In the name of GOD

POISONING

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OBJECTIVES:

- Understanding the importance of poisoning prevention
- Knowing the types of poisoning syndromes
- Acquaintance with different types of antidotes according to the type of poisoning
- The ability to perform the necessary measures for a poisoned patient

ETIOLOGY AND EPIDEMIOLOGY

- The most common agents ingested by young children include cosmetics, personal care products, cleaning solutions, and analgesics.
- fatal childhood poisonings were most commonly caused by analgesics, antihistamines , and sedative/hypnotics.
- Most ingestions occur at home (٩١%) and are single substance (٩٠%).

ETIOLOGY AND EPIDEMIOLOGY

- More than ٢ million human exposures are called into poison control centers in the United States each year. More than half of all exposures are in children with a male predominance in children under ١٣, but a female predominance in adolescence.

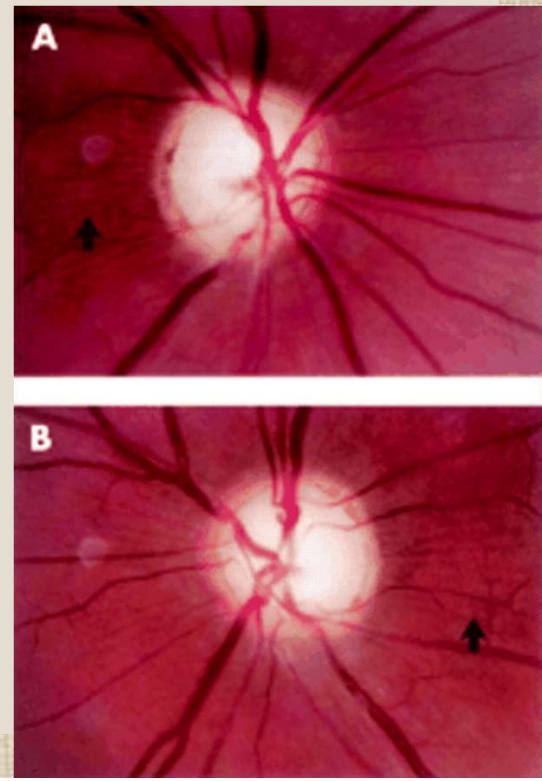
- Most ingestions in young children are unintentional, with intentional ingestions becoming more common in children ١٣ and older

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

Historical and Physical Findings in Poisoning:

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



Historical and Physical Findings in Poisoning:

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

Historical and Physical Findings in Poisoning:

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

Historical and Physical Findings in Poisoning:

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

CLINICAL MANIFESTATIONS

- 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.

CLINICAL MANIFESTATIONS

- A history and complete physical examination including vital signs often provide sufficient clues to distinguish between toxic ingestion and organic disease .

- Determination of all substances that the child was exposed to, type of medication, amount of medication, and time of exposure is crucial in directing interventions.

CLINICAL MANIFESTATIONS

A poisoned child can exhibit any one of six basic clinical patterns:

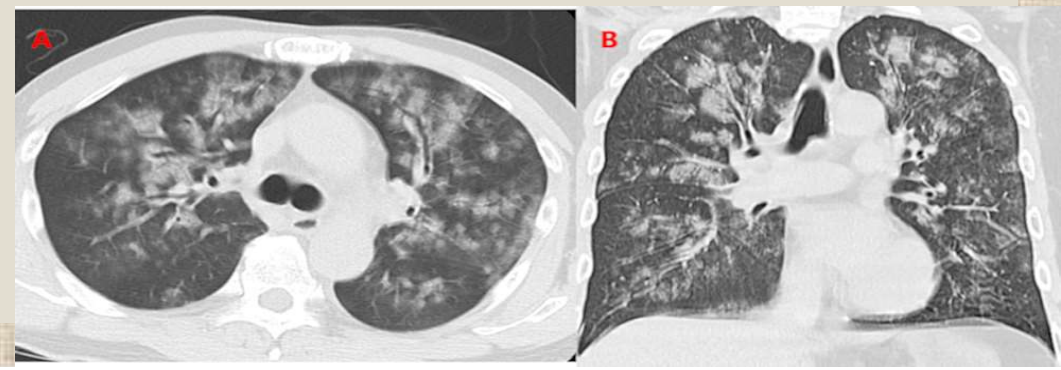
- Coma
- toxicity
- metabolic acidosis
- heart rhythm aberrations
- gastrointestinal symptoms
- seizures

COMA:

- coma is perhaps the most striking symptom of a poison ingestion.
- it also may be seen as a result of several other causes including trauma, a cerebrovascular accident, asphyxia, or meningitis.
- A careful history and clinical examination are needed to distinguish among these alternatives.

