Pwmed Dr manjhoot

Block k Forensic medicine notes

Comfiled by Wakeel Ahmed Nekmal

1. Corrosive Poisons

2. Acid

3. Alkali

4. Inorganic Metallic Irritants

5. Arsenic

6. Lead

7. Mercury

8. Copper

9. Thallium and Others

10. Non–Metallic Irritants

11. Cyanide Poison

12. Plant Organic Irritants

13. Animal Organic Irritants

14. Snake Bite and Management

15. Scorpion Bite

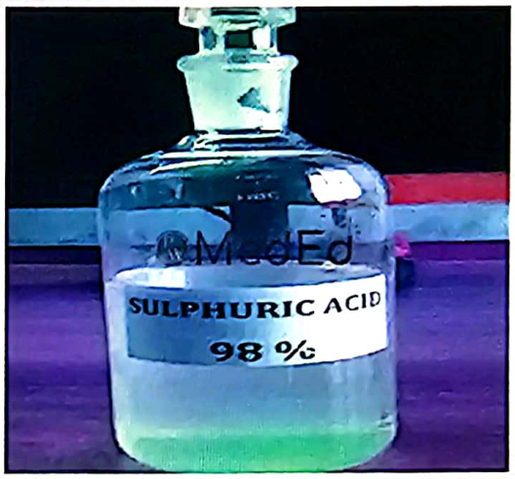
16. Agricultural Poisons

17. Abdominal Injuries

Corrosive Poisoning

Corrosives: Can be divided into 2 types:

1. Acids:



- Proton donor - PH < 3
- All acids does Coagulative necrosis except hydrofluoric acid
- No remote action except Carbolic acid
- Most common organ affected - Stomach

2. Alkali:

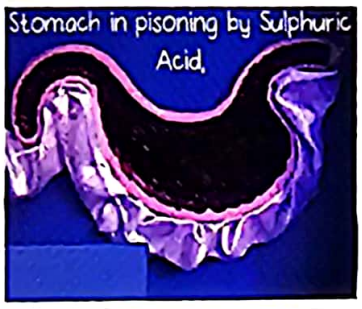
- Proton acceptor - PH>11
- Liquefactive necrosis
- Deeper penetration - More serious injury
- Most common organ affected- Esophagus

Effect	Alkali	Acid
Injury	Severe	Less severe
Necrosis	Liquefaction	Coagulation
Burns	Deep	2nd degree
Edema	Marked	Mild
Eschar	Soft, edematous, translucent	Hard
Charring	Not seen	Seen

1. SULFURIC ACID (USED AS BLEACHING ACID, CLEANSER)

- It is colorless, odorless, oily non fuming acid
- Fatal dosage - 5 to 10 ml
- Fatal period - 12 to 15 hours
- Most common presentation - Pharyngeal pain
- Pharyngeal stricture - Dysphagia- malnutrition- death
- Triad of Sulphuric acid poisoning
  1. Chalky white teeth
  2. Black tongue
  3. Perforation of the stomach

- Treatment: Calcium or magnesium oxide
- Chemical charring +++ (Blackening of surface)
- 326 A and 326 B
- Cause of death: Shock, perforation of stomach, peritonitis, laryngeal spasm.



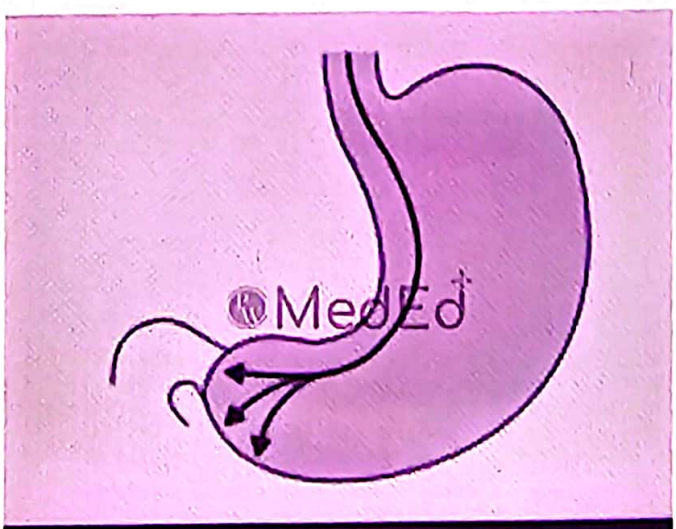
Postmortem findings:

1. Erosion of skin at angle of mouth, lips.
2. Corrosion of the trachea and larynx.
3. Blacking charring of stomach, peppery feel.
4. Perforation stomach
5. Toxic swelling of liver and kidney

Medicolegal aspects:

1. Accidental mistaken for glycerin
2. Suicide - Common
3. Homicide is rare
4. Abortifacient
5. Vitriolage

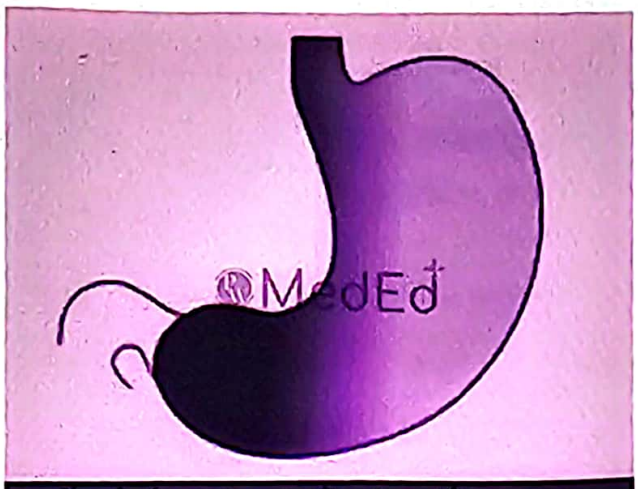
Extra edge:



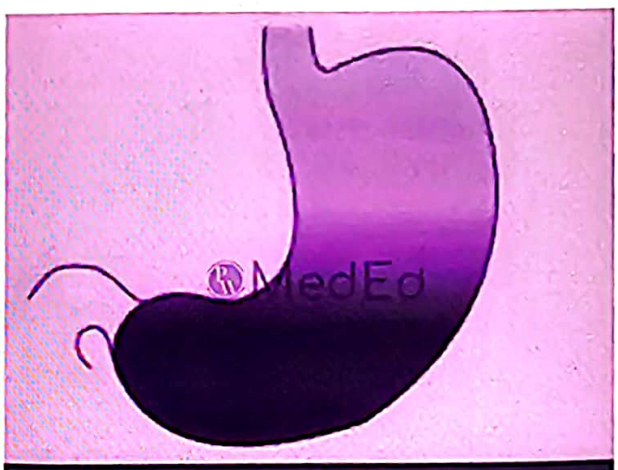
Magenstrasse is the term applied to the pathway acidic agents follow

- In Full stomach: Lesser curvature is affected

- In Empty stomach: Lower 2/3 of stomach affected



Shaded region indicates the area of greatest injury caused by acid ingestion in a food-filled stomach



Shaded region indicates the area of greatest injury caused by acid ingestion in an empty stomach

2. CARBOLIC ACID - PHENOL

- Used as Antiseptics and preservatives
- Fatal dosage - 10 to 20 ml
- Fatal period - 3 to 4 hours
- Symptoms: "5C"
  1. C: CNS depression
  2. C: Constricted pupil
  3. C: Carboluria (Green color urine) because of pyrocatechol and hydroquinone.
  4. C: Cartilage - Ochronosis
  5. C: Corneal deposition
- Cause of death - Syncope, asphyxia due to edema of glottis.
- Treatment - Symptomatic, gastric lavage can be done.

Medicolegal importance

1. Sucide
2. Abortion

3. Rarely homicide

Dettol - Chlorinated phenol with terpineol.

- Lysol - 50% sodium of cresol in saponified vegetable oil
- Metabolism - Liver hydroquinone and pyrocatechol - Green urine and ochronosis
- Signs and symptoms - Convulsions, spasms, miotic pupil, rapid irregular pulse, sweating

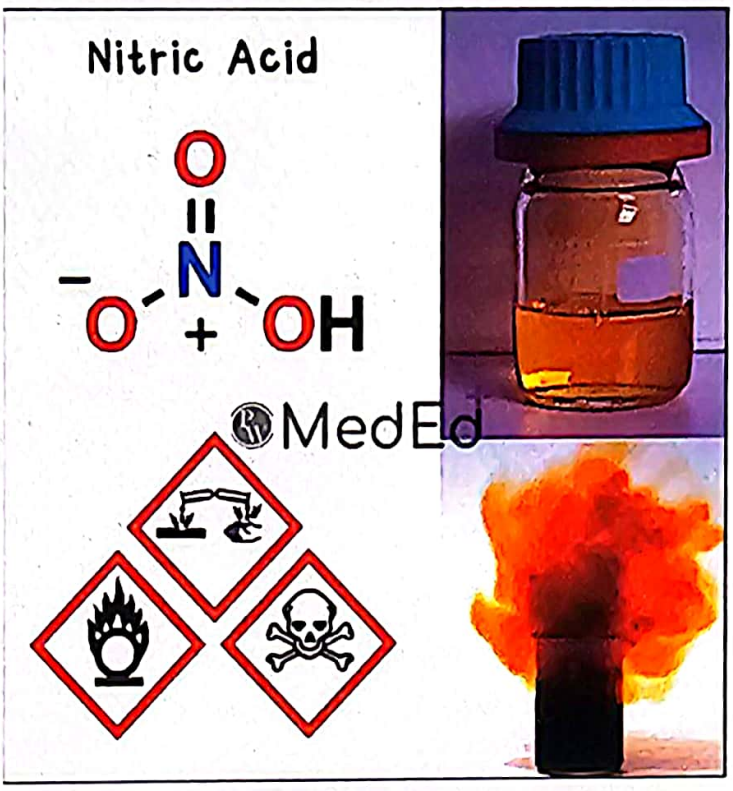
Postmortem findings

1. Graying brown discoloration at angle of mouth and lips, chin, front of body
2. Hardening of stomach (Lathery stomach)
3. Kidney - Hemorrhagic nephritis
4. Pulmonary edema
5. Delayed puterfaction

Medicolegal aspects

1. Accidental
2. Suicide - Common
3. Homicide is rare
4. Abortifacient

3. HNO3 (NITRIC ACID)



- Use as metal cleaners, dishwashing gel, wood working
- Colorless, pungent, fuming
- Yellow (Xanthopoetic)
- Fatal dosage - 10 to 15 ml
- Fatal period - 12 to 24 hours
- Cause of death - Circulatory collapse, respiratory distress

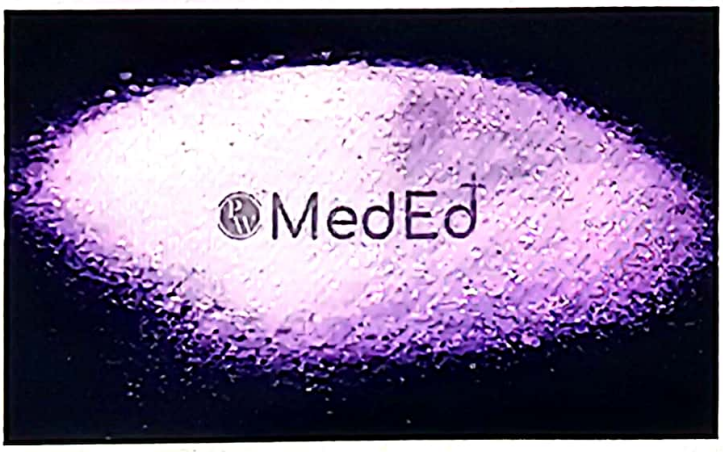
4. HYDROCHLORIC ACID

- Toilet bowl cleaners
- Colorless, pungent, fuming acid
- Fatal dosage - 15 to 30 ml
- Fatal period - 24 hours
- Cause of death - Shock, laryngeal spasm,

Medicolegal importance

1. Sucide (MC)
2. Accidental
3. Homicide (Rare)
4. Abortion

5. OXALIC ACID



- Used as Disinfectants, household things, anti rust products
- Usually available in form of Prismatic crystals
- Fatal dosage - 15 to 30 grams
- Fatal period - 1 to 2 hours
- Also known as Salt of sorrel, acid of sugar
- Leads to Hypocalcemia - Tetany
- Vomitus - coffee ground vomitus
- Oxaluria - Ca oxalate crystals
- Treatment - Calcium gluconate
- Present in beetroot leaves (Known as vegetable acid)

Medicolegal importance

1. Accidental poisoning
2. Suicidal poisoning
3. Forgery

STRONG ALKALIS (CAUSTIC ALKALIS)

- Ammonia, potassium hydroxide, sodium hydroxide, carbonates of ammonia, potassium and sodium.
- Fatal dosage - KOH and NAOH - 5 grams, Ammonia - 30 grams, Sodium and potassium - 15 to 30 grams.
- Fatal period - 24 hours
- Liquefactive necrosis

