



***KEROSENE &
CORROSIVES -
AUTOPSY FEATURES***

Asela Mendis

KEROSENE

Kerosene is a liquid mixture of chemicals produced from the distillation of crude oil.

- *Kerosene is not particularly poisonous.*
- *Frequent skin exposure may lead to skin damage (dermatitis).*

Toxicity occurs if kerosene is inhaled while being ingested.

Irritating to eyes and skin.

Aspiration may cause serious lung injury.

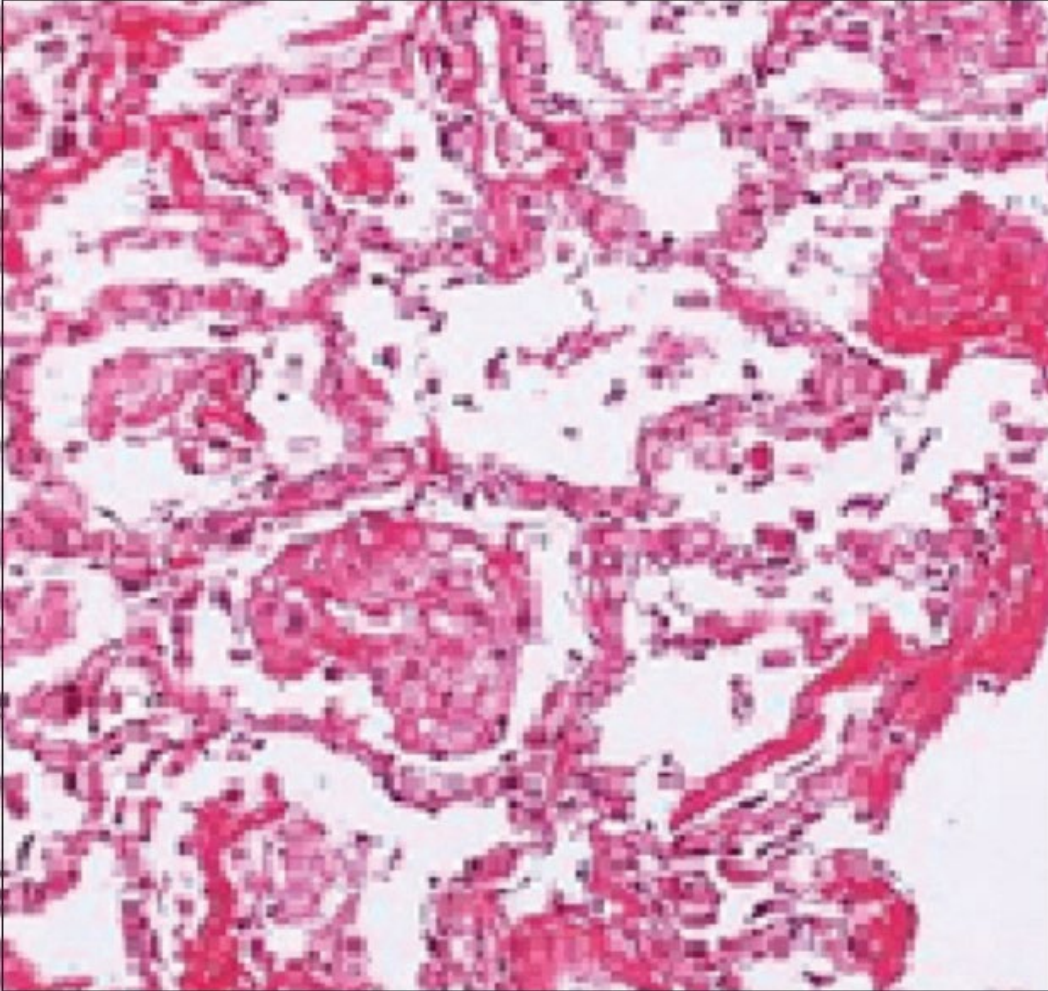
Hydrocarbon poisoning may result from

- ***Ingestion or inhalation.***
- ***Ingestion, most common among children < 5 yr, can result in aspiration pneumonitis.***
- ***Inhalation, most common among adolescents, can result in ventricular fibrillation, usually without warning symptoms.***
- ***Diagnosis of pneumonitis is by clinical evaluation, chest x-ray etc.***
- ***Gastric emptying is contraindicated because aspiration is a risk. Treatment is supportive.***

