Aortoesophagal Fistula Due to Caustic Ingestion

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Purpose. Caustic agent ingestion may produce corrosive lesions that can extend beyond adjacent organs. We report three cases of aortoesophageal fistulas (AEF) after caustic ingestion that were diagnosed by autopsy.

Results. AEF is a fatal complication that should be suspected in any patient with caustic ingestion who presents with gastrointestinal bleeding. A high index of suspicion, early recognition by gastrointestinal endoscopy, computed tomography scan, and aortography are important to improve the outcome.

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Introduction

Aortoesophageal fistulas (AEFs) constitute less than 10% of all aorto-enteric communications. Primary AEFs arise from communication between the native aorta and esophagus, whereas secondary AEFs are usually the consequence of communication between a suture line of a vascular graft and the esophagus. The development of AEFs after corrosive ingestion is an uncommon cause of AEFs that has been reported.

We report three cases of primary AEFs caused by corrosive ingestion with fatal outcomes.

Report

Case 1

A 37-year-old man was admitted to our hospital with a history of ingesting about three tablespoons of chloridric acid with suicidal intent. He was referred 12 hours after ingestion of caustic agents. He had no history of previous surgery. He had a history of major depression for six to seven years. On admission, he complained of perioral irritation and soreness, nausea, vomiting, dyspnea, odynophagia, and dysphagia. Vital signs were normal, and there was no clinical finding relevant to systemic toxicity. Physical examination revealed signs of burns and mucosa sludge in his oral cavity. He had respiratory stridor. On abdominal examination, there was no sign of an acute abdomen. At the time of admission, hemoglobin, white blood cell, and platelet counts were 12 mg/dl, 11200/ml, and 176000/ml, respectively. Upper gastrointestinal endoscopy showed diffuse mucosal erythema, ulceration, and patchy necrosis consistent with grade II and III of modified Di Costanzo classification. The patient was a candidate for surgical intervention, but due to the patient not giving informed consent, he was managed conservatively.

Five days after admission, mild episodes of hematemesis occurred, which were spontaneously controlled. On the 11th day of admission, after massive hematemesis, cardiac arrest occurred. Cardiopulmonary resuscitation was performed, but was not successful. Autopsy revealed a fistula between the middle third of the esophagus and thoracic aorta (Fig. 1).
Case 2

A 40-year-old man was admitted to our hospital complaining of upper gastrointestinal bleeding. His brother mentioned that he had ingested a pocket (about 30 grams) of arsenic-based depilatory substances, which was dissolved in water. The patient was hemodynamically unstable at admission, which was 36 hours after intoxication. On physical examination, there were ulcerations and sludge in the oral cavity, compatible with grade III of modified Di Costanzo classification. We tried to resuscitate the patient, but unfortunately he died before performing any diagnostic and therapeutic interventions. At autopsy, his esophagus and adjacent organs were necrotic. The origin of bleeding was the descending aorta that was fused to the esophagus.

Case 3

A 67-year-old man presented to our hospital with the chief complaint of hematemesis two months after ingestion of sulfuric acid. According to his medical history, there was a grade II esophageal lesion at his first admission due to caustic ingestion. During that admission, he had undergone laparatomy, gasterotomy, posterior gastric wall biopsy, feeding jejunostomy, and intraluminal esophageal stenting. Twenty one days later, the patient was discharged after esophageal stent removal and was in good condition and eating.

Two months later, he was admitted again for hematemesis. The patient was hemodynamically stable. Laboratory tests including Hb, WBC, and Plt were 9.5 g/dl, 13000/ml and 264000/ml, respectively. An upper GI endoscopy demonstrated linear ulcerations in the lower third of the esophagus. The patient died following a severe episode of hematemesis on the first night of hospitalization. The autopsy revealed an aortoesophageal fistula beyond the erosions seen in the endoscopy.

Discussion

Common causes of AEF are rupture of a thoracic aortic aneurysm, aortic dissection, foreign bodies, esophageal cancer, and thoracic surgery. Primary AEF has also been reported as a rare consequence of corrosive esophageal injury. Over six years we have investigated about 1260 patients with caustic ingestion. We identified only 3 AEF, but all had fatal outcomes.

Most aortoesophageal fistulas are diagnosed post-mortem after a massive exsanguinating hemorrhage. Reports of ante-mortem diagnosis and successful salvage of patients with aortoesophageal fistula are rare.

The classic symptoms of AEFs were described by Chiari in 1914. This triad consists of dysphagia or mid-thoracic pain, followed by a sentinel or herald bleed, and then an exsanguinating haemorrhage after a variable asymptomatic period. Presentation with the classic Chiari’s triad is usually diagnostic. However, most cases are diagnosed after death or at surgery. Sometimes when the condition is suspected after a herald bleed, investigations may help in confirming the diagnosis. Some patients may have recurrent small episodes of bleeding rather than a single sentinel bleed.

The first step in the management of patients with corrosive injury is to maintain airway patency and hemodynamic stability. In patients presenting with apnea and stridor, orotracheal intubation should be performed because of the possible development of edema. Endoscopy should be done in every stable patient in order to determine the severity of the lesion. Further treatment may be required related to the severity (endoscopic grade) and available complications.

When AEF is suspected an endoscopy, computed tomography scan, and aortography are useful for diagnosis and for identifying the site of the fistula.

Although some reports of aortic stenting and emboilizations are available for treatment of AEF for other reasons in caustic ingestion emergency surgery is the treatment of choice. Caustic injury leads to AEF and esophageal necrosis. Aortic repair and esophagectomy with delayed esophageal reconstruction are essential principles in the management of AEF. Early repair of AEF appears to result in better outcomes.
To our knowledge in English-language literature, there exists only one report of corrosive-induced aortoesophageal fistula managed successfully by combined surgical and endovascular treatment by means of a stent graft. This case was encountered in association with an extensive corrosive lesion.

We present these cases to increase awareness of AEF, which although rare and usually fatal, does occur in patients ingesting corrosive agents and should be suspected in any of these patients who present with gastrointestinal bleeding. Early recognition, a high index of suspicion, and improved critical care and emergency services before a fatal hemorrhage are very important and can reduce the rate of death in patients ingesting corrosive agents.

References


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