Esophageal stricture due to magnesium citrate powder ingestion: A unique case

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CASE PRESENTATION

A 40-year-old man presented to the emergency department with dyspnea and chest pain after ingesting one teaspoon of magnesium (Mg) citrate powder (630 mg of elemental Mg) prescribed by his naturopath for constipation. He failed to mix it with 180 mL (6 oz) of fluid as instructed on the product monograph. Potential adverse effects were not listed. He was initially treated for aspiration pneumonia but returned two weeks later with chest pain and dysphagia. Endoscopy demonstrated severe Los Angeles grade D esophagitis from 18 cm to the gastroesophageal junction. He was discharged with a prescription for an oral proton-pump inhibitor (PPI). Twenty-three days post-ingestion, he presented with progressively worsening dysphagia and odynophagia. A second endoscopy identified a caustic stricture 25 cm from the incisors. A 6 mm controlled radio expansion wire-guided balloon was used to attempt dilation but was stopped due to fresh heme and tearing. He was admitted to hospital for intravenous fluid and PPI therapy. An upper gastrointestinal barium radiograph identified a tapered narrowing initiating at 3 cm below the cricopharyngeus (Figure 1). The first attempt at endoscopic dilation using a 15 Fr Savary bougie was unsuccessful due to significant transmural inflammation. Next, an ultrathin endoscope was used to pass a guidewire into the stomach and a Hurricane biliary balloon (Boston Scientific, USA) was used to sequentially dilate the stricture to 6 mm to 8 mm. An 18 mm × 170 mm FCSEMS (Hanarostent-Esophagus CCC, MI Tech Co Ltd, USA) was advanced via guidewire and deployed using fluoroscopic guidance (Figure 2). The patient could tolerate a full fluid diet. Subsequent endoscopies at two-week intervals identified 5 cm of distal stent migration, for which proximal esophageal dilation and lasso repositioning were required. Two months after initial placement, the stent was removed and the esophagus was dilated to a diameter of 15 mm. At two follow-up endoscopies, stricturing had recurred, indicating a full-thickness stricture (Figure 3).

Surgical management was explored because his quality of life was severely affected. The surgeons performed endoscopy using a small-diameter Savary bougie dilator to determine the suitability of the gastric mucosa for an esophageal replacement conduit. After this, he developed two areas of contained perforation; he was admitted to hospital and treated with antibiotics. Seven months after initial ingestion, he underwent esophagectomy with gastric conduit and feeding jejunostomy.

DISCUSSION

In the United States, 5000 to 20,000 caustic ingestions occur annually (1,2). Primarily observed in children, a minority are encountered in the adult population and are often intentional. There are no reported cases of caustic ingestions secondary to Mg citrate, a weak base, in the medical literature. However, a search of the grey literature identified two personal accounts on public forums of 'inflamed esophagus' and 'burned esophagus' after ingestion of undiluted Mg citrate powder.

Figure 1) Barium upper gastrointestinal series: A tapered narrowing of the proximal esophagus begins 3 cm below the cricopharyngeus. The narrowing persists over 13 cm and is 2 mm in diameter, demonstrating long-segment stenosis of the esophagus

Figure 2) Fluoroscopy. Expandable metallic stent is demonstrated to be in satisfactory position after endoscopy and antegrade instrumentation of the midthoracic esophageal stricture including balloon dilation

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TABLE 1
Endoscopic grading of caustic mucosal injuries

Grade	Endoscopic findings
0	Normal
1	Edema and hyperemia of mucosa
2A	Friability, hemorrhages, erosions, blisters, whitish membranes, exudates and superficial ulcerations
2B	2a plus deep discrete or circumferential ulceration
3A	Small scattered areas of necrosis
3B	Extensive necrosis

Data adapted from reference 4

Acidic ingestions cause a coagulative necrosis leading to eschar formation (2,3). Alkali ingestions cause liquefactive necrosis, submucosal vascular thrombosis and transmural injury (2,3). Between the third week and several months after ingestion, scar retraction and stricture formation occur. An increase in gastroesophageal reflux from altered sphincter pressure exacerbates stricture formation (3). The quantity ingested, pH of substance, duration of exposure and physical state of the product predict severity of disease (2,3). Acute complications include mucosal injury, fistulae, perforation, mediastinitis and peritonitis, which, in rare cases, lead to death (2-4). Long-term complications include esophageal stricture, pyloric stenosis and esophageal squamous cell carcinoma.

Endoscopy should be performed within 24 h to assess severity of damage and prognosis; symptoms and examination are not reliable (2-4). Procedure-related perforation in early endoscopy is rare (3). The Zargar endoscopic classification is used to grade caustic mucosal injuries (Table 1) (4). Grades 0 to 2A have a good prognosis, with low likelihood of stricture formation (2-4). Grades 2B to 3A have a very high likelihood of stricture development (70% to 100%) (2-4). Grade 3B is associated with increased mortality and the need for surgery (2-4).

Animal studies have demonstrated benefit from steroids for prevention of stricture formation; however, human studies are inconclusive and use is only considered in grade 3 injuries (2,3). Concomitant prophylactic antibiotics are recommended when treating with steroids (3). Intraluminal stents may be helpful in preventing stricture formation (3). Treatment with antacids to prevent acceleration of stricture formation by any potential reflux is recommended, although evidence is limited (3). Treatment of developed strictures includes dilation and, potentially, surgery (3).

In our case, the stent was placed early; however, it failed due to repeated migration. Repeated dilations due to recurrent stricture formation also failed and, ultimately, the patient required surgical intervention. Our case is unique because it describes a severe esophageal stricture due to Mg citrate ingestion that was previously unreported. The patient

Figure 3) Barium upper gastrointestinal series five months post-ingestion demonstrating persistent stenosis

was not made aware of the potential risks before consuming this product, nor was it listed on the product monograph. Patients should be educated about the potential for side effects from natural supplements.

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