

A middle age addicted man with caustic stomach

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ABSTRACT

Background: The term caustic generally refers to alkaline and the term corrosive generally refers to acidic agents' injury; however, in medical literature caustic is frequently a term applied to both substances. Ingested alkali typically damage the esophagus more than stomach or duodenum, whereas acids usually cause more severe gastric injury. Since esophagus has a slightly alkaline pH, its epithelium is more resistant to acids, so that only 6 to 20% of those who ingest these substances present lesions in this organ.

Case : A middle-aged addicted man who drunk hydrochloric acid accidentally had extensive necrosis of the stomach with remarkable sparing of the esophagus on second look exploration. A total gastrectomy with a Roux-en-Y esophago-jejunostomy with feeding jejunostomy was performed.

Conclusion : In caustic GI injury, patients who are operated on and found to have no evidence of extensive esophago-gastric necrosis, a biopsy of the posterior gastric wall should be performed to exclude occult injury. If histologically there is a question of viability, a second look operation should be performed within 36 hours.

Keywords: Acids, Caustics, Chemical Burns, Esophagus, Stomach Diseases/Chemically Induced.

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INTRODUCTION

Ingestion of caustic substances can cause severe injury to the esophagus and stomach and the severity and extent of damage depends on factors such as corrosive properties of the ingested substances (acid or alkali, amount, concentration, physical form of the agent, and duration of contact with the mucosa) [1].

Children frequently expectorate most of the caustic agents before swallowing; in contrast suicidal patients often ingest larger amounts of these substances compared to those who accidentally swallow them. The most common substances are strong alkali [1,2]. The term "lye" implies substances that contain sodium or potassium hydroxide. Button batteries contain highly concentrated solutions, and damage occurs from alkali, local electrical discharge, and pressure necrosis from the foreign body. When a button battery is swallowed, burn occurs within hours and perforation within 4 Hours. Urgent endoscopy is indicated before some serious complication such as esophago-tracheal or esophago-aortic fistula develops [3].

Alkali ingestion injuries cause liquefaction necrosis and may produce deep burns that are staged similar to cutaneous burns (firstdegree=mucosal; second-degree=transmucosal; third-degree=transmural). Caustic ingestion can result in esophageal strictures in 5 to 20% of patients.

Esophageal strictures can be detected 1 to 3 months following the ingestion. The pattern of stricturing is variable but typically is a long, smooth, tapering stricture of the cervical and /or upper thoracic esophagus [4]. Since esophagus has a slightly alkaline pH, its epithelium is more resistant to acids, and only 6 to 20% of patients show lesions in this organ [5]. Both strong alkali and strong acids produce liquefaction or coagulation necrosis. Alkali dissolve tissues and therefore penetrate more deeply, while acids cause coagulative necrosis that limits their penetration [1,2].

The lesions caused by lye injury occur in three phases. First is coagulation of intracellular proteins which results in cell necrosis. Second is ulceration and granulation phase and third is the

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icatratization and scarring phase [4]. The food in the stomach tends to provide a protective effect against injury [6].

Due to lack of immediate symptoms, alkali ingestion is more common than acid ingestion. Acids cause an immediate burning sensation in the mouth, whereas alkali does not [1,2].

Upper GI endoscopy should be performed during the first 24 hours after ingestion in order to evaluate the extent of damage, establish prognosis and therapy [7]. Patients who ingest caustic substances can have a spectrum of presentations from asymptomatic to obtundation with evidence of visceral perforation [1,2,6]. There may be airway compromise with need for emergent cricothyroidotomy or tracheostomy. Up to 20% of patients with no oropharyngeal burns, might still have esophageal injury [4-6]. Laryngitis, hoarseness, or stridor indicate laryngeal injury and impending airway compromise. Chest pain, hypotension, peritonitis, and fever are strong indicators of visceral perforation [7].

Caustic ingestions result in esophageal strictures in 5 to 20% of patients [4]. Barium esophagram is the mainstay in the diagnosis of strictures secondary to caustic ingestion. Since injury is random and depends on the agent and the extent of contact, irregular eccentric strictures with areas of sacculation are common. In the most severe forms, caustic strictures can involve the entire thoracic esophagus, which would appear thread-like on esophagram [4].

