

Corrosives: fix, erode & destroy living tissues with which they come in contact with (acids + bases)

→ convert hemoglobin to hematin

→ Alkalies are more dangerous than acids

- alkalies  $\Rightarrow$  liquefaction necrosis  $\Rightarrow$  deeper penetration

- acids  $\Rightarrow$  coagulative necrosis  $\Rightarrow$  hard eschar (prevents deeper penetration)

[except hydrofluoric acid  $\Rightarrow$  liquefactive necrosis)



### Mineral acids

- HCl
- HNO<sub>3</sub>
- H<sub>2</sub>SO<sub>4</sub>

### Organic acids

- Carbolic acid
- Oxalic acid

### Caustic alkalis

- Ammonia
- Broken battery
- Cement burns  
(calcium oxide)
- Liquid Lye (NaOH)

### Other Corrosives

- KMnO<sub>4</sub>

## Mineral Acids:

→ Acid: tastes sour, reacts with metals & carbonates, turns blue litmus paper red.

→ Mineral acid: derived from one or more inorganic compounds; does not contain a carbon atom.

- release hydrogen ions when dissolved in water

- **MoA:** protein coagulation  $\rightarrow$  coagulative necrosis  $\rightarrow$  hard eschar formation

self-limiting  $\leftarrow$  limits further penetration of acid  $\leftarrow$

- concentrated forms excret with tissue water to generate significant heat

superimposed thermal injury  $\leftarrow$

# Hydrochloric Acid (HCl): colourless, fuming, pungent liquid

→ often yellow in colour due to impurities

- Uses:
  - Bleaching agent
  - Descaler in boilers
  - Dyeing industry
  - Laboratory reagent

## Signs & Symptoms in Acute Poisoning:

- corrosive action on skin is less than that of  $H_2SO_4$  (skin is not usually corroded)
- Mucous membrane is readily corroded & destroyed.
  - Grey or grey-white MM  $\rightarrow$  brown, black  $\rightarrow$  blue  
(due to production of acid hematin)

Inhalation: Same as  $HNO_3$

- i) Coughing & dyspnea
- ii) Sneezing
- iii) Intense irritation of throat & lungs
- iv) Suffocation [feeling of asphyxiation]
- v) Cyanosis

Contact with eyes: Same as  $HNO_3$

- i) Lacrimation
- ii) Photophobia

## Signs & Symptoms in Chronic Poisoning:

→ occurs due to chronic exposure to fumes

- i) Eyes - Conjunctivitis, corneal ulcers
- ii) Nose - Coryza (inflammation of nasal mucous membranes)
- iii) Oral cavity - inflammation of gums, loosening of teeth
- iv) GIT - pharyngitis
- v) Respiratory System - bronchitis

## Diagnosis: (For all acid corrosives)

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- i) Saliva  $\Rightarrow$  pH tested by litmus paper
- ii) Stain on clothing / environment  $\rightarrow$  add few drops of  $\text{Na}_2\text{CO}_3$  /  $\text{NaHCO}_3$     
bubbles indicate acid
- iii)  $\downarrow$  PT & PTT
- iv) Arterial pH  $< 7.22 \Rightarrow$  acid ingestion
- v) Chest & abdominal radiographs: may show signs of esophageal or gastric perforation [pneumomediastinum, pneumoperitoneum]
- vi) Endoscopy: should be performed within 12 hours [safer during this period; risk of perforation  $\uparrow$  after 24 hours]

Fatal Dose: 15 - 20 mL

Fatal Period: 12 - 24 hrs

Management: Same as  $\text{HNO}_3$  &  $\text{H}_2\text{SO}_4$

### I) Systemic Ingestion:

#### i) Grade 1 esophageal injury:

$\rightarrow$  diet as tolerated

$\rightarrow$  no further therapy needed; supportive care only

#### ii) Grade 2A esophageal injury:

$\rightarrow$  if unable to tolerate PO  $\rightarrow$  provide nutritional support via nasogastric tube / orogastric tube / percutaneous feeding tube or total parenteral nutrition (TPN)

$\rightarrow$  admission recommended; supportive care

iii) Grade 2B & 3 Esophageal Injuries:

→ Admission into ICU

→ Initiate early percutaneous feeding tube or TPN

→ Prevention of stricture formation:

- Antacids

- Antibiotics

- Corticosteroids

→ If stricture has developed:

- Nasogastric tubes

- TPN ; gastrostomy may be required

- Traditional treatment for focal esophageal stricture  $\Rightarrow$  esophageal dilatation with esophagoscopy.

→ Additional imaging or surgical exploration for gastric injuries.

