Spontaneous intramural esophageal hematoma (IEH) secondary to anticoagulation and/or thrombolysis therapy in the setting of a pulmonary embolism: a case report

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ABSTRACT

Intramural esophageal hematoma is part of a spectrum of esophageal injuries. Vomiting and straining, endoscopic procedures and bleeding disorders are the most common predisposing factors. However, it can also be an unusual complication of anticoagulation and/or thrombolysis therapy. The most common symptoms are retrosternal chest pain, dysphagia and hematemesis. Computed tomography is the modality of choice and treatment is medically conservative with the cessation of Warfarin and thrombolysis use. When anticoagulation and/or thrombolysis therapy is necessary, periodic reassessment for symptoms of intramural esophageal hematoma may be helpful for early identification and management. We described one case of intramural esophageal hematoma possibly resulting from anticoagulation and/or thrombolysis therapy in the setting of pulmonary embolism.

CASE REPORT

CASE REPORT

A 73-year-old male presented to the emergency department with a painfully swollen left leg and shortness of breath. He denied any recent travels, injury, immobility or history of deep vein thrombosis (DVT). His medical history is significant for peripheral vascular disease and hypertension for which he takes Accupril and Atenolol. Duplex venous ultrasonography of the left leg revealed extensive DVT and superficial vein thrombosis (SVT) (Figure 1). A computed tomography angiogram (CTA) of the pulmonary arteries demonstrated an extensive partially calcified thrombus in the inferior vena cava (IVC) with chronic features and bilateral pulmonary embolism (PE) (Figure 2). The patient was admitted, placed on anticoagulation therapy and discharged after 5 days with an international normalized ratio (INR) that ranged from 1.12-1.4 (normal 2-3) during his hospital stay.

A few weeks later, the patient was readmitted for an IVC filter placement and venous catheter-directed thrombolysis of his left iliac and left common femoral vein in an attempt to reduce his chronic thrombus burden. After 2 days of thrombolysis, he was started on anticoagulation therapy. His INR ranged from 1.93-2.19 during this time. On the 4th hospital day, the patient complained of severe chest pain and dysphagia. He was unable to tolerate liquids or small amounts of oral intake. The patient also developed hematuria and epistaxis. He appeared to be in distress, but his vital signs were normal. His cardiac enzymes were negative and had an INR of 2.74 and partial thromboplastin time (PTT) of 123.6s (normal 25-40s). A CT of the chest revealed an eccentric intramural esophageal hematoma (Figure 3) that was 5 cm wide and 21.7 cm long, with no deviation of the esophagus. An extensive hematology workup was negative. The patient had no prior

upper gastrointestinal complaints and no history of endoscopic procedures. Warfarin was discontinued and the patient was treated conservatively with nothing by mouth and parenteral nutrition. Ten days later, he had a normal coagulation profile and his dysphagia improved enough to tolerate a full liquid diet. A repeat CT of the chest showed a resolving intramural esophageal hematoma measuring 3.9 cm wide and 18.6 cm long (Figure 4). He was subsequently discharged.

The patient was readmitted the next day for severe chest pain and worsening dysphagia. His cardiac enzymes were negative and his coagulation profile remained normal. The patient's persistent and worsening dysphagia despite improving measurements on CT imaging prompted further investigation. An upper gastrointestinal endoscopy revealed a large organized clot that obliterated the esophageal lumen with circumferential erosion and ulceration in the middle portion of the esophagus from 30 cm down to 40 cm (Figure 5). The majority of the clot was removed using the Roth net, but a large clot still partially occluding the lumen can be seen burrowed in the submucosa of the distal esophagus (Figure 6). Although the patient's dysphagia greatly improved after the endoscopy, a CTA of the pulmonary arteries revealed a new PE involving the right pulmonary artery and extending into ipsilateral arteries supplying the upper, middle and lower lobes (Figure 7). The patient expired the next day.

DISCUSSION

Intramural esophageal hematoma (IEH) is characterized by a hemorrhagic episode that starts within the submucosa of the esophagus. The collection of blood forms a hematoma and may eventually progress to dissection of the submucosal layer [1]. IEH is part of a spectrum of esophageal injuries that ranges from local mucosal tears to transmural rupture of the esophagus, also known as Mallory-Weiss syndrome and Boerhaave's syndrome respectively.