The stomach should also be evaluated for the squeals of caustic injury. A repeat endoscopy and barium esophagram are recommended in follow-up at intervals of 1, 2 and 8 months, at which time 60%, 80% and nearly 100% of strictures will have been developed, respectively [1].

The presence of fever is strongly correlated with the presence of an esophageal lesion and

serial chest and abdominal radiographs are indicated to follow patients with questionable chest and abdominal exams [1].

Perforation usually occurs as a result of inflammation, vascular thrombosis, ulceration and necrosis of the esophageal wall 3 to 5 days after the ingestion [8]. Adequate pain relief with

narcotics and broad spectrum antibiotics (third generation cephalosporins) for grade III burns or perforations are recommended [1,2]. Because all corrosive agents are extremely hygroscopic, the caustic substances cling to the esophageal epithelium. Subsequent strictures occur at the intrinsically narrower areas of the esophagus, i.e: the crico-pharyngeus, middle esophagus, and gastroesophageal junction [2]. Severe and recalcitrant strictures may require esophageal replacement with colon or jejunum interpositional grafts, while less severe strictures are managed with dilation. Authorities suggest a baseline barium esophagogram 3 to 4 weeks after ingestion to establish a baseline for future stricture formation [9]. When esophageal replacement is necessary, most authors advocate resection of the esophagus. The excluded esophagus may become a posterior mediastinal mucocele that causes respiratory distress due to tracheo-bronchial compression. It is always preferable to place the esophageal substitute in the posterior mediastinum in the original esophageal bed [10].

For patients in respiratory distress, laryngoscopy should be performed in order to evaluate the need for tracheostomy [1-4]. In cases with epiglottitis or larynx edema, endotracheal intubation is contraindicated and tracheostomy should be performed for airway control [9].

If a patient presents within the first hour of ingestion, neutralization could be attempted [1,2]. Lye or other alkali can be neutralized with half strength vinegar, lemon juice or orange juice [1,2]. Acids can be neutralized with milk, egg white or antacids. Oral feeding can be started when the dysphagia of the initial phase has regressed [1] or when a patient can swallow saliva painlessly [2].

Currently, some recommend that the dilations should be started at the first day after the injury with the aim of preserving the esophageal lumen by removing adhesions that occur in the injured segments, although this approach is still controversial [1,10]. Undilatable strictures or strictures that recur rapidly or progress after dilatation would require esophageal resection and replacement [4].

The presence of air in the esophageal wall is a sign of muscle necrosis and impending perforation and is a strong indication for esophagectomy.

In patients who are operated on and found to have no evidence of extensive esophago-gastric necrosis, a biopsy of the posterior gastric wall should be performed to exclude occult injury. If histologically there is a question of viability, a second look operation should be performed within 36 hours. Whenever a stent is inserted, it should be kept in position for 21 days and removed after a satisfactory barium esophagogram [1,2,11].

If dysphagia does not develop within 8 months, it is unlikely for a stricture to occur[1]. Dilations are performed daily for 2 to 3 weeks then every other day for 2 to 3 weeks, followed by weekly sessions for 3 months [2, 5, 8].

If endoscopic dilation fails to reestablish an adequate lumen (40 French), surgical intervention is necessary. Most deaths are due to the sequels of perforation and Mediastinitis [10].

Late sequelae are esophageal stricture and esophageal squamous cell carcinoma (SCC) [11]. Caustic strictures can progress to SCC, however this typically occurs 30 or more years after the injury [4].

CASE PRESENTATION

A 43 year old opium-addicted male was admitted in the Emergency Department of Valiasr Hospital of Arak, Iran following accidental ingestion of hydrochloric acid about one hour before admission.

