally, the child’s torso and head should be erect during the procedure. This could be done in either sitting or standing position
Fig 4. Algorithm for interpretation of spirogram

**Step 1:**
- **Time Volume Curve**
  - Acceptable → No → Repeat the procedure
  - Yes

**Step 2a:**
- **FEV₁**
  - >80% → Normal (if FEV₁/FVC >80% & FEF₂₅-₇₅ >65%)
  - <80% → Lower 

**Step 2b:**
- **FEV₁/FVC**
  - >80% → s/o Restrictive lung disease
  - <80% → Obstruction

**Step 2c:**
- **FVC**
  - >80% (pure obstruction)
  - <80% (Obstruction and Restriction)

**Step 3:**
- **Flow volume loop** → Look for e/o upper or lower airway obstruction

- **Upper / Extrathoracic Obst:**
  - Lower limb (inspiratory) loop involved
- **Lower / Intrathoracic Obst:**
  - Upper limb (expiratory) loop involved
The fingers should be kept away from the vent holes and marker

- Maximum air is inspired with the mouth wide open
- The mouthpiece is placed in the mouth with lips pursed around it
- Blow out as hard and fast as possible with a short, sharp blast (like blowing a candle)
- The pointer is moved to zero after each recording
- The recording is done thrice waiting at least 2 seconds in between each recording
- The best of the three values is recorded and not the average.
- A record of the value obtained in the morning and evening is maintained

Peak expiratory flow rate (PEFR) correlates with FEF (max) in spirometry. The PEFR reading is correlated with the predicted values from standard normogram based on height of the child. Ideally this again has to be population based and in routine office practice a formula: \( \{(Ht \text{ in cms} - 100) \times 5\} + 100 = \text{PEFR } \text{Lt/min} \), can be used.  

PEFR in office practice

In a child with suspected asthma PEFM may help to confirm the diagnosis. PEFR values <80% of predicted values (by nomogram or the best personal recording of the patient during a symptom free period, whichever value is higher) are considered abnormal. Further confirmation is by doing a bronchodilator reversibility test i.e. either 3-4 puffs of short acting beta-2- agonist via MDI or a single dose beta-2- agonist via nebuliser is given and PEFR is recorded every 5 minutes for the next 15 minutes. If there is 15% improvement in the recording, it confirms reversible bronchospasm.

Subjects who use inhaled short-acting bronchodilators should be tested at least 4 to 6 hours after the last use of their inhaled bronchodilator to allow proper assessment of acute bronchodilator response (long-acting inhaled bronchodilators may need to be withheld for a more extended period) either for spirometry or PEFR. The length of the interval between administration of the bronchodilator and postbronchodilator testing varies, however a interval period of 15 minutes is being practiced.

If the PEFR values are normal in a suspected case of asthma, the patient is asked to record PEFR at least twice a day (8am and 8pm preferably) to check for diurnal variation. Normally the PEFR values are less in the mornings as compared to the evening values and the variation is normally less than 10%. If the diurnal variation is more than 20%, the possibility of asthma is considered.

Exercise test: The initial PEFR is recorded and the patient is asked to exercise vigorously for 5 to 6 minutes (e.g. by running up and down stairs). The PEFR is recorded every five minutes for the next 15 minutes post-exercise. In normal individuals there is no change in PEFR reading; however in asthmatics there will be a fall in PEFR by at least 15% at the end of the test. This fall in PEFR will be improved after inhaled bronchodilator. This test is especially useful for patients with exercise-induced asthma. A fall in PEFR value is usually said to precede an acute exacerbation of asthma. This can be used to predict an impending exacerbation in children who perceive symptoms of asthma poorly and can be helpful in initiating early reliever therapy to prevent acute exacerbation.

PEFR zones (Tri colour cards)

These are colour-coded zones that are based on the traffic light concept. They can be used as home monitor to check the well being of airways:
a) Red zone: “Danger” i.e. PEFR <50% of child’s best. This is a medical emergency and warrants hospitalization.

b) Yellow zone: “Alert” i.e. PEFR 50-80% of child’s best. The advice is to eliminate triggers and use reliever (bronchodilator) medicine.

c) Green zone: “Safe” i.e. PEFR >80% of child’s best. The advice is to avoid triggers.

In all 3 zones if the child is on preventor medication, it should be continued.

PEFR recording is an objective assessment in asthma like temperature recording in fever. PEFR monitoring is a useful tool in home management of asthma. In acute exacerbation of asthma, PEFR monitoring may worsen symptoms due to collapse of peripheral airway during forced expiration. Cough during recording can depict an abnormally high value of PEFR despite severe airway obstruction.

**Conclusion**

Spirometry and PEFM recording are both effort dependent. With proper calibration of the equipment, correct technique and interpretation of results, they are valuable tools in the assessment of lung diseases.

