HEAVY METALS

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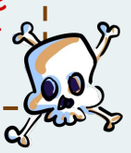
Objectives

? Not given……

Don't panic, there is a lot of information but focus on important things and get the CONCEPT! You can do it ^_*



NOTES EXTRA BOOK IMPORTANT GOLDEN NOTES

Iron (Fe)

So when we talk about Iron toxicity then we refer to the ingestion of iron **supplements**! People know the supplements

Suicide VS Accidental

-Children younger than age 6 years: Highest risk toxicity, accidental. Mother tablets, the color is appealing for them

-Adult: Suicide attempts. Intentional

Pharmacology (image)

-Normal serum iron levels : 50 to 150 micg/dL. Serum iron level isn't a great test because it bound

-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 mcg/dL. Serum transferrin better but cost more

-TIBC is higher than the serum iron level due to a low degree of saturation. (Normal)

-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.

-No way of actively excreting iron, only by cell loss (There is no physiologic excretion mechanism)

-In hemochromatosis, TIBC is totally saturated by significantly high levels of iron in that condition (click to see other conditions)

Dose

-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. Toxicity begins at level above 20 mg/kg

-Ingestion of more than 60 mg/kg may lead to severe morbidity.

-Pediatric patients are more susceptible to iron toxicity.

-50% mortality (LD50) is reported to be 200 to 250 mg/kg. 50% die no matter what you do

-Calculate iron tablets to know the exact dose (no free iron is given or used fortunately)

(Remember that iron is not normally excreted from our body so that's why we have to be careful and measure pt's level to avoid toxicity)

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**).

-Unbound (free) iron moves into cells and localizes near the mitochondrial cristae, resulting in uncoupling of oxidative phosphorylation and impairment of adenosine triphosphate (ATP) synthesis. No One will ask but it interrupts krebs cycle

-Cell membranes are injured by **free radical mediated lipid peroxidation**.

-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:

Important! People love to ask about it (acetaminophen and iron stages)

5 stages

I — Reflects the corrosive effects of iron on the gut. Vomiting occurs within 80 minutes 3-5 hours is the peak, but some has extended release of ingestion in more than 90%of symptomatic cases. Diarrhea, which can be bloody, follows.

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.

III — 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.

Metabolic derangements due to iron poisoning include hypoglycemia, leukocytosis, and severe lactic acidosis from hypoperfusion and interference with cellular respiration.

Early coagulation defects are probably related to direct effects of iron on vitamin K–dependent clotting factors. Later coagulation : hepatic failure.

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.

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.

Iron (Fe)

Diagnostic strategies:

GI Symptoms

The presence of gastrointestinal symptoms suggests a potentially serious ingestion, whereas their absence is reassuring.

It has a direct caustic effect on GI mucosa; don't forget CAUSTICS effects on GIT (check the lecture!!)

Radiograph

Radiopaque iron tablets can be seen on abdominal radiograph Help specifically in iron. Some formulations aren't radio-opaque. **Helpful** not diagnostic!

Serum Iron Level

A serum iron level: 3 to 5 hours (best) after ingestion, is the most useful laboratory test to evaluate the potential severity of an iron overdose. (important!)

Toxicity of Iron by Amount Ingested and Peak Serum Levels:

Elemental Iron (mg\kg)	Peak Serum Iron (micg\dL)	Toxicity
<20	50-150	None
20-40	<350	Minimal
40-60	350-500	Moderate
>60	>500	Severe

Because iron is rapidly cleared from the serum and deposited in the liver, iron levels may be deceptively low if measured late, even after a substantial ingestion.

-Pediatric toxicity more(susceptible)than adults and die in a lesser dose
-Ingestion of more than 60 mg\kg is TROUBLE!

Management:

Gastric Emptying	-Iron is not bound to activated charcoal. (Not helpful) -Neither gastric lavage nor ipecac effectively removes large numbers of pills. -Iron tablets clump together as their outer coatings dissolve.
Whole-Bowel Irrigation (WBI)	-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. -Whole-bowel irrigation is contraindicated in the presence of bowel obstruction, perforation, or ileus.
Deferoxamine (Combined with **WBI** at the same time)	-Deferoxamine chelates iron more toxic to form the water soluble compound ferrioxamine, which can be renally excreted or dialyzed. -100 mg : chelate 9.35 mg of elemental iron. Doses are not required -Deferoxamine may also limit the entrance of iron into the cell and chelate intracellular 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. -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.** -The presence of ferrioxamine turns the urine a "vin ros?" Red wine color, which reflects the excretion of chelated iron

Iron (Fe)

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.