- Esophageal injury
- Treatment - Symptomatic

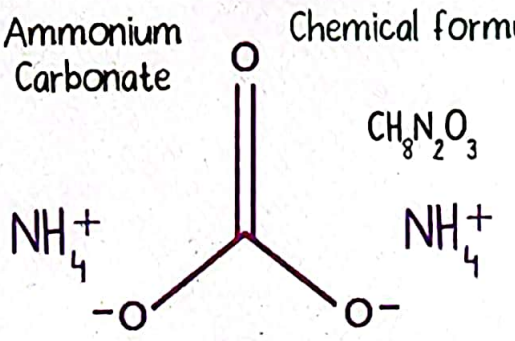
Medicolegal importance

1. Accidental
2. Homicidal
3. Suicidal

Ammonium Carbonate

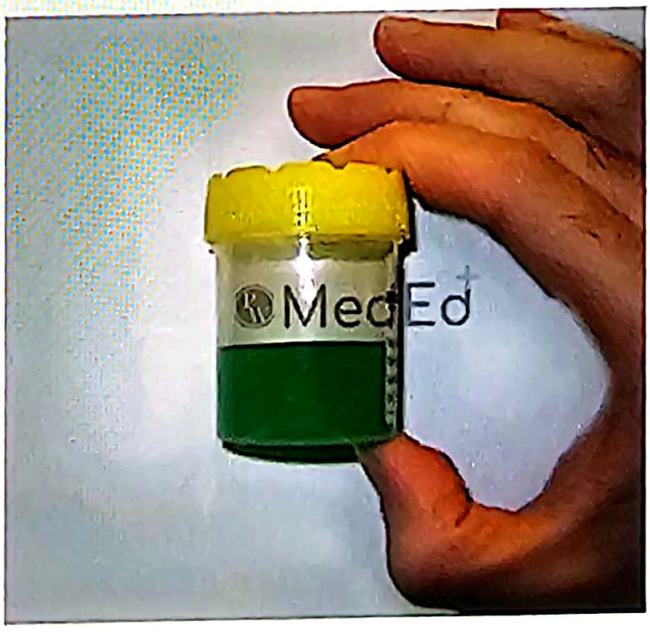
Chemical formula

$CH_8N_2O_3$

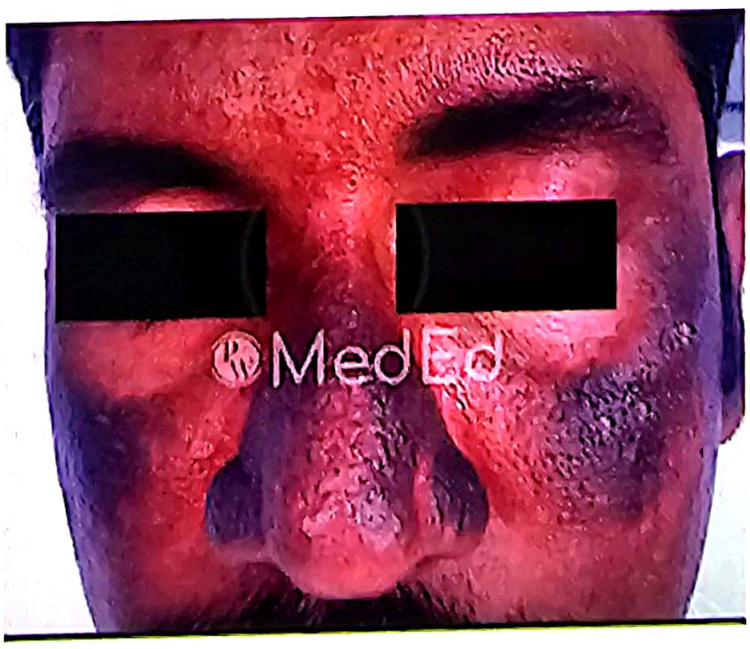


Exact mass: 96.05 Molecular Weight: 96.09

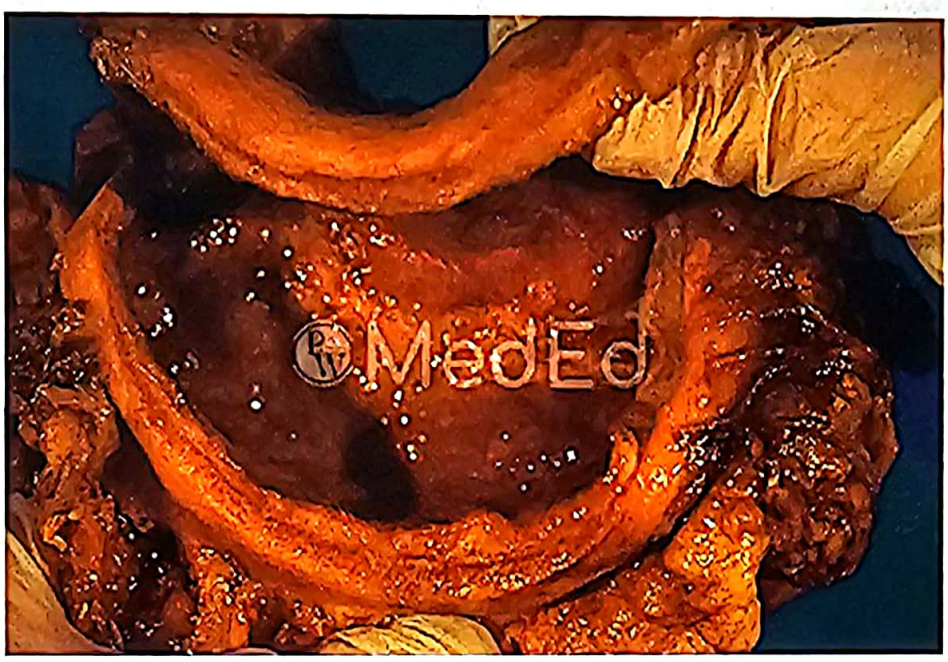
IMAGES



Carboluria seen in phenol

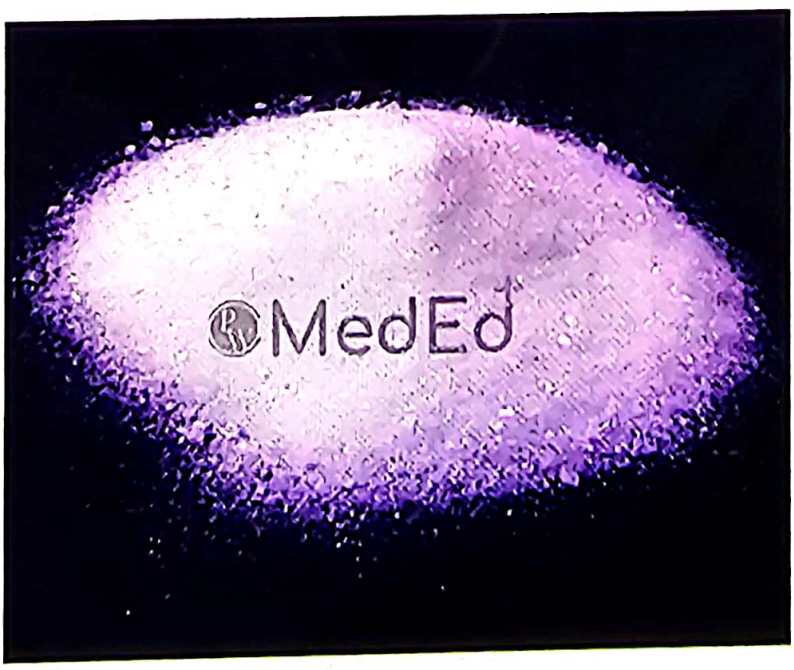


Ochronosis (Carbolic acid poisoning)

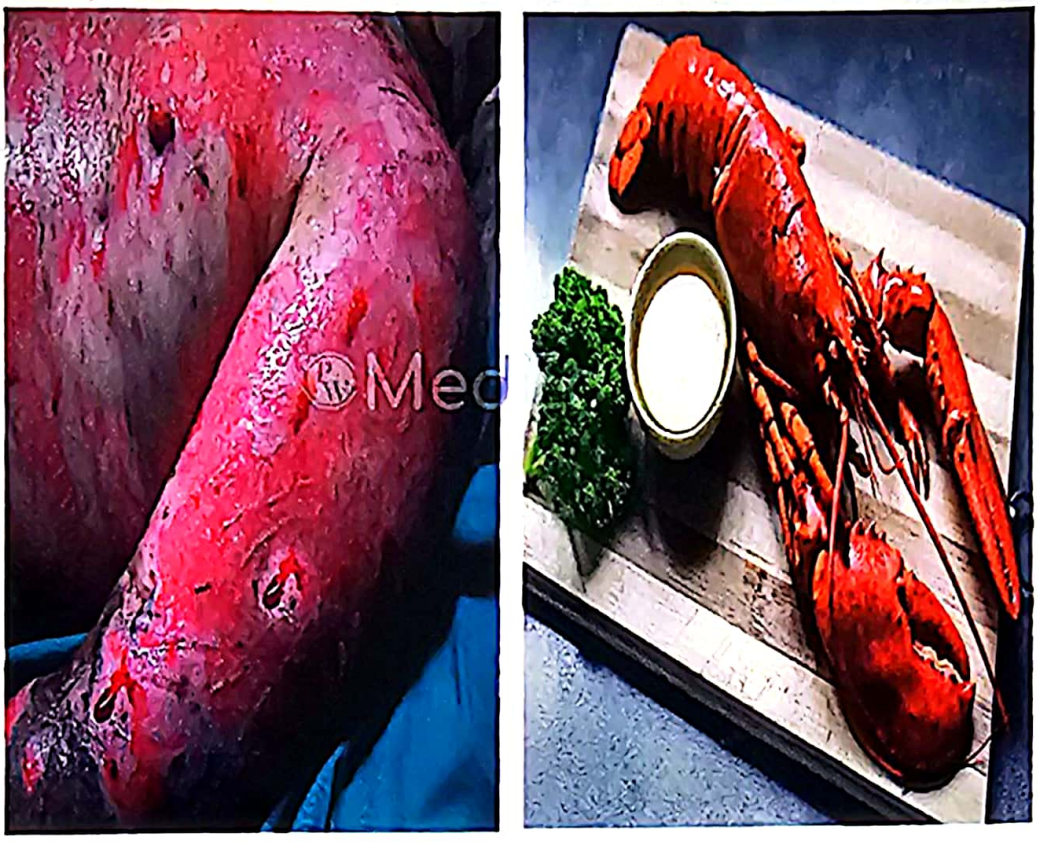


Hardening of stomach (Carbolic acid)

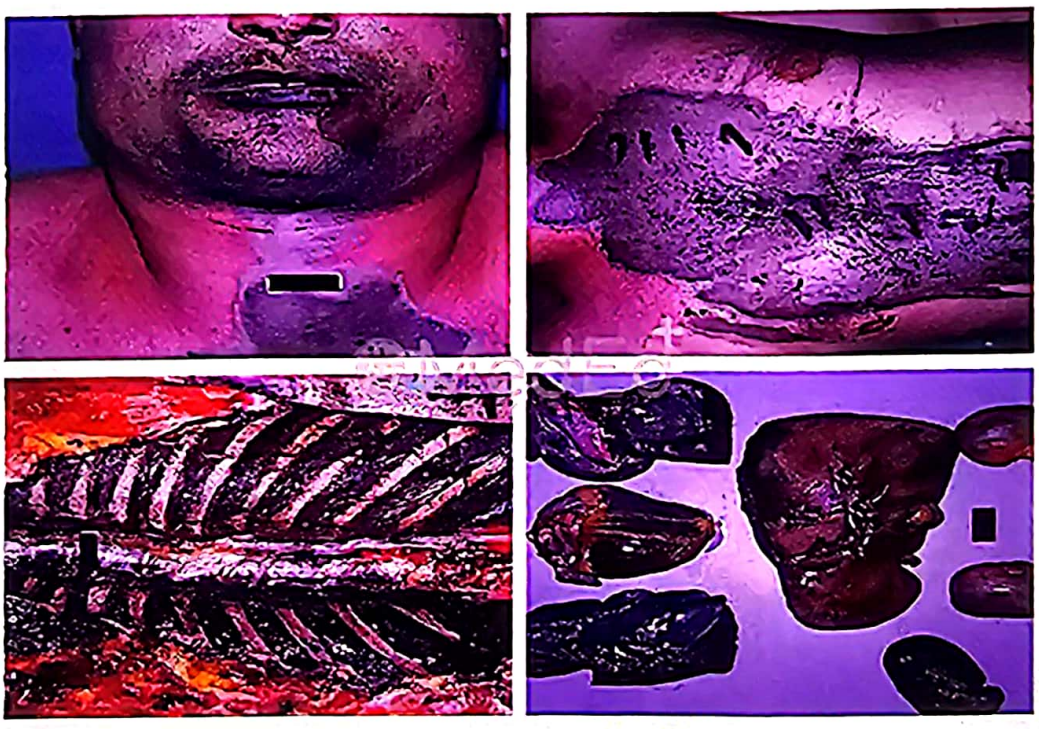
HNO_3



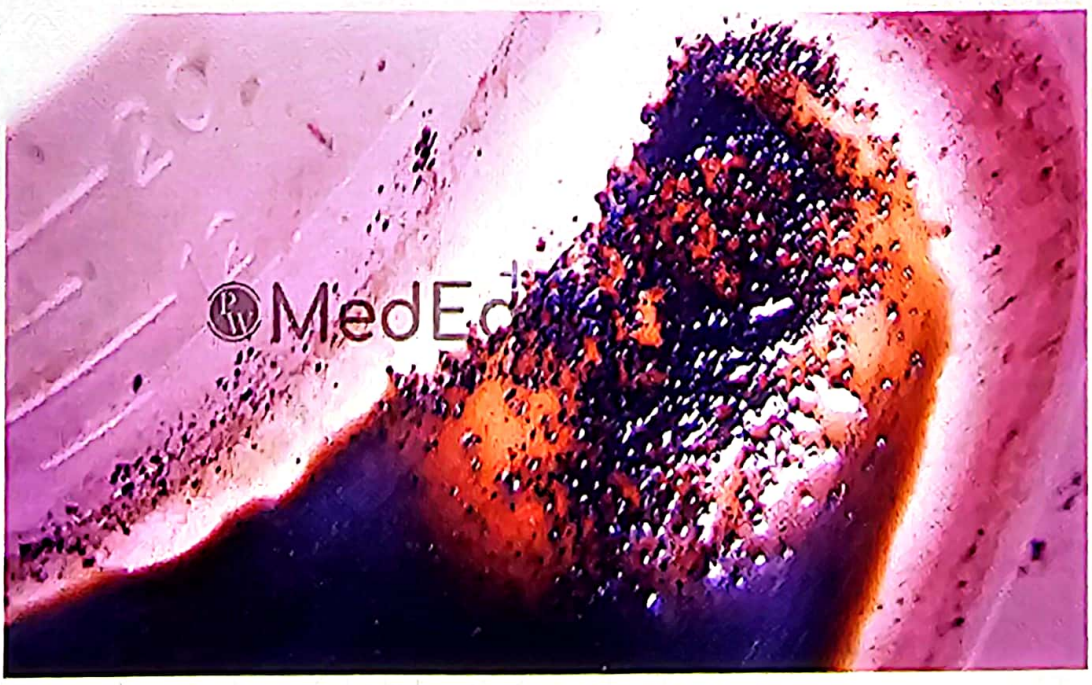
Oxalic acid



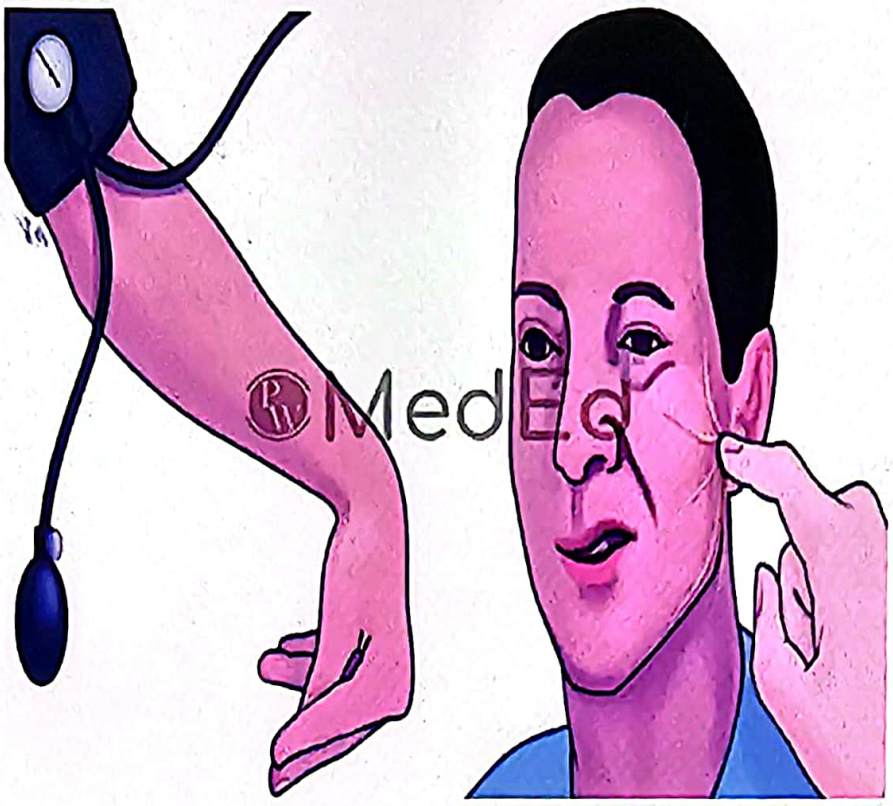
Boiled lobster syndrome (Boric acid)



