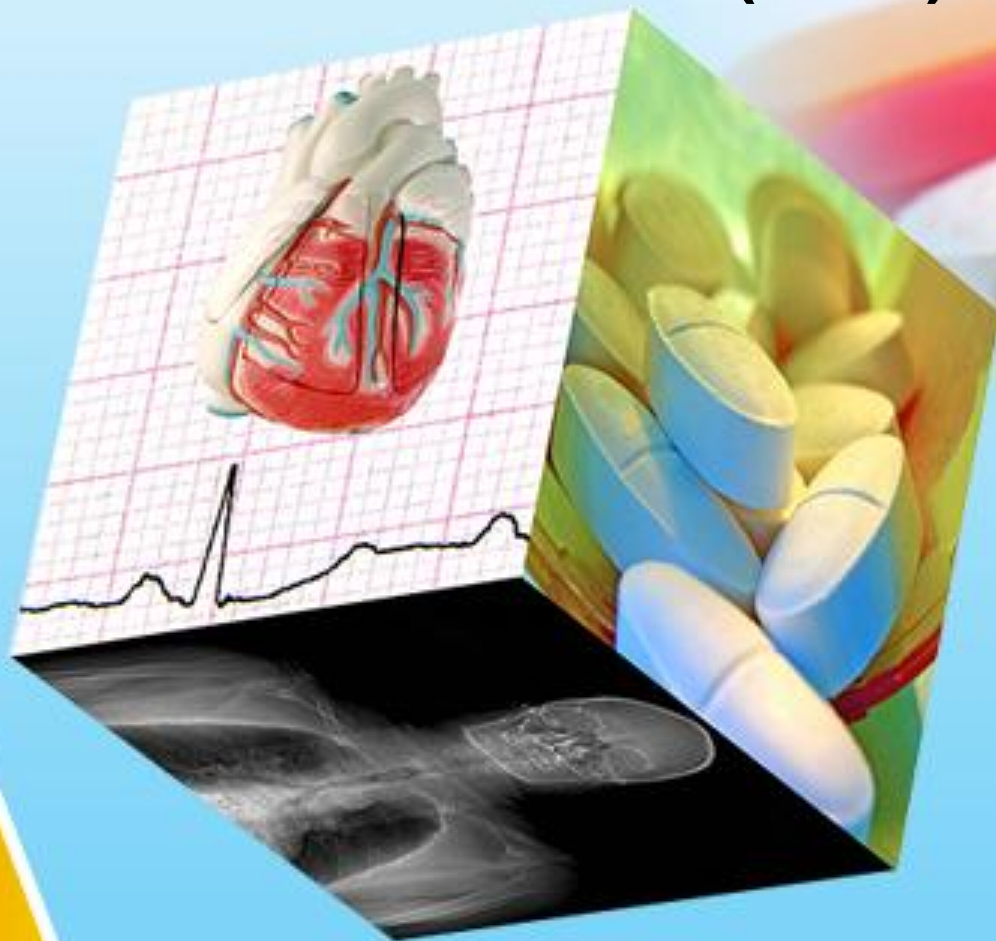


HEAVY METALS

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IRON



IRON

- children younger than age 6 years : minimally toxic.
- Adult : suicide attempts.



Principles of Disease Pharmacology



- Normal serum iron levels : 50 to 150 micg/dL.
- The total iron-binding capacity (TIBC), a crude measure of the ability of serum proteins—including transferrin—to bind iron, ranges from 300 to 400 micg/dL.