II) Contact with Skin & Eyes:

i) Wash the affected part with:

- plenty of water & soap or

- Sodium/potassium carbonate (in case of eyes, wash with very dilute solution of sodium bicarbonate), then instill a few drops of castor oil or olive oil.

ii) Neutralize  $\Rightarrow$  after washing, apply a thick paste of Magnesium oxide or carbonate.

PM Appearance:

i) Corrosion is less severe than in  $H_2SO_4$

ii) Stomach:

- brownish discolouration of mucosal folds

- contains brownish fluid

- perforation (rare)

iii) Respiratory passages & lungs: • Acute inflammation  
• Edema

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### Tests:

Ammonia test: place an open bottle of ammonia near stomach contents / vomitus / suspected poison  $\rightarrow$  thick white fumes of  $\text{NH}_4\text{Cl}$  are seen.

[Normal HCl of stomach is too dilute (0.2-0.5%) to produce similar fumes of  $\text{NH}_4\text{Cl}$ .]

Litmus test: suspected solution turns blue litmus red.

Silver Nitrate Test: suspected solution +  $\text{AgNO}_3 \rightarrow \text{AgCl}$  (thick white ppt.)

### MLI:

Suicide: • ingested as such  
• used in detergent suicides by producing  $\text{H}_2\text{S}$

Accident: • Gastrogenic  $\Rightarrow$  may be confused with antiseptics (negligence by medical staff)  
• In chemistry class / laboratories

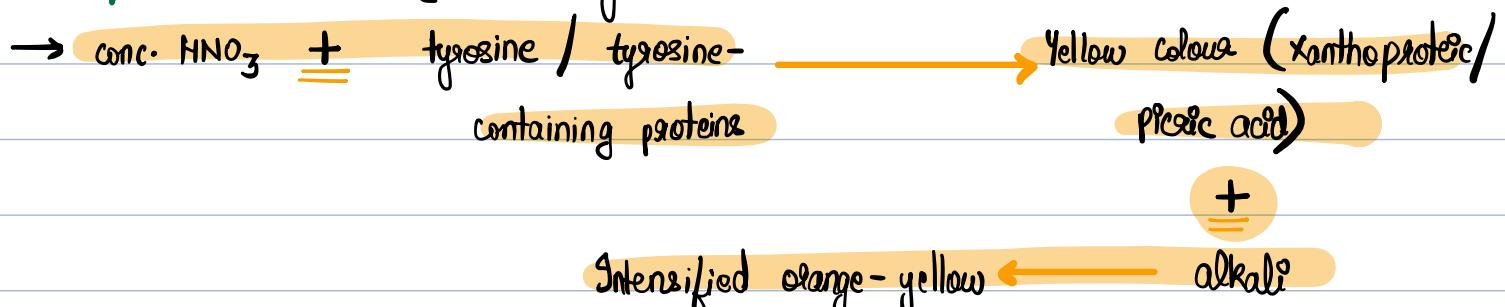
Homicidal: very rare (due to its corrosive nature)

Abortifacient: rarely introduced into vagina to produce abortion.

# Nitric Acid [HNO<sub>3</sub>]: clear, colourless, fuming, heavy liquid

→ peculiar choking odour.

Xanthoproteic Reaction: [xantho = yellow]



## Signs & Symptoms:

i) Dermal contact: yellow colour of skin (xanthoproteic reaction)

→ intense burning pain

→ immediate corrosion

→ destruction of skin

ii) Ingestion: same as H<sub>2</sub>SO<sub>4</sub>

→ Immediate: • Burning pain in mouth

• Dysphagia

• Epigastric pain (soon spreads all over thorax & abdomen)

• Odynophagia

• Pharyngeal pain

• Salivation

• Stador

→ Intense thirst, evacuations

→ Nausea, vomiting  $\Rightarrow$  brownish-black (due to acid hematin)

$\Rightarrow$  mucoid & strongly acidic

$\Rightarrow$  contains shades of charred wall of stomach

→ Voice  $\Rightarrow$  hoarse & husky (dysphonia)

→ Abdomen  $\Rightarrow$  distended & tender

$\Rightarrow$  tenesmus

→ Features of generalised shock

→ Metabolic acidosis (due to — absorption of acid — shock — severe tissue burns)

→ Leukocytosis

- Findings of face: • sunken eyes • dilated pupils • swollen lips, excoriated
- angle of mouth shows brown/black streaks from angle of mouth to side of chin
- corroded mucous membranes (mouth, throat, esophagus)
- chalky white teeth • black, sodden, swollen teeth
- Chemical peritonitis → % perforation occurs
- Late signs & symptom & sequelae of recovery: • permanent scars on skin
- stricture of oesophagus & stomach (hour-glass deformity)
- pyloric stenosis • increased propensity for carcinomas

