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Special Article

Management of Poisonings in Children

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ABSTRACT

Acute poisoning in children is common problem worldwide, prevalent in rural as well as urban areas. Exposure of a child to such a substance is usually accidental and can cause symptoms and signs of organ dysfunction leading to injury or death.

This review is intended to discuss general approach to various types of accidental poisoning based on altered physiology in general and history and a physical examination with intent to fit the clinical characteristics into a group of signs and symptoms associated with a particular substance. (also known as TOXIDROMES)

Fortunately, very few patients require hospital admission and even fewer patients need treatment in a pediatric

Intensive care unit. Most patients will need a period of observation in a monitored bed which could be located in the emergency ward or in a step down level of pediatric intensive care unit

The ones who need admission to pediatric intensive care unit are usually critical if organ dysfunction sets in. The challenge to the pediatric

Intensivist tends to be institution of supportive treatment promptly, as well as, to institute specific antedote therapy (if available) as soon as feasible. To the physician examining the patient a challenging issue is to determine which ingestions are potentially high risk and which are Inconsequential.

Alluminium phosphide, organophosphorous compounds, kerosens oil ingestion can result in life threatening problems in affected children, are therefore reviewed in greater detail. Most poisonings can be managed by supportive treatment in the PICU.

Key words: Poisoning, ingestion, children, toxic

Introduction

Acute poisoning in children is an important pediatric emergency and is a world wide problem. By definition, poisoning is exposure of an individual to a substance that can cause symptoms and signs of organ dysfunction leading to injury or death. The cause and types of poisoning vary in different parts of the world depending upon the factors such as demography, socioeconomic status, education, availability of poisonous substance, occupation prevalent in the society etc. Though poisoning is a common occurrence during childhood, fortunately, very few patients require hospital admission and even fewer patients need treatment in a Paediatric Intensive Care Unit. The challenge to the pediatric intensivist can be daunting in determining which ingestions are potentially high risk and which are inconsequential. The main reason in accidental poisoning is lack of supervision by the parents as well as storage of poisonous substances in easily accessible places.

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Epidemiology

- More than 90% of toxic exposures in children occur in the home.
- Mainly involve only a single substance.
- Ingestion most common route of poisoning in 70-80% cases (dermal, ophthalmic and inhalation routes each occurring in about 6.0% cases).
- Involve toddlers or preschoolers, is generally

accidental, and this age accounts for approx 85%–90% cases.
- Approx 50% of cases involve nondrug substances, such as common household products. Pharmaceutical preparations comprise the remainder.
- Accidental poisoning is the twelfth leading cause of admission in the pediatric ward in India and pediatric poisonings constitutes 0.23-3.3% of the total poisoning in various studies.
- More than 85% of pediatric poisoning exposures can be managed without direct medical intervention,
- The mortality ranges from 0.64-11.6%.

Pediatric fatalities are most often associated with the following agents: analgesics, hydrocarbons, antidepressants, gases and fumes, stimulants and street drugs, cardiovascular drugs, anticonvulsants, sedatives/hypnotics/antipsychotics, and chemicals including organophosphates.

Clinical Approach to the Poisoned Child The initial approach to the patient with a documented or suspected poisoning should be no different than that in any other sick child. A detailed history and physical examination serves as the foundation for a thoughtful differential diagnosis and the formation of an initial prognosis. The history and physical examination should not await the collection of body fluid and the results of a "tox screen."

Patient history

Thehistoryshouldincludethefive"W's":

Who: The patient's age, weight, relationship to others present, and gender;

What: The name and dosage of medication(s) or substance(s) of abuse or other co-ingestants and amount ingested;

When: The time and date of ingestion;

Where: The route of poisoning; and

Why: Whether intentional or accidental, and associated details.

Other important historical points include a review of systems, specifically the presence of seizures, agitation, coma, vomiting, headache, and shortness of breath. Clinicians should be aware that in some cases the history is unreliable. Patients may not know what they ingested, the patient's altered mental status or psychosis may impede communication, or the patient may intentionally mislead the clinician. Due to their varied nature and different actions, poisoning can present by myriad symptoms. However a sudden onset of organ dysfunction or clinical deterioration of a previously well child leads to a suspicion of poisoning. A number of medications have been determined to be potentially lethal to a child who weighs 10 kg and ingests of just one tablet, capsule, or teaspoonful (table 1)

The potential for toxicity increases with increased amount of exposure.

Physical Examination

The intent of this article is not to review every possible poisoning but to provide a methodical approach to organizing physical findings that can focus therapy and guide specific diagnostic evaluations. The signs and symptoms that suggest specific classes of poisoning are generally grouped into syndromes and referred to as toxidromes.

The classic toxidromes may be grouped into four categories: Anticholinergic, Cholinergic, Opiate-sedative-ethanol syndromes, Sympathomimetic. The agents and findings are shown in table 2.

Detailed general physical and systemic examinations of the patients also help in pointing to a specific poison/toxin as shown in table 3.

Vitals recording also helps in determining the particular toxidrome as shown in table 4.

Laboratory Evaluation

The laboratory evaluation generally confirms a diagnosis that has already been established based on the history and physical examination. In some circumstances, important decisions about therapy will be made based upon quantitative drug or toxin levels in blood specimens. These include acetaminophen, ethanol, methanol, ethylene glycol, lithium, salicylates, iron, lead, mercury, arsenic, phenobarbital, carbon monoxide, methemoglobin, and theophylline. A "negative" toxicology screen by no means excludes the possibility of a toxic exposure. In certain instances, toxins may be better detected in urine than in blood. Analysis of gastric contents may be helpful in elucidating a particular toxin if they are collected before absorption is likely.

