Necrosis of the Stomach Secondary to Ingestion of Corrosive Agents*

Report of Three Cases Requiring Total Gastrectomy

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The ingestion of corrosive agents frequently produces adverse effect on the esophagus and the stomach or on both. Agents of alkaline pH usually result in esophageal injury, and agents of acid pH often cause gastric damage. Squamous epithelium lining the esophagus is sensitive to alkaline agents; however, alkaline agents upon reaching the stomach are rapidly neutralized by the gastric acidity of the stomach. Conversely, esophageal mucosa is resistant to acid agents, which in turn produce severe inflammatory changes throughout the gastric wall. Corrosive agents regardless of their pH acidity or alkalinity can be destructive in some instances to both esophagus and stomach. Rarely does one note necrosis of the entire stomach secondary to the swallowing of corrosive agents. We present three such cases requiring total gastrectomy, seen by us over a one-month period at Cook County Hospital, Chicago.

Corrosive agents, when swallowed, frequently have an adverse effect on the esophagus and stomach. Although lye ingestion usually results in injury to the esophagus, we have noted gangrene of the stomach following ingestion of lye, as well as of acid.

In patients with a history of lye ingestion accompanied by abdominal findings on admission, one should be alert to the possibility of perforation of the esophagus or necrosis of the stomach, either of which requires exploration and definitive surgery.

Herrington,1 Marks and co-workers,2 and Boikan and Singer3 indicated that lye is the most commonly ingested alkaline corrosive agent producing esophageal stricture and hydrochloric acid is the most frequent agent producing gastric cicatrizing effects. The squamous epithelium lining the esophagus is usually sensitive to alkaline agents, but the alkali on reaching the level of the stomach, is almost always rapidly neutralized by the acidity present. Conversely the esophageal mucosa is extremely resistant to acid agents, which in turn produce severe inflammatory changes throughout the thickness of the gastric wall, with minimal damage inflicted to the antral and pyloric regions. Following ingestion of a corrosive agent of acid pH with resultant cicatrizing stenosis of the stomach, it has been estimated that in only approximately 20 percent of such cases is there any degree of esophageal involvement. When present, this is usually limited to a superficial mucosal reaction. With the ingestion of a corrosive alkaline pH, the stomach may also be involved in a small percentage of cases, and in some instances this reaction may be quite severe.

Harris,4 McLanahan,5 and Bolstad,6 have described pyloric stenosis after ingestion of alkaline corrosives and Bolstad7 made a careful search of the American literature over a 25-year period ending in 1948, and revealed only one case of esophageal stricture combined with pyloric stricture which eventually led to obstruction. Strange and associates,† presented a case of severe cicatricial injury of the stomach without esophageal involvement in a patient following the ingestion of chlorax (5.25 percent sodium hypochlorite by weight in
water, with a pH of 10.8). They reviewed the literature on reported injuries resulting from the ingestion of this substance and found only one case (a mild esophageal injury of a diffuse erythematous nature not resulting in a stricture), which was included in Uhde's report of 34 cases of chemical burns of the esophagus.

Citron and colleagues have outlined a group of corrosives frequently found in the house as well as common substances reported to have produced corrosive changes.

Herrington has indicated that the degree of damage inflicted to the stomach depends upon the amount of caustic ingested, its strength, the time length of contact with the stomach, whether or not food is present in the stomach at the time, and the relative tonicity of the pyloric sphincter. In most cases maximum damage takes place along the magenstrasse and in the antral and pyloric regions.

Berry and colleagues reported a case of necrosis of the entire stomach secondary to ingestion of a corrosive acid which was successfully treated by total gastrectomy. Middlekamp and co-workers give a classification for burns of the esophagus: first degree, hyperemia of the mucosa with superficial desquamation of the epithelium; second degree, superficial blisters, ulcers, hyperemia and patchy exudate on the mucosa; and third degree, hypoemia areas with total loss of esophageal granulation tissue. They pointed out an observation which has not been previously emphasized, that respiratory distress indicative of laryngeal involvement was frequently associated with third degree burns of the esophagus.

It is extremely rare that corrosive injury to the stomach is severe enough to warrant total gastrectomy. For this reason we were prompted to present three such cases seen by us in a one-month period at Cook County Hospital in Chicago.

CASE REPORTS

CASE 1

This 28-year-old well-developed, well-nourished Negro woman was admitted to Cook County Hospital on August 29, 1968, with a 24-hour history of lye ingestion (suicidal attempt). On admission the patient was noted to have severe burns of the mouth, tongue and pharynx. The abdomen was rigid, distended and tender throughout, with hypoactive bowel sounds, and the patient was complaining of severe chest pain.

Complete blood count, urinalysis, nonprotein nitrogen, blood glucose, serum electrolytes and chest x-ray film were within normal limits. X-ray examination of the abdomen revealed findings compatible with a reflex ileus.

After hydration, the patient's abdomen was explored; marked necrosis of the stomach was found. Total gastrectomy, partial duodenotomy, feeding jejunostomy and tracheostomy were done.

The mediastinum was markedly edematous and the muscularis of the entire esophagus was completely denuded with the mucosa protruding just beneath the pleura. Multiple friable perforations of the esophagus were noted.

Immediately following surgery the patient had cardiac arrest and despite vigorous resuscitative measures did not recover.

Examination of the removed stomach revealed measurements of 22 cm along the greater curvature and 8 cm along the lesser. The serosa was red in color; the mucosa was deep red with areas of necrosis.