[More excretions & abdominal distension than  $\text{H}_2\text{SO}_4$  owing to gas formation]

iii) Inhalation: same as HCl

- coughing & dyspnoea
- sneezing
- intense irritation of throat & lungs
- suffocation (feeling of asphyxiation)
- cyanosis

iv) Contact with eyes: same as HCl

- Lacrimation
- Photophobia

Fatal dose: 10 - 15 mL

Fatal period: 12 - 24 hours

Management: same as HCl

## I) Systemic Ingestion:

### i) Grade 1 esophageal injury:

→ diet as tolerated

→ no further therapy needed; supportive care only

### ii) Grade 2A esophageal injury:

→ if unable to tolerate PO → provide nutritional support via

nasogastric tube / orogastric tube / percutaneous feeding tube or  
total parenteral nutrition (TPN)

→ admission recommended; supportive care

### iii) Grade 2B & 3 Esophageal Injuries:

→ Admission into ICU

→ Initiate early percutaneous feeding tube or TPN

→ Prevention of stricture formation:

• Antacids

• Antibiotics

• Corticosteroids

→ If stricture has developed:

• Nasogastric tubes

• TPN; gastrostomy may be required

• Traditional treatment for focal esophageal stricture ⇒ esophageal dilatation with esophagoscopy.

→ Additional imaging or surgical exploration for gastric injuries.

## II) Contact with Skin & Eyes:

### i) Wash the affected part with:

• plenty of water & soap or

• sodium/potassium carbonate (in case of eyes, wash with very dilute solution of sodium bicarbonate), then instill a few drops of castor oil or olive oil.

ii) Neutralize  $\Rightarrow$  after washing, apply a thick paste of Magnesium oxide or carbonate.

### PM Appearance:

$\rightarrow$  Orange-yellow to brown stains on:

- skin of mouth (where contact has occurred)

$\rightarrow$  Esophagus: • softened mucous membrane

- yellow/brown in colour

$\rightarrow$  Stomach & duodenum: • mucous membrane — yellow-brown/green

— soft, friable

— ulcerated, easily detached

- walls may be perforated

$\rightarrow$  Inhalation of fumes:

- larynx, trachea, bronchi  $\Rightarrow$  congested

- lungs  $\Rightarrow$  congested & edematous

### Tests:

i) Litmus test: blue litmus turns red

ii) Stomach Contents / Vomit in a test tube + add strong  $\text{FeSO}_4$  sol. &  $\text{MgSO}_4$  gently from the sides of the test tube  $\rightarrow$  brown

$\text{HNO}_3$  is present.  $\leftarrow$  rings form at the junction of fluids  $\rightarrow$

iii) Stomach contents/vomit in a test tube  $\rightarrow$  drop a small piece of copper heat  $\rightarrow$  pungent dark brown fumes of Nitrogen dioxide

$\text{HNO}_3$  is present  $\leftarrow$

### MLI:

$\rightarrow$  mostly suicidal

$\rightarrow$  accident & homicide are rare

# Sulphuric Acid ( $H_2SO_4$ ):

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Signs & Symptoms: same as  $HNO_3$

## i) Ingestion:

- Immediate:
  - Burning pain in mouth
  - Dysphagia
  - Epigastric pain (soon spreads all over thorax & abdomen)
  - Odynophagia
  - Pharyngeal pain
  - Salivation
  - Stomatitis
- Intense thirst, ejections
- Nausea, vomiting ⇒ brownish-black (due to acid hematin)
  - ⇒ mucoid & strongly acidic
  - ⇒ contains shades of charred wall of stomach
- Voice ⇒ hoarse & husky (dysphonia)
- Abdomen ⇒ distended & tender
  - ⇒ tenesmus
- Features of generalised shock
- Metabolic acidosis (due to — absorption of acid — shock — severe tissue burns)
- Leukocytosis
- Findings of face:
  - sunken eyes
  - dilated pupils
  - swollen lips, excoriated
  - angle of mouth shows brown/black streaks from angle of mouth to side of chin
  - corroded mucous membranes (mouth, throat, esophagus)
  - chalky white teeth
  - black, sodden, swollen teeth
- Chemical peritonitis ⇒ of perforation occurs
- Late signs & symptom & sequelae of recovery:
  - permanent scars on skin
  - stricture of oesophagus & stomach (hour-glass deformity)
  - pyloric stenosis
  - increased propensity for carcinomas

