QUICK GUIDE FOR PEDIATRIC EMERGENCIES

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PEDIATRIC EQUIPMENT

	Newborn	6 months	1 yrs	2-3 yrs	4-6 yrs	7-10yrs	11-15yrs	>16 yrs
Weight	2-4	6-8	10	12-16	20-25	25-35	40-50	>50
Laryngoscope Blade	MIL 0	MIL 1	MIL 1 MAC 2	MIL 1 MAC 2	MIL 2 MAC 2	MIL 2 MAC 2	MIL 2 MAC 3	MIL 2 MAC 3
Endo tracheal tube	3.0-3.5	3.5	4.0	4.0-4.5	4.5-5.0	5.5-6.0	6.0-6.5	7.0-8.0
Laryngeal mask airway	1	1.5	2	2	2.5	2.5-3	3	4
Central venous line (Fr)	3-4	4	4-5	4-5	5	5	7	7
NG Tube (Fr)	5-8	8	10	10-12	12-14	12-14	14-18	14-18
Chest tube (Fr)	10-12	12-18	16-20	16-24	20-28	20-32	28-38	28-42
Foley (Fr)	8	8	8	8	8	8	10	12

ENDOTRACHEAL TUBE(ETT) FORMULAS

Uncuffed ETT size = Age(years)/4 + 4

cuffed ETT size = Age(years)/4 + 3.5

ETT depth (from lip to mid-trachea): ETT internal diameter (size) x 3

CHAPTER-1

ACUTE EXACERBATION OF ASTHMA

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms, such as wheeze, shortness of breath, chest tightness and cough, that vary over time and in intensity, together with variable expiratory airflow limitation.[1]

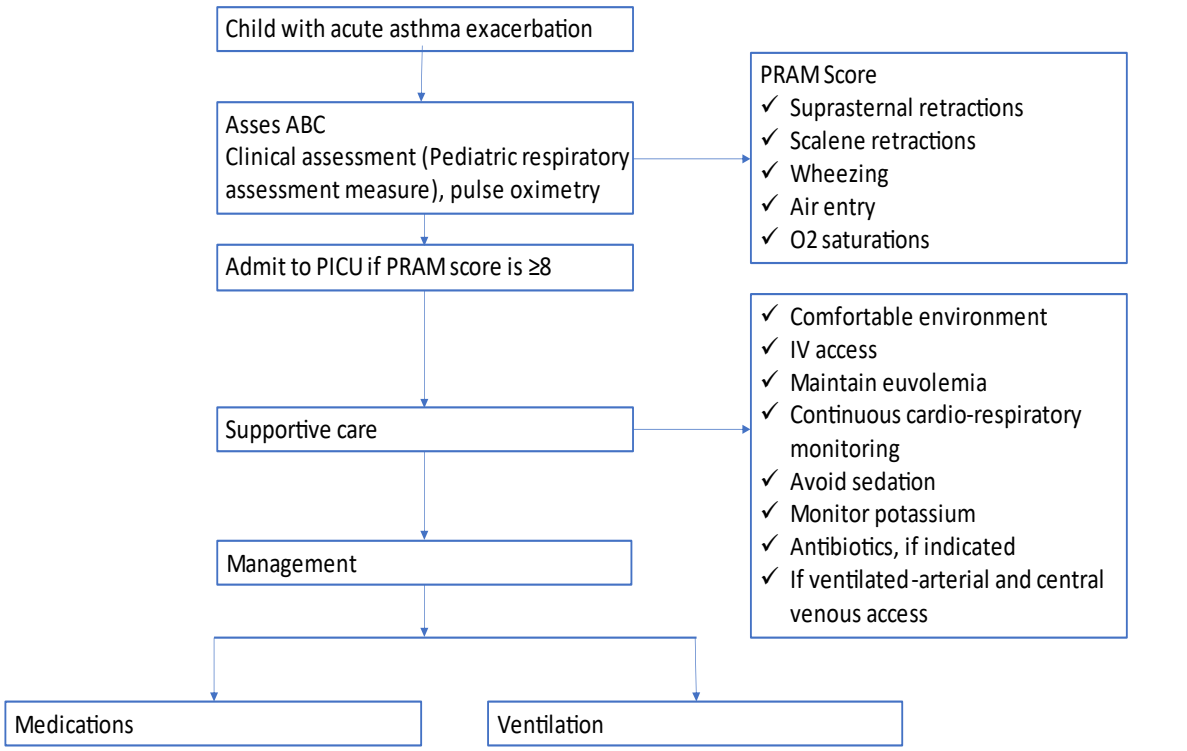
Exacerbations of asthma are episodes characterized by a progressive increase in symptoms of shortness of breath, cough, wheezing or chest tightness and progressive decrease in lung function, i.e., they represent a change from the patient's usual status that is sufficient to require a change in treatment.[2]

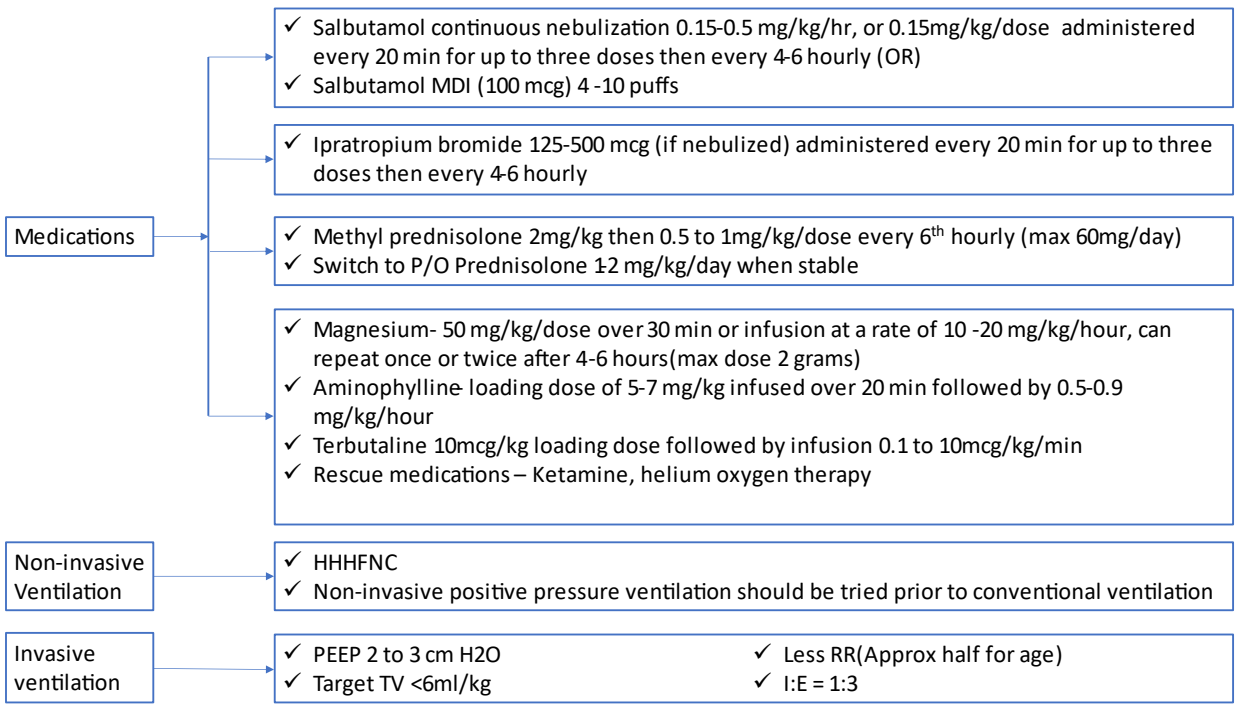
Management in an emergency depends on the severity at presentation and response to treatment.

Initial assessment of acute exacerbation of asthma:[4]

Symptom	**Mild**	**Moderate**	**Severe or life threatening**
Confused/drowsy	No	No	Agitated or altered
Oximetry on presentation (SpO2)	94%	94%-90%	Less that 90%
Talks in	Sentences	Phrases	Words or unable to speak
Pulse in	< 100 /min	100-200 /min	>200 /min or bradycardia
Central cyanosis	Absent	Absent	Likely to be present
Wheeze intensity	Variable, mainly at end exhalation	Moderate to loud, present in both phrases of respiration	Audible wheeze or silent chest due to poor air entry

Initial management of children who have acute asthma.[3]





CHAPTER-2

ANAPHYLAXIS

Anaphylaxis is an acute, potentially lethal, multisystem syndrome resulting from the sudden release of mast cell- and basophil-derived mediators into the circulation[5].

It most often results from immunologic reactions to foods, medications, and insect stings, although it can also be induced through nonimmunologic mechanisms by any agent capable of producing a sudden, systemic degranulation of mast cells or basophils[6].

CLINICAL CRITERIA FOR DIAGNOSING ANAPHYLAXIS[5]

Anaphylaxis is highly likely when any one of the following 3 criteria are fulfilled:
1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g., generalized hives, pruritus or flushing, swollen lips-tongue-uvula) AND AT LEAST ONE OF THE FOLLOWING a. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced peak expiratory flow, hypoxemia) b. Reduced Blood Pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence)
2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours): a. Involvement of the skin-mucosal tissue (e.g., generalized hives, itch-flush, swollen lips-tongue-uvula) b. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced Peak Expiratory Flow, hypoxemia) c. Reduced Blood Pressure or associated symptoms (e.g., hypotonia [collapse], syncope, incontinence) d. Persistent gastrointestinal symptoms (e.g., crampy abdominal pain, vomiting)
3. Reduced Blood Pressure after exposure to known allergen for that patient (minutes to several hours): a. Infants and children: low systolic Blood Pressure (age specific) or greater than 30% decrease in systolic Blood Pressure* b. Adults: systolic Blood Pressure of less than 90 mm Hg or greater than 30% decrease from that person's baseline

MANAGEMENT[7]:

Place patient in recumbent position

Airway: Immediate intubation if evidence of impending airway obstruction from angioedema. Delay may lead to complete obstruction.

Oxygen: Give 8 to 10 L/minute via facemask or up to 100% oxygen, as needed.

Epinephrine:

• The first and most important therapy in anaphylaxis is epinephrine. There are NO absolute contraindications to epinephrine in the setting of anaphylaxis.

• Epinephrine 0.01 mg/kg should be injected IM in the mid-outer thigh (maximum is 0.5 mg per dose)

• If there is no response or the response is inadequate, the injection can be repeated in 5 to 15 minutes (or more frequently). If epinephrine is injected promptly IM, patients respond to 1, 2, or, at most, 3 injections.

• If signs of poor perfusion are present or symptoms are not responding to epinephrine injections, prepare IV epinephrine for infusion 0.1 to 1.0mcg/kg/min

Normal saline rapid bolus: Treat poor perfusion with rapid infusion of 20 mL/kg. Reevaluate and repeat fluid boluses (20 mL/kg), as needed.

Salbutamol: For bronchospasm resistant to IM epinephrine, give Salbutamol 0.15 mg/kg (minimum dose: 2.5 mg) inhaled via nebulizer.

H1 antihistamine: Consider giving diphenhydramine 1 mg/kg (max 50 mg IV, over 5 minutes) or cetirizine (children aged 6 months to 5 years can receive 2.5 mg IV, those 6 to 11 years of age can receive 5 or 10 mg IV, over 2 minutes).

H2 antihistamine: consider giving IV ranitidine 1mg/kg/dose (max dose 50mg)

Glucocorticoid: Consider giving methylprednisolone 1 mg/kg (max 125 mg) IV.

Patient needs to be admitted and further managed in the ICU.