Direct Toxicity(Hydrocarbon):

- Hydrocarbon ingestion occasionally may result in systemic toxicity, but more often it leads to pulmonary toxicity.
- Hydrocarbons with low viscosity pose the greatest risk of producing aspiration pneumonia.
- Emesis or lavage should not be initiated in a child who has ingested volatile hydrocarbons.



Direct Toxicity(caustic):

- Caustic ingestions may cause dysphagia, epigastric pain, oral mucosal burns, and low-grade fever.
- Treatment depends on the agent ingested and the presence or absence of esophageal injury.
- Alkali agents may be solid, granular, or liquid. Liquid agents are tasteless and produce full-thickness liquefaction necrosis of the esophagus or oropharynx.

- Because acids taste sour, children usually stop drinking the solution, limiting the injury. Acids produce a coagulation necrosis, which limits the chemical from penetrating into deeper layers of the mucosa and damages tissue less severely than alkali.

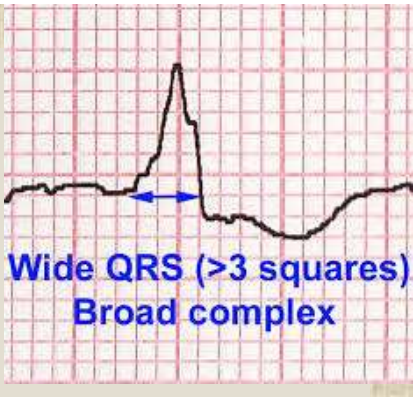
Direct Toxicity(caustic):

- When the esophageal lesions heal, strictures form. Ingestion of these agents also creates a long-term risk of esophageal carcinoma. Treatment includes dilation of late-forming strictures.
- Ingested button batteries also may produce a caustic mucosal injury.
- Batteries that remain in the esophagus may cause esophageal burns and erosion and should be removed with an endoscope.
- In addition, acid agents can injure the lungs (with hydrochloric acid fumes), oral mucosa, esophagus, and stomach.

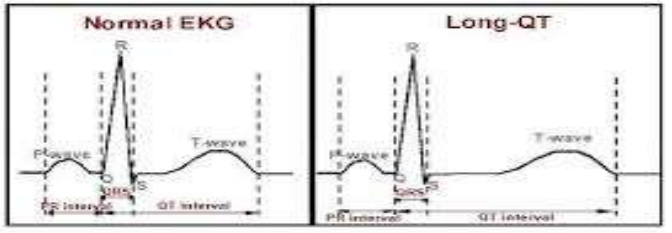
Metabolic Acidosis:

- A poisoned child may also have a high anion gap metabolic acidosis
- high anion gap metabolic acidosis is assessed easily by measuring arterial blood gases, serum electrolyte levels, and urine pH.
- An osmolar gap, if present, strongly suggests the presence of an unmeasured component, such as methanol or ethylene glycol.

Dysrhythmias:



- Prolonged Q-T intervals may suggest phenothiazine or antihistamine ingestion.
- widened QRS complexes are seen with ingestions of cyclic antidepressants and quinidine.
- Because many drug and chemical overdoses may lead to sinus tachycardia, this is not a useful or discriminating sign.
- sinus bradycardia suggests digoxin, cyanide, a cholinergic agent, or β blocker ingestion.



Gastrointestinal Symptoms:

- Gastrointestinal symptoms of poisoning include emesis, nausea, abdominal cramps, and diarrhea.

- These symptoms may be the result of direct toxic effects on the intestinal mucosa or of systemic toxicity after absorption.

Seizures:

- Seizures are the sixth major mode of presentation for children with toxic ingestions.
- poisoning is an uncommon cause of afebrile seizures.
- When seizures do occur with intoxication, they may be life-threatening and require aggressive therapeutic intervention.