Anion Gap: Electrolytes and blood urea nitrogen (BUN)/creatinine levels allow for the determination

of an anion gap acidosis, basic electrolytes, and the assessment of renal function. The anion gap calculation is:

Na(mEq/L) - [Cl(mEq/L) + HCO3 (mEq/L)]

The normal anion gap is generally 12+-4 mEq/L. Agents that cause an elevated anion gap metabolic acidosis cane be remembered by following mnemonics:

MUDPILES: **M**ethanol, **U**remia, **D**iabetic ketoacidosis, **P**araldehyde and phenformin **I**soniazidandiron, **L**acticacidosis, **E**thanol and ethylene glycol, **S**alicylates.

KUSMOUL: **K**etoacidosis, **U**remia, **S**alicylate poisoning, **M**ethanol, **O**rganic acidemias **U**remia, **L**actic acidosis

Electrocardiogram: The 12-lead electrocardiogram is an invaluable tool in the evaluation of potential intoxication,

Prolonged QT: arsenic, amiodarone, cyclic antidepressants, chloroquine, cisapride, diphen-hydramine, lithium phenothiazines procainamide quinidine sotalol

Wide QRS: amantadine, antihistamine, cyclic antidepressants, carbamazapine chloroquine, cocaine, phenothiazine, quinine quinidine, some beta blockers

Sinus Bradycardia: beta blockers, carbamates, calcium channel blockers, clonidine, cyanide, digitalis, organophosphates, opioids, sedative-hypnotices

Hemogram: Complete blood cell count with platelets and leukocyte differential, blood clotting parameters (prothrombin time and partial thromboplastin time), liver function tests, and possible electro-encephalogram may prove useful.

RadiologicImaging: Chest and abdominal x-rays are extremely helpful in locating a number of radiopaque pills or tablets and in elucidating aspiration or pulmonary edema.

Pneumonitis or pulmonary edema is caused by:

MOPS- **M**eprobamate, Methadone,
Opiods, Organophosphates,
Phenobarbital, propoxyphene, phenoxanthines,
Salicylates.

Radiopaque compounds may be grouped by the mnemonic "COINS":

Chloral hydrate, calcium carbonate and cocaine packets,
Opiate packets,
Iron and heavy metals (lead, arsenic, mercury),
Neuroleptics, and
Sustained–release or enteric coated tablets.

Management:

The general approach to the poisoned patient can be summarized as:

- stabilization,
- decontamination and
- enhanced elimination,
- antidotes- poison-specific treatment, and
- The familiar adage "TREAT THE PATIENT, NOT THE POISON" is appropriately stated

Initial steps in treating the poisoned patient.

STABILIZATION: Stabilization is the first priority in managing toxic ingestion. The patient should be rapidly assessed to determine adequacy of airway and ventilation, mental status, and cardiovascular function. At this point, the clinician should also search for and correct hypoxia and hypoglycemia.

Initial management priorities are

- A maintenance and protection of the airway
- B support of ventilation
- C support of circulation.
- D disability, drugs, decontamination, dextrose
- E exposure, electrocardiogram (ECG), elimination
- F find antidote

ADMISSION IN PICU. Any child with:

- coma, altered sensorium, agitation, seizure,
- inadequate respiration or respiratory distress
- impaired vital parameters-tachy/ bradycardia, hypo/ hypertension
- organ failure

DECONTAMINATION: Shown in table 5.

ELIMINATION: Shown in table 6.

Speci c poisonings - most commonly seen in Indian children

ALUMINIUM PHOSPHIDE (ALP), a widely used solid fumigant, was declared as an ideal fumigant pesticide in 1973 for its effectiveness, easy to use and low cost properties. Available as tablets of 'Celphos', 'Alphos' or 'Quickphos', each tablet weighing 3.0 g liberates 1.0 g of phosphine gas (PH_3). PH_3 being gaseous in nature diffuses uniformly throughout the stored grains, leaving non-toxic residues in the

form of phosphite and hypophosphite of aluminium without affecting the food value of grains.

Epidemiology: The poisoning has been steadily increasing and has achieved alarming epidemic proportion. The poisoning involving primarily the youth is mostly suicidal, occasionally accidental and rarely homicidal.

Mechanism of action: More recent studies reported extra-mitochondrial release of hydrogen peroxide and liberation of oxygen free radicals causing lipid peroxidation and protein denaturation of cell membrane leading to hypoxic cell damage. However, the exact mechanism of action of ALP is still unclear.

Diagnosis of ALP poisoning is based on: (a) history of ingestion of the poison, (b) clinical manifestations, (c) Foul or decaying fish like breath odour and (d) cardiac arrhythmias and metabolic acidosis. The confirmation of diagnosis is done by qualitative silver nitrate impregnated paper test for treatment purposes and by chemical analysis for medicolegal purposes.

Management: Unfortunately, there is no known antidote for aluminium phosphide intoxication. The main aim of management is to sustain life with appropriate resuscitation measures till PH_3 is excreted from the body. Hence early recognition and institution of therapy are mandatory. The steps to reduce mortality during first 24 hours include:

(a) Preventing absorption of PH_3 through GI tract that can be achieved by:
- Meticulous gastric lavage with potassium permenganate ($KMNO_4$) 1:1000 solution, to be repeated twice or thrice so as to remove / oxidise unabsorbed poison.
- Slurry of activated charcoal may be given to adsorb PH_3.
- Judicious use of antacid orally and H_2 blocker intravenously for symptomatic relief.
- Medicated liquid paraffin or magnesium sulphate may be given to accelerate its excretion through gut.
- Coconut oil given orally also has a positive clinical significance and can be added to the treatment protocol.