Pediatric Pearls
Stock adequate adrenaline & check expiry date every month Observe patient for half an hour after administration of both vaccines & drugs routinely. Identify anaphylaxis prone children and teach parents to administer adrenaline in emergency situations. EpiPen is an alternative but not available in India.

CHAPTER-3

BARBITURATE POISONING

Barbiturates are a class of sedative-hypnotic drugs. They are commonly used as antiepileptics (phenobarbital) and for the induction of general anesthesia (thiopental).

Signs and Symptoms[8]:

Decreased level of consciousness

Poor coordination

Vertigo

Nausea

Muscle weakness

Thirst

Oliguria

Decreased temperature

Dilated or contracted pupils

Bradycardia

Rapid and weak pulse

Fatal cases are marked by coma, hypotension and respiratory depression.

Investigations[8]:

Qualitative assessment of the amount of drug taken with a plasma or urine screening test.

Plasma barbiturate concentration and ingested dose have been implicated as the most important correlates of toxicity.

Blood glucose, pH, and serum electrolytes

Management[8,9,10]:

Assessing the patient's airway, breathing, and circulation. With significant sedation and respiratory depression, intubation and mechanical ventilation may become necessary.

Monitor and support Circulation by fluids, inotropes.

Gastric decontamination – Up to 6 hours post ingestion

Ensure airway is adequately protected.

Activated charcoal 1g/kg.

Multiple doses may be required, repeated q4-6hrly x 4-6 times.

Forced alkaline diuresis.

Administer NaHCO3 (D5 with 80-150 meq/L of NaHO3 and KCI 20meq/L) to maintain Urine pH 7.5-8. It is important to avoid hypokalemia when we maintain an alkaline urine pH

Ensure urine output of at least 2ml/kg/hr

Monitor for and treat dyselectrolytemia

Extracorporeal therapy (ECTR):

The use of ECTR should be restricted to cases of severe long-acting barbiturate poisoning.

The indications for ECTR in this setting are the presence of prolonged coma, respiratory depression necessitating mechanical ventilation, shock, persistent toxicity, or increasing or persistently elevated serum barbiturate concentrations despite treatment with multiple-dose activated charcoal.

Intermittent hemodialysis is the preferred mode of ECTR, and multiple-dose activated charcoal treatment should be continued during ECTR

Cessation of ECTR is indicated when clinical improvement is apparent.

CHAPTER-4
BRONCHIOLITIS

Bronchiolitis is a clinical diagnosis, recognized as “a constellation of clinical symptoms and signs including a viral upper respiratory prodrome followed by increased respiratory effort and wheezing in children less than 2 years of age[11].

Diagnosis[12,13]:

 Diagnosis is based on clinical assessment. Lower respiratory tract symptoms and signs (cough, tachypnea, crepitations and wheezing) follow a viral prodromal phase (coryza/low grade fever)

Do not routinely perform a chest X-ray in babies or children with bronchiolitis, because changes on X-ray may mimic pneumonia and should not be used to determine the need for antibiotics.

 Indications for chest X- ray:

If respiratory distress is severe needing an ICU care Course is atypical

There is uncertainty about the diagnosis

 Measure oxygen saturation in every baby and child presenting with suspected bronchiolitis

 Do not routinely carry out blood gas testing in babies or children with bronchiolitis. Consider carrying out capillary blood gas testing in babies and children with severe worsening respiratory distress.

Differential diagnosis[14]:

Consider a diagnosis of pneumonia if the baby or child has:

• High fever (over 39°C) and/or

• Persistently focal crackles.

Consider diagnosis of viral-induced wheeze or early-onset asthma rather than bronchiolitis in older infants and young children if they have:

• Persistent wheeze without crackles or

• Recurrent episodic wheeze or

• Personal or family history of atopy.

Management[12,13]:

Adequate hydration:

Give fluids by nasogastric or orogastric tube in babies and children with bronchiolitis if they cannot take enough fluid by mouth.

Give intravenous isotonic fluids (see the NICE guideline on intravenous fluid therapy in children) to babies and children who:

• Do not tolerate nasogastric or orogastric fluids or

• Have impending respiratory failure.

Oxygen supplementation to babies and children with bronchiolitis if their oxygen saturation is:

• Persistently less than 90%, for children aged 6 weeks and over

• Persistently less than 92%, for babies under 6 weeks or children of any age with underlying health conditions.

 Consider continuous positive airway pressure (CPAP) in babies and children with bronchiolitis who have impending respiratory failure.

 High-flow nasal cannula (HFNC) oxygen may have a role as a rescue therapy to reduce proportion of those requiring intensive care.

Upper airway suctioning to be performed in children who have respiratory distress or feeding difficulties because of upper airway secretions or apnea.

Do not use any of the following to treat bronchiolitis in babies or children:

• Chest physiotherapy

• Antibiotics

• Hypertonic saline

• Adrenaline (nebulized)

• Salbutamol

• Montelukast

• Ipratropium bromide

• Systemic or inhaled corticosteroids

• A combination of systemic corticosteroids and nebulized adrenaline.

• Steam inhalation

• RSV polyclonal immunoglobulin/palivizumab (no role in acute management but useful in prophylaxis)

Indications for admission[13]:

• A respiratory rate of over 60 breaths/minute

• Difficulty with breastfeeding or inadequate oral fluid intake (50% to 75% of usual volume, taking account of risk factors and using clinical judgement)

• Clinical dehydration

• Persistent oxygen saturation of less than 92%, when breathing air

• Consider ICU admission in case of apnea (observed or reported), sick looking child, severe respiratory distress (grunting, marked chest recession, or a respiratory rate of over 70 breaths/minute) and central cyanosis.

CHAPTER-5

CAMPHOR POISONING

Source[14]:

It is commonly seen in household items, including vaporized or topical cold preparations (for example Vicks Vaporub is 4.18% camphor and Tiger Balm), topical musculoskeletal anesthetics preparations (liniments), moth repellents, for performing rituals in religious ceremonies, and in antimicrobial preparations.

Camphor is also used in the Indian household in the form of small cubes for its fragrance.

Toxicity[14,15]:

 Major systemic toxicity has not been reported with ingestion of up to 30 mg/kg of camphor.

 The neurotoxic dose of camphor is 50 mg/kg body weight.

 The fatal dose is 500 mg/kg body weight.

 Camphor is a rapidly acting neurotoxin and can lead to excitation followed by depression of CNS. It is rapidly absorbed from GIT, leading to the rapid onset of action of toxic effects within 5–20 minutes, and peak effect occurs at 90 minutes.

 Seizures can occur after gastrointestinal, dermal, or inhalation exposures to camphor.

Clinical Presentation[14,16]:

 There may be a strong smell from the breath.

 Generalized warmth which progresses to burning sensation in pharynx and epigastrium and vomiting.

 Changes in mental status (confusion, restlessness, delirium, and hallucinations)

 Muscle twitching

 Myoclonus

 Ataxia

 Hyperreflexia

 Fasciculations

 Seizures (most common reported symptom)

Management 1,2,3:

 Support of Airway, Breathing and Circulation.

 Skin and ocular decontamination should be done by flushing with large amount of water.

 As camphor is rapidly absorbed from the stomach, gastric lavage and activated charcoal have a limited role in the management of camphor ingestion.

 Seizures should be managed with benzodiazepines (midazolam and lorazepam) and repeat doses may be needed. For uncontrolled seizures, a second anticonvulsant is needed, such as phenobarbitone or phenytoin.

Hemodialysis is not of much benefit.

CHAPTER-6

CORROSIVE POISONING

Caustic ingestions are seen most often in young children between one and three years of age and can cause severe acute injury and long-term complications, especially the development of esophageal strictures.

Accidental ingestion is common in younger children (< 5 years) while suicidal ingestion is more common in adolescents.

Source:

Toilet bowl cleaners, metal cleaners, bleach, drain cleaners, paint removers, hair straightener, car batteries, rust removers, laundry detergents, dish washer detergents.

PATHOPHYSIOLOGY[17]:

Alkali	Acids
✓ Alkaline agents tend to cause esophageal injury if the pH is above 11.5 to 12.5 via liquefaction necrosis. ✓ This type of injury leads to early disintegration of the mucosa, allowing deep penetration and even perforation	✓ Acids or corrosive agents tend to cause esophageal injury if the pH is less than 2, via coagulation necrosis. ✓ Esophageal injury from acids tend to be attenuated and perforation is less common because the coagulum that forms on the mucosal surface may limit deeper penetration of the caustic substance

CLINICAL MANIFESTATIONS[17]:

Gastrointestinal tract injury	✓ The most common symptom is dysphagia ✓ Patients may also present with drooling, retrosternal or abdominal pain, and hematemesis. ✓ Deep esophageal burns can be complicated by esophageal perforation, which can cause mediastinitis and the development of a tracheoesophageal fistula
Upper airway injury	✓ Symptoms suggesting upper airway injury include stridor, hoarseness, nasal flaring, and retractions.

Evaluation:

History and examination: History should seek to establish the timing of the exposure, including whether it was directly observed by a caretaker, and an estimation of the amount of the substance ingested.

Imaging

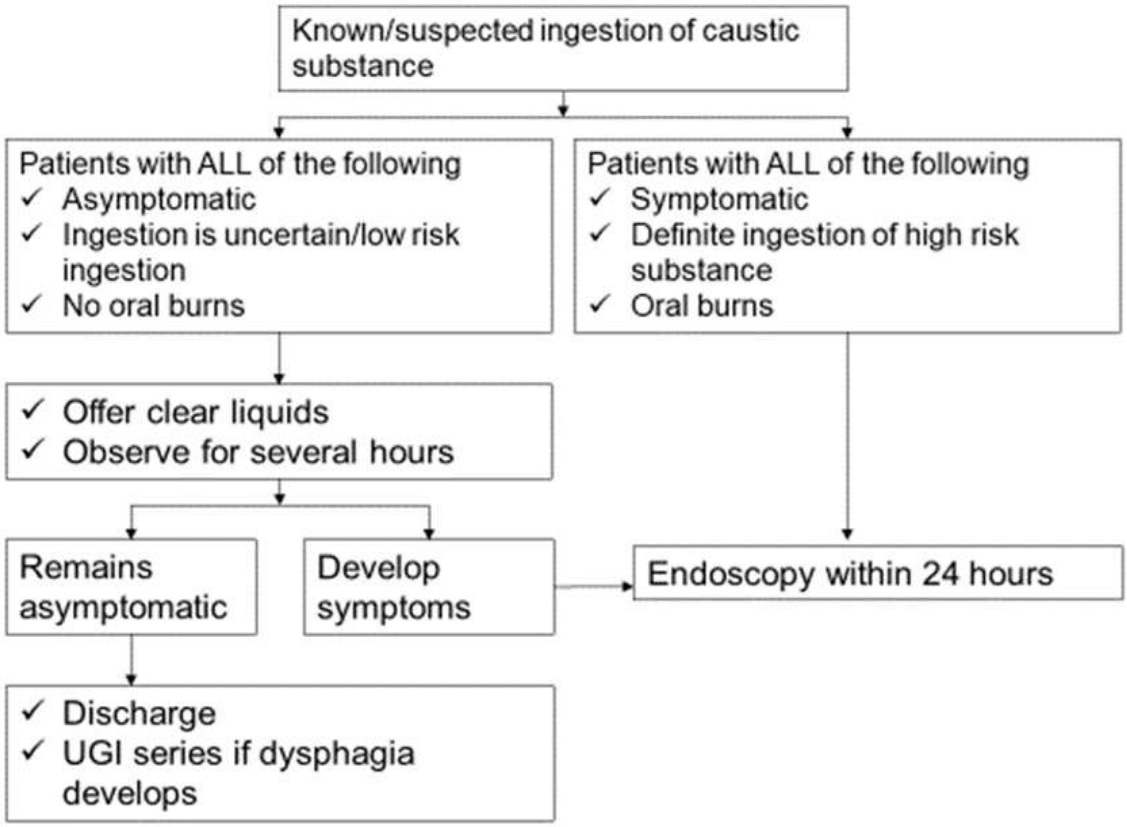
 A chest radiograph is appropriate in any patient with respiratory symptoms.