H_2SO_4 Postmortem findings



Coffee ground vomitus (Oxalic acid)



Tetany (due to Oxalic acid)

H_2SO_4 poisoning-Multiple perforation of stomach

Inorganic Metallic Irritants

ARSENIC

Per:

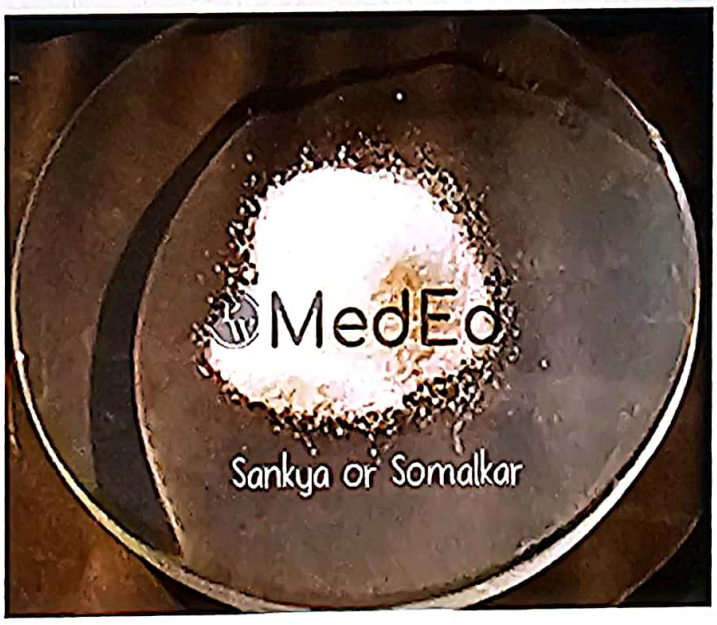
Arsenic is known as the King of all poisons. Napoleon Bonaparte died due to suspected Arsenic poisoning.

Arsenic and the Emperor

THE COMPOUND OF ARSENIC

1. Arsenic trioxide
2. Copper arsenite (Scheele's green)
3. Copper aceto-arsenite (Paris green)
4. Arsenic sulfide
5. Endemic toxicity in soil & water (MC route)

- Action of arsenic - Cellular respiration
- It can lead to bladder cancer, skin cancer.
- Fatal dosage - 100 -200 mg (1 mg/kg) arsenic trioxide
- Fulminant - >3 grams - shock and death due to peripheral vascular poisoning.



Paris Green-Copper Acetoarsenite

Copper Arsenite-Schlees Green

ACUTE ARSENIC TOXICITY

Clinical features:

1. Pain (first) in throat before vomiting
2. Purging follows vomiting

3. Rice watery stool - Severe diarrhea
4. Tenesmus and pain around the anus.
5. Renal failure- oliguria, uremia.
6. CVS - Hypotension, ARDS, pulmonary edema
7. CNS -Tremors, convulsions, coma.
8. Arsine gas - Hemolysis, liver and kidney failure

ARSENIC POISONING AND CHOLERAA

S.No.	Symptoms	Arsenic poisoning	Cholera
1	Pain in throat	Before vomiting	Not so
2	Diarrhea	Follows vomiting	Precedes vomiting
3	Vomitus	Contains mucus, bile and streaks	Watery
4	Stools	Rice water in the beginning and bloody later	Rice watery throughout and passed as involuntary jet
5	Tenesmus	Present	Absent
6	Anal irritation	Present	Absent
7	Voice	Not affected	Peculiar rough and whistling
8	Conjunctivae	Inflamed	Not so
9	Analysis of excreta	Presence of arsenic	Presence of vibrio cholerae
10	Circumstantial evidence	Arsenic poisoning	Cholera in that locality

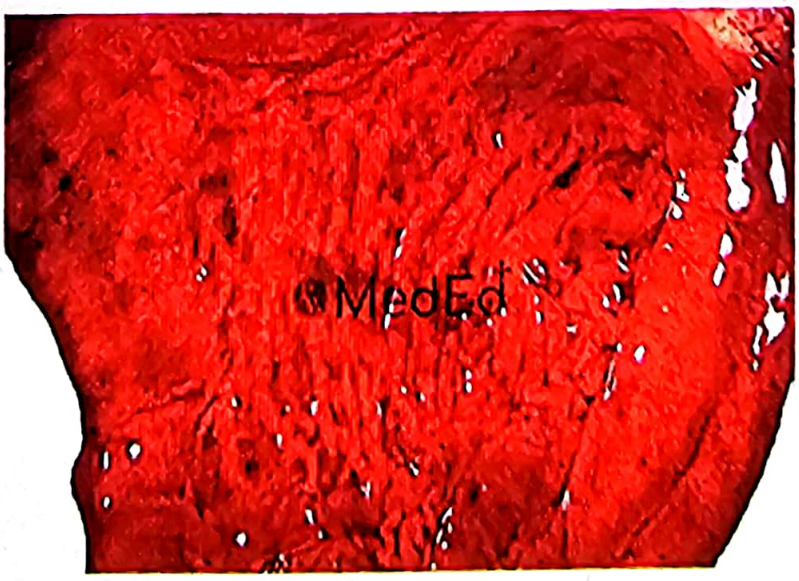
Management

Investigation: Urine, blood, hair, nails, X-ray bands, ECG-ST depression, marsh, reinsch, Gutzeit, NAA (Neutron activation analysis).

Treatment: Gastric lavage, BAL-3 mg/kg IM (DOC) , DMSA, DMPS

- BAL C/I in Cadmium poison, iron poison and peanut allergy

Post mortem finding: Red velvety stomach



1. CPC +++
2. Lungs- pulmonary edema
3. Stomach-red, petechiae, hemorrhages

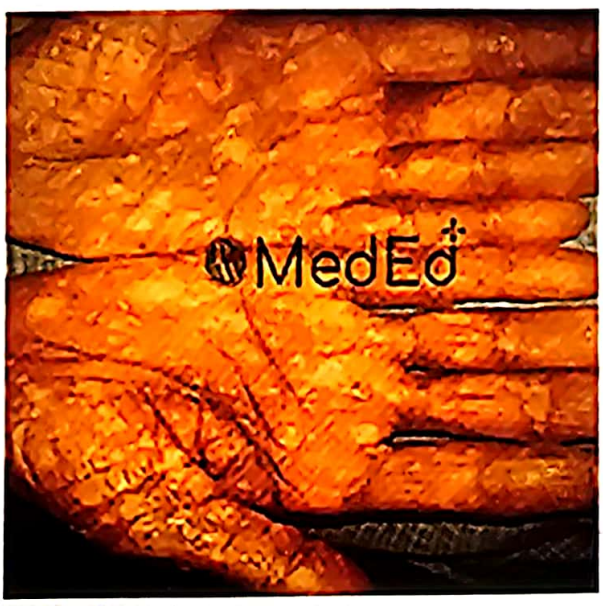
CHRONIC ARSENIC POISONING

Signs and symptoms:

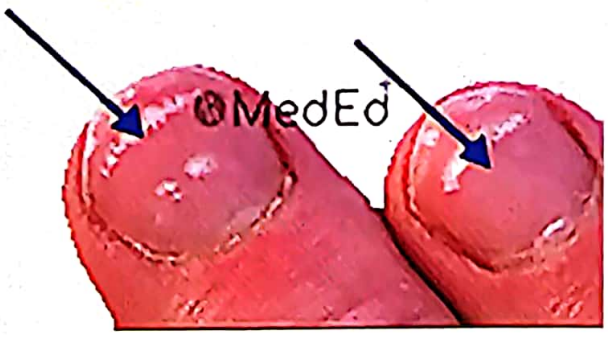
1. Skin and nails - Aldrich mees line, rain drop pigmentation, hyperkeratosis, leucomelanosis, black foot disease, BBC (Basal cell carcinoma).
2. Liver kidney heart - Failure
3. Bone marrow - Suppression, anemia, leukemia
4. GIT - Nausea, vomiting abdominal pain
5. CNS - Encephalopathy, Mixed neuropathy (Painful swelling-erythromelalgia)

- Incase of Lead there is motor neuropathy
- Mee's line - Arsenic, thallum, fluorosis

Rain Drop Pigmentation



Aldrich MEE S Line



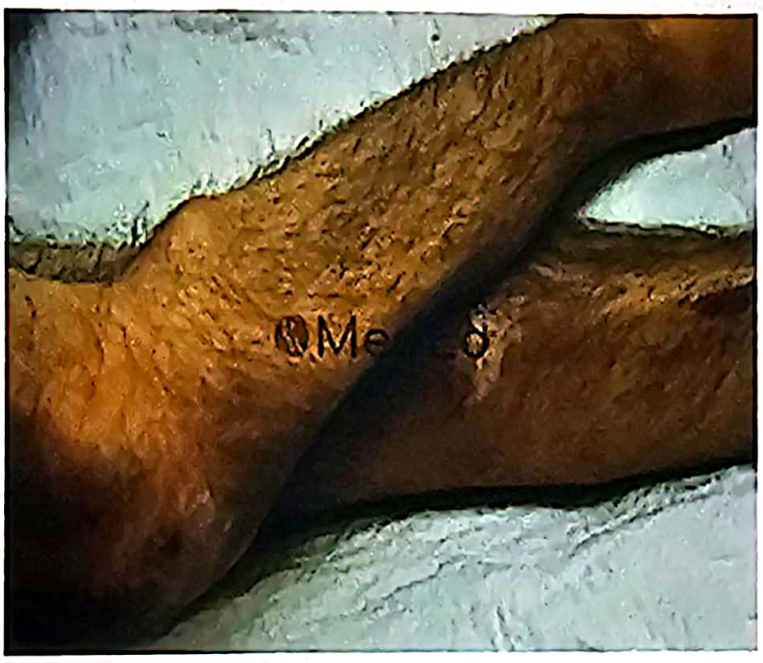
Treatment

1. Decontamination
2. BAL (DOC)
3. Vitamin B, IV sodium thiosulfate
4. Antioxidants

Post mortem findings

- Emaciation, pigmentation, keratosis, alopecia, jaundice, wasting of muscles, white streak on nails.
- Stomach - chronic gastritis
- Liver- hepatomegaly, cirrhosis

- Kidney - Acute tubular necrosis
- Heart - Myocardial necrosis



Medicolegal aspects

1. Homicide - Pan, food, drink
2. Accidental - Well water
3. Suicidal - Very rare due to pain
4. Cattle poison
5. Occupational disease - Mining, glass, semiconductor industry

MNEMONIC: BONAPARTE - RRR

B - Black foot disease

O - Oxide (Arsenic trioxide - Sankya, somalkar, white arsenic), (120-200mg), (2 mg/kg), garlic odor

N - Neoplasia (skin, lung, bladder)/Nails & Hair

A - Alopecia & anemia/Addison like disease/Ashes/Aphrodisiac/Abortion stick/Apoptosis

P - Peripheral neuropathy (sensory > motor, distal muscle (glove stocking)/PDH inhibition)

A - Aldrich mees line

R - Rigor mortis lasts longer and retards putrefaction

T - Teratogen

E - Enteritis/Endocardial hemorrhage

R - Raindrop pigmentation/Measles rash

R - Red velvety stomach (Hemorrhagic gastroenteritis)

R - Reinsch test (Marsch, gutzeit test)

Inorganic Metallic Irritants: Lead

Persona

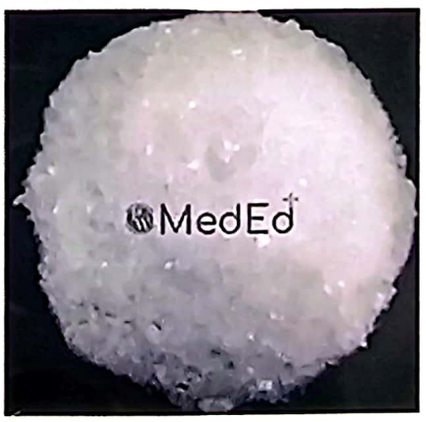
INTRODUCTION

- 1 in 3 children have lead poisoning
- Lead poisoning is also called Plumbism, Saturnism, Painter[]s colic, Miner's disease.

TOXIC COMPOUNDS AND ITS USES

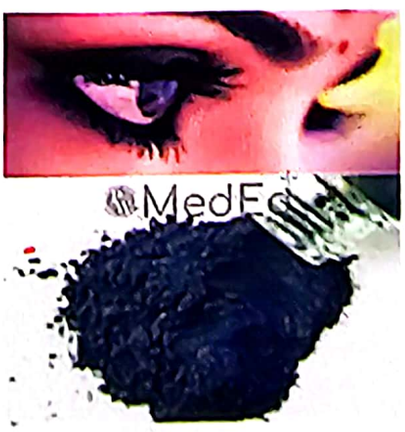
Compounds	Uses
Lead acetate (sugar of lead)	Earlier used as an astringent and local sedative for sprains
Lead tetroxide (red lead or vermilion)	Used as Sindoor
Tetraethyl lead	Anti-knock for petrol
Lead sulfide (surma; least toxic)	Applied on the eyes
Lead carbonate	Manufacturing of paints

Lead Tetroxide



Lead Acetate

Lead Carbonate



Lead Sulfide

MECHANISM OF LEAD TOXICITYA

- Oxidative phosphorylation
- It affects enzymes like pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase, pyruvate kinase, etc.
- Affects myelin sheath and nerves
- Causes encephalopathy in children
- Heme synthesis
- Due to inhibition of ALA dehydratase and ferrochelatase

ACUTE TOXICITY

- Lead carbonate . 40g
- Lead acetate - 20g
- Fatal period - 1 to 2 days

Signs and symptoms:

- Manifests as GIT and CNS disturbances
- GIT: metallic taste
- Dry throat
- Vomiting and nausea
- Burning abdominal pain (colic)
- Blood stained diarrhea leading to circulatory collapse

CNS:

- Headache
- Lethargy
- Paresthesia
- Myalgia
- Insomnia
- Coma
- Death

Laboratory diagnosis:

- Porphyrinuria due to coproporphyrin III
- Blood lead level >70 - 100 µg/dL
- Porphyrin >35 µg/dL
- Urine lead level >0.15-0.3mg/L

Treatment:

- Gastric lavage
- Whole bowel irrigation Calcium disodium ethylene diamine tetraacetic acid 50mg/kg/day in 4-6 divided doses in a continuous infusion for 5 days.
- Vitamin C

CHRONIC LEAD POISONING

Also known as Saturnism, Painter's colic, Miner's disease.