The 5 subtypes of IEH are categorized by the nature of the hemorrhage: traumatic, emetogenic, abnormal hemostasis-related, aorta-related and spontaneous [2]. Spontaneous submucosal hematoma of the esophagus is further sub-classified into factors that increase the tendency of bleeding such as drugs or an underlying disease [2, 3]. The most common predisposing factors for IEH are endoscopic procedures, vomiting and straining and bleeding disorders [4, 5].

IEH is more commonly seen in middle-aged patients, with a slight predominance in females. The 4 stages of IEH manifestation are evaluated according to the degree of luminal involvement. Stage I and Stage II are characterized by isolated hematoma and hematoma with surrounding tissue edema respectively. Stage III involves a hematoma with edema and compression of the esophageal lumen while Stage IV is the complete obliteration of the esophageal lumen with formation of hematoma, edema and organized clot formation [3, 6]. As such, the patient described in this case had Stage IV spontaneous eccentric IEH without deviation of the esophagus. The subsequent circumferential erosion, ulceration and clot formation found on endoscopic evaluation is believed to be a complication of the intrinsic intramural hematoma, where there is erosion from the submucosa to the esophageal surface. In addition, the absence of vomiting, retching or esophageal instrumentation prior to the patient's initial presentation and the ensuing negative workup for coagulopathy and cardiac causes suggests that the esophageal hematoma developed secondary to his anticoagulation and/or thrombolysis use.

Chest radiography plays a limited role in IEH, although a widened mediastinum and hyperlucent mass at the expected location of the esophagus may be suggestive [4]. Water-soluble contrast-enhanced esophagography can be useful in patients who do not first present with dysphagia/odynophagia. A well-defined filling defect indicates a focal hematoma. In the event of an intramural dissection of the esophagus, the creation of a false and true lumen is seen as a 'double-barreled esophagus' [4]. Although esophagography is a useful study, many patients present initially with symptoms of dysphagia, so the procedure may be difficult to perform.

Multidetector non-contrast Computed Tomography (CT) is the modality of choice in the evaluation of IEH [4, 5]. CT is a non-invasive imaging modality that is able to demonstrate the complex anatomy of the esophagus and assess its wall integrity while also evaluating for other mediastinal structures [5]. The characteristic finding is concentric (symmetric) or eccentric (asymmetric) mural thickening of the esophageal wall with a high attenuation esophageal mass that is non-enhancing (Figure 3) [1]. This mass may extend along the esophageal wall causing varying degrees of luminal obliteration [5]. An upper gastrointestinal endoscopy often reveals raised purplish red lesions most commonly located in the submucosa [1, 7]. In this case, an organized clot was found on endoscopy to be superimposed distally on circumferential ulceration of the esophageal wall causing obliteration of the lumen (Figure 4). Endoscopic ultrasound demonstrating a periesophageal lesion confined in the submucosal layer without communication to the aorta can be a useful supplemental study to aid in the diagnosis of IEH [8].

Anticoagulation and/or thrombolysis therapy is an unusual cause of IEH, with only a handful of documented cases [3, 9, 10], especially in the absence of endoscopic trauma or bleeding disorders. Chest pain, dysphagia/odynophagia and hematemesis are the most common initial symptoms of IEH [4, 7]. There are 35% of patients who present with this triad of symptoms while at least 50% of patients present with at least two of the above symptoms [6]. As such, it is important to differentiate IEH from cardiac ischemic events because although it is a relatively benign condition, it could be worsened by anticoagulation therapy [3, 6, 10]. In the setting where anticoagulation and/or thrombolysis are necessary, such as a PE, it may be beneficial to perform periodic reassessments for new-onset chest pain and dysphagia/odynophagia so IEH can be recognized and managed earlier.

The differential diagnoses for IEH includes all causes of symmetric and asymmetric esophageal wall thickening [1]. Most notably are artoesophageal fistula, Boerhaave syndrome,

achalasia, foregut duplication cysts and esophageal diverticula. It is also important to rule out the more immediate causes of chest pain such as an acute myocardial infarction by electrocardiogram and serial cardiac enzyme investigations.

