Esophageal Rupture Presenting with Hematemesis

by Chaya Abelow, Arkady Broder, Angelo Reyes, Seth A. Cohen

A n otherwise healthy 80 year-old woman presented to the emergency room with six hours of hematemesis and chest pain. The patient had been in her usual state of health until 12 hours earlier when she developed nausea and two episodes of non-bloody vomiting after eating breakfast. Several hours later she had another episode of vomiting, this time with hematemesis and subsequent chest pain, prompting her to call an ambulance. On arrival in the emergency room, the patient was hypertensive and tachycardic. She complained of crushing chest pain, which required 8 mg of morphine for pain relief. On exam there was no crepitus on her neck or chest; abdominal and rectal exams were unremarkable. Initial laboratory values revealed WBC of 13,200 with 89% neutrophils; hemoglobin of 12.7 g/dl; BUN of 45 mg/dl. A chest x-ray was normal. The patient was admitted to the intensive care unit (ICU) where she continued to have chest pain and more frequent hematemesis. Emergent endoscopy, after endotrachial intubation to protect the airway, revealed a tear in the proximal and middle third of the esophagus (Figures 1 and 2). Additionally, the patient was found to have subcutaneous emphysema. Computed tomography (CT) scan of the chest with contrast confirmed esophageal rupture at the level of the carina and extensive pneumomediastinum with

Chaya Abelow, MD¹ Arkady Broder, MD¹ Angelo Reyes, MD² Seth A. Cohen, MD¹ ¹Department of Medicine and Division of Digestive Diseases: CA, AB, SC ²Division of Cardiothoracic Surgery, Beth Israel Medical Center, NY

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Figure 3. CT Chest with oral and intravenous contrast demonstrating esophageal perforation and oral contrast extravasating into the mediastinum.

Figure 4. En-bloc. esophagus gross pathology specimen Note the full thickness extended transmural tear.

air extending into the soft tissues of the lower neck and anterior chest wall (Figure 3). At surgery, an esophageal tear was found from the level of the aortic arch down to the esophageal-gastric junction; given the extent of the injury and concern about viability of the esophageal tissue a complete esophagectomy with a spit fistula was performed. Pathologic examination of the esophagus showed squamous esophageal tissue with deep ulceration and acute inflammation (Figure 4). The patient tolerated the surgical procedure and recovery was uneventful.

Spontaneous esophageal rupture is seen in less than 25% of all perforation cases and remains a rare entity in the United States. This syndrome was first described in 1724 by Herman Boerhaave and believed to be a result of barotrauma during vomiting. In 90% of cases, the rupture occurs in the left lateral distal third of the esophagus where the vessels and nerves enter because of a presumed anatomic weakness. Early diagnosis and treatment of Boerhaave’s is essential as mortality is estimated at 20-40% among treated patients and essentially 100% among those untreated.

The clinical presentation can vary widely, leading to incorrect diagnosis in up to 60% of cases. The most common symptoms of thoracic esophageal perforation are vomiting, chest pain and dyspnea. Notably, hematemesis is very unusual and rarely reported; in this patient, the large amount of ongoing hematemesis obscured a prompt diagnosis. Physical exam may be unremarkable as subcutaneous emphysema is a late and therefore insensitive finding. Alternatively, those patients who present late or in whom diagnosis is delayed may have nonspecific signs of sepsis and shock.

Accurate and timely diagnosis therefore requires a high index of suspicion. Initial evaluation should include a chest x-ray, as it may be abnormal in up to 90% of cases. Some of the findings include subcutaneous emphysema, pneumomediastinum, pleural effusion, pneumothorax or hydropneumothorax. However, a negative chest x-ray does not rule out esophageal perforation, as was demonstrated in our patient. CT of the chest with oral and intravenous contrast is considered the gold standard for confirming the diagnosis in 92-100% of cases and it offers the added benefit of detailing any additional involvement of surrounding structures. An esophagram with water-soluble contrast may be a viable alternative, but is less sensitive and specific for esophageal rupture, particularly in the cervical region. Outside of therapeutic intervention, upper endoscopy is generally not recommended for diagnosis as both the endoscope and insufflation of air can expand the perforation and introduce air into the mediastinum. In this patient the extent of the hematemesis obscured a prompt diagnosis and led the physicians to treat it as a case of upper GI bleeding. In retrospect, however, the degree of chest pain should have prompted a CT scan of

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the chest. Endoscopy did yield the correct diagnosis but contributed to the pneumomediastinum by introducing air under pressure.

In all cases, initial treatment should be directed at resuscitation of the patient and limitation of mediastinal damage with strict NPO and broad-spectrum antibiotics. Definitive treatment depends on extent and location of the perforation, rapidity of diagnosis and the presence of any underlying disease of the esophagus. Medical management alone may be sufficient in patients who are diagnosed early and who do not have signs of infection or involvement of the pleura. Endoscopic luminal stent placement may be another alternative to the treatment of localized and mostly contained esophageal perforations. Most patients, however, will ultimately require surgical therapy. 3

References


Answers to this month’s crossword puzzle:

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1 2 3 4 5 6 7 8
S T R I C T U R E S  B E A T
T I R R O M I D I R
O U N C E N A S O G E N E
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G Y I H S S
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A B I O P S Y P A T E N T
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