(b) Reducing organ toxicity: In the absence of specific antidote and high affinity of PH_3 for enzyme systems, organ toxicity develops rapidly. Most of the organ toxicity is hypoxic due to oxidant injury produced by PH_3 and heart is the most vulnerable organ. Hence, use of magnesium sulphate as an antioxidant has shown significant reduction in the mortality. Magnesium sulphate in addition to having antioxidant effect, is also useful as an anti-arrhythmic and anti-hypoxic agent in ALP poisoning and as such acts as a double edged weapon for protection of the cells in the presence of hypoxia.

(c) Enhancing PH_3 excretion: PH_3 is stable and partially water-soluble. It is excreted through breath and urine, therefore, adequate hydration and renal perfusion by low dose dopamine, 4 - 6 mg / kg / minute must be maintained. Though contraindicated in shock, diuretics may be tried in patients with stable blood pressure of around 80 mm Hg.

(d) Supportive measures:

Hypoxia is managed by oxygen inhalation, airway patency by endotracheal intubation or assisted ventilation if necessary. Shock is managed by intravenous fluids, given during first 3 to 6 hours, guided by CVP, Blood pressure should be maintained above 70 mm of Hg. Low dose dopamine (4 – 6 mg/kg/minute) and hydrocortisone (5-8mg/kg I.V. after 4 to 6 hours) have been reported to be effective. Steroids combat shock, check the capillary leakage in the lungs and potentiate the responsiveness of shock to catecholamines.

Arrhythmias (both tachy- and bradyarrhythmias) may be controlled by magnesium sulfate that has a membrane stabilizing effect. Conventional anti-arrhythmic drugs like digoxin, xylocaine, etc are reported to be ineffective.

Acute Respiratory Distress Syndrome (ARDS) can be managed by delivering 100% oxygen by face masks fitted with reservoir inspiratory bags at moderate flow rate of 5 to 10 L, to achieve PO_2 of 60 to 70 with lowest inspired fraction of O_2. Mechanical respiratory support and positive end expiratory pressure (PEEP) therapy is given in haemodynamically unstable patients.

Figure 1 shows the flowchart in managing this dreadful poison.

ORGANOPHOSPHORUS COMPOUNDS (OPC) are widely used as agricultural, industrial and domestic insecticides. Poisoning with OPC may occur in

isolation after exposure or in epidemics after ingestion of contaminated foodstuffs. Most of these compounds are available either as organophosphates (Malathion, Parathion, Methylparathion, Isomalathion, Diazinon, Dichlorovas, etc.) or carbamates (Carbanyl, Matacil, etc). They are used as sprays after dilution with organic solvents or water. The available formulations contain 1 to 95% of an active ingredient and accordingly the toxicity varies widely.

Mechanism of action: Organophosphorus compounds are potent inhibitors of true acetylcholinestrase (AchE) present in central nervous system (CNS) and the red blood cells (RBC) and pseudo-cholinesterase (Pseudo-ChE) present in liver, plasma and serum. The pharmacological and toxicological effects are due to accumulation of acetylcholine at synapses resulting in initial stimulation followed by paralysis of neurotransmission at cholinergic synapsis present in CNS, somatic nerves, autonomic ganglion, para-sympathetic nerve endings and some sympathetic nerve endings like in sweat glands. **Diagnosis** is based on: (1) history and circumstances leading to exposure, (2) clinical manifestations (3) clinical and therapeutic response to atropine and oximes. Confirmation of diagnosis is by measurement of anticholinestrase enzyme in RBCs or plasma pseudocholinestrase enzyme for treatment purpose and chemical analysis of body fluids (blood, urine, gastric lavage) for medicolegal purpose.

Management: All cases of OPC poisoning should be sent to hospital as quickly as possible. Although symptoms may develop rapidly, delay in onset or steady increase in severity may be seen up to 24 hours after ingestion.

Therapy consists of (a). Evacuation of stomach (b). Prevention of absorption from other sites by removing the contaminated clothing and meticulous washing

Summary:

Assessment and Management of A Poisoned Patient*

STABILIZE PATIENT

GOALS
- Identify the toxin
- Assess the presence and degree of toxicity
- Consider if a non-toxicological condition could be the cause of or contributing to clinical presentation

History	**Physical Examination**	**Laboratory***
Important but often: • Unreliable • Unavailable • Incomplete	A focused examination to include: • Vital signs • Arousal levels, Eyes • Muscle tone Reflexes • Skin, Odors • Look for toxidromes or toxic signs	• Selective/Progressive • Toxicological testing

MANAGEMENT
- **A** Airway
- **B** Breathing
- **C** Circulation
- **D** Drugs (glucose, thiamine, naloxone) Decontamination
- **E** Climination
- **F** Find an antidote
- **G** General Management (complications)

Notes *Use in conjuction with Laboratory Guideline for the Investigation of the Poisoned Patient.

of skin with alkaline soap or sodium bicarbonate solution. (c). Supportive measures. Maintain ABC. (d). Administration of specific antidote

- Intermittent atropine therapy is given in the dose of 0.05 mg/kg I.V slowly in children, every 5 to 10 minutes till parasympathetic manifestations are controlled or early signs of atropinization appear. Tachycardia and papillary dilation are not reliable indicators of atropinization. The maintenance dose is given after the initial bolus at a continuous infusion at the rate of 0.02 – 0.08 mg/kg/hr. continued for 3 – 5 days and slowly withdrawn on 6th or 7th day. Sudden withdrawal may produce relapse or exacerbation of signs and symptoms.
- Some studies have reported that high dose continuous administration of atropine (150 mg in 5% Dextrose) drip over a period of 6 hours is equally effective as intermittent therapy.
- Oximes (cholinesterase enzyme reactivators) as for example praldoxime is given (table 7) mixed in 250 ml of normal saline and infused over 30 minutes. The dose may be repeated after one hour and subsequently every 6 - 12 hours if muscle or diaphragmatic weakness or coma are not relieved. In severe poisoning praldoxime can be administered by continuous infusion (9 - 19 mg/kg/hr after an initial bolus). The titration should be based on clinical response and the oxime therapy should be monitored by measuring cholinesterase activity.