He was pale and emaciated with a blood pressure of 85/60 mmHg, pulse rate = 90/min, respiratory rate = 20, temperature = 37 °C. It was determined that the patient was not at significant risk of airway compromise and chest x-ray was normal; therefore, we decided to put him under close observation for any signs of deterioration. Total parenteral nutrition was initiated and the patient remained nil per os (NPO). There was no respiratory distress or subcutaneous emphysema. Chest examination was unremarkable but he had abdominal tenderness, rebound tenderness, and guarding. CBC demonstrated leukocytosis (WBCs=17800); however CxR and abdominal

x-ray were normal. An endoscopy was performed within 12 hours post-admission that showed bullae formation in the esophagus and upper stomach. There was some bloody fluid in the stomach but no active bleeding, the first and second parts of duodenum were normal. The endoscopic diagnosis was caustic agent esophagitis and gastritis grade II.

At exploratory laparotomy, there was no visceral perforation, but *multiple foci of patchy discoloration of gastric wall* were found. A biopsy of posterior gastric lumen was conducted, at the second look laparotomy which was executed 48 hours later, due to full thick necrosis of gastric wall, total gastrectomy, Roux-en-Y esophageo-jejunostomy anastomosis, with feeding jejunostomy was performed.

Esophagogram on the 11th day after admission showed that contrast media entered into one of the bronchial branches of the left lower lobe of the lung. A repeat endoscopy, performed on the 17th day, demonstrated no fistula and regular diet high in calorie and protein was started orally. Esophagoscopy on the 24th day after the admission was also normal.

The patient was discharged on oral feeding after two months and was followed and visited for about a year.

DISCUSSION

This article describes a case of caustic injury to the upper gastro-intestinal tract. Ingestion of corrosive substances always presents diagnostic and therapeutic challenges to physicians [1-4].

Individual case studies or case series have led to the suggestion that acids cause maximal damage to the stomach and relatively minor damage to the esophagus because of the rapid transit [12].

Our patient's endoscopy showed bullae formation in esophagus and upper stomach, bloody fluid in the stomach without active bleeding, caustic esophagitis and gastritis grade II. It is suggested that all patients with 2nd degree or greater corrosive burns should be administered parenteral broad spectrum antibiotics. Intravenous proton pump inhibitors are also widely used to minimize the insult to

the injured gastric mucosa [1-3,6]. Therefore, we administered a third generation cephalosporin and pantoprazole to the patient.

In view of the high probability of slow but relentless progression of transmural necrosis, there should be a low threshold for consideration of laparotomy at the earliest suspicion [2,6,8].

Because of the poor prognosis with these patients, two days later a second look exploration was performed, gastrotomy showed extensive mucosal and transmural necrosis of the stomach and immediate total gastrectomy was performed.

If the esophagus is relatively spared with moderate injury to the stomach, the patient is kept on regular observation to monitor the progression of the gastric burn and if the stomach appears soft and necrotic, as in this patient, a gastrotomy is performed and the extent of mucosal and transmural necrosis is assessed before planning resection. The resection is usually either distal or total gastrectomy. The decision for the line of section should be made after gastrotomy, since mucosal necrosis is more extensive than what appears from the serosal side [2,7]. The more severe injury to stomach than that of the esophagus can be explained on the basis of rapid transit through esophagus, reservoir function of stomach that allows accumulation of large volume of acids in stomach, reflex pylorospasm which increases the contact time between the acid and the stomach mucosa. Isolated acid corrosive injury to stomach though rare, has been reported in the literature [13].

Our knowledge about the best management approach in caustic or corrosive injury of gastrointestinal tract is limited. Each patient must be evaluated individually as the clinical picture varies widely and signs and symptoms alone are unreliable guides to the extent of the injury [14].

CONCLUSION

Patients who ingest caustic substances can present in any condition, from asymptomatic to frankly toxic with evidence of visceral perforation, mediastinitis, and even death. Alkali ingestion, because of its lack of immediate symptoms, is more common than

acid ingestion. Alkali typically damage the esophagus more than stomach or duodenum, whereas acids cause more severe gastric injury. Our case shows that acid agents can cause severe gastric injury and full necrosis, with minor or no injury to the esophagus without the need for esophagectomy.

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