Common Iron Preparation

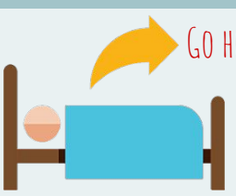
Compound	% of Elemental Iron
Ferrous Sulfate	20
Ferrous Fumarate	33
Ferrous Gluconate	12
Ferric pyrophosphate	30
Ferrocholinate	14
Ferroglycine Sulfate	16
Ferrous Sulfate (Dried)	33
Ferrous Carbonate (Anhydrous)	38
Carbonyl Iron	100

-Understand that each formula has its own % of elemental iron.
-Some newer formulations are designed to be non-toxic and slowly absorbed so it is hard to harm yourself if you are intentionally overdosed yourself!

Disposition

The asymptomatic and less than 20 mg/kg (discharge home) of elemental iron can be observed without further therapy.

Patient remains asymptomatic after 6 hours of observation, discharge is recommended



Lead (Pb)

Lead poisoning is a disease of industrialization.

Lead Containing Products

-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

Doctor talked about it all (Well, it could help in answering scenario-based questions!)

Route of Exposure

-Exposure usually results from ingestion or inhalation.
-It may result from direct skin contact with organic lead compounds or from retained bullets in or near joints. No cellular effect only toxic

Hobbies & Activities at Risk of Lead Exposure

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.

Activities

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.

Hobbies

Children VS Adult TOXICITY

Children typically present to the emergency department. Very common in children and very toxic

1-Following an ingestion of lead
2-Symptomatic with a possible exposure history
3-Referred for management of an elevated BLL (Blood Lead Level)

Lead toxicity in adults most often results from inhalational exposure in the workplace, as well as from hobbies and related activities. Social history is important(occupational history)

Lead Toxicity:

Pharmacology

-There is no known biological 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 and interferes with critical enzymatic reactions.
-Its toxic effects are most **prominent** in: Hematopoietic,Neurologic and Renal systems. Brain,Blood, Kidneys, Peripheral nerves. (question)

Hematopoietic Effects:
-Anemia: normochromic or hypochromic. Basophilic stippling (specific for lead)
-The severity of the anemia correlates directly with the BLL(Blood Lead Level)

Neurologic Effects:
-In the peripheral nervous system, segmental demyelination and degeneration of motor axons result in peripheral neuropathies.
-**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, over aggressive behavior, learning disabilities, criminal behavior, and subclinical sensorineural hearing loss.
-Adults and children with acute toxicity may present with lead encephalopathy associated with increased capillary permeability and cerebral edema.

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

Lead (Pb)

Clinical Features of Pb Toxicity

Chronic Toxicity	-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.	
Acute Toxicity	-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.
CNS Toxicity	-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:

Blood Lead Level

-Although capillary lead levels correlate well with BLLs (Blood lead level), the most informative biomarker is a BLL.
-The Centers for Disease Control and Prevention has defined a chronic BLL of greater than 10 micg/dL as toxic for a child. Permanent sequelae
-Acute exposure can result in levels up to 100 micg/dL.

Serum Lead Levels and Symptomatology:

Level (micg\dL)	Symptoms in adults	Symptoms in children
10	None	-Decreased intelligence -Decreased growth -Decreased hearing
20	-Increased protoporphyrin[1] -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):

Other Lab Tests

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

Imaging

-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 shown in radiographs of long bones. (CLICK TO SEE IT!)

1-Or known as ZPP (Zinc Protoporphyrin) is a compound found in RBCs when heme production is inhibited by lead and\or by lack of Iron. It increases in lead poisoning.

Lead (Pb)

Management

Whole-Bowel Irrigation

When to use it:
-Severe poisoning
-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. <10 normal, >70 toxic

Dimercaprol

-For these seriously poisoned patients, dimercaprol (or British anti lewisite [BAL]) should be the first chelator given.
-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. (G6PD >_<)
-Since dimercaprol is diluted in peanut oil, it is contraindicated in patients allergic to peanuts.