 Computed tomography (CT) or magnetic resonance angiography are sometimes needed to evaluate patients for the possibility of esophageal perforation with erosion into vascular structures, including an aorto-esophageal fistula.

Management[17,18]:

 The initial step in management of caustic ingestion is supportive care and close observation, with an emphasis on preventing vomiting, choking, and aspiration.

 Gastric decontamination – lavage, activated charcoal are contraindicated



Zargar classification for corrosive esophageal injury[17,18]

Injury	Findings	Management	
Grade 0	Normal mucosa	✓ Feed as tolerated ✓ PPI for 1 week if symptomatic ✓ UGI series in 2 to 3 weeks if dysphagia develops or for grade 2A lesions	
Grade 1 (superficial)	Mucosal edema and hyperemia		
Grade 2 (transmucosal)	Friability, hemorrhages, erosions, blisters, whitish membranes, and superficial ulcerations		
Grade 2A	No deep focal or circumferential ulcers		
Grade 2B	Deep focal or circumferential ulcers	✓ Place NG tube during endoscopy ✓ Corticosteroids 3 days course	✓ PPI for one week ✓ Antibiotics for 1 week ✓ UGI series on 2 to 3 weeks or if dysphagia develops any time ✓ Dilatation as needed if esophageal strictures develop
Grade 3	Areas of multiple ulceration and areas of brown-black or greyish discoloration suggesting necrosis	✓ Place NG tube during endoscopy or consider gastrostomy ✓ No corticosteroids	
Grade 3A	Small scattered areas of focal necrosis		
Grade 3B	Extensive necrosis		

UGI: upper gastrointestinal

PPI: proton pump inhibitors

NG: Nasogastric

CHAPTER-7

CROUP

Viral croup is an age-specific viral syndrome characterized by laryngeal and subglottic edema that primarily affects infants and children between 6 months and 3 years of age.[19]

The 3 hallmark signs of croup are hoarseness, cough, and stridor.[19]

Parainfluenza types 1, 2, and 3 are the most common viruses causing croup.[19]

Diagnosis [20,21]:

It is based on clinical assessment. Low grade fever, barking cough, variable degrees of stridor and respiratory distress are the common presenting manifestations.

Viral cultures and rapid antigen testing should be reserved for patients in whom initial treatment is ineffective.[22]

X- ray of neck/chest is not needed unless there is doubt about the diagnosis.

Westley Croup Score[20]

Clinical feature	Assigned score
Level of consciousness	
Normal, including sleep	0
Disoriented	5
Cyanosis	
None	0
With agitation	4
At rest	5
Stridor	
None	0
With agitation	1
At rest	2
Air entry	
Normal	0
Decreased	1
Markedly decreased	2
Retractions	
None	0
mild	1
moderate	2
severe	3
Total	

Differential Diagnosis[20]

Bacterial Tracheitis

Retropharyngeal/Para pharyngeal abscess

Epiglottitis

Foreign body

Management[20,22]:

Score total	***Severity***	***Description***	***Management***
≤2	Mild	✓ Occasional barky cough ✓ no stridor at rest ✓ mild or no retractions	✓ Home treatment –Symptomatic care including antipyretics and oral fluids ✓ Outpatient treatment – Single dose of oral dexamethasone 0.15 to 0.6 mg/kg (maximum 16 mg) or oral prednisolone (1 mg/kg)
3 to 7	Moderate	✓ Frequent barky cough stridor at rest ✓ mild to moderate retractions	✓ Single dose of oral dexamethasone 0.6 mg/kg (max 16 mg) ✓ Nebulized Epinephrine 0.5ml/Kg (max dose infants 2.5ml, children 5.0ml) ✓ Hospitalization is generally not needed but may be warranted for persistent or worsening symptoms after treatment with glucocorticoid and epinephrine
8 to 11	Severe	✓ Frequent barky cough ✓ stridor at rest ✓ marked retractions significant distress	✓ Single dose of oral/IM/IV dexamethasone 0.6 mg/kg (maximum 16 mg) ✓ Repeated doses of nebulized epinephrine may be needed ✓ O2 in non threatening manner ✓ Inpatient admission is generally required unless marked improvement occurs after treatment with glucocorticoid and nebulized epinephrine
≥12	Impending respiratory failure	✓ ↓consciousness ✓ stridor at rest ✓ severe retractions ✓ poor air entry ✓ cyanosis or pallor	✓ Single dose of IM/IV dexamethasone 0.6 mg/kg (maximum 16 mg) ✓ Repeated doses of nebulized epinephrine may be needed ✓ *O2 in non-threatening manner* ✓ PICU admission is generally required ✓ Consultation with anesthesiologist or ENT surgeon may be warranted to arrange for intubation in a controlled setting

Pediatric Pearls
▪ Always rule out a foreign body (FB) ▪ Since patients deteriorate rapidly, educate parents about worsening of symptoms. ▪ Steroids are useful. ▪ Nebulize Epinephrine at the right doses

CHAPTER-8

CYANOTIC SPELL

Hyper cyanotic (or "Tet") spells present as periods of profound cyanosis that occur because of episodes of almost total RVOT obstruction.

It is commonly seen with Tetralogy of Fallot; may also be seen in other Cyanotic Congenital heart diseases like tricuspid atresia with restrictive VSD, VSD with severe PS.

Precipitating factors[23]:

- Sympathetic stimulation, including pain and anxiety (e.g., during venipuncture)
- Exercise
- Breath holding or Valsalva maneuver
- Crying, feeding, and defecation
- Vasodilatation and decrease in SVR (e.g., hot baths)
- Hypoxia
- Hypercarbia
- Acidosis
- Induction of anesthesia
- Sympathomimetic drugs

Common age of Presentation[25]:

Less than 2 years, peaking around 4-6 months

Clinical presentation[23]:

- Classically, the child becomes restless, agitated, and breathless.
- Older children may squat into a knee-to-chest position as a learnt mechanism to increase SVR and consequently reduce right-to-left shunt.
- The child will be cyanosed and tachycardic.
- A reduced murmur may be heard because of reduced blood flow through the RVOT.
- In severe attacks, the conscious level may fall or the patient may have a seizure.

Treatment[24,25]:

Management is aimed at improving oxygenation, decreasing obstruction to right ventricular outflow, increasing pulmonary blood flow and systemic vascular resistance.

INTERVENTION	Mechanism of action
knee-chest position	Increases systemic vascular resistance (SVR), which promotes movement of blood from the RV into the pulmonary circulation
Oxygen – Nonthreatening manner	Pulmonary vasodilator and a systemic vasoconstrictor.
Intravenous (IV) fluid bolus (normal saline 10 to 20 mL/kg)	Fluids improve RV filling and pulmonary flow.
IV morphine (0.1 mg/kg per dose) or intranasal fentanyl or midazolam	Reduces hyperpnea
IV beta blocker (e.g., propranolol 0.1 mg/kg per dose or esmolol 0.1 mg/kg per dose). If single doses are ineffective, a continuous IV infusion of esmolol (50 to 75 mcg/kg/min) can be provided.	relaxation of the RVOT with improved pulmonary blood flow.
Sodium Bicarbonate 1-2meq/kg IV	Corrects established acidosis
IV phenylephrine (bolus dose of 5 to 20 mcg/kg per dose followed by continuous infusion)	increases systemic afterload which promotes RV flow into the pulmonary circulation rather than the aorta.
Ketamine 0.25-1mg/kg IV	Increases systemic vascular resistance and cause sedation

If the spell does not abort, the child needs to be shifted to the PICU for esmolol/propranolol and phenylephrine infusion and plan for emergency shunting procedure.

CHAPTER-9

DIABETIC KETOACIDOSIS

Diagnose DKA in children and young people who have.

 Acidosis (indicated by blood pH below 7.3 or plasma bicarbonate below 15 mmol/litre) and

 Ketonemia (indicated by blood beta-hydroxybutyrate above 3 mmol/litre)

Blood glucose levels are generally high (above 11 mmol/l) but children and young people with known diabetes may develop DKA with normal blood glucose levels.

Clinical Presentation[26]:

Clinical History:

 Polyuria

 Polydipsia

 Weight loss

 Abdominal pain

 Weakness

 Vomiting

 Confusion

Clinical Signs:

 Clinical dehydration

 Deep sighing respiration (Kussmaul)

 Smell of ketones

 Lethargy, drowsiness

Management:

General resuscitation: Airway, breathing and circulation

Initial fluid bolus

 All children and young people with mild, moderate or severe DKA who are not shocked and are felt to require IV fluids should receive a 10 ml/kg 0.9% sodium chloride bolus over 60 minutes.

 Patients with shock require appropriate restoration of their circulation and circulatory volume. Shocked patients should receive a 20 ml/kg bolus of 0.9% saline over 15 minutes.

 Following the initial 20 ml/kg bolus shocked patients should be reassessed and further boluses of 10 ml/kg may be given if required to restore adequate circulation up to a total of 40 ml/kg at which stage inotropes should be considered.

 Once circulating blood volume has been restored and the child adequately resuscitated, calculate fluid requirements as follows:

 Requirement = Deficit + Maintenance

Assume a 5% fluid deficit in children and young people in mild DKA (indicated by a blood pH 7.2-7.29 &/or bicarbonate <15)
Assume a 7% fluid deficit in children and young people in moderate DKA (indicated by a blood pH of 7.1- 7.19 &/or bicarbonate <10)
Assume a 10% fluid deficit in children and young people in severe DKA (indicated by a blood pH <7.1 &/or bicarbonate <5)

 Maintenance fluid volumes should be calculated using the Holliday – Segar formula

 The deficit should be replaced over 48 hours alongside appropriate maintenance fluids

 Hourly rate = ({Deficit – initial bolus} / 48hr) + Maintenance per hour

 Use 0.9% sodium chloride with 20 mmol potassium chloride in 500 ml (40 mmol per litre) until blood glucose levels are less than 14 mmol/l.

 Once blood glucose < 14mmol add 5% glucose to 0.9% sodium chloride with 20 mmol KCl per 500 ml. Reduce insulin infusion to 0.05 units/kg/hr

Insulin:

 Once rehydration fluids and potassium are running, blood glucose levels will start to fall. There is some evidence that cerebral oedema is more likely if insulin is started early. Do not give bolus doses of intravenous insulin.

 Therefore, start an intravenous insulin infusion 1-2 hours after beginning intravenous fluid therapy

 Use a soluble insulin infusion at a dosage between 0.05 and 0.1 units/kg/hour.

Bicarbonate:

 Do not give intravenous sodium bicarbonate to children and young people with DKA. Only consider bicarbonate if there is life threatening hyperkalemia or in severe acidosis with impaired myocardial contractility.

Cerebral Oedema:

Immediately assess a child or young person with DKA for suspected cerebral oedema if they have any of these early manifestations:

- Headache
- Agitation or irritability
- Unexpected fall in heart rate
- Increased blood pressure

If a child or young person develops any of these signs –

- Deterioration in level of consciousness
- Abnormalities of breathing pattern, for example respiratory pauses &/or drop in SaO2.
- Oculomotor palsies
- Abnormal posturing
- Pupillary inequality or dilatation.

