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Aortoesophageal Fistula Secondary to Reflux Esophagitis

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Key Words

Aorta · Esophagitis · Esophageal fistula

Abstract

Aortoenteric fistula is a rare cause of massive upper gastrointestinal bleeding and is in the overwhelming majority of cases due to erosion of a suture line of a prosthetic vascular graft into the bowel. We report the case of a massive fatal gastrointestinal hemorrhage from an aortoenteric fistula secondary to erosion from reflux esophagitis. Proper management requires expedient radiographic and endoscopic evaluation, and even with appropriate management mortality remains extremely high.

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Introduction

Life-threatening upper gastrointestinal hemorrhage is most often caused by esophageal varices or gastroduodenal ulcers. Aortoenteric fistulas are very rarely the etiology of such bleeding. An early review of fatal upper gastrointestinal hemorrhage noted that 6.1% of the deaths were due to aortoenteric fistulas [1]. The duodenum was the most common segment of bowel involved, with only one-fifth of cases involving the esophagus.

The most frequent cause of a primary aortoesophageal fistula is a ruptured thoracic aneurysm, accounting for almost 75% of cases [2]. The next most common etiology is an esophageal foreign body erosion into the aorta, such as a fish bone perforation [3]. Other causes include pseudoaneurysm formation after repair of thoracic aortic aneurysm [4], malignant tumors of the esophagus or bronchus, and iatrogenic perforation of the esophagus leading to fistula formation. Chronic ulcerative esophagitis is an extremely rare cause of aortoesophageal fistula and represents only 0.5% of reported cases [5]. We report the case of massive upper gastrointestinal bleeding from an aortoesophageal fistula secondary to chronic reflux esophagitis.

Case Report

A 62-year-old man was admitted to the hospital for vomiting and dehydration. Initial surgical consultation was obtained to evaluate a reducible right inguinal hernia. His past medical history was remarkable for schizoaffective disorder and psychogenic polydipsia. Chest radiographs revealed a right lower lobe lung infiltrate. Despite a 10-day course of intravenous antibiotics he showed a persistent leukocytosis. Computed tomography of his chest was obtained to further evaluate his pneumonia. This study demonstrated a hiatus hernia, bibasilar atelectasis in his lungs, and a small area of contrast extravasation from the anterior surface of his aorta (fig. 1).

The patient improved clinically except for a slowly rising leukocyte count. Eleven days after admission the patient collapsed in his room, and was found to have a blood pressure of 80/30 mm Hg. After transfer to the intensive care unit and fluid resuscitation his blood pressure and heart rate normalized. Nasogastric suctioning was initiated and yielded the return of frank blood. During placement of central venous and arterial access lines the patient began to have pulsatile bleeding from his mouth.

At this point the patient was taken to the operating room for emergent abdominal exploration for what was expected to be a briskly bleeding peptic ulcer. Upon entry to the abdomen a massively distended stomach was encountered. A longitudinal gastrotomy was performed and 3 l of at first old, then fresh blood was evacuated. This revealed an area of pulsatile bleeding on the pos-

Fig. 1. Computed tomogram of the chest with intravenous contrast. The large arrow indicates the gastroesophageal junction. The small arrow shows extravasation of blood from the aorta.

terior surface of the stomach above the crura of the diaphragm. Upon digital palpation a 1-cm defect was palpable between the stomach and aorta.

While the aortic defect was digitally occluded, a left thoracotomy was performed and the midthoracic aorta cross-clamped. The aortic wall was debrided and a polytetrafluoroethylene patch sewn in place. After the clamp was removed no further bleeding was noted. The patient, however, developed intractable ventricular arrhythmias probably secondary to prolonged hypotension and expired in the operating room.

The pathology report of the stomach and esophagus revealed an ulcerative candida esophagitis and chronic gastritis with no evidence of malignancy.

Discussion

An aortoesophageal fistula is a connection between the aorta and the esophagus, which allows high-pressure blood from the aorta to pump into the low-pressure gastrointestinal tract. This type of fistula is a very rare cause of upper gastrointestinal hemorrhage and is almost uniformly fatal. Since its initial description in 1818 by Dubrueil [6], there has been little improvement in patient survival.

Chiari [7] first described the 'aortoesophageal syndrome' as a midthoracic pain followed by a sentinel hemorrhage, and exsanguination hours to days later. This constellation of findings was named 'Chiari's triad'. A 30-year review of aortoesophageal fistulas by Hollander and Quick [8] found midthoracic pain in 59% of patients, dysphagia in 45%, and sentinel hemorrhage in 65%. In toto, only 45% of the patients met Chiari's triad.

To assure any chance of survival, the diagnosis must be made rapidly after a sentinel hemorrhage. Only 25% of patients re-bleed within 6 h; thus there is often inadequate time for diagnosis. The bright red bleeding of the oxygenated arterial blood in this condition can help distinguish it from the hemorrhage of esophageal varices – the most common cause of massive upper gastrointestinal bleeding. The clinical manifestation of pulsatile oral hemorrhage was demonstrated in our patient. Chest radiographs can suggest the presence of a thoracic aneurysm with aortography or computed tomography used for confirmation and delineation of the pathology. Aortography will however only delineate the fistulous tract if the rate of bleeding is 0.5–1 ml/min [3]. Some investigators feel that aortography is the best diagnostic study to demonstrate the fistula. This approach can, however, produce a false negative result secondary to clot formation.

Esophagogastroduodenoscopy is recommended in that it excludes other causes of bleeding while identifying mass lesions, esophagitis, ulcerations or foreign bodies. The fistula site is occasionally identified on visual examination. Barium esophography has been reported as a sensitive method for demonstrating the fistula, yet this study still produces a high rate of false negative or inconclusive results. Brisk bleeding often precludes any diagnostic tests, and while placement of a Sengstaken-Blakemore tube may be used as a temporizing measure, the risk of tube placement into the aortic lumen is a real risk. Aggressive fluid and blood resuscitation are the mainstays of therapy while the operating room is being prepared.

There have been no reported survivors with non-surgical management. The standard approach is via a left thoracotomy.

After the aorta has been controlled proximally and the fistula exposed, treatment options include simple closure, resection with insertion of prosthetic graft, or bypass grafting from the aortic arch to distal thoracic aorta. Management of the esophagus depends on the size of the perforation and etiology of the fistula. Small perforations due to aneurysmal erosion can be closed primarily after debridement and reinforced with pericardial fat. If the esophagus contains the primary pathologic process, as in reflux esophagitis or malignancy, it is generally too friable to be closed primarily. In this situation, a thoracic esophageal resection and cervical esophagostomy and gastrostomy are indicated. Gastrointestinal continuity can be restored at a later time by gastric pull-up or colonic interposition techniques. Interposition of a pericardial or pleural flap, or vascularized intercostal muscle flaps between the aorta and reconstructed gastrointestinal tract is recommended to prevent repeat fistulization.

Aortoenteric fistulae due to any cause are frequently fatal events. A high index of suspicion is mandated on the part of the treating physician to include this rare, but lethal, process in the differential diagnosis of upper gastrointestinal bleeding.

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