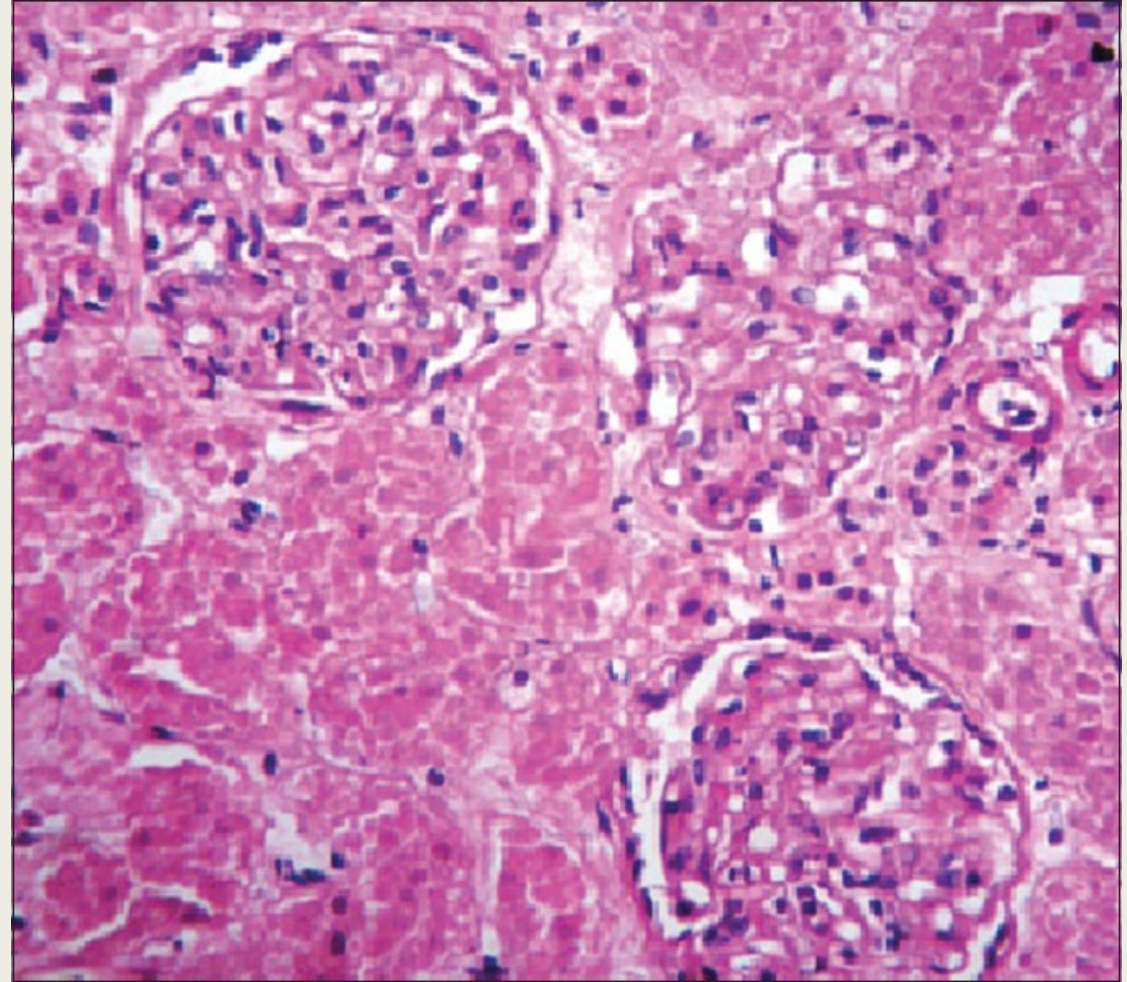
- *If ingested in large amounts, may be absorbed systemically and cause CNS or hepatic toxicity, which is more likely with halogenated hydrocarbons (eg, carbon tetrachloride, trichloroethylene).*