Drugs Associated With Major Modes of Presentation:

COMMON TOXIC CAUSES OF CARDIAC ARRHYTHMIA

Amphetamine	Antiarrhythmics	Anticholinergics	Antihistamines
Arsenic	Carbon monoxide	Chloral hydrate	Cocaine
Cyanide	Cyclic antidepressants	Digitalis	Freon
Phenothiazines	Physostigmine	Propranolol	Quinine quinidine
Theophylline			

CAUSES OF COMA

Alcohol	Anticholinergics	Antihistamines	Barbiturates
Carbon monoxide	Clonidine	Cyanide	Cyclic antidepressants
Hypoglycemic agents	Lead	Lithium	Methemoglobinemia
Methyldopa	Narcotics	Phencyclidine	Phenothiazines
Salicylates			

Drugs Associated With Major Modes of Presentation:

COMMON AGENTS CAUSING SEIZURES (MNEMONIC = CAPS)

C:

Camphor Carbamazepine Carbon monoxide Cocaine Cyanide

A:

Aminophylline Amphetamine Anticholinergics Antidepressants (cyclic)

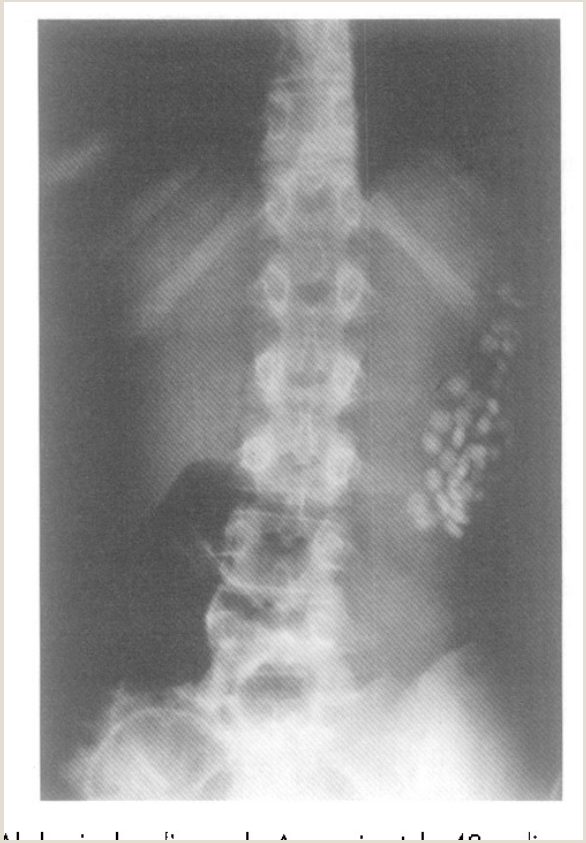
P:

Pb (lead) (also lithium) Pesticide (organophosphate) Phencyclidine
Phenol Phenothiazines Propoxyphene

S:

Salicylates Strychnine

Toxic Syndromes:



AGENT	MANIFESTATIONS
Acetaminophen	Nausea, vomiting, pallor, delayed jaundice–hepatic failure (72-96 hr)
Amphetamine, cocaine, and sympathomimetics	Tachycardia, hypertension, hyperthermia, psychosis and paranoia, seizures, mydriasis, diaphoresis, piloerection, aggressive behavior
Anticholinergics	Mania, delirium, fever, red dry skin, dry mouth, tachycardia, mydriasis, urinary retention
Carbon monoxide	Headache, dizziness, coma, other systems affected
Cyanide	Coma, convulsions, hyperpnea, bitter almond odor
Ethylene glycol (antifreeze)	Metabolic acidosis, hyperosmolarity, hypocalcemia, oxalate crystalluria
Iron	Vomiting (bloody), diarrhea, hypotension, hepatic failure, leukocytosis, hyperglycemia, radiopaque pills on KUB, late intestinal stricture, *Yersinia* sepsis
Narcotics	Coma, respiratory depression, hypotension, pinpoint pupils, bradycardia
Cholinergics (organophosphates, nicotine)	Miosis, salivation, urination, diaphoresis, lacrimation, bronchospasm (bronchorrhea), muscle weakness and fasciculations, emesis, defecation, coma, confusion, pulmonary edema, bradycardia
Salicylates	Tachypnea, fever, lethargy, coma, vomiting, diaphoresis, alkalosis (early), acidosis (late)
Cyclic antidepressants	Coma, convulsions, mydriasis, hyperreflexia, arrhythmia (prolonged Q-T interval), cardiac arrest, shock

LABORATORY AND IMAGING STUDIES:

- BLOOD:Laboratory studies helpful in initial management include specific toxin-drug assays; measurement of arterial blood gases and electrolytes, osmoles, and glucose; and calculation of the anion or osmolar gap.

- ECG: A full ١٢-lead electrocardiogram should be part of the initial evaluation in all patients suspected of ingesting toxic substances.

