

# Caustics

# What are Caustics Chemicals?

- Chemicals that cause tissue injury on contact with mucosal and epithelial surfaces.
- Include:
  - Alkaline
  - Acidic

# Alkalis

- Alkalis accept protons, resulting in the formation of conjugate acids and free hydroxide ions.
- Turn **Pink Litmus Blues**
- Lye is an example of an alkali and refers to both sodium hydroxide ( $\text{NaOH}$ ) and potassium hydroxide ( $\text{KOH}$ ).
- Ammonia ( $\text{NH}_3$ ) is another common alkaline corrosive.



# Acids

- Acids are proton donors, as they dissociate into conjugate bases and free hydrogen ions in solution.

Acidic caustics include

- hydrochloric acid (HCl) found in **Rust Removal or Toilet bowl cleaner**
- sulfuric acid ( $\text{H}_2\text{SO}_4$ ) acidic found in drain cleaners



# Perspective

- The severity of caustic agents typically increases with a pH less than 3 or greater than 11.
- On the contrary, hydrofluoric acid (HF) is a relatively weak acid that can cause necrotizing injury and life-threatening systemic toxicity.



HF burns, not evident until a day after



# Other Caustics:



Other chemicals that have caustic properties

Phenol

Formaldehyde

Iodine

concentrated hydrogen peroxide.

# Common Caustics Products



- **Liquid drain cleaners** have high concentrations of alkali (30% KOH) or acid (93% H<sub>2</sub>SO<sub>4</sub>).
- **Industrial and farms (dairy pipeline) cleaners** containing liquid NaOH and KOH (in concentrations of 8–25%)
- **Swimming pool cleaners** also contain caustics in high concentrations.

# Household Cleaning Products That Contain

APPLICATION	PRODUCT (MANUFACTURER) CHEMICAL
Drain cleaner—liquid	Heavy Duty Liquid Drain Opener (Share), $\text{H}_2\text{SO}_4$ 93% Drain Out Extra (Iron Out), KOH 30% Liquid-Plumr (Clorox), NaOH 0.5–2%, $\text{NaOCl}$ 5–10% Maximum Strength Drain Opener (Enforcer), KOH 1–10%, $\text{NaOCl}$ < 5% Drain Care Professional Strength Drain Opener, NaOH 5–15%
Drain cleaner—crystals	Heavy Duty Crystal Drain Opener (Roebic), NaOH 100% Crystal Drain Opener (Rohyme), NaOH 74% Crystal Drain Out (Iron Out), NaOH 30–60% Drano Pipe Cleaner (Johnson), NaOH 54%



# Household Cleaning Products That Contain Caustic Chemicals

Oven cleaner	Easy Off Heavy Duty Oven Cleaner (Reckitt), NaOH 4–6%
Rust remover	Rust Remover/Carpet Care (Johnson Wax Prof), HCl 10% Rust Stain Remover (Whink), HF 2.5–3% Rust Stripper (Certified), NaOH 50–75% Naval Jelly Rust Remover (Loctite), Phosphoric acid 25–30%
Toilet bowl cleaner	Instant Power Toilet Bowl Cleaner (Scotch), HCl 26% Bowl and Porcelain Cleaner (Cleanline), HCl 0.10% Bowl/Tile/Porcelain Cleaner (Share), Phosphoric acid 15–25% Husky 303 Toilet Bowl Cleaner, HCl 23% Misty Bolex Bowl Cleaner, HCl 26%
Swimming pool cleaner	Muriatic Acid, Aqua Chem (Recreational Water), HCl 31%

# Accidental Versus Intentional Ingestion



- Intentional ingestions have a greater degree of oropharyngeal sparing due to rapid swallowing but have a higher likelihood of serious injury.
- More than half of suicidal patients who ingest caustic agents have a history of psychiatric illness

# Solid Versus Liquid Corrosives:



- Crystals and solid particles have prolonged tissue adherence, causing more severe burns.
- Usually limited by immediate oral pain, causing them to be spit out sooner than a liquid agent.
- The ingestion of granular automatic dishwashing detergents is associated with devastating injuries

# Solid Versus Liquid Ingestions



- Crystal drain cleaners have lye concentration as high as 74% NaOH and may cause proximal esophageal injury.

# Solid Versus Liquid Ingestions



- Liquid household bleach typically contains dilute (5.25%) sodium hypochlorite ( $\text{NaHClO}$ ), and ingestion rarely causes injury.
- **Industrial-strength bleach** may contain significantly higher concentrations of  $\text{NaHClO}$ ,
- Toilet bowl cleaners contain hydrochloric acid as high as 26%
- Anticorrosive cleaners, such as 31% muriatic acid ( $\text{HCl}$ ), are sold in gallon containers for home use and as swimming pool cleaners.

