CAUSTICS :
INORGANIC ACIDS AND ALKALIS

INTRODUCTION

- **CAUSTICS**

- Caustic or corrosive agents have the potential to cause tissue injury (burning, corroding, dissolving, or eating away) by the process of chemical reaction on contact with mucosal surfaces.

- A wide variety of chemical and physical agents may cause corrosive injury. These include mineral and organic acids, alkalis, oxidizing agents, denaturants, some hydrocarbons, and agents causing exothermic reactions.

Table 5.1: Common Caustics

Acids		*Alkalis*	*Others*
Inorganic (Mineral)	**Organic**		
Boric	Acetic	Ammonia	Hydrogen peroxide
Chromic	Carbolic	Calcium hydroxide	Iodine
Hydrochloric	Citric	Potassium carbonate	Potassium permanganate
Nitric	Formic	Potassium hydroxide	Quaternary ammonium compounds
Phosphoric	Oxalic	Sodium carbonate	
Sulfuric		Sodium hydroxide	

MECHANISM OF ACTION

- The structure of biological proteins involves not only a specific amino acid sequence, but also a three dimensional structure dependent on weak forces, such as hydrogen bonding or van der Waal's forces.
- These three dimensional structures are key elements for the biological activity on the proteins, and are easily disrupted by external factors.
- Application of heat or chemicals, especially pH disturbances, can cause the structures to fall apart.

- In thermal injuries, there is a rapid coagulation of protein due to irreversible cross-linking reactions, whereas in chemical burns the protein destruction is continued by other mechanisms, mainly hydrolysis. These mechanisms may continue so long as traces of the offending agent are present, especially in deeper layers.

- Acids are hydrogen containing substances that on dissociation in water produce hydronium ions. They are potent desiccants with the ability to produce coagulation necrosis of tissues on contact

The severity of a chemical burn injury can be determined by:

- ✓ concentration, pH
- ✓ quantity of burning agent, molarity
- ✓ duration of skin contact
- ✓ penetration
- ✓ mechanism of action

SULFURIC ACID

SULFURIC ACID

- **Synonym** : Oil of vitriol; Oleum; Battery acid

- **Physical Appearance_** : heavy, oily, colourless, odourless, non-fuming liquid, hygroscopic.

- **Uses/Sources**:

I. Raw material in the manufacture of a number of chemicals.

e.g. acetic acid, hydrochloric acid, dyes, pharmaceuticals, detergents, paint, etc.

I. Storage batteries utilise sulfuric acid as an electrolyte. Sulfuric acid is also used in the leather, fur, food processing, wool, and uranium industries, for gas drying, and as a laboratory reagent .

II. Sulfuric acid can be formed in smog from the photochemical oxidation of sulfur dioxide to sulfur trioxide and subsequent reaction with water. It is a major component of acid rain

- **Usual Fatal Dose** :

  About 20 to 30 ml of concentrate sulfuric acid. Deaths have been reported with ingestion of as little as 3.5 ml.

- **Toxicokinetics** :

  Systemic absorption of sulfuric acid is negligible.

- **Mode of Action** :

  Produces coagulation necrosis of tissues on contact

Clinical Features

- ✓ Contact with the eyes can cause severe injury including conjunctivitis, periorbital oedema, corneal oedema and ulceration, necrotising keratitis, and iridocyclitis.

- ✓ Tongue is usually swollen, and blackish or brownish in colour. Teeth become chalky white. There may be constant drooling of saliva which is indicative of oesophageal injury.

- ✓ Damage to the larynx during swallowing; may be indicated as dysphonia, dysphagia, and dyspnoea

- ✓ Burning pain from the mouth to the stomach. Abdominal pain is often severe.
- ✓ The vomitus is brownish or blackish in colour due to altered blood (coffee grounds vomit), and may contain shreds of the charred wall of the stomach
- ✓ Metabolic acidosis, particularly following ingestion. Acidosis may be due to severe tissue burns and shock, as well as absorption of acid.