## ii) Contact with Eyes:

- Conjunctivitis
- Corneal edema & ulceration
- Endocyclitis
- Necrotizing keratitis
- Periorbital edema

## iii) Contact with Skin:

- intense burning pain
- Immediate corrosion
- destruction of skin

## Diagnosis: (For all acid corrosives)

- i) Saliva  $\Rightarrow$  pH tested by litmus paper
- ii) Stain on clothing / environment  $\rightarrow$  add few drops of  $\text{Na}_2\text{CO}_3$  /  $\text{NaHCO}_3$  bubbles indicate acid
- iii)  $\downarrow$  PT & PTT
- iv) Axillary pH  $< 7.22 \Rightarrow$  acid ingestion
- v) Chest & abdominal radiographs: may show signs of esophageal or gastric perforation [pneumomediastinum, pneumoperitoneum]
- vi) Endoscopy: should be performed within 12 hours [safe during this period; risk of perforation  $\uparrow$  after 24 hours]

Specific for  $\text{H}_2\text{SO}_4$ :

$\rightarrow$  vomitus  $\rightarrow$  add 10%  $\text{BaCl}_2$   $\rightarrow$  Heavy white ppt.  $[\text{BaSO}_4]$

## Fatal Dose: 10-15 mL

## Fatal Period: 12 - 24 hours.

## Cause of Death:

### i) Immediate:

- Circulatory collapse (due to trauma from corrosive injury)
- spasm or edema of glottis
- perforation of stomach

### ii) Delayed:

- Hypostatic pneumonia
- Renal failure
- Secondary infection
- Starvation (due to esophageal strictures)

## Complications:

### i) Immediate:

- Atelectasis
- GI hemorrhage
- Esophageal & gastric perforation
- Tracheobronchial necrosis

### ii) Late:

- Esophageal strictures
- Pyloric stenosis
- Upper airway obstruction
- Carcinoma

## Management:

### I) Systemic Ingestion: same as HCl & HNO<sub>3</sub>

#### i) Grade 1 esophageal injury:

- diet as tolerated
- no further therapy needed; supportive care only

ii) Grade 2A esophageal injury:

→ if unable to tolerate PO → provide nutritional support via nasogastric tube / orogastric tube / percutaneous feeding tube or total parenteral nutrition (TPN)

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iii) Grade 2B & 3 Esophageal Injuries:

→ Admission into ICU

→ Initiate early percutaneous feeding tube or TPN

→ Prevention of stricture formation:

- Antacids
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→ If stricture has developed:

- Nasogastric tubes
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- Traditional treatment for focal esophageal stricture ⇒ esophageal dilatation with esophagoscopy.

→ Additional imaging or surgical exploration for gastric injuries.

II) Contact with Skin & Eyes: same as HCl & HNO<sub>3</sub>

## i) Wash the affected part with:

- plenty of water & soap or
- sodium/potassium carbonate (in case of eyes, wash with very dilute solution of sodium bicarbonate), then instill a few drops of castor oil or olive oil.

## ii) Neutralize ⇒ after washing, apply a thick paste of Magnesium oxide or carbonate.

## Progress of Injury:

- Perforation occurs immediately.
- If perforation does not occur, the following events occur
  - **immediately to 4th day:** inflammation
  - **4th day to 3 weeks:** neovascularization & fibroblast proliferation take place ⇒ new collagen is laid down, damaged tissue is replaced by granulation tissue.
  - **8 weeks onwards:** Remodelling occurs.
    - progressive narrowing of oesophageal lumen.
    - dense scar formation ⇒ stricture ⇒ dysphagia ⇒ significant nutrient deficit.

## PM Appearances:

### a) External:

- clothing: acid burns, stains
- linear burns: couring down the angles of the mouth
- burns: lip / chin / chest / hands
- Swelling: of lips & mouth (due to inflammation)
- colour of burnt areas: grayish white → brown / black & leathery  
(may simulate abrasions)

### b) Internal:

- Esophagus: perforation is rare

#### Stomach:

- **Corrosion & perforation are common**
- If corrosion is absent, there will be:
  - inflammation
  - swelling due to edema
  - severe interstitial haemorrhage
- **Consistency of stomach:** soft, spongy, black mass which readily disintegrates when touched

- Lesser curvature may be affected more (∴ acid may travel along magenstrasse)
- Pyloric region: acid cannot pass through & collect here (due to pyloric spasm)
- Antral pooling occurs.
  - Mucosal ridges: show more injury (∴ they are raised)
  - Colour of mucosa: black, charred appearance

