CASE REPORT

Esophageal Hematoma: An Unusual Complication of Thrombolysis

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Abstract

Esophageal hematoma is a rare cause of chest pain. Esophageal hematomas can be spontaneous or secondary to trauma, toxic ingestion, or intervention. They develop from the dissection of the mucosa from the muscular layers of the esophageal wall. The cardinal symptom is severe chest pain; other symptoms include dysphagia, odynophagia, and hematemesis. Patients with spontaneous esophageal hematomas are commonly misdiagnosed to have an ischemic cardiac event. Distinction from a cardiac ischemic event is critical because this condition is worsened by anticoagulation. We describe one case of intramural hematoma with chest pain that was successfully managed by conservative treatment. (J Dig Endosc 2010;1:26-28)

Keywords: Esophageal hematoma, thrombolysis, anticoagulation

Case report

A 60-year-old female presented to emergency department of another hospital with sudden onset of dyspnea and chest pain. She had past history of diabetes mellitus for 10 years and hypertension for 5 years with no family history of coronary artery disease. In addition to medications for diabetes and hypertension she was taking aspirin prior to the initial presentation. Her electrocardiogram showed ST elevation in inferior leads with T inversion in lead II. Thrombolysis was done with streptokinase along with standard therapy of antiplatelets, betablockers, ACE inhibitors and statins. Her Trop T was negative and echocardiography showed basal and mid inferior wall hypokinesia, consistent with myocardial infarction; left ventricular function was good. Repeat electrocardiograms at 90 minutes and 24 hours were unchanged. She had recurrent episodes of angina, which were managed with low molecular weight heparin and nitrates. On the second day she developed sudden onset of chest pain with breathlessness, which was not relieved with nitrates, and ECG showed no fresh changes. She developed recurrent (six) episodes of hematemesis of around 100ml each. She was referred to our hospital for further management. There was no history of vomiting or retching either at initial presentation or before the hematemesis. Antithrombotics and antiplatelets were stopped. She was pale with tachycardia and maintaining normal supine blood pressure. Her hemoglobin was 7.4 gm for which fresh blood was transfused. Emergency upper gastrointestinal endoscopy (EGD) was done in the intensive care unit, which showed diffuse submural hematoma extending form upper esophageal sphincter to gastro-esophageal junction, occupying ½ to ¾ of circumference along with oozing of blood at some points. Patient was managed conservatively with blood transfusion. CT scan of chest with aortogram (Fig. 1) showed grossly distended esophagus, throughout its entire length extending from the level of C7 upto esophago-gastric junction. The esophagus appeared to be filled by hyperdense soft tissue density lesion along its entire length with compromise of the lumen with no

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leak of oral contrast. She was kept nil orally for 1 week, after which diet was started, initially liquids followed by soft diet. We repeated an endoscopy (Fig. 2) after 2 weeks, which showed longitudinal esophageal ulcers involving the entire length of the esophagus, suggestive of resolving esophageal hematoma. After complete recovery the patient was discharged after 3 weeks of hospitalization.

**Figure 1:** CT chest showed a hyperdense mass occupying almost the whole esophageal lumen. Note intraluminal air pockets.

**Figure 2:** Endoscopy done at 2 weeks showing resolution of the hematoma and linear ulcer over the entire length of the esophagus.

**Discussion**

The three different types of acute esophageal injury are a mucosal tear (Mallory–Weiss syndrome), full-thickness rupture (Boerhaave’s syndrome) and dissecting intramural haematoma. Dissecting intramural haematoma of the oesophagus is a rare condition in which intramural hemorrhage leads to submucosal dissection of the esophageal wall. Symptoms include retrosternal chest pain, dysphagia/odynophagia, and hematemesis. Thirty-five percent of patients present with this triad, and half present with at least two symptoms (1). Bleeding occurs later, after rupture of the hematoma into the lumen. Similar pain and associated symptoms may also be seen in myocardial infarction, aortic dissection, prolonged esophageal spasm, or acute drug-induced midesophageal obstruction and ulceration.

Esophageal hematomas may be spontaneous or associated with sudden changes in the transmural wall pressures due to a variety of causes. The etiology of the spontaneous intramural hematoma of the esophagus remains controversial. The most common predisposing factors for esophageal hematoma are vomiting (2), esophageal instrumentation (3), hematological or bleeding disorders, and anticoagulation therapy. Other less frequent causes reported include food impaction (4), pill ingestion (5), and trauma. The condition has to be differentiated from Mallory-Weiss tear and Boerhaave’s syndrome.

The diagnosis of esophageal hematoma can be safely made with several complimentary investigations. Endoscopy is the preferred investigation, especially when hematemesis is the presenting symptom (6). Endoscopy reveals a bulging, purplish lesion with a smooth, normal overlying mucosa. Occasionally there may be mucosal ulcerations and necrosis in the presence of extensive submucosal extension, as was seen in our case. The endoscopic appearance can be mistaken for a vascular or hemorrhagic tumor or for a large esophageal varix. Ouatu-Lascar et al has proposed that esophageal hematomas can be further classified according to the degree of involvement of the lumen in four stages (7). Stage 1: hematoma without surrounding tissue edema, Stage II: hematoma with surrounding tissue edema, Stage III: hematoma with edema plus compression of esophageal lumen and Stage IV: complete obliteration of the lumen with hematoma, edema, and organized clot formation.

Other investigations include contrast swallow, CT scan or MRI, and endoscopic ultrasound. The contrast esophagogram reveals a ‘double-barrelled esophagus’ (the ‘mucosal stripe sign’) or an elongated tubular filling defect. MRI or CT scan, shows eccentric thickening of the esophageal wall.

Despite dramatic presentation, most cases of intramural hematomas resolve spontaneously. Treatment is therefore conservative and should include correction of the coagulopathy, nil by mouth, intravenous alimentation and
antibiotics in severe cases (8). Surgery is reserved only for rare instances, to drain a hematoma and close an esophageal mucosal tear (9). In contrast, aortoesophageal fistula is a life threatening disease that may present as submucosal hematoma, with a very poor prognosis if surgical intervention is delayed. Physicians should be aware of and differentiate aortoesophageal fistula from intramural hematoma. The classic endoscopic finding of aorto-esophageal fistula is a pulsatile esophageal mass. Serial endoscopy reveals that the dissected mucosal layer sloughs away leaving a large longitudinal ulcer.

Since the diagnosis of myocardial infarction was not well established in our patient and he was taking aspirin regularly, the possibility of initial presentation as a result of esophageal hematoma cannot be totally excluded and extensive lesion occurred secondary to thrombolysis. But absence of vomiting, retching or esophageal instrumentation prior to the initial presentation suggest that esophageal hematoma developed following thrombolysis.

Conclusions

Intramural esophageal hematoma is a rare condition, which should be considered in patients presenting with acute chest pain, hematemesis and dysphagia/odynophagia, particularly in the presence of a bleeding disorder or where there has been recent administration of antiplatelets, anticoagulants or thrombolytics. It has a good prognosis when treated conservatively with surgical intervention reserved for those with severe hemorrhage or esophageal perforation.

References