Esophageal foreign body causing direct aortic injury

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BRIEF COMMUNICATION

Foreign bodies in the esophagus are uncommon causes of esophageal perforation. Many nonperforating cases are successfully managed by flexible gastroscopy. However, complicated foreign bodies such as those that result in esophageal perforation and vascular injury are best managed surgically. Gastroscopy remains the primary method of diagnosis. A case of a 59-year-old woman who developed retrosternal and intrascapular pain, odynophagia and hematemesis after eating fish is reported. Flexible gastroscopy showed arterial bleeding from the midthoracic esophagus. Computed tomography scan localized a 3 cm fish bone perforating the esophagus with surrounding hematoma. An aortogram did not reveal an actively bleeding aortoesophageal fistula. The fish bone was surgically removed and the patient recovered with no postoperative complications. This case illustrates the importance of early consideration for surgical intervention when confronted with a brisk arterial bleed from the esophagus with suggestive history of foreign body ingestion.

Key Words: Esophageal perforation; Foreign body; H ematemesis

Esophageal perforation and its complications have been well documented in the literature (1-3). Documented etiologies include tumors, iatrogenic causes, trauma and foreign body ingestion. Foreign body ingestion is an uncommon cause of esophageal perforation but the complications associated with a prolonged impaction can result in considerable morbidity and mortality. Although there have been many cases reported of aortoesophageal fistulae following foreign body ingestion, little has been published about direct aortic injury from a foreign body. Because esophageal perforation and vascular injury usually present as hematemesis or melena, gastroenterologists should be aware of this diagnostic possibility. We present a case of esophageal perforation associated with direct vascular injury related to an aortic puncture caused by a fish bone.

CASE PRESENTATION

A previously healthy 59-year-old woman presented to a local infirmary with a 3 h history of retrosternal chest pain. The pain began immediately after eating a meal of fish. The restrosternal chest pain was pleuritic in nature. The patient had odynophagia. She was treated conservatively with antacids and discharged home. She returned two days later because the pain persisted and began to radiate to the intrascapular region. She became diaphoretic and pale. There were no complaints of nausea or vomiting. The patient was then seen at a peripheral hospital where the possibility of aortic dissection was considered. She was transferred to this hospital for further investigation.

The patient was seen by the cardiovascular surgery service during which she had two episodes of fresh hematemesis. Her hemoglobin was 118 g/L at the peripheral hospital and dropped to 100 g/L after the hematemesis. There was no antecedent history of melena or gastrointestinal bleeding in the past. She was a lifelong nonsmoker, did not use alcohol and there was no history of acetylsalicylic acid or nonsteroidal anti-inflammatory drug use. Her hepatitis B and C status was unknown. There was no history of peptic ulcer disease.

On physical examination, she was pale, diaphoretic and drowsy. The blood pressure was 102/42 mmHg with no pulsus paradoxus. The heart rate was 64 beats/min and she was afebrile. Her peripheral pulses were symmetric. There was no...
palpable subcutaneous emphysema. The remainder of the examination was unremarkable.

Laboratory examination revealed a normal platelet count and coagulation status. The electrocardiogram was normal. A chest x-ray did not show a widened mediastinum nor any subcutaneous or mediastinal air.

On endoscopy, a fresh arterial bleed was seen at approximately 20 cm from the upper alveolar ridge. A fish bone was embedded in the wall of the esophagus. A contrast enhanced computed tomography (CT) examination of the thorax was subsequently performed. The fish bone, measuring approximately 3 cm in length, was seen lying almost horizontally in the proximal thoracic esophagus at the level of the crossing left brachiocephalic vein. Its medial end was seen posterior to the trachea while its lateral end was seen directly abutting the medial aspect of the transverse portion of the thoracic aorta (Figures 1 and 2). Increased attenuation in the mediastinal fat surrounding the thoracic esophagus was present, as were small locules of extraluminal air compatible with perforation (Figure 3). Small bilateral pleural effusions with accompanying bilateral atelectasis were also noted. An aortogram was also performed, which did not show an aortic leak. The patient was resuscitated with three units of packed red blood cells and underwent a right hemithoracotomy the following day.

