Nonsurgical management of severe esophageal and gastric injury following alkali ingestion

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The ingestion of caustic substances may result in significant gastrointestinal injury. Endoscopy can play a major role in the initial evaluation and subsequent therapy of such injuries. The case of a 50-year-old man who ingested an alkaline floor stripper is described, including the endoscopic management of esophageal and pyloric strictures, with good functional results. The role of endoscopy, steroids and acid suppression in the management of such patients is also explored.

Key Words: *Caustic ingestion; Endoscopic therapy; Esophageal stricture; Pyloric stricture*

La prise en charge non chirurgicale d'une grave lésion œsophagienne et gastrique après l'ingestion d'un alcali

L'ingestion de substances caustiques peut provoquer d'importantes lésions gastro-intestinales. L'endoscopie occupe une place importante dans l'évaluation initiale et la thérapie subséquente de ces lésions. Les auteurs décrivent le cas d'un homme de 50 ans qui a ingéré un décapant à plancher alcalin, y compris la prise en charge endoscopique des constrictions œsophagiennes et pyloriques, et les bons résultats fonctionnels. Ils explorent également le rôle de l'endoscopie, des stéroïdes et de la suppression acide dans la prise en charge d'un tel patient.

The ingestion of corrosive substances often results in significant proximal gastrointestinal mucosal injury, occasionally perforation, and sometimes death. Despite regulations for improved labelling of such harmful substances, the rate of ingestion ranges from 5000 to 20,000 ingestions per year in the United States (1,2). While the majority of these are accidental ingestions involving children, a substantial minority involve adults, often with suicidal intent (3-5). The natural history of such ingestions is dependent on numerous factors, including the quantity ingested, the pH of the material, the duration of exposure and its physical state (ie, solid or liquid) (1,2,6). Acidic substances result in coagulative necrosis, with a protective eschar forming and limiting deep injury. In contrast, alkaline substances cause a liquefactive necrosis, with substantial submucosal vascular thrombosis, often producing transmural injury (1,2,4,6).

Endoscopy plays an important role in the initial assessment of patients who have ingested a caustic material by visually defining the extent and severity of injury. Esophageal stricturing is a common complication, and antegrade dilation often provides excellent relief (7-10). Pyloric stenosis may also result from caustic injury and this complication may be amenable to endoscopic intervention (8,11-14). The present report describes the care of a patient who suffered significant esophageal and gastric injury following large volume alkali ingestion. The ensuing complications, including esophageal and pyloric stricturing, were managed entirely endoscopically, with a good functional outcome.

CASE PRESENTATION

A 50-year-old male prisoner ingested 300 mL of liquid floor wax stripper, mixed with grape juice. The liquid's active ingredients included sodium carbonate, sodium metasilicate and sodium hydroxide, and its pH was 13.4. On the patient's arrival to the emergency room, he was experiencing difficulty swallowing his saliva. He also described oropharyngeal burning pain. He denied shortness of breath or chest pain. His vital signs were normal. The oropharynx had significant chemical burns, manifested by erythema, diffuse edema and mucosal sloughing. General physical examination was otherwise unremarkable. Laboratory tests, including complete blood count, electrolytes, creatinine levels and arterial blood gas were normal. The electrocardiogram, chest x-ray and abdominal films were also normal. A laryngoscopy was performed, and although laryngeal erythema and edema were evident, it was determined that the patient was not at significant risk of airway compromise.

Endoscopy performed 12 h after ingestion revealed significant injury to the oropharynx, as well as edema and erythema in the larynx and vocal cords. The proximal esophagus sustained near-circumferential injury manifested by edema, the presence of a yellow-white coagulum and friability. The distal esophagus was more severely injured with circumferential edema, tissue sloughing and erythema (Figure 1A). Frank tissue necrosis was evident in the body and antrum of the stomach, visualized as black eschar, edema and hemorrhage (Figure 1B). The pylorus was edematous but could be intubated. The proximal duodenal cap had mild injury, but distal to this, the duodenal mucosa appeared normal.

