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CAUSTICS



Objectives



Not given):

Enjoy studying this lecture! It
will bring back your chemistry
class memories (:



NOTES EXTRA BOOK IMPORTANT GOLDEN NOTES

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Caustics

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Perspective:



Caustic Chemicals

7

pH

Basically, when a substance donates H ions like an acid comes in contact with a base which donates OH ions they produce water and neutralize each other.

Chemicals that cause tissue injury or burns on contact with mucosal and epithelial surfaces by causing protein denaturation or degeneration.

The severity of caustic agents typically increases with a pH less than 3 or greater than 11.

An acid or a base that has a pH ranges between 3 to 11 may cause a little bit reaction on contact but no significant burns.

≠

On the contrary, hydrofluoric acid (HF) is a relatively weak acid that can cause necrotizing injury and life-threatening systemic toxicity.

It doesn't have a very low pH but it releases fluoride ions (F) which are very necrotizing agents themselves. It can cause systemic manifestations.

→ It includes:

Acids

Alkaline

-Acids are proton donors, as they dissociate into conjugate bases and free hydrogen ions in solution. $\text{pH} < 7$, strong acids $\text{pH} = 2-3$
-Turn Pink Litmus red

Acidic caustics include:

-The **commonest** Hydrochloric acid (HCl) found in Rust Removal or Toilet bowl cleaner
-Sulfuric acid (H_2SO_4) acidic found in drain cleaners

-Alkalis accept protons, resulting in the formation of conjugate acids and free hydroxide ions. $\text{pH} > 7$
-Turn Pink Litmus Blues fast bedside test to determine the type of caustic

Alkalis caustics include:

-Lye which refers to both sodium hydroxide (NaOH) and potassium hydroxide (KOH).
-Ammonia (NH_3) It is an alkaline that doesn't have OH ions to donate but it accepts H ions to form ammonium NH_4 , it is commonly used in fertilizers and agriculture as a common source of nitrogen.

Common Caustics

1

Liquid drain cleaners

Have high concentrations of alkali (30% KOH) or acid (93% H_2SO_4).

2

Industrial and farms (dairy pipeline) cleaners

Containing liquid NaOH and KOH (in concentrations of 8-25%)

they are stronger acids than those used in houses

3

Swimming pool cleaners

Contain caustics in high concentrations.

Other chemicals with caustic properties

Phenol

Formaldehyde

Iodine

concentrated hydrogen peroxide.



Caustics



Household Cleaning Products That Contain Caustic Chemicals

| Application | Product | Manufacturer | Chemical formula |
|--------------------------|--|---|---------------------------|
| Drain Cleaner (Liquid) | Heavy duty liquid drain opener | Share | H_2SO_4 93% |
| | Drain out extra | Iron out | KOH 30% |
| | Liquid-plumr | Colorx <small>the commonest caustic</small> | NaOH 0.5-2%, NaOCl 5-10%. |
| | Maximum strength drain opener | Enforcer | KOH 1-10%, NaOCl < 5%. |
| | Drain care professional strength drain opener. | - | NaOH 5-15%. |
| Drain Cleaner (Crystals) | Heavy duty crystal drain opener | Robic | NaOH 100% |
| | Crystal drain opener | Rohyme | NaOH 70% |
| | Crystal drain out | Iron out | NaOH 30-60% |
| | Drano pipe cleaner | Johnson | NaOH 54% |
| 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 50-70% |
| | Naval jelly rust remover | Loctite | Phosphoric acid 25-30% |
| Toilet Bowl Cleaner | Instant power toilet bowl cleaner | Scotch | HCl 26% |
| | Bowl/ tile / porcelain cleaner | Share | Phosphoric acid 15-25% |
| | Husky 303 toilet bowl cleaner | - | HCl 26% |
| | Misty bolex bowl cleaner | - | HCl 26 % |
| Swimming Pool Cleaner | Muriatic acid, aqua chem | Recreational water | HCl 31% |

Caustics



- ❑ 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

Intentional VS Accidental INGESTION

Suicidal ingestion is more likely to be taken in bigger volumes, and patients know that it is a burning substance so they will swallow it fast causing no or mild oral injuries but more significant deeper burns.
In accidental ingestion when an individual feels the sudden burning sensation he will spit the substance out decreasing the chances of getting deeper burns.

