Ingestion of Caustic Agents

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Caustic ingestion is a serious avoidable medical problem with a variety of clinical presentations and a complicated clinical course.

There is a bimodal occurrence with regards to the age groups involved. 80% of caustic ingestion occurs accidentally in young children (< 5 years old). This accounts for 0.3% of annual paediatric admissions in the Gambia. The second peak of occurrence occurs in an older age group. In adults, ingestion of caustic substances is frequently intentional, involving larger amounts of substance, and subsequently the injuries sustained are often are life-threatening.

Agents

Caustic agents can generally be classified into either acidic (pH less than 3) or alkali (bases with pH greater than 11), however neutral agents also ingested and still cause harm.

Below is a table of the commonly ingested caustic agents;

Agent type	Examples
Alkali	Sodium hydroxide AKA caustic soda, Potassium hydroxide (oven cleaners, liquid drain cleaners, disk batteries) Calcium and lithium hydroxide (hair products) Ammonia (household cleaners) Dishwasher detergents
Acid	Sulfuric acid, hydrochloric acid, nitric acid (toilet cleaners, pool cleaners, rust removers)
Other	Sodium hypochlorite (usually neutral) Peroxide Kerosene

Mechanism of injury

The literature on the damage caused by the differing substances generally suggests alkali compounds cause more severe injuries with liquids specifically often causing greater damage than solids. With alkaline liquids, liquefactive necrosis occurs with diffusion into deeper layers of the injured mucosa.

Acids are conventionally thought to cause coagulation necrosis that forms an eschar, preventing deep tissue penetration however there are documented cases of acids also causing deep tissue damage.
Neutral agents, depending on the substance, often cause tissue irritation, oedema and subsequent tissue damage. With bleaches the oesophagus isn't usually greatly damaged however laryngeal oedema may cause rapid airway compromise.

When ingested caustic substances can cause widespread injury to the lips, oral cavity, pharynx, and the upper airway. Often it is the damage to the oesophagus that is the most serious resulting in the greatest number of short and long term complications.
The nature of the injury is dependent on several factors, mainly the type of agent, the amount consumed, the concentration, and the length of time the agent is in contact with a given tissue.

Symptoms

These differ and usually correlate to the agent volume, concentration, and damage caused

- Obvious burns to the lips, mouth, and oropharyx.
- Laryngeal or epiglottic oedema may present with
  - Stridor, aphonia/dysphonia, wheezing, or dyspnoea.
- Pulmonary oedema suggests likely lower respiratory exposure
- Other non-specific symptoms include
  - nausea, recurrent emesis, hematemesis, dysphagia, odynophagia, and drooling.
- The presence of abdominal pain or rigidity as well as substernal/chest or back pain may be a sign of severe burn or perforation.
- Oesophageal perforation may result in mediastinitis, with severe chest pain, tachycardia, fever, tachypnoea, and shock.

Management

Treatment of caustic ingestion is largely supportive

As with all acutely unwell patients an ABCD approach should be adopted.

A - The airway must firstly be assessed and if unstable then intubation under direct vision is advised. If intubation is difficult or fails then a surgical airway should be secured. It is airway compromise that will kill these patients very acutely after the injury.

After the airway has been protected a thorough history and examination can be conducted to establish the extent of the injuries. Particular focus should be given to establishing the substance ingested, volumes involved and timing of events. It is also important to consider whether the ingestant contains any poisonous substances and if so vital to consult appropriate literature to manage this aspect.

B/C - Chest and abdominal x-rays are useful aid on detecting possible oesophageal perforation (free air in the mediastinum / diaphragm). This will also guide the development of an aspiration pneumonia.

The patient should be kept nil by mouth with aggressive IV hydration provided. Biochemical markers can establish the presence of acidosis or organ failure.

Dilution (with milk) is said to be only useful in the first few minutes after ingestion.

DO NOT encourage gastric emptying by emesis or lavage. It is contraindicated as it re-exposes the GI tract to the substance

DO NOT attempt to neutralize the substance as this produces an exothermic reaction and can subsequently cause heat damage.

It is important to note that these injuries like other burns are dynamic over the hours, days and weeks after initial ingestion. Therefore, it is important to continue to reassess the patient and manage the possible complications if or when they development.

Complications

The most significant and long lasting injuries occur when there is damage to the oesophagus. Transmural oesophageal burns to the oesophagus may be associated with up to a 20% mortality rate. Endoscopy is used to establish the extend of the damage however the timing of when to conduct oesophagoscopy is strongly debated with no consensus in the literature. Each case is unique and pros and cons for endoscopy discussed on a case by case basis.

There are two significant complications seen in oesophageal injuries;

1. **Oesophageal stricture formation**
   Occurs in around 10 – 20% of oesophageal injuries
   Can occur as early as three weeks post injury
   Symptoms include
   - Severe dysphagia
   - Obstructive symptoms (80%)

   May require long term parenteral feeding or often balloon dilatation of the stricture. In severe cases grafting and even oesophageal reconstruction is required

2. **Oesophageal perforation**
   Perforation of the oesophagus, stomach or duodenum may lead to peritonitis, shock, and death
   Such injuries require surgical interventions such as exploratory laparotomy

Oesophageal injury predisposes the patient to oesophageal carcinoma by 1000-fold for 10 to 25 years. Long term follow-up is inevitable.

Morbid functional complications include nasopharyngeal reflux, hypopharyngeal and laryngeal stenosis, and tongue fixation.