**References**


**NEWS AND NOTES**

**JOHAR, AUGUST 18-20, 2006**

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NUTRITION, HEALTH AND WELL-BEING OF CHILDREN

* Mehbaran Singh

Abstract: Health and well being of children depends upon an interaction between their genetic potential for physical growth and mental development and intake of optimal nutrition, exposure to safe and stimulating environment and freedom from common day-to-day infections. The last 3 months of fetal life, first 3 years of postnatal life (pre-school years) and adolescence are most critical and vulnerable to the adverse effects of malnutrition. Due to control of florid or severe cases of malnutrition, the child survival has improved but the quality of life and human resource development has not improved due to wide-spread prevalence of subclinical or biochemical deficiencies of micronutrients.

Keywords: micro nutrients, child health

Mothers are creators of progeny and their health and wellbeing is closely interlinked with the health of their children. Therefore, a life-cycle approach should be followed to provide optimal nutrition and health care to girl children from infancy through childhood, adolescence, pregnancy and lactation.

Breast feeding is the best feeding for every baby and must be promoted and universalized to provide a best start in life. Nursing mother should receive nutritional supplements to improve the quality of breast milk. Breast feeding should be continued as long as feasible and adequate intake of home-cooked cereal-based semi-liquid complementary feeds should be introduced at six months of age taking due precautions to prevent bacterial contamination and infections. Food fussiness should be prevented by intelligent and relaxed handling of pre-school children during meal times. School-going children and adolescents should be encouraged to take a balanced nutritious diet to prevent both undernutrition as well as obesity. The practice of missing the breakfast must be condemned and all efforts should be made to ensure that breakfast is the most wholesome meal of the day. Children should be discouraged to follow the unhealthy dietary practices like consumption of soft drinks and junk food and instead encouraged to become more milk-friendly.

Children should be encouraged to take a balanced nutritious diet to ensure adequate intake of macronutrients (carbohydrates, proteins and fats), micronutrients (vitamins and minerals), phytonutrients, antioxidants and fiber. But many a times, it is not possible to ensure intake of 100% RDAs of micronutrients from dietary sources alone because most children do not like to take green leafy vegetables or fruits, and they are fussy, choosy and rebellious in their food habits. The situation may be worse among vegetarians because of poor availability of iron, zinc, vitamin B<sub>12</sub>, and decosa hexaenoic acid (DHA). In view of high prevalence of subclinical deficiencies of micronutrients, it is thus mandatory that nutritional supplements should be given to children (especially during pre-school years and adolescence) to ensure that their full genetic potential for physical growth

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and mental development is achieved in order to provide a solid foundation to our society.

The health and well being of children depends upon the interaction between their genetic potential and exogenous factors like adequacy of nutrition, safety of environment, social interactions, physical activity and stimulation. Nutrition has a global role to promote physical growth, enhance neuromotor development, boost host defences to ward off common day-to-day infections, retard the process of aging, and prevent occurrence of age-related degenerative diseases and thus improve the quality of life.

**Nutritional status of children**

Unlike offsprings of other mammals who are on their feet at birth and can search for their food, human babies are entirely at the mercy of their caretakers for at least 3-5 years of life. They have high energy and nutrient requirements due to rapid growth velocity and neuromotor development. Because of high incidence of low birth weight babies and unsatisfactory feeding practices, nutritional disorders are common in developing countries. Nutritional status is further compromised due to occurrence of recurrent respiratory and GI infections because of poor environmental sanitation, overcrowding and lack of safe drinking water.

The florid cases of kwashiorkor, severe protein-energy malnutrition and various syndromes due to gross deficiencies of single nutrients (like scurvy, rickets, pellagra, beri-beri etc.) have become rare. Nevertheless, there are still widespread diseases of public health relevance due to deficiency of micronutrients like iron deficiency anemia, iodine deficiency disorders and milder forms of vitamin A deficiency. However, though deficiency of isolated or single nutrient is rare in clinical practice an individual is more likely to have a deficiency of multiple micronutrients.

Due to control of florid or severe cases of malnutrition, the child survival has improved but the quality of life and human resource development has not improved. Over 50 percent of under-5 children in India are stunted due to intrauterine growth retardation and post-natal protein-calorie malnutrition. According to National Nutrition Bureau of India, 80-90% children take less than 30% RDA of green leafy vegetables. Therefore, iron consumption is inadequate in 90% of individuals in India. Dietary surveys have shown that two-thirds of adolescents consume less than 70% RDA of vitamin A and riboflavin. Intake of calcium, vitamin B complex and vitamin C is also inadequate. Studies conducted at National Institute of Nutrition, Hyderabad have shown that over 50% of apparently healthy school going children have subclinical or biochemical deficiencies of micronutrients. There is increasing evidence that deficiency of certain micronutrients may adversely affect the physical and mental growth of children. With progressive elimination of protein-energy deficits in the diets, deficiencies of micronutrients are emerging as the major bottleneck to compromise optimal physical growth and mental development of children.