Aortoesophageal fistula (AEF) is a rare subtype of IEH that may be a life-threatening cause of upper gastrointestinal bleeding secondary to aortic and esophageal conditions such as ruptured aortic aneurysm or foreign body aspiration respectively [1]. The classic diagnostic triad consists of midthoracic pain, sentinel hemorrhage and subsequent fatal exsanguination [11]. Chest radiograph demonstrates mediastinal widening and barium esophagography may reveal esophageal narrowing associated with extravasation of contrast material into the aorta [12]. Although CT is the diagnostic modality of choice, a definite fistula may not always be seen. If present however, the CT will show an enhancing submucosal esophageal mass with a fistulous tract between the aorta and the esophagus [1, 4]. The presence of a hyperattenuating enhancing esophageal mass with an associated aneurysm of the adjacent aorta may also be indicative of AEF [4]. In contrast to IEH, the classic endoscopic finding of AEF is a pulsatile esophageal mass [3].

Boerhaave syndrome is a complete transmural tear of the esophagus that occurs most commonly from increased intra-esophageal pressure such as violent straining or vomiting [1]. Patients typically present with a triad of symptoms that include vomiting, sudden severe chest pain and subcutaneous emphysema [1]. A chest radiograph may show mediastinal widening, left-sided pleural effusions or hydropneumothorax, subcutaneous emphysema and radiolucent streaks of gas along the aorta or in the neck [1, 13]. Esophagography with nonionic, water-soluble contrast agent is the diagnostic modality of choice, especially when perforation is suspected because barium increases the risk of mediastinitis [1, 13]. The presence of extravasation or submucosal collection of contrast material is diagnostic. If results are unequivocal, helical contrast-enhanced CT is performed to demonstrate extra-luminal gas and/or oral contrast medium in the lower mediastinum [8]. The presence of supradiaphgramatic, periesophageal air collection in the lower one-third of the esophagus is characteristic of Boerhaave syndrome [1].

Achalasia is an esophageal motility disorder that is characterized by the inability of the lower esophageal sphincter to relax. As a result, patients present with regurgitation in addition to dysphagia and chest pain. Chest radiograph may show mediastinal widening with double contour of mediastinal borders, anterior tracheal bowing or air-fluid levels in the mediastinum with small or absent gastric air bubbles [14, 15]. The diagnostic modality of choice is barium esophagography which demonstrates a markedly dilated esophagus, absent primary peristalsis and the classic 'bird-beak' deformity - a V-shaped, smooth and symmetrically tapered narrowing of the distal esophagus as it extends into the gastroesophageal junction (GEJ) [14, 15]. Helical CT with sagittal reconstructions may also show dilatation of the esophagus with a diameter greater than 4 cm, decreased or normal wall thickness, air fluid levels within a dilated esophagus and abrupt, smooth narrowing of the distal esophagus near the GEJ [14].

Foregut duplication cysts can occur anywhere along the alimentary tract and are the result of abnormal embryologic development where groups of cells are sequestered from the primitive foregut [16]. They are classified as cystic or tubular duplications. Although less common, tubular duplications may occasionally communicate directly with the esophageal lumen. Most patients are asymptomatic, but symptoms caused by obstruction, bleeding or an infected cyst may occur [16], resulting in dysphagia and pain. Grayscale ultrasound of the bowel wall is the best diagnostic imaging tool for intraabdominal duplication cysts, which occur 75% of the time [17]. The combination of an echogenic inner mucosal layer, hypoechoic muscular layer and echogenic outer serosa of the bowel wall is the pathognomonic finding for duplication cysts [17]. CT imaging is recommended for intrathoracic lesions, which are characterized by a homogeneous mass with low attenuation and a smooth border [16]. Tubular duplication cysts may show two esophageal lumina with a thin enhancing rim in the esophageal wall, delineating the mucosal linings [16]. Debris within the cysts may also be seen.

Esophageal diverticula are saccular outpouchings of the esophageal wall that are formed due to pulsion or traction of the esophagus. In addition, barium esophagography is the diagnostic imaging of choice [18]. The most common sign and symptoms include regurgitation and aspiration of undigested food, halitosis and hoarseness with or without a neck mass [19]. Pulsion-type diverticulum, such as Zenker diverticulum, results from increased intraluminal esophageal pressure and involves only the mucosa [17, 19]. A barium-filled sac with wide, rounded contours characterize pulsion diverticula on barium esophagography [18]. They also remain filled even after the esophagus has emptied of barium due to the absence of a muscular layer [16]. Traction diverticulum is formed due to fibrosis of bordering periesophageal tissue and involves all layers of the esophageal wall, including muscle [16]. On a barium esophagography, traction diverticula presents with a tented or triangular-shaped outpouching that tends to empty when the esophagus collapses [16, 18].