# Factors that influence the extent of injury

- Type of agent (Acid/Alkali)
- Solid/Liquid
- Concentration of solution
- Volume
- Viscosity
- Duration of contact
- pH
- Presence or absence of food in the stomach.

# Mechanism of Injury: Acids



- Acidic compounds desiccate epithelial cells and cause **coagulation necrosis**
- An **eschar is formed** thereby limiting further penetration.
- Acids tend to have a strong odor and cause immediate pain on contact, the quantity ingested is usually small
- Because of **resistance of squamous epithelium** to coagulation necrosis, acids are thought to be less likely to cause esophageal and pharyngeal injury
- Acids can be absorbed systemically, causing metabolic acidosis, as well as damage to the spleen, liver, biliary tract, pancreas, and kidneys.

# Mechanism of Injury: Alkalis



- Alkaline contact causes **liquefaction necrosis**, fat saponification, and protein disruption, allowing further penetration of the alkali
- The depth of the necrosis depends on the concentration of the alkali
- A concentration of 30% NaOH in contact with tissue for 1 second results in a full-thickness burn.
- Alkalis are colorless, odorless, and unlike acids, do not cause immediate pain on contact.

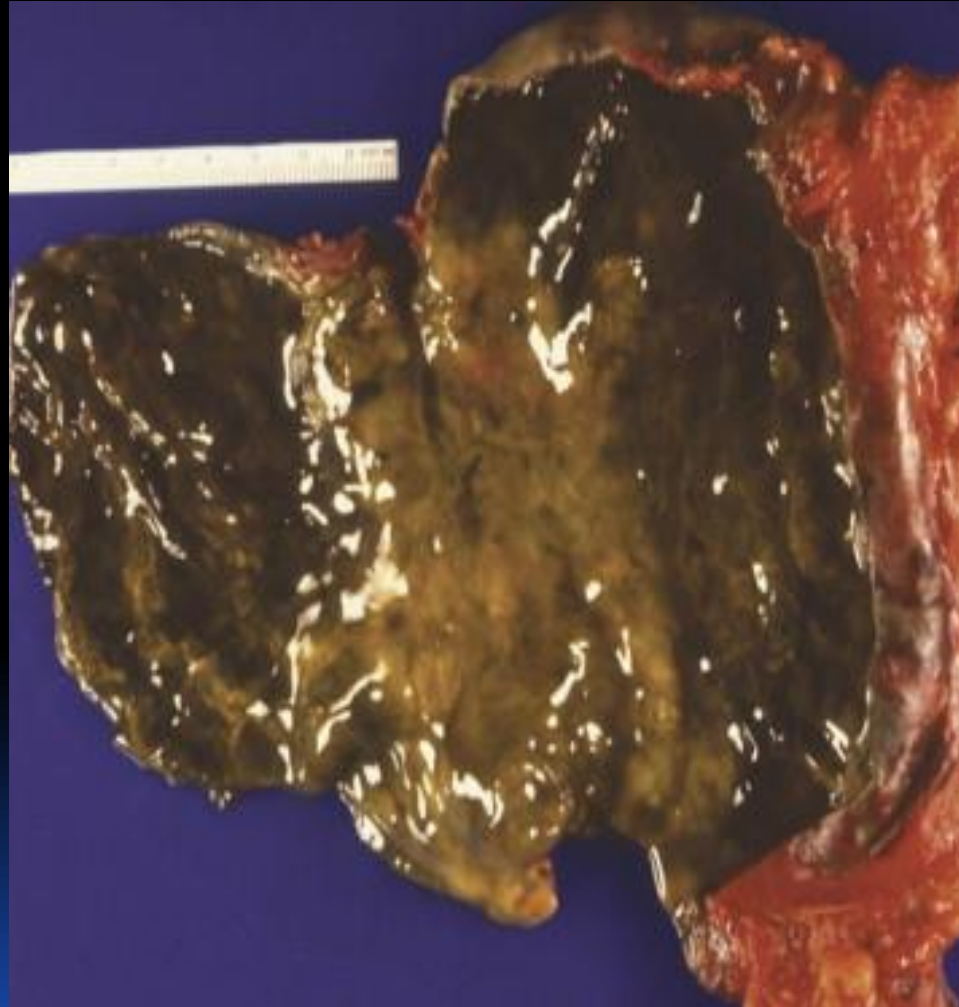


# Mechanism of Injury: Alkalis



- Alkaline ingestions typically involve the squamous epithelial cells of the oropharynx, hypopharynx, and esophagus.
- The narrow portions of the esophagus, where pooling of secretions can occur, are also commonly involved.
- Alkalis may also cause gastric necrosis and perforation.
- The esophagus can also be injured. Burns below the pylorus carry a worse prognosis than burns above the pylorus (50% vs. 9% mortality).