- **Chronic Exposure**

- ✓ Occupational exposure to sulfuric acid mist can cause erosion of teeth over a period of time, as also increased incidence of upper respiratory infections.

- ✓ Sulfuric acid can react with other substances to form mutagenic and possibly carcinogenic products such as alkyl sulfates. chronic exposure to sulfuric acid fumes may be linked to carcinoma of the vocal cords and nasopharyngeal carcinoma

- **Diagnosis**

- ✓ Fresh stains in clothing may be tested by adding a few drops of sodium carbonate. Production of effervescence (bubbles) is indicative of an acid stain.

- ✓ If vomitus or stomach contents are available, add 10% barium chloride. A heavy. white precipitate forms which is insoluble on adding 1 ml nitric acid

- ✓ Litmus test: The pH of the saliva can be tested with a litmus paper to determine whether the chemical ingested is an acid or an alkali (turns red in acid, and blue in alkaline solution).

- **Treatment**

- ✓ Following measures are contraindicated: oral feeds, induction of vomiting, stomach wash, and use of activated charcoal

- ✓ Remove all contaminated clothes and irrigate exposed skin copiously with saline. Non-adherent gauze and wrapping may be used.

- ✓ Administration of water or milk if the patient is seen within 30 minutes of ingestion (120–240 ml in an adult, 60–120 ml in a child). But no attempt must be made at neutralisation with alkalis, since the resulting exothermic reaction can cause more harm than benefit.

- ✓ Studies indicate that even administration of buffering agents such as antacids can produce significant exothermic reaction.

- ✓ Eye injury should be dealt with by retraction of eyelids and prolonged irrigation for at least 15 to 30 minutes with normal saline or lactated Ringer's solution, or tap water if nothing else is available. Anaesthetic agents and lid retractors may be necessary. It is desirable to continue with the irrigation until normal pH of ocular secretions is restored, which can be tested with litmus paper . Slit lamp examination is mandatory after decontamination, to assess the extent of corneal damage.

- ✓ **Oral feeds**: Depends on degree of damage as assessed by early endoscopy. The following is a rough guide
- Mild (GRADE I): May have oral feedings on first day.
- Moderate (GRADE II): May have liquids after 48 to 72 hours.
- Severe (GRADE III): Jejunostomy tube feedings after 48 to 72 hours.

- Respiratory distress due to laryngeal oedema should be treated with 100% oxygen and cricothyroidotomy.
- Administration of steroids has been shown to delay stricture formation (in animals) when given within 48 hours of acid ingestion, but the practice is generally not recommended because of increased risk of perforation.
- Dosage recommended is 60 to 100 mg/day of prednisolone for the first 4 days, followed by 40 mg/day for the next 4 days, and finally 20 mg/day for the subsequent 7 to 10 days. In children, the appropriate dose is 2 mg/kg/day.
- Alternatively 0.1 mg/kg of dexamethasone or 1 to 2 mg/kg of prednisone can be given for 3 weeks and then tapered off.

- Administer antibiotics only if infection occurs. Prophylactic use is not advisable unless corticosteroid therapy is being undertaken.
- For severe pain, powerful analgesics such as morphine have to be given.
- The use of flexible fibre optic endoscopy is now standard practice in the first 24 to 48 hours of ingestion to assess the extent of oesophageal and gastric damage.
- Emergency laparotomy is mandatory if there is perforation or peritonitis.
- Follow-up is therefore essential to look for signs of obstruction, nausea, anorexia, weight loss. Surgical procedures such as dilatation, colonic bypass, and oesophagi gastrostomy may have to be undertaken.

NITRIC ACID

NITRIC ACID

- **Synonym :** Aqua Fortis; Azotic acid; Engraver's acid; Hydrogen nitrate.

- **Physical Appearance** : Nitric acid is a colourless or yellowish fuming liquid with an acrid, penetrating odour. It is essentially a solution of nitrogen dioxide (NO_2) in water and is available commercially in several forms.

- **Uses/Sources** :

✓ Glassblowing, engraving and electroplating, underground blasting operations, farming , welding, fire fighting, and industrial chemistry

✓ Nitric acid is formed in photochemical smog from the reaction between nitric oxide and hydrocarbons.

