Nonsurgical management of severe esophageal and gastric injury following alkali ingestion

Ramy D Abaskharoun MD FRCPC, William T Depew BSc MD FRCPC, Lawrence C Hookey BSc MD FRCPC

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 (1,2,6).

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 dilatation 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...
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 post-ingestion. 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 post-ingestion 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 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 post-ingestion. B Endoscopic image of a distorted antrum 10 days post-ingestion. Active ulceration and a narrowed but patent pylorus are demonstrated.
Can J Gastroenterol Vol 21 No 11 November 2007

bleeding, perforation, fistula formation or death (15-18).

resulting from an intense fibrotic reaction that may occur in
the weeks following ingestion (4). The natural history of such injuries, if perforation is avoided, is ulceration, frequently followed by strictures
ingestion (2,4). The natural history of such injuries, if perforation is avoided, is ulceration, frequently followed by strictures

Alkali ingestion results in liquefactive necrosis, which is often
to be predictive of complications of caustic ingestion such as
bleeding and perforation, or late complications of stricture
formation can be anticipated. Table 1 outlines a classification sys-
muscular injury. Two retrospective case reviews (15,19) described caustic in-
gestion 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
oro-pharyngeal 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 sys-

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 con-
trast 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 fur-
ther 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

Six further endoscopies were performed on him as an outpa-
tient over the next three months. Active ulceration and inflam-
amination 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 nar-
rrowed distally. The antrum was contracted and the barium
flowed easily into the duodenum (Figure 3). The patient was fol-
lowed for 18 months after his ingestion, and experienced no dys-
phagia 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 perfora-
tion 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

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

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

Two retrospective case reviews (15,19) described caustic inges-
tion 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
oro-pharyngeal 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 sys-

The present case invites discussion on steroid use to reduce
the severity and persistence of esophageal and/or pyloric stric-
tures. A controlled trial of intravenous prednisolone with
step-down to oral prednisone in children has not shown a difference in stricture formation rates (23). Intraluminal steroid injections have been 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 intraluminal steroids in the setting of such severe and persistent 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 postgestation. 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 and active stricture was questioned, and the patient was empirically treated with proton pump inhibitor therapy. The gastric body was biopsied approximately 12 weeks postgestation, 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.

REFERENCES