Treat them Immediately for cerebral oedema using the most readily available of

- Hypertonic saline (2.7% or 3% 2.5-5 ml/kg over 10-15 minutes) or
- Mannitol (20% 0.5-1 g/kg over 10-15 minutes)
- Neuro-protection bundle
- In addition, fluids should be restricted to ½ maintenance rates
- Inform pediatric intensivist immediately.

CHAPTER-10

DIARRHEA WITH DEHYDRATION

 Acute diarrhea is the second leading cause of under-five mortality in India.

 It is defined as the passage of frequent watery stools (>3/24 h). Recent change in consistency of stools is more important than frequency.

 Acute diarrhea is caused by variety of viral, bacterial and parasitic agents. The common ones are: Rotavirus, E. coli, Shigella, Cholera, and Salmonella. Campylobacter jejuni, Giardia and E. histolytica are also not uncommon

Clinical Evaluation[27]:

The objectives of initial clinical evaluation of the patient in emergency department are:

 Assessment of the severity of the illness, grade of dehydration and rehydration needs.

 Identification of likely causes on the basis of the history and clinical findings.

A rapid assessment of airway, breathing and circulation should be done in all children at presentation. Tachycardia, prolonged capillary refill time, decreased urine output, altered mental status and hypotension indicates presence of shock and calls for oxygen, and fluid resuscitation with 20 ml/kg normal saline bolus.

Management[27,28]:

Classification	No Dehydration	Some dehydration	Severe dehydration
Clinical features	No signs of dehydration	Two or more of the following Restless irritable Sunken eyes Very thirsty Skin pinch goes back slowly	Two or more of the following Lethargy Coma Sunken eyes Not able to drink water Skin pinch goes back slowly
Investigations			Blood urea nitrogen Serum creatinine Serum electrolytes complete blood count Urine and stool tests as appropriate
Rehydration therapy	none	ORS 50 – 100ml/kg ORS over 3 to 4 hours	RL/NS in 20ml/kg body weight till perfusion and

			mental status improves then administer 100ml/kg of ORS over 4 hours or 5% dextrose with ½ saline IV at twice the maintenance fluid rate
Replacement of losses	<10kg- 60 to 120ml of ORS/stool >10kg- 120 to 240ml of ORS/stool	<10kg- 50 to 100ml of ORS/stool >10kg- 100 to 200ml of ORS/stool	<10kg- 50 to 100ml of ORS/stool >10kg- 100 to 200ml of ORS/stool If unable to drink, administer same volume via NG or IV 5% dextrose with ½ saline in same dose
Nutrition	Continue breast feeding Age-appropriate normal diet Assure adequate calorie intake	Continue breast feeding Age-appropriate normal diet Assure adequate calorie intake	Continue breast feeding Age-appropriate normal diet Assure adequate calorie intake
Zinc therapy	Zinc supplements are given for 10–14 d during and after diarrhea in the doses of 10 mg/kg for infants < 6 month of age and 20 mg/d for the children >6 month of age		
Antimicrobial therapy	✓ Diarrhea with clinical signs of sepsis ✓ Diarrhea in a child with severe malnutrition ✓ Neonates and very young infants (< 3month) with fever (> 38.5°C) ✓ Dysentery (bloody stools) and diarrhea during outbreak of shigellosis		

CHAPTER-11

DISC BATTERY INGESTION

 An increasing number of button battery (BB) ingestions has been described worldwide, mainly because of the wide abundance of batteries in consumer electronics.

 Recognizing BB ingestion is very important because of the extremely narrow 2-hour time window to remove BB impacted in the esophagus.[29]

Clinical features:

 Presenting symptoms differ according to the impaction site. Most witnessed ingestions present with acute gastrointestinal or respiratory symptoms, such as vomiting, drooling, dysphagia, odynophagia, irritability, coughing, stridor, and shortness of breath.

 Illustratively, most complications occur after unwitnessed ingestions leading to delayed diagnosis, as symptoms are variable and nonspecific.

 Esophageal battery impaction has the highest risk of complications, especially in children <6 years of age and in batteries >20 mm in diameter.

Investigations:

 Two-view (anterior-posterior and lateral) X-ray is paramount to diagnose BB ingestion and confirm its location.

 Contrast studies with CT scanning (or MRI scanning after battery removal only) are necessary to identify complications, such a mediastinitis, fistulas, and spondylodiscitis.

 In delayed diagnosis of an esophageal impaction (first confirmation of the BB on X-ray >12 hours after ingestion or time point of removal >12 hours after ingestion) regardless of symptoms (serial) CT/MRI scans of the chest and neck should also be considered as the BB may have been lodged in the esophagus previously.

Management:

Esophageal and Airway Impaction:

 When the battery is located in the esophagus, immediate endoscopic removal is necessary, if possible, within 2 hours of ingestion.

 If the ingested battery is located in the airway or in the gastrointestinal tract above the clavicles, an ENT doctor should be consulted to remove objects from the upper airways or upper part of the esophagus by rigid endoscopy.

Location Beyond the Esophagus

 Once the BB passed the esophagus almost three-quarters of ingested batteries pass spontaneously within 4 days. According to the NASPGHAN guideline, removal is, therefore, advised if a BB is still in the stomach after 2 to 4 days.

 ESPGHAN recommends that once the BB has passed the esophagus, asymptomatic cases should be followed-up after 7 to 14 days with an X-ray to confirm passage unless the battery has been noticed in the stools by the parents. Only if the battery still has not passed the stomach by 7 to 14 days, endoscopic removal is necessary as by then the chance that it will pass spontaneously is expected to be minimal.

 Removal of gastric BB is necessary in symptomatic cases, in case of co-ingestion with a magnet or in delayed diagnosis.

pH Neutralization Strategies to Mitigate Injury Progression After Ingestion:

 Mitigation strategies with honey and sucralfate can be considered in specific cases while waiting for endoscopy but should not delay it.

 early and frequent ingestion of honey, and if available, sucralfate in the clinical setting may have the potential to reduce injury severity and improve patient outcomes.

 Another mitigation strategy is neutralization of accumulated tissue hydroxide through acetic acid irrigation immediately. following battery removal and may be considered an option. This can be done with 50 to 150 mL 0.25% sterile acetic acid and should only be considered if signs of perforation are absent.

CHAPTER-12

GENERAL APPROACH TO A CHILD WITH POISONING

Poisoning is a commonly encountered pediatric emergency.

Recognition of the Child with Poisoning

• Acute onset of symptoms in a previously well child.

• Circumstantial evidence- empty bottles, tablets, spillage of household chemical near child.

• History may be available in a few cases

Management

1. Cardiopulmonary assessment and support of Airway, Breathing and Circulation.
2. Prevention or reduction of absorption
3. Enhancement of excretion
4. Supportive treatment
5. Administration of antidote

1. Cardiopulmonary assessment and support of Airway, Breathing and Circulation.

Initial management consists of stabilization of Airway, Breathing and Circulation.

Indications for RSI

• Depressed sensorium GCS < 8 or fall in GCS by 2 from the time of initial assessment.

• Absent / poor cough & gag reflex.

• Hypoventilation.

• Fluid/catecholamine unresponsive shock.

• Severe metabolic acidosis.

Sensorium needs to be assessed by GCS or AVPU at hourly intervals until the situation is under control.

Papillary size and reaction need to be checked

Check blood sugar (bedside)

If hypoglycemic – treat with 10% dextrose 5ml/kg bolus.

If bedside sugar is not available – trial of 10% dextrose 5ml/kg can be given in all patients with altered sensorium.

In all patients with depressed sensorium +/- depressed respiration, a trial of IV Naloxone 0.1mg/kg (max 2mg can be given).

Expected response – increase in rate & depth of respiration, improvement in sensorium.

2. Prevention / Reduction of Absorption

a) Skin Decontamination

• Particularly in organo phosphorous poisoning.

• Remove clothes.

• Clean skin / eyes with copious amounts of water.

• Medical personnel to take appropriate precaution by wearing gloves, protective clothing while handing.

b). Gastric Decontamination

i. Gastric Lavage

Useful only if patient arrives within 1 hour of ingestion of toxin.

Contra Indicated

• If Protective airway reflexes are lost

• Hydrocarbon/Corrosive poisoning

• Injury to GI Tract

Method

• Large bore single lumen (24-32f) NG/OG tube.

• After confirming position, normal saline (NS) 10-15ml/kg to be instilled & withdrawn repeatedly until clear fluid is aspirated.

ii. Activated charcoal (AC)

Binds to toxin & reduces absorption.

Indications

In patients presenting up to 4 hours after toxin ingestion.

Not useful (PHAILS – Mnemonic)

- P - Pesticide (Rodenticide)
- H - Hydrocarbon
- A -Acid, Alkaline, Alcohol'
- I -Iron
- L -Lithium
- S -Solvents

Dose: 1g/kg

Administered as slurry via NG tube.

Ensure the airway is adequately protected.

In children with normal sensorium, can be taken orally as a slurry mixed with juice/ cool drink to improve palatability.

Contra Indication

Absent airway reflexes.

Not useful in poisons not absorbed by activated charcoal (PHAILS).

iii. Multiple doses Activated Charcoal

Indications

- Salicylates.
- Tricyclic anti-depressants.
- Sustained release / Enteric coated tablets.
- Substances with increased enterohepatic circulation – Phenobarbitone, Carbamazepine, Theophylline.

3. Enhancement of Excretion:

Whole Bowel Irrigation (WBI)

Indication – Iron poisoning, Sustained release preparations.

Dose – 30ml/kg/hr 9500ml/hr) of polyethylene glycol given through nasogastic tube

End Point – Clear effluent after several hours.

Contra – Indications –Absent airway reflexes, GI bleeds, bowel obstruction or perforation

4. Supportive Management

• Shock / hypotension'

• Electrolyte imbalance

• Metabolic disturbance

• Bleeding

• Liver failure

• Psychiatry referral where relevant

5. Antidotes commonly available

Drug	**Antidote and dosage**
Acetaminophen	N – acetylcysteine 150mg/kg over 15 min, 50mg/kg, over 4hrs, then 100mg/kg over 16 hrs/Oral-140mg/kg, then 70mg/kg every 4hrs for 17 doses.
Organophosphates	Atropine 0.05mg/kg doubled every 3-5min until symptoms resolve Pralidoxime if patient has muscle weakness, respiratory depression within 36 hrs of ingestion 25-50mg/kg over 30-60 min then 10-20mg/kg/hr.
Iron	Deferoxamine 15mg/kg/hr up to 6-8g/day
Opiates	Naloxone 0.05-0.1mg/kg titrated to symptom reversal.
Methemoglobinemia (Commonly seen with dapsone & INH)	Methylene Blue 1-2 mg/kg over 5 min
Valproic Acid induced. Hyperammonemia or elevated AST/ALT	L- carnitine 100mg/kg in for divided doses.

Medico legal notification needs to be done in all cases of poisoning – accidental or intentional.

Pediatric Pearls
Antidotes and Drugs used in poisoning. ▪ N – acetylcysteine ▪ Atropine ▪ Desferrioxamine

- Naloxone
- Methylene Blue
- L – Carnitine
- Activated Charcoal
- Polythylene glycol

Common house hold items of low toxicity

Children often ingest many household items kept within their reach due to their curious nature. Most of these unintentional ingestions are usually in small quantities only. Some of these are harmless and require only reassurance.

A list of substances that are harmless and require no treatment apart from reassurance is mentioned below.