- **Usual Fatal Dose** : 20 to 30 ml
- **Toxicokinetics** : Systemic absorption is negligible.
- **Mode of Action** : Nitric acid is a powerful oxidising agent and reacts with organic matter to produce trinitrophenol, liberating nitrogen monoxide(xanthoproteic reaction). Corrosion is less severe when compared to sulfuric acid
- **Clinical Features**

✓ Corroded areas appear yellowish due to xanthoproteic reaction . Stains on clothing and teeth also appear yellowish.

✓ Abdominal distension due to gas formation.

✓ Inhalation of fumes can produce coughing, rhinorrhoea, lacrimation, dyspnoea, and pulmonary oedema

- **Diagnosis**:

- ✓ **Litmus test**
- ✓ Drop a small piece of **copper** into the stomach contents and heat it. Pungent, dark brown heavy fumes shows nitric acid is present in sufficient concentration.

- **Treatment:**

- ✓ Same as for sulfuric acid.
- ✓ Respiratory distress is present more often.
- ✓ Requires special attention and provide supportive measures.

HYDROCHLORIC ACID

HYDROCHLORIC ACID

- **Synonyms :**Muriatic acid; Spirit of salts.

- **Physical Appearance :**Hydrochloric acid is a colourless, fuming liquid which may acquire a yellowish tinge on exposure to air . It is actually hydrogen chloride in water.

- **Uses:** Bleaching agent (less than 10% HCl),Dyeing industry ,Metal refinery, Flux for soldering ,Metal cleaner, drain cleaner , Laboratory reagent.

- **Usual Fatal Dose:** About 30 to 40 ml.

- **Diagnosis** :

Litmus test

- ✓ If an open bottle of concentrate ammonia solution is placed near the stomach contents or vomitus, copious white fumes of ammonium chloride will be seen. Though normal stomach contents contain hydrochloric acid, this is usually too dilute (0.2 to 0.5%) to vitiate the value of this test.

- Treatment :

- ✓ Same as for sulfuric acid, except that corroded areas are more likely to be greyish, and symptoms are generally less severe. But respiratory manifestations are more pronounced

HYDROFLURIC ACID

HYDROFLURIC ACID

- **Physical Appearance :** Colourless, fuming liquid.
- **Uses :** Industry: 90% solution: petroleum refining, pharmaceutics, and germicides. 10% solution: tanning, glass and metal etching, and rust removal. Laboratory chemical. Window cleaning.
- **Usual Fatal Dose :** Unclear, but is probably in the range of 10 to 15 ml.
- **Toxicokinetics:** Ingestion of hydrofluoric acid may be associated with significant systemic absorption and manifestations such as hypocalcaemia, acidosis, and shock.

- **Mode of Action :**

- ✓ It is a unique acid, in that most of its toxicity is due to the anion, fluoride, and not to the cation, hydrogen. Most acids cause burns and necrosis from liberated hydrogen ions .
- ✓ Hydrofluoric acid burns result in severe progressive tissue and bone destruction, and severe pain.
- ✓ The fluoride ion then proceeds to affect tissue integrity and metabolism in three ways:

1. Liquefactive necrosis.
2. Decalcification and destruction of bone.
3. Production of insoluble salts—calcium and magnesium fluoride.

These effects result in hypocalcaemia and hypomagnesaemia.

- **Clinical Features:**

- ✓ Severe pain in fingers with destruction and loss of nail, and sometimes even the entire terminal phalanx.
- ✓ The fluoride ion may cause decalcification and corrosion of bone beneath the area of dermal burn.
- ✓ Bone destruction is extremely painful.
- ✓ Inhalation causes severe throat irritation, cough, dyspnoea, cyanosis, lung injury and non cardiogenic pulmonary oedema.
- ✓ Ingestion is associated with severe, burning pain followed by retching and vomiting. There is often haemorrhagic gastritis and frank hematemesis.
- ✓ Hypocalcaemia, hypomagnesaemia, hyperkalaemia, metabolic acidosis.

