Poisoning

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When to suspect poisoning??

Any child who presents with unexplained symptoms including altered mental status, seizure, cardiovascular compromise, or metabolic abnormality should be considered to have ingested a poison until proven otherwise. The index of suspicion should be raised if the child is in the "at-risk" age group (one to four years of age) and/or has a previous history of ingestion or, in adolescents, a prior history of substance use disorder. A history and physical examination by someone who understands the signs and symptoms of various ingestions often provide sufficient clues to distinguish between toxic ingestion and organic disease.

Signs and symptoms of different poisons

TABLE 45.1 Historical and Physical Findings in Poisoning	
ODOR	
Bitter almonds	Cyanide
Acetone	Isopropyl alcohol, methanol, paraldehyde, salicylate
Alcohol	Ethanol
Wintergreen	Methyl salicylate
Garlic	Arsenic, thallium, organophosphates, selenium
Violets	Turpentine
OCULAR SIGNS	
Miosis	Narcotics (except propoxyphene, meperidine, and pentazocine), organophosphates, muscarinic mushrooms, clonidine, phenothiazines, chloral hydrate, barbiturates (late)
Mydriasis	Atropine, cocaine, amphetamines, antihistamines, cyclic antidepressants, PCP, LSD
Nystagmus	Phenytoin, barbiturates, ethanol, carbamazepine, PCP, ketamine, dextromethorphan
Lacrimation	Organophosphates, irritant gas or vapors
Retinal hyperemia	Methanol
Poor vision	Methanol, botulism, carbon monoxide
CUTANEOUS SIGNS	
Needle tracks	Heroin, PCP, amphetamine
Dry, hot skin	Anticholinergic agents, botulism
Diaphoresis	Organophosphates, muscarinic mushrooms, aspirin, cocaine
Alopecia	Thallium, arsenic, lead, mercury
Erythema	Boric acid, mercury, cyanide, anticholinergics
ORAL SIGNS	
Salivation	Organophosphates, salicylate, corrosives, strychnine, ketamine
Dry mouth	Amphetamine, anticholinergics, antihistamine
Burns	Corrosives, oxalate-containing plants
Gum lines	Lead, mercury, arsenic
Dysphagia	Corrosives, botulism

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INTESTINAL SIGNS	
Diarrhea	Antimicrobials, arsenic, iron, boric acid, cholinergics
Constipation	Lead, narcotics, botulism
Hematemesis	Corrosives, iron, salicylates, NSAIDs
CARDIAC SIGNS	
Tachycardia	Atropine, aspirin, amphetamine, cocaine, cyclic antidepressants, theophylline
Bradycardia	Digitalis, narcotics, clonidine, organophosphates, β blockers, calcium channel blockers
Hypertension	Amphetamine, LSD, cocaine, PCP
Hypotension	Phenothiazines, barbiturates, cyclic antidepressants, iron, β blockers, calcium channel blockers, clonidine, narcotics
RESPIRATORY SIGNS	
Depressed respiration	Alcohol, narcotics, barbiturates
Increased respiration	Amphetamines, aspirin, ethylene glycol, carbon monoxide, cyanide
Pulmonary edema	Hydrocarbons, organophosphates
CENTRAL NERVOUS SYSTEM SIGNS	
Ataxia	Alcohol, barbiturates, anticholinergics, narcotics
Coma	Sedatives, narcotics, barbiturates, salicylate, cyanide, carbon monoxide, cyclic antidepressants, alcohol
Hyperpyrexia	Anticholinergics, salicylates, amphetamine, cocaine
Muscle fasciculation	Organophosphates, theophylline
Muscle rigidity	Cyclic antidepressants, PCP, phenothiazines, haloperidol
Peripheral neuropathy	Lead, arsenic, mercury, organophosphates
Altered behavior	LSD, PCP, amphetamines, cocaine, alcohol, anticholinergics

LSD, Lysergic acid diethylamide; *MSG*, monosodium glutamate; *NSAID*, nonsteroidal antiinflammatory drug; *PCP*, phencyclidine.

General Management

1.Supportive Care

Supportive care is the mainstay of treatment in most cases. Prompt attention must be given to protecting and maintaining the airway, establishing effective breathing, and supporting the circulation. This management sequence takes precedence over other diagnostic or therapeutic procedures. If the level of consciousness is depressed and a toxic substance is suspected, glucose (1 g/kg intravenously), 100% oxygen, and naloxone should be administered.

2. Gastrointestinal decontamination: The intent of gastrointestinal decontamination is to prevent the absorption of a potentially toxic ingested substance and, in theory, to prevent the poisoning.

3.Enhanced elimination Multiple-dose activated charcoal should be considered only if a patient has ingested a life-threatening amount of carbam-azepine, dapsone, phenobarbital, quinine, or theophylline.

Alkalinization of urine may be helpful for salicylate or methotrexate ingestion. Dialysis may be used for substances that have a low volume of distribution, low molecular weight, low protein binding, and high degree of water solubility, such as methanol, ethylene glycol, salicylates, theophylline, bromide,

and lithium.

4. Specific Antidotes

TABLE 45.6 Specific Antidotes

POISON	ANTIDOTE	DOSAGE	COMMENTS
Acetaminophen	*N*-Acetylcysteine	140 mg/kg PO initial dose, then 70 mg/kg PO q4hr × 17 doses 150 mg/kg IV over 1 hr, followed by 50 mg/kg IV over 4 hr, followed by 100 mg/kg IV over 16 hr	Most effective within 16 hr of ingestion
Benzodiazepine	Flumazenil	0.2 mg IV, may repeat to 1 mg max	Possible seizures, arrhythmias, DO NOT USE FOR UNKNOWN INGESTIONS
β-Blocking agents	Glucagon	0.15 mg/kg IV, followed by infusion of 0.05-0.15 mg/kg/hr	
Carbon monoxide	Oxygen	100%; hyperbaric O_2	Half-life of carboxyhemoglobin is 5 hr in room air but 1.5 hr in 100% O_2
Cyclic antidepressants	Sodium bicarbonate	1-2 mEq/kg IV, followed by continuous infusion; titrated to produce pH of 7.5-7.55	Follow potassium levels and replace as needed
Iron	Deferoxamine	Infusion of 5-15 mg/kg/hr IV (max 6 g/24 hr)	Hypotension (worse with rapid infusion rates)