IEH has a generally good prognosis as most cases resolve spontaneously. However, in patients with multiple other co-morbidities, mortality can reach 7-9% [10]. Treatment is conservative with correction of the underlying cause, nothing per oral and intravenous alimentation [2, 3]. Antibiotics may be required only in severe cases. While surgery is rarely necessary in IEH, AEF has a very poor prognosis if surgical intervention is delayed [1]. As such, it is important to be able to differentiate between these two diseases. Subsequent CT imaging may be necessary to confirm the resolution of the hematoma.

TEACHING POINT

Anticoagulation therapy and thrombolysis are rare secondary causes of IEH, especially in the absence of endoscopic trauma or bleeding disorders. In the setting where anticoagulation or thrombolysis are necessary, such as a PE, it may be beneficial to perform periodic reassessments for new-onset chest pain and dysphagia/odynophagia so intramural esophageal hematoma can be recognized and managed earlier. It is also important to differentiate intramural esophageal hematoma from aortoesophageal fistula, as prompt surgery intervention in the latter leads to a better prognosis.

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FIGURES

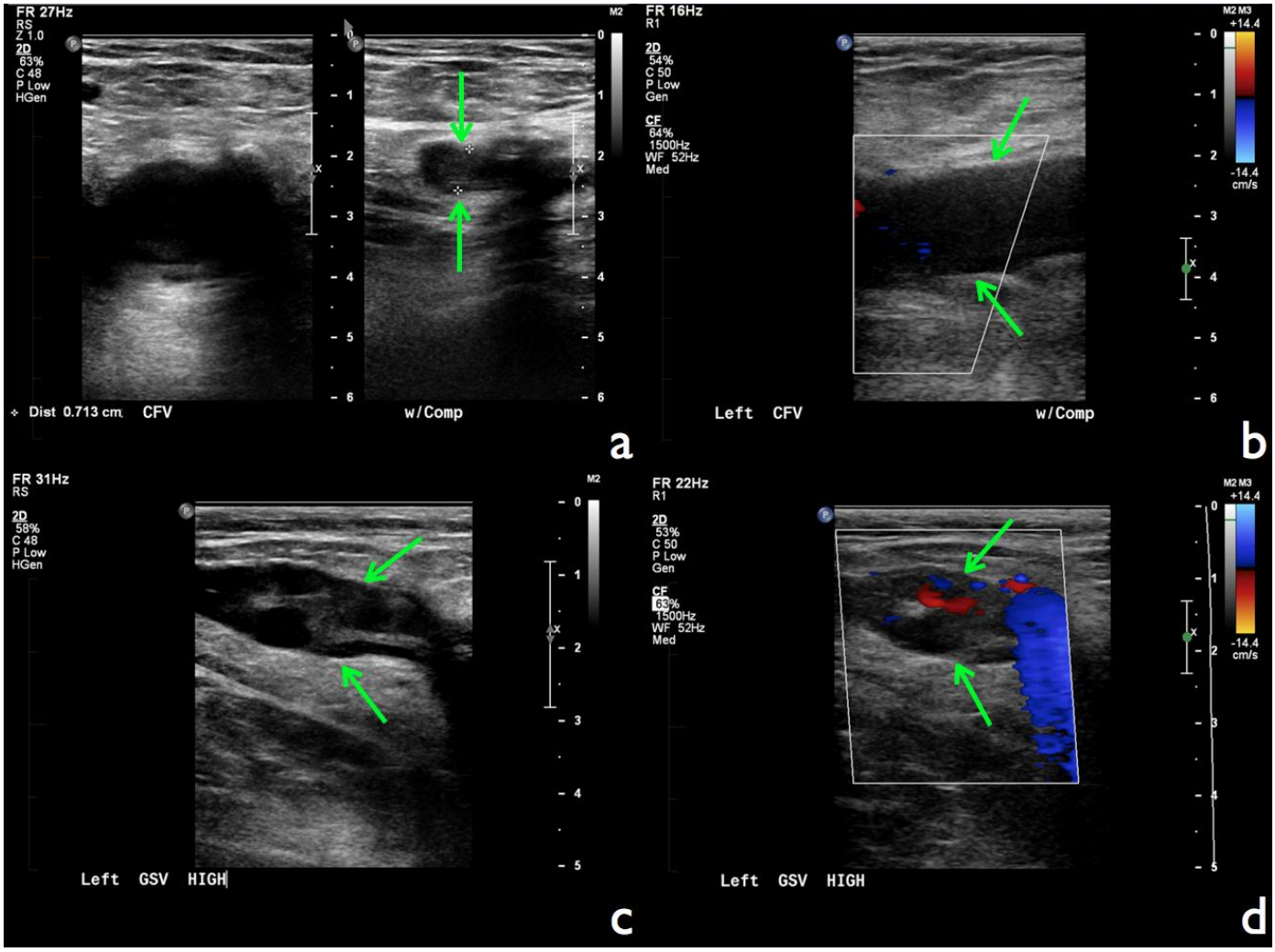

Figure 1: 73-year-old male with extensive deep and superficial vein thrombosis. (a) 2D gray scale venous ultrasonography of the left common femoral vein before and after compression demonstrates a non-compressible echogenic material that is consistent with a clot (arrows). (b) 2D color Doppler venous ultrasonography reveals obstruction of the left common femoral vein upon compression (arrows). (c) 2D gray scale venous ultrasonography of the left greater saphenous vein demonstrates echogenic material consistent with a thrombus (arrows). (d) 2D color Doppler venous ultrasonography shows obstruction of blood flow in the left greater saphenous vein (arrows).