- **Treatment**

- ✓ Wash burnt areas with water, preferably under a shower or tap for at least 15 to 30 minutes
- ✓ Soak the affected portion in iced solution of 25% magnesium sulphate, or any high molecular weight quaternary ammonium compound such as benzethonium chloride or benzalkonium.
- ✓ If Hydrofluoric Acid has been ingested, attempt immediate administration of a fluoride binding substance such as milk, chewable calcium carbonate tablets, or milk of magnesia. Avoid large amounts of liquid, since this may induce vomiting.
- ✓ Inhalation injury is treated by removing the victim from the scene into fresh air, followed by decontamination of the clothes and skin.

- ✓ Ocular exposure should be treated by irrigation of the eye for at least 30 minutes. Local ophthalmic anaesthetic drops may be instilled to obtain patient compliance for the prolonged irrigation. The pH of the eye fluid should be periodically checked with litmus paper, and irrigation is continued until it is normal.
- ✓ Stomach wash is risky and best avoided. But it may be done if spontaneous vomiting has not occurred, and the time between ingestion and treatment is less than 90 minutes. Addition of 10% calcium gluconate to the lavage fluid may provide some free calcium to bind the fluoride.
- ✓ Topical Skin Therapy: For exposure to weak solutions of hydrofluoric acid (less than 20%), local application of 2.5% calcium gluconate gel is the treatment of choice. After applying the gel, an occlusive barrier can be used (e.g. vinyl gloves or plastic wrap).

PHOSPHORIC ACID

PHOSPHORIC ACID

- **Physical Appearance**: Phosphoric acid is a clear, colourless, odourless, unstable, orthorhombic crystalline solid, or a syrupy liquid with a pleasing acid taste (when suitably diluted).

- **Usual Fatal Dose:** About 300 to 500 ml.

Uses :

- ✓ Phosphoric acid is used as a flavouring material, an acidulant, and a synergistic antioxidant and sequestrant in carbonated beverages.
- ✓ Phosphoric acid is also utilised in dental ceramics, for water treatment, in engraving processes, for the rustproofing of metals prior to painting, for metal pickling, in the coagulation of rubber latex, and as an analytic reagent in laboratories.
- ✓ Dilute phosphoric acid has been used in preparations for the management of nausea and vomiting, and in Great Britain, a technical grade of orthophosphoric acid in water, 1:330, is an approved disinfectant for foot-and-mouth disease.

- **Clinical Features**

✓ Corrosion of GI tract, sour acrid taste, vomiting, abdominal pain, bloody diarrhoea, acidosis, convulsions, coma, death.

✓ Hypophosphatemia and hypocalcaemia are common findings. There may also be hypomagnesaemia.

- **Diagnosis**

✓ Add 2 ml of ammonium molybdate solution, followed by 1 ml of nitric acid to a small quantity of gastric aspirate and heat for 1 minute. A canary yellow precipitate is specific for phosphoric acid.

- **Treatment**

✓ On general lines the treatment is same as that for sulfuric acid.

✓ Convulsions can be managed with benzodiazepines or barbiturates.

✓ Hyperphosphataemia (if severe) can be treated by haemodialysis.

BORIC ACID

BORIC ACID

- **Physical Appearance**
  - ✓ Boric acid is a white powder or crystalline solid.
  - ✓ It is an odourless solid.
  - ✓ Sodium tetra borate decahydrate and pentahydrate are white odourless, crystalline solids.
- **Usual Fatal Dose**
  - ✓ About 15 to 30 grams.

Uses:

1. Medical
   - ✓ Borates have been used in a wide variety of pharmaceutical preparations including medicated powders, skin lotions, mouthwash, toothpaste, and eyewash solutions.
   - ✓ Boric acid and borax are used in cosmetics and oral hygiene products.
   - ✓ Boric acid has been used as a preservative for urine samples.

2. Household
   - ✓ Borates have been used as a home remedy for diaper rash and oral discomfort in infants.
   - ✓ Boric acid powder mixed with flour or sugar is used to kill ants and cockroaches in the home. Commercially available insecticides and herbicides used in the home may contain borates.