- *After ingestion of even a very small amount of liquid hydrocarbon, patients initially cough, choke, and may vomit.*
- *Young children may have cyanosis, hold their breath, and cough persistently.*
- *Older children and adults may report burning in the stomach.*

- ***Aspiration pneumonitis causes hypoxia and respiratory distress.***



Diffuse alveolar damage with hyaline membranes, Hematoxylin and Eosin ×20



Extensive tubular necrosis affecting predominantly proximal tubules but intact glomeruli. Hematoxylin and Eosin ×20

Immediate Signs or Symptoms of Acute Exposure

- ***Inhalation: May cause headache, dizziness, drowsiness, incoordination and euphoria. Aspiration into the lungs causes pneumonitis with choking, cough, wheeze, breathlessness, cyanosis and fever.***

- ***Ingestion: Often no symptoms occur but there may be nausea, vomiting and occasionally diarrhoea.***

- ***Ocular: This product is expected to be pH neutral but may be irritating to the eyes causing an immediate stinging and burning sensation with lacrimation.***

- ***Dermal: Irritant. Drying and cracking due to defatting action. There may be transient pain with erythema, blistering and superficial burns.***

Histopathology

- ***Bronchospasm***
- ***Interstitial inflammation***
- ***Atelectasis***
- ***Emphyema***
- ***Hyperaemia***
- ***PMN infiltration***
- ***Bronchial and bronchiolar necrosis***
- ***Vascular thrombosis***
- ***Intra-alveolar haemorrhage and oedema***

Corrosives



Corrosives are materials that can attack and chemically destroy exposed body tissues. Corrosives can also damage or even destroy metal. They begin to cause damage as soon as they touch the skin, eyes, respiratory tract, digestive tract, or the metal.

- ***Strong acids – HCl, HNO₃, H₂SO₄ etc..***
- ***Alkalis***
- ***Metallic salts - Zn Chloride, Cu sulphate, K cyanide etc.***

Most corrosives are either acids or bases. Common acids include hydrochloric acid, sulfuric acid, nitric acid, chromic acid, acetic acid and hydrofluoric acid.

Common bases are ammonium hydroxide, potassium hydroxide (caustic potash) and sodium hydroxide (caustic soda).

Mechanisms of action

- (1) Oxidation: The protein denaturation - sodium hypochlorite, potassium permanganate, and chromic acid
- (2) Reduction: hydrochloric acid, nitric acid and alkyl mercuric compounds.
- (3) Corrosion: It causes protein denaturation on contact. - phenols, sodium hypochlorite, and white phosphorous.
- (4) Protoplasmic poisons: formic and acetic acids, while inhibitors include oxalic and hydrofluoric acids.
- (5) Vesicants: mustard gas, dimethyl sulfoxide (DMSO), and Lewisite.
- (6) Desiccants: Sulphuric and Hydrochloric acids

The severity of a burn injury is determined by:

- (a) Concentration,
- (b) Quantity of burning agent,
- (c) Duration of skin contact,
- (d) Penetration and,
- (e) Mechanism of action.