Intraoperative findings included perforations of the left and right lateral aspects of the midthoracic esophagus by a 3 cm fish bone. The fish bone was embedded in the right lateral wall of the esophagus, approximately 3 cm above the azygos vein. A hematoma surrounding the transverse aorta was seen.

Postoperatively, the patient received antibiotics for 10 days, and a follow-up gastrograffin swallow was normal. She was discharged two weeks after presentation. She remained well two months after presentation.

**DISCUSSION**

Esophageal perforation occurs in 1% to 4% of instances of foreign body ingestion (1,2). These complications carry a considerable morbidity from mediastinitis, parapharyngeal abscess, pericarditis, pneumothorax, pyopneumothorax and pneumomediastinum (2). In a series of 511 cases of esophageal perforation, there is a reported mortality of 22% (4). Foreign body perforation was the etiology of 7% in that series. This condition, although uncommon, must be recognized in the appropriate clinical context.

Local inflammation results in erosion by the foreign body to adjacent structures. A reas of anatomic constriction have an increased risk of foreign body impaction and are common sites of perforation. The aorta is often involved and there are case
reports of aortoesophageal fistulae developing over weeks to years (5). Aortoesophageal fistula is a diagnosis that is almost invariably fatal. Sloop and Thompson (6) described the clinical features of this condition. A ‘signal hemorrhage’ precedes exsanguination, the timing of which is unpredictable. A fistulized tract through inflammatory tissues is thought to be the cause of the exsanguination, but the signal hemorrhage is thought to be due to local arterial bleeding (6). The current case was that of persistent pain and odynophagia for two days before the presentation of the signal hemorrhage. This demonstrates the extreme variability of the time course in vascular injury from foreign body esophageal perforation.

Although the bleeding seen on gastroscopy could have been a result of esophageal venous plexus injury, it is more likely that aortic injury had occurred. Bleeding from esophageal perforation most commonly results in intramural hematomas, but focal thickening of the esophageal wall was not seen. There are no case reports of intramural esophageal bleeding resulting in mediastinal hematomas as was seen in this case.

Direct visualization by gastroscopy remains an important method in the diagnosis of esophageal foreign bodies. Many therapeutic gastroscopic manoeuvres are used to disimpact the esophagus (7). We suggest that flexible gastroscopy is contraindicated in a case of established esophageal perforation. Although few case studies exist on flexible gastroscopy in esophageal perforation with subsequent conservative management of mediastinitis (8), it is reasonable to suggest that air insufflation during this procedure would lead to further morbidity. Characteristics of the suspected foreign body (coins [9], fishbones, needles [10]), duration of foreign body impaction, underlying esophageal disease and clinical presentation with complications are required to assess the probability of esophageal perforation. In the present case, there was no objective x-ray evidence that esophageal perforation had occurred before the endoscopy. However, we suggest that if there is radiological evidence, or if clinical suspicion remains high for esophageal perforation, rigid esophagoscopy or CT scanning should be performed.

The role of CT in foreign body esophageal perforation has been documented. A retrospective study of the CT scans of 12 patients with esophageal perforation revealed that extraluminal air was the most useful finding (11). The evaluated features, however, are not pathognomonic for esophageal perforation (12). The role of CT is not to confirm a highly suspicious case of esophageal perforation due to a foreign body but to provide further evidence in atypical cases. In the case presented, the fish bone was easily seen and the surrounding hematoma confirmed the observation that vascular injury was likely.

Nonoperative treatment of esophageal perforation without vascular injury may result in complications such as mediastinitis. Definitive treatment is controversial and some have advocated conservative treatment, even in cases of mediastinitis (8). Novel endoscopic treatments have been attempted, including endoscopic clipping of esophageal perforations (13). Some selective cases have resolved in a nonoperative approach (14) but if there is a question of vascular injury, as with this case, operative management is the best choice (15).

The above case illustrates the importance of a detailed history, physical examination and timely investigations when dealing with a foreign body perforation of the esophagus resulting in vascular injury. In suspected esophageal perforation, CT scan remains the diagnostic modality of choice. If the CT scan does not show evidence of perforation but clinical suspicion remains high, the next diagnostic modality should be rigid esophagoscopy or surgery.

REFERENCES