3. Industrial
   - ✓ Borates are used in making heat-resistant glass, glazes, enamels, fire-resistant materials and agents, paints, photographic agents, and as insecticides and herbicides.
   - ✓ They are used to preserve wood, and also as flame retardants in wood and textiles.

- Toxicokinetics
  - ✓ Absorption occurs through GI tract when ingested (though quite slowly), through lungs when inhaled (especially in the form of pentaborate gas), and possibly also through skin.
  - ✓ Serum and urine borate levels do not correlate well with the clinical state, though symptoms of toxicity generally occur only when blood levels exceed 100 to 150 mg/ml.

- **Clinical features**

1. **Acute Poisoning**
   - ✓ GIT: nausea, vomiting (bluish green), hematemesis, diarrhoea (bluish green), epigastric pain.
   - ✓ CNS: headache, tremor, convulsions, delirium, coma.
   - ✓ CVS: hypotension, shock.
   - ✓ Renal: oliguria, anuria, renal failure.
   - ✓ Acid-base: metabolic acidosis.
   - ✓ Dermal: erythema, desquamation, and exfoliation ("boiled lobster syndrome")

2. **Chronic Poisoning—**
   - ✓ Usually seen in children who have been treated with a boric acid preparation for diaper rash.
   - ✓ Apart from skin manifestations, there may be oliguria, renal tubular necrosis, and renal failure.
   - ✓ There may also be hypo- or hyperthermia, alopecia, and hypoplastic anaemia.
   - ✓ Fatalities have been reported.

- **Diagnosis**:

- ✓ **Urine test**: One drop of the patient's urine acidified with HCl is added to turmeric paper. Development of a brownish red colour is suggestive of boric acid or borates.

- ✓ Blood borate levels may be useful to establish the diagnosis of borate intoxication.

- ✓ Monitor renal function tests, cardiovascular status, fluid and electrolyte balance in symptomatic patients

- **Treatment**

- ✓ Induction of emesis or gastric lavage.
- ✓ Administration of a cathartic (e.g. magnesium sulfate).
- ✓ Forced diuresis: Rule out renal damage. Urinary elimination may be enhanced by administration of 0.45% saline in 5% dextrose in water IV, along with a diuretic (e.g. furosemide 1 mg/kg, up to 40 mg/dose). Urine flow should be maintained between 3 to 6 ml/kg/hour.
- ✓ Peritoneal or haemodialysis.
- ✓ Supportive measures: Correction of shock, convulsions, etc.
- ✓ Skin exposure must be treated by washing the affected area several times with soap and water. Eye contact is treated by irrigation with water for at least 20 minutes. Ophthalmological consultation may be required.

CHROMIC ACID

CHROMIC ACID

- **Source:**
- ✓ It is a derivative of the metal chromium, being one of the hexavalent chromium compounds, while bivalent and trivalent compounds include chromic oxide, chromic phosphate, and chromic sulfate.

- ✓ Corrosive in nature, and can cause oral burns and tissue ulceration.

- **Clinical features**
- ✓ Vomiting, diarrhoea, GI bleeding, and manifestations of renal failure.
- ✓ Intense gastrointestinal irritation or ulceration and corrosion, epigastric pain.
- ✓ vertigo, fever, muscle cramps, haemorrhagic diathesis, intravascular haemolysis, circulatory collapse, peripheral vascular collapse.
- ✓ Liver damage, acute multisystem shock, coma, and even death, depending on the dose.

- Treatment
- ✓ Chelation therapy with BAL may be helpful. Haemodialysis and exchange transfusion have also been successfully tried. Dimercapto-propane-sulfonic acid (DMPS) is a new drug with promising results.
- ✓ Dichromate (sodium, potassium, and ammonium) are important hexavalent chromium compounds which display some significant differences when compared to other chromium compounds. Ingestion of dichromates can cause vertigo, abdominal pain, vomiting, convulsions, severe coagulopathy, intravascular haemolysis, and hepatorenal failure.
- ✓ Chronic inhalation of chromate dust causes conjunctivitis, lacrimation, ulceration of nasal septum, and respiratory cancer.
- ✓ Treatment of acute dichromate poisoning involves administration of BAL and large doses of ascorbic acid (IV). Stomach wash can also be done with a solution of ascorbic acid. There are reports of favourable results with N-acetylcysteine.