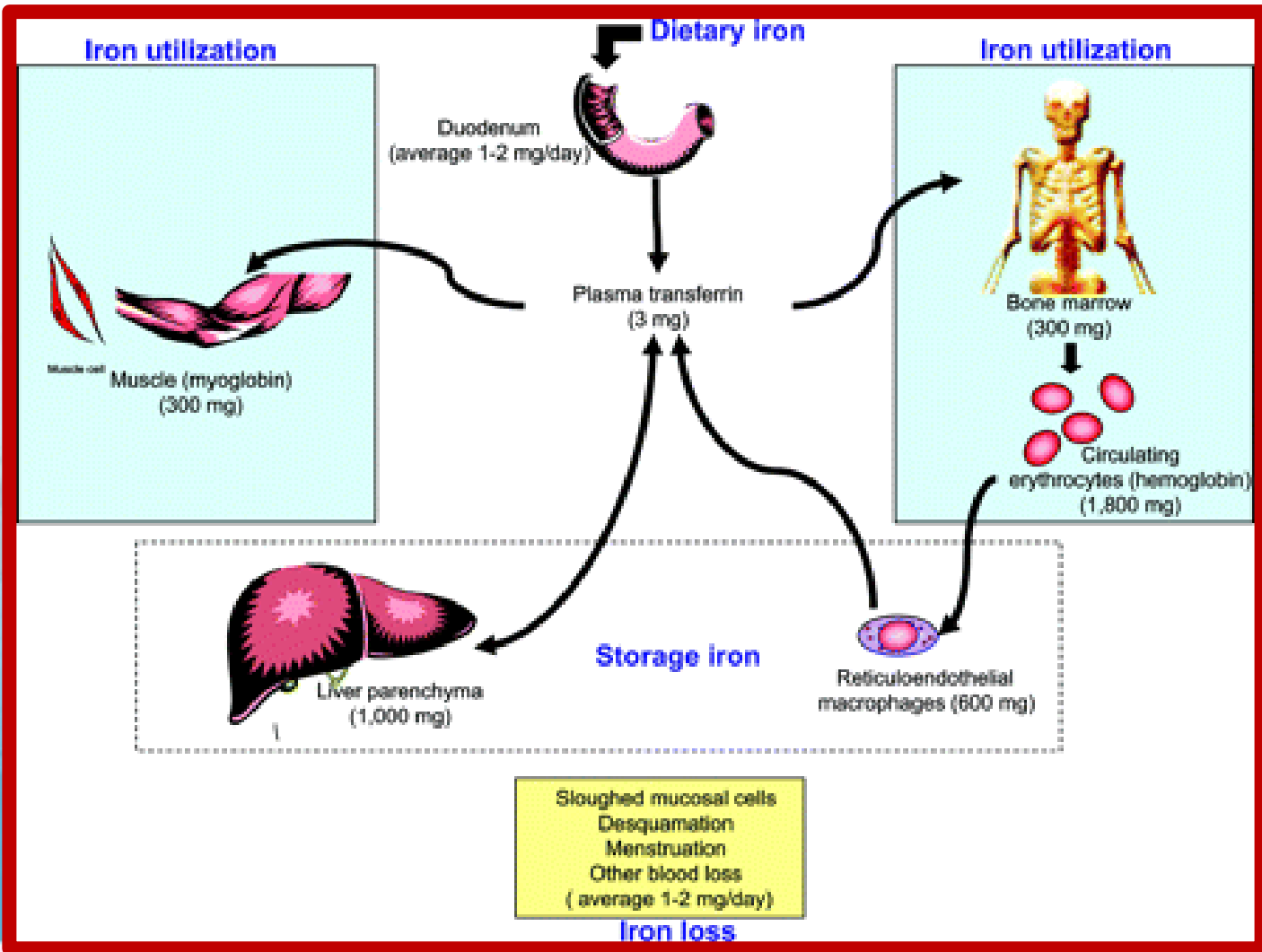


Principles of Disease Pharmacology



- It is higher than the serum iron level due to a low degree of saturation.
- When iron levels rise following a significant iron overdose, transferrin becomes saturated so that excess iron circulates as free iron in the serum.
- This unbound iron is directly toxic to target organs.





Principles of Disease Pharmacology



- Ingestions of less than 20 mg/kg of elemental iron usually cause no symptoms.
- Ingestion of 20 to 60 mg/kg results in mild to moderate symptoms
- ingestion of more than 60 mg/kg may lead to severe morbidity.
- 50% mortality (LD_{50}) is reported to be 200 to 250 mg/kg.



Common Iron Preparations

COMPOUND	PERCENTAGE OF ELEMENTAL IRON
Ferrous sulfate	20
Ferrous fumarate	33
Ferrous gluconate	12
Ferric pyrophosphate	30
Ferrocholate	14
Ferroglycine sulfate	16
Ferrous sulfate, dried	33
Ferrous carbonate, anhydrous	38
Carbonyl iron	100

Pathophysiology

- Two distinct toxic effects:
- (1) it causes direct caustic injury to the gastrointestinal mucosa
- (2) it impairs cellular metabolism, primarily of the heart, liver, and central nervous system (CNS).



Pathophysiology

- Unbound (free) iron moves into cells and localizes near the mitochondrial cristae, resulting in uncoupling of oxidative phosphorylation and impairment of adenosine triphosphate synthesis.
- Cell membranes are injured by free radical-mediated lipid peroxidation.



Pathophysiology

- Iron increases capillary permeability and induces both arteriolar and venodilation.
- Myocardial toxicity decreases cardiac output.
- Hydration of the iron molecule creates an excess of unbuffered protons, worsening metabolic acidosis.
- This multitude of effects, combined with severe gastrointestinal fluid losses, can lead to the development of shock, cardiovascular collapse, and death.

Clinical Features

Five stages.

- Phase I reflects the corrosive effects of iron on the gut. Vomiting occurs within 80 minutes of ingestion in more than 90% of symptomatic cases. Diarrhea, which can be bloody, follows.
- Phase II represents an apparent (but not complete) recovery that lasts less than 24 hours but can extend up to 2 days. Most patients recover after this point.



Clinical Features



- Phase III is characterized by the recurrence of GI symptoms, severe lethargy or coma, anion gap metabolic acidosis, leukocytosis, coagulopathy, renal failure, and cardiovascular collapse.