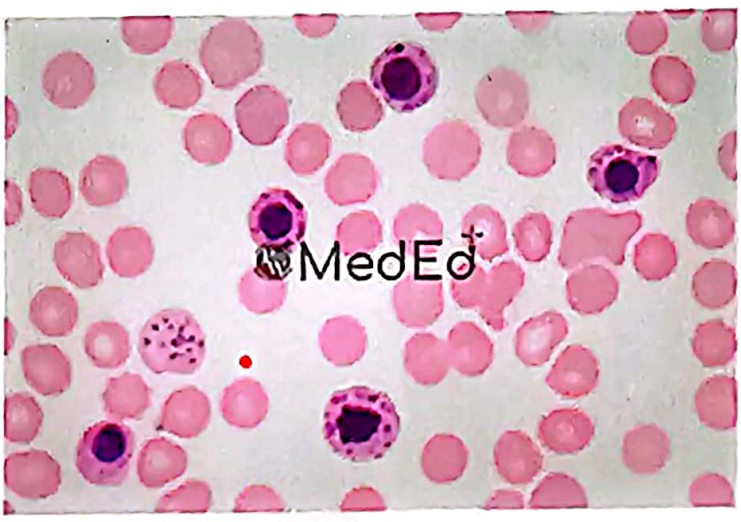
Causes:

- Lead dust
- PICA
- Ingestion
- Air pollution
- Occupational hazards

Signs and symptoms:

Remember as "ABCDEFGHI"

- Anemia
- Burtonian line/basophilic stippling



Basophilic stippling

Burtonian line: bluish line at the junction of teeth and gum.

- Constipation, colicky pain
- Drops/dotted retina
- Encephalopathy - common in children
- Facial pallor/Fanconi syndrome/ foul smell
- Earliest symptoms – facial pallor
- Growth retardation, growth
- Hypertension, hyperuricemia, headache, hallucination
- Impotence, infertility, insomnia, irritability
- Kidney (Nephropathy)

Diagnosis

- Anemia - IDA (iron deficiency anemia)
- Punctate basophilia >200 cells/cu mm
- Elevated free erythrocyte protoporphyrin (>35µg/dL) level and azotemia.
- Urine lead level >80µg/dL (in 24-hour sample)

- Whole blood lead level – blood lead >70µg/dL (severe toxicity) and >50µg/dL (moderate toxicity).
- Coproporphyrin in urine >15µg/dL
- Delta amino levulinic acid in urine >5 mg/dL
- Plasma lead level >0.1mg/dL

Treatment:

- CaNa EDTA:
- Chelation therapy is indicated for adults with blood lead >70µg/dL and for children with encephalopathy or blood lead >45 µg/dL.
- BAL:

  Chelator of choice in case of renal impairment
- Succimer (DMSA) is given to mild to moderate toxicity

Medicolegal aspects:

- Abortion
- Occupational hazard
- Retained bullets
- Spinal tap in lead poisoning leads to herniation and death

MNEMONIC

- BAHUBALI:
  - Burtonian line
  - Anemia and facial pallor
  - Headache, hallucinations, hypertension and hyperesthesia.
  - Uroporphyrin level increased
  - Basophilic stippling
  - Arthralgia, abortion
  - Lead palsy and lead band on X-Ray
  - Impotence, irritability, insomnia, infertility

Inorganic Metallic Irritants: Mercury

TOXIC COMPOUNDS OF MERCURY

Personal Not

1. Mercuric chloride:

- Fatal dose = 1-4 g, 3-5 days
- Colorless, odorless prismatic crystal
- Most common cause for acute poisoning
- Most toxic inorganic salt

2. Mercurous chloride:

- Abortifacient

3. Mercury thiocyanate:

- Diwali winding snake

4. Mercuric sulfide:

- Sindoor
- Red crystalline powder

5. Mercuric oxide:

- Batteries

6. Organic methyl and ethyl mercury:

- Most poisonous
- Minamata disease, mad hatter's disease
- Metallic mercury used in dental amalgam is not poisonous

ACTION OF MERCURY

- Toxic to CNS - causes tremors called "hatter shake".
- Cellular respiration
- Pulmonary irritant
- 3 target organs - brain, kidney, GIT.

ACUTE POISONING DUE TO MERCURY SALTS:

System	Signs and Symptoms
GIT	○ Metallic taste
	○ Feeling of constriction in throat

System	Signs and Symptoms
GIT	o Hoarse voice
	o Hot burning pain from mouth to stomach
	o Pain in the lower abdomen followed by nausea, retching, and vomiting.
	o Vomiting grayish, slimy, mucoid material with blood and shred of mucus.
	o Followed by bloody diarrhea and tenesmus.
Renal	o Oliguria
	o Albuminuria
	o Hematuria ending with renal failure
CVS	o Hypertension
	o Tachycardia
	o Difficulty in breathing
	o Circulatory collapse

Diagnosis:

- DMPS provoked urine challenge test
- Blood mercury level
- Urine mercury level - NAA
- Nanomedicine

Treatment:

- Acute poisoning:
- Gastric lavage with sodium formaldehyde
- BAL
- DMSA or SUCCIMER
- D penicillamine

Postmortem findings:

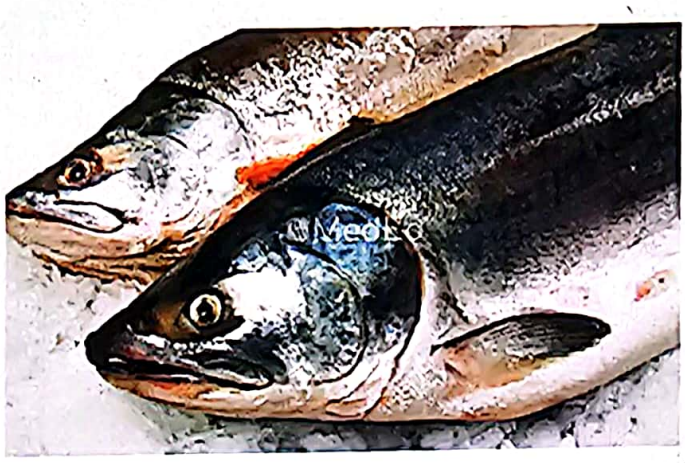
- Petechial hemorrhage
- Congestion
- Cyanosis
- Kidney - PCT necrosis
- GIT - inflammation
- Liver - congested
- Heart - fatty degeneration

CHRONIC MERCURY POISONING

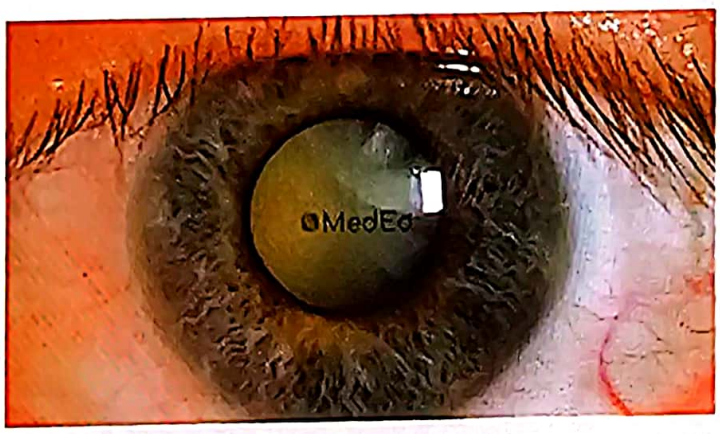
Clinical features:

- Remember as "M3 BATTLE"
- Minamata disease
- Mercuria lentis

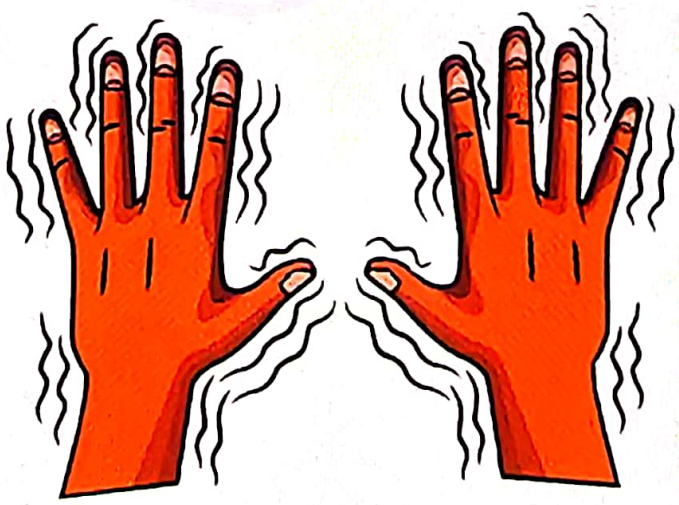
- Membranous nephritis
- Brown malt reflex/black line on gums and salivation
- Acrodynia/pink's disease
- Tremors, tonicity decreases
- Lens affected
- Erethism



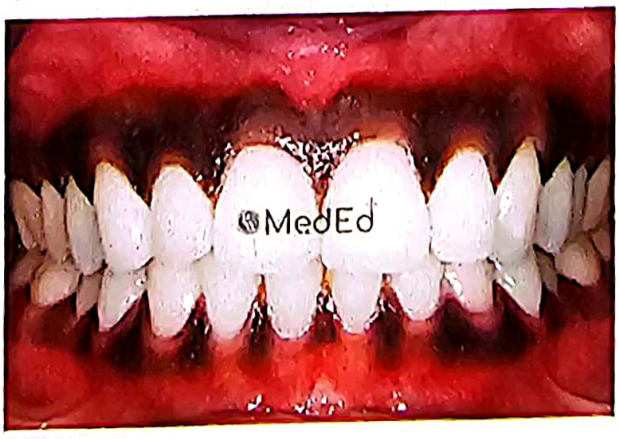
Minamata disease due to consumption of Poisonous fish



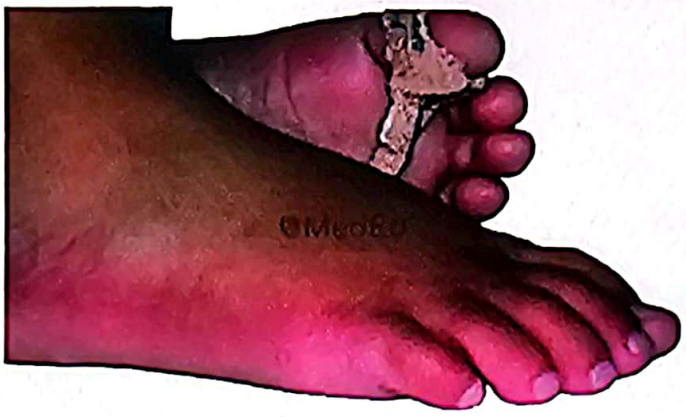
Deposition of Mercury In anterior lens capsule



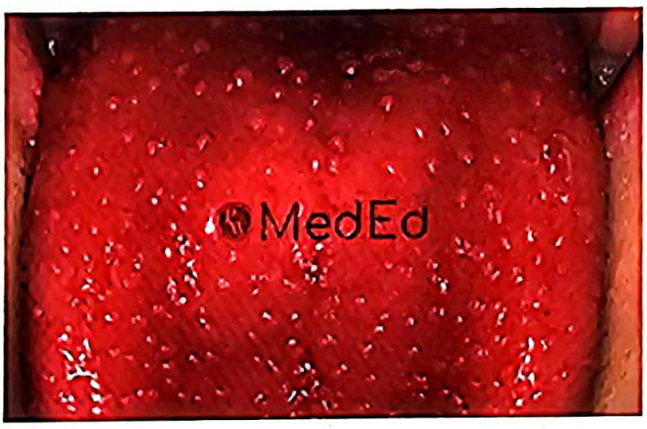
Tremors in Mercury Poisoning



Black line at junction of teeth and gums



Acrodynia



Strawberry tongue

Diagnosis:

- Blood >35µg/L
- Hair
- Urine >150µg/L

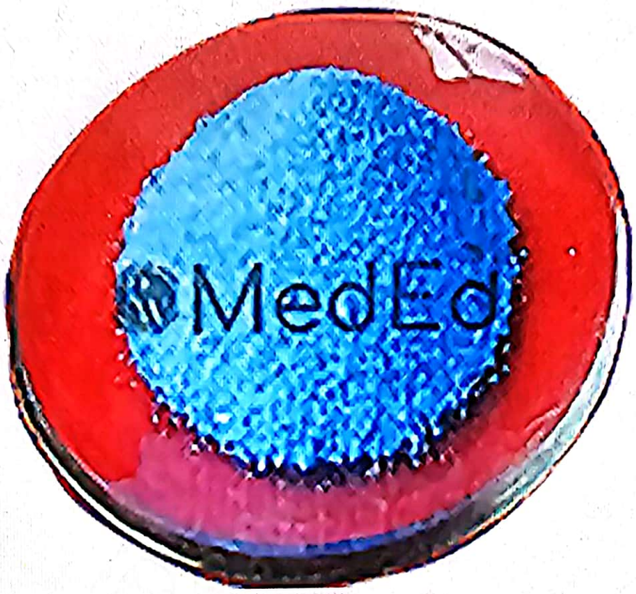
Treatment:

- BAL
- DMSA – Drug of choice
- N-acetyl D penicillamine
- DMPS
- BAL is contraindicated in organic mercury poisoning due to increased neurotoxicity.

Inorganic Metallic Irritants

COPPER: COPPER SULFATE-BLUE VITRIOL

Pers



- Metallic Copper is non-poisonous.
- Copper Containing Enzyme - Catalase, cytochrome oxidase, dopamine beta hydroxylase, cytochrome C oxidase, Serum Ceruloplasmin.

Toxic Compounds

- Copper Sulfate (Blue Vitriol)
- Copper Subacetate (Verdigris)
- Copper carbonate

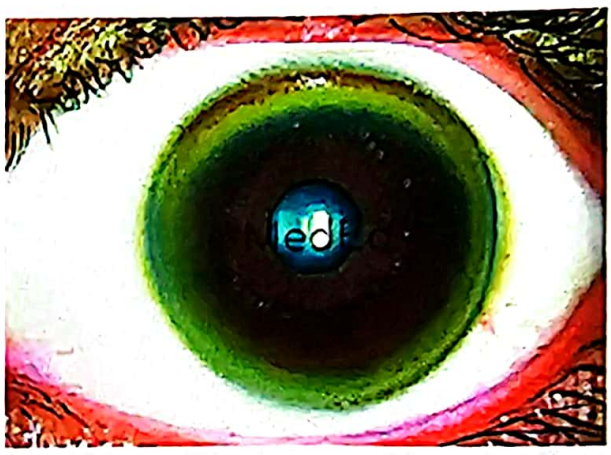
SCHEELEs GREEN (COPPER ARSENITE): Paris green -Copper acetoarsenite

Signs and symptoms of Acute poisoning

- Metallic taste, Hypersalivation, Abdominal pain
- Renal failure
- Hepatic failure
- Muscle cramps
- Hypotension shock
- Frontal headache, lethargy, drowsiness
- Fatal dosage: copper subacetate-125 grams, copper sulfate- 10 to 20 grams
- Treatment: D penicillamine, DMSA, DMPS, EDTA, BAL, tetrathiomolybdate.