KEROSENE OIL: Among house hold products kerosene was the leading poison taken due to its high use as cooking fuel and lightening in the house. This substance was usually stored in the soft drink or mineral water bottle and is easily ingested by children. Kerosene oil ingestion results in toxicity of gastrointestinal, respiratory and central nervous system. Aspiration pneumonia is the commonest complication. Management is shown in figure 2.

Conclusions: After resuscitation, stabilization, and initial evaluation and management, interventions that can be used in the ICU to decrease toxin absorption or enhance elimination. Specific interventions or antidotes may be indicated in certain poisonings. The management of poisoning emergencies in the pediatric emergency care units has changed over the last few decades. Most pediatric poisoning exposures have favorable outcomes, with very few deaths reported but supportive care that includes attention to airway and monitoring is also important to improve outcomes. Critical care practitioners should be familiar with the evaluation of these patients and knowledgeable about beneficial interventions.

Key messages:

- Supportive care remains the crux of therapy for all poisonings. Aggressive airway management with attention to airway reflexes is by far the most important management principle. Careful evaluation and maintenance of ventilation is critical. Hemodynamic compromise must be recognized and corrected.
- The laboratory evaluation generally serves to confirm a diagnosis that has been made based upon the history and physical examination. The negative toxicology screen does not exclude the possibility of a toxic exposure. Identification of toxidromes requires meticulous attention to clinical signs and symptoms.
- Familiarity with specific toxins and their antidotes enables immediate initiation of therapy, as well as the ability to definitively identify certain toxins.
- Prevention strategies are key in decreasing morbidity and mortality.

Management of Poisonings in children
Tables and Figures

Table 1: Toxicity levels of selected medications and medication classes

Agents	Minimum potential lethal dose	Maximum dose available	Potentially fatal units in a 10-kg child
Antimalarials			
Chloroquine	20 mg/kg	500 mg	1
Hydroxychloroquine	20 mg/kg	200 mg	1
Camphor	100 mg/kg	200 mg/mL	5 mL

Imidazolines			
Clonidine	0.01 mg/kg	0.3 mg; 7.5 mg/patch	1
Tetrahydrozoline	2.5 to 5 mL	0.1%	2.5 to 5 mL
Methyl Salicylates	150 to 200 mg/kg	1400 mg/mL	1.1 to 1.4 mL
Sulfonylureas			
Glipizide	0.1 mg/kg	5 mg	1
Glyburide	0.1 mg/kg	10 mg	1
From Matteucci MJ. One pill can kill: Assessing the potential for fatal poisonings in children. *Pediatr Ann* 2005;34:964-968.			

Table 2: Agents and toxidromes

Anticholinergic toxidrome*	**Agents**
Agitation, Delirium, Coma, Mydriasis Dry mouth Warm, dry, flushed skin, Fever Tachycardia, Hypertension Urinary retention, Decreased bowel sounds Associated expressions "Mad as a Hatter" "Blind as a Bat" "Red as a Beet" "Hot as a Hare" "Dry as a Bone"	Antihistamines—diphenhydramine, hydroxyzine, Antiparkinsonian drugs, Antispasmodics, Atropine, Alkaloids Belladonna, Benztropine mesylate Carbamazepine, Cyclobenzaprine, Cyclopentolate eyedrops Hyoscyamine Jimsonweed Oxybutynin Phenothiazines Scopolamine, some mushrooms TriCyclic antidepressants, Trihexyphenidyl
Cholinergic toxidrome	**Agents**
Muscarinic effects (parasympathetic) DUMBBELS: Diarrhea, Urinary incontinence, Miosis, Bradycardia Bronchorrhea, Emesis, Lacrimation Salivation SLUDGE: Salivation, Lacrimation, Urinary incontinence, Diarrhea/ Diaphoresis, GI upset/hyperactive bowel, Emesis Nicotinic effects (Sympathetic and parasympathetic autonomic ganglia) Mydriasis, Fasciculations, Weakness Paralysis, Tachycardia, Hypertension Agitation, Diaphragmatic failure Central effects Lethargy, Coma, Agitation, Seizures, Headache, Confusion, Ataxia, Respiratory depression	Organophosphate insecticides Drugs for myasthenia gravis (e.g., pyridostigmine) Nicotine Carbamate insecticides
Opioid Toxidrome	**Agents**
Central nervous system depression, hypothermia, hypotension, hypoventilation, miosis bradycardia decreased GI motility (Triad of- respiratory depression, coma, miosis, exception meperidine-mydriasis and seizures).	opium and opioids (substances derived from opium and synthetically derived agents with similar properties). Codeine, hydrocodone, oxycodone, Diphenoxylate Fentanyl patches Heroin Methadone propoxyphene (clonidine-centrally acting included as diminishes sympathetic tone, causing triad)
Sympathomimetic toxidrome*	**Agents**
Agitation, Seizures Mydriasis Tachycardia, Hypertension Diaphoresis, Cool Skin Pallor Fever, sweating	Albuterol, Amphetamines Caffeine, Catecholamines, Cocaine Ephedrine Ketamine Lysergic acid diethylamide (LSD) Methamphetamine Phencyclidine (PCP), Phenylephrine (devcongestants), Phenylpropanolamine, Pseudoephedrine Terbutaline, Theophylline

*Both almost similar clinical features except skin dry, warm, and bowel sounds decreased in **Anticholinergic toxidrome.** Skin cool, diaphoretic, and hyperactive bowel sounds in **Sympathomimetic toxidrome.**

Table 3: Various signs and poison involved.