### Preservation of Samples:

- i) Organs: vitreous humor, lungs
- ii) Clothings: may reveal acid

### Tests:

- i) Litmus test: suspected solutions turns blue litmus red
- ii) suspect material + BaCl or BaNO<sub>3</sub> → white ppt. of BaSO<sub>4</sub>
- iii) Pour suspected material over organic matter (e.g. cotton) → Charring

### MLI:

- i) Vitriolage: (aka Vitriol throwing) throwing of a corrosive substance over the face of adversary due to jealousy or revenge
  - aim is not to kill but to disfigure.
  - corrosive is filled inside an easily breakable container & thrown over the victim

#### → Outcomes of vitriolage:

- destruction of garments
- disfigurement of face (if not treated promptly)
- blindness (sometimes)
- Contractures around joints (may cause restriction of movements)
- death (may occur)

→ ML investigation:

- clothes must be collected & sent for chemical examination
- plain water swabs from affected areas (for chemical examination)

→ MLI of vitriolage:

- it is a grievous hurt
- all hospitals must provide immediate first-aid or medical treatment free of cost to any victim of vitriolage [s. 357C, CrPC]
- if treatment is not provided, punishment is 1y or fine or both [s. 166B, IPC].

ii) Accidental poisoning:

→ due to  $H_2SO_4$  being mistaken for glycerine / castor oil / linseed oil.

iii) suicide:

→ taken orally for suicide

iv) Homicide:

→ cannot be used for homicide (due to its corrosive nature)

v) Disposal of dead bodies:

→  $H_2SO_4$  may be used for criminal disposal of dead bodies after murder.

vi) For blinding an enemy or to extort confessions (used by the police)vii) Occupational hazard:

→ inadvertent inhalation of vapour in chemical factories.

viii) Abortifacient:

→ injected in vagina for criminal abortion.

ix) Battery acid: (30 - 35%  $H_2SO_4$ )

→ used in illicit manufacture of several narcotic drugs

x) Self defense:

→ acids have been used by women for defending themselves against sexual assault.

# Carbolic Acid [Phenol, $C_6H_5OH$ ]:

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## Properties:

- pure acid consists of short, colourless, prismatic, needle-like crystals
- commercial phenol is a brownish liquid containing impurities like cresol.
- on exposure to air  $\Rightarrow$  crystals turn pink & liquefy.
- characteristic carbolic/ phenolic smell
- taste: burning sweetish
- sparingly soluble in water
- Not a true acid (does not turn blue litmus red)
- used as antiseptic, disinfectant.

## Toxicokinetics:

- Absorption: readily absorbed from all routes
- Excretion: phenol is converted to pyrocatechol & hydroxyquinone in the liver  $\longrightarrow$  excreted in urine  $\longrightarrow$  further oxidation in urine  $\longrightarrow$  green-coloured urine
  - initially  $\Rightarrow$  colourless/ light-green
  - later  $\Rightarrow$  dark green
  - long-standing  $\Rightarrow$  almost black

→ time required for complete excretion = 36 hours.

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## Signs & Symptoms of Acute Poisoning:

### i) Local:

#### a) Skin:

- numbness (due to damage to nerve endings)

→ Burns: carbonic acid precipitates proteins & coagulates cell contents

- contact with skin → painless white opaque eschar → falls off in a few days → leaves a brown stain
- lytic burns produce a brownish purple colour.

b) Digestive tract:

- nausea, vomiting
- hot burning pain extending from mouth to stomach → tingling → anaesthesia
- corrosion of lips, mouth, tongue → Soon harden & become white.
- difficulty in deglutition & speech.

ii) Systemic:

a) General:

- pupils are contracted usually
- stertorous breathing
- Pulse: feeble, irregular, rapid
- Face:
  - covered with cold sweat
  - dusky cyanosis.

b) CNS:

- initial stimulation → later: depression (especially respiratory centre)
  - agitation
  - lockjaw
  - convulsions, seizures
  - headache, giddiness, confusion
  - unconsciousness, coma towards the end.

c) CVS:

- Hypotension, arrhythmias.

d) Respiratory system:

- Breathing: slow, laboured, short, infrequent gasps → progresses to respiratory failure & arrest
- laryngeal & pulmonary edema (due to irritation)
- bronchitis, bronchopneumonia (due to aspiration of vomit)

e) Liver: signs of hepatotoxicityf) Blood:

- Hemolysis
- Methemoglobinemia

g) Acid-Base Balance:

- Metabolic acidosis
- Respiratory alkalosis

h) Urine:

- scanty (suppressed)
- contains albumin & free Hb
- colour: green

i) Rare symptoms:

- fine, rapid, rhythmic contractions of the periosteal musculature resembling the chewing movements of a rabbit (rabbit syndrome)