CHRONIC COPPER POISONING

- Copper fumes in welders - metal fumes fever
- Wilson disease
- Renal damage
- Anemia
- Colicky abdominal pain
- Green color line on gums (Clapton lines)
- Jaundice/KF ring/chalcosis oculi/Sunflower cataract
- Vineyard sprayer's lung disease



Kayser - Fleischer Ring around the periphery of cornea

Management of chronic copper poisoning

Diagnosis: ↓ Serum ceruloplasmin, and ↑ 24-hour urinary copper excretion.

Treatment:

- Dimercaprol (BAL) - 5 to 15 mg/kg/4 hours IM until urinary excretion drops below 50 mcg/24 hours
- D - penicillamine, oral zinc, unithiol, ca edta, liver transplant, ammonium tetrathiomolybdate.

Medicolegal Aspects:

- Suicide
- Accidental - Copper cooking utensils, IUCD
- Green color of vegetables
- Cattle poison
- Rarely homicide

Inorganic Metallic Irritants

THALLIUM POISONING

- Poisoner's poison
- Ideal homicidal (colorless, tasteless, odorless, slow acting & wide clinical symptom)

Aunt thally Caroline Grills - killed 4 by mixing thallium with tea and cake (killed stepmother, sister in law & two other family members. Motive - property greed)

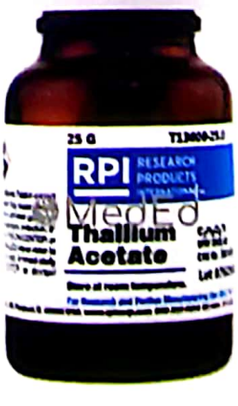
Toxic compounds of thallium:

- Thallium acetate - colorless, tasteless- Rodenticide, fireworks, Insecticides
- Thallium sulfate - Ants & Rats

Mechanism:

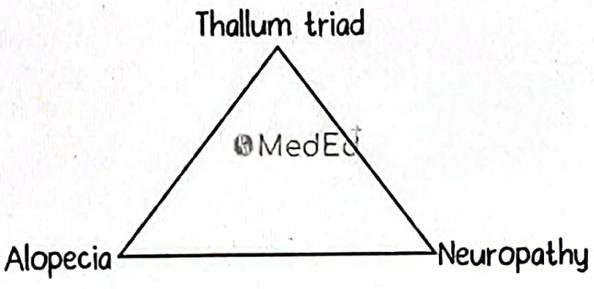
- Corrosive - GI symptoms
- Replaces the potassium in enzymes
- Wallerian degeneration of neurons
- Myelin sheath affected
- Hair loss - destruction of hair follicles & stunted mitosis in hair follicles

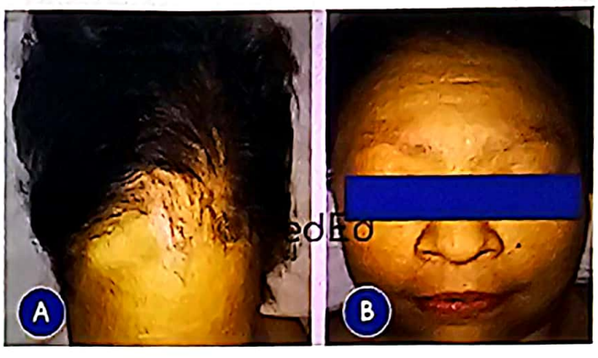
Thallium sulphate:



Signs & Symptoms:

- Fatal dosage - 200 mg - 1 gram, 8 mg/kg, 24 to 36 hours.
- Gastroenteritis
- Peripheral neuropathy - proximal, symmetrical, mixed.
- Cranial nerve palsies, delirium, convulsions, coma
- Loss of hairs in 2 weeks - scalp, eyebrow, axilla, and body hair - brown black pigmentation at hair root.
- Mees line





Period after poisoning	Symptoms
3-4 hours	Nausea, vomiting, diarrhea, and hematemesis
5 hours - 14 days	Disorientation, coma, psychosis, seizures, heart failure, acute pulmonary edema,optic neuritis,acne,hyperhidrosis,gray-gum lines, hair root pigmentation, and anhidrosis.
15-30 days	Dry and scaly skin, alopecia & white stripes on nails
> 30 days	Ataxia, foot hyperextension, and memory loss

Treatment:

- Potassium chloride
- Gastric lavage with sodium iodide - Thallium iodide
- Activated charcoal
- Prussian blue/Berlin blue- Oral therapy
- Pilocarpine
- Hemodialysis

Post mortem changes:

- CPC++
- Liver- Centrilobular necrosis & fatty degeneration
- Spleen - congested
- Brain - congested

Medico-legal aspects:

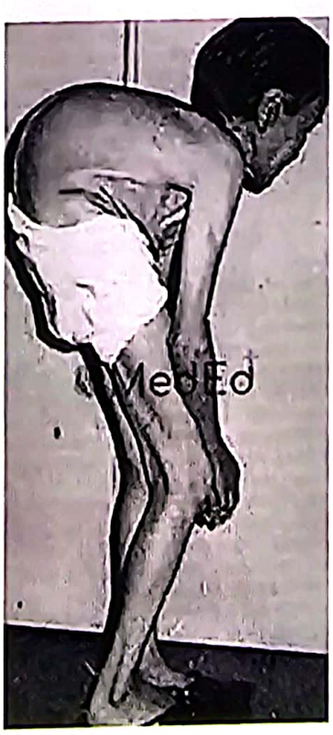
- Ideal homicidal
- Accidental
- Abortion
- Suicidal

- Occupational

CADMIUM POISONING

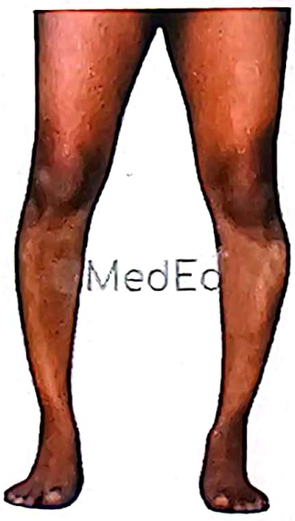
- Ailing bones & Failing kidneys

itai- itai disease (ouch ouch disease):



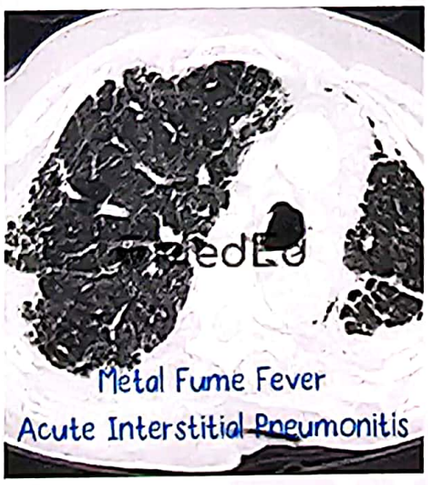
Cadmium poisoning: Seen in welding, electroplating, jewelry making, nickel - cadmium batteries,smokers, shellfish.

Mechanism: Substitution of cadmium in place of zinc. programmed cell death, formation of cadmium - metallothionein complex leading to nephrotoxicity.



Osteomalacia & Pathological Fracture

Abdominal Pain



Metal Fume Fever
Acute Interstitial Pneumonitis

Neuropathy

Findings:

- Cyanosis (cadmium blue)
- Yellowish teeth (cadmium ring formation)
- Chronic renal failure (hypercalciuria, proteinuria, azotemia)
- Carcinogenic - pancreas, bladder

Treatment:

- Acute- CA EDTA- IM-75mg/kg/24 hours or DMSA 10 mg/kg orall
- Chronic - No chelation therapy, symptomatic management only (BAL is contraindicated)

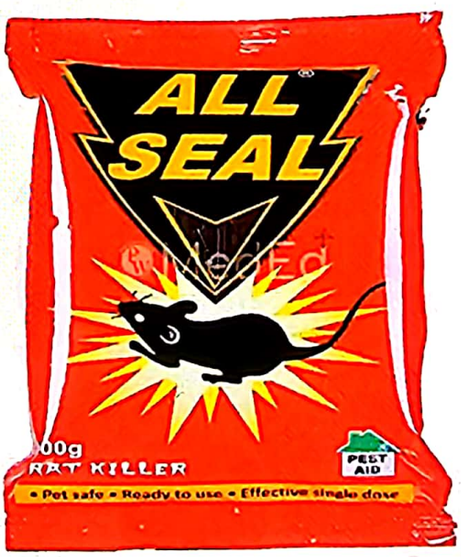
BARIUM POISONING

Barium chloride:

Barium carbonate:

Rat poison: barium carbonate, hydroxide, or chloride (50 - 70 mg/kg)

Barium poisoning:



- Fatal dosage- 1 gram, Fatal period - 12 hours
- Barium induced hypokalemia, paralysis & areflexia (within 2 hours, normal K+ levels - 3.5 to 5.5MEq/L)
- Fatigue due to vomiting, diarrhea, lactic acidosis, ventricular arrhythmia, & respiratory paralysis.

Treatment:

- Gastric lavage
- Potassium administration
- IV Na_2SO_4 OR $MgSO_4$
- Assisted ventilation

ZINC POISONING

- Zinc sulfate (white vitriol)
- Zinc phosphide - Rodenticide

Symptoms:

- GI symptoms
- Metal fume fever - zinc oxide vapor
- Phosphine gas - Cardiac arrhythmias

Metal fume fever:

- Smelter shake, Brass chills, Monday morning fever
- Self limiting febrile illness due to inhalation of metal oxide fumes of:

1. Zinc oxides
2. Copper
3. Magnesium
4. Nickel, Mercury, Chromium, Cadmium

- Fumes release cytokines in the lungs.

Non-Metallic Irritants

PHOSPHORUS (LIGHT BRINGING)

TYPES OF PHOSPHORUS

1. Red phosphorus:

- Non poisonous & Amorphous

2. White phosphorus/Yellow phosphorus:

- Inflammable
- Poisonous
- Luminescent in dark
- Crystalline
- Garlic odor
- Phosphine
- Fertilizer/Insecticide/Rat killer/incendiary bombs/ smoke screen fireworks

Signs and symptoms

- Contact injury - second & third degree yellow burn with garlic smell
- Acute ingestion -

1. GIT symptoms - Luminescent vomitus feces (6 hours)
2. Second stage - symptoms free (1-4 days)
3. Liver & Kidney failure

- Fatal dosage - 60 to 120 (1 mg/kg), 24 hours
- Cause of death - Cardiac arrhythmias due to hypocalcemia, Hyperkalemia
- Treatment - Symptomatic/copper sulfate/Liver transplantation
- Post mortem findings - Hypostasis/ CPC+++/Swollen yellow liver/Toxic hepatitis
- Medicolegal aspects - Accidental/Homicidal by mixing with alcohol/Industrial/Bomb grenade.
- Chronic poisoning - Teeth showing multiple discharging sinuses/Lucifer jaw.

Hydrocyanic acid

CYANIDE POISONING

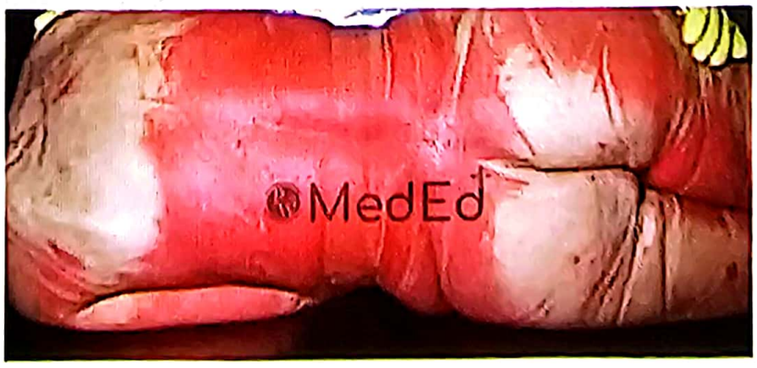
Interesting fact - Apple seeds contain a protein called amygdalin, a substance that releases cyanide when comes in contact with human digestive enzymes, but acute toxicity is seen as very rare.

CYANIDE (PRUSSIC ACID, SCHEELE'S ACID)

Fatal dose:

- Pure acid: 50-60 mg (2-10 mins)
- NaCN & KCN: 200-300 mg (30 mins)
- Inhalational: 270 ppm (immediate)

- Fatal period: 2 to 8 minutes
- Mechanism of action: It acts on ETC, complex IV (Cytochrome oxidase), affects cellular respiration & ATP production, which eventually leads to death of the patient.
- Ideal suicidal agent: Cyanide is considered as ideal suicidal substance.
- Smell: Pure acid is colorless gas with bitter almond smell (sex linked recessive trait)
- Postmortem change: Pink postmortem hypostasis



SYMPTOMS	SIGNS
Headache	Altered mental status (e.g., confusion, disorientation
Confusion	Seizures or Coma
Dyspnea	Mydriasis
Chest tightness	Tachypnea/Hyperpnea (early)
Nausea	Bradypnea/Apnea (late) Hypertension (early)/Hypotension (late) Cardiovascular collapse Vomiting Plasma lactate concentration >8 mmol/L

LAB FINDINGS OF CYANIDE POISONING

- Narrow A-V oxygen difference
- Increased anion gap acidosis
- An elevated lactic acid levels

Lee Jone test - Greenish blue color (due to formation of ferrocyanide)

Differential diagnosis: Organophosphates

Treatment:

- 100% oxygen
- Gastric lavage with sodium thiosulphate
- Activated charcoal is ineffective
- 3 step-cyanide antidote:

1. Amyl nitrate-inhalation therapy
2. Sodium nitrite (unstable)-intravenous
3. Sodium thiosulphate (soluble stable compound)-intravenous therapy

- Mercury cyanide-BAL
- KCN/NaCN-Hydroxocobalamin

POST-MORTEM CHANGES

- Blood stained froth (also seen in OP poisoning,opium poisoning, drowning)
- CPC++/Cerebral edema
- Smell of bitter almond
- Cranial cavity opened first
- Spleen is the best specimen
- Autopsy should be done in negative pressure room with protective devices

- Pink postmortem hypostasis
- Early rigor mortis
- Extremely volatile substance-collect samples in air tight bottles. Lungs should be preserved.

Medicolegal Aspects:

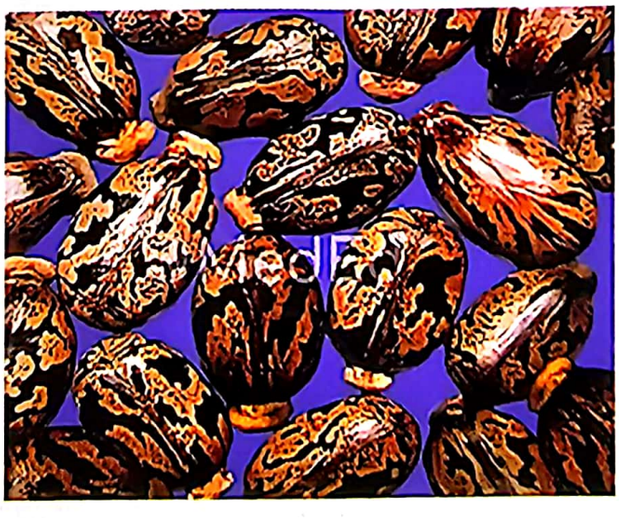
- Ideal suicidal
- Homicidal
- Accidental
- Judicial

Plant Organic Irritants

Important plant organic irritants:

- Ricinus Communis (castor plant)
- Croton Taglium (jamalgota)
- Abrus Precautorius (rati)
- Semicarpus Anacardium (laundry seed)
- Capsicum Annum (red chilly)
- Calotropis (madar)

1. RICINUS COMMUNIS



- It grows all over India, especially in wastelands.
- Seeds are polished and mottled with light brown markings.
- The active principle is toxalbumin. (inhibits protein synthesis and causes hemolysis)
- It can cause liver, brain, kidney and GIT hemorrhage and causes death in 3 to 4 days.