- Bar soap, Shampoo, hand lotion, creams, detergents, toothpaste, shaving cream and lotion
- Clay, dehumidifying packets, crayon, candle, chalk
- Thermometer, pencil, Ink

Removal necessary large amount ingested for –

Colognes, after shave lotion, hair tonic, deodorants, hair dyes, >20match sticks

Devices used for CPAP delivery (Patient interface)

CHAPTER-14

HYPERTENSIVE CRISIS IN CHILDREN

The definition of HTN in the pediatric population has evolved with the development of evidence-based normative data based on age, sex, and height percentile.

Prehypertension[1,2]	**Pediatric HTN**[1,2]	**Stage 1 HTN**[1,2]	**Stage 2 HTN**[1,2]
SBP and/or DBP measurements that range from the 90th percentile to less than the 95th percentile.	SBP and/or DBP ≥95th percentile for sex, age, and height percentile on ≥3 separate occasions.	SBP and/or DBP measurements that range from the 95th percentile to 5 mmHg above the 99th percentile	SBP and/or DBP measurements that are >5 mmHg above the 99th percentile

Hypertensive crisis[1,2]**:** used to describe an acute elevation in BP that can cause rapid end-organ damage.	
Hypertensive urgency	**Hypertensive emergency**
✓ Elevated BP without the presence of acute target-organ damage. ✓ May manifest symptoms such as headache and nausea	✓ Elevated BP with acute target-organ injury. ✓ Organ injury occurs in the central nervous system, kidneys, or cardiovascular system

LAB investigations[30]**:**

Initial investigations in the ED depend on findings from the history and physical exam

 Urinalysis: Presence of hematuria and proteinuria

 Serum chemistries: blood urea nitrogen, creatinine for baseline renal function, as well as for evidence of hypokalemic alkalosis found in HTN associated with renal vein stenosis

 Compete blood count

 Electrocardiogram and chest X-ray should be considered to evaluate for signs of congestive heart failure or myocardial hypertrophy.

 Computed tomography of the head may be warranted in the presence of abnormal neurological findings.

Management 30,31:

 The majority of mild-to-moderate pediatric HTN seen in the ED does not require immediate intervention. These cases mandate close outpatient follow-up, evaluation, and management.

 Hypertensive crises, however, necessitate immediate intervention to safely and effectively lower the BP. Current recommendations advise no more than a 25% reduction of SBP in the first 8 hours, followed by a gradual return to normal BP over 26 to 48 hours.

 Hypertensive emergencies are most commonly treated with intravenous (IV) medications. This allows for easier titration and control of the rate of decrease in BP.

 Rule out elevated intracranial pressure before lowering BP.

Most Useful				
Drug	Class	Dose	Route	Comments
Esmolol	β-blocker	100–500 mcg/kg/min	IV infusion	Very short-acting—constant infusion preferred. May cause profound bradycardia. Produced modest reductions in BP in a pediatric clinical trial.
Hydralazine	Vasodilator	0.2–0.6 mg/kg/dose	iv, im	Should be given every 4 hours when given iv bolus. Recommended dose is lower than FDA label.
Labetalol	α- and β- blocker	bolus:0.2–1 mg/kg/dose up to 40 mg/dose infusion: 0.25–3.0 mg/kg/hr	iv bolus or infusion	Asthma and overt heart failure are relative contra-indications.
Nicardipine	Calcium channel blocker	1–3 mcg/kg/min	iv infusion	May cause reflex tachycardia.
Sodium nitroprusside	Vasodilator	0.53–10 mcg/kg/min	iv infusion	Monitor cyanide levels with prolonged (>72 hr.) use or in renal failure; or co-administer with sodium thiosulfate.
Occasionally useful				
Clonidine	Central α-agonist	0.05–0.1 mg/dose may be repeated up to 0.8 mg total dose	po	Side effects include dry mouth and sedation.
Enalaprilat	ACE inhibitor	0.05–0.1 mg/kg/dose up to 1.25 mg/dose	iv bolus	May cause prolonged hypotension and acute renal failure, especially in neonates.
Fenoldopam	Dopamine receptor agonist	0.2–0.8 mcg/kg/min	iv infusion	Produced modest reductions in BP in a pediatric clinical trial in patients up to 12 years.
Minoxidil	Vasodilator	0.1–0.2 mg/kg/dose	po	Most potent oral vasodilator; long-acting.
Isradipine	Calcium channel blocker	0.05–0.1 mg/kg/dose	po	Stable suspension can be compounded.

Pediatric Pearls
▪ Gradually reduce blood pressure ▪ Avoid sublingual Nifedipine. ▪ Aggressively evaluate for etiology and end organ damage ▪ Check the fundus

CHAPTER-15

IRON POISONING

Source: Iron capsules, liquid preparation or chewable iron tablets

Liquid formulations and chewable iron are not radiopaque.

Toxic dose: more than 60mg/kg of elemental iron.

Mechanism of Toxicity

Due to direct caustic effects on the GI mucosa & presence of free iron in the circulation.

Clinical Presentation

1. Stage of GI Symptoms (Less Than 6hrs)

 GI symptoms like vomiting, abdominal pain – early onset indicates severe toxicity.

 Doses of 30mg/kg of elemental iron could cause GI symptoms.

 If there are no GI symptoms up to 6hrs post ingestion, it is unlikely that there is significant toxicity.

2. **Stage of Relative Stability (6-24hrs)**

GI symptoms subside and child appears stable.

Subtle signs of mild GI bleeding, hyperventilation, prolonged CFT may go unnoticed.

3. **Stage of Shock (24-48HRS)**

This is the stage of significant shock, metabolic acidosis & even GI bleeding. Shock could be a combination of hypovolemic, distributive and myocardial components.

4. **Hepatotoxic Stage (may start after 2-3days)**

In this stage, features of liver failure are evident through elevated liver enzymes and prolonged prothrombin time.

5. **Stage of Gastric Scarring (2-6weeks) Post Ingestion**

Gastrointestinal strictures are formed.

Management

Investigations

X – Ray Abdomen: To look for concretion of iron capsules. (Presence of radiographic evidence and severe metabolic acidosis indicates significant toxicity).

 Serum Iron Levels : Through not readily available, can be used to predict outcome

<350mcg/dl –mild

350-500mcg/dl –moderate

>500mcg/dl – severe toxicity

 Total Blood Count – may reveal leukocytosis

 Blood sugar – usually more than 150mg/dl

 S-Electrolytes particularly serum HCO3

 BUN, Creatinine, LFT.

Treatment

Assess & stabilize Airway, Breathing and Circulation.

Decontamination

 NG or Orogastric Lavage: Limited by the tube size through which effective evacuation of iron concretions are not possible.

CHAPTER-16

KEROSENE POISONING

 Kerosene is an aliphatic hydrocarbon commonly used in households and industries, especially in developing countries.

 It is used in households for cooking, lighting, heating, and in paints and pesticides.

 Exposure occurs accidently in children when they consume the kerosene stored in juice bottles, beverages bottles, and colorful packing within their reach.

Mechanism of Toxicity[34]

 Due to its low viscosity, low surface tension, and high volatility, there are high chances of aspiration, which usually occurs at the time of ingestion (coughing and gagging) or vomiting (spontaneous or induced) after ingestion.

 Aspiration of even small amounts of kerosene (<1 mL) can lead to serious, potentially life-threatening toxicity.

 Kerosene once aspirated spread rapidly across the surfaces of airway and alveoli leading to inactivation of type II pneumocytes, and resulting surfactant deficiency, intra-alveolar hemorrhage, inflammation, and necrosis.

Clinical Features[33]

Respiratory system	Tachypnea, dyspnea, cyanosis, grunting, bronchospasm, intercostal retractions, use of accessory muscles On auscultation, decreased breath sounds, diminished resonance, bronchial breath sounds, crepitations
Central nervous system	Drowsiness, which is often mild and transient. Other signs of CNS impairment include restlessness, stupor, and convulsions, dizziness, euphoria, headache
Gastrointestinal system	Vomiting, abdominal pain, constipation, nausea, diarrhea and Malena
Fever	Fever, often seen within 30 minutes, is generally triggered by inflammatory response due chemical irritation. However, if the fever persists beyond 48 hours, bacterial infection should be considered

Investigations[34]:

Chest X ray:

 Indicated in all children with a history of kerosene ingestion and respiratory symptoms.

 Chest radiographs may be normal initially but may show abnormalities within 6 hours of aspiration in the majority of symptomatic cases.

 Typical radiographic findings are increased broncho vascular markings and infiltrate involving bibasilar and perihilar regions. Lobar consolidation, pneumothorax, pneumomediastinum, and pleural effusion are uncommon.

Arterial blood gas to know about oxygenation and ventilation status in children with respiratory distress as well as the need for escalation of respiratory support and response to it.

Management[34]:

 The mainstay of treatment for kerosene poisoning is supportive care.

 Remove the patient from exposure and remove soiled clothing

 In the case of accidental ingestion, the emesis and gastric lavage are contraindicated as there is a higher risk of aspiration than systemic absorption.

 There is no clear role of corticosteroids or prophylactic antibiotics. Antibiotics are reserved for children with definite evidence of infection.

 If aspiration pneumonitis develops, the respiratory treatment is supportive. The airway patency should be evaluated and established. Any child with respiratory symptoms (grunting, tachypnea, or cyanosis) should be started on humidified oxygen and requires an arterial blood gas analysis. Oxygen support, respiratory monitoring, intake and output monitoring, and supportive care are important management strategies. It is important to realize that children with respiratory compromise at presentation to the emergency department can suffer rapid deterioration.

 For children who have severe distress with hypoxemia unresponsive to supplemental oxygen and/or severe central nervous system involvement require early intubation and mechanical ventilation.

CHAPTER-17

NAPHTHALENE POISONING

Source[35,36]: Mothballs, Toilet bowl deodorizers.

 The naphthalene content of one mothball varies from 0.5 to 5 g.

 The fatal dose of naphthalene in children is unknown, but even one mothball can result in toxicity, and there are reports of deaths following the ingestion of naphthalene balls.

 The absorption of naphthalene from GIT is erratic in the pediatric population. Other routes of exposure are inhalation and skin contact.

Clinical Features[35]:

 Headache

 Vomiting

 Diarrhea

 Abdominal pain

 Fever

 Altered mental status.

 Significant clinical manifestations in naphthalene poisoning occur due to hemolysis leading to anemia, jaundice, hemoglobinuria, methemoglobinemia, and acute kidney injury.

 Skin exposure – dermatitis

Investigations[35]:

 Complete blood count, peripheral blood film for evidence of hemolysis

 Urine and plasma hemoglobin

 Lactate dehydrogenase (LDH)

 Renal and liver function tests

 Acid–base status

 Urine routine examination

 Methemoglobin levels

 G6PD levels need to be monitored in children with naphthalene poisoning.

Management[37]:

Specific Treatment –

 Specific treatment includes the use of methylene blue and exchange transfusion.

 Exchange transfusion is the treatment of choice in patients with G6PD deficiency as methylene blue itself may induce hemolysis and cause paradoxical methaemoglobinaemia in these patients.

 NAC may be used in the treatment of methaemoglobinaemia as a reducing agent especially in patients with G6PD deficiency.

Supportive Treatment –

 Supportive treatment to maintain the airway, breathing and circulation (which may include endotracheal intubation, mechanical ventilation and use of inotropes).

CHAPTER-18

ORGANOPHOSPHATES POISONING

There are 2 groups of organophosphorus compounds: organophosphates and carbamates.

Source:

Organophosphates: - malathion, parathion, HETP

Carbamate: Baygon, Carbofiran

Mechanism of Poisoning

Inhibition of acetylcholinesterase, thereby causing increased cholinergic activity.

