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**Case Report**

# Fatal Caustic Ingestion: A Case Report & Short Review

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**Abstract:** Caustics cause both clinical and histologic damage on contact with tissue surfaces. They are available in both solid and liquid forms, with variations in viscosity, concentration of solution, and pH. The severity of a caustic injury may not be immediately evident in patients who present shortly after exposure. An acid is a proton donator and causes significant injury, generally at a pH below 3. The extent of injury is determined by duration of contact; ability of the substance to penetrate tissues; volume, pH, and concentration; the presence or absence of food in the stomach; and a property known as titratable acid reserve (TAR). Here we are presenting fatal intentional consumption of caustic with autopsy findings followed by short review of literature.
**Keywords:** Caustic, Sulphuric Acid

## INTRODUCTION

Caustics cause both clinical and histologic damage on contact with tissue surfaces. They are available in both solid and liquid forms, with variations in viscosity, concentration of solution, and pH. Usually, children are unintentionally exposed to household products. Adults may be exposed to household or industrial products that result in either occupational exposure or are used in suicide attempts [1]. Although less frequent, intentional exposures by adults are invariably more significant. One study noted that although children comprised 39% of admissions for caustic ingestions, adults comprised 81% of patients requiring treatment [2]. The severity of a caustic injury may not be immediately evident in patients who present shortly after exposure. Predicting which patients will require immediate interventions to prevent morbidity and mortality requires multiple clinical and laboratory parameters. Here we are presenting one such case of corrosive acid poisoning with autopsy findings followed by short review of literature.

## CASE REPORT

Here is 34 year old man labourer by occupation living in the old Delhi. Allegedly he consumed unknown poison at his home due to some quarrel at home at around 8:00 PM. Then he was taken to a Lok Nayak hospital, New Delhi, where he expired after 4 hours after admission. The body was subjected to Medico-legal post mortem examination 22hours after death at Department of Forensic Medicine, Maulana Azad Medical College, New Delhi.

Autopsy Examination: The dead body was of an adult male aged about 34 years, moderately built and moderately nourished. Both the eyes were closed, cornea hazy and conjunctivae were congested. Brownish to blackish excoriations were present over lips. Brownish to blackish excoriations in the form of marks of trickling present, over chin, chest, front aspect of lower part of shirt and upper part of pant.

Post-mortem staining was present over back of the body. Rigor present could be demonstrated involving all major joints.

Brownish to blackish excoriations and ulcerations present over mucosa of mouth, tongue and pharynx. Teeth were glistening white. While cutting the tongue gritty sensation was felt. On cut section, hardening of tongue muscles present.

**Fig. 1: Excoriations and ulcerations over mucosa of tongue**

On opening the abdomen, 300ml of brownish turbid fluid was present in the peritoneal cavity. Small and large intestines were congested and its visceral surface covered with turbid fluid.

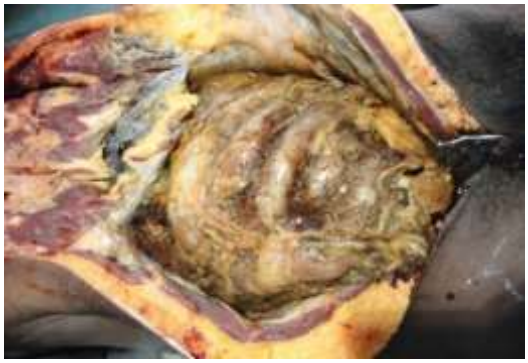

**Fig. 2: Congestion of intestines covered with turbid fluid**

Blackish mass covered with blood clots noted at left hypogastric region. On dissection, within the mass, remnants of stomach parts were found with blackish edges.

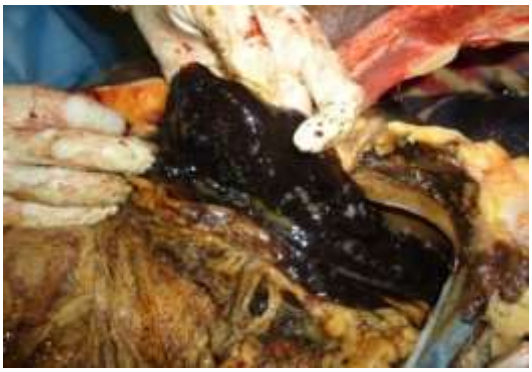

**Fig. 3: Stomach converted to Blackish hemorrhagic mass**

On opening thoracic cavity and on dissection of oesophagus, mucosa showed loss of rugosity with brownish to blackish excoriations and ulcerations at places.