Serum iron levels may have fallen to normal during this phase due to distribution into the tissues.



Clinical Features



- Phase IV, characterized by fulminant hepatic failure, occurs 2 to 5 days after ingestion. This is relatively rare, appears to be dose related, and is usually fatal.
- Phase V represents the consequences of healing the injured gastrointestinal mucosa.

It is characterized by pyloric or proximal bowel scarring, which is sometimes associated with obstruction.



Diagnostic Strategies

- The presence of gastrointestinal symptoms suggests a potentially serious ingestion, whereas their absence is reassuring.
- A serum iron level: 3 to 5 hours after ingestion, is the most useful laboratory test to evaluate the potential severity of an iron overdose.



Diagnostic Strategies

- Peak serum iron levels of less than 350 micg/dL are with minimal toxicity,
- 350 to 500 micg/dL with moderate toxicity
- **and greater than 500 micg/dL with potentially severe toxicity.**



Toxicity of Iron by Amount Ingested and Peak Serum Levels

ELEMENTAL IRON (MG/KG)	PEAK SERUM IRON (μ G/DL)	TOXICITY
<20	50–150	None
20–40	150–300	Mild
40–60	300–500	Moderate
>60	>500	Severe



Radiopaque iron tablets (arrow) seen on abdominal radiograph



Management

Gastric Emptying

- Iron is not bound to activated charcoal
- gastric lavage nor ipecac : NOT EFFECTIVE!
- Iron tablets clump together as their outer coatings dissolve.
- Gastrotomy has been
 - performed to remove iron
 - from the stomach.



Whole-Bowel Irrigation

- Polyethylene glycol electrolyte lavage solution (PEG-ELS) (CoLyte, NuLytely, or GoLYTELY) is routinely recommended.
- The solution is either taken orally or administered through a nasogastric tube.
- Rate of administration of PEG-ELS is 20 to 40 mL/kg/hr in young children and 1.5 to 2 L/hr for teenagers or adults, continued until the rectal effluent is clear and there is no radiographic evidence of pill fragments.



Management

- Whole-bowel irrigation is contraindicated in the presence of bowel obstruction, perforation, or ileus.



Management

- Hemodialysis and hemoperfusion are not effective in removing iron due to its large volume of distribution.
- Exchange transfusions have been recommended for severely symptomatic patients with serum iron levels exceeding 1000 micg/dL.



Management

Deferoxamine

- Deferoxamine chelates iron to form the water soluble compound ferrioxamine, which can be renally excreted or dialyzed.
- 100 mg : chelate 9.35 mg of elemental iron.
- Because of its short half-life, it is administered as a continuous infusion at a dose of 15 mg/kg/hr for up to 24 hours.
- The maximum rate of administration is 35 mg/kg/hr.



Management

Deferoxamine



- Rapid administration of deferoxamine can lead to hypotension, which is treated by reducing the initial rate of the infusion and slowly increasing it to the desired rate.
- Pregnancy is not a contraindication to deferoxamine.



Management

Deferoxamine

- The presence of ferrioxamine turns the urine a “vin ros?” color, which reflects the excretion of chelated iron.



LEAD



LEAD

- Lead poisoning is a disease of industrialization.
- Exposure usually results from ingestion or inhalation.
- it may results from direct skin contact with organic lead compounds or from retained bullets in or near joints.



LEAD

- Lead-based paint
- Curtain weights
- Buckshot
- Fishing weights
- Lead-contaminated soil or water
- Food or beverages stored or prepared in lead-soldered cans
- Lead-glazed pottery
- Lead crystal decanters



LEAD

- Children typically present to the emergency department
- (1) following an ingestion of lead
- (2) symptomatic with a possible exposure history
- (3) referred for management of an elevated BLL.

Lead toxicity in adults most often results from inhalational exposure in the workplace, as well as from hobbies and related activities.



LEAD

- Battery manufacture, radiator repair, bridge and ship construction or demolition, soldering or welding, cable or tin can production, stained glass manufacture, lead-glazed or crystal pottery making, glass production, firing range operation, and lead-based paint abatement.
- Hobbies at risk include making glazed pottery, target shooting at indoor firing ranges, soldering lead, smelting lead in the preparation of buckshot and fishing sinkers, repairing cars or boats, and remodeling homes.



Principles of Disease Pharmacology



- There is no known biologic need for lead.
- Its absorption is highest in malnourished children (approximately 40%) and in pregnant women.
- Although 90 to 95% of lead is stored in cortical bone and teeth, it is also found in the brain, liver, and kidneys.
- Approximately 75% of the absorbed lead is eliminated by the kidneys, with the remainder absorbed through the skin, hair, sweat, nails, and gastrointestinal tract.



Pathophysiology

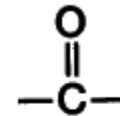
- Lead binds to sulfhydryl groups : interferes with critical enzymatic reactions.