- Unbroken seeds and castor oil are non-poisonous.
- Fatal dosage is 10 to 20 seeds (5 micrograms per kg, inhaled or injected)
- Fatal period is 3 to 5 days.
- Signs and symptoms include pulmonary oedema, convulsions, diarrhea, uremia and jaundice.
- Treatment includes gastric lavage, intravenous glucose and saline and blood transfusion.
- Medicolegal aspects can be bioterrorism, accidental, homicide and conjunctivitis.

2. CROTON TIGLIUM

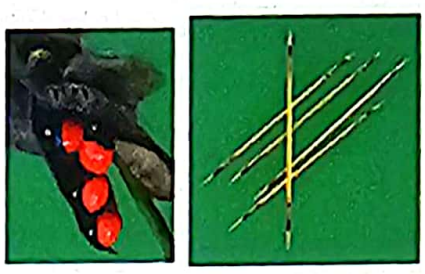
- Active principles are Crotin and Crotonoside.
- Seeds are dark brown, non-shiny and non-mottled.
- Signs and symptoms include pulmonary oedema, convulsions, diarrhea, uremia and jaundice.
- The fatal dosage is 4 crushed seeds or 3 drops of oil.
- Fatal period is 6 hours to 3 days.
- Medicolegal aspects can be accidental, arrow poison and abortion.

3. ABRUS PRECAUTORIUS

- Active principles are abrin (thermolabile toxalbumin), abrine (hemagglutinin) and abralin.
- Seeds are scarlet red with black spots on one end.
- They are egg-shaped, tasteless and odorless.
- Signs and symptoms include bloody diarrhea, hemotoxicity, dilated pupils and viper snake bite (cattle poison).
- Fatal dosage is 1 to 2 crushed seeds.

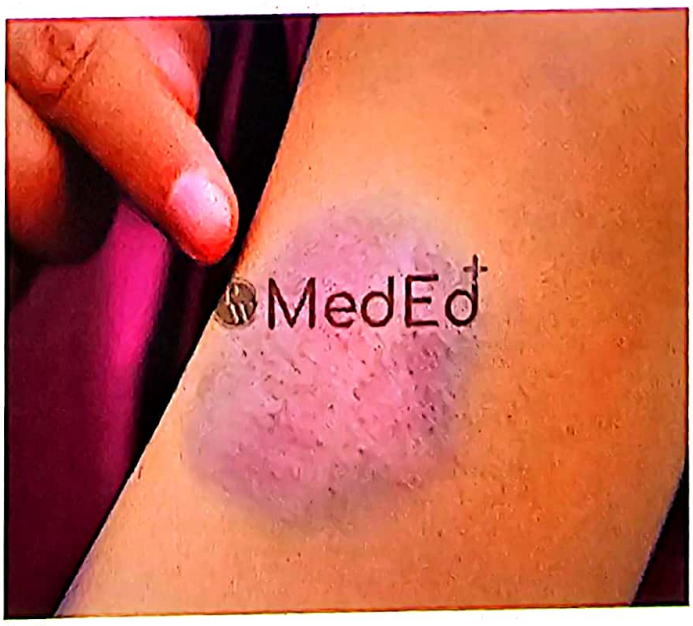
- Fatal period is 3 to 5 days.
- Post-mortem findings show CPC present and hemorrhages.
- Medicolegal aspects can be accidental, arrow poison, abortion, sui needle and suicidal.
    - Seeds decorticate
    - Datura
    - Opium
    - Onion

    → Spirit + water → sharp pointed needles → sun dried (15 mm long, 90 to 120mg weight)
    - 2 needles stuck into a wooden handle
    - Blown over the animal



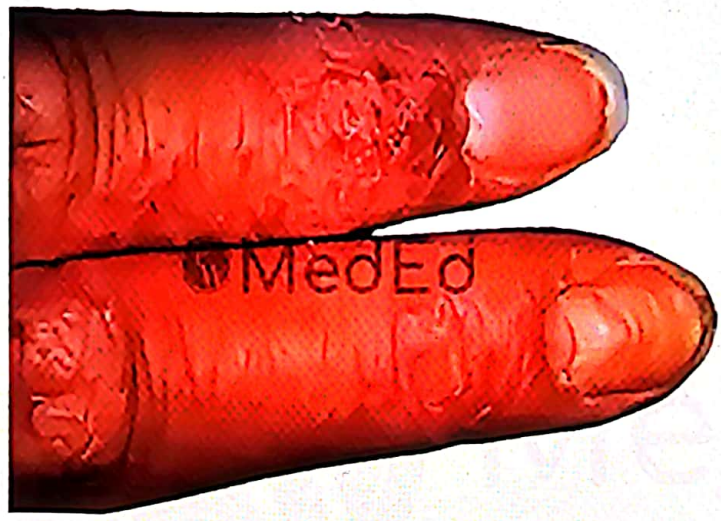
4. SEMICARPUS ANACARDIUM

- Active principles are bhilawanalol, semicarpol, cardol and catechol.
- Seeds are heart-shaped with rough projections at base.
- They are black or brown in colour.
- Fatal dose is 5 to 8 crushed seeds.
- Fatal period is 12 to 24 hours.
- Post-mortem findings show CPC present and haemorrhages.
- Medicolegal aspects can be accidental, arrow poison, abortion and artificial bruises.



5. CAPSICUM ANNUM

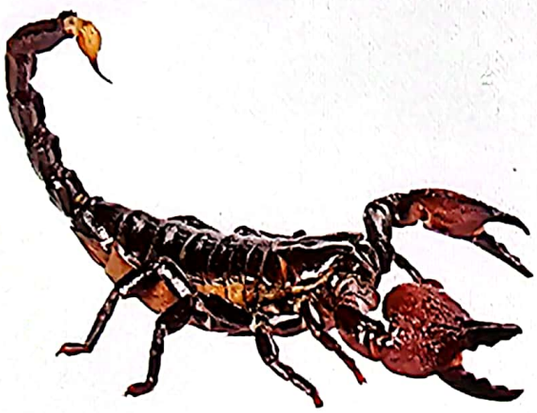
- It resembles dhatra seeds.
- It causes Hunan hand.



6. CALOTROPIS

- Calotropis gigantea is purple in color.
- Calotropis procera is white in color.
- Active principles are uscharin, calotoxin, calactin, gigatin and calotropin.
- Post-mortem findings show CPC present and hemorrhages.
- Medicolegal aspects can be accidental, arrow poison, abortion, artificial bruises, infanticide, cattle poison and dried root - snake bite (used by snake charmers).

Animal Organic Irritants (Snakes) Part-1



BIG FOUR SNAKES

Common Cobra:

- Hood and spectacle marks are seen.
- It is neurotoxic.
- The site of action of venom is Post synaptic.

Common Krait:

- Steal black in color.
- It has a white transverse line.
- Most common in India.

Russell viper:

- It has a triangular head, constricted neck, broad body and pointed tail.
- It is hemotoxic.
- It causes DIC and death.

Saw scaled viper:

- It has a triangular head, constricted neck, broad body and pointed tail with a white wavy line over the snake.

- It is hemotoxic.
- Treatment: Polyvalent antivenom

15 MG. 30 MINS-6 HOURS
NAJA NAJA

8 MG, 1 to 2 days
BIRD FOOT MARKS, E CARINATUS

(40 MG , 1 TO 2 DAYS),
DABOIA RUSSELLI

3 TO 6 MG, 18 HOURS, 8 CERULEUS
MOST COMMON (INDIA)

Features	Common Cobra	King Cobra	Common Krait	Banded Krait	Russell's Viper
Head and Neck	Hood present, bears a double/ single spectacle mark	Hooded without spectacle mark	Head covered with large shields	Head covered with large shields	Flat, triangular with distinct 'V' mark and small scales
Belly	Smooth scales	Scales looks shiny, but it is dry to touch	Creamy white	Triangular in cross section	White with broad plates
Back	Spectacled white or yellow pattern, which sometimes forms ragged bands	Yellow or black bands or broad chevron like markings	Single/double white bands with central row of hexagonal scales	Alternate black and yellowish bands	Three rows of diamond - shaped black/brown spots
Color	Brown/Black/ Green	Yellow/Green/ Brown/black with white cross-bands meters	Steel-Blue/black	Resembles common Krait	Brown/Buff
Length	1.5-2 meters	3-4 meters	1.25-1.5 meters	2 meters	1.5 meters
Habitat	Throughout India	Thick jungle/forests	Close to human dwelling	Assam, Bengal, South India	Throughout India

COBRA V/S VIPER

S.NO.	Feature	Cobra	Viper
1.	Head	Small, covered by large scales or shields	Large, broader than the body, triangular, covered by small scales
2.	Body	Long and cylindrical	Short with narrow neck
3.	Pupils	Circular/round	Vertical, slit-like
4.	Maxillary bone	Carries poison fangs and other teeth	Carries only poison fangs
5.	Fangs	Grooved, short and fixed	Canalized, long and movable
6.	Venom	Neurotoxic	Hemotoxic
7.	Tail	Less tapering	Tapering
8.	Other teeth	Pressed in upper jaw	Absent
9.	Reproduction	Oviparous	Ovo-viviparous

CLASSIFICATION OF POISONOUS SNAKES

Elapidae:

3 to 4 meters long,
Chevron like markings

Large head scales, bell, Hexagonal scales, steel black

Alternate black and yellow bands

Cause of death in Elapidae: Respiratory paralysis, convulsions.

SIGNS AND SYMPTOMS: (5 'D' AND 2 'P')

- 5 'D':
  1. Dyspnea
  2. Dysphonia
  3. Dysarthria
  4. Diplopia
  5. Dysphagia

- 2 'P':
  1. Ptosis
  2. Paralysis

VIPERIDAE

- Venom - 90% Enzymes - Endothelial cells - Hemolysis and Cellulitis
- Cause of death is DIC.

Small head scales, distinct 'V'
Mark on head, brown spots

HYDROPIDAE

- 20 types in India.
- Sea snakes - Myotoxic.

Points	Poisonous snakes	Non poisonous
1. Belly scales	Large:They cover the entire breadth of belly	Small: They never cover
2. Head scale	a) Usually small in vipers b) May be large in pit vipers c) Cobras and Coral snakes where third labial touches the eye and nasal shiels d) Kraits, where there is no pit and the third labial does not touch the nose and eye	Are usually large with exceptions as outlined under poisonous snakes
3. Fangs	Are hollow like hypodermic needle	Short and solid
4. Tail	Compressed	Not markedly compressed
5. Habit	Usually nocturnal	Not so
6.Teeth bite marks	Two fang marks with or without marks of teeth	Two fang marks with number of small teeth marks

NON - POISONOUS SNAKES - COLUBRIDAE

1. Rat snake
2. Vine snake
3. Sand boa
4. Mud snake

Identify:

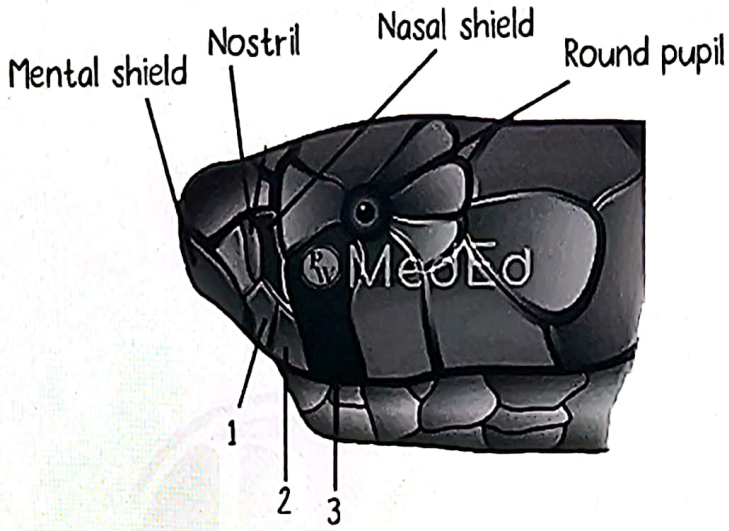
Answer: Common Krait

Answer: Viper

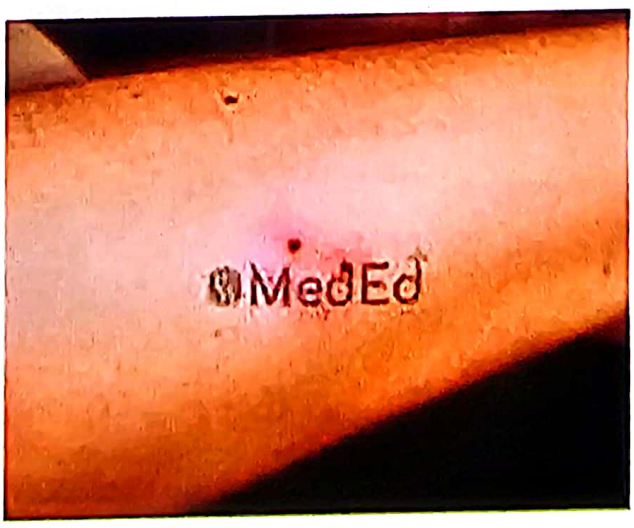
Answer: Banded Krait

King Cobra

Common Cobra

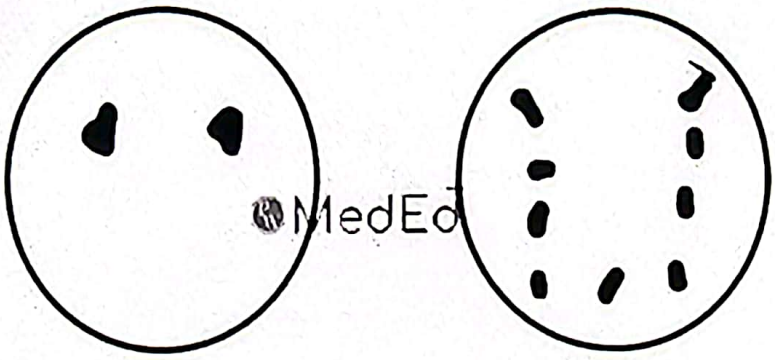


- 70% bites in India - Non -Venomous, 15% are dry bite, 15% - Ophitoxaemia.
- Dry bites most common with cobra
- Early and intense pain - significant envenomation
- Snake venom - Vipers(75 to 90% enzymes), Elapidae 25 to 70% enzymes



MARKERS OF SEVERE ENVENOMATION

- Rapid early extension of local swelling from the site of the bite.
- Early tender enlargement of lymph nodes.
- Early systemic signs
- Early spontaneous systemic bleeding (Gum bleeding)
- Dark brown urine



Poisonous snakes bite

Non poisonous snake bite

FATAL DOSAGE OF DRIED VENOM

- Cobra - 15 mg - alpha bungarotoxin - post synaptic
- Common Krait - 3 to 6 mg - beta bungarotoxin - presynaptic
- Saw scaled viper - 8 mg
- Russel viper - 40 mg
- Poison apparatus - Modified Salivary gland
- Bites on the head and trunk are dangerous.
- Venom is non-poisonous when taken orally.