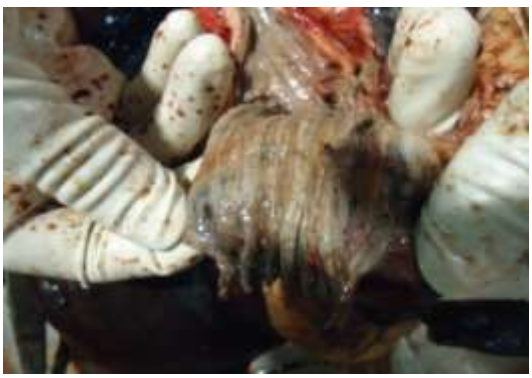

**Fig. 4: Oesophageal mucosa showing brownish to blackish excoriations and ulcerations**

Both lungs were congested and edematous. Cut section, showed congestion. Both the kidneys were congested. Cut section, showed congestion and obliteration of cortico-medullary junction. Liver and spleen were congested and hardened at places. Blood and viscera were preserved, packed, labelled, sealed and handed over to concerned Police for toxicological analysis in Forensic Science Laboratory. Toxicology report showed traces of sulphur atoms in stomach and intestines, indicating the poison to be Sulphuric acid. Finally, we opined with our post-mortem findings and toxicology report, the cause of death as "Shock as a result of peritonitis due to consumption of sulphuric acid".

## DISCUSSION

An acid is a proton donator [3] and causes significant injury, generally at a pH below 3. The extent of injury is determined by duration of contact; ability of the substance to penetrate tissues; volume, pH, and concentration; the presence or absence of food in the stomach; and a property known as titratable acid reserve (TAR). TAR quantifies the amount of neutralizing xenobiotic needed to bring the pH of a caustic to that of physiologic tissues. Neutralization of caustics takes place at the expense of the tissues, resulting in the release of thermal energy, producing burns. Generally, as the TAR of caustics increases, so does their ability to produce tissue damage. Some xenobiotics, such as zinc chloride and phenol, have a high TAR and are capable of producing severe burns even though they possess a near-physiologic pH.

Following exposure to an acid, hydrogen ($H^+$) ions desiccate epithelial cells, producing an eschar and resulting in what is histologically referred to as coagulation necrosis. This process leads to edema, erythema, mucosal sloughing, ulceration, and necrosis of tissues. Dissociated anions of the acid ($Cl^-$, $SO_4^{2-}$, $PO_4^{3-}$) also act as reducing agents further injuring tissue. In this case, it has been noted the erythema, mucosal sloughing, ulceration, and necrosis esophagus and stomach.

Ophthalmic exposure to acids results in coagulative necrosis that tends to prevent further penetration into deeper layers of the eye. In most series, following an acid ingestion, both the gastric and esophageal mucosa is equally affected [4]. On occasion, the esophagus may be spared damage while severe injury is noted in the stomach [5]. This tends to be a rarer finding than concomitant injury to both stomach and esophagus, and is probably related to the rapid transit time of liquid acids through the upper gastrointestinal tract. Skip lesions from acid ingestions may be a function of viscosity and contact time [3]. Additionally, acid-induced pylorospasm may lead to gastric outlet obstruction, antral pooling, and perforation [6].

Esophageal burns, secondary to acid exposures, are classified based on endoscopic visualization that employs a grading system similar to that used with burns of the skin [1]. That is:

- Grade I burns are generally described as hyperemia or edema of the mucosa without evidence of ulcer formation.
- Grade II burns include submucosal lesions, ulcerations, and exudates. Some authors further divide grade II lesions into
  - Grade IIa, noncircumferential lesions, and
  - Grade IIb, near-circumferential injuries.
- Grade III burns are defined as deep ulcers and necrosis into the periesophageal tissues.

In this, it is noted all the 3 grades at various levels in the oesophagus.

## CONCLUSION

Titratable Acid Reserve (TAR) quantifies the amount of neutralizing xenobiotic needed to bring the pH of a caustic to that of physiologic tissues. Generally, in cases of consumption of strong acids with less duration of survival, the most common cause of death would be peritonitis.

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