TABLE **45.6** Specific Antidotes—cont'd

POISON	ANTIDOTE	DOSAGE	COMMENTS
Lead	Edetate calcium disodium (EDTA)	35-50 mg/kg/day IV × 5 days; continuous infusion or divided q12hr	
	British anti-Lewisite (BAL; dimercaprol)	4 mg/kg/dose IM q4hr × 2-7 days	May cause sterile abscesses Prepared in peanut oil, do not u in patients with peanut allergy
	Succimer (2,3-dimercaptosuccinic acid ([DMSA])	10 mg/kg/dose PO tid × 5 days, then 10 mg/kg/dose PO bid × 14 days	Few toxic effects, requires lead- home plus compliant family
Nitrites/ methemoglobinemia*	Methylene blue	1-2 mg/kg IV, repeat q 30-60 min if needed; treat for levels >30%	Exchange transfusion may be needed for severe methemoglobinemia; methylene blue overdose also causes methemoglobinemia
Opiates	Naloxone	0.1 mg/kg IV, ET, SC, IM for children, up to 2 mg, repeat as needed	Naloxone causes no respiratory depression
Organophosphates	Atropine	0.02-0.05 mg/kg IV/IO, repeat every 20-30 min as needed	Physiological: blocks acetylcholi
	Pralidoxime (2 PAM; Protopam)	25-50 mg/kg IV over 5-10 min (max 2000 mg/dose); may repeat after 1-2 hr, then q10-12hr as needed	Specific: disrupts phosphate-cholinesterase bond

Iron poisoning

- Iron poisoning is a common toxicologic emergency in young children.
- Children may show signs of toxicity with ingestions of 10-20 mg/kg of elemental iron. Serious toxicity is likely with ingestions of more than 50 mg/kg.
- **Pathophysiology:**

The absorption of iron is normally very tightly controlled by the gastrointestinal (GI) system. However, in overdose, local damage to the GI mucosa allows unregulated absorption, which leads to potentially toxic serum levels.

Phases of toxicity

Phase 1; initial toxicity, predominantly manifests as GI effects. This phase begins during the first 6 hours after ingestion and is associated with vomiting, diarrhea, and abdominal pain. Both hematemesis and hemetachezia may develop, predominantly due to direct local corrosive effects of iron on the gastric and intestinal mucosa. Early hypovolemia may result from GI bleeding, diarrhea, and third spacing due to inflammation. This hypovolemia can contribute to tissue hypoperfusion and metabolic acidosis.

Convulsions, shock, and coma may complicate this phase if the circulatory blood volume is sufficiently compromised. In these cases, the patient progresses directly to phase 3, possibly within several hours.

- **Phase 2:** is known as the latent phase and typically occurs 4-12 hours post ingestion. It is usually associated with an improvement in GI symptoms, especially when supportive care is provided during phase 1. During this time, iron is absorbed by various tissues, and systemic acidosis increases. Clinically, the patient may appear to improve, especially to nonmedical personnel, because the vomiting that occurs in phase 1 subsides. However, the vital signs worsen (eg, progressive tachycardia, developing hypotension) and laboratory analysis demonstrates progressive metabolic acidosis and, potentially, the beginning of other end-organ dysfunction (ie, elevation of transaminase levels).

- **Phase 3:** typically begins within 12-24 hours post-ingestion, Phase 3 consists of marked systemic toxicity caused by this mitochondrial damage and hepatocellular injury. GI fluid losses lead to hypovolemic shock and acidosis. Cardiovascular symptoms include decreased heart rate, decreased myocardial activity, decreased cardiac output, and increased pulmonary vascular resistance. The decrease in cardiac output may be related to a decrease in myocardial contractility exacerbated by the acidosis and hypovolemia. Free radicals from the iron absorption may induce damage and play a role in the impaired cardiac function.

- The systemic iron poisoning in phase 3 is associated with a positive anion gap metabolic acidosis.

- A coagulopathy is observed and may be due to two different mechanisms. First, free iron may exhibit a direct inhibitory effect on the formation of thrombin and thrombin's effect on fibrinogen in vitro. This may result in a coagulopathy. Later, reduced levels of clotting factors may be secondary to hepatic failure.

- **Phase 4:** may occur 2-3 days postingestion. Iron is absorbed by Kupffer cells and hepatocytes, exceeding the storage capacity of ferritin and causing oxidative damage. Pathologic changes include cloudy swelling, periportal hepatic necrosis, and elevated transaminase levels. This may result in hepatic failure.

- **Phase 5:** occurs 2-6 weeks post ingestion and is characterized by late scarring of the GI tract, which causes pyloric obstruction or hepatic cirrhosis.

Differential Diagnosis

- Gastroenteritis
- MUDPILES
  - Methanol, Uremia, Diabetic ketoacidosis, Paraldehyde, **Iron** (or Isoniazid), Lactic acidosis, Ethylene glycol, Salicylates

Workup

- Clinical Diagnosis
  - Hx of ingestion
  - Physical Exam findings
- Serum levels, imaging studies are **adjuncts**
  - Mild - Less than 300 µg/dL
  - Moderate - 300-500 µg/dL
  - Severe - More than 500 µg/dL
  - **Peak serum levels after 2-6 hours of ingestion**
  - Abdominal x-ray may show undissolved tablets

Treatment

- Asymptomatic -> Follow up at home
- Symptomatic ->
  - Establish IV access
  - Labs -> CBC (anemia), VBGs (Acidosis), Serum Iron
  - Abdominal X-ray
- Ipecac not recommended, Gastric lavage not recommended, Activated Charcoal not helpful, Whole Bowel Irrigation can be useful

- Chelating Agent -> **Deferoxamine**
  - Iron levels > 500µg/dL
  - Significant Acidosis
  - Ongoing symptoms

Deferoxamine

- IV or IM (IV preferred)
- Dose -> **15 mg/kg/hr**
- When to stop?
  - If mild-moderate -> 6-12 hours
  - If severe -> 24 hours
  - Use your clinical judgment
- Adverse Effects?
  - Rare
  - Hypotension
  - ARDS
  - Yersinia sepsis
  - Pink urine?

Deferasirox (Exjade)

- Oral iron chelating agent
- Useful in iron toxicity associated with RBC transfusion

Carbon Monoxide

Carbon Monoxide

- Odorless, colorless
- Product of **inefficient** carbon-containing fuel combustion
  - Kerosine heaters
  - Closed space fire
  - Automobiles
  - Water heaters
  - … etc.