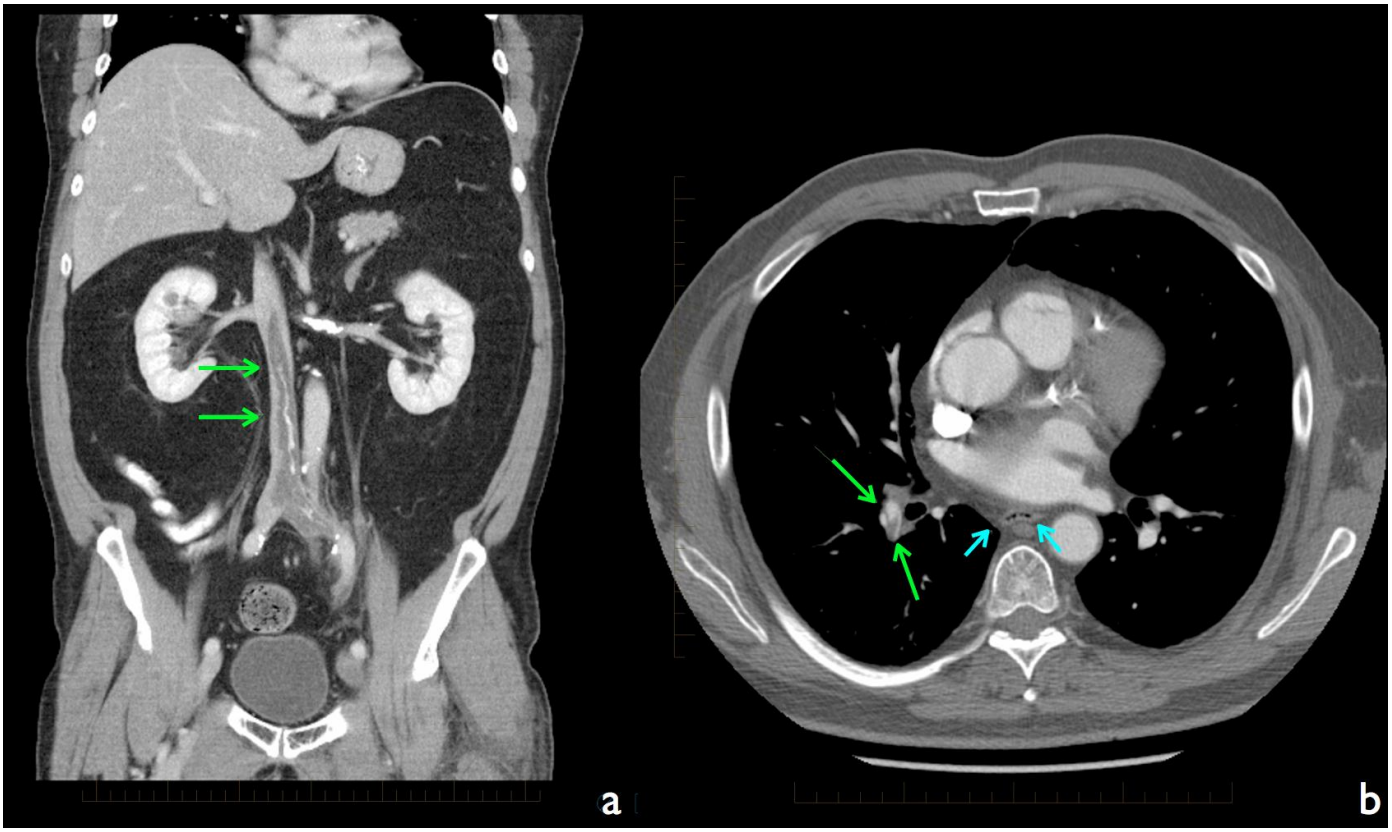

Figure 2: 73-year-old man with an IVC thrombus and pulmonary embolism. (a) Coronal contrast-enhanced CT of the abdomen and pelvis in the portal venous phase demonstrates an extensive, partially calcified thrombus (arrows) with chronic features within the inferior vena cava (IVC) beginning at the level of the renal vein and extending into the left common iliac vein and right internal iliac veins. (b) Axial contrast enhanced CT Angiogram (CTA) of the pulmonary arteries in the pulmonary arterial phase demonstrates filling defects located in the pulmonary arteries of the right main lower lobe (green arrows) consistent with a PE. The esophagus is unremarkable (blue arrows). (Protocol: 192 mA/195 mA respectively, 120 kVp, 5.0 mm thickness, 125cc of Isovue 370)

