Case Report

# Delayed death due to Hydrochloric acid containing toilet-cleaner poisoning: A case report

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## ABSTRACT

Corrosive injuries of the stomach are common in developing countries. The mortality and morbidity from corrosive poisonings is high in India and depends on the severity of initial damage caused by the corrosive agents before the patient reaches to hospital. An average home contains a dozen of different cleaning products. These account for a large number of accidental and suicidal poisonings. We discuss a case intentional self-harm by ingesting toilet cleaner containing higher amounts of hydrochloric acid and presented to the emergency department of Sri Aurobindo Medical College & Post Graduate institute, Indore. The different aspects of the case are discussed in detail in this paper.

**Keyword :** hydrochloric acid; toilet - cleaner; corrosive

## INTRODUCTION

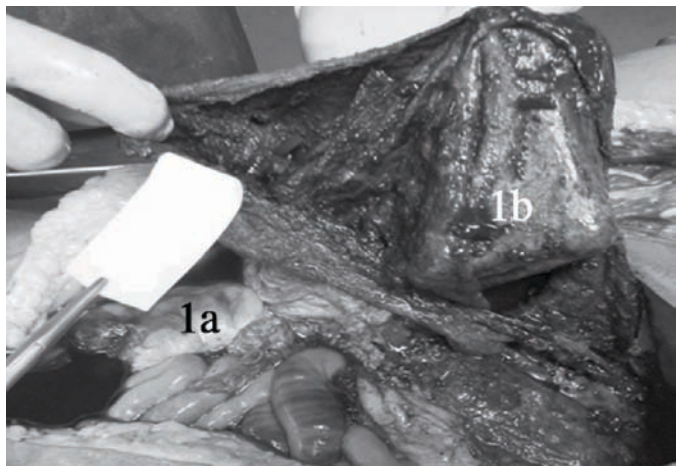
Corrosives are the group of chemicals that have the capacity to cause tissue injury on contact by a chemical reaction. Corrosives and caustics are synonyms, both mean 'something that eats away'. Acids and alkalis are the two primary types of agents most often responsible for caustic exposures.[1] In developing countries, accidental or suicidal ingestion of acids is encountered more often than in developed countries where alkaline corrosives are more frequent.[2] They most commonly affect the gastrointestinal tract, respiratory system and eyes. We discuss a case of suicidal corrosive acid ingestion in which patient succumbed to death due to perforation peritonitis after 4 days of hospital stay.

## CASE-REPORT

A 25 years old female was brought to the casualty with the history of acid ingestion (Toilet cleaner containing 10% of Hydrochloric acid) at home after having quarrel with her husband. As per the case sheet records she had complains of abdominal pain and two episodes of vomiting. Gastric lavage was not done. Patient was drowsy with B.P. - 120/70 mmHg and Pulse - 80/min. Patient was immediately shifted to ICU. Patient was haemo-dynamically stable and had no respiratory or metabolic acid-base imbalance by the fourth day. Sudden clinical deterioration was observed on fourth day with free fluid in abdomen. She succumbed to death after four days of admission. Body was subjected to medicolegal autopsy.

Autopsy findings showed body was moderately built and nourished. Both the lips were brownish black in colour with evidence of acid corrosion over parts of cheek, chin, neck & chest, trickling down from the angle of mouth. There was corrosion of mouth and tongue with chalky white teeth. Surgical tracheotomy wound was present on front of neck. Rigor mortis was present all over the body. Post mortem lividity was purplish and present on back of chest & abdomen, fixed. Pleural cavity had 100 ml of red colored fluid. Trachea contained scanty mucoid froth. The oesophagus was corroded and its mucosa was shred off. The peritoneal cavity was having 100 ml of black coloured liquid and was showing features of chemical peritonitis. The serosal surface of stomach was black with multiple areas of full thickness perforation with black irregular softened edges along lesser curvature and pre-pyloric area. Mucosal surface of stomach was charred and perforated containing about 50 mg of black coloured material (Figure 1). Peritoneum was dull and inflamed. Small intestine especially duodenum also had black colored charred material.