Corrosives

- ❑ Crystals and solid particles have prolonged tissue adherence, causing more severe burns. **Small surface area deeper 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 VS Liquid

- ❑ Crystal drain cleaners have lye concentration as high as 74% NaOH and may cause proximal esophageal injury.
- ❑ Liquid household bleach typically contains dilute (5.25%) sodium hypochlorite (NaHClO), and ingestion rarely causes injury.
- ❑ Industrial-strength bleach may contain significantly higher concentrations of NaHClO, Toilet bowl cleaners contain hydrochloric acid as high as 26%
- ❑ Anticorrosive cleaners, such as 31% muriatic acid (HCl), are sold in gallon containers for home use and as swimming pool cleaners.

Ingestion

-Liquid caustics usually spread out to a bigger surface area causing superficial burns but if it was a strong acid or base then it will cause deeper burns.
-Viscous caustics will stick longer causing deeper burns.



Factors that influence the extent of injury:

- 1-Important in crystals and solids because they keep in contact and going deeper and deeper (duration of contact)
- 2-Food might dilute caustics and limit the amount of burning but also presence of food or other toxins may induce vomiting causing more oral and esophageal burns

Type of agent (Acid/Alkali)

Solid/Liquid

Concentration of solution

Volume

Viscosity

Duration of contact¹

pH

Presence or absence of food in the stomach²

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Caustics

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Mechanism of Injury:

Acids

-Acidic compounds desiccate epithelial cells and cause **coagulation necrosis** when an acid comes in contact with epithelial cells it will coagulate the proteins into a membrane so it will limit the injuries caused by the acid, -then 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 in accidental ingestion it will be spat out immediately

-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. The effect of ingested acid on the oral mucosa and upper esophagus will be less comparing to the stomach because of basicity of saliva, burns of stomach will be augmented by the acidity of gastric secretions

Alkalis

-Alkaline contact causes **liquefaction necrosis**, fat saponification, and protein disruption, allowing further penetration of the alkali higher chances of transmural burns
- 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. Soapy taste

-Alkaline ingestions typically involve the squamous epithelial cells of the oropharynx, hypopharynx, and esophagus. Oral mucosa is alkaline in nature so acid will be neutralized in the mouth
-The narrow portions of the esophagus, where pooling of secretions can occur, are also commonly involved. Upper GI burns will be deeper than in acid burns because of saliva
-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).



Stages of Caustic Injury:

Classically, the damage occurs in following steps:

1 Necrosis

2 Invasion by bacteria and polymorphonuclear leukocytes.

3 Vascular thrombosis follows, increasing the damage along with inflammation.

Over the next 2 to 5 days, superficial layers of tissue begin to slough and form granulation tissue. This is the point when bowel wall is in weakest status and it will be prone to perforation

4 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 that will increase the strength of bowel wall and decrease of delayed perforations

Esophageal stricture and obstruction may form over a period of weeks to years from contraction of the scar.

Food bolus obstruction in esophagus will make it difficult for the patient to swallow.
Lower bowel obstruction will cause vomiting



Caustics



Degree of burn:

Caustic injury is graded into 3 degrees/Grades based on endoscopy:

| First Degree | Second Degree | Third Degree |
|---|---|---|
| Hyperemia | superficial ulcers, white membrane, exudates, friability and hemorrhage <i>assets by endoscopy</i> | Transmural involvement with deep injury, necrotic mucosa, or frank perforation of the stomach or esophagus. |
| Edema | 2A Non circumferential | |
| | 2B Circumferential | |
| This division is important for long term prognosis as 2B has higher for developing an obstruction | | |



The initial grade of burn on esophagoscopy correlates with the risk of stricture formation. Chances of stricture formation are:

30% Grade 2A Burns: 15 to 30% develop strictures

75% Grade 2B: upto 75% develop strictures

90% 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.

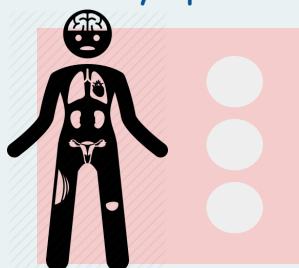
Acid-base reaction for neutralization produces heat that may cause burns in addition to chemical burn except in clorox ingestion when drinking milk is recommended



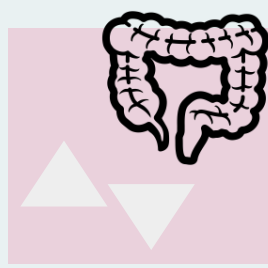
Clinical Features:

-Clinical features depends on the site and severity of burns.

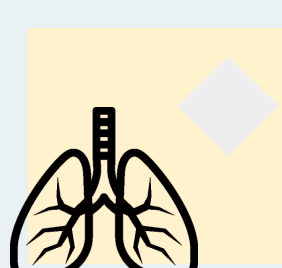
-Patients with acid or alkali ingestions present with similar initial constellation of signs and symptoms.