Clinical syndrome[35]

1. Acute Toxicity:

Muscarinic effects	(SLUDGE), Salivation, Lacrimation, Urination, Defecation, Gastrointestinal cramps, emesis.
Nicotinic effects	Fasciculations, weakness, flaccidity.
CNS effects	Seizures, agitation, coma, delirium.
CVS	Bradycardia, heart block, cardiac arrest.
RS	Bronchorrhea, bronchospasm.
Eyes	Miosis, blurred vision.

2. Intermediate syndrome:

- Occurs 24-96 hours after exposure.
- Bulbar, respiratory, and proximal muscle weakness are prominent features.
- Generally, resolves in 1-3 weeks.

3. Organophosphorus Agent-Induced Delayed Peripheral Neuropathy (OPIDN):

- Usually occurs several weeks after exposure.
- Primarily motor involvement.
- May resolve spontaneously but can result in permanent neurologic dysfunction.

Management [36,37]:

Diagnostic evaluation of acute toxicity:

1. Atropine challenge if diagnosis is in doubt (1 mg IV in adults, 0.01 to 0.02 mg/kg in children)

 Absence of anticholinergic signs (tachycardia, mydriasis, decreased bowel sounds, dry skin) strongly suggests poisoning with organophosphate or carbamate

2. Draw blood sample for measurement of RBC acetylcholinesterase activity to confirm diagnosis

General

 Check airway, breathing, and circulation.

 Place patient in the left lateral position, preferably with head lower than the feet, to reduce risk of aspiration of stomach contents.

 Provide high flow oxygen, if available. Intubate the patient if their airway or breathing is compromised

 Effective skin & mucosal decontamination with copious amounts of water to prevent dermal absorption. May need to be repeated periodically, if dermal inoculation is the mode of poisoning.

Specific Management

1. Atropine:

 Dose 0.05mg/kg I.V. bolus to be given every 10-15 minutes, dose may be doubled as required) Reverses muscarinic effects only end point for administration: Drying up to secretions, Tachycardia is not a contraindication for atropine.

 Repeat atropine dose 0.05mg/kg after 5 to 10 min if no improvement after first dose. Repeat boluses until heart rate is appropriate for age and systolic BP >5th centile and chest are clear

 Once patient is stable, start infusion of atropine at approximately 20% of the cumulative bolus doses administered.

2. Pralidoxime:

 Cholinesterase reactivator

 Mechanism: regeneration of acetylcholinesterase

 Dose: 25-50mg/kg I.V over 20-30 min followed by infusion at 10 – 20mg/kg/hour in saline.

Continuing PAM infusion till atropine is no longer required for 12-24 hours and patient is extubated.

CHAPTER-19

OPIATE TOXICITY

Source: Cough syrups containing codeine, Lomotil & opiate containing analgesics

Clinical features[39]:

The classic findings of opioid toxicity are:

 Miosis

 Central nervous system (CNS) depression

 Respiratory depression

 Other findings, including hyporeflexia, hypothermia, dermal "track marks", flushing, pruritus, bradycardia, hypotension, or decreased bowel sounds.

System	Manifestations
Pulmonary	Respiratory depression, noncardiogenic pulmonary edema
Cardiovascular	Orthostatic hypotension, peripheral vasodilation, dysrhythmias
Neurologic	Analgesia, euphoria, dysphoria, miosis, mydriasis, seizures
Gastrointestinal	Nausea, vomiting, constipation
Dermatologic	Flushing, pruritus
Reproductive	Amenorrhea, anovulation

Ancillary studies[39]:

Children and adolescents with altered mental status and suspected opioid intoxication warrant the following studies:

 Blood glucose.

 Pulse oximetry and, if respiratory status does not improve after naloxone administration, blood gas measurement.

 Serum acetaminophen concentration if suicidal intent or if ingestion of combination agents (e.g., acetaminophen with hydrocodone or oxycodone)

 Serum ethanol level (adolescents with recreational or suicidal overdose)

 Electrocardiogram, especially in patients with methadone overdose to evaluate for prolonged QTc.

 Chest radiograph, in patients with persistent respiratory findings suggestive of pulmonary aspiration or noncardiogenic pulmonary edema.

 Rapid urine pregnancy test in post menarcheal girls

 Urinalysis, serum electrolytes, blood urea nitrogen, creatinine, and creatine kinase in patients at risk for rhabdomyolysis (e.g, prolonged immobilization, muscular rigidity)

Management[38,39]

 Support Airway, Breathing and Circulation

 Continuous monitoring of SpO2, ECG and q15-30 min BP checks

 Gastric lavage only if the child presents within 1 hour of consumption

 Activated charcoal should be given only if patient is seen within 4 hours of ingestion & af6er protecting the airway.

Specific Antidote:

Give naloxone for deep coma and respiratory depression as follows:

 Children <20 kg: 0.1 mg/kg IV or IO (maximum 2 mg per dose) except neonates

 Children over 20 kg: 2 mg IV or IO

 Adolescents suspected of opioid dependents and overdose: 0.04 to 0.4 mg per dose repeated every 3-5 minutes and titrated to patient response

 If no effect, repeat the naloxone dose every one to two minutes to a maximum total dose of 10 mg

 Patients with recurrent toxicity may receive additional bolus doses or a continuous naloxone infusion. Begin the infusion rate at 2/3 of the total dose of naloxone needed to restore breathing, delivered every hour.

If the patient develops respiratory depression despite the naloxone infusion (this may happen 20 to 30 minutes after starting infusion) administer a naloxone bolus (using half the original effective bolus dose) and repeat, if necessary, until adequate ventilation returns, then increase the infusion rate.

If the patient develops signs of opioid withdrawal, stop the infusion. If respiratory depression returns, start the infusion at half the original rate.

CHAPTER-20

PARACETAMOL TOXICITY

Toxic dose[40]:

>150 mg/kg in 24 hours	Child
>7.5 gm in 24 hours	Adult / adolescent

Clinical features[40, 41]:

Mean onset of symptoms is – 6 hrs. after ingestion.

Stage	Time frame	Symptoms
1	0.5 – 24 h	Unspecific symptoms like diarrhea, nausea, malaise
2	24 – 48 h	Abdominal pain, abnormal laboratory values
3	3 – 4 days	Peak of hepatic dysfunction, stage 1 symptoms may reappear
4	4 – 14 days	Recovery or complete liver failure

Investigations[41]:

• All patients need a baseline measurement

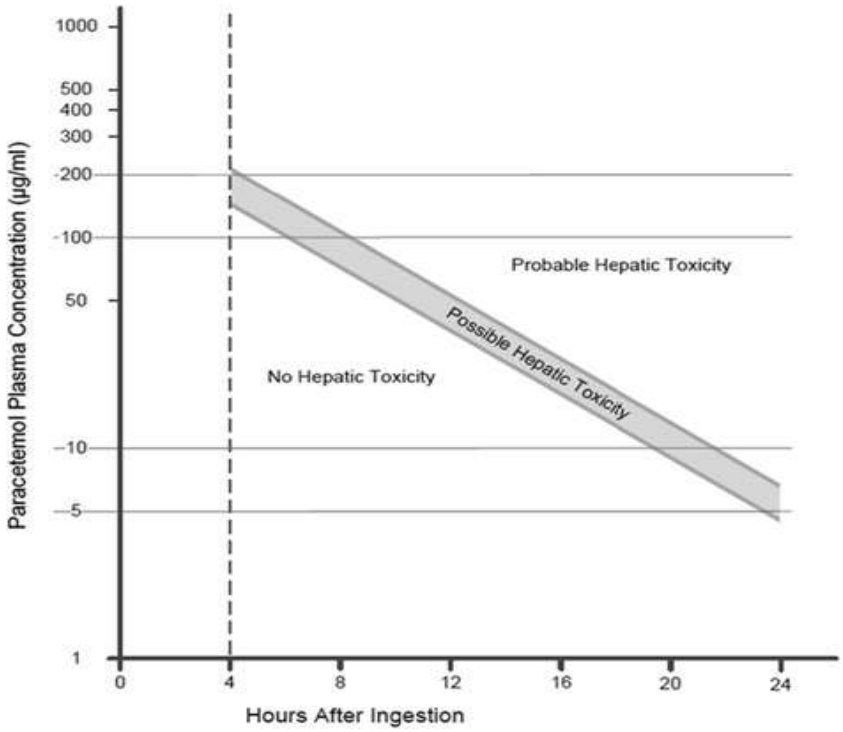
- Electrolytes
- Liver enzymes
- Renal functions,
- Markers of coagulopathy
- ABG analysis

• If the patient was asymptomatic at presentation, the routine baseline investigations should be repeated after 48 hours.

• In symptomatic patients, they are done as and when required depending on the clinical status.

• Blood paracetamol level estimated after 4 hours of ingestion of a large single dose of paracetamol should be plotted against.

Rumack–Matthew nomogram to estimate the risk of toxicity and need for NAC (in asymptomatic patients).



Management[41, 42]:

 Assess and support airway, breathing, circulation – Sensorium is usually normal in paracetamol poisoning.

 Gastric lavage only if child presents within 1 hour of ingestion.

 Activated charcoal 1 g/kg up to 4 hrs. after ingestion.

Specific Antidote

N – acetyl cysteine Oral or IV

Indication

• Patients who have ingested >150 mg/kg paracetamol are symptomatic.

• If the blood paracetamol level at 4 hours indicates toxicity(140–150 µg/mL).

• Patients with delayed presentation (>24 hours after ingestion) and laboratory evidence of hepatotoxicity and a history of excessive acetaminophen ingestion.

Time of administration:

Ideally within 8 hrs. after ingestion. However, it can still be effective days after the ingestion, when patients are already in hepatic failure and acetaminophen levels are no longer detectable.

Oral administration:

140 mg/kg first dose followed by 70 mg/kg every 4th hourly for 17 doses.

IV – 21 hr. regimen:

	<20kg	20-40kg
Loading dose	150 mg/kg in 3 mL/kg of diluent given IV over 60 minutes	150 mg/kg in 100 mL of diluent given IV over 60 minutes
Second dose	50 mg/kg in 7 mL/kg of diluent given IV over 4 hours	50 mg/kg in 250 mL of diluent given IV over 4 hours
Third dose	100 mg/kg in 14 mL/kg of diluent given IV over 16 hours	100 mg/kg in 500 mL of diluent administered over 16 hours

Adverse reaction with IV – Flushing, itching, hives to be treated with antihistaminic.

When to stop:

• Asymptomatic (e.g., no right upper quadrant pain)

• Acetaminophen concentration is nondetectable

• The serum transaminase activity is decreasing significantly (has decreased to the normal range or to <50 percent of the peak value)

King's College Hospital criteria for liver transplantation in paracetamol induced acute liver failure[43].

• Arterial pH <7.3 (irrespective of the grade of encephalopathy)

OR

• Grade III or IV encephalopathy AND

Prothrombin time >100 seconds (INR>6.5) AND

Serum creatinine >3.4mg/dL (301 µmol/L)

CHAPTER-21

POLYTRAUMA

The golden hour of stabilization:

Primary survey:

Includes stabilization Airway, Breathing and Circulation and disability (neurologic) assessment.

Airway and Breathing:

Maintain 'C' spine immobilization.

↓

Open airway by jaw thrust manoeuvres

↓

Administer O2 by face mask (if hypoxic)

Intubate (by RSI) if:

- GCS ≤ 8 or GCS falls by more than two from time of initial assessment
- Hypoxia
- Increased work of breathing
- Flail chest is present
- Protective airway reflexes are lost
- Decompensated shock persists despite conventional management.

Use an orogastric tube to deflate the stomach.

