Corrosive Injury to the Upper Gastrointestinal Tract
Due to Acid Ingestion
—Report of Two Cases—

Takaya Tanaka, Naoshi Takeyama, Mineo Matsubara,
Hideharu Yamanaka*, Koshiro Hioki* and Masakatsu Yamamoto*
Emergency Care Unit and *Department of Surgery Kansai Medical University
Moriguchi, Osaka 570, Japan

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Abstract
The authors report two patients who ingested hydrochloric acid, one of whom died of septic shock 3 days after ingesting the acid. Both patients developed delayed, severe constriction of the entire esophagus and the stomach. Ingestion of acid often causes severe, extensive corrosive damage to the upper gastrointestinal tract. Appropriate management of this life-threatening condition requires an awareness of its subtle manifestations as well as a carefully planned surgical approach. The authors describe their cases in detail and discuss the early and late clinical manifestations and the current treatment options.

Introduction
Acid ingestion usually involves children who accidentally drink acidic household cleaners or battery fluid or adults attempting suicide. Most often, the result is corrosive tissue injury rather than immediate death. The damage to the gastrointestinal tract caused by acids differs in nature and site from that caused by alkaline agents, whereas acids frequently produce stomach burns but usually spare the esophagus, alkaline substances effect the esophageal mucosa most severely and only occasionally produce gastric injury.

In this report, we describe the cases of two adults who developed severe constriction of the entire esophagus and stomach as a result of acid ingestion. One patient died 3 days later of septic shock.

Case Reports
Case 1. A 40-year-old male with a history of schizophrenia attempted suicide 1 day prior
to admission by drinking 500 ml of the liquid toilet bowl cleaner Sanpol® which contains 0.9% hydrochloric acid. On arrival at the hospital emergency room, he was in shock, with severe abdominal pain and violent vomiting. He also exhibited confusion, lethargy, tachypnea, and profound metabolic acidosis. There were extensive burns of the mouth, tongue, and soft palate. The abdomen was rigid, with diffuse tenderness and minimal rebound tenderness. Abdominal x-rays taken over the next 2 days showed no evidence of free air but suggested paralytic ileus. Emergency esophagogastroduodenoscopy revealed multiple ulcerations and moderate bleeding of the esophagus, stomach, and duodenum.

The patient was treated with fluids, an antacid, dopamine, antibiotics, and a steroid, and his vital signs gradually improved. However, hepatic and renal parameters, including S-GOT, S-GPT, alkaline phosphatase, bilirubin, BUN, and S-creatinine, showed rapid elevation and he developed disseminated intravascular coagulation. His temperature fluctuated between 39.0 and 40.5°C, and diffuse abdominal tenderness persisted. Because he was unable to swallow saliva, a tracheostomy was performed. Despite these measures, 36 hours after admission the patient again lapsed into shock and died.

At autopsy, the distal esophagus, stomach (Fig. 1), duodenum, and proximal jejunum were observed to have undergone severe hemorrhagic necrosis, but there was no evidence of

![Fig. 1 Cross section of the stomach showing discoloration and mucosal hemorrhagic necrosis.](image)
perforation. Microscopic examination revealed necrosis of the stomach mucosa and smooth muscle and thrombosis of the arterioles, with no adjacent normal-appearing stomach.

Case 2. A 48-year-old female with a history of reactive depression attempted suicide by stabbing herself in the left hypochondrium with a dagger. On admission she was stuporous but responded to stimuli, and was noted to have localized erosion of the mouth and tongue. Nasogastric suction yielded dark blood. At this time, it was not known that prior to the stabbing incident the patient had ingested large quantities of Sanpol®.

Emergency laparotomy initially disclosed only penetration of the left lobe of the liver and free blood in the abdominal cavity. Subsequently and unexpectedly, the stomach was found to be distended with liquid and clotted. The surface of the stomach was thickened, hyperemic and edematous; the posterior surface was grayish-red, without perforation. The duodenum and the small and large bowel appeared normal. Because of the patient's precarious condition, resection of the stomach was seemed inadvisable at this time. Only hemostasis and suturing of the penetrating wound of the left liver were performed.

Postoperatively, gastric lavage was carried out on the assumption of ingestion of a corrosive substance. On the seventh hospital day, esophagogastroduodenoscopy revealed confluent

Fig. 2 Esophageal stricture 5 weeks after acid ingestion.