**Nutritional status and infections**

Studies during the past two decades have demonstrated the importance of optimal nutrition for the functional integrity of the immune system. Both under nutrition and over-nutrition as well as deficiencies and excess of single nutrients have been shown to have adverse effects on the immune system. Recently, studies have shown that immunological dysfunction is the earliest marker of deficiency of micronutrients. Every few days our body replaces one quarter of our immune cells. For example, neutrophils have a half life of merely 36 hours! Therefore, the immune system needs continuous supply of vitamins and minerals for their regeneration.
A large number of vitamins (vitamins A, E, C, pyridoxine, folic acid) and trace minerals (iron, zinc, selenium, copper) are credited to enhance cell-mediated and humoral immune responses. There is increasing clinical and laboratory evidence to suggest that children with subclinical deficiencies of various micronutrients are more vulnerable to develop a variety of common day-to-day infections. They are likely to have more severe infections with prolonged convalescence. Infective illnesses are recognized to adversely affect the nutritional status by causing anorexia, tissue catabolism and enhanced utilization of micronutrients, thus setting up a vicious cycle of undernutrition, recurrent infections and unsatisfactory physical growth (Figure 1). During antigen-antibody fight, there is increased production of reactive oxygen-free radicals which may further adversely affect the integrity of immune cells by damaging their mitochondria. There is enough clinical and laboratory evidence to suggest that deficiency of micronutrients is associated with increased incidence and severity of common day-to-day infections which can be prevented or controlled by giving nutritional supplements.

Physical growth

Over 50 percent of under-five children are stunted in India. They have sub-optimal vigour and stamina, poor neuromotor coordination, learning skills and mental capabilities. Vitamins and trace minerals are required for production of various enzymes, hormones and biochemical mediators for regulation of biological processes. They are required for energy production, synthesis of RNA and DNA and for providing protection against reactive oxygen-free radicals. Interaction between sub-optimal nutrition and occurrence of repeated infections is the leading cause of growth retardation in children in developing countries. Dietary inadequacies and recurrent infections, interact in a mutually reinforcing manner to further aggravate nutritional status. Calcium, phosphorus, vitamins A, C, D and K are required to maintain the integrity and mineralisation of bones.

Brain development

It is not generally realized that neurons are more sensitive to nutrients and dietary chemicals compared to other body cells. Optimal nutrition during pregnancy and first 3 years of life is most crucial because 70 percent of the human brain develops during fetal life and remaining 30 percent during pre-school years. Micronutrients are required for production of several enzymes and co-factors for a number of metabolic pathways. It has been well known that pellagra (niacin deficiency) leads to reduced cognition and dementia. A number of other B-complex vitamins especially B₁, B₂, B₆, B₁₂ and folic acid are needed for synthesis of several neurotransmitters. Deficiency of folate, B₆, B₁₂ and choline are associated with elevation of plasma homocysteine level which may lead to thromboembolic complications and stroke. Iodine is required for synthesis of tri-iodothyronine and thyroxin. Iron is required for proper functioning of neurotransmission system by production of dopamine, serotonin and GABA. Iron deficiency has been shown to adversely affect brain stem development.
auditory activity and visual evoked potentials which may persist even after correction of iron deficiency anemia. Zinc is a component of a large number of metalloenzymes and there is high concentration of zinc in the brain. Copper is an important component of cytochrome-C oxidase and superoxide dismutase in the brain. Fish and fish oils are important sources of omega-3 fatty acids and decosahexaenoic acid (DHA). Omega-3 fatty acids are credited to reduce cellular and vascular inflammation in the brain, promote vasodilatation and ensure integrity of brain cell membranes to keep them soft and pliable. DHA is the building material for fabrication of synaptic communications and constitute almost one-half of the total fat in the brain cell membranes. It increases the level of “feel good” neurotransmitter serotonin and the “memory boosting” chemical acetylcholine. According to WHO, pregnant women and nursing mothers should take 2.6g omega-3 fatty acids and 100-300 mg DHA per day to meet the nutritional needs of the baby intra-uterine and her breast fed infant. The brain-friendly nutrients are listed in Table 1.