Pathophysiology

- CO affinity to Hb is **~240x higher than O2**
- CO displaces O2 -> HbCO complexes -> reduced oxygen delivery -> **tissue hypoxia**
- CO displaces NO -> NO binds to free radicals -> further cellular damage and vasodilation

Presentation

- Early
  - **headache**, malaise, nausea, and vomiting
- At higher exposure levels
  - mental status changes, **confusion**, ataxia, syncope, tachycardia, and tachypnea
- Severe
  - **coma**, seizures, myocardial ischemia, acidosis, cardiovascular collapse, and potentially **death.**
- Clinical features **DO NOT** correlate with HbCO levels
- Physical Exam
  - CVS and CNS

Investigations and Labs

- At ER
  - CO oximetry (SpO2 is not useful)
  - ABGs or VBGs
  - Creatine Kinase
  - Pregnancy test
  - ECG

	Carbon Monoxide Poisoning	Cyanide Poisoning
Hx	**Closed-space fires** Can affect multiple individuals from the same place	Combustion of **certain substances** (plastics, rubber, …etc) Cyanide containing foods
Oxygen-myoglobin dissociation curve	**Left-shift**	Normal
Clinical features	**Headache, dizziness**, N/V, altered mental status, **inhalation injury** (associated with fires)	Smell of **bitter almonds** Confusion, vertigo, N/V, arrhythmias
Post mortem changes	Cherry-red livor mortis with bullous skin lesions	Cherry-red livor mortis
Labs	High HbCO on Co oximetry PaO2 normal High anion-gap metabolic acidosis	PaO2 Normal High anion-gap metabolic acidosis

Management

- Prevention is essential
  - Proper ventilation
  - CO monitors

- **Oxygen therapy**
  - 100% oxygen via nonrebreather mask
  - When to stop?
    - 6 hours asymptomatic
    - HbCO normalizes (~ <3-5%)

Management

- **Hyperbaric Oxygen**
  - Severe intoxication
  - Controversial
  - Indications
    - HbCO > 25% (if pregnant > 15%)
    - Neurological manifestations
    - Acute MI

- Treatment lasts for 6-24 hours

ACETAMINOPHEN

LUJAIN ALWLAIDAT

- Acetaminophen (APAP / N-acetyl-para-aminophenol) is the most widely used analgesic and antipyretic in pediatrics, available in multiple formulations, strengths, and combinations. Consequently, APAP is commonly available in the home, where it can be unintentionally ingested by young children, taken in an intentional overdose by adolescents and adults, or inappropriately dosed in all ages

- In the United States, APAP toxicity remains the most common cause of acute liver failure and is the leading cause of intentional poisoning death.

Pathophysiology

- APAP toxicity results from the formation of a highly reactive intermediate metabolite, N -acetyl-p -benzoquinone imine (NAPQI)
- In therapeutic use, only a small percentage of a dose (approximately 5%) is metabolized by the hepatic cytochrome P450 enzyme CYP2E1 to NAPQI, which is then immediately joined with glutathione to form a nontoxic mercapturic acid conjugate . In overdose, glutathione stores are overwhelmed, and free NAPQI is able to combine with hepatic macromolecules to produce hepaticellular necrosis

- The single acute toxic dose of APAP is generally considered to be >200 mg/kg in children and >7.5-10 g in adolescents and adults. Repeated administration of APAP at supratherapeutic doses (>90 mg/kg/day for consecutive days) can lead to hepatic injury or failure in some children, especially in the setting of fever, dehydration, poor nutrition, and other conditions that serve to reduce glutathione stores.
- Any child with a history of acute ingestion of >200 mg/kg (unusual in children <6 yr) or with acute intentional ingestion of any amount should be referred to a healthcare facility for clinical assessment and measurement of a serum APAP level.

presentation

- Classically, 4 general stages of APAP toxicity have been described
- The initial signs are nonspecific (i.e., nausea and vomiting) and may not be present. Thus the diagnosis of APAP toxicity cannot be based on clinical symptoms alone, but instead requires consideration of the combination of the patient's history, symptoms, and laboratory findings.
- If a toxic ingestion is suspected, a serum APAP level should be measured 4 hr after the reported time of ingestion. For patients who present to medical care more than 4 hr after ingestion, a stat APAP level should be obtained. APAP levels obtained <4 hr after ingestion , unless “nondetectable” are different to interpret and cannot to be used to estimate the potential for toxicity . Other important baseline lab tests include hepatic transaminase , renal function tests , and coagulation parameters .

Classic Stages in Clinical Course of Acetaminophen Toxicity

STAGE	TIME AFTER INGESTION	CHARACTERISTICS
I	0.5-24 hr	Anorexia, vomiting, malaise Lab tests typically normal, except for acetaminophen level
II	24-48 hr	Resolution of earlier symptoms; right upper quadrant abdominal pain and tenderness; elevated hepatic transaminases (aspartate > alanine), INR
III	3-5 days	Peak transaminase elevations; development of liver failure, multi organ-system failure, death *or* recovery begins
IV	4 days to 2 wk	Resolution of liver function abnormalities Clinical recovery precedes histologic recovery

treatment

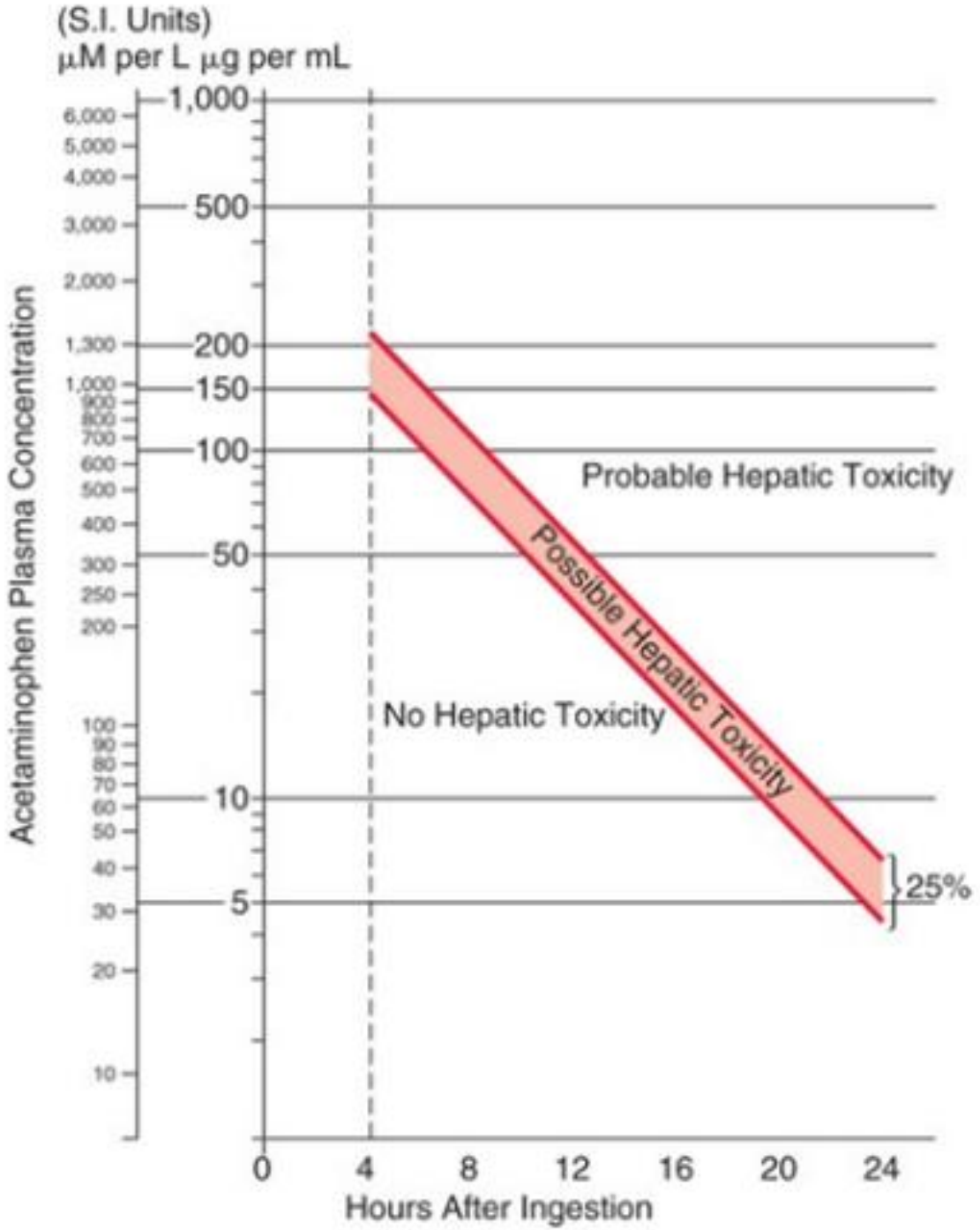
- When considering the treatment of a patient poisoned or potentially poisoned with APAP, and after assessment of the ABCs, it is helpful to place the patient into one of the following four categories.

