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Endoscopic evolution of a caustic injury to the gastric antrum

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Caustic injury to the upper GI tract can occur due to accidental or intentional ingestion of caustic substances. We present a case of severe caustic injury of the gastric antrum complicated by prepyloric stenosis, and illustrate the evolution and resolution of the injury with serial endoscopic images over a 175-day period.

CASE REPORT

A 25-year-old female nursing student presented with hematemesis, epigastric pain, hoarse voice, and dysphagia with inability to swallow saliva 30 minutes after intentional ingestion of approximately 1 tablespoon of granular caustic soda (sodium hydroxide).

Endoscopy was performed 12 hours after the initial ingestion (day 0) and revealed oral cavity edema without overt ulceration and moderately severe distal esophageal inflammation without ulceration or bleeding. **Figure 1** demonstrates grade 2B circumferential caustic injury of the distal one third of the stomach (grade 2B injury defined as deep focal or circumferential ulceration¹); extensive mucosal necrosis and edema are evident. This area was not traversed due to the risk of perforation.

Intravenous antibiotics and a continuous infusion of proton pump inhibitor were administered. Oral intake of fluids resumed on day 6, permitted because of resolution of dysphagia and abdominal pain. She was discharged on day 16 on a liquid diet and oral proton pump inhibitor.

Progress endoscopies were undertaken on days 2, 7, and 14. By day 14, the esophagus had completely healed, while the area of severe involvement in the gastric antrum had

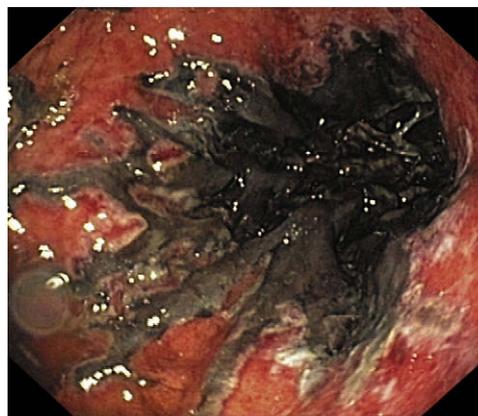


Figure 1. Day 0. Grade 2B circumferential caustic injury of the distal one third of the stomach (antral/prepyloric region), which is hyperemic and edematous, with areas of ulceration and mucosal necrosis.

subsided to 50%. **Figure 2** (day 14) demonstrates grade 2A antral injury, defined as superficial ulceration with bleeding and exudate.¹ The pylorus was traversed for the first time; the duodenum appeared normal.

Symptoms of early satiety developed after 1 month, and endoscopy on day 35 revealed severe pinhole stenosis at the site of previous severe injury (**Figs. 3A and B**). Serial incremental dilations of the stricture, from 12 mm to 20 mm, with a controlled radial expansion (CRE) balloon (Boston Scientific, Boston, Mass) were performed over 5 sessions from day 42 to day 70, permitting resumption of a normal diet. Neither recurrence of stenosis nor symptoms of gastric outlet obstruction occurred between day 70 (**Fig. 4**) and the following scheduled endoscopy at day 175 (**Fig. 5**).



Figure 2. Day 14. The area of injury has retracted; there is ulceration and exudate.

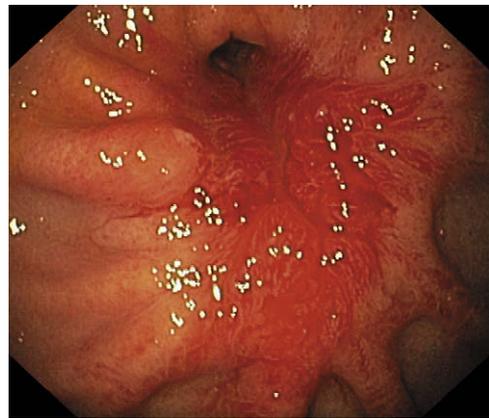


Figure 4. Day 70. Gastric antral stenosis 1 week after the third dilatation (18 mm; subsequently dilated to 20 mm).

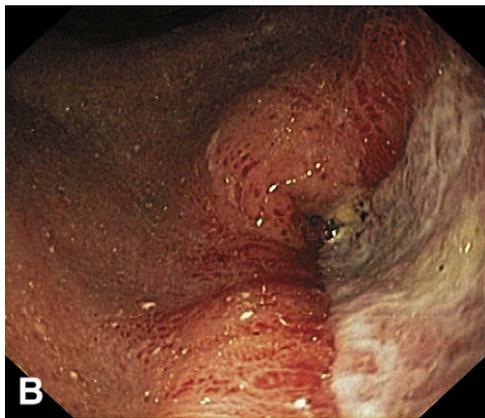
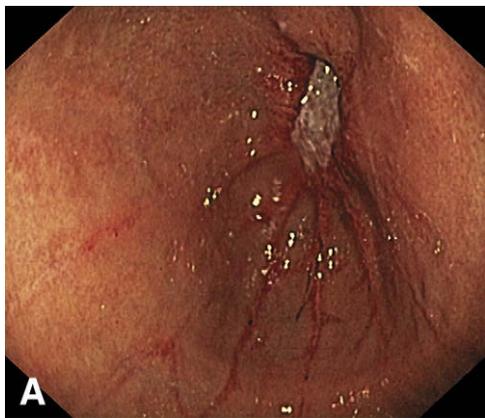


Figure 3. A and B, Day 35. Severe pinhole stenosis at the site of injury.