In consultation with an esophageal surgeon, who viewed the endoscopic images of the extent of injury, surgical and nonsurgical options were discussed with the patient. In the present case, it was decided to admit the patient under close observation for any signs of deterioration. Total parenteral nutrition was initiated and the patient remained nil per os. Systemic steroids were not administered. A repeat endoscopy was performed one day after ingestion. The esophageal injury had not progressed, and

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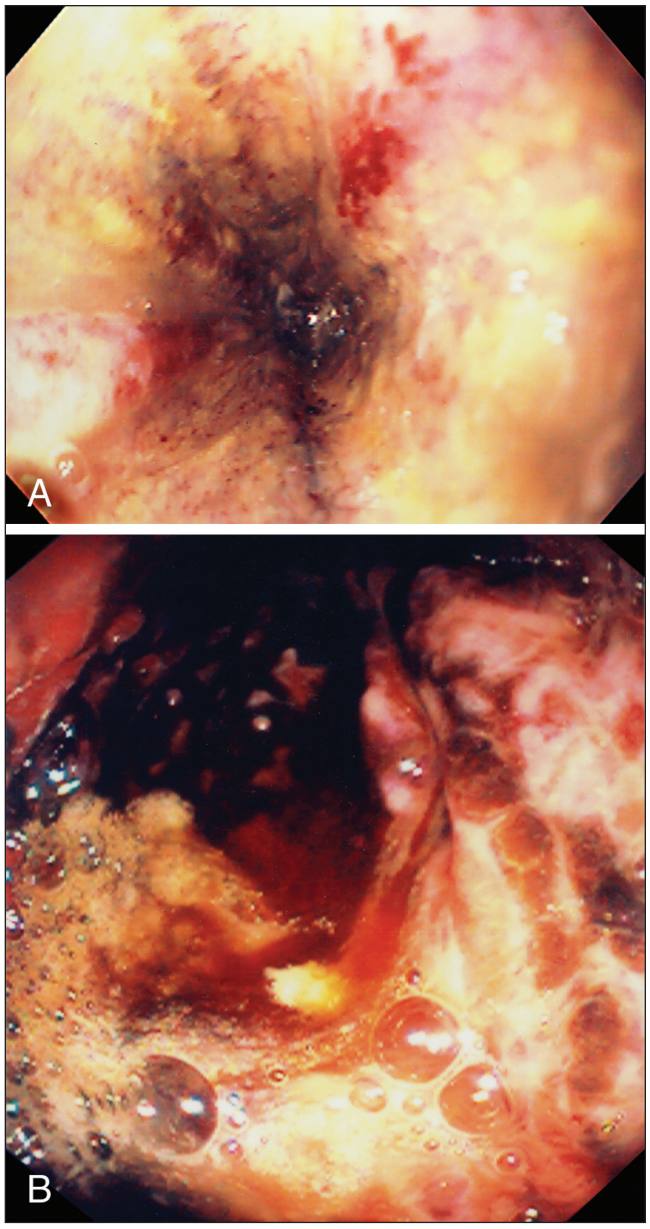

Figure 1) *Photos taken during endoscopy 12 h following alkali ingestion, demonstrating edema, mucosal sloughing and hemorrhage of the distal esophagus* (**A**)*, and severe mucosal injury with hemorrhage, edema and black eschar in the stomach* (**B**)