Drugs for intubation in the hemodynamically stable child:

Atropine (0.02 mg/Kg, max 1 mg)

Lidocaine (1 mg/Kg, if TBI is also present)

Fentanyl (1-2 µg/Kg)

Midazolam (0.1 mg/Kg) or Propofol (1-2 mg/Kg)

Paralyse with vecuronium (0.3 mg/Kg).

Drugs for intubation in the haemodynamically unstable child :

Atropine (0.02 mg/Kg),

Lidocaine 1 mg /Kg (if TBI is also present)

Ketamine 1-2mg/kg (in the absence of TBI) or

Fentanyl (1-2 µg/Kg)

Paralyse with Vecuronium (0.3 mg/Kg)

Circulation:

Obtain two large bore IV accesses

↓

Obtain samples for CBC, grouping and cross matching, PT, PTT (if bleeding) electrolytes, SGOT, SGPT, BUN, Se Creatinine

↓

If B. P < 5th centile, administer NS 20ml/Kg over 5-10 min and repeat x3 times till B.P improves.

↓

Consider blood loss (abdomen, retroperitoneum, pelvis, thigh, chest) if

B.P does not improve and transfuse PRBC 20ml/Kg

↓

Simultaneously start dopamine +/- noradrenaline infusion.

↓

Catheterise bladder if there is no blood at urethral meatus

Disability:

Neurologic assessment is made initially by the GCS score and periodically reassessed; if <8, the child needs intubation.

If TBI is suspected, non contrast CT scan needs to be done.

Secondary Survey:

Perform a detailed head to toe examination to asses extent of injuries.

Maintain 'C' spine immobilization.

Palpate for swelling and depression.

Control bleed

Look for bruising / swelling over:

- Chest (rib fracture/underlying lung contusion)
- Abdomen (liver/splenic laceration) Auscultate chest (air entry)
- Consider needle throracostomy if pneumothorax is suspected

Auscultate abdomen (bowel sounds)

Look for blood at urethral meatus – if present do not catheterize bladder.

Choice of imaging

Chest X RAY – AP View

X – ray Neck – Lateral view

CT brain if TBI is suspected.

X – ray limb if fracture is suspected

Focused abdominal sonography in trauma (FAST), if there is doubt about Abdominal trauma and patient is too sick to be shifted to CT room.

CT abdomen with contrast is the imaging of choice when abdominal trauma is strongly suspected.

Shift the child for imaging only when Airway Breathing and Circulation have been stabilized.

Involve Pediatric surgeon, Neurosurgeon and Orthopedic surgeon in patient care after the child has been stabilized.

Pediatric Pearls
Maintain ‘c’ spine immobilization at all times

CHAPTER-22

PYRETHRIN & PYRETHROID POISONING

Pyrethrin and synthetic pyrethroids are active ingredients of common house hold insecticides and mosquito repellants.

Most cases of pyrethrin toxicity are allergic reactions.

Synthetic pyrethroids cause no allergic reaction, but can cause systemic toxicity following ingestion, dermal exposure and inhalation.

Commercially available pyrethroids and their clinical features[1]:	
✓ Bifenthrin ✓ Permethrin ✓ Phenothrin ✓ PR allethrin ✓ Resmethrin ✓ Tefluthrin	✓ Cyhalothrin ✓ Cypermethrin ✓ Deltamethrin ✓ Fenvalerate ✓ Fenpropathrin ✓ Tralomethrin
Type I syndrome "T syndrome"	**Type II syndrome "CS syndrome"**
✓ Severe fine tremor ✓ Marked reflex hyperexcitability sympathetic activation. ✓ Paresthesia (dermal exposure)	✓ Choreoathetosis ✓ Salivation ✓ Coarse tremor ✓ Increased extensor tone ✓ Moderate reflex hyperexcitability sympathetic activation

Diagnosis[44]:

The diagnosis of pyrethroid poisoning is based mainly on the clinical presentation and compound identification on the container brought by the patient or relatives.

Management[44,45]:

 Management is largely supportive and symptomatic because there is no available antidote.

 Optimization of the airway, breathing, and circulation is vital.

 Immediate decontamination of skin with soap and water can be considered; however, there is no evidence that it reduces toxicity.

 Gastric lavage is best avoided in the case of pyrethroid ingestion as the risk of aspiration pneumonia with the solvent is high. Evidence for the use of activated charcoal is limited; however, this can be considered if the patient is present within 1 hour of ingestion.

Systemic toxicity

- Essentially supportive management
- Benzodiazepines for tremors/ seizure.
- Topical Vitamin E for paraesthesia
- Supportive treatment for allergic reaction.

Majority of patients recover within 1-6 days without any neurological sequelae.

CHAPTER-23

RODENTICIDE POISONING

Historically, various ingredients like strychnine, heavy metals, yellow phosphorous, and zinc phosphide have been used as rat poison. The majority of rodenticides we encounter today. Contain warfarin type anticoagulants and long acting brodifacoum also called superwarfarins (100 times > potent and longer acting 16-69 days).

Mechanism of action

Blockage of production of Vitamin K depends on coagulation factors.

Signs and symptoms

Bleeding manifestations

Cardiac arrythmias, refractory shock or cardiac arrest

Onset of symptoms may be delayed (In case of thallium, arsenic, SMFA)

Investigations

Complete Blood count

PT, aPTT, INR – Repeat daily up to 75 hours, even if initial values are normal Blood grouping/cross matching.

Management

Support of Airway, Breathing and Circulation

Gastric decontamination with activated charcoal 1g/kg with adequate airway protection. (Preferably those who present to ER <1hour of ingestion)

Antidote – Vitamin K 0.3 mg/kg I.V over 5 minutes q8hrs (Max – 10mg) if coagulopathic (INR>2)

Prolonged Treatment with high dose may be needed. If frank bleeding – fresh frozen plasma transfusion needed.

CHAPTER-24

SALICYLATE POISONING

Source:

Tablets containing aspirin, oil of wintergreen

Clinical presentation

Nausea, vomiting, hyperpyrexia, hyperventilation, tinnitus, coma, convulsions.

Less common

- Respiratory depression
- Pulmonary edema
- Acute tubular necrosis
- Hepatotoxicity
- SIADH

Investigations

Arterial blood gas – in all suspected cases – Mixed respiratory alkalosis & metabolic acidosis

S. Electrolyte, BUN, S. Creatinine, S. Ketones

Salicylate level – Not available in most laboratories

Treatment:

Support Airway, Breathing and Circulation.

Gastric lavage – limited value

Activated charcoal – after protecting the airway

Multiple doses of activated charcoal may be required (every 4-6 hours up to 6 doses)

Increase elimination.

a. Alkalinixation 80-150 meq/l of sodium bicarbonate with 5% Dextrose to run at one and a half maintainance to maintain urine pH > 7. Add KCI to IVF 20-40 meq/l (to be checked every 6 hrs).

b. Dialysis

Indication for hemodialysis

- Renal failure
- Congestive cardiac failure
- Extremely elevated salicylate levels (> 100mg/dl)

CHAPTER-25

SCORPION STING – MANAGEMENT

46,47,48

Grade	Manifestation	Treatment
Grade I	✓ Isolated pain	✓ Ice packs ✓ NSAIDS ✓ Local anesthetic agents
Grade II	✓ Hypertension ✓ Sweating ✓ Vomiting ✓ Priapism ✓ Fever ✓ shivering	✓ Encourage IV fluids/IV fluids if oral intake is poor ✓ Antivenom ✓ Prazosin(30mcg/kg) stat; repeat as and when necessary ✓ Oral paracetamol if necessary
Grade III	✓ Cardiogenic shock ✓ Pulmonary edema ✓ Altered consciousness	✓ Oxygen therapy ✓ CVP guided fluid therapy ✓ Positive pressure ventilation if needed ✓ Antivenom ✓ Dobutamine ✓ Nitroprusside or nitroglycerine, if normotensive ✓ Prazosin(30mcg/kg) stat; repeat as and when necessary
Grade IV	✓ Tachycardia ✓ Hypotension with or without pulmonary edema	✓ Oxygen therapy ✓ CVP guided fluid therapy ✓ Positive pressure ventilation if needed ✓ Antivenom ✓ Dobutamine ✓ Prazosin(30mcg/kg) stat; repeat as and when necessary

Investigations:

Aimed at identifying complications

 Electrocardiogram

Chest X ray

 Echocardiography

 Biochemical abnormalities: these are elevated potassium, amylase, lactate dehydrogenase levels, raised liver enzymes, glucose and free fatty acids, reduced cholesterol and triglyceride levels.

Pediatric Pearls
Do not use sustained release, use only crossed tablets or Prazosin

CHAPTER-26

SEPTIC SHOCK

Definitions

Sepsis: Systemic inflammatory response syndrome caused by an infection.

Severe sepsis: Sepsis with at least one organ system dysfunction.

Septic shock: Sepsis with cardiovascular dysfunction causing inadequate supply & deficit of oxygen and metabolic substrates to tissues.

Management principles:

 Document baseline observations and continue ongoing monitoring to assess the response to treatment.

 Provision of supplemental oxygen by providing appropriate airway and ventilatory support.

 Stabilization of cardiovascular function with fluid resuscitation to optimize preload and appropriate inotropic and vasoactive therapy to optimize perfusion pressure.

 Treatment of underlying cause (antimicrobial therapy to cover likely pathogens, drainage of pus wherever appropriate).

Steps in management of child with septic shock:

1. Recognize septic shock and classify the physiological state:

Clinical definition:

Presence of sepsis with any of the two signs of poor systemic perfusion mentioned below

a. Decreased pulse volume (week or absent dorsalis pedis pulse)

b. Prolonged capillary refill time (> 3 sec)

c. Central (rectal/oral) to peripheral (toe skin) temperature defference > 3° C

d. Oliguria (< 1ml/kg/hour)

e. Unexplained metabolic acidosis or increased lactate (> 2mmol)

Hypotension – Systolic BP <5th percentile for age : < 70mmHg in infants; 70+ (age in years x 2) after one year of age. Hypotension occurs late in children with septic shock

2. Therapeutic end points and monitoring parameters

Breathing

 Normal respiratory rate for age

 Normal work of breathing (In the absence of pneumonia, lung pathology)

 SpO2 > 92%

Circulation

 Normal heart rate for age

 Normal pulses with no difference between central and peripheral pulse

Warm and pick extremities

 Capillary refill time < 2 s

 Normal range of systolic BP for age with normal pulse pressure

Renal

 Urine output - > 1ml/kg/hour in children upto 50kg or 30-50 ml/hr in children weighing greater than 50kgs

Neurological.

 Return baseline mental status (AVPU scale) in a child who is not intubated and sedated.

 Brisk response to light and equal pupils – in a sedated or paralyzed child.

3. Emergency department management of septic shock (Flow Chart – 1)

Emergency management – Golden hour of sepsis management

a. Recognition of shock state and providing respiratory support (0.5 min)

b. Establishment of intravenous/intraosseous access and fluid resuscitation to Optimize preload (5-40min)

c. Vasoactive drug therapy and Optimization of perfusion pressure (20-60 min)

d. Correction of metabolic impairments (20-60 min)

 The first dose of broad-spectrum antibiotic has to be given during the golden hour of resuscitation. Commonly used antibiotics include third-generation cephalosporin such as Ceftriaxone and aminoglycoside such as Amikacin for community acquired infections and immunocompromised patients to be treated accordingly.

 While vasoactive drugs are instituted for restoration of cardiac output and perfusion pressure, hypoglycemia and hypocalcemia, both of which might impair cardiac performances should be corrected.

 Stress dose Hydrocortisone 50 mg/ m2/dose stat followed by 50 mg/m2/day in 4 divided doses should be given in catecholamine resistant shock.