■ ***Corrosive substance ingestion in the acute phase***

Result in injuries of the larynx

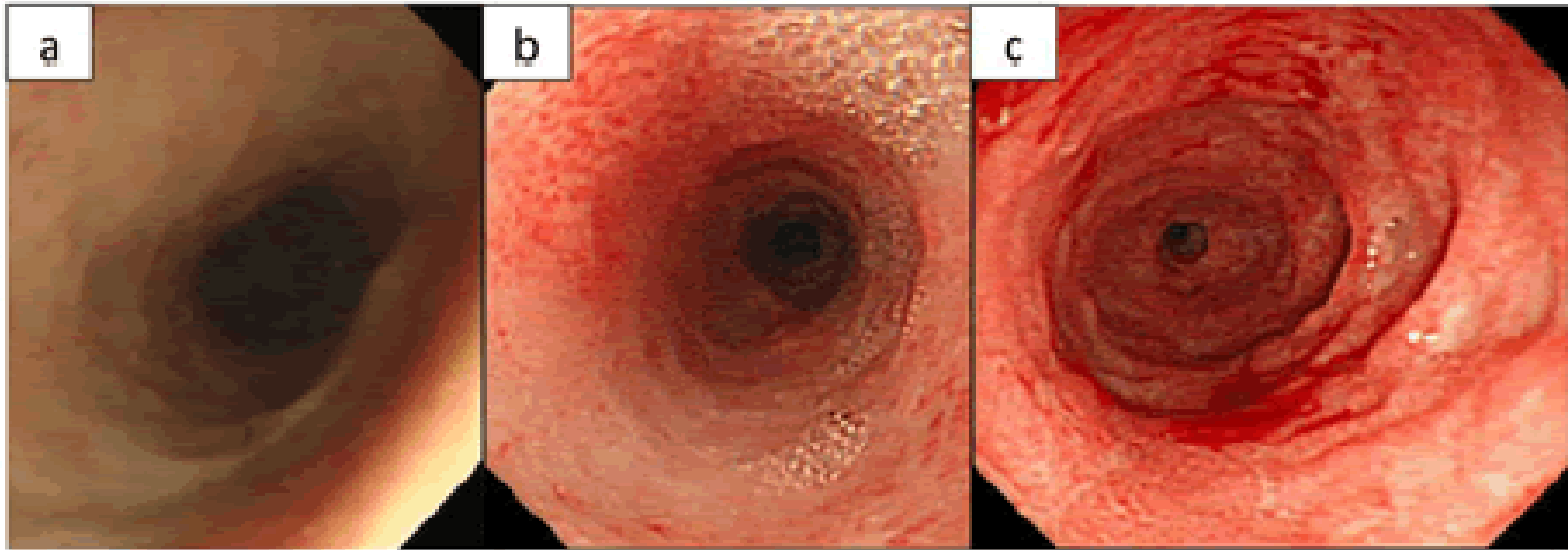
Cause laryngospasm associated with dyspnoea, tachypnoea,

Dysphonia and aphonia.

Aspiration of the corrosive substance may cause endotracheal or bronchial necrosis with mediastinitis, often leading to fatal outcome

- ***Strictures and stenosis of the oesophagus***
- ***Stenosis of gastric antrum and pylorus***