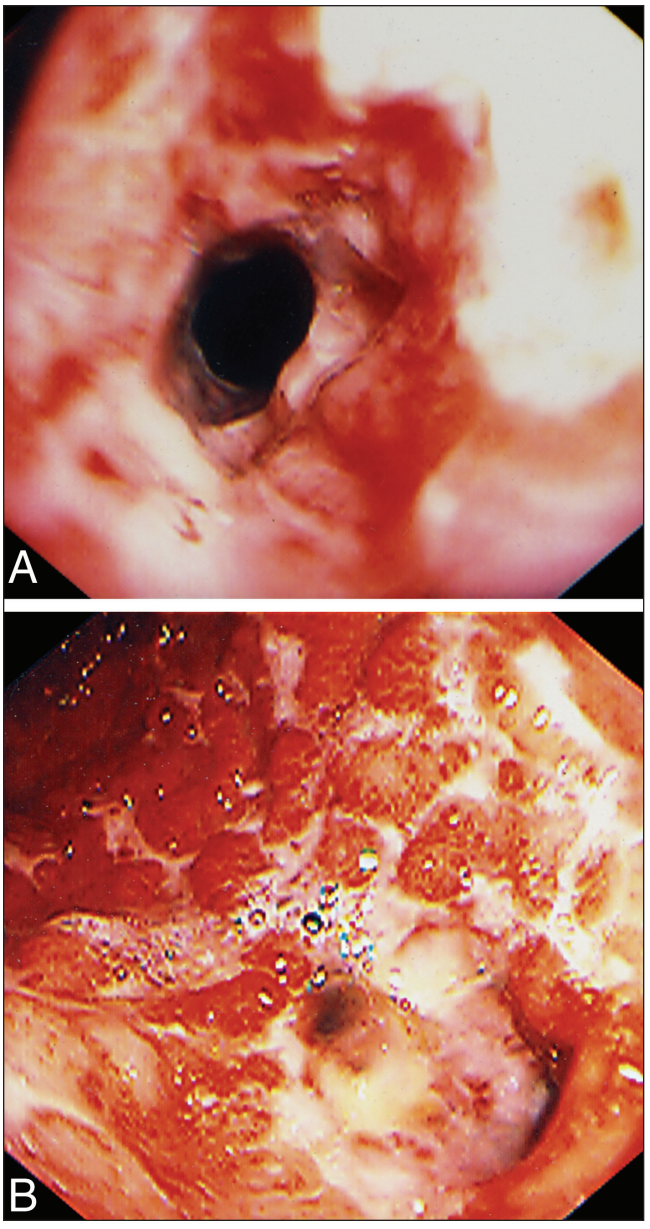

Figure 2) A *Endoscopic view of an esophageal stricture and inflammation 35 cm from the incisors, 10 days postingestion.* **B** *Endoscopic image of a distorted antrum 10 days postingestion. Active ulceration and a narrowed but patent pylorus are demonstrated*

the gastric body and antrum remained severely inflamed and injured. Conservative management and close observation were continued. During the next week, the patient's oropharyngeal symptoms improved and he was able to swallow secretions.

A repeat endoscopy was performed 10 days postingestion. The esophageal mucosa was healing but ulceration was still present, particularly at the site of two early strictures, 25 cm and 35 cm from the incisors (Figure 2A). The proximal stricture was noncircumferential and the adult gastroscope (diameter 9.4 mm) passed through it easily. The distal stricture was circumferential and narrowed the lumen more significantly, only allowing passage of the pediatric gastroscope (diameter 8.7 mm). The stomach was distorted by a contracted antrum with continued active ulceration (Figure 2B). The pylorus was patent but did have mucosal erosions and ulceration. The duodenal cap had returned to normal. The esophageal strictures were serially dilated with Savary dilators over a guidewire under fluoroscopic control, starting at a diameter of 9.3 mm (28 Fr) to a maximum diameter of 12 mm (36 Fr). The patient was started on clear fluids and tolerated this well.