CHAPTER-27

SNAKE ENVENOMATION

Prehospital care

RIGHT

R: Reassure

I : Immobilise the bitten extremity in a neutral position

G : Go to

H : the Hospital

T : Tell the doctor about – ptosis, gum bleeds, abdominal pain (Krait bites) Constriction bands, pressure immobilization are not recommended.

If tourniquet has been applied, do not release it immediately.

Supportive care:

Support of Airway, Breathing and Circulation (the general indications for intubation hold good). Continuous monitoring of HR, SpO2, breathing pattern and Q15min-Q30min B.P checks are needed.

Investigations to be sent:

Complete blood count, bleeding/clotting time, PT/PTT, BUN, Se. creatinine, grouping and cross matching, SGOT/SGPT.

Indications for Anti Snake Venom (ASV)

Preferable administered within 4 hours of the bite. But can be given up to 96 hours of the bite.

Severe local symptoms:

Rapid spread of the swelling, crossing a joint or involving half of the bitten limb, in the absence of a torniquet.

Development of enlarged, tender lymph nodes, draining the bitten limb.

Systemic Envenomation

- Neurologic signs: Ptosis, external opthalmoplegia, altered sensorium, or seizures

- Cardiovascular abnormalities: Hypotension, shock, arrhythmias, abnormal ECG.
- Haemostatic abnormalities: Spontaneous bleeding manifestations, coagulopathy (>20 min whole blood clotting time or prolonged PT/PTT, thrombpcytopenia
- Acute renal failure, haemoglobinuria/myoglobinuria
- Respiratory distress, signs of upper airway obstruction.
- 8-10 vials of ASV are dissolved in 5-10ml/kg of isotonic fluid (150-300ml of NS or 5% Dextrose) and administered over 1-3 hours. If after 1-2 hours of administration, the child does not improve or worsens, a second and final dose should be given (max 25 vials).

Close monitoring for an anaphylactic reaction needs to be done. If any of these appears, ASV is discontinued and treatment for anaphylaxis (O2, fluid boluses, adrenaline 0.01mg/kg of 1;1000 solution IM, H1 and H2 blockers, IV steroids) started. Later ASV is restarted at a slower rate (of infusion) (1-2ml/kg/hr) and gradually increased.

Local wound management

The wound is cleaned thoroughly and left open. IV amoxicillin/clavulanate to be administered if the wound is open with severe local symptoms. Fasciotomy may be needed if there is evidence of compartment syndrome.

CHAPTER-28

STATUS EPILEPTICS

• Status epilepticus is defined as a seizure with 5 minutes or more of continuous clinical and/or electrographic seizure activity or recurrent seizure activity without recovery between seizures.[49]

• International League Against Epilepsy definition of status epilepticus indicates that emergency treatment of status epilepticus should be started at t1 and long-term consequences may occur.[50,51]

Status Epilepticus Type	**Time 1 (Treatment Started)**	**Time 2 (Consequences Expected)**
Tonic-clonic	5 min	30 min
Focal with impaired consciousness	10 min	>60 min
Absence	15 min	Unknown

• SE may also be an acute symptom of medical or neurologic disease. The more common examples of the latter includes[49]:

- Central nervous system infections (e.g., meningitis, encephalitis, intracranial abscess)
- Acute hypoxic-ischemic insult
- Metabolic disease (e.g., hypoglycemia, inborn error of metabolism, hepatic encephalopathy)
- Electrolyte imbalance (hyponatremia, hypocalcemia.)
- Traumatic brain injury
- Drugs, intoxication, poisoning
- Cerebrovascular event

Management[50]:

Stabilization phase (Seizure 0 to 5 minutes)	• Check and maintain ABC • Give high flow oxygen • Check blood glucose level • Establish IV access and collect samples for electrolytes, calcium, haematological investigations and toxicology screen	
Early status epilepticus (Seizure 5 to 30 minutes)	• IV LORAZEPAM: 0.1mg/kg (max 4mg/dose) slowly over 2 to 5 minutes • IV DIAZEPAM: 0.2mg/kg (max 10mg/dose) slowly over 2 to 5 minutes	• If no IV access, consider • INTRANASAL MIDAZOLAM 0.2mg/kg (max 5mg/dose) • PR DIAZEPAM 0.5mg/kg (max 20mg/dose)

		• IM MIDAZOLAM 0.2mg/kg (max 10mg/dose)
Established status epilepticus (Seizure 30 to 60min)	• IV PHENYTOIN 20mg/kg (max 1000mg) infusion @ 1mg/kg/min with continuous cardiac monitoring • IV LEVETIRACETAM 20 - 60mg/kg (max 4500mg) @ 5mg/kg/min • IV SODIUM VALPROATE 20 - 40mg/kg (max 3000mg) @ 5mg/kg/min • IV PHENOBARBITONE 20 - 40mg/kg (max 1000mg) @ 2mg/kg/min	
Refractory status epilepticus (Seizure more than 60min)	• RAPID SEQUENCE INTUBATION • IV MIDAZOLAM 0.2mg/kg bolus F/B 1mcg/kg/min, increasing to 1 mcg/kg/min every 10 to 15 minutes up to maximum of 30mcg/kg/min • HIGH DOSE PHENOBARBITONE COMA • Others: INJ THIOPENTONE, SHORT TERM PROPOFOL, INJ KETAMINE	
Super refractory status epilepticus (Seizure more than 24 hours)	TOPIRAMATE INHALED ANAESTHETICS Ketogenic diet Epilepsy surgery Electroconvulsive therapy and deep brain stimulation	

CHAPTER-29

TRICYCLIC ANTIDEPRESSANTS POISONING

TCA are used as antidepressants, anti –migraine medicine, and in treatment of attention deficit hyperactivity disorder, cyclical vomiting, neuropathic pain, and nocturnal enuresis and sleep disturbances.

E.g. Imipramine, Amitriptyline, Mapritiline, Amoxapine, Doxapane

Mechanism of toxicity

- Direct myocardial depression
- Anticholinergic effect
- Depletion of Norepinephrine

Symptoms

- CVS

a. Dysrhythmia – Sinus tachycardia, tosades, Ventricular tachycardia

b. Condution delay

c. Hypotension

- CNS – Altered mental status, seizures
- Anti cholinergic toxicity – Altered mental status, hyperthermia, urinary retention, and paralytic ileus.
- Pulmonary toxicity – Acute lung injury.
- Acronym – feature 3C & A – Cardiac, coma, convulsion & acidosis.

Management

- Continuous ECG monitoring is mandatory.
- Support Airway, Breathing and Circulation

Gastric decontamination

Gastric lavage may be done upto 4 hours after ingestion, only after ensuring airway is protected.

Activated charcoal (AC)

 1 g/kg

 Multiple doses are indicated due to delayed gastric emptying.

Symptomatic management

 In symptomatic / unstable patients at presentation or with rapid deterioration, support airway by early intubation

Treat arrhythmias

 Mainstay of treatment – Sodium bicarbonate

 Initial 1-2 meq/kg bolus followed by infusion Sodium bicarbonate containing solution as maintenance fluid (Sodium bicarbonate 150 meq/l in 5% Dextrose. Add potassium 20-40 meq/l of IV fluid) until arrhythmias are corrected & ORS duration < 100 ms.

 Goal is to maintain pH > 7.45, Severe intoxication pH; 7.5 – 7.55.

 In arrhythmia unresponsive to Sodium bicarbonate, IV lignocaine or 3% NaCI can be considered.

Treatment of hypotension

 NS bolus of 10 ml/kg – NS bolus to be administered cautiously due to concomitant myocardial depression, risk of pulmonary edema and acute lung injury.

 Monitor for pulmonary edema.

 If fluid unresponsive add vasopressors like Norepinephrine and Epinephrine to maintain BP.

Treatment of Seizures

 Phenobarbitone to be used.

 Phenytoin is contra – indicated.

CHAPTER-30

TRAUMATIC BRAIN INJURY (TBI)

TBI is categorized by means of the Glasgow Coma Scale (GCS) as

Severe: GCS ≤ 8

Moderate: GCS 9-13

Minor: GCS 14 and 15

Minor Head Trauma:

The decision on imaging (Ct brain), observation in the ER, admission or discharge is based on the risk stratification.

High Risk (CT recommended)

- Altered mental status
- Focal neurologic findings
- Signs of depressed or basilar skull fracture by clinical examination
- Excessive irritability (inconsolable)
- Seizures
- Vomiting ≥ 5 episodes or ≥ 6 episodes per hour
- Loss of consciousness ≥ 1 minute

Intermediate Risk (Observation and/or CT scan)

- Vomiting 3-4 episodes from time of injury.
- Loss of consciousness ≤ 1 minute
- H/o lethargy or irritability, now resolved
- Behaviour not at baseline, reported by caretaker
- Non acute, skull fracture (≥ 24 hours old)
- Frontal, parietotemporal, occipital swelling ≥ 3cm in younger children (< 2 years)

Low Risk (Recommend observation either in ER or at home)

- Low energy mechanisms (fall ≤ 3 feet)
- No signs or symptoms ≥ 2 hours since injury

Older age (especially ≥ 2 years) is more reassuring.

Children with normal CT and/or normal examination can be discharged after 6 hours of observation. The possibility of delayed bleed should be emphasized and the family asked to return if any of the symptoms mentioned (vomiting, drowsiness/excessive irritability, seizures) appear.

Severe Traumatic Brain injury

The "Golden Hour" management is aimed at preventing and minimizing secondary insults to the brain. The initial priorities are management of the airway, breathing and circulation, simultaneously instituting measures to lower the ICP.

Airway and Breathing

Maintain 'C' spine immobilization at all times

Administer O2 by face mask and open airway by jaw thrust maneuver.

Intubate (by RSI) if:

- GCS < 8 or falls by more than two from the time of initial assessment.
- Hypoxic
- Respiratory efforts are poor.
- Protective airway reflexes are lost.

Drugs for intubation in the hemodynamically stable child:

Atropine (0.02 mg/Kg, max 1 mg)

Lidocaine (1mg/Kg)

Propofol (1-2 mg/Kg) or Midazolam (0.1 mg/Kg)

Fentanyl (1-2 µg/Kg) and

Paralyze with Vecuronium (0.3 mg/Kg)

Use an orogastric tube to deflate the stomach.

Ventilate at rates appropriate for age (ETCO2 35-40 mm Hg)

Hyperventilate only if signs of impending herniation are present, which includes.

- Unequal pupils.
- Midpoint fixed/dilated pupils.
- Decorticate/decerebrate posturing.

 Cheyne Stokes/irregular breathing /hyperventilation.

Circulation

Obtain two large bore IV access

↓

If B.P < 5th centile, administer 20 ml/Kg of NS over 5 -10 min and repeat x 3 times till B. P improves.

↓

Start dopamine +/- noradrenaline if B.P is low despite 3 fluid boluses. (Threshold for starting inotrops is low in TBI, to optimize Cerebral perfusion Pressure.

↓

Consider blood loss in polytrauma victims and transfuse PRBC (20 ml/Kg)

ICP Lowering Measures

Head end elevation to 30°

Provide adequate analgesia/sedation.

Lidocaine 1mg/Kg before any painful procedure.

Seizure control as per status epilepticus protocol

Ventilate at rates appropriate for age.

Hyperventilate (ETCO2 35-40 mm Hg) only if signs of impending herniation are present.

Hyperosmolar therapy: Mannitol 1ml/Kg of 20% solution, in haemodynamically stable children, 3% Nacl 5ml/Kg in hemodynamically unstable children, followed by infusion of the same at 0.5-1 ml/Kg/hour.

Shift the child for imaging only after airway, breathing and circulation are stabilized.

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