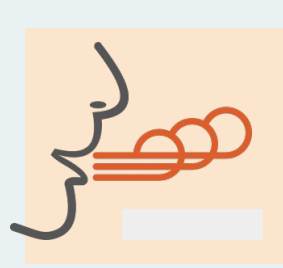
[SYSTEMATIC]



[GI TRACT]



[RESPIRATORY]



[AIRWAY]

LOCAL EFFECTS



Clinical Features



Help!

Airway edema and esophageal/gastric perforation are most emergent issues
Small ingestion of potent substances can be as serious as larger ingestions.

Airway

- Laryngeal edema occurs over a matter of minutes to hours.
- Tracheal necrosis is one of the most frequent causes of death after caustic ingestion.

GIT

- More than 40% of patients reporting to have "only taken a lick" have esophageal burns.
- Patients present with oral pain (41%), abdominal pain (34%), vomiting (19%), and drooling (19%).
- Visible burns to the face, lips & oral cavity. Burns can occur from spills or **contamination after vomiting**.
- Symptoms include dysphagia and food impaction (Dysphagia usually subsides in 3 to 4 days)

Systematic

- Systemic toxicity; hypovolemic shock; and hemodynamic instability with hypotension, tachycardia, fever, and acidosis are ominous findings.
- Peritoneal signs suggest hollow viscus perforation or extension of the burn to adjoining visceral areas.
- The fulminant course of some acid ingestion 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.

Respiratory

- Some have wheezing and coughing. Others present with stridor and dysphonia.
- Chest pain is common.

-Mild burns will present with pain, swelling of airways, spitting out saliva and vomiting.
-Severe burns will have more systemic manifestations.

The acidity of the stomach doesn't mean that it won't be affected by acids; strong acids can add to this acidity and cause harm



Caustics



Cont. Clinical Features:

Complications:

- ▲ Patients with significant esophageal burns, particularly those that are circumferential, may develop esophageal stricture 80% of strictures become apparent in 2 to 8 weeks
- ▲ 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



Significant acid ingestions may be devastating and result in a higher mortality rate than alkali ingestions.

Prediction:

Oropharyngeal burns alone do not appear predictive of more distal injury

Prolonged drooling and dysphagia predicted significant lesions with 100% sensitivity and 90% specificity. Even if the oral mucosa is not injured

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

In late presentation 4 or 5 days after ingestion, chances of infection, sepsis and obstruction will increase

Most of patients with oral burns will have also deeper burns that will develop swelling and obstruction of the air way, so patients must be intubated early before respiratory symptoms starts, especially if there is mild cough or mild hoarsens of the voice

Even after 30 or 40 years, people who had history of caustic ingestion will have higher risk (up to 1000-3000 compared to general population) develop esophageal cancer



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. CT has higher sensitivity and specificity for proliferation of organs

ABG to monitor systemic metabolic acidosis. In cases of intentional overdose, coingestants should be considered. If we suspected lungs or airway injury



- 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



- Check Hb if internal bleeding is suspected
- Check liver and renal functions
- Litmus test if the caustic nature is not known
- Chest x-ray to rule out aspiration pneumonia
- Abdominal x-ray for free air that may have been caused by perforation

primary management is to stop the ongoing burns and to manage the ABCs and vital signs.
-if the patient has mild burns in the mouth we should ask him to gargle without swallowing the water.
-if the patient is in shock give fluid poulses
-if the patient has electrolytes imbalance correct it
-any airway burns intubate the patient
-if the patient is vomiting blood do blood transfusion
Then after that let the specialist asses the depth and severity of burns.

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Caustics

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Management:

Fluid

- ◆ 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.
- ◆ IV access and vigorous fluid resuscitation.



- ◆ Contaminated clothing should be treated as hazardous waste and disposed of using proper precautions.
- ◆ Activated charcoal, and performing gastric lavage are not indicated.
- ◆ Careful nasogastric aspiration may be useful in the setting of significant acid ingestions presenting immediately after ingestion



Decontamination

Intubation

- ◆ 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



- ◆ 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



Drugs & Surgery

Early and continuous respiratory and hemodynamic monitoring is essential.

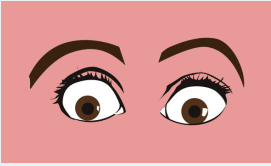
Do not neutralize the ingested corrosive with weak acids or alkalis due to thermal reactions and worsening injury.