Table 1. Smart nutrients for the brain

| Omega-3 fatty acids, decosahexaenoic acid (DHA) and arachidonic acid. |
| Vitamin B complex, folic acid, vitamin C, vitamin E |
| Iodine, iron, zinc, copper and selenium |
| Essential amino acids including taurine |
| Glucose |
| Choline |
| Antioxidants |

Life-cycle approach for the care of girl children

Health and well being of mothers and their children are intimately linked. Healthy mothers produce healthy babies while sick and malnourished mothers produce high-risk and low birth weight babies. The health and growth of the fetus in the womb is dependent upon the health, well being and nutritional status of the mother (rather than the father!) because she is both the seed as well as the soil where baby is nurtured for 9 months. Moreover, healthy, educated and well-informed mothers are in a better position to look after the health needs of their children. Therefore, a life-cycle approach should be followed to provide optimal nutrition and health care to girl children from infancy through childhood, adolescence, pregnancy and lactation (Figure 2). During adolescence, girls must be given balanced nutrition with supplements of iron, folic acid, calcium and phosphorus to build adequate nutritional stores to meet the nutritional demands of pregnancy and lactation.

Breast feeding

Breast milk is nutritionally complete and biologically most compatible drink and every baby must receive exclusive breast feeding during first 6 months of life. Breast milk is a nutritionally complete food for infants and contains several brain-friendly nutrients like lactose, DHA, zinc, choline, iodine and taurine. Breastfed babies have been shown to have 8 IQ points higher cognition compared to bottle fed infants. The baby should be put to the breast as soon as the mother has recovered from labor. Prelacteal feeds should not be given and colostrum should never be discarded because it is replete with protective antibodies. The baby should be given demand feeding and each breast should be completely emptied so that the baby gets both the fore milk as well as the hind milk. The nutritional quality of breast milk can be enhanced by improving the diet and providing nutritional supplements to the lactating mother. During the period of exclusive breast feeding, there is no need to provide any nutritional supplements to the baby.
**Complementary feeds**

Milk alone is a complete food to meet all the nutritional requirements during first 6 months of life. Most nutritional problems start at weaning time due to delay or unsatisfactory weaning foods and risk of contamination. There is a potential risk of occurrence of diarrhea and growth faltering during the process of weaning. Breast feeding should be continued for atleast 1 year or even longer and semi-solid cereal-based weaning foods should be introduced around 6 months of age. During weaning, bottle feeding should not be introduced but milk-products can be given. Milk can be offered with a cup or a glass after one year of age when breast feeding is gradually tapered off. The child should be offered calorie-dense, home-cooked, properly mashed cereal-based foods. A variety of food items which are liked and enjoyed by children should be offered like *khichri*, ragi, soft porridge, *suji kheer*, rice *kheer*, custard, rice and *dal*, bread and *dal*, mashed vegetables, yoghurt and banana. Butter and *ghee* should be added to increase caloric density. Mother should be advised to start with 1-2 teaspoons of semisolid foods at a time and gradually increase the quantity. Egg can be introduced at 6 months and minced meat, chicken and fish can be offered after the age of 9 months. The weaning foods should be given atleast 4-5 times/day by the end of first year. During weaning safe drinking water should be offered while maintaining strict personal hygiene to prevent contamination and risk of bacterial infection. WHO has published international guidelines for best feeding practices during the early years of life.
During weaning period supplements of micronutrients should be provided. All efforts should be made to provide optimal nutrition to children during first 3 years of life which are most crucial for optimal physical growth and mental development. It is well recognized that the stature achieved by the child at 3 years of age is a good predictor of ultimate adult height. During this period growth parameters should be monitored on Road-to-Health charts. The National Center of Health Statistics (NCHS) has published revised growth charts which should be used.

Table 2 highlights the common myths and food fads which must be discouraged by health education and awareness programs to promote optimal nutrition of children.

### Table 2. Common myths and food fads

<table>
<thead>
<tr>
<th>Myth</th>
<th>Reason</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children with diarrhea are often starved to give rest to the gut.</td>
<td></td>
</tr>
<tr>
<td>“Weaning” for some mothers implies stopping breast feeding.</td>
<td></td>
</tr>
<tr>
<td>“Weaning” is commonly delayed until “Anna Prasana” ceremony is held.</td>
<td></td>
</tr>
<tr>
<td>Many mothers have a fancy for fruit juices which are “useless” to provide energy and proteins. They are associated with a high risk of bacterial contamination.</td>
<td></td>
</tr>
<tr>
<td>Mothers often give watery soups and “dal ka pani” which has poor nutritive value.</td>
<td></td>
</tr>
<tr>
<td>Children with fever are often starved or given only grapes and pomegranate!</td>
<td></td>
</tr>
</tbody>
</table>