Fatal dose: 10 - 15 g

Fatal period: 3-4 hours

Management:i) Contact:

- remove clothing, clean skin

- Management of dermal burns: irrigation should be done with poly ethylene glycol solution (water can worsen the injury)

- If PEG solution is not available  $\Rightarrow$  use water mixed with soap
- $\rightarrow$  Washing:
  - ethyl alcohol (10%)
  - methylated spirit
  - olive oil

## ii) Ingestion:

$\rightarrow$  Emetics (generally fail due to anaesthetic effect)

### Gastric lavage:

- wash carefully with lukewarm water mixed with any of the following
  - Castor oil
  - Glycerine (10%)
  - saccharated lime
  - Olive oil
  - Soap solution
  - $Mg / Na$  sulphate
- continue washing till washings are clear, colourless & odourless
- after completion of lavage, leave - medicinal liquid paraffin (250 cc)  
(in stomach)
  - $Mg SO_4$  (30g)

### Demulcents

$\rightarrow$  Normal saline with  $NaHCO_3$  (7g/l)

$\rightarrow$  Hemodialysis (in case of renal failure)

$\rightarrow$  To correct methemoglobinemia
 

- methylene blue IV
- exchange transfusion (if meth Hb  $> 70\%$ )

## Cause of death:

- i) Asphyxia
  - failure of respiration
  - edema of glottis
  - complications (ex: bronchopneumonia)

## ii) Syncope

## Signs & Symptoms of Chronic Poisoning: Phenol meroasmus

- i) General:- Anorexia
  - Vertigo
  - Headache
  - Weight loss

ii) Dark urine

iii) Pigmentation:

- yellowish (ochre-like) discolouration of cartilage, sclera & skin  $\Rightarrow$  Ochronosis  
[seen on microscopic examination]
- Macroscopically — affected tissues appear bluish grey
- D/d — Alkaptonuria

### MLI:

- used for suicide
- homicidal  $\Rightarrow$  rare because of odour & taste
- Accidental  $\Rightarrow$  due to carelessness in storage
  - $\Rightarrow$  misguided lay treatment
- introduced into vagina & uterus for criminal abortion.

### Tests:

1 ml of solution to be tested (urine) + few drops of 10% ferric chloride  $\xrightarrow{\text{Bluish colour}}$  (phenol is present)  
(salicylates also give positive test)

i) External:

- smell of phenol from body
- corrosion of skin around mouth; trickling marks at the angle of mouth (grayish/brownish)
- Tongue: white, swollen, hardened
- Lips, mouth, throat: mucous membrane is coagulated, corrugated, opaque, soddened, shows numerous small submucous haemorrhages.

ii) Internal:

- **Esophagus** - mucosa same as that of mouth & throat
  - mucosa arranged in longitudinal folds
- **Stomach** - contains reddish fluid mixed with mucus & shards of epithelium
  - smell of phenol
  - mucosa is same as that of mouth & throat
  - thick & leathery
  - necrotic mucosa showing partial separation, with severe congestion (fusiform show more damage)
- **Duodenum, jejunum, upper part of ileum:** similar changes (lesser extent)
- **Respiratory tract:** - coagulation necrosis of mucosa
  - severe congestion of submucous layers
  - laryngeal & pulmonary edema
- **Liver, Spleen:** - whitish hardened patch where the stomach is in contact with them
- **Kidney:** haemorrhagic nephritis
- **Brain:** congested, oedematous
- **Blood:** dark, semifluid, partially coagulated.

# Oxalic Acid:

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- Two forms:
  - anhydrous
  - dihydrate (more common)
- colourless, transparent prismatic crystals
- resembles in appearance to magnesium sulfate & zinc sulfate
- Oxalic acid may be administered in mistake for Epsom salts.

Feature	Oxalic acid	$\text{Mg SO}_4$ / $\text{Zn SO}_4$
Taste	Sour	Bitter
Reaction	Strongly acidic ( $\text{pH} < 7$ )	Neutral ( $\text{pH} = 7$ )
On heating with sodium	Sublimates	Does not sublimate
On heating with bicarbonate	Effervesces	Does not effervesce
Application on ink stains or plant moulds	Stains disappear	Do not disappear

## Occurrence:

- present in many plants
- kidney stones: Calcium oxalate  $\Rightarrow$  most common component
- Bacteria: produce oxalates from oxidation of carbohydrates.