OCULAR SIGNS	
Miosis	barbiturates (late), chloral hydrate, clonidine, diazepam, muscarinic mushrooms, Narcotics (except meperidine), Narcotics (except Demerol and Lomotil), organophosphates, phenothiazines, PCP phenothiazines,
Mydriasis	Anticholinergics, Atropine, alcohol, amphetamines, antihistamines, cocaine, cyanide, carbon monoxide, glutethimide, LSD, methanol, sympathomimetics, tricyclic antidepressants
Nystagmus	barbiturates, carbamazepine, carbon monoxide, ethanol, glutethimide, Phenytoin, PCP,
Lacrimation	Organophosphates, irritant gas or vapors
Retinal hyperemia	Methanol
Poor vision	Methanol, botulism, carbon monoxide
SKIN	
Cyanosis (unresponsive to oxygen)	Nitrates, nitrites, phenacetin, benzocaine
Red flush, Erythema	anticholinergics, boric acid, Carbon monoxide, cyanide, mercury
Sweating, Diaphoresis	Amphetamines, barbiturates cocaine LSD, muscarinic mushrooms, nitrates, organophosphates, salicylates
Bullae	Barbiturates, carbon monoxide
Jaundice	Acetaminophen, mushrooms, carbon tetrachloride, iron, phosphorus
Purpura	Aspirin, warfarin, snakebite
Needle tracks	Heroin, PCP, amphetamines
Dry, hot skin	Anticholinergic agents, botulism
Alopecia	Thallium, arsenic, lead, mercury
TEMPERATURE	
Hypothermia	carbon monoxide, clonidine, ethanol, Sedative hypnotics, phenothiazines, TCAs,
Hyperthermia	Anticholinergics, amphetamines, cocaine, phenothiazines, salicylates, TCAs, theophylline
ORAL SIGNS	
Salivation	carbamates, corrosives, Organophosphates, salicylates, strychnine,
Dry mouth	Amphetamines, anticholinergics, antihistamine
Burns	Corrosives, oxalate-containing plants
Gum lines	Lead, mercury, arsenic
Dysphagia	Corrosives, botulism
CARDIAC SIGNS	
Tachycardia	Atropine (Anticholinergics), alcohol, antidepressants, amphetamines, cocaine, salicylates, sympathomimetics, theophylline
Bradycardia	β blockers, calcium channel blockers, clonidine, Digitalis, hypnotics, mushrooms, narcotics, organophosphates, sedative
Hypertension	Amphetamines, cocaine, LSD, PCP
Hypotension	Phenothiazines, barbiturates, cyclic antidepressants, iron, β blockers, calcium channel blockers
Arrhythmias	Amphetamine Antiarrhythmics Anticholinergics, Antihistamines Arsenic antidepressants, β-blockers, carbon monoxide, Chloral hydrate Cocaine cyanide, Cyclic antidepressants digoxin, organophosphates, phenothiazines, theophylline Quinine, quinidine
RESPIRATORY SIGNS	
Depressed respiration	Alcohol, barbiturates, narcotics, sedative/hypnotics
Increased respiration	Amphetamines, carbon monoxide, cyanide, ethylene glycol, salicylates
Pulmonary edema, Pneumonia	Aspiration, Hydrocarbons, heroin, narcotics organophosphates, salicylates, sympathomimetics,
Kussmaul	ethylene glycol, Methanol, salicylates
Wheezing	Organophosphates
GASTROINTESTINAL SYSTEM	
Vomiting, diarrhea, abd pain, Cramps	Iron, phosphorus, heavy metals, lithium, mushrooms, Antimicrobials, arsenic, boric acid, fluoride, iron, lead organophosphates
Constipation	Lead, narcotics, botulism
Hematemesis	Aminophylline, corrosives, iron, Lead, salicylates

CNS SIGNS	
Ataxia or nystagmus	Alcohol, Antihistamines, antidepressants, anticholinergics, Bromides, barbiturates, Anticonvulsants (especially phenytoin, carbamazepine, Barbiturates), Carbon monoxide narcotics, Organic solvents Piperazine
Coma	Alcohols, anticholinergics, Antihistamines barbiturates, cyanide, Clonidine carbon monoxide, cyclic antidepressants, Sedatives, Lead narcotics, Methyldopa Narcotics Phencyclidine Phenothiazines PCP, organophosphates, salicylates, lead, sedative hypnotics, Hypoglycemic agents narcotics, tricyclic antidepressants, salicylates, organophosphates, barbiturates
Hyperpyrexia	Anticholinergics, quinine, salicylates, LSD, phenothiazines, amphetamines, cocaine
Muscle fasciculation	Organophosphates, theophylline
Muscle rigidity, Myoclonus	Cyclic antidepressants, PCP, phenothiazines, haloperidol, Anticholinergics
Paresthesia	Cocaine, camphor, PCP, MSG
Peripheral neuropathy	Lead, arsenic, mercury, organophosphates
Altered behavior	LSD, PCP, amphetamines, cocaine, alcohol, anticholinergics, camphor
Seizures **(MNEMONIC = CAPS)**	**C**amphor, Carbamazepine, Carbon monoxide, Cocaine, Cyanide, **A**minophylline, amphetamines, Anticholinergics, antihistamines, Antidepressants (cyclic) **P**b (lead), phenothiazines, propoxyphene Pesticide (organophosphate), Phencyclidine, Phenol **S**alicylates, strychnine
Delirium/psychosis	Anticholinergics, sympathomimetics, alcohol, phenothiazines, PCP, LSD, marijuana, cocaine, heroin, methaqualone, heavy metals
Weakness, paralysis	Organophosphates, carbamates, heavy metals
Hypertonus	Anticholinergics, strychnine, phenothiazines
Nystagmus	Diphenylhydantoin, barbiturates, carbamazepine, PCP, carbon monoxide, glutethimide, ethanol
Extrapyramidal	Phenothiazines, haloperidol, metoclopramide