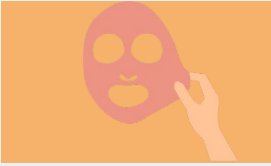
Most of the times especially in children caustics is ingested by accident, the manifestations is usually mild (no visible or very superficial burns, patient is active and cooperative, no drooling), in this case the patient should be asked to drink some water:

- If the patient is able to swallow it and doesn't have dysphagia (remember it has high sensitivity and specificity for deep burns) try to neutralize the burns by some milk, observe for several hours then discharge the patient.
- If dysphagia is definite and deeper burns are suspected we should arrange for endoscopy to assess the extent and depth of burns.
- Endoscopy will not help only in management it will also help to know the long term prognosis of an injury BUT it has to be done by the early window period if it is available, if not we should wait few days before performing it.

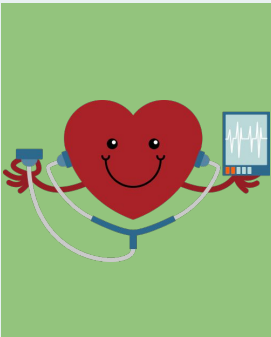
{ Special Cases }



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 **through cornea**

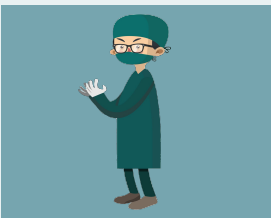


Dermal caustic exposures can also result in significant burn injuries. Clothing removal, copious irrigation, and local wound debridement are the most important initial treatment



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**. **ECG monitoring and calcium replacement** if the patient has **hypoglycemia**

Cardiac and serum calcium monitoring are warranted in all.



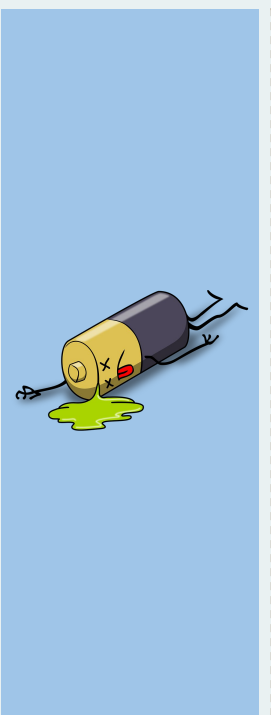
Povidone-iodine (Betadine): is used as a surgical scrub and is not a caustic agent, but ingestion of tincture of iodine can cause severe gastrointestinal injury and is potentially life threatening. Gastric irrigation with starch or milk in these cases may convert iodine to less toxic iodide.



Concentrated hydrogen peroxide (H₂O₂): found in hair bleaching substances. 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 H₂O₂ ingestion.



Button batteries are usually made of a metallic salt (lithium, mercury, nickel, zinc, cadmium, or silver) bathed in NaOH or KOH. **alkaline batteries**

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.

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.



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.

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.

Give sodium bicarbonate

Phenol is well absorbed through the skin, dermal exposure may result in systemic toxicity.

Can cause CNS manifestation. If Seizures happen use benzodiazepines

Dermal decontamination of phenol exposures with LMW polyethylene glycol has been suggested but water may prove just as useful

Anything reaches the cecum will come out, unless it gets stuck there and produces symptoms (nausea, vomiting) so you have to remove it

Summary

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Acids $\text{pH} < 7$

Proton donors;

The **commonest** Hydrochloric acid (HCl)
Sulfuric acid (H_2SO_4)

Coagulation necrosis → then eschar formation

Strong odor, immediate pain on contact

Resistance of squamous epithelium to coagulation necrosis → systemic absorption (metabolic acidosis)

burns of stomach will be augmented by the acidity of gastric secretions

Alkalis $\text{pH} > 7$

Accept protons;

Alkalis caustics include:

- Lye (NaOH and KOH).
- Ammonia (NH_3)

Liquefaction necrosis, fat saponification, and protein disruption

Colorless, odorless, no immediate pain on contact

- Typically involve the squamous epithelial cells of the oropharynx and esophagus
 - Gastric necrosis and perforation.
- Burns below the pylorus carry a worse prognosis

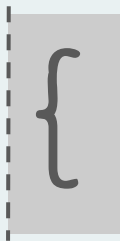
DIAGNOSIS:

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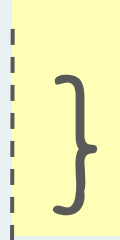
MANAGEMENT:

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- If dysphagia is definite and deeper burns are suspected we should arrange for endoscopy to assess the extent and depth of burns.
- Endoscopy will not help only in management it will also help to know the long term prognosis of an injury BUT it has to be done by the early window period if it is available, if not we should wait few days before performing it.
- Do not neutralize the ingested corrosive with weak acids or alkalis due to thermal reactions and worsening injury.
- Early and continuous respiratory and hemodynamic monitoring is essential.