1. Prophylactic.
2. Hepatic Injury.
3. Acute Liver Failure.
4. Repeated Supratherapeutic Ingestion.

1 Prophylactic :

- normal aspartate transaminase (AST)
- Known level of APAP and the ingestion is within 24 hr of the level being drawn, treatment decisions are based on where the level falls on the Rumack-Matthew nomogram .
- Any patient with a serum APAP level in the possible or probable hepatotoxicity range per the nomogram should be treated with N - acetylcysteine (NAC)
- This nomogram is only intended for use in patients who present within 24 hr of a single acute APAP ingestion with a known time of ingestion
- If treatment is recommended, they should receive NAC as either oral Mucomyst or IV Acetadote for 24 or 21 hr, respectively
- Repeat AST and APAP concentration drawn toward the end of that interval should be obtained. If the AST remains normal and the APAP becomes nondetectable, treatment may be discontinued
- If the AST becomes elevated, the patient moves into the next category of treatment (injury).
- If APAP is still present, treatment should be continued until the level is nondetectable. In the case of a patient with a documented APAP level, normal AST, and an unknown time of ingestion, treatment should ensue until the level is nondetectable, with normal transaminases.

- The importance of instituting therapy with either IV or oral NAC no later than 8 hr from the time of ingestion cannot be overemphasized. No patient, regardless of the size of the ingestion, who receives NAC within 8 hr of overdose should die from liver failure. The longer from the 8 hr mark the initiation of therapy is delayed, the greater the risk of acute liver failure. Any patient presenting close to or beyond the 8 hr mark after an APAP overdose should be empirically started on NAC pending laboratory results.



- **Rumack-Matthew nomogram for acetaminophen poisoning, a semilogarithmic plot of plasma acetaminophen concentrations vs time. Cautions for the use of this chart: The time coordinates refer to time after ingestion; serum concentrations obtained before 4 hr are not interpretable; and the graph should be used only in relation to a single acute ingestion with a known time of ingestion. This nomogram is not useful for chronic exposures or unknown time of ingestion and should be used with caution in the setting of co-ingestants that that slow gastrointestinal motility. The lower solid line is typically used in the United States to define toxicity and direct treatment, whereas the upper line is generally used in Europe. (From Rumack BH, Hess AJ, editors: Poisindex , Denver, 1995, Micromedix. Adapted from Rumack BH, Matthew H: Acetaminophen poisoning and toxicity, Pediatrics 55:871–876, 1975**

2 Hepatic Injury.

- These patients are exhibiting evidence of hepatocellular necrosis
- manifested first as elevated liver transaminases (usually AST first, then alanine transaminase [ALT]), followed by a rise in the INR.
- Any patient in this category requires therapy with NAC (IV or oral).
- When to discontinue therapy in the clinically well patient remains controversial, but in general the transaminases and INR have peaked and fallen significantly “toward” normal (they do not need to be normal).
- Most patients' liver enzymes will peak 3 or 4 days after their ingestion.

3 Acute Liver Failure.

- The King's College criteria are used to determine which patients should be referred for consideration of liver transplant.
- These criteria include acidemia (serum pH 6), renal dysfunction (creatinine >3.4 mg/dL), and grade III or IV hepatic encephalopathy
- A serum lactic acid >3 mmol/L (after IV fluids) adds to both sensitivity and specificity of the criteria to predict death without liver transplant.
- The degree of transaminase elevation does not factor in to this decision-making process.

•4 Repeated Supratherapeutic Ingestion.

- APAP is particularly prone to unintentional overdose through the ingestion of multiple medications containing the drug or simply because people assume it to be safe at any dose , Ingestion of amounts significantly greater than the recommended daily dose for several days or more puts one at risk for liver injury
- because the Rumack-Matthew nomogram is not helpful in this scenario, a conservative approach is taken

Antidote

- In the asymptomatic patient, if the AST is normal and the APAP is <10mg/mL , no therapy is indicated.
- A normal AST and an elevated APAP warrants NAC dosing for at least long enough for the drug to metabolize while the AST remains normal . An elevated AST puts the patient in the “hepatic injury” category previously described.
- A patient presenting with symptoms (i.e., right upper quadrant pain, vomiting, jaundice) should be empirically started on NAC pending lab results

•NAC is available in oral and IV forms, and both are considered equally efficacious.

• The IV form is used in patients with intractable vomiting, those with evidence of hepatic failure, and pregnant patients.

• Oral NAC has an unpleasant taste and smell and can be mixed in soft drink or fruit juice or given by NG tube to improve tolerability of the oral regimen

•Administration of IV NAC (as a standard 3% solution to avoid administering excess free water, typically in 5% dextrose), especially the initial loading dose, is associated in some patients with the development of anaphylactoid reactions (non–immunoglobulin E mediated)

•These reactions are typically managed by stopping the infusion; treating with diphenhydramine, albuterol, and/or epinephrine as indicated; and restarting the infusion at a slower rate once symptoms have resolved

- IV dosing, however, delivers less medication to the liver compared with the oral regimen
- As a result, many toxicologists now recommend higher doses of the IV formulation in patients with large overdoses. Transaminases, synthetic function, and renal function should be followed daily while the patient is being treated with NAC
- Patients with worsening hepatic function or clinical status might benefit from more frequent lab monitoring

Organophosphate poisoning

baild

- Organophosphates are chemicals in insecticide used extensively in agriculture
- Toxicity results from ingestion of, or exposure to, agricultural pesticides.
- Its lipid soluble ,absorbable by skin,conjuctiva,respiratory system and gastrointestinal system.