Calcium Disodium Ethylenediaminetetraacetic Acid

-Dimercaprol is followed by calcium disodium ethylenediaminetetraacetic acid (CaNa2EDTA) (very specific), a highly effective lead chelator.
-The dosage of "CaNa2EDTA" 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 1g in children and 2g in adults**.
-"CaNa2EDTA" **should be given** only with adequate urine flow or with hemodialysis in renal failure. Renal excretion only!
-For **less** seriously poisoned patients, the dosage of "CaNa2EDTA" is 50 mg/kg/day or 1000 mg/m2 /day, given in two to four divided doses for up to 5 days. Reduce dose!

2,3-dimercaptosuccinic acid (DMSA)

-(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.

Oral d-penicillamine

-Oral d-penicillamine (not often used) 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.

-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.
-No need for chelation for children with a BLL lower than 45 micg/dL.

Acute Lead Encephalopathy

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

Lead (Pb)

Management

BLL

What does it tell ?

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.

Screening

Children with lead levels of 10 to 19 micg/dL need:

- Family counseling
- Careful follow-up
- Frequent screening of BLL

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.

FOREIGN BODY INGESTION

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.

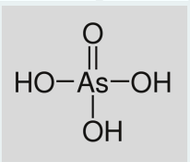
ADMISSION

Patients discharged home on oral chelation therapy **should not return to a contaminated environment.**

DISCHARGE

Arsenic (As)

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

Uses of 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.

Napoleon Bonaparte

Arsenic Toxicity:

Well, Arsenic can remind you of malaria! Both affect liver and both bind to hemoglobin and cause Hemolysis!! And guess what? Both are found where contaminated water can be found (;

Pharmacology	Pathophysiology	Clinical Features
-Arsenic has no metabolic or biologic function. -Of the two inorganic forms, trivalent arsenite (very toxic gas) (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. -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. -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	-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.**	-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 alkalinization can be used to decrease renal deposition of hemoglobin. -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.** (While Cyanide causes bitter almond odor) -Encephalopathy with seizures and coma, respiratory failure associated with ARDS* and dysrhythmias associated with cardiac conduction disturbances. Multi organ dysfunction -**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. *Adult Respiratory Distress Syndrome

Arsenic had killed one of the famous historical figures named Napoleon Bonaparte, that's why you have to go to next slide and check the remaining clinical Features and more two things (;

{ Arsenic (As) }

Clinical Features:

Hematemesis and hematuria, that always makes you think of arsenic as an agent that hates BLOOD so it will try to get it out of the body!!

Acute effect of arsenic poisoning :

Gastrointestinal

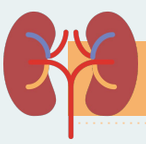
Violent gastroenteritis
Hematemesis/hematochezia
Jaundice "remember, it causes hemolysis!"
Pancreatitis
Dysphagia
Hepatomegaly

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

Arsenic poisoning should also be considered in any patient with a history of severe or recurrent gastroenteritis or abdominal pain and unexplained dermatologic lesions associated with peripheral neuropathy. New undiagnosed rash; think Arsenic chronic toxicity

Weeks to Months later on : Characteristic lines in the nails (Mees' lines), sensorimotor neuropathy hyperkeratosis of the palms and soles (binds to sulfhydryl groups in keratin)

{ Arsenic (As) }

Diagnostic strategies:

Normal arsenic levels are:

Blood	Urine
5 micg/L or less	(Gold standard) less than 50 micg/day in a (24-hour urine) important!!!

Any urine level above 100 µg/day or 50 micg/L (single reading) **necessitates treatment**

Management:

Support

The initial management should address life threatening conditions with supportive management of shock, dysrhythmias,and seizures.

Blood

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

Chelation Therapy

-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(BAL) is the preferred chelator in patients who are critically ill.
-DMSA is a water-soluble analogue of dimercaprol that can be given orally

No Activated charcoal, does not adsorb arsenic.

{ Mercury (Hg) }

Mercury is a silver white metal
The only metal that is liquid at room temperature. (It could be a hint!!)
It has a long history of medicinal uses.

Uses of Mercury:

Significant poisoning at home

-Sphygmomanometer mercury spilled then was aerosolized by vacuuming
-Mercury was heated on the kitchen stove to extract gold from ore.

Industrial Uses

The manufacture of fluorescent lights, batteries, polyvinyl chloride, and latex paint

Mercury is a common pollutant of air and water because of its many industrial uses.