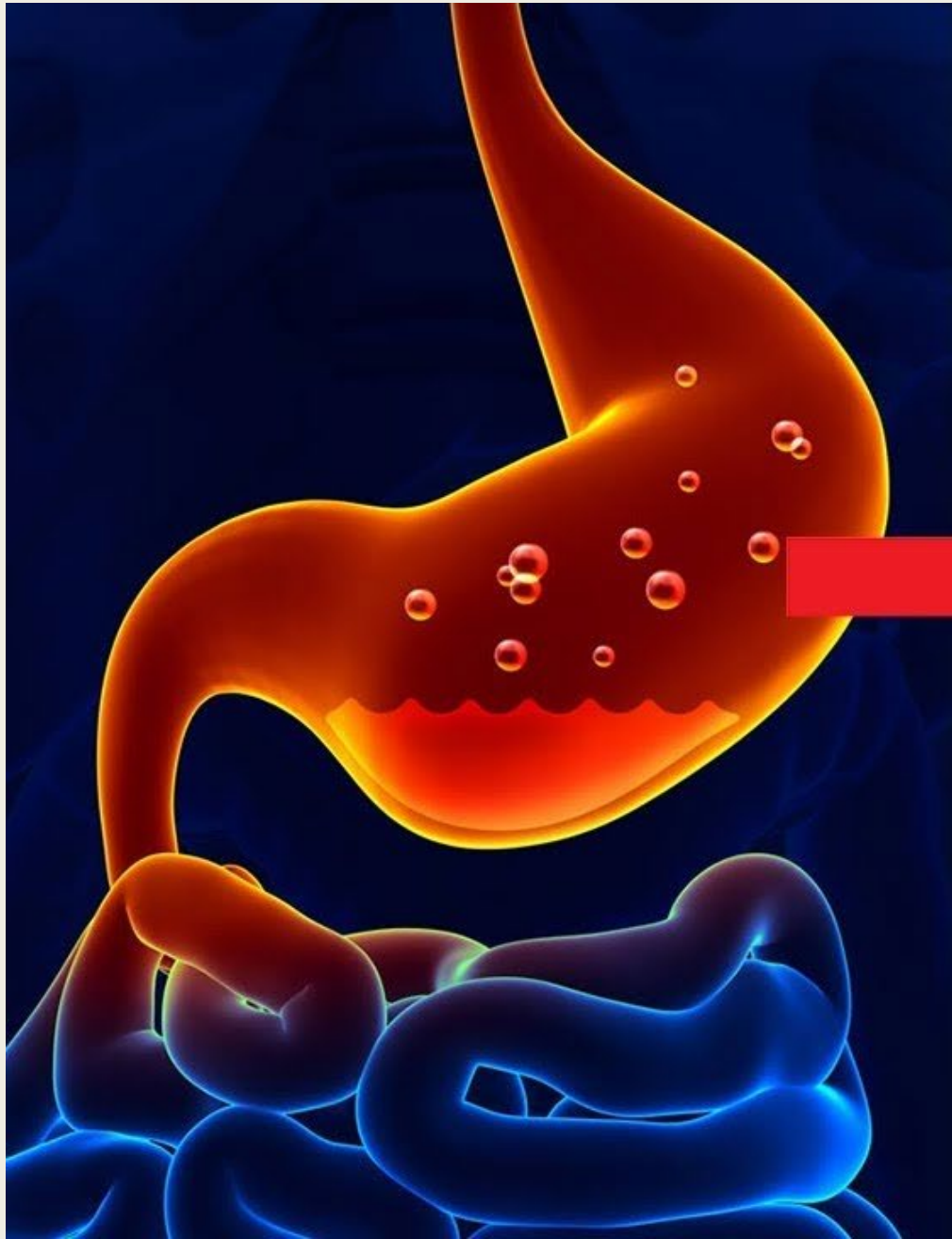
a. On day 11, the EGD showed generalized ulceration of the esophagus and multiple gastric ulcers

b. On day 32, the EGD showed advanced esophageal stenosis after corrosive esophagitis

c. On day 37, the EGD showed advanced stenosis where passage was difficult even with a thin transnasal endoscope 41 cm from the incisor