S.No.	Feature	Neurotoxic Venom	Vasculotoxic Venom
1.	Action	It causes muscular weakness of legs and paralysis of muscles of face, throat and respiration	It causes enzymatic destruction of cell walls and coagulation disorders
2.	Site	Acts on motor nerve cells and resemble curare	Acts on endothelial cells of blood vessels, and red cells are lysed-hemolysis
3.	Local symptoms	Minimum	Severe swelling, oozing of blood and spreading cellulitis
4.	Symptoms	Cobra venom produces both convulsions and paralysis, while krait causes only paralysis	Hemorrhage from external orifices is common
5.	Examples	Elapids, such as cobra or kraits	Vipers

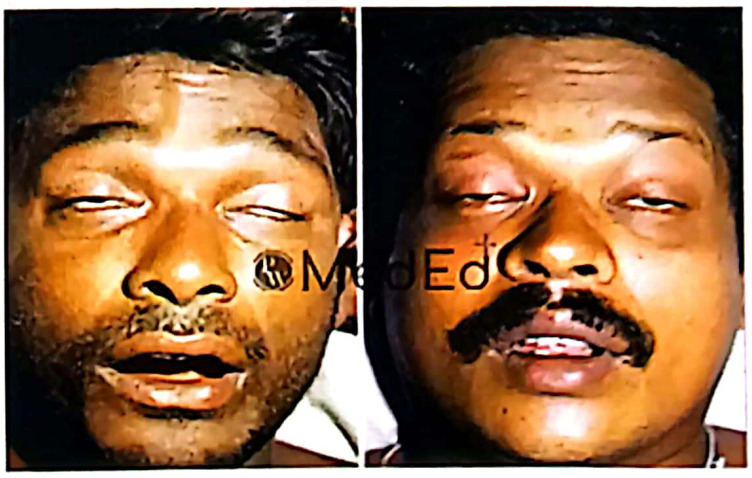
Note: Myotoxic venom produces generalized muscular pain ending in respiratory failure in fatal cases. Eg: Sea snakes.

Toxicology Snake Bite Management

Personal N

COMMON COBRA BITE

- Toxins: acetylcholine esterases, alpha bungarotoxin and cobrotoxin
- Site of action: postsynaptic
- AchE: decrease so muscle paralysis seen
- Neurotoxic: act on motor nerve cells and resemble curare
- Local symptoms appear in 6 to 8 minutes - small reddish wheal, burning pain, local necrosis causes wet gangrene with putrid smell in 1 to 2 days, skip lesions.



- Systemic symptoms appear after about 30 minutes: convulsions + paralysis, cyanosis, paralyzed tongue with hypersalivation.
- Sleepy, weakness in legs, nausea, vomiting, not able to speak, staggering gait, poor neck lift, falling po_2.
- Earliest sign: ptosis followed by external ophthalmoplegia
- Paralysis complete by 2 hours
- Respiratory failure

COMMON KRAIT BITE

- Toxins: beta bungarotoxin, acetylcholinesterase
- Site of action: presynaptic
- Similar signs and symptoms of cobra but less rapid
- Painless, no froth, no nausea and vomiting
- Abdominal pain, ptosis, dysarthria, dysphagia, quadriparesis
- No swelling, no burning pain at the site of bite

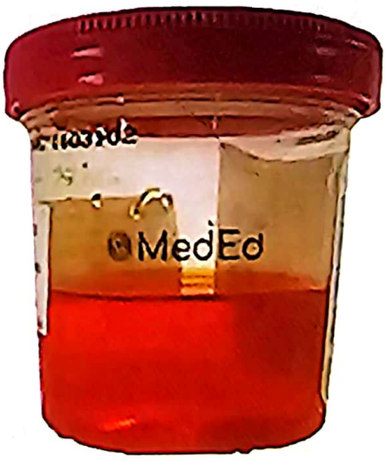
Common krait snake

- More paralysis
- Mild convulsions
- More drowsiness and intoxication is more intense
- Albumin in urine
- Nocturnal bite: people sleeping on the floor, victim early morning wake up with paralysis

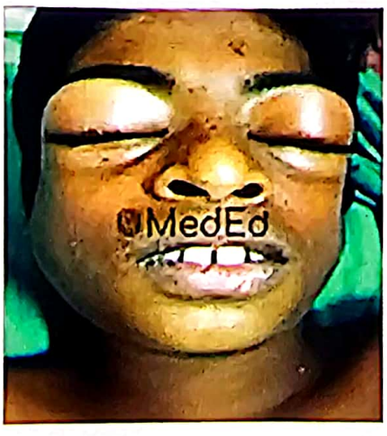
VIPER BITE

- Toxins: hyaluronidase, serine protease, phospholipase A2
- More local reactions like pain, oozing of blood
- Local necrosis leading to gangrene
- Blisters
- Bilateral parotid swelling (viper head), conjunctival edema, SCH
- Petechial hemorrhage, epistaxis, gum bleeding, hemoptysis, hematemesis, hematuria, rectal bleeding.
- Acute renal failure, anuria, oliguria, increased serum creatinine
- DIC, increased clotting time and bleeding time
- Hemotoxic

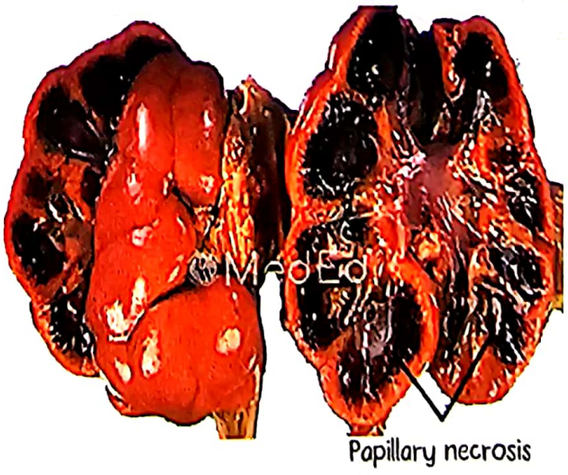
Saw scaled viper



Hematuria

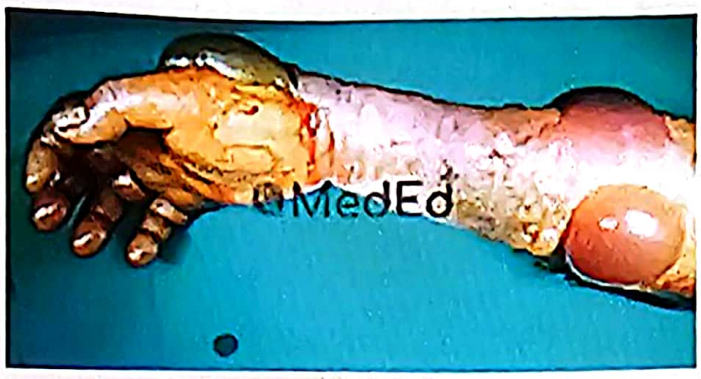


Viper head



Papillary necrosis

Kidney necrosis



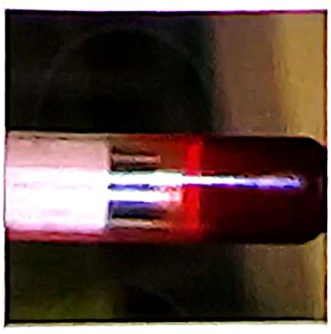
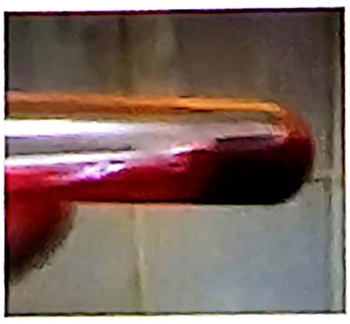
Blisters

SEA SNAKE

- Painless with minimal local symptoms
- Enlarged lymph nodes
- Sweating, vomiting, thrust
- Rhabdomyolysis (increase hb, increase myoglobin and kidney failure)
- Shoulder girdle muscle
- Lock jaw, trismus
- Myoglobinuria, hyperkalemia
- Generalized muscle pain ending in respiratory failure

Investigations:

- Enzyme immunoassay (most common) and radioimmunoassay (most sensitive)
- 20 minutes whole blood clotting test (20 WBCT)
- Single breath counting test

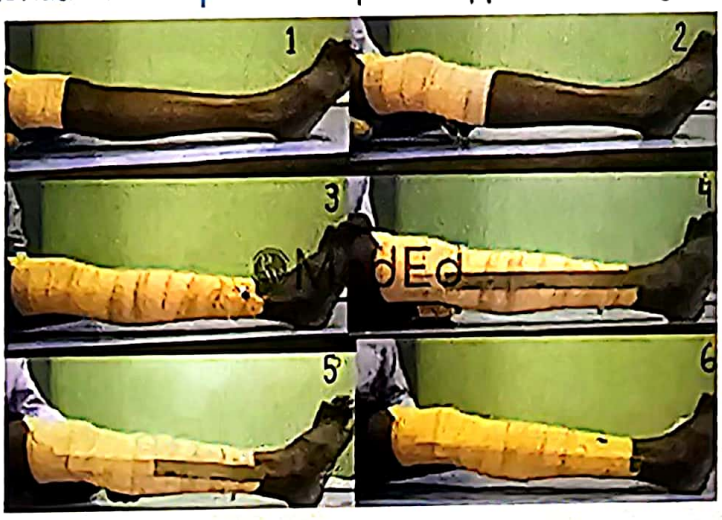
Whole Blood Clotting Test (WBCT)		
Draw 2 mL of venous blood and transfer directly into a clean and dry glass tube. Leave it upright, open, undisturbed for 20 and/or 30 minutes at room temp.	After exactly 20 minutes, pick up the tube and invert it. If a solid clot is retained, the test indicates normal coagulation.	If clot breaks down quickly upon inversion of the tube or fails to coagulate, the test indicates a coagulopathy.
		
Collection: a blood sample for WBCT testing immediately after collection.	Normal: a solid clot is retaining upon inversion of the tube at 20 or 30 minutes (Grade 0, no coagulopathy).	Abnormal: clot degrades rapidly (Grade 1, friable clot) or fails to coagulate whatsoever (Grade 2).

Whole blood clotting test

SNAKE BITE MANAGEMENT

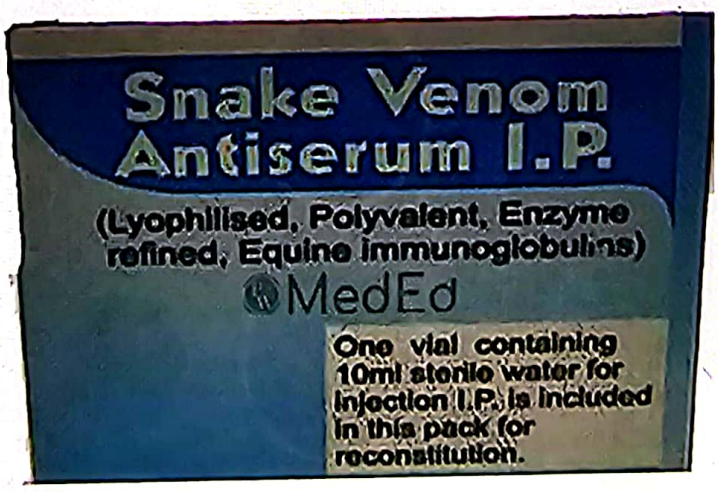
	Do it	Don'ts
R	Reassurance	Tight tourniquet
I	Immobilisation	Incision and suction of wound
GH	Get to Hospital immediately	Washing the wound
T	Telling the doctor about emergence of symptoms	Electro/cryotherapy

- Premium non nocere (first do no harm - incision over the bite, mouth suctioning, tourniquet around the limb, use of snake stones, icepack, electric shock)
- Reassurance
- Immobilization (splint or sling)
- Pressure immobilization - Sutherland wrap (if patient is > 1 hour from medical care) in elapidae bites. (not in vipers)
- Pressure pad or Monash technique - hard pad is applied directly to the wound.



Sutherland wrap (50-60 mmhg pressure)

Polyvalent antisnake venom:



Side effect of polyvalent anti snake venom: Anaphylaxis, serum sickness

How to give:

- Slow iv - 8 to 10 vials over a period of 1 hour
- Repeat doses 6 hour rule in vipers and 1 to 2 hour rule in elapidae
- Half life 90 hours/no test required/children same dose
- Max 20 in neurotoxic - 30 vials in vipers

- Ideally within 4 hours
- Procedure - freeze died antivenom dissolved in water 10 ml of viral, about 80 to 100 ml diluted in 200 to 500 ml of isotonic solution of Normal saline and given slow iv.

Supportive treatment:

- Broad spectrum antibiotics
- Tetanus toxoid
- Atropine followed by neostigmine in cobra bites
- Endotracheal intubation
- Dopamine - renal failure
- Hypovolemic shock - normal saline, ringer lactate
- Fresh frozen plasma
- Heparin

Snake bite envenomation severity scale - SESS

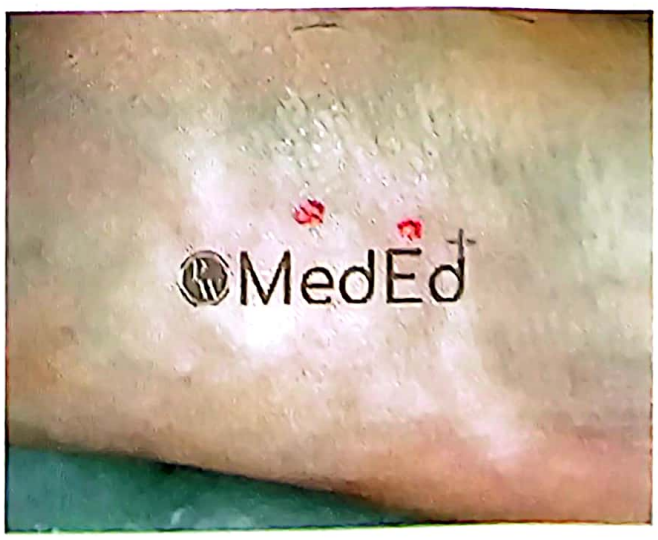
No envenomation	:	Absence of local or systemic reactions, Fang marks (+/-)
Mild envenomation	:	Fang marks (+), moderate pain, minimal local edema (0-15cm), erythema (+), ecchymosis (+/-), no systemic reactions.
Moderate envenomation	:	Fang marks (+), severe pain, moderate local edema (15-30cm), erythema and ecchymosis (+), systemic weakness, sweating, syncope, nausea, vomiting, anemior thrombocytopenia.
Severe envenomation	:	Fang marks (+), severe pain, severe local oedema (>30cm), erythema and ecchymosis (+), hypotension, paresthesia, coma, pulmonary edema, respiratory failure.