## Uses:

- book binding
- bleach
- cleaning copper
- ink remover
- photography
- illegal:
  - removing writing & signature from papers
  - removing election stain marks on fingers

## Mechanism of Action:

Locally: acts as a corrosive

Systemically: Reacts with Ca in plasma → forms calcium oxalate

i) Hypocalcemia

ii) Precipitation of Ca oxalate crystals in liver, kidneys, heart & lungs

iii) Excretion of envelop-shaped crystals of calcium oxalate in urine

## Signs & Symptoms:

### i) Contact:

→ skin: may be damaged &/or discoloured

→ mucosa of eye, mouth: greatly damaged → scalded appearance

(sometimes, production of acid haematin may give rise to black colour)

### ii) Ingestion:

#### \* Immediate:

→ burning, sour/bitter taste in mouth

→ sense of constriction around the throat

→ intense thirst

→ mouth may appear scalded or black

→ severe pain begins in epigastrium & soon radiates all over the abdomen

→ abdomen is tender

→ persistent vomiting, eructations & diarrhoea.

• vomitus contains altered blood & mucus → coffee-ground appearance

→ signs & symptoms due to hypocalcemia: • tetany

• numbness & tingling of fingertips & legs

• Chvostek sign positive

\* Delayed: due to renal failure (calcium oxalate crystals in kidneys)

→ urine: scanty / suppressed

Contains traces of blood, albumin & Ca oxalate crystals.

## Management:

- i) Gastric lavage: with Ca salts → converts acid into insoluble calcium oxalate
- ii) Antidote: Calcium salts (chloride, gluconate, lactate, chalk powder)
- iii) Calcium gluconate IV: 10ml of 10% at frequent intervals
- iv) Parathyroid extract: 100 units IM in severe cases (mobilizes  $\text{Ca}^{2+}$  from bones)
- v) Dialysis or exchange transfusion: for renal failure
- vi) Miscellaneous:
  - demulcents
  - symptomatic
  - evacuation of bowel by castor oil / enema

Fatal dose: 600 mg/kg

Fatal period: 1-2 hours

## PM appearances:

i) Mucus membrane of tongue, mouth, pharynx, esophagus:

conc.	→ whitened (as in bleached)	→ similar to scalded appearance
	sol.	→ sometimes brown/black due to formation of acid hematin
		weak sol.

ii) Esophagus: corrugated mucosa, longitudinal erosions

iii) Stomach:

- Mucosa:
  - soft & reddened
  - punctate erosions
  - corrosion

- may be black (acid hematin)
- numerous dark brown/black streaks running longitudinally

→ Contents: gelatinous & brownish (acid hematin)

→ Perforation (rare)

iv) **Intestine:** corrosion in upper part of duodenum

v) **Liver:** hepatic centrolobular necrosis

vi) **Kidneys:**

- congested & swollen by edema
- swelling & retraction of glomeruli
- renal tubules are full of Ca oxalate & necrosed.

vii) **Urinary bladder:** urine with calcium oxalate crystals (envelope-shaped)

viii) **All intestinal organs:** congestion

ix) **Crystals of calcium oxalate:** in renal cortex & vessels & capillaries of liver, lung, heart.

### Test:

Suspected soln +  $\text{Ba NO}_3$   $\longrightarrow$  White ppt. of Barium oxalate  
(soluble in  $\text{HCl}$  or  $\text{HNO}_3$ )

### MLI:

→ accidental poisoning: due to mistaken identity with Epsom salt or  $\text{Zn SO}_4$

→ homicide: rare (due to acid sour taste)

→ suicide: rare (due to severe pain & burning)

→ Abortifacient: by vaginal injection

## Ammonia: (caustic alkali)

→ At room temperature,  $\text{NH}_3$  is highly water-soluble, colorless, irritant gas with a pungent choking odour

→ Ammonium hydroxide (aka Liquor ammonia / ammonia water / aqua ammonia)

- solution of ammonia in water (~ 30% ammonia)

### Signs & Symptoms: (seen on inhalation)

i) Head, ears, eyes, nose, throat (HEENT): facial & oral burns & ulcerations

ii) Respiratory system: • cough • oxygen desaturation • rhonchi • salivation  
• stridor • tachypnoea • wheezing

iii) CNS: loss of consciousness

### Diagnosis: HCl test

Place an open bottle of conc. HCl near stomach contents / vomitus / suspected poison

→ thick white fumes of  $\text{NH}_4\text{Cl}$  (confirmatory)

### Management:

→ mainly supportive → treating hypoxia, bronchospasm, acute lung injury (ALI), hypovolemia, burns of skin & eyes.

