

# Endoscopic appearance of esophageal hematomas

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## INTRODUCTION

Esophageal hematomas develop from the dissection of the mucosa from the muscular layers of the esophageal wall and represent an uncommon condition affecting all ages<sup>[1-3]</sup>. Although the most common cause of esophageal hematomas is iatrogenic mechanical injury-induced by prolonged nasogastric intubation, difficult or forceful endoscopic intubation, or the result of variceal injection sclerotherapy—some may be spontaneous, particularly in patients receiving anticoagulants<sup>[3-6]</sup>. Presenting symptoms most commonly include dysphagia, hematemesis, and sub-sternal or epigastric pain<sup>[5,9]</sup>.

In this report, we present four cases of esophageal hematomas diagnosed endoscopically, describe their clinical and endoscopic characteristics and propose a classification. As our cases exemplify, therapy is conservative and a favorable prognosis is the rule. Although the diagnosis has traditionally been made by barium esophagography<sup>[1]</sup> or CAT scanning<sup>[1,10]</sup>, the increased use of endoscopy will allow increased recognition of esophageal hematomas even at a subclinical (asymptomatic) stage and ultimately lead to a better understanding of their etiopathogenesis and their prevention.

## CASE REPORTS

**Case 1** A 90-year-old man with a history of

hypertension, congestive heart failure, and mild aortic stenosis was referred for upper endoscopy to determine the cause of iron deficiency anemia. A colonoscopy had previously revealed diverticular disease. Laboratory values included a hematocrit of 30%, iron of 90 mg/L and a TIBC of 358. Coagulation studies were normal. Endoscopy revealed salmon-colored mucosa extending from 28 cm-34 cm from the incisors, consistent with Barrett's esophagus. A densely red, elevated lesion, consistent with a hematoma was noted at 19 cm (Figure 1a). No therapy was given. A follow-up endoscopy, performed one week later, showed complete resolution of the hematoma. Because of its location, the hematoma was attributed to a difficult, possibly traumatic, endoscopic intubation.

**Case 2** A 59-year-old man was referred for upper endoscopy because of chronic reflux symptoms. His medications included omeprazole 20 mg po-daily, and aspirin 325 mg po-daily. Endoscopy revealed confluent esophageal erosions and ulcers consistent with Savary stage III esophagitis from 36 cm-33 cm. Upon biopsy of the esophagus, a purple-red, elevated lesion, consistent with a hematoma was noted at 35 cm (Figure 1b). Histologic examination of the biopsy sample revealed changes consistent with reflux esophagitis. The hematoma was attributed to the mucosal biopsy obtained in the context of aspirin intake.

**Case 3** A 90-year-old man with solid food dysphagia was referred for esophageal dilation. He had a history of metastatic (stage 4) squamous esophageal cancer treated with radiation therapy and esophageal dilation. He had mild anemia, but no thrombocytopenia or coagulation abnormalities. Endoscopy revealed a mid-esophageal stricture at 5 cm-29 cm which was dilated with minimal resistance using Savary dilators over a guide wire. This resulted in a 1 cm-long submucosal hematoma at 26 cm (Figure 1c). No biopsy was obtained. Because the patient's dysphagia resolved, there was no follow-up endoscopy.

**Case 4** A 51-year-old male with a history of rheumatic heart disease, status post aortic valve replacement, was hospitalized for mitral valve replacement. Because his postoperative course was complicated by enterococcal sepsis, a transesophageal echocardiogram was performed which excluded the presence of endocarditis. The