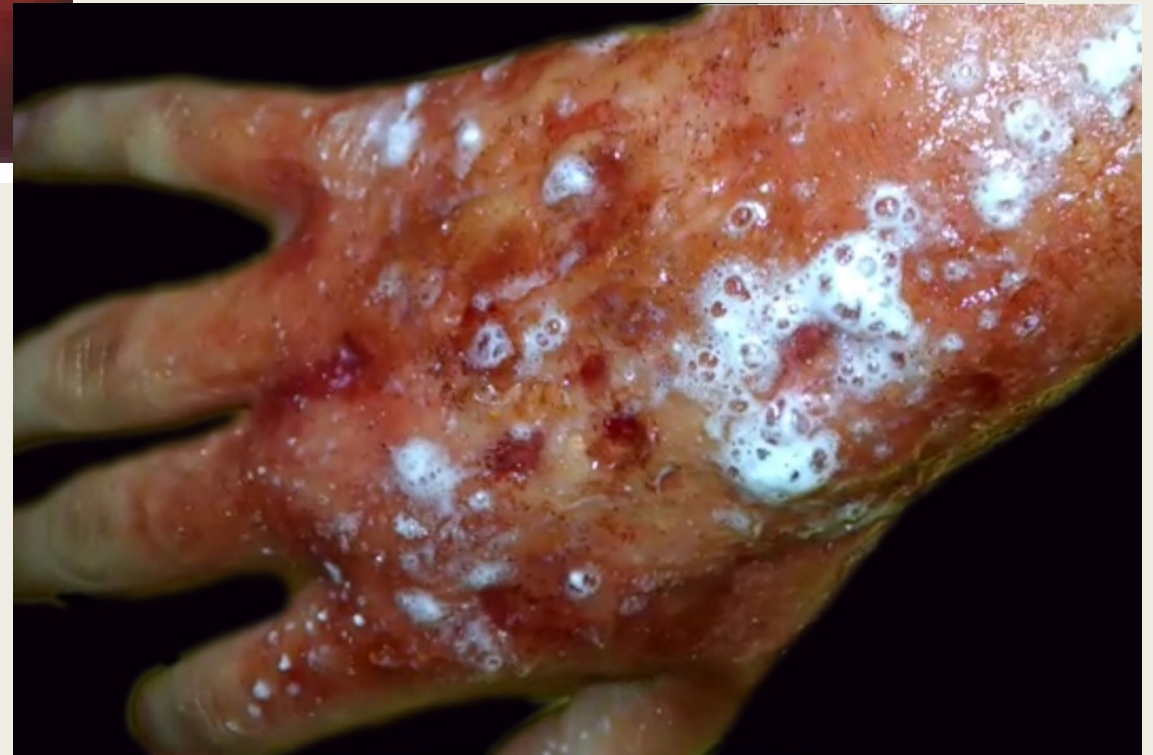
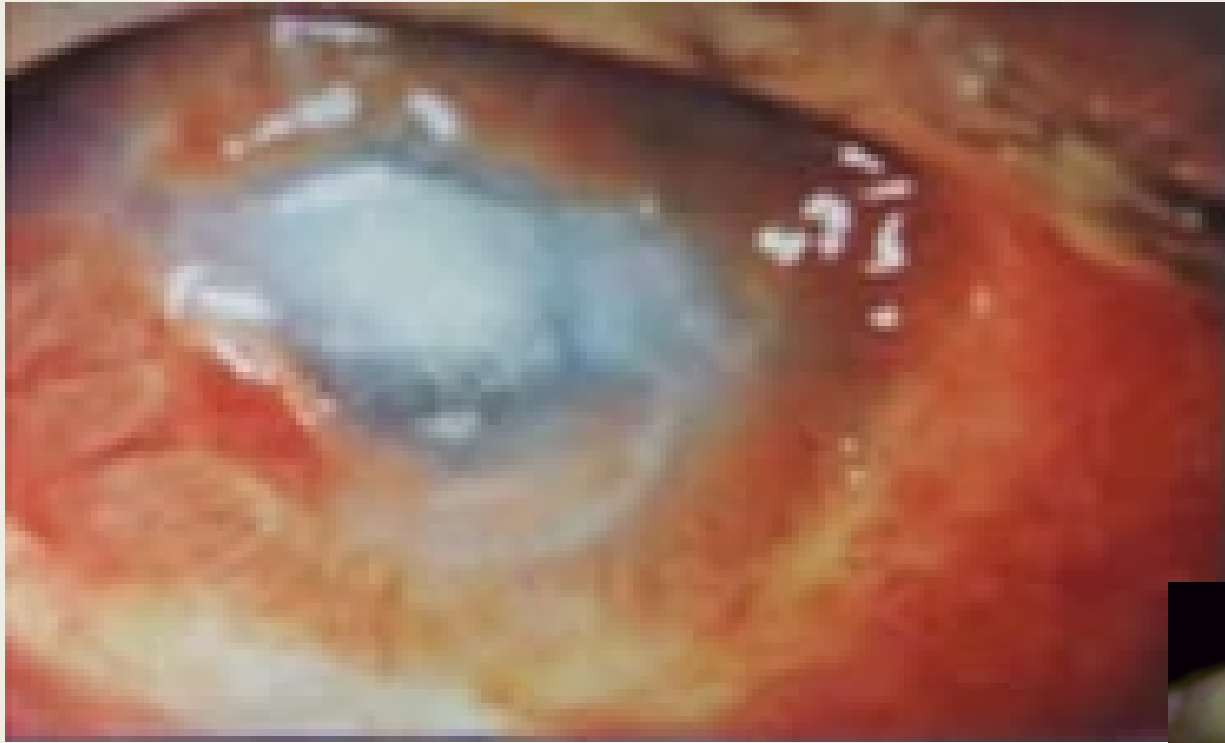
Figure 2: Esophagogastroduodenoscopy.