How toxic is your knowledge



Q1: A 10 year old child is brought to the hospital after ingestion of some poison. He is conscious and dynamically stable, but complains of severe oral pain and inability to swallow. On examination, he has swollen lips, tongue, and drooling of saliva. Which of the following is the likely cause of this presentation?

- a. Caustics
- b. Paracetamol
- c. Aspirin
- d. Pesticides

Q2: A 8 year old child ingest drain cleaners and upper GI endoscopy has been performed. What is the type of necrosis to be seen in the above patient?

- a. Fibrinous
- b. Liquefactive
- c. Coagulative
- d. Caseous

Q3: Most lye solutions used as all purpose cleaners and for industrial purposes contain which one of the following?

- a. Alkaline substance
- b. Acidic solutions
- c. Neutral pH solution
- d. Mixture of acidic and alkaline solution

Q4: Which one of the following is an acid Caustic ?

- A. Sodium hydroxide
- B. Potassium hydroxide
- C. Sulfuric acid
- D. Ammonia

Q5: You are treating a seven-year-old child who accidentally drank acid. He has severe oral pain with ulcers in his mouth and is drooling saliva. He is crying with a hoarse weak voice and is coughing constantly. He is complaining of retrosternal chest and abdominal pain. He is slightly tachycardic and tachypneic and looks dehydrated. What is the first step in the management of this patient?

- A. Secure an IV line and give him intravenous fluid boluses
- B. Request an urgent Gastroenterology consultation for performing Upper GI endoscopy
- C. Request for blood work including hemoglobin, counts, renal function and electrolytes
- D. Gently suction his airway, give him oxygen and prepare for elective intubation

Q6: A 5-year-old boy presents with history of drooling of saliva and inability to eat or drink because of dysphagia for the last 6 hours. X-ray shows that there is a button battery lodged in the mid-esophagus.

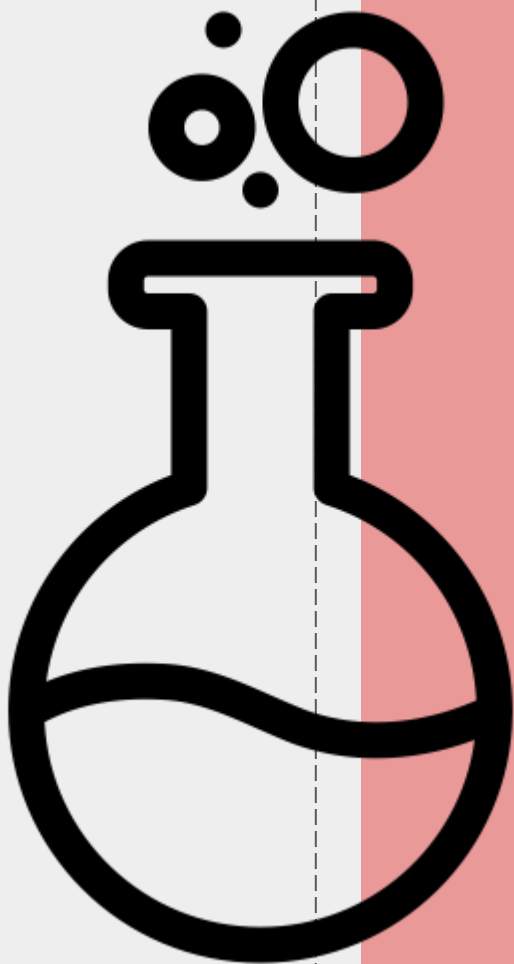
What would be your next step in the management of this patient?

- A. Expectation observation for the battery to descend to the stomach
- B. Arrange for urgent endoscopy removal of the battery
- C. Start IV steroids to prevent perforation of the esophagus
- D. Start prophylactic Antibiotics to prevent secondary infection



- 1-A
- 2-C
- 3-A
- 4-C
- 5-D
- 6-B

THANK YOU AND GOOD LUCK!



VERY TOXIC BUT YOU ARE
GONNA DO IT!

A+ is yours (:

- Email us at:

436toxicology@gmail.com

How well do you think we have done? We are waiting for your feedback!



Click here!

- THEME WAS DESIGNED BY: ASEEL BADUKHON
- LOGO WAS DESIGNED BY: NORAH ALHOGAIL