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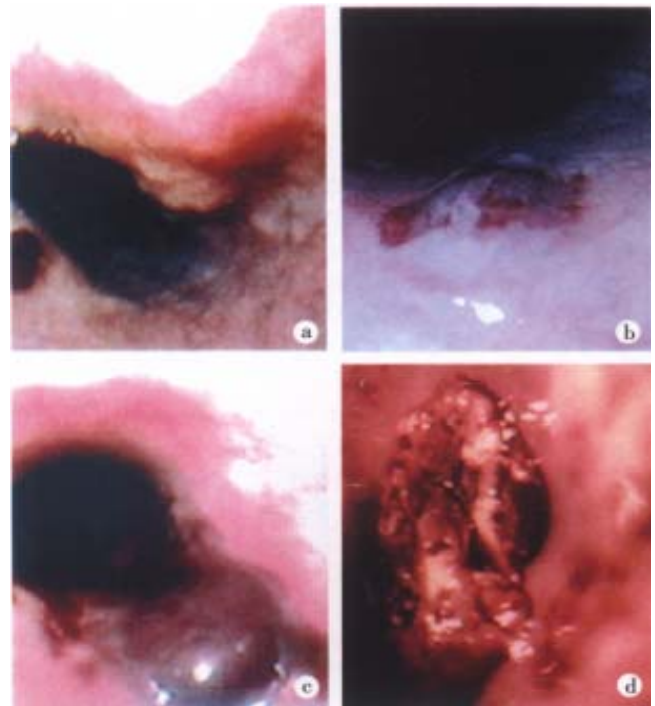
following day, hematemesis was noted. Coagulation parameters were within the expected therapeutic range for prosthetic valves. An emergency upper endoscopy revealed a 3 cm-long, dark red esophageal mass at the GE junction, completely obliterating the esophageal lumen (Figure 1d). Biopsies obtained from the surface of the mass revealed organized clot and acute esophageal mucosal inflammation (not shown). Broad spectrum antibiotics and H<sub>2</sub> receptor antagonists were administered intravenously; effective anticoagulation was also continued. Although there was no more evidence of gastrointestinal hemorrhage, the patient eventually succumbed to sepsis ten days later. Autopsy revealed severe herpetic tracheobronchitis without any evidence of esophageal injury.

## DISCUSSION

Esophageal hematomas appear as raised purplish-red lesions, mostly sub-mucosal in location, but occasionally obliterating the esophageal lumen. Depending on their etiology, they may be classified as spontaneous or traumatic. Based on the above observations, we propose that esophageal hematomas can be further classified according to the degree of involvement of the lumen in four stages (Table 1). Esophageal hematoma formation<sup>[11,12]</sup>, is an uncommon condition in which an intramural hemorrhage leads to a variable degree of submucosal dissection of the esophageal wall ranging from single or multiple localized hematomas to complete dissection of the esophagus<sup>[1]</sup>. Esophageal hematomas may be spontaneous, associated with sudden changes in the transmural wall pressures due to a variety of causes including coughing, retching or protracted vomiting<sup>[13]</sup>, or may result from iatrogenic instrumentation, such as esophageal variceal sclerotherapy<sup>[3,14,15]</sup> or transesophageal ultrasonography<sup>[6]</sup> (Table 2). Hematomas may also be associated with direct trauma from abrasive foodstuffs such as taco shells<sup>[16]</sup>, pill-induced esophageal injury<sup>[17]</sup>, or with a coagulopathy, with little or no history of esophageal barotrauma<sup>[5]</sup>. Other probable causes include esophageal stricture, diverticulum, esophageal arteriovenous malformation, and aspirin use<sup>[18]</sup>. The majority of patients with esophageal hematomas are older than 70 yr, although isolated reports of patients as young as 21 yr have been made. In some studies, there seems to be a predilection for the female gender<sup>[1]</sup>.

**Table 1 Stages of esophageal hematomas**

I: Hematoma without surrounding tissue edema
II: Hematoma with surrounding tissue edema
III: Hematoma with edema plus compression of esophageal lumen
IV: Complete obliteration of the lumen with hematoma, edema, and organized clot formation



**Figure 1** Endoscopic appearance of esophageal hematoma as. a: Stage I esophageal hematoma. There is no surrounding edema. Patient was asymptomatic. b: Stage II esophageal hematoma after an esophageal biopsy. Note the surrounding tissue edema. c: Stage III hematoma induced by esophageal dilation of a stricture. There is edema and separation of the surface mucosal layer that is partially obliterating the lumen. Patient was asymptomatic. d: Stage IV esophageal hematoma related to recent transesophageal echocardiography. Edema and clot formation led to complete obliteration of the esophageal lumen and hematemesis.