- *Corrosives can burn and destroy body tissues on contact.*
- *Some corrosives are toxic and can cause other health problems.*









Acids

- Acids are proton donors. They release hydrogen ions and reduce pH from 7 down to values as low as 0.
- Acids with a pH less than 2 can produce coagulation necrosis on contact with the skin

Akali

- Bases are proton acceptors. They will strip hydrogen ions from protonated amine groups and carboxylic groups.
- Alkalis with a pH greater than 11.5 produce severe tissue injury through liquefaction necrosis

Alkali burns

- *More severe burns than acids.*
- *Destroy cell structure - extend deep in to tissues.*

The mechanism by which strong alkali injuries are caused involves three factors:

1. Saponification of fat

An exothermic reaction producing a significant amount of heat, which causes severe tissue damage. Destruction of fat allows an increase in water penetration of the alkali into the burn eschar, destroying the natural water barrier that lipids provide.

2. Extraction of considerable water from cells causes damage

Due to the hygroscopic nature of alkalis, causing extensive cell death and damage to tissues.

3. Alkalis dissolve proteins of the tissues to form alkaline proteinates, which are soluble and contain hydroxyl ions (OH). These ions cause further chemical reaction which initiate deeper injury of the tissue (liquefaction necrosis).

Strong alkali

- Lime, sodium hydroxide and potassium hydroxide
- They are capable of deep penetration, and tissue
- destruction continues long after the initial exposure
- Systemic effects can also occur due to substantial
- absorption of the chemical.

Consequences of caustic injury

- ***Caustic injury may cause the following:***

Occurs within seconds of exposure to caustic agent

Ulceration and perforation: Occurs within 24-72 hours of exposure

Fibrosis: Occurs within 14-21 days of exposure

Stricture: Occurs after weeks to years of exposure

Carcinoma formation: Occurs after decades of alkali exposure.



Fig. 1 – Patient who tried to remove a tattoo with a chemical product.



Fig. 3 - Formic acid burn. (a and b) Early copious water irrigation was established for an hour. Both cornea were affected and were immediately explored by an ophthalmologist. (c) After 2 weeks treatment with topical silver sulphadiazine cream, the frontal, nasal and periorbital areas, where the eyelids are specially affected, have not still epithelized. (d) Although these areas were grafted, and a temporal tarsorrhaphy was kept in place for 2 weeks, the patient developed an ectropion in the four eyelids.

Sulphuric acid



Fig. 5 - Sulphuric acid burn. Brown discoloration is typical.

Hydrochloric acid

- In contact with the skin, hydrochloric acid denatures the proteins into chloride salts.
- Pulmonary damage (upper airway oedema, pulmonary inflammation) due to fumes

Hydrofluoric acid

- Hydrofluoric acid causes severe burns and systemic effects,
 - Superficial burns.
 - Liquefactive necrosis of the soft tissue.
 - Systemic hypocalcaemia and
 - Hypomagnesaemia
 - Ventricular fibrillation



HNO_3



HCl



HF



Sulphuric

Drip marks



Important medico-legal issues

- Cause of death
- Circumstances
- Category of hurt
- Causative Agent
- Reconstruction of the event

Thank You