DISCUSSION

Caustic soda is the main ingredient in common household cleaning products, and its ingestion can cause a spectrum of injuries to the upper aero-digestive tract, ranging from esophagitis and gastric ulceration to potentially life-threatening oropharyngeal and laryngeal injuries with respiratory compromise, esophageal necrosis with perforation and mediastinitis, and gastric perforation with



Figure 5. Day 175. Advanced healing of the gastric injury. The lumen remains substantially narrowed but without recurrence of clinically significant symptoms.

peritonitis. Esophageal injury is reported more commonly than gastric injury.²

In those for whom immediate surgery is not indicated, conservative management includes the use of empiric antibiotic therapy and vigilant monitoring for extra-intestinal complications such as airway and respiratory compromise.³ As healing occurs, those with at least grade 2B caustic injury have a 70% chance of stricture formation necessitating mechanical dilatation or surgery.² In cases of gastric injury, critical antropyloric stenosis can develop rapidly, with a median time to symptom onset of 2 months.³ Systemic corticosteroids and antidotes such as milk and water have been used; however, their benefits remain unproven.^{4,5} The role of proton pump inhibition has not been studied in this group of patients.

This case demonstrates the evolution of severe gastric antral injury due to caustic soda ingestion, and is, to our knowledge, the first case report in the published literature to endoscopically document the complete process in a single patient. In cases of severe injury, vigilant clinical follow-up facilitates early endoscopic assessment and low-risk treatment before critical, potentially endoscopically

refractory high-grade stenosis develops. Further studies are required to establish the optimal clinical and endoscopic follow-up protocol.

DISCLOSURE

All authors disclosed no financial relationships relevant to this publication.

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EUS-guided FNA diagnosis of primary splenic lymphoma

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Primary lymphoma of the spleen associated with occlusion of the splenic vein has been reported only once.¹ We report a case of primary splenic lymphoma associated with splenic vein thrombosis (SVT), which led to sinistral (left-sided) portal hypertension (HTN) related GI bleeding. EUS-guided FNA (EUS-FNA) was used to confirm the diagnosis and led to appropriate therapy.

CASE REPORT

A 51-year-old man with a history of HTN was admitted with an embolic stroke. A week prior, he had been to a local hospital emergency department with a 1-week history of melanic stools and weakness. His hemoglobin level was 5.5 gm/dL (normal 12.9-16.9 gm/dL), which required a blood transfusion. An EGD at that time revealed old blood in the stomach and “enlarged folds” in the gastric fundus. Given the concern for an embolic source of his stroke and recent GI bleeding, a GI consultation was requested for clearance for anticoagulation. An EGD was repeated, which revealed a normal esophagus and isolated gastric varices with red wale signs. A CT revealed a complex 21-mm × 24-mm solid lesion located between the pancreatic tail and splenic hilum, which caused focal splenic vein occlusion (Fig. 1). There was also a

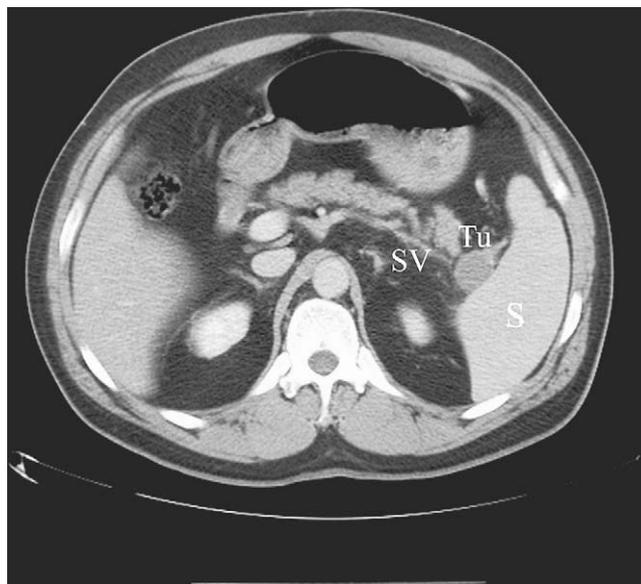


Figure 1. CT in the portal-vein contrast phase, demonstrating solid, round mass (*Tu*) in the hilum of the spleen (*S*), with attenuation of the splenic vein (*SV*).

50-mm × 39-mm oval hypodense splenic lesion (Fig. 2). EUS confirmed 2 oval, well-defined hypoechoic lesions, likely perisplenic lymph nodes, around the pancreatic tail and splenic hilum, one of which involved the splenic