Pathophysiology of Caustic Ingestion

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Introduction

A 2005 report from the American Association of Poison Control Centers indicates that there were over 200,000 exposures to caustic substances in the form of household and industrial products, including acids (sulfuric acid, hydrochloric acid), alkanis (potassium hydroxide, sodium hydroxide), and other agents. While the majority of cases involved exposure of a body surface, such as the face, eyes, and extremities, ingestion of the caustic agent was the leading cause of death [1]. Of the different types of ingested caustics, the most commonly involved are:

A. strong alkalis, such as sodium hydroxide and potassium hydroxide, which exist in different forms, including granular, paste, or liquid. The most common household forms of the alkalis include washing detergents, drain cleaners, soaps, cosmetics, and clinitest tablets. Alkalis are found in high concentrations in button batteries, which can cause severe injury if they leak after ingestion of the battery.

B. strong acids can be found in battery fluids (sulfuric acid), toilet bowel cleaners (sulfuric and hydrochloric acids), antirust compounds (oxalic and hydrochloric acids) and slate cleaners (hydrochloric acid) [2].

Many medications can cause damage to the esophageal mucosa on contact. This can occur when patients take pills without water or in a recumbent position, or if there is a predisposing factor to esophageal injury. Examples for medications causing this type of mucosal injury to the esophagus include potassium chloride, tetracycline, aspirin, and doxycycline. It is noteworthy that potassium hydroxide ingestion can cause perforations of the brachial artery, left atrium, and aorta [3].

The majority of caustic ingestions in the United States involve strong alkalis with concentrations exceeding 50%. The Federation Caustic Poison Act of 1927 was the first major government effort for poison prevention in the United States that aimed to decrease the incidence of caustic ingestion, which mostly occurs in children. This was followed by the Poison Prevention Packaging Act and the Hazardous Substances Act in the 1970s. Both restricted the concentration of household cleaning agents to less than 10%, and also mandated the use of child resistant containers and warning labels. However, these measures have not totally prevented the occurrence of serious injuries from ingestion of caustic products [4].

Factors Affecting the Extent and Severity of Gastrointestinal Injury due to Caustic Ingestion (Fig. 1).

Physical Form (solid, liquid, or gel)

The form of an ingested corrosive determines the pattern and distribution of injury in the gastrointestinal tract. Liquid alkalis cause diffuse and circumferential burns
while solids tend to produce localized burns especially at sites of anatomic constrictions [5]. The amount of ingested caustic is also dependent on the physical form. While the liquid form is easily swallowed, the crystallized corrosive tends to cause marked oral pain, which limits further swallowing. Granular automatic dishwashing powder has also been associated with severe injury to the gastrointestinal tract [6].

**Contact Time**

Logically, the longer the contact-time between the caustic compound and tissue, the more profound the injury. For example, exposure of rat esophagus to caustic soda for a period of 10 min causes damage to the esophagus and if prolonged to 120 min, leads to perforation [7].

**Concentration**

As expected, the damage induced by a caustic solution is positively correlated to its concentration in solution. For example, exposure of rat esophagus to 1.83 % caustic soda largely causes epithelial necrosis, while 7.33 % produces additional submucosal damage, and a concentration of 14.33 % further extends the damage to the muscle and adventitial layers [7].

**pH, pKa, and Titratable Acid/alkaline Reserve**

Alkalis are known to cause liquifactive necrosis, with greater penetration into the tissue layers, while acids induce a coagulative necrosis that limits tissue penetration [8]. Potential injury to tissues by a corrosive cannot be predicted based solely on pH. There are many other determinants of the ability of a corrosive to damage tissues, including solution strength. The term ‘strength’ refers to the willingness of the alkali to dissociate in aqueous solution, and is expressed by the equilibrium constant, pKa, which is the pH at which the alkali is 50 % dissociated to its conjugate acid. Strong alkalis are compounds that have a pKa greater than 14 or are capable of complete dissociation in water [9].

Titratable acid or alkaline reserve (TAR) is a better indicator of the ability of a caustic to injure the tissue. It is defined as the amount of acid or alkali that needs to be added to the caustic in order to reach neutral pH. Higher TAR values generally