Esophagogastric Hematoma Mimicking a Malignant Neoplasm: Clinical Manifestations, Diagnosis, and Treatment

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Esophagogastric hematoma is a rare condition occurring spontaneously or after esophageal instrumentation. In this report, we describe a patient with acute dysphagia in whom a lower esophageal mass was detected radiographically. Upper endoscopy revealed an esophageal mass that extended from the mid-esophagus to the gastroesophageal junction and was associated with a malignant-appearing ulcerated mass (5 to 6 cm) in the cardia. Gastric cancer with esophageal extension was the presumptive diagnosis. Computed tomography showed that the esophageal mass had a density similar to blood, a finding suggesting the presence of an esophageal hematoma. Biopsy specimens of the ulcer revealed acute inflammation but no malignant involvement. The patient was treated conservatively, and the initial symptoms resolved. Esophagogastric hematomas can mimic a neoplasm; thus, establishing the correct diagnosis is important because this condition has a favorable prognosis, and only conservative treatment is needed.

REPORT OF CASES

Case 1

A 70-year-old man was referred to Mayo Clinic Rochester for evaluation of acute dysphagia and epigastric pain of 3 days' duration. He had no nausea, vomiting, anorexia, or weight loss. Past medical history included rheumatoid arthritis, prostate cancer, and coronary artery disease, for which he had undergone coronary bypass. His daily medications were n-penicillamine, prednisone, and aspirin. Physical examination revealed stable vital signs, a sternotomy scar, and joint deformity consistent with rheumatoid arthritis. Laboratory tests yielded a hemoglobin concentration of 15 g/dL, leukocyte count of 15 x 10^9/L, and platelet count of 135 x 10^9/L. A chest roentgenogram demonstrated a small left pleural effusion. An upper gastrointestinal series performed elsewhere revealed a lower esophageal stricture or mass. Upper endoscopy demonstrated an ulcerated, partially obstructing esophageal mass with dark discoloration extending from the mid-esophagus through the gastroesophageal junction (Fig. 1). A friable malignant-appearing ulcerated mass (5 to 6 cm) was detected in the cardia (Fig. 2); biopsy specimens were obtained. The initial endoscopic impression was gastric cancer with submucosal esophageal extension. The histologic interpretation of the biopsy specimens was reactive fundic mucosa and a hemorrhagic ulcer base with no evidence of malignant disease. Several radiologic studies were performed for further assessment of the abnormal findings on endoscopy. A radiographic contrast study with use of meglumine diatrizoate showed no extravasation of contrast medium. Computed tomography (CT) of the chest demonstrated a tubular submucosal esophageal mass with a density consistent with blood and a soft tissue mass at the gastroesophageal junction (Fig. 3). An upper gastrointestinal series showed a linear collection of contrast medium tracking along the right side of the esophagus, from the carina to the gastroesophageal junction (Fig. 4).

Because gastric cancer with an associated esophageal hematoma remained the foremost diagnosis, surgical consultation was sought. An operation was deferred. On further questioning, the patient’s wife revealed that the patient was taking 12 aspirin tablets per day. The patient was treated conservatively with omeprazole, 20 mg daily, and had an uncomplicated hospitalization except for pneu-
Fig. 1 (case 1). On endoscopy, ulcerated submucosal mass is evident in mid-esophagus.

Fig. 2 (case 1). On endoscopy, large deep malignant-appearing ulcer is evident within cardia.

Fig. 3 (case 1). Chest computed tomographic scan, demonstrating soft tissue mass within esophagus that displaces lumen to the left. This appearance would be consistent with submucosal site of mass (arrows). Bilateral pleural effusions and coronary artery calcification are incidentally noted.

monia. At dismissal, the clinical diagnosis was aspirin-induced gastric ulcer and submucosal esophagogastric hematoma. A follow-up upper endoscopy 1 month later showed mild distal esophagitis and complete resolution of the esophagogastric abnormalities.

Case 2
A 31-year-old woman came to our institution because of upper gastrointestinal bleeding. Her past medical history was remarkable for systemic lupus erythematosus, deep venous thrombosis, and paroxysmal atrial fibrillation. Her medications included sodium warfarin, prednisone, digoxin, and one aspirin tablet per day. Laboratory tests yielded an international normalized ratio of 11.1 and a hemoglobin concentration of 9 g/dL. Upper endoscopy showed a malignant-appearing ulcerated gastric mass. An abdominal CT scan revealed pronounced thickening of the fundic wall, suggesting intramural edema or a hematoma rather than malignant disease. Biopsy specimens of the gastric mass revealed inflammatory tissue but no evidence of malignant involvement, an outcome supporting the diagnosis of a gastric hematoma. A follow-up upper endoscopy 1 month later showed complete disappearance of the mass, and thus the benign nature of the disease was confirmed.