Table 4: Common Toxidrome Findings

Physical Findings	**Adrenergic**	**Anti-cholinergic**	**Anti-cholinesterase**	**OPIOID**	**Sedative-hypnotic**
RR	Increased	No change	No change	Decreased	Decreased
HR	Increased	Increased	Decreased	Normal/ decreased	Normal/ decreased
Temp	Increased	Increased	No change	Normal/ decreased	Normal/ decreased
BP	Increased	NoChange/increased	No change	Normal/ decreased	Normal/ decreased
Mental status	Alert/ agitated	Depressed/ Confused/ hallucinate	Depressed/ Confused/	Depressed	Depressed
pupils	Dilated	Dilated	Constrict	Constrict	Normal
Mucus membrane	Wet	Dry	Wet	Normal	Normal
skin	Diaphoretic	Dry	Diaphoretic	Normal	Normal

Table 5: Agents Used for Gastrointestinal Decontamination in Children

Agent	**Dose**	**Risks**	**indications**	**Contraindications**
Activated charcoal*†	1 to 2 g per kg (maximum of 50 to 60 g) diluted with fruit juice or bottled drinks orally or through orogastric tube in an uncooperative child. Due to its constipatory effect, a dose of cathartic is added with the first dose. Also 0.25–0.50 g/kg every 2–4 hr	• Aspiration • Constipation • Vomiting • Ileus • seizures. If aspirated • pneomonitis • empyema • pneumothorax	• Antidepressants • Carbamazepine • Dapsone • Dextropropoxyphene • Digoxin • Diisopyraminde • Nadolol • opioids • Phenobarbitol • Phenylbutazone • phenothiazines Phenytoin • phencyclidine • Piroxicam • Quinine • salicylates • Sotalol • Theophylline	Toxins Ineffectively Bound by Activated Charcoal can be remembered by mnemonic PHAILS P – Pesticides, petroleum distillates, unprotected airway H – Hydrocarbons, heavy metals, >1h A – Acids, alkali, alcohols, altered level of consciousness, aspiration risk I – Iron, ileus, intestinal obstruction L – Lithium, lack of gag reflex S – Solvents, seizures

Agent	Dose	Risks	indications	Contraindications
Gastric lavage*†	10 to 15 mL per kg saline instilled via large-bore orogastric tube, repeated until aspirates clear	Esophageal/ laryngeal trauma, aspiration, nausea/vomiting, impaired level of consciousness	ß-blockers, calcium channel blockers, cyclic antidepressants, theophylline iron, tricyclic antidepressants, lithium. Mixed hydrocarbons- mnemonic for such hydrocarbons is CHAMP, Camphor, halogenated hydrocarbons, aromatic hydrocarbons, (heavy) metal-containing hydrocarbons, and pesticide-containing hydrocarbons.	Unprotected airway, ingestion of hydrocarbons or corrosives, risk of perforation or hemorrhage increased intracranial pressure or uncontrolled hyper- tension
Polyethylene glycol (used with whole bowel irrigation)	30 mL/kg/hr 500 mL per hour for children nine months to five years of age1,000 mL per hour for children six to 12 years of age	Vomiting, cramping Fluid and electrolyte imbalance	Metals, lithium iron, lithium or lead Sustained-release preparations theophylline, or calcium channel blockers Ingestion of pharmaceutical patches Massive overdoses Concretions of pills	Unprotected airway, intractable vomiting, gastrointestinal hemorrhage, ileus, perforation, obstruction
Sorbitol (used with activated charcoal)	1 to 2 g per kg	Hypernatremia, dehydration	Same as charcoal	Obstruction, perforation, ileus

*— May not be beneficial if given more than one hour after ingestion. Tablets, powder-filled capsules complete intestinal absorption can be delayed by as much as 3–6 hr
†— Not routinely recommended

Table 6: Enhancing Elimination

Method	Method	Risks	indications
DIURESIS: 2-5 mL/kg/hr Urinary alkalinization with sodium bicarbonate	sodium bicarbonate (50-75 mEq/L) to the IV fluids..	fluid overload, with cerebral edema, pulmonary edema, and hyponatremia.	salicylate, phenobarbital, chlorpropamide, and the chlorophenoxy herbicides primidone
DIALYSIS	Hemodialysis Peritoneal dialysis	Fluid and electrolyte disturbance	methanol, ethylene glycol, and large symptomatic ingestions of salicylates or theophylline. lithium, atenolol, sotalol valproic acid, and phenobarbital

Table 7: Specific poisonings and their antidotes.