Its toxic effects are most prominent in

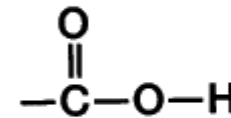
- Hematopoietic
- Neurologic
- Renal systems.



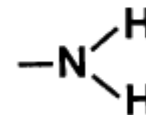
Hydroxyl group



Carbonyl group



Carboxyl group



Amino group



Sulfhydryl group



Pathophysiology

- Anemia : normochromic or hypochromic.
- The severity of the anemia correlates directly with the BLL. Blood Lead Level
- In the peripheral nervous system, segmental demyelination and degeneration of motor axons result in peripheral neuropathies.



Pathophysiology

- Wrist drop and foot drop are characteristic of adult lead poisoning.
- Lead toxicity also causes neuropsychiatric disorders.
- In children, LOW (IQ) scores, hyperactivity, decreased attention span, overaggressive behavior, learning disabilities, criminal behavior, and subclinical sensorineural hearing loss.

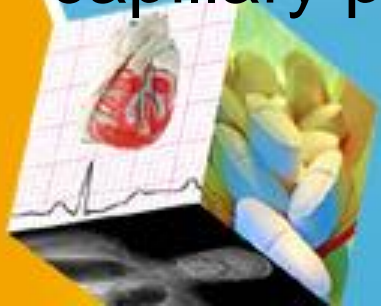


Pathophysiology



- Lead nephropathy is fibrosis in the proximal tubules, with relative sparing of the glomeruli.
- Hyperuricemic gout (“saturnine gout”) can result from increased reuptake of uric acid by the tubular cells.
- Lead poisoning has also been correlated with hypertension.

Adults and children with acute toxicity may present with lead encephalopathy associated with increased capillary permeability and cerebral edema



Clinical Features

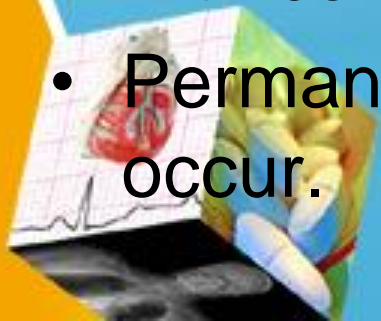
- Symptoms of chronic, mild lead poisoning are slow in onset and nonspecific.
- The diagnosis is suspected by obtaining an accurate and comprehensive history of exposure to lead.



Clinical Features



- Acute exposure to lead can result in symptomatic poisoning.
- “Lead colic” is characterized by cramping abdominal pain with nausea, vomiting, constipation, and, occasionally, diarrhea.
- Fatigue, anemia, peripheral neuropathy, renal impairment, and hepatic and CNS dysfunction.
- The CNS toxicity may manifest as mild headache or personality changes to full-blown encephalopathy with coma, convulsions, and papilledema.
- Permanent neurologic and behavioral sequelae may occur.



Diagnostic Strategies

- Chronic BLL of greater than 10 micg/dL is toxic for a child.
- Acute exposure can result in levels up to 100 micg/dL.



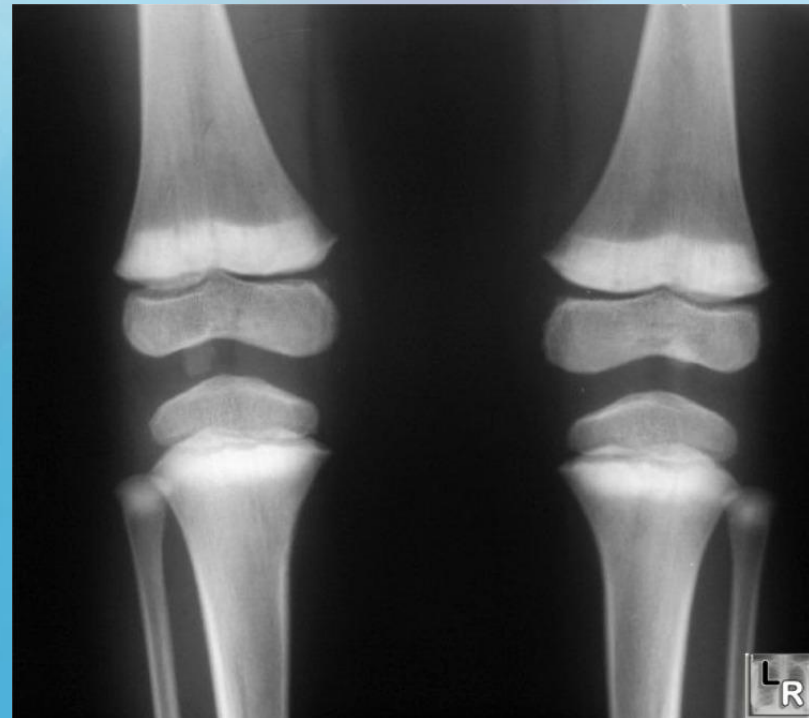
Diagnostic Strategies

- blood cell count, serum glucose, blood urea nitrogen, creatinine, electrolyte levels, and urinalysis.
- A peripheral smear may show basophilic stippling.
- Markers of hepatic injury may be elevated following acute exposure.