DISCUSSION
Intramural esophageal hematoma is a rare condition of esophageal injury deep to the mucosal layer without full-thickness esophageal perforation. The mechanism of injury may be similar to that of the Mallory-Weiss and Boerhaave syndromes. Esophageal hematomas have been reported in patients whose ages range from 21 to 87 years; mean age is 58 years. Unlike the Mallory-Weiss and Boerhaave syndromes, which typically occur in men, the esophageal hematoma has a 2:1 female preponderance.
Fig. 4 (case 1). Upper gastrointestinal series with use of contrast material, showing opacification of two lumens. Larger lumen represents normal esophageal lumen. Smaller lumen (false lumen) represents contrast material that has dissected outside lumen within the esophageal hematoma (double-barrel sign).

The term "esophageal apoplexy" was introduced by Smith and associates and is defined as spontaneous intramural esophageal hemorrhage. The pathogenesis of esophageal hematoma is unknown. Criblez and colleagues reviewed 91 cases of esophageal hematoma that appeared in the literature. Precipitating factors were identified in 63% of all patients: vomiting in 22%, instrumentation in 17%, hemostasis abnormalities in 21%, and spontaneous occurrence in 37%. Shay and coworkers observed that, in most patients with normal hemostasis, the hematomas occurred distally after vomiting and were thought to be due to the Mallory-Weiss syndrome. In patients with impaired hemostasis, the hematoma occurred proximally or at multiple sites without the characteristic emesis noted in most patients. In these patients, investigators suggested that submucosal bleeding occurs initially as a result of abnormal hemostasis and spontaneous mucosal rupture. Intramural hematomas have been described in other organs in association with coagulopathy. Submucosal distention may result in acute pain with subsequent mucosal rupture leading to hematemesis. The hematoma may dissect into the proximal stomach and mimic a gastroesophageal junction or proximal gastric mass (as in our patients).

The characteristic clinical manifestations include chest pain, dysphagia, and hematemesis; 35% of patients have this triad. Pain, initially in 66 to 87% of patients, is retrosternal or epigastric, develops gradually, and may become severe and exacerbated with swallowing. Dysphagia occurs in 26% of patients. Hematemesis is less common and is associated with minimal blood loss.

Upper gastrointestinal series characteristically reveal a "double-barrel" sign, as in one of our patients (case 1) (Fig. 4). This reflects a mucosal tear that allows escape of contrast material into the esophageal hematoma. Other roentgenographic findings include luminal narrowing and filling defects. CT scanning can be most helpful in confirming the absence of esophageal perforation and in assessing the mediastinum for other pathologic disorders such as tumors. In our patients, CT was helpful in defining the esophagogastric mass as a hematoma and not a tumor, as was initially suspected. Endoscopy may be unnecessary when the clinical picture and radiologic findings are characteristic of an esophageal hematoma. Nonetheless, endoscopy is likely to be the first major diagnostic intervention in light of the clinical manifestations of these patients. Directed histologic sampling can be expected to support the diagnosis. Endoscopic ultrasonography can also be useful for excluding a malignant process; it allows better characterization of the abnormal findings confined within the sonographic layers of the gut wall. Our first patient (case 1) did not initially reveal that he was taking an extremely high dose of aspirin. The combination of non-steroidal anti-inflammatory drugs and corticosteroids is known for its ulcerogenic properties, and corticosteroids may also interfere with healing of tissue. Both these factors may have contributed to our patient's clinical manifestations. With this additional information, we can assume that aspirin and corticosteroids caused the development of a gastric ulcer, complicated by erosion into an intramural blood vessel, which in combination with aspirin-induced platelet dysfunction resulted in a spontaneous submucosal esophagogastric hematoma. Similarly, in our second patient (case 2), the combination of prednisone, aspirin, and sodium warfarin resulted in a large intramural gastric hematoma that was mistaken for a malignant mass. Aspirin use has been described in 16% of patients with esophageal hematomas.

The correct diagnosis of an intramural hematoma, esophageal or, less commonly, gastric, is important because of the excellent prognosis associated with conservative therapy. Most cases resolve spontaneously with-
Although the endoscopic findings in our patients were suggestive of a malignant esophagogastric neoplasm, the CT findings and histopathologic features indicated a benign condition. This was supported by the patients' clinical response to conservative therapy.

**CONCLUSION**

Shortly after the initial assessment of a patient in whom an esophagogastric hematoma is suspected, follow-up with endoscopy or CT can exclude malignant disease by demonstrating spontaneous resolution. Recognition of an esophagogastric hematoma is critical because of the favorable prognosis without the need to perform invasive diagnostic tests or an operation.

**REFERENCES**