A repeat endoscopy was performed on postingestion day 17. The distal stricture was narrowed more significantly than previously and would not allow passage of even the pediatric endoscope (diameter 8.7 mm). This stricture also continued to have active ulceration. Again, under fluoroscopic control, the strictures were dilated, to a maximum diameter of 10 mm (30 Fr). The stomach contained copious fluid, even though the patient had nothing to drink for the preceding 16 h. The pylorus was visualized and it continued to have active ulceration. It was strictured and would not allow passage of the

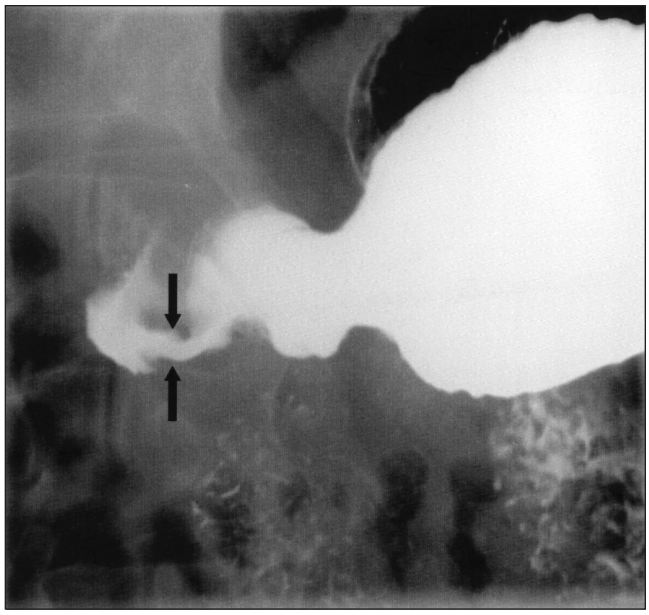

Figure 3) *Barium contrast radiograph of the stomach demonstrating flow of barium through a patent pylorus (arrows) into the duodenum*

pediatric endoscope. A wire was passed through the pylorus into the duodenum, under fluoroscopic control. This stricture was dilated using a 12 mm hydrostatic balloon (CRE, Boston Scientific, Canada), filled with saline and radio-opaque contrast dye in a one to one ratio, under 8 atm pressure for 60 s. Under fluoroscopy, a very short balloon waist, suggesting a short pyloric stricture, was successfully dilated. The endoscope was then manoeuvred through the pylorus into the duodenum without difficulty.

Over the next three weeks, the patient underwent four further endoscopies with esophageal and pyloric dilations. After this, he denied any dysphagia. His diet was advanced to solids. There was no longer any endoscopic evidence of gastric stasis or retention. He was discharged on oral proton pump inhibitors.

Six further endoscopies were performed on him as an outpatient over the next three months. Active ulceration and inflammation at the distal esophageal stricture continued until 12 weeks after the ingestion. Up to four months after ingesting the alkali, dilations of the distal stricture were necessary, but only required one pass of an 18 mm (54 Fr) dilator. An upper gastrointestinal barium radiograph was performed three months postingestion. The esophagus was patent, although mildly narrowed distally. The antrum was contracted and the barium flowed easily into the duodenum (Figure 3). The patient was followed for 18 months after his ingestion, and experienced no dysphagia or symptoms of gastric stasis.

DISCUSSION

Alkali ingestion results in liquefactive necrosis, which is often more severe than the coagulative necrosis resulting from acid ingestion (2,4). The natural history of such injuries, if perforation is avoided, is ulceration, frequently followed by strictures resulting from an intense fibrotic reaction that may occur in the weeks following ingestion (4).

Endoscopically visualized esophageal injury has been shown to be predictive of complications of caustic ingestion such as bleeding, perforation, fistula formation or death (15-18).

TABLE 1
Zargar Endoscopic Classification Scheme for caustic mucosal injury

Grade	Definition
0	Normal examination
1	Edema and hyperemia of the mucosa
2A	Superficial ulcerations, exudates, whitish membranes, blisters, erosions, hemorrhages and friability
2B	Grade 2A plus deep discrete or circumferential ulceration
3A	Small, scattered areas of necrosis
3B	Extensive necrosis

Adapted from reference 17, with permission from the American Society for Gastrointestinal Endoscopy