Microscopic multiple sections showed smooth muscles with extensive areas of necrosis, engorged vessels, fibrin and neutrophilic cell infiltration. Pathologic diagnosis of the gastrectomy specimen revealed marked necrosis of gastric wall (Fig 1 and 2).

CASE 2

This 30-year-old well-developed, well-nourished Negro man was admitted to Cook County Hospital on September 16, 1968. The patient stated that he had been vomiting blood for three hours. He was a heavy drinker (alcohol) and gave a
history of peptic ulcer disease.

When examined the patient was in no acute distress with vital signs within normal limits. Nasogastric tube was productive of bright red blood; stool was 4+ positive for occult blood; and hematocrit ranged between 44 to 46. Since the patient continued to bleed, he was taken to the operating room on September 20, where a pyloroplasty and vagotomy were done. At surgery the liver was adherent to the lesser curvature of the stomach and the rugal folds were absent. The stomach was edematous, hyperemic and friable, and contained organized clot. A frozen section of gastric wall was negative for malignancy. A clinical diagnosis of atrophic toxic gastritis was considered.

For three days postoperatively, the patient continued to run a febrile course of 103°F with right upper quadrant guarding and tenderness. On September 23, a diagnosis of "gastric disruption (Bshelf) with intraperitoneal spill" was made and confirmed by giving the patient 5 ml of methylene blue by mouth. The patient was hydrated, taken to the operating room and reexplored. A diffuse purulent gangrene was noted throughout the entire stomach and disruption of the pyloroplasty was present. The distal esophagus appeared normal. A total gastrectomy, splenectomy, tube duodenostomy, feeding Wetzel jejunostomy, and cervical esophagostomy were done.

The removed stomach revealed greater curvature 24 cm in length, 12 cm proximal circumference, and 5 cm distal circumference. The mucosa showed multiple dark-brown necrotic areas of hemorrhage and multiple pseudopolypoid structures, the largest measuring up to 1 cm.
On arrival the patient vomited black material and noted a burning sensation in the epigastrium and in the throat. Speaking was painful. Because of the development of abdominal pain and tenderness, the patient was taken to the operating room on September 24 for exploration. The serosa of the stomach was gangrenous along the greater curvature from fundus to antrum; on opening the stomach, the mucosa was noted to be necrotic from esophagus to pylorus. The duodenum appeared perfectly normal, but the mucosa of the distal esophagus was gangrenous.

Total gastrectomy and a feeding and decompression jejunostomy were done, along with a double-barrel cervical esophagostomy. The cervical esophagus revealed a gray-green discoloration.

The excised stomach measured 28 cm along its greater curvature and 11 cm along the lesser curvature. The mucosa was dark-brown in color with areas of ulceration. The folds were prominent and the wall edematous.

Microscopic sections of stomach showed extensive areas of necrosis with ghost-like appearance of the glands, engorged vessels, hemorrhage, fibrin deposits and marked neutrophilic cell infiltration of the submucosa, muscularis and serosa (Fig 5 and 6).

Patient was treated with corticosteroids and multiple postoperative esophageal dilatations and esophagosopies were done. A massive amount of greenish pus poured from the distal esophagostoma, and the lower end of the esophagus with friable foul smelling necrotic material observed on inspection through a pediatric esophagoscope.

It became readily apparent that the thoracic esophagus could not be used for reconstruction purposes. Laryngoscopy and retrograde esophagoscopy of the proximal left cervical esophagostomy revealed a scarred contracted friable segment, but it was felt that the proximal segment of the esophagostoma near the pharynx could be used for anastomosis.

On December 5, the patient underwent a subternal colon bypass (using the left colon). The patient tolerated the procedure well and was sent to the recovery room in good condition. Microscopic section of tissue from the proximal esophagus which had been submitted for study at the time of surgery revealed bundles of skeletal muscle fibers intermingled with intervening fibrous connective tissue. The latter contained focal aggregates of chronic inflammatory cells, with some foreign body type of giant cells and amorphous material. The diagnosis was cicatrization of skeletal muscle with foreign body reaction. On the fourth postoperative day the cervical anastomosis was noted to leak.

On February 6, 1969, the patient underwent an esophagoplasty. There was no continuity whatsoever of the previous anastomosis. A split thickness skin graft was then taken from the lateral surface of the left deltoid area, a celestine tube was painted with bees dermatome glue and skin graft applied (deepside out) to the celestine tube. The tube was then inserted into the lumen of the pharynx and colon. Anastomosis was done using 5-0 chronic interrupted sutures. The deep neck tissues were then approximated with 4-0 chromic and skin edges approximated with 5-0 nylon.

**DISCUSSION**

Three adult cases of extensive necrosis of the entire stomach secondary to the ingestion of corrosive agents which required total gastrectomy, are presented.

At the time of gastrectomy a feeding tube jejunostomy should be done to maintain the nutritional status of the patient until such time that a procedure to establish gastrointestinal continuity is done. Supportive measures include sedation, antibiotics, whole blood, parenteral fluids and electrolyte replacement as well as psychiatric support and therapy.

If the esophagus is not perforated, it is not necessary to drain or remove the esophagus as this only adds to the surgical risk. A cervical esophagostomy (double-barrel) is done at the time of initial surgery to divert sputum from the damaged esophagus.

The final and definitive procedures are dependent upon the degree and extent of cicatricial injury to the esophagus.

At a later date a second stage colon or small bowel bypass (preferable colon bypass) may be done as a definitive procedure to reestablish gastrointestinal continuity.

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