

## Case Report

# Acute Gastric Perforation after Acid Ingestion

Feryal Gün, Latif Abbasoğlu, Alaaddin Çelik

*Istanbul University, Istanbul Medical School, Department of Pediatric Surgery, Turkey*

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### ABSTRACT

Gastric outlet obstruction is a common late result after acid ingestion; early complications, such as gastric necrosis or perforations are unusual. This is a report of a patient with the history of strong acid ingestion who underwent total gastrec-

tomy due to perforation and extensive necrosis of the stomach. *JPGN* 35:360–362, 2002. **Key Words:** Acid ingestion—Gastric perforation—Gastrectomy. © 2002 Lippincott Williams & Wilkins, Inc.

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### INTRODUCTION

Corrosive injury after ingestion of caustic household products is a common problem in children (1,2). The ingestion of alkalis primarily damages oropharynx and esophagus whereas the injury from ingestion of strong acids usually involves the distal part of the esophagus and stomach (2,3). The acid pools in the antrum and causes gastric outlet obstruction more commonly because of cicatricial antral stenosis (1,4). Concentrated acids, if swallowed in large amounts, may even lead to early perforation of the stomach if it is in a fasting state (2–4). We report a case of acute gastric perforation due to acid ingestion and discuss its clinical presentation, course, and management.

### CASE REPORT

A 2-year-old boy was admitted to our unit 2 hours after ingesting an unknown amount of scale dissolvent (25% nitric acid). He was lethargic. His respirations were shallow and he was vomiting blood. His vital signs were temperature, 36°C; pulse rate, 154/min; respiratory rate, 40/min; and blood pressure, 110/60 mm Hg. On physical examination, there were erythemas of the lips and oropharynx, and first- and second-degree skin burn on the neck. Abdominal examination revealed tenderness and voluntary guarding. Laboratory studies disclosed the following values: WBC count, 34,100 /mm<sup>3</sup>; hemoglobin,

8.6 g/dl; glucose, 520 mg/dl; Na, 127 mEq/L. Arterial blood gas values showed pH, 7.11; pO<sub>2</sub>, 133 mm Hg; and pCO<sub>2</sub>, 55 mm Hg. Renal, hepatic, and pancreatic function test results were within normal limits. An abdominal roentgenogram showed free air under the diaphragm suggesting a perforation in the gastrointestinal tract. He experienced a respiratory arrest. He was intubated, intravenous antibiotics were given and he was transported to the operating room immediately.

Laparotomy revealed extensive gastric necrosis on both the anterior and posterior walls with a linear perforation of 4 to 5 cm along the greater curvature starting from fundus of the stomach (Fig. 1). The esophagogastric junction was not damaged. A total gastrectomy was performed, remodeling the gastrointestinal tract by anastomosing the distal end of the esophagus to the jejunum with a Braun anastomosis, and closing the duodenal stump. The resected stomach measured 9 cm in length and 6 cm in width. The mucosa was dark brown and black in color. Microscopic sections of stomach showed extensive areas of necrosis and edema. On the second postoperative day, weaning from mechanical ventilation was achieved. He was given total parenteral nutrition for 10 days. On the seventh day, barium meal revealed a satisfactory esophago-jejunal anastomosis, and oral feeding was started and well tolerated (Fig. 2). However, at the end of the first month a stricture was detected at the anastomotic region which was dilated. He is currently asymptomatic, 18 months after the dilatation.

### DISCUSSION

Acidic substance ingestion is less common in children than alkali ingestion. The acidic products ingested are

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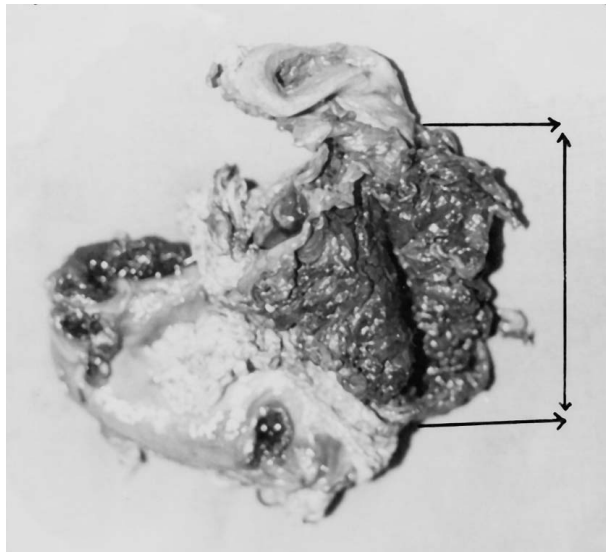
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Correspondence to: Feryal Gün, Hareket Ordusu sok. Isil apt. No: 12/17 Bahçelievler, Istanbul, 34590 Turkey (e-mail: gunferyal@yahoo.com).

usually household cleaning substances. In contrast with alkaline caustics that damage the esophagus and usually spare the stomach, acidic solutions frequently pass through the esophagus without causing mucosal injury but producing coagulation necrosis of the stomach as in a thermal burn (1,2). Although the esophagus is usually spared after acid ingestion due to rapid transit and the resistance of squamous epithelium, isolated cases of esophageal acid injury have been reported (4,5). Acid ingestion may induce pyloric spasm and produces antral mucosal edema, inflammation, and finally pyloric stricture due to extensive fibrosis (2-4,6). Gastric burns caused by alkali corrosives have also been reported (7).

The extent and the severity of corrosive gastric injury is directly related to the concentration and amount of acidic substance as well as the length of time in the stomach, and to the amount of gastric content at the time of ingestion (1,4,6,8). The most-frequently encountered acidic substances are hydrochloric acid, sulfuric acid, or, as in our case, nitric acid.

Strong acids reaching the stomach may cause perforation and peritonitis in 24 to 48 hours if a large volume is involved and if the organ is empty (2,3,9). In our case, perforation appeared 2 hours after ingestion.

An immediate total gastrectomy may be indicated, as in our case, if there is perforation or extensive necrosis of the stomach wall (2-4,7,10). Otherwise, these patients should be kept under close observation and fiberoptic endoscopy is performed to determine the extent of the damage (1,6). Total gastrectomy in children is more troublesome than in adults because of side effects such as anemia, vitamin B<sub>12</sub> deficiency, and growth retardation (11,12). Our patient is doing well so far with respect to these complications.



**FIG. 1.** Extensive necrotic areas and perforation site of the stomach.



**FIG. 2.** Barium meal showing patent esophagojejunal anastomosis.

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