# Gastric mucosa after ingestion of 35% potassium hydroxide



# Gastric serosa after ingestion of 35% Sulfuric acid



# Esophagus after ingestion of 35% potassium hydroxide.



# Stages of Caustic Injury



- Classically, the damage occurs in following steps.
  - Necrosis
  - invasion by bacteria and polymorphonuclear leukocytes.
  - Vascular thrombosis follows, increasing the damage.
    - Over the next 2 to 5 days, superficial layers of tissue begin to slough.
  - Healing: The tensile strength of the healing tissue may be quite low for up to 3 weeks increasing chance of delayed perforation
  - Between 1 week and several months, granulation tissue forms, collagen is deposited, and re-epithelization
- Esophageal stricture may form over a period of weeks to years from contraction of the scar.

# Degree of Burn:



- Caustic injury is graded into 3 degrees/Grades based on endoscopy
  - First: edema and hyperemia
  - Second: superficial ulcers, white membrane, exudates, friability and hemorrhage
    - Grade 2A: non circumferential
    - Grade 2B: Circumferential
  - Third: Transmural involvement with deep injury, necrotic mucosa, or frank perforation of the stomach or esophagus.
- The initial grade of burn on esophagoscopy correlates with the risk of stricture formation.

# Chances of stricture Formation



- Grade 2A Burns: 15 to 30% develop strictures
- Grade 2B: upto 75% develop strictures
- Grade 3: 90% result in stricture
  
- Whether heat from the exothermic reaction increases the injury has never been quantified, but it has led to concerns regarding initial dilution or gastric lavage.

# CLINICAL FEATURES



- Airway edema and esophageal/gastric perforation are most emergent issues
- Laryngeal edema occurs over a matter of minutes to hours.
- Systemic toxicity; hypovolemic shock; and hemodynamic instability with hypotension, tachycardia, fever, and acidosis are ominous findings.
- Small ingestion of potent substances can be as serious as larger ingestions.
- More than 40% of patients reporting to have “only taken a lick” have esophageal burns.



# CLINICAL FEATURES



- Patients present with oral pain (41%), abd pain (34%), vomiting (19%), and drooling (19%).
- Some have wheezing and coughing. Others present with stridor and dysphonia.
- Chest pain is common.
- Visible burns to the face, lips & oral cavity. Burns can occur from spills or contamination after vomiting
- Peritoneal signs suggest hollow viscous perforation or extension of the burn to adjoining visceral areas.

Lip burn after exposure to 35%  
potassium hydroxide.



# CLINICAL FEATURES



- Tracheal necrosis is one of the most frequent causes of death after caustic ingestion.
- Oropharyngeal burns alone do not appear predictive of more distal injury

**Prolonged drooling and dysphagia predicted significant lesions with 100% sensitivity and 90% specificity.**

**Vomiting and stridor may also be more predictive of burn injury.**

# CLINICAL FEATURES



- Dysphagia usually subsides in 3 to 4 days.
- Patients with significant esophageal burns, particularly those that are circumferential, may develop esophageal stricture
- 80% of strictures become apparent in 2 to 8 weeks.
- Symptoms include dysphagia and food impaction..

# CLINICAL FEATURES



- Patients have an increase in esophageal cancer (1000-fold to 3000-fold increases) that develops 40 to 50 years after the caustic ingestion.
- A recent long-term study showed that 1.8% of patients who ingested caustic soda developed esophageal cancer.
- Nearly 3% of esophageal cancer patients have a history of caustic ingestion

# CLINICAL FEATURES



- Significant acid ingestions may be devastating and result in a higher mortality rate than alkali ingestions.
- The fulminant course of some acid ingestions may be due to systemic absorption of the acid, resulting in metabolic acidosis (which may also be the result of extensive tissue necrosis), hemolysis, and renal failure.

# DIAGNOSTIC STRATEGIES

- Patients with chest and abdominal pain should have a chest radiograph and decubitus or upright abdominal studies to identify peritoneal and mediastinal air, denoting perforation or pleural effusion.
- Any suggestion of abdominal involvement should prompt abdominal CT or US.
- ABG to monitor systemic metabolic acidosis.
- In cases of intentional overdose, co-ingestants should be considered.