Two retrospective case reviews (15,19) described caustic ingestion in children and adults, and showed that symptoms alone were not predictors of endoscopic injury. Furthermore, two other reports (17,18) have demonstrated that the degree of oropharyngeal injury does not predict the severity of the esophagogastric injury related to the caustic ingestion. This has led to the practice of early endoscopy in the evaluation of cases of caustic ingestion such that early complications such as bleeding and perforation, or late complications of stricture formation can be anticipated. Table 1 outlines a classification system developed by Zargar et al (17). Generally, patients with grade 0, 1 or 2A injury escape with no sequelae. Patients with grade 2B to 3 injury have a high rate of developing strictures. Patients with grade 3 injury are at risk for both systemic complications requiring intensive care unit admission and local complications of bleeding and perforation. Most deaths occur in patients with grade 3 injury (16-18).

Endoscopic antegrade dilation has been described previously in similar clinical settings (20-22). However, when such information is provided, the timing of such dilations appears to be later than performed in the present case (23,24). We undertook an early dilation program due to the intense fibrotic reaction resulting in significant early stricturing. If we had elected to wait longer before dilation, it is plausible that access through the stricture in an antegrade fashion may not have been safe, or possible. The interval between dilations was decided based on literature showing that these strictures can be more difficult to manage (24), and also based on our initial experience and judgment of this particular patient's response to dilation. We believe that with strictures that still contain active inflammation and ulceration, an aggressive dilation strategy is necessary to prevent them from becoming progressively more obstructive between endoscopies.

One report (24) comparing the treatment of corrosive and peptic strictures described a high initial success rate with dilation of corrosive strictures (93.6%). However, the authors also demonstrated increased recurrence rates (94.4% versus 54.6%, at 60 months) with corrosive strictures compared with peptic strictures, respectively. However, the majority of these recurrences occurred early (ie, within the first 18 months), and the rate of symptomatic recurrence normalized by 36 months of follow-up examinations and treatment. The observed rate of perforation for dilation of corrosive strictures was 0.8%, compared with 0.3% for peptic strictures.

The present case invites discussion on steroid use to reduce the severity and persistence of esophageal and/or pyloric strictures. A controlled trial of intravenous prednisolone with

step-down to oral prednisone in children has not shown a difference in stricture formation rates (23). Intralesional steroid injections have been described in case reports and small case series (20-22,25-30). However, this intervention is most often described in peptic strictures, with only a few cases involving caustic injury (20,21,26,30). In the present case, the authors were reluctant to use intralesional steroids in the setting of such severe and persistently active mucosal injury. Only one previous report (26) commented on whether active ulceration was present when this therapy was employed. The patient described in the present report continued to have active ulceration at the distal stricture site until 12 weeks postingestion. At this point, the stricture was managed much more easily, with an increased interval between dilations. The patient was successfully managed without steroids, but the possibility exists that their use may have reduced the total number of dilations required.

In spite of obvious gastric retention, evident by copious fluid in the stomach during endoscopy, the patient never experienced symptoms of gastric stasis. The pyloric stricture responded well to balloon dilation, requiring four dilations until patency was maintained. A progression to larger balloons was not undertaken due to the lack of symptoms and the resolution of endoscopically evident gastric retention. Such success has been reported previously, but only in a small number of cases (20,26).

The role of acid suppression in this setting is unclear. Previous authors have documented initial achlorhydria postcorrosive gastric injury, sometimes with a return of normal gastric acid secretion (6,13). The role of acid in the persistent ulceration in the esophagus and antrum was questioned, and the patient was empirically treated with proton pump inhibitor therapy. The gastric body was biopsied approximately 12 weeks postingestion, and parietal cells were abundant, suggesting that achlorhydria was not part of the sequelae of alkali ingestion in this case.

Although, in the past, many severe caustic ingestion injuries required laparotomy for the assessment and potential resection of necrosis, the case described was managed conservatively with observation, total parenteral nutrition and endoscopy, in consultation with an esophageal surgeon. The authors advocate an individualized approach to each patient, taking into account the extent and type of injury and the patient's clinical condition.

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