Pathophysiology

- **absorbed** through the skin, lungs, and gastrointestinal tract
- They **bind** to acetylcholinesterase (AChE) and render this enzyme non functional. (inhabitantion)
- leads to an **overabundance** of acetylcholine (accumulation)
- After some period of time the acetylcholinesterase-organophosphorus compound undergoes a conformational change, known as "**aging**" which renders the enzyme irreversibly resistant to reactivation by an antidotal oxime
- acetylcholine leads to stimulation muscrinic and nicotinic receptors and make CNS symptoms

Presentation

- symptoms of organophosphate poisoning fall into the following three broad categories:
- Muscarinic
- Nicotinic
- CNS

Muscarinic findings

- Diaphoresis and diarrhea, urination, miosis, bradycardia, bronchorrhea, bronchospasm, emesis, lacrimation and salivation (DUMBELS)
- Pulmonary edema
- Increased pulmonary and oropharyngeal secretions
- Sweating
- Abdominal cramping and intestinal hypermotility

Nicotinic findings

- Muscle fasciculations (twitching)
- Fatigue
- Paralysis
- Respiratory muscle weakness
- Tachycardia
- Hypertension

CNS findings

- Anxiety
- Restlessness
- Confusion
- Headache
- Slurred speech
- Ataxia
- Seizures
- Coma
- Central respiratory paralysis
- Altered level of consciousness and/or hypotonia

Management

- Initially ..
- — patients with markedly depressed mental states require 100% oxygen and immediate endotracheal intubation .
- — adequate volume resuscitation with isotonic crystalloid (normal saline or lactated Ringer’s solution) should be performed .
- — using ANTIDOTES (Atropine, pralidoxime)

Antidote : Atropine

- competes with acetylcholine at muscarinic receptors,
- Dose : administered beginning at a dose of 0.05 mg/kg IV , IF effect is noted, the dose should be doubled every 3-5 minutes until pulmonary muscarinic signs and symptoms are alleviated

Antidote : Pralidoxime

- Pralidoxime (2-PAM) : effective in treating both muscarinic and nicotinic symptoms.
- breaks the bond between the organophosphate and the enzyme, reactivating AChE. Pralidoxime is only effective if it is used before the bond ages and becomes permanent
- current World Health Organization recommendation for IV bolus therapy with pralidoxime is at least 25 to 50 mg/kg for children, based upon the severity of symptoms and should be administered slowly over 30 minutes,

Caustic ingestions (acid and alkali)

- Caustic ingestions are seen most often in young children between 1-3 years of age
- can cause severe acute injury and long-term complications, especially the development of esophageal strictures

Pathophysiology

- Alkali –tend to cause esophageal injury if the pH is above 11.5 to 12.5 , via liquefaction necrosis Penetration into the esophageal wall
- Acids –tend to cause esophageal injury if the pH is less than 2, via coagulation necrosis

Presentation

- 1- Gastrointestinal tract injury :The most common symptom is dysphagia
  , esophageal injury : 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 .
- 2- UPPER AIRWAY INJURY :● May be asymptmatic . ● stridor, hoarseness, nasal flaring,and retractions.

  Such symptoms suggest injury to the epiglottis, which can be severe and may require emergency intubation or tracheotomy

Management

- Stabilization and supportive care
- NPO
- Asymptomatic patients: should be observed for several hours to monitor fluid intake and overall statusendoscopy usually is unnecessary .
- Symptomatic patients :may require intensive care, endotracheal intubation, and/or tracheostomy.All patients with symptoms also should be evaluated with upper endoscopy to evaluate the extent of the injury, unless endoscopy is contraindicated because of respiratory compromise.

Foreign body & hydrocarbon ingestion

- Done by:Rana jassem Alhajri

Foreign body ingestion

- Foreign body ingestion most often occurs when a non-edible object is swallowed and enters the digestive tract. However, the condition can also refer to edible items that become lodged before reaching the stomach

- The majority of foreign body ingestions occur in children between the ages of six months and three years.

- Commonly ingested objects include coins > button batteries > toys > magnets > safety pins > bones > food boluses

- Most cases passes harmlessly, however, complications may occur, which include:

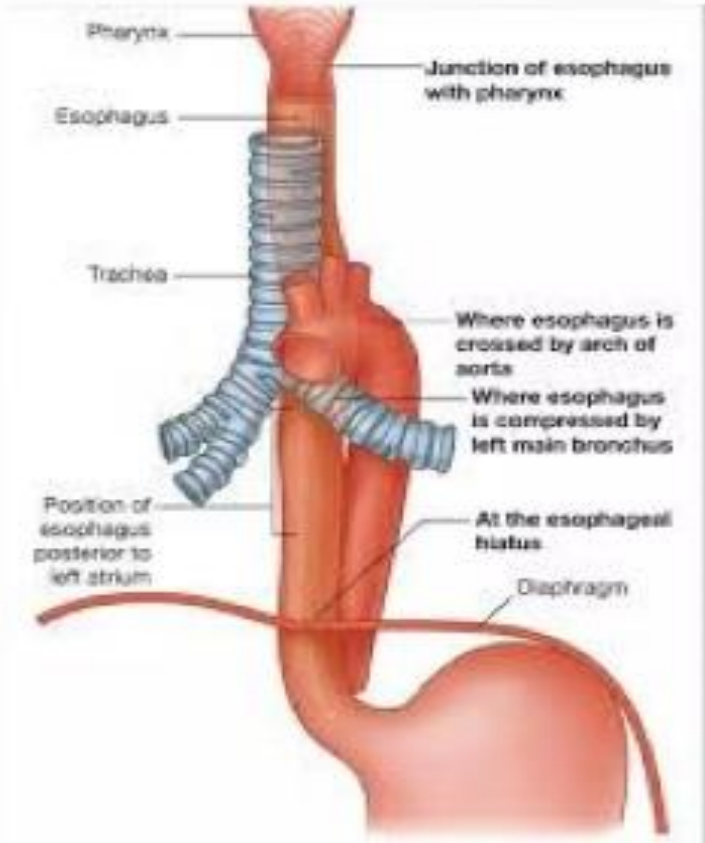
- Systemic reaction\allergy
- GI mucosa erosions\perforation
- Peretonitis
- Pneumothorax
- Aortoenteric fisutla

- The injury may be: mechanical, chemical or electrical

- It is important to localize the location of FB; to determine the appropriate approach

- The location is classified into: esophagus & stomach\lower GIT.
- Most complications occurs due esophageal impaction.