**Table 2 Causes of esophageal hematomas**

Spontaneous	Associated with coughing, retching, protracted vomiting Esophageal barotrauma Pill-induced esophageal injury Use of anticoagulants or aspirin Esophageal diverticulum A-V malformation
Traumatic	Endoscopic intubation, biopsy, dilation Variceal sclerotherapy Transesophageal echocardiography Foreign body ingestion

The clinical presentation is variable. Although mostly asymptomatic, esophageal hematomas may present with dramatic chest pain, severe dysphagia, and hematemesis. In such instances, they should be differentiated from Mallory-Weiss tear and Boerhaave's syndrome, conditions which they may closely mimic<sup>[19-21]</sup>. Typically barium swallow or CAT scan have been used for the diagnosis, showing intraluminal filling defects or a double-barrelled appearance of the esophagus<sup>[22]</sup>. More recently, endoscopy is the preferred investigation, especially when hematemesis is the presenting symptom<sup>[23]</sup>. Because spontaneous resolution is the rule, the treatment of esophageal hematomas is conservative

and consists of a regimen of nil by mouth, intravenous alimentation and antibiotics in severe cases<sup>[1]</sup>. Surgery is reserved only for rare instances, to drain a hematoma and close an esophageal mucosal tear<sup>[24]</sup>.

In our study, all four patients were male, the hematoma was visualized endoscopically, and was traumatic either from insertion of the scope (cases 1 and 4), or from endoscopic manipulation (cases 2 and 3). In 3 out of 4 of our patients the hematomas were asymptomatic. Because of the increased utilization of endoscopy for diagnosis and therapy, it is expected that esophageal hematomas will be encountered more frequently. This is particularly true in cases of esophageal dilation of strictures, repetitive multiple biopsies for surveillance of Barrett's esophagus, esophageal variceal sclerotherapy, and transesophageal echocardiography.

Hematomas related sclerotherapy seem to occur 2 d-4 d after injection, when tissue necrosis that extends into the submucosa reaches its maximum. Contributing factors include volume of sclerosant per injection, interval between treatments, paravariceal versus intra-variceal injection, bleeding diathesis, and occurrence of retching or prolonged Valsalva during or shortly after the treatment<sup>[3-5]</sup>. Early endoscopy is useful in establishing the diagnosis and reveals an intraluminal bulge of the mucosa with dark blue discoloration.

The blind insertion of a transesophageal endosonographic probe for diagnosis of valvular heart disease<sup>[25]</sup>, and its increased utilization during cardiothoracic surgery<sup>[6]</sup> represents an additional risk for esophageal hematoma formation, as exemplified in case 4 of this report. A recent review of the literature on the association of transesophageal echocardiography during cardiac surgery and gastrointestinal bleeding revealed that the overall percentage of postoperative gastrointestinal complaints following the procedure was 11%, and frank upper gastrointestinal bleeding was 2.1%<sup>[6]</sup>. Another multicenter survey of 10419 examinations on the safety of transesophageal echocardiography revealed only 2 patients who developed bleeding complications which necessitated interruption of the examination<sup>[7]</sup>. Realising that the risk of gastrointestinal bleeding is small but present, most authors recommend careful review of upper digestive symptoms to rule out preexisting esophageal pathology, ample lubrication of the endosonographic tip, and avoidance of fixation of the probe in a flexed position for prolonged periods in order to avoid contact pressure and esophageal mucosal damage<sup>[8]</sup>.

In conclusion, our study reviews the endoscopic features of esophageal hematomas and classifies

them in terms of etiology and appearance. With the widespread use of esophageal instrumentation, these lesions will be increasingly recognized endoscopically. Depending on their severity and endoscopic stage, esophageal hematomas may or may not be associated with symptoms. Treatment is generally conservative and favorable prognosis is the rule.

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