**Food fussiness**

<table>
<thead>
<tr>
<th>Myth</th>
<th>Reason</th>
</tr>
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<tr>
<td>Due to excessive concern and over protection because of one or two child norm in the society, feeding of preschool children demand considerable art and tact to tackle the problem of food fussiness and food preferences. The development of food fussiness should be prevented by avoiding overindulgence and not paying excessive concern and attention to the child’s food. The individual likes and dislikes of the child should be identified and honored and he should be offered a variety of food items to break the monotony. Parents should adopt a relaxed attitude at meal times and let the child enjoy what he likes to eat. There should be “intelligent neglect” of the child. Children do have a rebellious attitude and many a times a negative statement like “Kabir would not get his food today” may evoke a positive response. The best way to make the child eat is “not to try”. The child should be encouraged to self-feed even if he creates a mess. Most children would like to eat when other family members are eating or even take a bite from their plate. After giving a reasonable time, the plate should be quietly removed even if the child has not finished without showing any concern and anxiety. The whole family including grandparents must participate in the mission approach to change the “blackmailing” behavior of the child. Because food fussiness is a behavior disorder and it is not due to “weak liver” or loss of appetite, the role of tonics and appetizers is doubtful. The emphasis should be placed on changing the attitude of the family.</td>
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**Diet of school-going children**

During adolescence, there is a rapid spurt of physical growth and sexual maturation. Almost 20% of stature and 50% of bone mass are laid down during adolescence. Adequate intake of calories (3000 kcal/d) should be ensured by consumption of plenty of pulses, legumes, fresh green leafy vegetables, seasonal fruits, milk and dairy products. It has been shown that well-nourished girls have higher premenarcheal growth velocities and reach menarche earlier while undernourished girls continue to grow more slowly for a longer period because menarche is delayed. During this period, junk food should be avoided and children given extra calories, proteins and micronutrients like calcium, iron, iodine and zinc. Intake of soft drinks should be discouraged due to...
their health hazards and efforts should be made to make children more milk-friendly.

**Soft drinks and junk food**

Most children hate to take milk and find it boring, colorless, insipid and conventional. They usually get hooked to take soft drinks due to catchy and aggressive advertisements. In some children milk may cause abdominal pain and flatulence. Soft drinks provide empty calories and are devoid of wholesome nutrients. Caffeinated drinks may cause restlessness, insomnia and addiction. They may predispose to development of osteoporosis due to calciuria and displacement of milk intake. Dental caries may occur due to exposure of teeth to the acidic pH of soft drinks. The coloring agents in the drinks may cause allergic disorders. The presence of phosphoric acid, fizz and bubbles may cause damage to the mucosa of the gut. According to the reports published by Bhabha Atomic Research Centre, there may be excessive production of a toxic agent called hydroxyl methyl furfural when soft drinks are stored in room temperature in hot summer months. And there is an issue and controversy regarding the presence of excessive amounts of pesticides in soft drinks. In view of the aforementioned facts children should be discouraged to take soft drinks, and there is a need to print a statutory warning on soft drink bottles that “excessive intake of soft drinks may be hazardous to the health of children”. Instead children should be encouraged to take milk and dairy products. Milk drinks should be made available in different flavours in tetrapacks having attractive and catchy motifs. Nutritional supplements can be added to the milk to change its color, taste, flavour and nutritional value to make children more milk-friendly. When a child dislikes to take milk or milk intake is associated with bloating or abdominal discomfort, he should be encouraged to take milk products like youghurt, kheer, porridge, custard and cheese.

Due to changes in life-style, indulgence in soft drinks and junk food, adoption of sedentary habits, almost 25% of adolescent children belonging to well-to-do urban families are overweight or overtly obese. They are also an important cause of constipation due to lack of fiber. Junk food like hot dogs, hamburgers, French fries, pizzas, samosas, pakoras, kachories, milk shakes, soft drinks, desserts etc. are loaded with calories, saturated fats, transfatty acids and excessive amount of omega-6 fatty acids leading to obesity and adverse health consequences later in life. Studies have shown that fast foods provide additional 187 kcal/day leading to additional weight gain of 3 kg/year. Children should be discouraged to take junk food or alternatively fast food items should be made more health-friendly by reducing their content of saturated fats and ensuring liberal use of health-friendly omega-3 fatty acids, vegetables and fruits in their production.

**Meal proportions and their distribution**

There is a popular saying that “Eat breakfast like a king, lunch like a prince and dinner like a pauper”. Breakfast should be the most wholesome meal of the day because it is taken after a long gap of fast and it must have enough calories and essential nutrients to start the day with optimal energy and enthusiasm. Instead, studies have shown that over 50% of school going children in our country miss their breakfast because they sleep late, get up late in the morning and there is not enough time to take breakfast before leaving for school. There is evidence to suggest that skipping of breakfast may adversely affect their vigour, zest, memory, learning capability, academic performance, emotional and psychological well being. Hungry children are also more likely to have headaches and tummy aches. Parents must be informed about the health hazards of missing the breakfast and they should be motivated to provide wholesome breakfast to their children. The habit
of taking snacks (like potato wafers or chips, French fries, namkeens, biscuits etc) in-between meals, binging and fasting must be condemned because of potential risk of causing obesity.