LABORATORY AND IMAGING STUDIES:

- URIN:Urine screens for drugs of abuse or to confirm suspected ingestion of medications in the home may be revealing. Quantitative toxicology assays are important for some agents not only for identifying the specific drug, but also for providing guidance for therapy, anticipating complications, and estimating the prognosis

LABORATORY AND IMAGING STUDIES:

- Toxicology
- ABG
- Electrolytes
- Glucose
- Anion Gap
- ECG
- KUB(some cases)

Screening Laboratory Clues in Toxicological Diagnosis:

HYPOGLYCEMIA:
Ethanol
Isoniazid
Insulin
Propranolol
Oral hypoglycemic agents

ANION GAP METABOLIC ACIDOSIS (MNEMONIC = MUDPILES)
Methanol,* metformin
Uremia*
Diabetic ketoacidosis*
Paraldehyde,* phenformin
Isoniazid, iron
Lactic acidosis (cyanide, carbon monoxide)
Ethanol,* ethylene glycol*
Salicylates, starvation, seizures

Screening Laboratory Clues in Toxicological Diagnosis:

HYPERGLYCEMIA:
Salicylates
Isoniazid
Iron
Phenothiazines
sympathomimetics

HYPOCALCEMIA:
Oxalate
Ethylene glycol
fluoride

RADIOPAQUE SUBSTANCE ON KUB (MNEMONIC = CHIPPED)
Chloral hydrate, calcium carbonate
Heavy metals (lead, zinc, barium, arsenic, lithium, bismuth as in Pepto-Bismol)
Iron
Phenothiazines
Play-Doh, potassium chloride
Enteric-coated pills
Dental amalgam

TREATMENT:

The four foci of treatment for poisonings are:

- supportive care
- Decontamination
- enhanced elimination
- specific antidotes

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.(ABC)
- If the level of consciousness is depressed and a toxic substance is suspected, glucose (١ g/kg intravenously), ١٠٠% oxygen, and naloxone should be administered.

Gastrointestinal Decontamination:

- The intent of gastrointestinal decontamination is to prevent the absorption of a potentially toxic ingested substance
- **Syrup of ipecac:** should not be administered routinely to poisoned patients because of potential complications and lack of evidence that it improves outcome.
- **gastric lavage:** should not be used routinely, if ever, in the management of poisoned patients because of the lack of efficacy and potential complications.
- **Single-dose activated charcoal:** decreases drug absorption when used within ١ hour of ingestion; however, it has not been shown to improve outcome.

Gastrointestinal Decontamination:

- **Charcoal is ineffective** against caustic or corrosive agents, hydrocarbons, heavy metals (arsenic, lead, mercury, iron, lithium), glycols, and water-insoluble compounds.

- The administration of a cathartic (sorbitol or magnesium citrate) alone has no role in the management of the poisoned patient.

- **Whole-bowel irrigation using polyethylene glycol** (GoLYTELY) as a nonabsorbable cathartic may be effective for toxic ingestion of sustained-release or enteric-coated drugs.

Enhanced Elimination:

- Multiple-dose activated charcoal should be considered only if a patient has ingested a life-threatening amount of carbamazepine, 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.

specific antidotes:

POISON	ANTIDOTE
Acetaminophen	*N*-Acetylcysteine
Benzodiazepine	Flumazenil
β-Blocking agents	Glucagon
Carbon monoxide	Oxygen
Cyclic antidepressants	Sodium bicarbonate
Iron	Deferoxamine

POISON	ANTIDOTE
Lead	Edetate calcium disodium (EDTA) British anti-Lewisite (BAL; dimercaprol) Succimer (2,3-dimercaptosuccinic acid ([DMSA])
Nitrites/ methemoglobinemia*	Methylene blue
Opiates	Naloxone
Organophosphates	Atropine Pralidoxime (2 PAM; Protopam)

PROGNOSIS:

- Most poisonings result in minimal or no toxicity, or have minor effects.

- Intentional ingestions result in a much higher rate (٥٫٢%) of major effects or death compared with unintentional ingestions (٠٫٢%).

- Adolescents are more likely to have a moderate, major, or fatal effect from ingestion compared to younger children (١٧٫٣% of teens compared with ١٫١% of children under ٦ years).

PREVENTION:

- Properly educating parents regarding safe storage of medications and household toxins is necessary for preventing ingestions.

- If a child has ingested poison, a poison control center should be called.

REFERENCES:

- Nelson Essentials of Pediatrics ٢٠٢٣
- Nelson Textbook of Pediatrics ٢٠٢٠

Thank you for your attention