- about 70% at the upper esophageal sphincter or thoracic inlet
- about 15% in the mid-esophagus at the level of the aortic notch
- about 15% just above lower esophageal sphincter



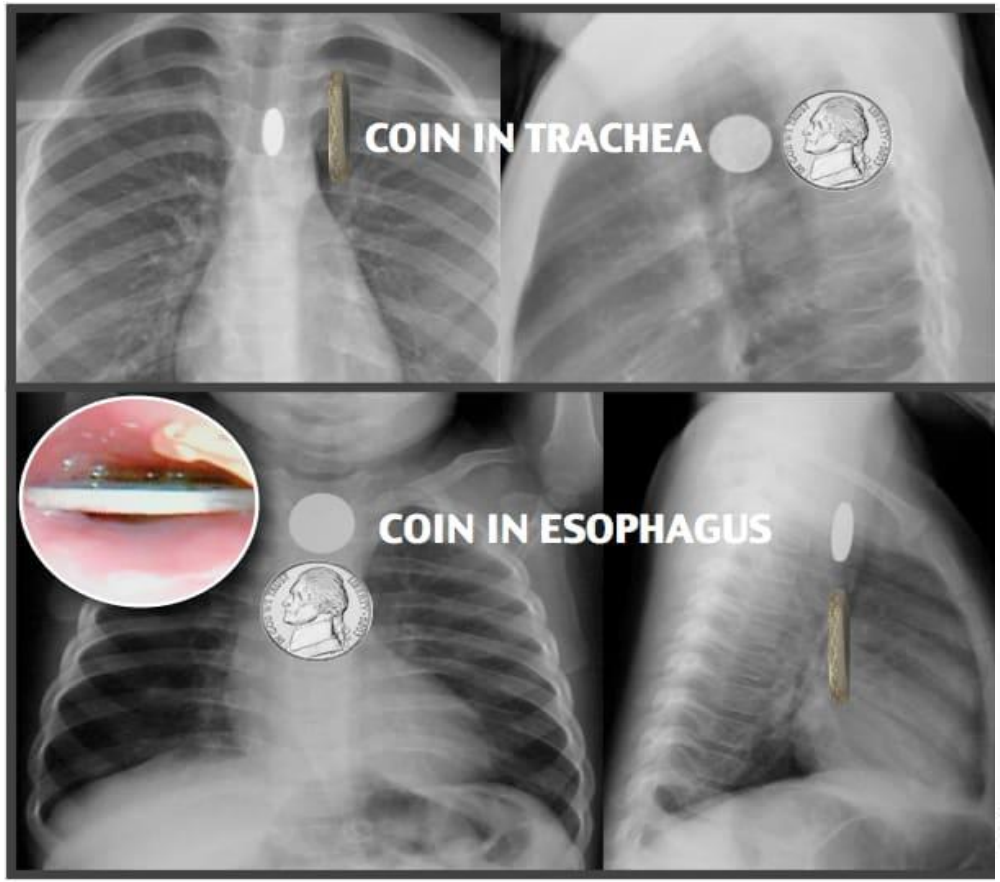
- Symptoms of FB in esophagus: dysphagia, food refusal, cough, drooling, stridor & chest pain\sore throat

- Symptoms of FB in lower GIT: abdominal pain\distention, vomiting, hematochezia & fever

Imaging

- Start by single X-ray for the neck, chest & entire abdomen.
- If the object in the esophagus, frontal and lateral views are required to precisely locate the object.
- If below diaphragm, no further imaging are required.
- If the object is radiolucent, endoscopy is superior to barium studies; as it also used to remove the object

- General rule: if the coin is in the esophagus, it appears in coronal orientation, while it looks in saggital orientation if in the trachea.
- Disk batteries have distinctive double ring sign