Conclusion

All efforts should be made to ensure that children take a well balanced nutritious food by encouraging them to consume green leafy vegetables, lentils, soyabean, seasonal fruits, milk and dairy products, eggs, fish, chicken etc. However, the prevailing dogma in nutritional science that a balanced diet is sufficient to meet all the nutritional requirements has been challenged. According to the recommendations of United Nations Sub-committee on Nutrition it is not possible to meet the requirements of 100% recommended dietary allowances (RDAs) of micronutrients from dietary sources alone\textsuperscript{24}. The situation is worse among vegetarians and young children because they are fussy, choosy and rebellious in their food habits. Nutritional supplements are thus mandatory to improve physical growth and mental development and prevent occurrence of common day-to-day infections. Healthy children do provide a solid foundation to the society inorder to ensure optimal human resource development of a country and focus on their optimal nutrition is of fundamental importance to achieve that goal.

References


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**NEWS AND NOTES**

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RADIOLOGIST TALKS TO YOU

HEPATOMEGALY AND HEPATIC MASSES - I

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**Ramalingam A

Mass lesions in the liver may be solid or cystic, single or multiple, they may be incidental findings, or present as hepatomegaly. Ultrasound is the primary screening technique and can be very informative.

Abscesses are the commonest space occupying lesions (SOL) encountered in an enlarged liver (Fig.1). They may present acutely with pain and fever. The ultrasound appearance may vary depending on the duration of the abscess. Evolving abscesses tend to have an appearance that resemble a solid lesion. As the pus liquefies they become cystic or hypoechoic. Doppler ultrasound will help in differentiating between an abscess and a mass lesion; vessels will be seen traversing through a mass lesion due to increased vascularity, whereas in an abscess they may be splayed out. On plain CT, an abscess is hypodense relative to the surrounding liver, similar to other cystic lesion. Contrast CT however shows the pathognomonic feature of an enhancing ring around the non-enhancing collection of pus in an abscess.

Hydatid cyst is another hypoechoic cystic lesion (Fig 2). These may be unilocular or multilocular (a single large cyst with multiple daughter cysts within it). Degeneration or damage of the cyst wall leads to a contained internal rupture of the membranes. The detached membranes float within the cyst, an appearance likened to a serpent. Later, the cyst wall undergoes calcification which is seen as white curvilinear rim. A hydatid cyst may also get

Fig. 1. Liver abscess (SOL)

Fig. 2. Hydatid Cyst - US

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infected and at that stage it is not possible to
differentiate it from an abscess, even by CT.

Cysts can also be a part of the autosomal
dominant polycystic kidney disease (Fig 3).
Simple bile cysts are rare and can be mistaken
for the unilocular type of hydatid cyst. One
differentiating feature that will help is the margin
of the cysts. Bile cysts tend to have a scalloped
edge (Fig. 4).

Another rare lesion is the mesenchymal
hamartoma, a developmental anomaly which arises
from the mesoderm of the portal tract. They
present in children less than 2 years of age as
painless hepatomegaly. They are slow growing but
can suddenly increase in size due to rapid fluid
collection. On ultrasound, there is a well defined
mass which is either cystic or solid. It is clearly
demarcated from the surrounding liver and is
usually found on its undersurface. On real time
ultrasound the mass moves with respiration along
with the liver. The mass shown in Fig 5 is cystic
with some solid elements. The intrahepatic biliary
radicals are normal. CT supports the sonographic
findings and shows the extent of the mass with thick
enhancing septae running between the low
attenuating cystic parts (Fig. 6). In MRI, the solid
mesenchymal component is hypointense while the
cystic parts are hyperintense in T2W images.
ABDOMINAL EPILEPSY AS A CAUSE OF RECURRENT ABDOMINAL PAIN

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**Pangrikar AG

Abdominal pain or discomfort with or without nausea and vomiting or other sensations sometimes occurs in association with a convulsive disorder. A concept that abdominal epilepsy is an entity has been proposed by number of authors in the past. Recent reports are few and the criteria for diagnosis have become hazy. Trousseau first described abdominal epilepsy in 1868.

The association of abdominal symptoms with epilepsy has been recognized for many years. The invention and clinical application of electroencephalogram (EEG) in 1920’s shifted the focus of medical attention from abdomen to brain where, for the most part, it has remained to this date. Modern medical science has rediscovered abdominal connection in epilepsy. Common clinical features of abdominal epilepsy include abdominal pain, nausea, bloating, and diarrhoea with nervous system manifestations such as headache, confusion and syncope.