ALKALIS

Alkalis

Alkalis common in poisoning includes:

- ✓ Ammonium
- ✓ sodium and potassium hydroxide
- ✓ Sodium and potassium carbonate
- ✓ Sodium hypochlorite is also increasingly being implicated.

Physical appearance

- ✓ Most of these occurs as White powder or Colorless solution
- ✓ Ammonia gas – colorless with pungent choking odor.

Fatal dose

- ✓ 10-15gm for most alkalis

Uses

- ✓ Ammonium hydroxide- Paint, oil, dirt remover, refrigerant
- ✓ Potassium carbonate-Household cleaning agent.
- ✓ Sodium hypochlorite-Household bleach
- ✓ Sodium carbonate-Household cleaning agent, detergent
- ✓ Potassium hydroxide-Drain cleaner.
- ✓ Sodium hydroxide-Drain cleaner ,oven cleaner
- ✓ Ammonium gas-Smelling gas

Mode of action

- ✓ Locally it cause tissue injury by liquefactive necrosis, it is the process that involves saponification of fats and solubilization of proteins.
- ✓ Cell death occurs from emulsification and disruption of cellular membrane
- ✓ The most severely injured tissues are those that first contact the alkalis , which is squamous epithelial cells of oropharynx, hypo pharynx, and esophagus
- ✓ Esophagus will be more severely affected than stomach, but ingestion of large quantities severely affects both.
- ✓ Tissue edema occurs immediately , may persist for 48hrs , and may eventually progress sufficiently to create airway obstruction .
- ✓ Overtime , if the injury was severe enough, granulation tissue starts to replace necrotic tissue.

- ✓ Over the 2-4 weeks, scar tissue remodels and may thicken and contract enough to form strictures
- ✓ The most severe burns also may be associated with oesophageal perforation.

Mechanism of action

Hydroxide ions saponify FAs of the cell membrane

↓

Protein disruption,solubilisation

↓

Liquefaction necrosis,further penetration of alkali

Clinical features

- ✓ Corrosion of GI mucosa with greyish pseudo membrane formation.
- ✓ Esophagus will be severely affected resulting in
  - Dysphagia
  - Vomiting
  - Drooling
  - Hematemesis.

- ✓ Abdominal pain, diarrhea
- ✓ Ophthalmologic problems
- ✓ Ammonia ingestion- Respiratory symptom (inhalation of fumes while swallowing)

Diagnosis

1. In stomach content

- White , solid , lumps , flakes or granules
- Turns litmus paper blue
- Becomes warm on addition of water
- Sharp penetrating odor in case of ammonia

2. Platinum wire flame test - Touch platinum wire to unknown substance and then place in the flame, sodium gives an yellow flame and potassium gives an purple flames

3. Fume test for ammonia - Place an open bottle of concentrate HCL near a sample, copious white fumes of ammonium chloride will spread out from a sample if ammonia is present

Treatment

- Respiratory distress (especially in case of ammonia) may require endotracheal intubation.
- Oxygen must be administered as required.
- Fist aid – diluents such as Milk or water may be given for alkali ingestion.(Adult-one or more glass)
- Withhold all oral feeds initially.
- Assess the electrolyte balance.

- Patient with 2nd or 3rd degree esophageal burn , intraluminal stent may be useful because perforation which require surgical repair
- Antibiotic should be administered Only if there are sign of perforation or secondary infection.
- An oesophagogram should be done for 3 weeks to evaluate the formation of strictures , patient who have suffered from strictures formation require long term endoscopic follow up
- Injuries to the eyes and skin should be irrigated copiously with water or saline Ophthalmologic is mandatory. Topical antibiotics and steroids may help.