# DIAGNOSTIC STRATEGIES

- Patients with S&S (vomiting, drooling, stridor, or dyspnea) should undergo endoscopy within 12 to 24 hours to define the extent of burn
- Endoscopy is contraindicated in patients with possible or known perforation
- The finding of frank necrosis or obliteration of the lumen should result in termination of the procedure
- Endoscopy performed too early may miss the extent or depth of tissue injury.
- Hypoxia warrant immediate bronchoscopy



# MANAGEMENT



- In alert patients who are not vomiting and can tolerate liquids, small volumes (1–2 cups) of water or milk can be considered within the first few minutes after ingestion
- Forcing fluids is never indicated
- Do not neutralize the ingested corrosive with weak acids or alkalis due to thermal reactions and worsening injury

# MANAGEMENT



- Early and continuous respiratory and hemodynamic monitoring is essential.
- Contaminated clothing should be treated as hazardous waste and disposed of using proper precautions
- Activated charcoal, and performing gastric lavage are not indicated.
- Careful naso-gastric aspiration may be useful in the setting of significant acid ingestions presenting immediately after ingestion

# MANAGEMENT



- Early endotracheal intubation is warranted with airway compromise suggested by hoarseness, throat pain, drooling, or edema.
- Intubation should be undertaken early before edema and secretions threaten the airway and make intubation difficult
  - No Blind nasotracheal intubation
  - When oral intubation is anticipated to be difficult awake fiberoptic intubation or primary surgical cricothyrotomy may be necessary
- IV access and vigorous fluid resuscitation

# MANAGEMENT



- Surgical exploration is indicated for free air, peritonitis, increasing and severe chest and abdominal pain, and hypotension.
- Corticosteroid therapy remains controversial.
- Prophylactic antibiotics may potentially mask evidence of impending perforation

# SPECIAL CASES



- **Ocular alkali** exposures are true ophthalmologic emergencies.
- Immediate and aggressive lavage with at least 2 L of normal saline per eye is indicated in all cases except for frank perforation
- **Dermal caustic exposures** can also result in significant burn injuries. Clothing removal, copious irrigation, and local wound debridement are the most important initial treatment

# SPECIAL CASES



- **Hydrofluoric acid:** Although a relatively weak acid the dissociated fluoride anions are problematic because of extreme electro-negativity
- Deaths from HF exposure have occurred after ingestion, after skin contact in areas as small as 1% BSA with concentrated HF & inhalation of HF vapor
- Systemic toxicity is characterized by immediate and profound **hypocalcemia and dysrhythmias**
- Cardiac and serum calcium monitoring are warranted in all

# SPECIAL CASES

- **Povidone-iodine** (Betadine): is used as a surgical scrub and is not a caustic agent, but ingestion of tincture of iodine can cause severe gastrointestinal injury and is potentially life-threatening.
- Gastric irrigation with starch or milk in these cases may convert iodine to less toxic iodide.
- **Phenol or Formaldehyde** can also cause severe caustic injury to the gastrointestinal tract
- Both phenol and formaldehyde are general protoplasmic poisons and can cause protein denaturation and coagulation necrosis.

# SPECIAL CASES

- Systemic symptoms, including dysrhythmias, hypotension, seizures, and coma, may result from phenol ingestion.
- Acidosis may be prominent after formaldehyde ingestion due to its metabolism to formic acid
- Phenol is well absorbed through the skin, dermal exposure may result in systemic toxicity
- Dermal decontamination of phenol exposures with LMW polyethylene glycol has been suggested but water may prove just as useful



# SPECIAL CASES



## Concentrated hydrogen peroxide ( $H_2O_2$ )

Ingestion may cause gastrointestinal burn injury and the formation of gas emboli

- Radiographic evaluation for the presence of gas in the chest or abdominal cavities, including the portal system, should be performed in symptomatic patients
- Hyperbaric oxygen has been used successfully to treat gas emboli from  $H_2O_2$  ingestion.

# SPECIAL CASES



- **Button batteries** are usually made of a metallic salt (lithium, mercury, nickel, zinc, cadmium, or silver) bathed in NaOH or KOH.
- Obstruction can cause pressure necrosis, caustic injury due to leakage of alkaline medium, or electrical injury.
- Ulceration, perforation, and possible fistula formation occur but are uncommon.
- Heavy-metal toxicity in this setting has not been reported

# SPECIAL CASES



- Evaluation of button battery ingestions requires radiography to assess the position of the foreign body.
- Batteries lodged in the airway or esophagus require expeditious removal.
- Gastric or intestinal batteries can be treated with watchful waiting.
- Follow-up radiographs should be obtained in 1 week if the battery has not passed.