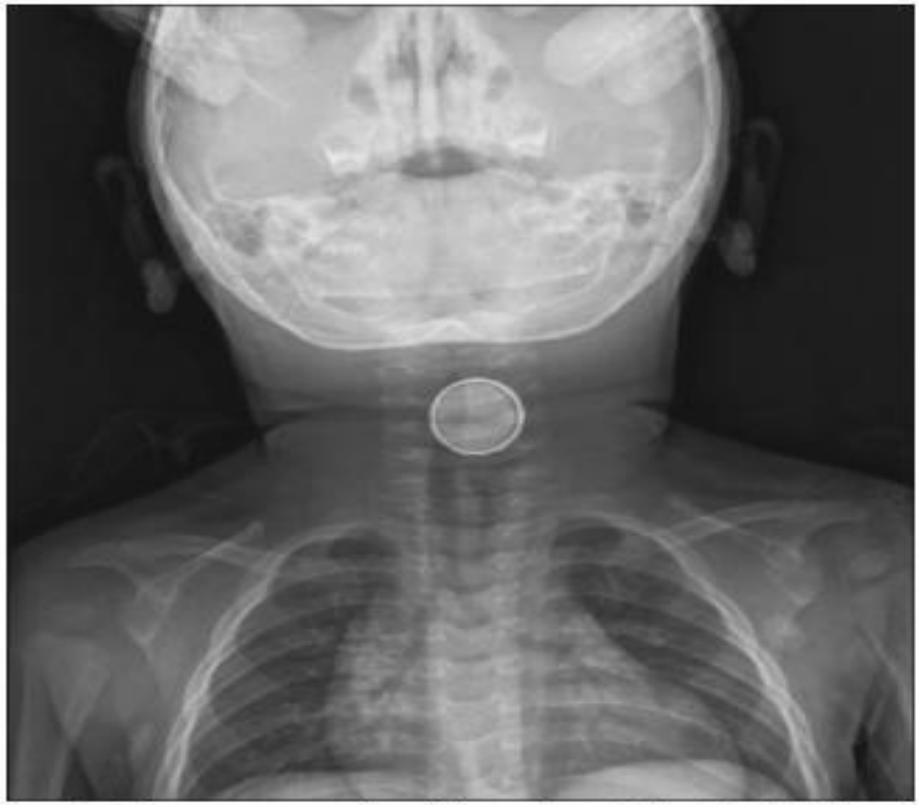
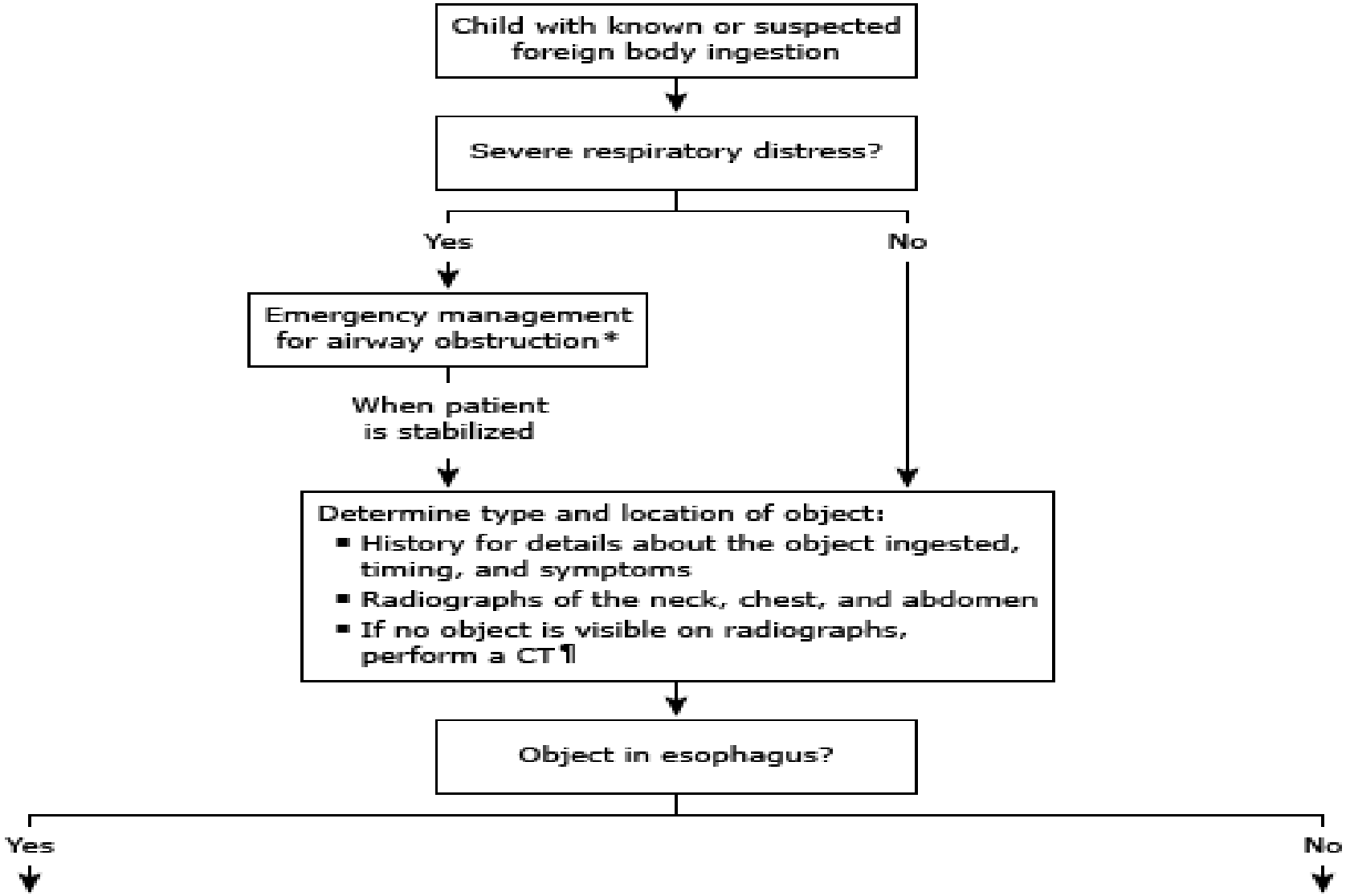
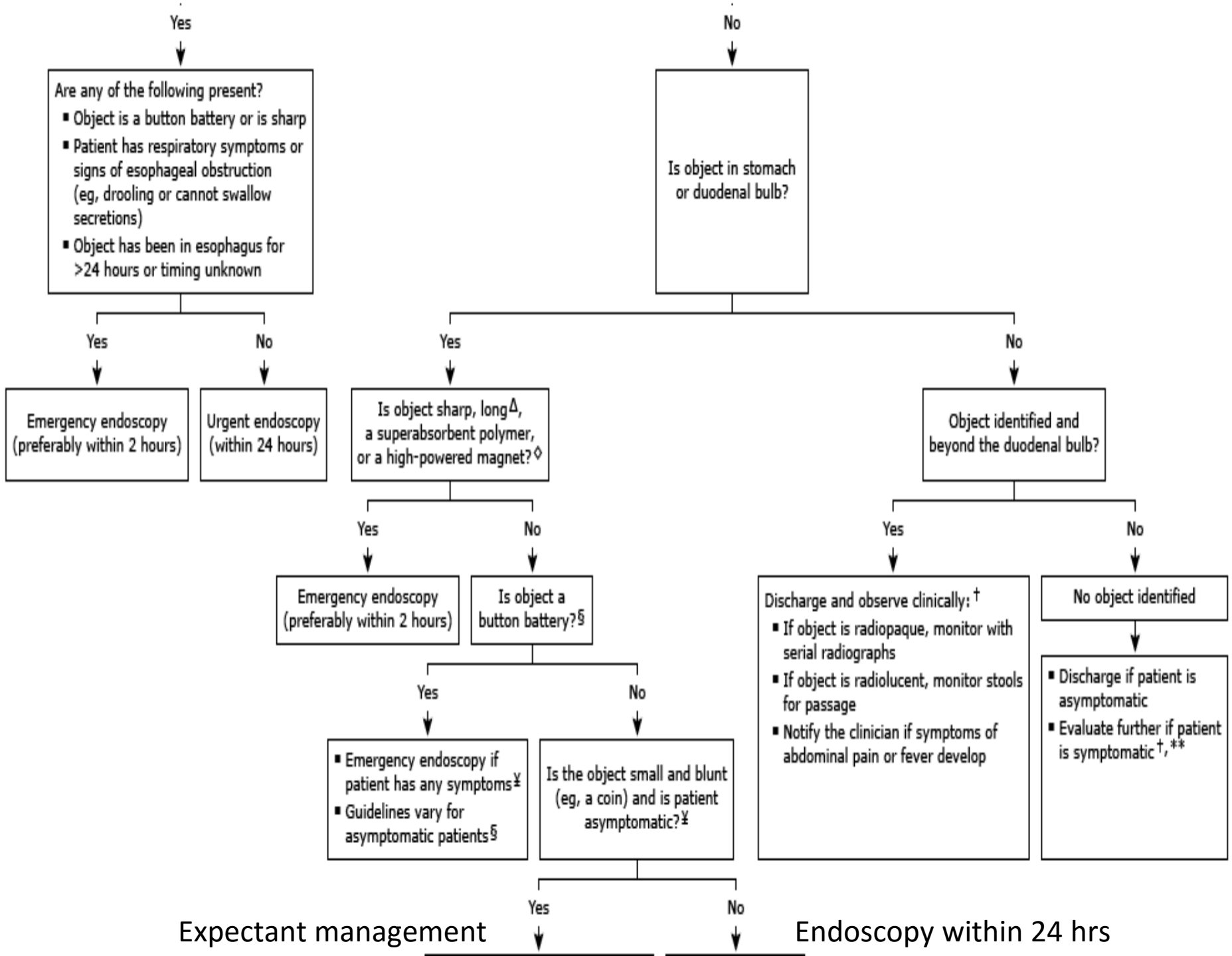


Figure 1 – This anteroposterior radiograph shows a foreign body with a double-density shadow characteristic of a button battery.