Fig. 3 Gastric contraction 5 weeks after acid ingestion.

white plaques, with the adjacent erythematous mucosa containing multiple large ulcerations throughout the entire esophagus, stomach, and second portion of the duodenum. After a postoperative symptom-free period of 26 days, the patient developed postprandial vomiting, and an upper gastrointestinal series obtained 5 weeks after ingestion of the acid demonstrated severe contraction of the esophagus and stomach (Figs. 2 and 3). Seven weeks after admission, total esophagogastrectomy and substernal bypass with closure of the duodenal stump were performed, the descending colon being interposed between the cervical esophagus and proximal jejunum. The resected esophagus and stomach were severely constricted and inelastic. The mucosa had been replaced by granulation tissue and contained multiple ulcerations (Fig. 4). The patient's recovery was uneventful and she is able to eat without difficulty.

Discussion

These two cases illustrate the range of responses to ingestion of a corrosive substance. The factors that determine the extent of damage after ingestion of a corrosive agent are the amount of normal material in the stomach, the amount and concentration of the corrosive substance, the duration of its contact with the gastrointestinal mucosa, and the tone of the
pyloric sphincter. A full stomach buffers the acid's effects and tends to minimize the injury. It is well known that acids tend to destroy the stomach and spare the esophagus, owing to the resistance of the esophageal squamous epithelium and rapid esophageal transit time. On the other hand, alkaline agents usually injure the esophagus and spare the stomach, due to the neutralizing effect of the acid normally present in the stomach. Nevertheless, ingestion of large amounts of either type of substance causes severe damage to both organs in 10 to 20 % of cases. Concomitant damage to both organs has been encountered in only six of 30 reported cases since 1960.

The effects of acid on the gastric mucosa are similar to the consequences of external body burns. Essentially, the depth of the burn determines the late sequelae; the depth is classified as superficial, deep, or transmural. The last is a feature of severe damage in which extensive necrosis, maceration, and acute gangrenous perforation develop in an occasional patient within 24 to 48 hours after ingestion, resulting in peritonitis, mediastinitis, septic shock, renal failure, and death. However, in our fatal case, death resulted from septic shock in the absence of perforation. Daly and Cardona reported death from extensive necrosis of the stomach in a patient being treated for esophageal stricture.

The natural history of corrosive gastritis, as described by Marks et al., has been confirmed. Necrosis with saponification occurs in the acute phase immediately following ingestion and lasts from 1 to 4 days. Three to five days after the chemical insult, extremely hemorrhagic erosions and ulcerations appear, with sloughing of the superficial necrotic tissue. Such changes alone are not indicative of nonviability of the damaged organ. However, as ulcerations heal and fibrosis develops, decreasing vascularity and increasing rigidity are seen, and the healed mucosa becomes smooth and pale. Maximal involvement invariably occurs at the antrum, and results in pyloric or prepyloric stenosis within 3 weeks to 4 months after ingestion.

Endoscopy is safe and of crucial importance in the management of corrosive injury to the upper part of the gastrointestinal tract. The risk of instrumental perforation of the esophagus during the acute stage is the principal deterrent to routine early endoscopy. Chung and Den-Besten suggested that under such circumstances, the examination be terminated and then repeated at 48-hour intervals, in the interest of early inspection of the upper gastrointestinal tract. An aggressive endoscopic approach has several advantages: 1) The depth and extent of esophageal and gastric involvement are often clear after one or two further examinations; 2) in the absence of mucosal gangrene, conservative management, with monitoring by repeated endoscopy, may be carried out with relative safety; and 3) accurate mapping of the extent of the mucosal lesion, which frequently cannot be accomplished by serosal
examination, allows surgical intervention prior to life-threatening perforation, or when healing of the acute lesion has progressed to the point where elective resection can be carried out with maximum safety.

There is no general agreement as to the initial treatment following ingestion of acid. The value of nasogastric intubation, gastric lavage, administration of milk, and induction of vomiting is controversial. Antacids are probably indicated, especially if given early. Spain et al. and Rosenberg et al. reported that steroids, or with antibiotics, inhibited fibroplasia and exerted an anti-inflammatory effect. There is no clear evidence, however, that steroids or antibiotics are beneficial.

The most controversial aspect of the management of acid ingestion is surgical intervention. Ultimately, most patients who survive the ingestion require surgery. Some authors have recommended early operation as a means of averting late complications. Most, however, advocate postponing surgery until the full extent of scarring and deformity of the stomach is evident, except in cases of severe hemorrhage and/or free perforation. During emergency surgery, removal of necrotic tissue is essential and may necessitate total esophageal resection, gastric resection, pancreatoduodenectomy, and small bowel resection. However, the mortality exceeds 40%.

Attempts at definitive restoration of intestinal continuity should be deferred at the time of emergency surgery, as anastomoses of edematous friable segments of esophagus, stomach, or small bowel are prone to disruption. Ingestion of a highly concentrated acid carries a particular prognosis because of both early and late complications. Both may develop insidiously and have a deceptively quiet course, as occurred in our two cases.

References