We report a 10 year male child presenting with recurrent abdominal pain of long duration who had undergone several investigations and treatments at various hospitals before being diagnosed as having abdominal epilepsy.

Case Report

10 year male child born of third degree consanguinity was brought for episodic pain in abdomen since last five years. Pain was localized to upper abdomen and peri umbilical region as a feeling of upward thrust, lasting for 2 to 3 minutes and subsided on its own. Pain was associated with lacrimation, palpitation, unresponsiveness and incontinence of bowel with each episode. Initially such episodes occurred at monthly intervals, frequency increased to every fortnightly and now since last 6 months twice a week. There was no incontinence of urine, vomiting, emotional insult or any association with specific foodstuff or altered pattern of defecation.

When child reported to us, he had already undergone following investigations. Plain x-ray abdomen, ultrasonography of abdomen thrice by different persons and were reported normal. Urine and stool examinations revealed nothing abnormal. Barium meal followthrough done at other hospital did not reveal any abnormality.

He had repeated antihelminthic, antiamoebic and antispasmodic treatments but had no response. He is a school going child with average school performance with normal development. His anthropometric measurements, physical examination and hemogram were normal. Urine examination was negative for porphobilinogen.

With the typical history, we did his electroencephalogram (EEG) which revealed focal spike and sharp waves around left temporal region (Fig. 1). CT brain was normal. Child was started on anticonvulsant carbamazepine. After one year of starting anticonvulsants, he is totally symptom free.
Discussion

Abdominal epilepsy is a rare disorder, difficult to diagnose. It is paroxysmal in nature and may be confused with abdominal migraine.\(^3\)

One area of particular concern for pediatrician is proper diagnosis of abdominal epilepsy. The epigastric aura (with or without a rising sensation) as seen in our case is the most common premonitory symptom in complex partial seizure. Vomiting and abdominal pain may occur in association with altered responsiveness, other signs of epilepsy and abnormal EEG findings. The onset of the attack is usually paroxysmal and postictal sleepiness is the rule.

Attacks of abdominal epilepsy usually lasts for minutes and usually responds to anticonvulsants.\(^3\)

The pathophysiology of abdominal epilepsy remains unclear. Temporal lobe seizure activity usually arises in or involves the amygdala. It is not surprising therefore that patients who have seizures involving temporal lobe to have GI symptoms, since discharges arising in amygdala can be transmitted to gut via dense direct projections to the dorsal motor nucleus of the vagus. In addition, sympathetic pathways from amygdala to GI tract can be activated via hypothalamus. On the other hand, it is not clear that the initial disturbance in abdominal epilepsy arises in brain. There are direct sensory pathways from the bowel via vagus nerve to the solitary nucleus of medulla which is heavily connected to amygdala. These can be activated during intestinal contractions.\(^2\)

Usually, abdominal epilepsy is idiopathic. However it may be rarely secondary to developmental brain disorders like polymicrogyria. Hence computed tomographic scan as well as MRI may be indicated in patients with abdominal epilepsy.\(^4\)

Many a times abdominal epilepsy is misdiagnosed as psychogenic pain, however, a triad of paroxysmal pain, abnormal electroencephalogram (EEG) and remarkable response to anticonvulsants confirms the diagnosis.\(^5\)

Though the condition is rare, it should be considered in differential diagnosis in a child with unexplained paroxysmal abdominal pain.\(^6\) It is suggested that no one symptom satisfies the requirement for diagnosis. The rigid criteria required for the diagnosis of other forms of

![Fig.1: Showing Polygraphic EEG record with paroxysmal spikes and waves in the left temporal region.](image)
epilepsy should also be applied to this syndrome. The conclusion that abdominal pain is of central origin should be made on positive rather than negative ground.

References


NEWS AND NOTES

XX SOUTH ZONE CONFERENCE OF IAP
SOUTH PEDICON 2006, SALEM
XXXI ANNUAL CONFERENCE OF IAP-TNSC
ORGANISED BY THE IAP-SALEM DISTRICT BRANCH
VENUE: HOTEL CENNEY’S GATEWAY, SALEM
DATE: AUGUST 18TH – 20TH 2006

Greetings from IAP Salem District Branch. It is our privilege to welcome you in Salem, for the South Pedicon 2006. A fabulous mixture of academics and cultural feast awaits everyone.
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Mode of payment by Demand Draft, Drawn in favour of “South Pedicon 2006” payable at Salem.