Management


Child with known or suspected foreign body ingestion
Severe respiratory distress?
Yes
No
Emergency management for airway obstruction*
When patient is stabilized
Determine type and location of object:
History for details about the object ingested, timing, and symptoms
Radiographs of the neck, chest, and abdomen
If no object is visible on radiographs, perform a CT¶
Object in esophagus?
Yes
No


Yes
No
Are any of the following present?
▪ Object is a button battery or is sharp
▪ Patient has respiratory symptoms or signs of esophageal obstruction (eg, drooling or cannot swallow secretions)
▪ Object has been in esophagus for >24 hours or timing unknown
Is object in stomach or duodenal bulb?
Yes
No
Emergency endoscopy (preferably within 2 hours)
Urgent endoscopy (within 24 hours)
Is object sharp, long Δ, a superabsorbent polymer, or a high-powered magnet? ◊
Object identified and beyond the duodenal bulb?
Yes
No
Emergency endoscopy (preferably within 2 hours)
Is object a button battery? §
Discharge and observe clinically: †
▪ If object is radiopaque, monitor with serial radiographs
▪ If object is radiolucent, monitor stools for passage
▪ Notify the clinician if symptoms of abdominal pain or fever develop
No object identified
▪ Discharge if patient is asymptomatic
▪ Evaluate further if patient is symptomatic †, **
▪ Emergency endoscopy if patient has any symptoms ¥
▪ Guidelines vary for asymptomatic patients §
Is the object small and blunt (eg, a coin) and is patient asymptomatic? ¥
Expectant management
Endoscopy within 24 hrs

Emergency management of airway obstruction

- 1. manual maneuvers:
- 2. instrumentation: ambu-bag, oropharyngeal or endo-tracheal tube
- 3. surgical: creaction of airway bypass “trachiostomy”

Hydrocarbon ingestion

- Hydrocarbon exposures account for over 28,000 cases reported annually to United States regional poison control centers and are an important cause of poisoning worldwide [1]. About 85 percent of hydrocarbon exposures are unintentional. Children five years of age and younger account for the majority of the nearly 14,000 annual pediatric exposures

Pathophysiology

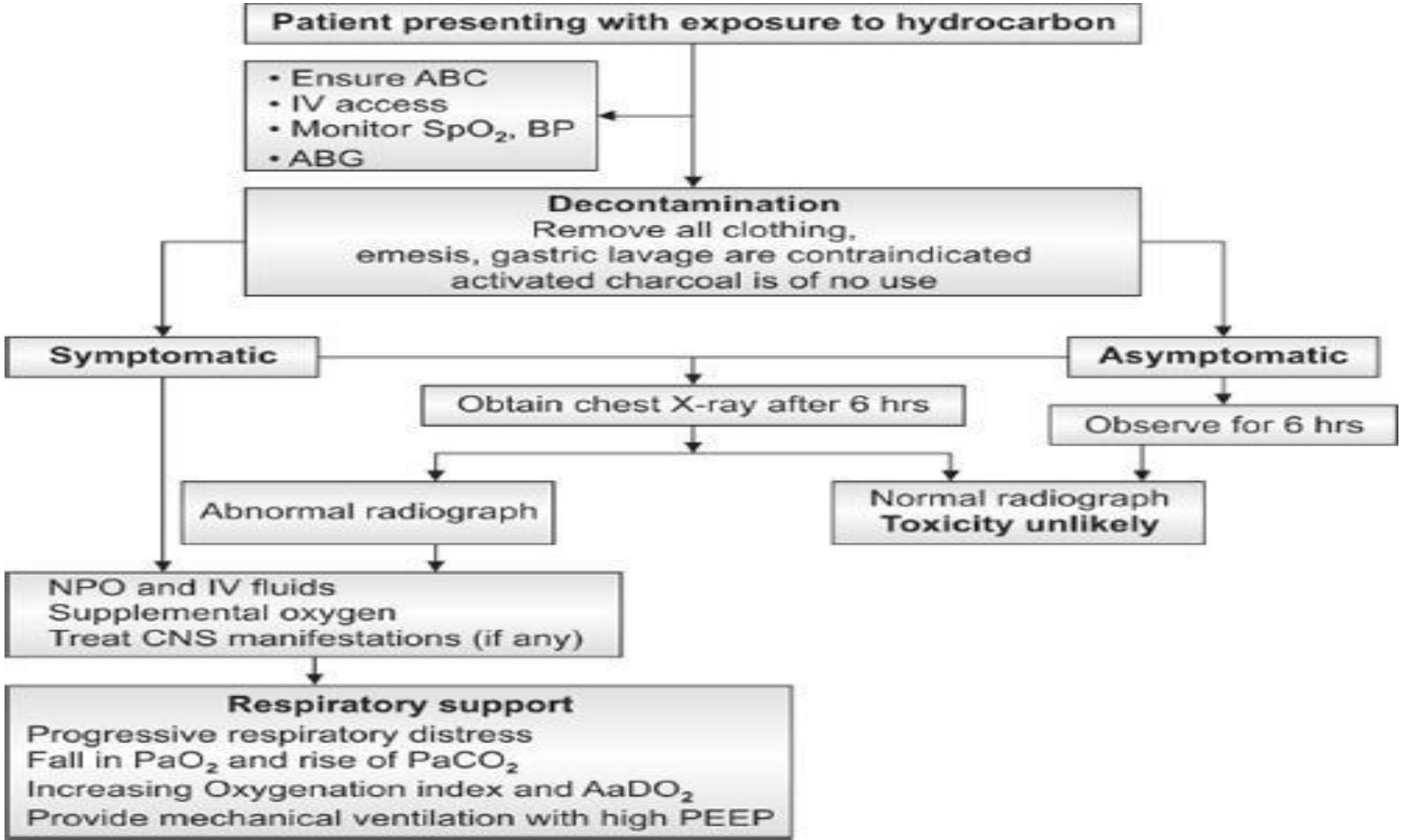
The toxicity of hydrocarbons is due to **their low surface tension and vapour pressure which helps them spread over large surface area of the lungs and cause chemical pneumonitis.** While lower surface tension helps in spreading over a large area, lower viscosity enhances penetration into distal airways leading to severe necrotizing pneumonia.

Thus compounds like kerosene, gasoline and naphtha with **high volatility, low viscosity, and low surface tension** are more likely to be aspirated and cause severe lung injury.

Presentation

- RS: cough, gagging, wheezing, hemoptysis & tachypnea
- Cardiac: arrhythmias & myocardial injury
- CNS: seizures, coma & CNC depression
- GI: nausea, vomiting, diarrhea & heartburn

Management


Patient presenting with exposure to hydrocarbon
• Ensure ABC
• IV access
• Monitor SpO_2, BP
• ABG
Decontamination
Remove all clothing,
emesis, gastric lavage are contraindicated
activated charcoal is of no use
Symptomatic
Asymptomatic
Obtain chest X-ray after 6 hrs
Observe for 6 hrs
Abnormal radiograph
Normal radiograph
Toxicity unlikely
NPO and IV fluids
Supplemental oxygen
Treat CNS manifestations (if any)
Respiratory support
Progressive respiratory distress
Fall in PaO_2 and rise of $PaCO_2$
Increasing Oxygenation index and $AaDO_2$
Provide mechanical ventilation with high PEEP

Hydrocarbon pneumotitis