Contra-Indications

- Emesis and Gastric lavage -it can re-expose the GI tract to the caustic
- Attempts to neutralize the alkalis are contraindicated because severe exothermic reaction may results , it will produce heat that may worsen tissue damage
- Catharsis
- Activated charcoal - it may infiltrate burned tissue and interfere with endoscopic evaluation

OTHER CAUSTICS

1. Potassium permanganate

Fatal dose: 5 to 10 grams

Mode of action: It is an irritant and in highly concentrated form It acts as a corrosive. It also exhibits systemic toxicity.

Clinical features: Burning , abdominal pain , vomiting , inflammatory edema in lungs leads to dyspnoea, severe thirst

Complications: Shock, hepatic and renal failure , pancreatitis and methaemoglobinaemia

Diagnosis: Stains will be decolourised by oxalic acid or Hydrogen peroxide

Treatment

- Dilution with water, if corrosion is not severe it can be done with dilute hydrogen peroxide.
- Methaemoglobinaemia can be treated with methylene blue, 1 to 2mg/kg/dose IV over 5mins as needed
- Chelation with EDTA has been used in patient with manganese intoxication
- Supportive measures

2. Hydrogen peroxide

Fatal dose: Not clearly known, fatalities with industrial grade solution

Mode of action: It decomposes to water and oxygen. When used in closed space or under pressure , liberated oxygen cannot escape , systemic oxygen embolization and subcutaneous emphysema can occur.

Clinical features: Gastric distention, GI irritation, perforation, vomiting, gingival ulceration, convulsion , metabolic acidosis, burns and gangrene

Treatment

- Airway management , endotracheal intubation, oxygen administration and mechanical ventilation.
- Administer water immediately to dilute the peroxide , spontaneous vomiting is common.
- Laparotomy may be required if there is evidence of air in the GI tract.
- Hyperbaric oxygen therapy may be help to alleviate life threatening gas embolism
- Supportive therapy with particular reference to control of metabolic acidosis and convulsion.

3. Iodine

Fatal dose: 2 to 5gm of free iodine

1 to 2 ounces of strong iodine tincture

Mode of action: It is an intense irritant ,systemic toxicity is due to combination of free iodine with serum sodium bicarbonate leading to metabolic acidosis

Clinical features: Conjunctivitis, cough, burning, salivation, metallic taste, vomiting, diarrhoea, ocular burn, edema, tachycardia, hypotension, metabolic acidosis, renal failure, convulsion, hypersensitivity reactions, thyrotoxicosis.

Treatment

- Treatment is primarily supportive.
- There is no specific antidote.
- Decontamination of exposed area (skin or eyes) with water
- Sodium bicarbonate IV for metabolic acidosis
- Intake of sodium chloride which promotes the excretion of iodides

CASE PRESENTATION

- An 18 year old, unmarried male gold smith was brought to psychiatry out patient of a general hospital by his relatives. The patient had strong ideas of suicide by hanging, drowning and consumption of large amount of sedative tablet.
- On further enquiry he was found to have symptoms of sadness, easy fatigability, lack of concentration, sleeplessness, lack of appetite, loss of interest in work and daily activities of three week duration
- Physical examination was unremarkable, Thus the patient was diagnosed of Major depressive Disorder.
- Considering the suicidal intentions in the patient, emergency ECT was planned. While making arrangements for administration of ECT he was made to wait at the reception area with his relatives.
- After half an hour he was taken to the ECT table when they noticed a pungent odor emanating from the patient. Hence, the administration of ECT was withheld and observation was continued.
- Patient became increasingly restless and was clutching his abdomen

- His BP- 130/70mmHg
- PR-120bpm
- Pupils were midsize and non reactive to light
- At this stage a tentative diagnosis of acid poisoning was made
- He was given albumin of 2 eggs and 3 bananas. Patient was shifted to emergency ward.
- As the patient developed difficulty in breathing and became semiconscious, he was put on assisted breathing
- The patients condition detoriated and he died after 2 hours
- An autopsy was performed 6 hours after the death. The autopsy confirmed death due to sulfuric acid poisoning