Diagnostic Strategies

- In cases of altered mental status, seizures, or coma, a CT of the head will show cerebral edema associated with acute lead encephalopathy
- In children :
“lead bands” or
“lead lines” that
are characteristic of
chronic exposures.



Serum Lead Levels and Symptomatology

LEVEL ($\mu\text{G}/\text{DL}$)	SYMPTOMS	
	ADULTS	CHILDREN
10	None	Decreased intelligence Decreased hearing Decreased growth
20	Increased protoporphyrin No symptoms	Decreased nerve conduction velocity Increased protoporphyrin
30	Increased blood pressure Decreased hearing	Decreased vitamin D metabolism
40	Peripheral neuropathies Nephropathy Infertility (men)	Decreased hemoglobin synthesis
50	Decreased hemoglobin synthesis	Lead colic
70	Anemia	Anemia Encephalopathy Nephropathy
100	Encephalopathy	Death

Management

Acute Lead Encephalopathy

- Standard measures to control cerebral edema, including intubation and neurosurgical consultation for invasive monitoring of ICP are indicated.



whole-bowel irrigation

1. Severe poisoning
 2. Radiopacities
- Activated charcoal does not adsorb lead.



Chelation Therapy

- Any patient with a BLL greater than 70 micg/dL, or with signs suggestive of encephalopathy, will require admission for parenteral chelation therapy.
- For these seriously poisoned patients, dimercaprol (or British anti lewisite [BAL]) should be the first chelator given.



Chelation Therapy



- The dosage is 3 to 5 mg/kg (25 mg/kg/day), given by deep intramuscular injection every 4 hs for 2 days, followed by a dose every 4 to 6 hs for 2 more days and then every 4 to 12 hs for up to 7 days.
- Dimercaprol forms complexes that undergo both renal and biliary excretion.
- Adverse reactions to dimercaprol include nausea, vomiting, urticaria, pyrexia, hypertension, and hemolysis in patients with glucose-6-phosphate dehydrogenase deficiency.



Chelation Therapy

- Since dimercaprol is diluted in peanut oil, it is contraindicated in patients allergic to peanuts.
- Dimercaprol is followed by calcium disodium ethylenediaminetetraacetic acid (**CaNa₂EDTA**)



Chelation Therapy

- The dosage of CaNa_2EDTA for patients with acute lead encephalopathy is 75 mg/kg/day or 1500 mg/m²/day given IV or IM in two to four divided doses, with a maximum daily dose of 1 g in children and 2 g in adults.
- CaNa_2EDTA should be given only with adequate urine flow or with hemodialysis in renal failure.



Chelation Therapy

- The need for parenteral chelation therapy in asymptomatic or minimally symptomatic children is guided by the BLL.
- A BLL of more than 69 micg/dL mandates hospitalization and parenteral chelation therapy.
- For less seriously poisoned patients, the dosage of CaNa_2EDTA is 50 mg/kg/day or 1000 mg/m²/day, given in two to four divided doses for up to 5 days.



Chelation Therapy

- (2,3-dimercaptosuccinic acid :DMSA)

for Serum lead levels of 45 to 69 micg/dL in patients without vomiting or CNS symptoms can be managed in the outpatient setting.

- The initial dose of DMSA is 10 mg/kg every 8 hours for 5 days, then 10 mg/kg every 12 hours for 14 days.



Chelation Therapy

- Oral d-penicillamine should be used only in patients who do not tolerate DMSA.
- The usual oral dose of d-penicillamine is 25 mg/kg every 6 hours for 5 days.
- d-Penicillamine is less efficacious than DMSA and has more adverse reactions.
- Penicillin allergy is a contraindication to the use of d-penicillamine.



Chelation Therapy



A BLL between 20 and 44 micg/dL in a patient who is asymptomatic or minimally symptomatic requires a more aggressive medical and environmental evaluation.



Chelation Therapy



- No need for chelation for children with a BLL lower than 45 micg/dL.
- Children with lead levels of 10 to 19 micg/dL need :
 - * family counseling
 - * careful follow-up
 - * frequent screening of BLLs



Chelation Therapy



- The treatment of adults with chronic poisoning is less aggressive than for children.
- If gastrointestinal symptoms or CNS problems are present, hospitalization with parenteral chelation therapy is indicated.
- In the asymptomatic adult or the adult with only mild clinical problems, the only intervention needed is cessation of exposure.



Disposition

- Patients who have ingested a single lead foreign body (e.g., fishing sinker) will usually pass it harmlessly.
- If the foreign body remains in the gastrointestinal tract after 2 weeks, removal should be considered to prevent lead toxicity.