Poisoning	Antidote	Dose	Route	Adverse Effects/Warnings
Anticholinergic atropine sulfate	physostigmine	0.02 mg/kg; slow push; may repeat q5-10 min to 2 mg max	IV/ IM	Bradycardia, asystole, seizures, bronchospasm, vomiting, headache Note: Do not use with cyclic antidepressants
Benzodiazepine	flumazenil	0.2 mg over 30 sec; if inadequate response, repeat q1min to 1 mg max	IV	Nausea, vomiting, facial flushing, agitation, headache, dizziness, seizures. Do not use for unknown or antidepressant ingestions Note: May not reverse respiratory depression
Beta blocker	glucagon	0.05 mg/kg bolus followed by infusion of 0.05 mg/kg/hr	IV	Hyperglycemia, nausea, and vomiting
Calcium channel blockers	Calcium chloride 10% glucagon	0.1-0.2 ml/kg IV bolus Same as above	IV	Hyperglycemia, nausea, and vomiting
Carbon monoxide	oxygen	100%, hyperbaric	Inhalation	Half-life of carboxyhemoglobin is 5 hr in room air, but 1.5 hr in 100% O_2 and 15–30 min in 3 atmospheres hyperbaric

Cyanide	amyl nitrite, sodium nitrite, or thiosulfate	Amyl 0.3ml inhaled over 15sec q30sec, sod nit 180-240mg/m2 IVx1, sod thio 7g/m2 IVx1.	Inhalation IV	Syncope, hypotension, tachycardia,, methemoglobinemia
Digoxin	Fragment ag binding (Digibind/ Digifab)	One vial binds 0.6 mg of digitalis estimated from serum level	IV	Allergic reactions (rare), return of condition being treated with digitalis glycoside
Ethylene glycol Methanol	ethanol fomepizole	0.8-2 ml 10%/kg/h 15 mg/kg load; 10 mg/kg q12h for 4 doses; 15 mg/kg q12h until level <20 mg/dL No specific dose for children.	IV IV	Vertigo, flushing, sedation, seizures, hypotension, hypoglycemia Infuse slowly over 30 min; increase doses to q4h if dialysis is concurrent
Extrapyramidal antipsychotic	diphenhydramine HCl benztropine mesylate	5 mg/kg divide q8h; 300 mg/ 24 hr max 0.02-0.05 mg/kg/dose qd or bid (4 mg max)	IV/ PO IV/ PO	Sedation or paradoxical agitation, ataxia Sedation, blurred vision, dry mouth, and tachycardia
Heavy metal Arsenic, mercury, lead other metals	BAL in oil dimercaprol Dimercaptosuccinic acid (DMSA) EDTA, calcium	3-5 mg/kg/dose q4hr, for the first day; subsequent dosing depends on toxin. 10 mg/ kg/dose q8h for 5 days, then 10 mg/kg q12h for 14 days. 1-1.5 g/m^2/24 hr in divided doses q12h for 5 days IV	Deep IM PO	Local injection site pain and sterile abscess, nausea, vomiting, fever, salivation, nephrotoxicity. Nausea and vomiting; repeated courses may be needed. Nausea, vomiting, fever, hypertension, arthralgias, allergic reactions, local inflammation, and nephrotoxicity (maintain adequate hydration)
Heparin	protamine sulfate	Intravenous, 1 mg of protamine sulfate for approximately every 100 USP units of heparin	IV	Hypotension, bradycardia, anaphylaxis, flushing, bleeding
Iron	deferioxamine	Infusion of 15 mg/kg/hr (max 6 g/24 hr)	IV (preferred)	Hypotension (minimized by avoiding rapid infusion rates)
Isoniazid	pyridoxine	Isoniazid; dose = dose	IV	Uncommon
Methanol	ethanol fomepizole	750 mg/kg loading dose followed by 80-150 mg/kg/hr infusion of 5% or 10% ethanol	IV/ PO	Nausea, vomiting, sedation
Methemoglobinemia	methylene blue	0.1-0.2 mL/kg of 1% solution, slow infusion, may be repeated q30-60 min	IV	Nausea, vomiting, headache, dizziness
Opioid	naloxone	0.01 mg/kg; if no effect, give 0.1 mg/kg; may be repeated as needed; may give continuous infusion	IV	Acute withdrawal symptoms if given to addicted patients
Organophosphate and carbamate pesticides;	Atropine pralidoxime chloride	0.05 mg/kg repeated q5-10 min as needed. until secretions substantially reduced Dilute in 1-2 mL of NS for ET instillation. 25-50 mg/kg over 5-10 min (max 200 mg/min); can be repeated after 1-2 hr then q10-12 hr as needed	IV/ ET IV/ IM	Tachycardia, dry mouth, blurred vision, and urinary retention Nausea, dizziness, headache, tachycardia, muscle rigidity, and bronchospasm (rapid administration)
Oral hypoglycemics sulfonylureas	Octreotide	1–2 μg/kg q8 hr	IV/ SC	Used in addition to high-dose glucose; may add glucagon
Paracetamol	N-acetylcysteine	140 mg/kg loading followed by 70 mg/kg q4h for 17 doses	PO	Nausea, vomiting

Salicylates	Sodium bicarbonate	1-2mEq+40 mEq KCl in 1 lit D5W (urine output 1-2ml/kg/ hr, urine pH 7.5)	IV	Metabolic alkalosis, hypernatremia, edema
Tricyclic antidepressants	Sodium bicarbonate	1-2 mEq/kg	IV	Metabolic alkalosis, hypernatremia, edema
Warfarin	vitamin K phytomenadione and fresh frozen plasma	5–10 mg	IV/ SC	Monitor prothrombin time; give fresh frozen plasma for acute bleeding; repeat vitamin K for superwarfarin