Markers of severe envenomation:

- Very venomous snake
- Rapid swelling extension
- Early lymph nodes enlargement
- Early systemic symptoms
- Early spontaneous bleeding

Postmortem findings:

- Snake bite mark - fang mark
- CPC - congestion, petechial hemorrhage, cyanosis
- Enlarged lymph nodes
- Cellulitis, hemorrhage, swelling



Medicolegal aspects of snake bite:

- Accidental - most common manner
- Homicidal - throwing snake on the bed of sleeping person (302 IPC)
- Suicidal
- Cattle poison
- Medical negligence

Toxicology Animal organic irritants

SCORPION

Personal N

One Liners:

- Eight legs
- Indian red scorpion (Mesobuthus tamulus)
- Toxicity more than snakes
- Cause of death in scorpion poison: Acute pulmonary edema
- Hemotoxic or neurotoxic or cardiotoxic
- The main toxins include phospholipase, acetylcholinesterase, hyaluronidase, serotonin and neurotoxins. The venom affects channels with prolongation of action potentials, as well as depolarization of nerves.
- Prominent radiating pain with hyperesthesia
- Clinical Symptoms - Sweating, salivation, muscle fasciculation
- Adrenals - potent stimulator

Treatment:

- Pressure immobilization
- Prazosin- 30 microgram/kg/dose orally till warm extremities
- Scorpion antivenom - Best
- Xylocaine - pain management
- Calcium gluconate - Cramps & edema
- Barbiturates - Convulsions
- Atropine - Pulmonary edema
- Medicolegal aspects are: Accidental, CPC++, Anaphylaxis at major organs

HONEY BEE/WASP

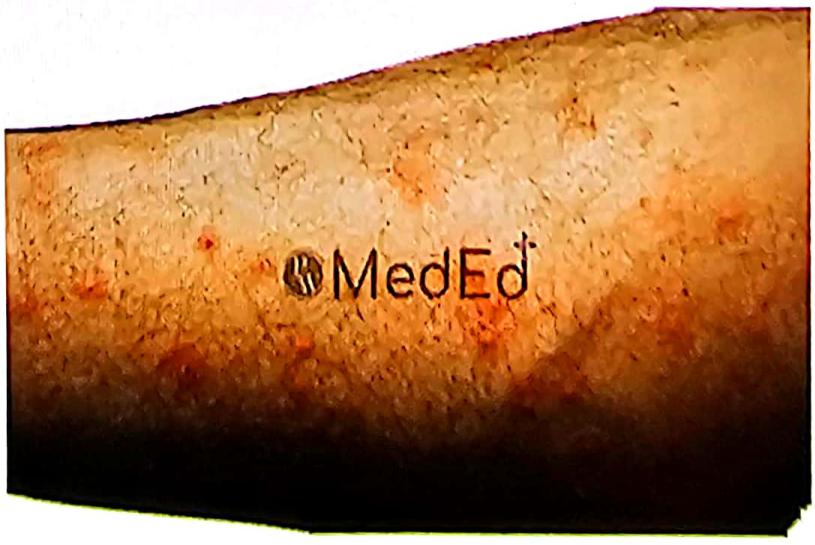
Honey bee induces release of	Wasp induces the release of
Dopamine	Serotonin
Histamine	Kinin
Neurotoxins	

- Cause of death: Anaphylactic reactions
- Fatal period: within 2- 15 minutes of bee bite (50 to 100 honey bee bites leads to death)

Treatment:

- Adrenaline - Antidote of choice
- Calcium gluconate
- Glucocorticoids

Interesting fact - A honey bee has a barbed stinger that usually remains in the skin following sting. A honeybee dies following stinging because it eviscerates itself while leaving its stinger and venom sac behind.



SPANISH FLY

- Spanish fly (Blister beetle)
- 2 cm long, and 0.6 cm broad
- Organic irritants- Redness & Burning pain & priapism
- Leads to Abortion, Convulsions and coma

- Fatal dose - 15 to 20 mg of cantharidin, Fatal period - 24 to 36 hours.
- Post mortem findings - Congested stomach, petechial hemorrhages, Hemorrhages at the kidney, live & brain, bladder, heart and lung.
- Medicolegal importance: Abortion, Aphrodisiac, accidental poisoning.

BROWN RECLUSE SPIDER

- Leads to Intravascular hemorrhage
- Acute renal failure
- Classical finding - Wheal - vasodilation (red) → vasoconstriction (white) → necrosis (Blue pre necrotic necrosis) - Red - White - Blue sign
- Well known species- "Mysore oriental" (Indian Tarantula)

POISONOUS LIZARD (GILA MONSTER)

Not present in India

Agricultural Poison - Organo compounds

- Commonest method of suicide, Insecticides, Herbicides, Antihelmenthics, Nerve gases, Ophthalmic agents.

ALKYL PHOSPHATES

Mnemonic-Moth

- Malathion: (Kill bug): 1 gram
- OMPA: 175mg
- TEPP: 100mg
- HEPP-100mg

Mnemonic-Mpdp

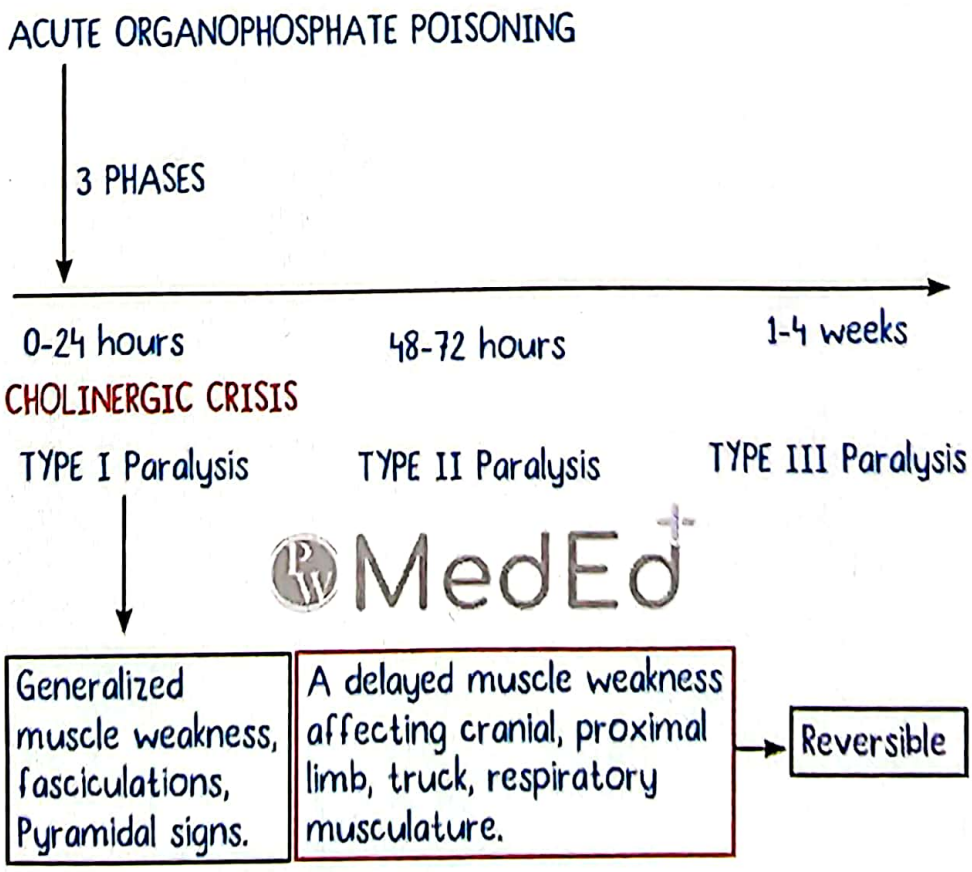
- Methyl parathion (Meta acid)
- Parathion (Follidol): 175mg (1 drop)
- Diazinon (Tik 20): 1 gram
- Paraoxon: 1 gram

MECHANISM OF ACTION

- Acetylcholinesterase inhibition
- Butyrylcholinesterase inhibition
- Irreversible
- Aging
- Carbamates: limited to CNS toxicity
- Transdermal/Transconjunctival/Ingestion/Inhalation

OP poisoning clinical features: MTF (Onset 30 mins to 2 hours):

- Salivation/seizure/sweating
- Lacrimation: Chromodacryrrhoea: porphyrins in lacrimal gland
- Urination
- Diarrhea
- Gastric upset
- Emesis
- Miosis/muscle cramps
- Tachycardia
- Fasciculations



Type II paralysis is the main cause of morbidity, mortality and high cost of treatment of acute organophosphate poisoing in hospital patients in India.

LABORATORY DIAGNOSIS

- History & Clinical features
- Estimation of cholinesterase activity: (Ach E) Mild < 10%, Moderate 10 : 50 %, Severe 50: 90%.
- P Nitrophenol test
- Mass spectrometry
- Electrophysiological test

Differential diagnosis:

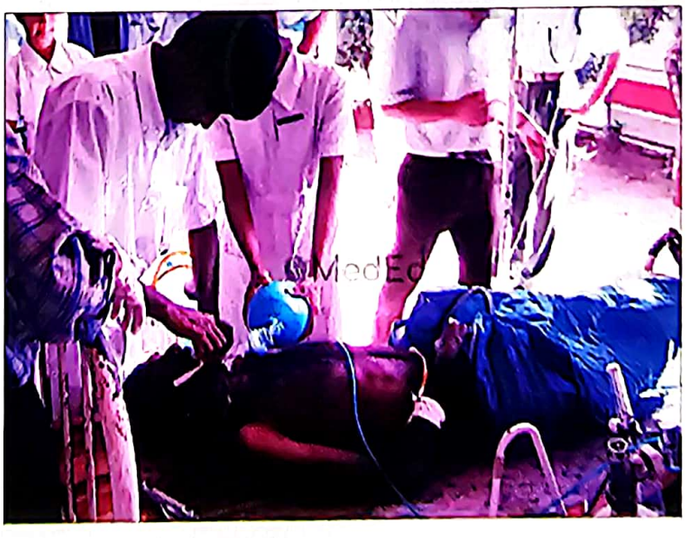
- Narcotics
- Pneumonia
- Ketoacidosis
- Meningitis

Management:

- ABCD
- Remove the patient from the site of exposure, wash with soap & water.
- Gastric lavage + Activated charcoal
- Atropine 2 to 4 mg I V
- Pralidoxime/Diacetyl Monoxime: reverse muscle weakness
- Diazepam

Medicolegal Aspects:

- Misdiagnosis: potential medical negligence
- Suicide
- Chemical warfare agent
- Accidental poisoning



Case of acute organophosphorus poisoning

Abdominal Injury

ABDOMINAL TRAUMA

- Most common injury overall: spleen
  ⇒ New update: liver > spleen
- Most common organ injury in blunt trauma: spleen
- Most common organ injury in penetrating: liver
- Most common bowel injury in penetrating: jejunum
- Most common seat belt associated: mesenteric tear

ABDOMINAL TRAUMA

Blunt trauma

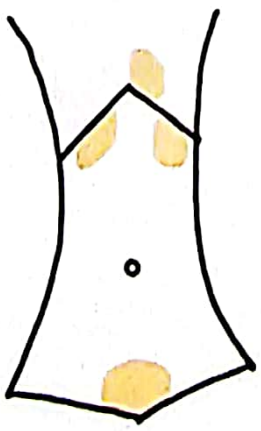
↓

Examine abdomen + Focussed Assessment with Sonography in Trauma (FAST)

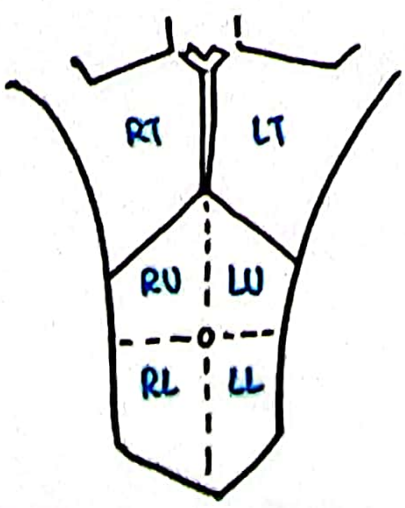
- Minimum fluid collection required for FAST (+) = ≥100CC

Sides:

1) Peri hepatic
2) Peri cardial
3) Peri splenic
4) Pelvic



eFAST: extended FAST – 6 site assessment



Penetrating

↓

1) Gunshot wound → Exploratory laparotomy
2) Stab wound

. Abdominal Wall Injuries:

Injury Points:

Abrasions/Contusions: Superficial injuries due to blunt trauma.

Lacerations: Cuts or tears caused by sharp objects.

Hematomas: Blood collection indicating internal damage.

Forensic Importance:

Indicates the type and direction of force.

Pattern of injury helps identify the weapon or object used.

Can indicate defensive wounds in an assault case.

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2. Esophageal Injuries:

Injury Points:

Perforation: Caused by sharp objects, strangulation, or forceful swallowing.

Tear (Boerhaave Syndrome): Caused by violent vomiting or trauma.

Forensic Importance:

Suggests forceful injury, likely homicide or suicide.

May involve force-feeding or torture cases.

Can indicate poisoning or caustic substance ingestion.

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3. Stomach Injuries:

Injury Points:

Perforation/Rupture: Due to stab wounds, gunshots, or blunt force trauma.

Bleeding Ulcers: May be misinterpreted in forensic cases.

Forensic Importance:

Presence of undigested food can estimate the time of death.

Poison residues can confirm homicidal poisoning.

Injury type can differentiate between accidental and intentional harm.

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4. Intestinal Injuries:

Injury Points:

Perforation/Rupture: Common in blunt trauma.

Mesenteric Tear: Severe injury causing internal bleeding.

Forensic Importance:

Indicates significant force, often seen in road traffic accidents.

Suggests delayed medical care if peritonitis is present.

Helps identify the manner of injury (accident vs. assault).

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5. Liver Injuries:

Injury Points:

Lacerations/Ruptures: Due to blunt trauma or stab wounds.

Hematomas: Indicate delayed internal bleeding.

Forensic Importance:

Massive bleeding may cause rapid death.

Right upper abdomen trauma suggests forceful impact.

Can reveal the mechanism of injury in homicides or accidents.

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6. Spleen Injuries:

Injury Points:

Laceration/Fracture: Common in left-sided blunt trauma.

Rupture: Leads to life-threatening internal bleeding.

Forensic Importance:

Suggests direct trauma, common in car accidents or falls.

Can indicate homicide if seen with multiple injuries.

Injury patterns help reconstruct the event timeline.

Overall Forensic Considerations:

Nature of Injury: Determines if the injury was accidental, homicidal, or suicidal.

Weapon Type: Identified through wound characteristics.

Time of Injury: Assessed by tissue healing and blood clotting stages.

Cause of Death: Massive internal bleeding, shock, or infection (peritonitis).

These aspects help forensic experts determine the

circumstances surrounding the injury or death.