Disposition

- Patients who are significantly symptomatic after an acute lead exposure and children with a BLL of 69 micg/dL or greater require hospitalization and chelation therapy.
- Patients discharged home on oral chelation therapy should not return to a contaminated environment.



ARSENIC



ARSENIC

- Arsenic (As), a tasteless, odorless substance that looks like sugar, has an infamous history as an agent of homicide.

- Epidemic poisoning.

Found in: smelters and electric power plants that burn arsenic-rich coal.



ARSENIC

- It is used in industry as a wood preservative and in the production of glass and microcircuits.
- Inorganic arsenicals are also used in rodenticides, fungicides, insecticides, paint, and tanning agents and as defoliants in the cotton industry.
- Arsenic is still used for medicinal purposes in the treatment of trypanosomiasis, amebiasis, and leukemia.



ARSENIC

- It has also been found as a contaminant in herbal remedies and drugs such as opium.
- Contaminated drinking water in underdeveloped countries.



Principles of Disease

Pharmacology

- Arsenic has no metabolic or biologic function.
- Of the two inorganic forms, trivalent arsenite (As^{3+}) is highly lipid soluble and is 5 to 10 times more toxic than the pentavalent arsenate (As^{5+}) form.
- The more toxic lipophilic trivalent arsenite form has a lower gastrointestinal absorption but is well absorbed by the skin.



Principles of Disease

Pharmacology

- Absorbed arsenic is bound by hemoglobin, leukocytes, and plasma proteins.
- It is cleared from the intravascular compartment within 24 hours and concentrates in the liver, kidneys, spleen, lungs, and gastrointestinal tract.
- Arsenic crosses the placenta and can also accumulate in the fetus.
- Its affinity for sulfhydryl groups in keratin makes arsenic detectable in the hair, skin, and nails.



Principles of Disease

Pharmacology

- **Arsine (AsH_3)**, a colorless and almost odorless gas, is extremely toxic.
- It is immediately lethal at 250 ppm.
- The excretion of arsenic and its metabolites occurs mainly through the kidneys.



Pathophysiology

- Arsenic binds avidly to sulfhydryl groups, inhibiting critical enzymes such as lactate dehydrogenase and glyceraldehyde-3-phosphate dehydrogenase, a critical step in glycolysis.
- It disrupts oxidative phosphorylation by replacing phosphorus in the formation of phosphate bonds (arsenolysis).
- Arsine causes massive hemolysis.



Clinical Features

- Acute exposure to arsine gas is characterized by severe hemolysis that is associated with renal tubular injury.
- Gastrointestinal symptoms are common, and CNS and liver dysfunction can occur.
- The mortality rate is 25 to 30%.
- Exchange transfusions and plasma exchange have been used to remove arsine, which is tightly bound to the erythrocytes.
- Urinary alkalization can be used to decrease renal deposition of hemoglobin.



Clinical Features

- GIT: nausea, vomiting, abdominal pain, and diarrhea
Initial manifestations of acute exposure to arsenic salts.
- Hematemesis and Hematochezia.
- Within 30 to 60 minutes of exposure, patients complain of a metallic or garlicky taste.
- Encephalopathy with seizures and coma, respiratory failure associated with ARDS and dysrhythmias associated with cardiac conduction disturbances.



Clinical Features

- Severe poisoning : cardiovascular collapse and death.
- Less common complications include hepatitis, rhabdomyolysis, hemolytic anemia, renal failure, unilateral facial nerve palsy, pancreatitis, pericarditis, pleuritis ,and fetal demise.



ACUTE EFFECTS OF ARSENIC POISONING

Gastrointestinal

Violent gastroenteritis
Hematemesis/hematochezia
Jaundice
Pancreatitis
Dysphagia
Hepatomegaly

Cardiovascular

Third spacing with shock
Sinus/ventricular tachycardia
Prolonged QT interval, ST depression, T wave inversion
Torsades de pointes
Pericarditis

Respiratory

Respiratory failure
Adult respiratory distress syndrome
Pulmonary edema
Pneumonia

Renal

Proteinuria
Hematuria
Oliguria
Renal failure

Neurologic

Headache
Drowsiness
Delirium
Coma
Encephalopathy
Seizures

Clinical Features

Weeks to Months later on :

Characteristic lines in the nails (Mees' lines),
sensorimotor neuropathy
hyperkeratosis of the palms and soles.

.



Diagnostic Strategies

- Normal arsenic levels are
Blood: 5 micg/L
Urine : less than 50 micg/day in a (24-hour urine)
- Any urine level above 100 micg/day or 50 micg/L necessitates treatment.



Management

- The initial management should address life-threatening conditions with supportive management of shock, dysrhythmias, and seizures.
- No Activated charcoal, does not adsorb arsenic



Management

- Hemodialysis removes arsenic in the setting of acute renal failure.
- Exchange transfusions or plasma exchange should be considered very early after an arsine exposure



Management

- With a known history of exposure in asymptomatic patient, chelation should start as early as possible without waiting for laboratory confirmation of the arsenic levels.
- Intramuscular dimercaprol is the preferred chelator in patients who are critically ill.
- DMSA is a water-soluble analogue of dimercaprol that can be given orally.