Fatal dose: 15-20 mL

## Button Battery Ingestion: (caustic alkali)

Signs & symptoms:

- irritability
- dysphagia
- refusal to eat
- pain
- vomiting

Diagnosis: X-ray

Management: Endoscoping or surgical removal is necessary because of:

- burns
- perforation
- metal poisoning

## Cement Burns: (caustic alkali)

### Mechanism of Action:

→ cement contains lime (calcium oxide) → penetrates clothing → reacts with sweat  
 exothermic reaction

→ even when not exposed to moisture → dry powder is very hygroscopic → causes  
 dessication injury

→ Hydrated calcium oxide  $\xrightarrow{\text{converts to}}$  Calcium hydroxide  $\xrightarrow{\text{Hydroxyl ion causes skin damage}}$

Signs & Symptoms: cement burns have an insidious onset

→ only mild irritation initially.

## Management:

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- remove soaked clothing
- abundant washing of wounds
- Neutralization with buffered phosphate solution

## Liquid Lye (NaOH / Caustic Soda): (caustic alkali)

- NaOH is found in many industrial solvents & cleaners

Signs & symptoms: • Burns      • Irritation      • Necrosis of skin & underlying tissues

Management: As for general alkalis

MLI: may be seen in vehicular accidents as air bag ruptures.

## Potassium Permanganate (KMnO<sub>4</sub>): aka Condy's crystals

- dark purple slender crystals having a sweet astringent taste
- strong oxidizing agent ∴ used as a disinfectant

### Mechanism of Action:

→ Before absorption (in solid state / strong solution): acts as corrosive / strong irritant  
Causes coagulation necrosis ←

→ After absorption: cardiac stoppage

### Signs & symptoms:

#### i) Ingestion:

→ intense thirst, nausea, vomiting, diarrhea

• Vomitus is purple brown

• Stools are black due to manganese sulfide

→ Burning pain from mouth to stomach

→ Dysarthria, dysphagia

- RS: severe inflammatory edema →
  - dyspnea
  - stridor
  - persistent spasmodic cough
- Purple brown discolouration: of skin, mucus membranes (with which it comes in contact)
  - lips, gums, teeth, tongue, tonsils, pharynx
  - colour changes to brown in a few moments & then to coal black due to conversion to manganese dioxide.

- Systemic: • Methemoglobinemia (because of oxidizing nature of  $\text{KMnO}_4$ )

#### ii) Local Application: locally applied as abortifacient -

- vaginal & cervical burns, erosions & ulcerations → severe scarring
- extensive bleeding → shock

#### Diagnosis:

- i) Stains → Place a drop of  $\text{H}_2\text{O}_2$  or soln. of oxalic acid & traces of  $\text{H}_2\text{SO}_4$
- ii) ↑ in serum & urine manganese levels

stains disappear ←

Fatal dose: 5-10g

Fatal period: Few hours

#### Management:

→ Immediate dilution (with water/milk)

→ Activated charcoal

→ Demulcents

→ Gastric lavage:

- may have to be done carefully (since  $\text{KMnO}_4$  is a corrosive)

- fluids used - dilute  $\text{H}_2\text{O}_2$

- 20% sodium thiosulphate

- end point - when returning fluid is colourless

→ Methemoglobinemia: treated with methylene blue 1-2 mg/kg IV over 5 min every 4 hours.

→ Chelation: done when manganese toxicity is suspected

- Administer EDTA & sodium para-aminosalicylic acid.

→ Supportive & symptomatic

### PM appearances:

→ Mucus membranes of GIT: (from lips to intestines) ⇒ corrosion, necrosis, haemorrhage

→ Liver & kidneys: degenerative changes seen.

### MLI:

#### i) Manner of poisoning:

→ Suicidal: most commonly

→ Accidental:

- in children who eat crystals mistaking them for candies
- when ingested/ injected as an abortifacient

→ Homicidal: very rare (due to its colour & peculiar taste)

#### ii) Production of fictitious injuries: by applying a tablet to the skin for 10-20 min

lesions similar to those of tertiary syphilis may be produced



	<u>HCl</u>	<u>HNO<sub>3</sub></u>	<u>HaSO<sub>4</sub></u>
signs & symptoms	intro + inhalation + eyes + chaotic	xanthopotic + dermal + ingestion + inhalation + eyes	ingestion + eyes + + skin
Management	systemic ingestion + skin, eyes	systemic ingestion + skin, eyes	systemic ingestion + skin, eyes
PM appearance	corrosion + stomach + RS	skin + oesophagus + stomach, duodenum inhalation	external + internal
Tests	Ammonia + litmus + AgNO <sub>3</sub>	litmus + brown ring + Cu, heat	10% BaCl <sub>2</sub>
MLI	— — —	— — —	— — —