Various other sources of mercury have also been implicated in intoxication.

Sources of Mercury:

Elemental	Salts	Organic
-Spill from mercury containing devices -Gastrointestinal exposure from ruptured Cantor or Miller-Abbott tube -Inhalational exposure in workplace/home -Deliberate injection or ingestion -Accidental ingestion	-Accidental disk battery ingestion -Deliberate ingestion -Laxative abuse	-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

-Elemental mercury is very bad for your lungs (inhalational is the worst)
-Salts (industrial exposure/inorganic)
-Organic (food sources "deep sea fish")
-Salts and organic mercury cause **neuropsychiatric symptoms >> Mad hatter disease**

Elemental mercury injection
leads to Multi organ failure

IV mercury leads to mercury pulmonary emboli which has a very unique appearance on X-ray (click on lung icon to see it!)

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.
-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 diverticula.
-Mercury is absorbed through the skin at 1% of the rate of inhaled mercury and is **not a concern**.

Organic Mercury	VS	Inorganic 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.		-Have two different forms: Hg1+ (mercurous) and Hg2+ (mercuric). -Ingestion of either salt leads to significant gastrointestinal and renal toxicity

{ Mercury (Hg) }

Mercury Toxicity:

Pathophysiology	Clinical Features	Diagnostic Strategies
-Mercury binds sulfhydryl groups. (Also, arsenic and lead do!) 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.	-Inhalation of elemental mercury onset of shortness of breath, fever, and chills that progresses to pneumonitis and respiratory distress. (Always, inhalational Hg means damage to your lungs!!) -Aspiration of liquid metallic mercury during medical procedures results in the rapid onset of tracheobronchial hemorrhage. -Acute ingestion of inorganic salts typically causes a corrosive gastroenteritis with third spacing and hemorrhage. -Patients complain of a **metallic taste** (Arsenic also causes this but it could be garlicky 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 (It is down there ^-*)	-"Normal" mercury levels are considered to be • Blood: less than 10 micg/L not helpful • Blood level more than 35 micg/L needs Rx. not helpful • 24-hour urine mercury levels is the most helpful test in confirming exposure and monitoring the effectiveness of chelation

Mercury Intoxication Syndromes

Types of mercury route of exposure	Signs and Symptoms
Inhalation of metallic mercury	Hypoxemia, Respiratory distress, ARDs*, Dyspnea, chest tightness, Fever, Chills, Burning in mouth and throat, Nausea, Vomiting, Bloody diarrhea and Renal tubular necrosis
Aspiration of metallic mercury	Aspiration pneumonitis, ARDs*
Subacute/chronic inhalation of metallic mercury	Metal fume fever, Neuropsychiatric symptoms, Renal dysfunction and 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 (AKA Mad hatter Syndrome)(also organic mercury causes it) , acrodynia
Organomercury exposure (methyl-diethyl-)	Delayed neurologic problems (ataxia, tremor, dysarthria), Visual field constriction, Hearing loss, Spasticity, hyper-reflexia

*Acute respiratory distress syndrome.

{ Mercury (Hg) }

Management

Acute Toxicity

-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.
-Charcoal absorbs very little and is not recommended.

Acute Inhalation Exposure

Patient should be removed from the source , supportive management provided.

Acute Aspiration

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

Self-injecti on

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

Chelation Therapy:

1 BAL(succimer)

-(don't give to organic(fish) causes neuronal dysfunction) 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(found is seafood)

2 DMSA

Used for both acute and chronic mercury poisoning and may be the best chelator for methylmercury.

2 d-Penicillamine

-It is also used.
-It should be administered **only after thorough gastrointestinal decontamination** because mercury absorption from the intestinal lumen is **enhanced by the penicillamine**.

Ingested metallic mercury is generally harmless unless its passage is impaired by entrapment in a diverticulum or the appendix.

There is no role for prophylactic antibiotics or steroids.

Summary

Iron (FE)

-Children younger than age 6 years : highest risk toxicity, accidental.
-Adult : suicide attempts.

-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).

-6 hrs monitoring with <20 mg/kg trx Whole bowel irrigation + deferoxamine.