MERCURY



MERCURY

- Mercury is a silver white metal
- the only metal that is liquid at room temperature.
- It has a long history of medicinal uses.
- Significant poisoning at home:
 - *Sphygmomanometer mercury spilled then was aerosolized by vacuuming
 - * mercury was heated on the kitchen stove to extract gold from ore.



MERCURY

- Various other sources of mercury have also been implicated in intoxication.
- Because of many industrial uses that include the manufacture of fluorescent lights, batteries, polyvinyl chloride, and latex paint, mercury is a common pollutant of air and water.



SOURCES OF MERCURY

Elemental

- Spill from mercury-containing devices
- Gastrointestinal exposure from ruptured Cantor or Miller-Abbott tube
- Inhalational exposure in the workplace/home
- Deliberate injection or ingestion
- Accidental ingestion

Salts

- Accidental disk battery ingestion
- Deliberate ingestion
- Laxative abuse

Organic

- Oral/dermal exposure to mercurochrome or thimerosal
- Repeated injections of drugs containing thimerosal as a preservative
- Exposure from occupational or agricultural accidents
- Water/soil pollution
- Consumption of contaminated seafood
- Exposure to paint containing mercury

Principles of Disease

Pharmacology



- The most familiar form of mercury is elemental or metallic mercury, also known as “**quicksilver**.”
- A common route : inhalation of volatilized vapor.
- After inhalation, 74% of the metallic mercury is retained in the lungs. This can result in severe pneumonitis and ARDS



Principles of Disease

Pharmacology

- Aspiration of elemental mercury results in primary pulmonary toxicity, in addition to CNS and renal toxicities.
- Elemental mercury is not absorbed by the gastrointestinal tract, so ingestion does not normally lead to systemic toxicity unless it becomes trapped in diverticulae.
- Mercury is absorbed through the skin at 1% of the rate of inhaled mercury and is not a concern.

Principles of Disease

Pharmacology

- Inorganic mercury

have two different forms: Hg^{1+} (*mercurous*) and Hg^{2+} (*mercuric*).

Ingestion of either salt leads to significant gastrointestinal and renal toxicity.



- The organic mercury

The major route of exposure to this type of mercury is through ingestion, but these compounds are also readily absorbed through the skin.

- These organic forms classically result in delayed neurotoxicity with prominent ataxia, tremor, dysarthria, and tunnel vision.



Pathophysiology

- Mercury binds sulfhydryl groups.
- Nephrotoxicity :direct damage and an immune reaction in the kidney.
- The skin changes: immune reaction.
- Mercury increases catecholamine level resulting in hypertension and tachycardia.
- Atrophy of the cerebellum.



Clinical Features

- Inhalation of elemental mercury onset of shortness of breath, fever, and chills that progresses to pneumonitis and respiratory distress.
- Aspiration of liquid metallic mercury during medical procedures results in the rapid onset of tracheobronchial hemorrhage



Clinical Features

- Acute ingestion of inorganic salts typically causes a corrosive gastroenteritis with third spacing and hemorrhage.
- Patients complain of a metallic taste in the mouth and may have a grayish discoloration of the mucous membranes.
- Massive fluid loss results in shock and acute tubular necrosis.



Mercury Intoxication Syndromes

TYPE OF MERCURY/ROUTE OF EXPOSURE	SIGNS/SYMPTOMS
Inhalation of metallic mercury	Hypoxemia Respiratory distress, ARDS Dyspnea, chest tightness Fever, chills Burning in mouth and throat Nausea, vomiting Bloody diarrhea Renal tubular necrosis
Aspiration of metallic mercury	Aspiration pneumonitis ARDS
Subacute/chronic inhalation of metallic mercury	Metal fume fever Neuropsychiatric symptoms Renal dysfunction Skin changes
Ingestion of inorganic mercury salts	Severe hemorrhagic gastroenteritis, shock, hypovolemia, third spacing Acute tubular necrosis in 24 hr, with albuminuria and hematuria
Subacute/chronic inhalation of inorganic mercury	Neurasthenia, erethism, acrodynia
Organomercury exposure (methyl-, diethyl-)	Delayed neurologic problems (ataxia, tremor, dysarthria), visual field constriction, hearing loss, spasticity, hyper-reflexia

ARDS, acute respiratory distress syndrome.

Diagnostic Strategies

- “Normal” mercury levels are considered to be
- Blood: less than 10 micg/L
- Blood level more than 35 micg/L needs Rx.



Management

- Initial management in the acutely poisoned patient should be aggressive support and decontamination.
- Gastric lavage with protein-containing solutions (e.g., milk and egg whites) may be beneficial in the decontamination of the gastrointestinal tract following ingestion of mercury salts.