**Fig.1:** Diffuse, confluent patches of sub-mucosal hemorrhage and necrosis involving the entire mucosal surface of stomach. 1a: showing a collection of free fluid containing altered-blood tinged in the peritoneal cavity. 1b: showing a perforation present over the pre-pyloric area of stomach.

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Toxicological analysis of stomach contents showed curdy white precipitate with silver nitrate test, which was insoluble in nitric acid and soluble in ammonia, suggesting presence of Hydrochloric acid. (Figure 2). It's noteworthy that this test for Hydrochloric acid came positive even after four days of consumption and hospitalization.

The opinion as to the cause of death of the deceased is attributed to the corrosive acid consumption and the complications like chemical peritonitis thereof.

**Fig.2:** Silver nitrate test confirming presence of Hydrochloric acid demonstrating the solubility of the white precipitate in Ammonia and Nitric acid.


## DISCUSSION

Exposure to corrosive agents continues to be a leading toxicological source of injury for children and adults. A study was performed on the pattern and outcome of acute poisoning cases in a tertiary care hospital in Karnataka, which concludes that out of 136 patients of various poisoning cases, 5.9% of them were due to corrosives and also very high mortality rate that is 23.8 % in cases of corrosive poisonings.[3] Gastric burns occur following ingestion of corrosive materials, either accidently by children and alcoholics, or intentionally by emotionally disturbed individuals. Ingestion with suicidal intent is due to free availability of the caustic agents in the market. Acids affect the stomach more commonly than alkalis.[4] Acids cause mucosal damage by coagulation necrosis and require a longer duration of contact. On the other hand, alkalis cause liquefaction necrosis[5] and are more viscous, and tend to adhere to the esophageal mucosa with only a relatively small amount reaching the stomach. The extent of esophageal damage is greater with alkalis than acids.[6] However, alkali damage of the stomach has also been reported.[7]

In most instances, the gastric burn injury is greatest along the lesser curvature and in the prepyloric area. The explanation for this distribution was provided by Testa in 1938. He introduced caustic soda mixed with barium into the esophagus of dogs and demonstrated by x-ray that the alkali barium bolus flowed along the lesser curvature, produced severe pylorospasm, and was retained in the pre-pyloric area.[8] Perforation of the stomach or the esophagus can occur at any time during the first 2 weeks. Hence, any change in the clinical condition of the patient such as worsening of abdominal pain or the appearance of chest pain should be promptly investigated by radiologic studies.[9]

The extent of gastric injury appears to be related to the nature, volume, and concentration of corrosives ingested; the length of time they remain in contact with the stomach; the content of the stomach at the time of ingestion; and the relative tonicity of the pyloric sphincter.[8] A full stomach tends to minimize injury because it will dilute and act as a buffer for the ingested acid.[10] At times only superficial mucosal injuries occur which heal uneventfully. In those who die late subsequent to acid exposure, a spectrum of predictable injuries are seen, which involve the submucosa and muscularis producing various degrees of deformity including antral stenosis, hourglass strictures, and rigidity simulating infiltrating gastric carcinoma. Severe caustic injuries of the stomach may result in perforation of its wall and development of acute abdomen. This requires emergency surgery. These injuries may appear in the first 48 hours or they may be delayed until the 14$^{th}$ day after corrosive ingestion.[8] Perforation injuries are reported in sulphuric acid ingestion cases,[8,10,11] however perforation due to hydrochloric acid consumption is relatively rare. A perforation of stomach on 4$^{th}$ day after initial uneventful hospital stay emphasizes the need for imperative repeated daily abdominal examinations to be performed during the first two weeks following corrosive ingestions.

## CONCLUSION

The key to improving the survival of such patients is its early detection of agent and also complications like perforation and to give them supportive care with maintenance of nutrition and control of sepsis. Parents should be made aware of the need to keep household corrosives safely away from children to prevent such incidences.

## CONFLICTS OF INTEREST

Declared none.

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