Arsenic (As)

-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.
-It is used in industry as a wood preservative and in the production of glass and microcircuits.
-Arsine gas supportive causing massive hemolysis
-Urine is gold standard
-Associated "Mees lines" and can be found in hair and skin

Lead (Pb)

-Lead poisoning is a disease of industrialization.
-Exposure usually results from ingestion or inhalation.
-Nonspecific brain,blood and renal findings Trx BAL+EDTA (IV therapy) for severe(inpatient) DMSA oral for outpatient
-Level <45 does not need chelation,>69 or symptoms needs chelation, all need environmental assessment and repeat levels

Mercury (Hg)

-Mercury is a silver white metal the only metal that is liquid at room temperature.
-It has a long history of medicinal uses.
-Mercury Multiple forms, (quicksilver) harmless if ingested
-Inorganic causes mad hatter's disease and organic
-found in seafood (don't use BAL)

How Toxic is your knowledge

1-Which one of the following agents and antidotes are correctly paired?
A. Anticholinergic overdose – Flumazenil.
B. Aspirin overdose – N-acetyl cysteine (NAC).
C. Iron overdose – Deferoxamine
D. Paracetamol overdose – Physostigmine.

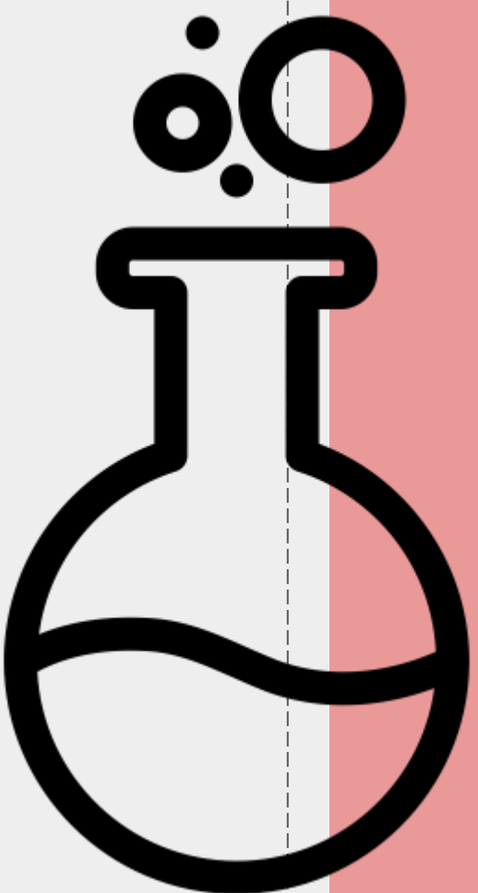
2-Which one of the following has the highest elemental iron composition?
A. Ferrous sulfate.
B. Ferrocholinate.
C. Carbonyl iron.
D. Ferrous gluconate.

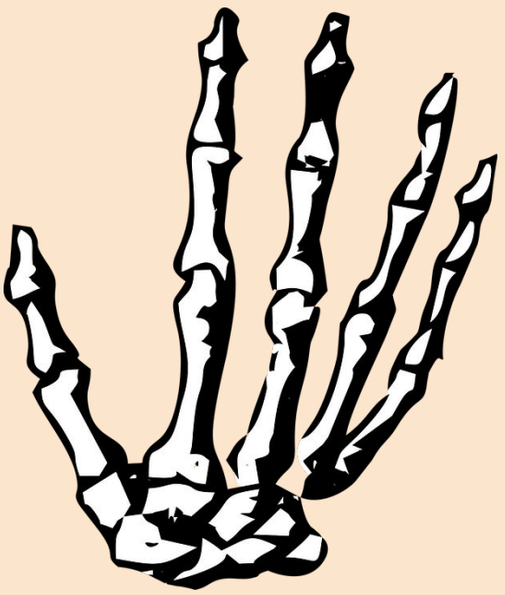
3-Which one of the following is antidote for arsenic poisoning?
A. Nalmefene.
B. Glucagon.
C. D-penicillamine
D. Hydroxocobalamin.

4-What is the type of mercury intoxication can cause systemic mercury poisoning?
A. Heated mercury to extract gold
B.Sphygmomanometer broke and swollen the mercury.
C.Exposure of mercury to skin
D.Exposure of mercury to mucus membrane

1-C
2-C
3-C
4-A

Thank you and good luck!





Very Toxic but you are gonna do it!

A+ is yours (:

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