Management

- Charcoal adsorbs very little and is not recommended
- Ingested metallic mercury is generally harmless unless its passage is impaired by entrapment in a diverticulum or the appendix.



Management

- For acute inhalational exposures
- patient should be removed from the source
- supportive management provided.

There is no role for prophylactic antibiotics or steroids.

Suction and postural drainage are indicated in cases of acute aspiration of metallic mercury.

Self-injection of metallic mercury often requires surgical debridement of infiltrated tissue



Chelation Therapy

- BAL is used for clinically significant acute inorganic mercury intoxication.
- Because it increases brain mercury levels in patients with methylmercury poisoning, BAL is contraindicated for patients poisoned with organic mercury compounds.



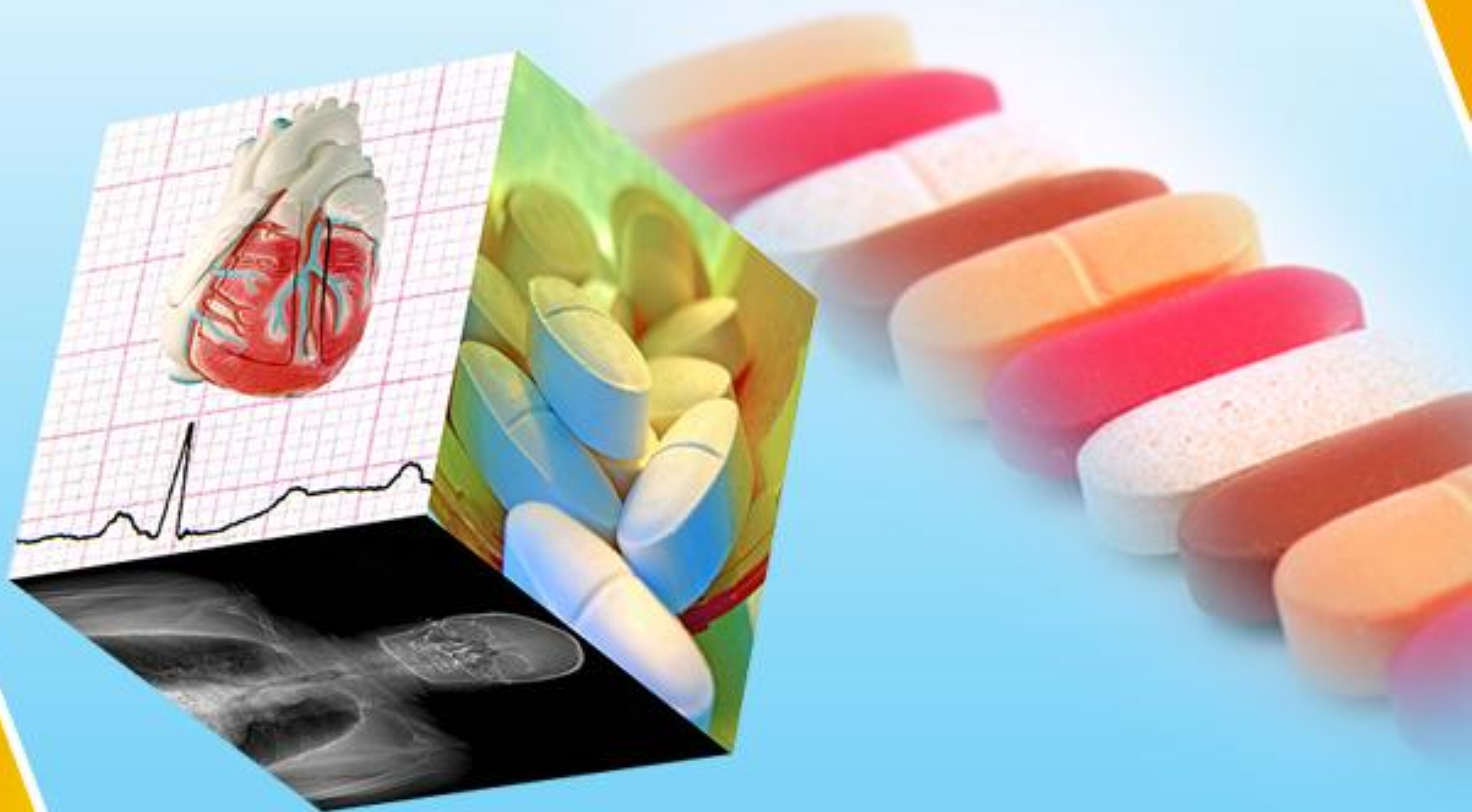
Chelation Therapy

- DMSA :used for both acute and chronic mercury poisoning and may be the best chelator for methylmercury.
- d-Penicillamine is also used.

It should be administered only after thorough gastrointestinal decontamination because mercury absorption from the intestinal lumen is enhanced by the penicillamines.



<http://fac.ksu.edu.sa/talmezeiny/home>



THANKS