For Registration and other inquiries contact: Organising Secretary, South Pedicon 2006, Pranav Hospitals Pvt.Ltd., 108/38, Brindhavan Road, Salem – 636 004.
Ph.No: 0427 – 2336787, 2336788 E-mail: southpedicon2006@rediffmail.com
For details regarding paper presentation please refer our website: www.southpedicon2006.com
ACQUIRED VELOPALATOPHARYNGEAL PALSY

* Rana K S  
** Behera M K  
*** Sood A

Abstract: Unilateral velopalato pharyngeal palsy is rare. The exact etiology of this condition is not known, though a post viral immune mediated response has been postulated. The present case developed features of isolated 9th nerve palsy after a viral illness. Rest of the neurological and local examination of the neck was normal. Neuro imaging of the head and neck was normal. He has completely recovered after a course of steroid.

Key words: Velopalatopharyngeal palsy.

Unilateral palatal palsy is uncommon. The etiology and pathogenesis of this condition remains unclear till date. This is usually a benign and a self-limiting condition. Viral infections have been postulated as a possible cause. We report a male child with multiple neurofibromas, who developed acute onset right-sided velopalatopharyngeal palsy and subsequently recovered completely.

Case Report

An eight years old male child was admitted with history of sudden onset nasal regurgitation of fluids in right nostril, nasal intonation of voice and difficulty in swallowing of seven days duration with history of running nose and dry cough, a week before the onset of illness. His symptoms increased over the next few days and then was static. There was no history of any visual and auditory difficulty, drooling of saliva and difficulty in chewing, breathing or coughing. Examination revealed absent palatal movements on a deviation of uvula to left while demonstrating gag reflex. There was no other neurological deficit. Examination also showed that he had multiple neurofibromas measuring 10 to 30mm on the inner aspect of left arm. He did not have any other neurocutaneous markers or dysmorphic features. Local neck examination and that of the ear, nose and throat including laryngoscopic examination of larynx was normal.

Hemogram and metabolic parameters were normal. CSF analysis, Brainstem Auditory Evoked Response, Magnetic Resonance Imaging and Angiography (BAER, MRI & MRA) of the brain and neck revealed no abnormality.

He was managed with oral prednisolone (1.5mg/kg) initially for two weeks and subsequently tapered over the next two weeks. The child started improving within the first week of treatment and the nasal regurgitation and nasal intonation subsided. He is being followed up and is presently asymptomatic.

Discussion

Acquired velopalatopharyngeal hemi-paralysis, a rare condition, is seen mostly in children with a male preponderance and is usually unilateral. It was first described by Edin in 1972. Most cases (>96%) had acute onset of nasal intonation,
unilateral nasal escape of fluids with varying degrees of dysphagia\(^3\). The present child had all these symptoms. No definite etiology has been found out but post viral immune mediated involvement of the ninth nerve has been postulated. Viral studies have always been negative. CSF examination has shown elevated IgG and IgG/albumin ratio. Respiratory infection prior to the illness has been documented in 35% cases\(^4\). Although unilateral absence of palatal reflex mostly indicates IX cranial nerve lesion, vagal involvement can also rarely result in unilateral palatal paralysis\(^5\). For definite diagnosis of isolated velopalatopharyngeal palsy, vocal cords involvement must be excluded. Hoarseness of voice and a bovine cough indicate a vocal cord palsy. A direct laryngoscopic examination is essential to rule out vocal cord involvement. A meticulous clinical search should be done for multiple cranial nerve palsies due to brainstem involvement because of vascular causes, tumors, syringobulbia, motor neuron disease and inflammatory diseases. Lesions around the ears and pharynx can also cause multiple cranial nerve palsies because of the close proximity of extra cranial course of the last four cranial nerves after their exit from cranial foramina. Isolated lesion of nucleus ambiguus leads to a combined palatopharyngeal and laryngeal palsy. Rarely an isolated injury to cephalic portion of the nucleus can lead to isolated palatopharyngeal palsy with laryngeal sparing\(^5\). Velopalatopharyngeal insufficiency secondary to local causes like submucosal cleft palates easily differentiated by long history and by local examination. The cranial MRI (with angiography) is the diagnostic modality of choice and is invariably normal. In the present case also, MRI with angiography of the head and neck was normal.

No specific treatment is required. Prognosis is excellent. Steroids have been used empirically. Recurrence is rare in children. A self limiting course is the norm with complete recovery in >85% cases over 2-3 weeks\(^3\). Multiple neurofibromas in this child were an incidental finding.

To conclude, sudden onset unilateral velopalatopharyngeal palsy in children is rare. It is a benign and self-limiting condition with almost complete recovery. Post viral infectious immune reaction is one possibility and that is why some people recommend short course of steroids.

Reference

PEDICON 2007
44th Annual Conference of the Indian Academy of Pediatrics, 12-14 January 2007
&
IAP-AAP CME 2007, 11th January 2007

Registration form

IMPORTANT: Please note:

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• Registration fees for all categories have been mentioned in Indian rupees, except the categories of foreign delegates which are in US dollars

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