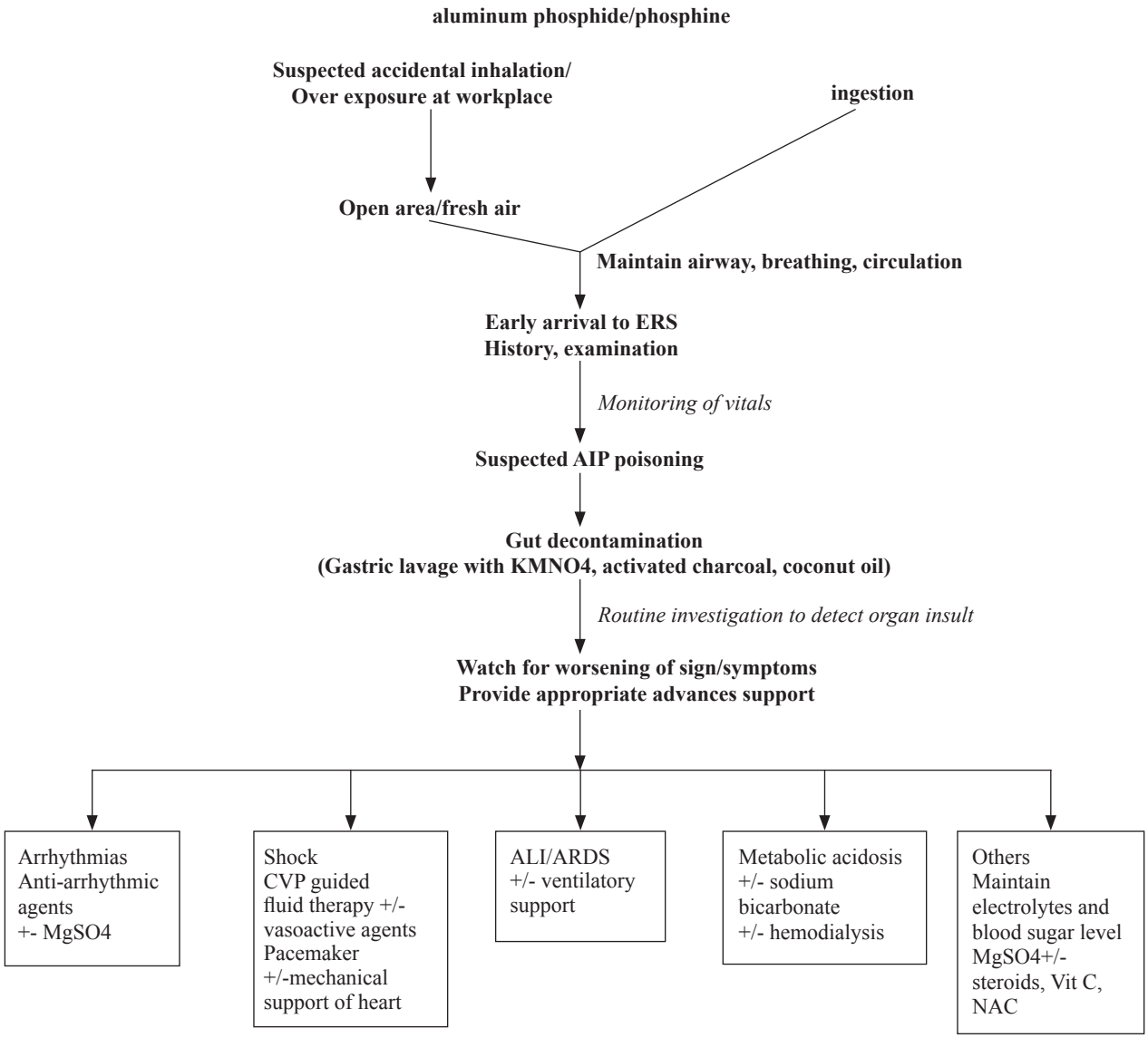

Figure 1: Emergency management of aluminum phosphide/phosphine poisoning

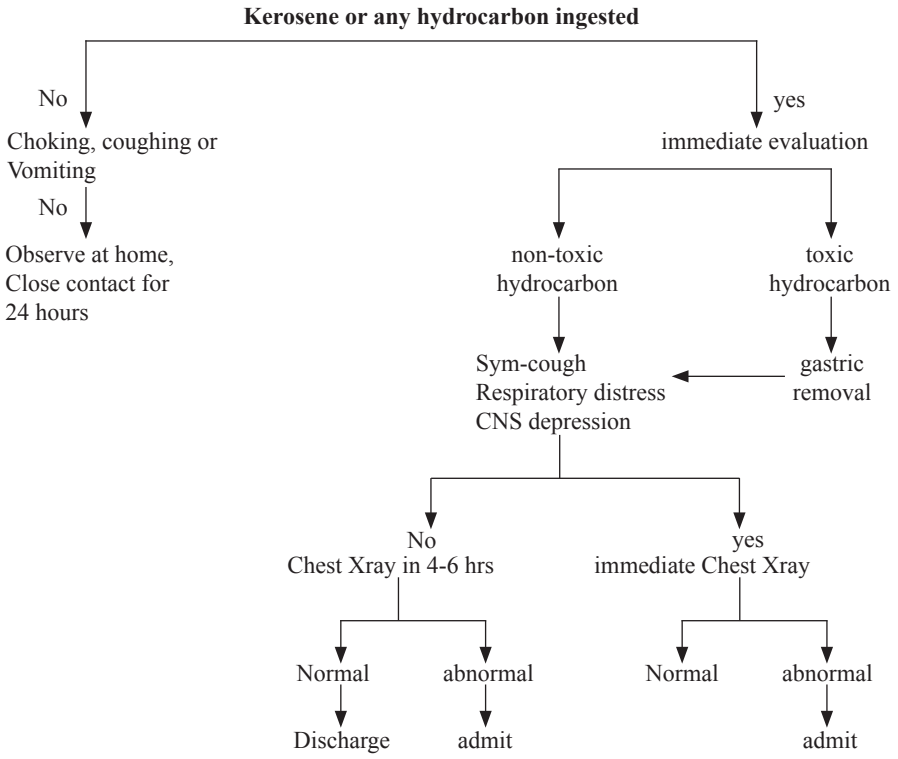

Figure 2: Approach to kerosene/petrol ingestion

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