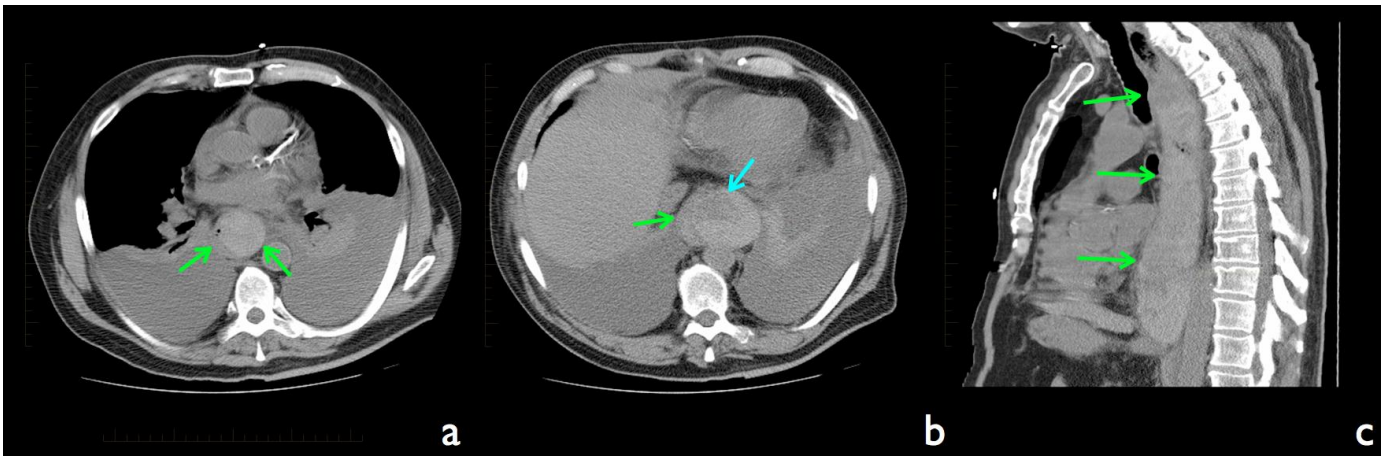

Figure 3: 73-year-old man with intramural esophageal hematoma. (a) Axial non-contrast CT of the chest demonstrates a high attenuation mass in the proximal esophagus consistent with an esophageal hematoma measuring 5.0 x 5.7 x 24.9 cm (anteroposterior (AP) x transverse (TR) x cranio-caudal (CC)) (green arrows). (b) Axial non-contrast CT of the chest demonstrates significant mural thickening of the esophageal wall (green arrow) as well as a high attenuation mass in the distal esophagus (blue arrow). (c) Para-sagittal non-contrast CT of the chest demonstrates the extension of the high attenuation mass along the mid to distal esophagus (green arrows). (Protocol: 436 mA, 120 kVp, 5.0 mm thickness)

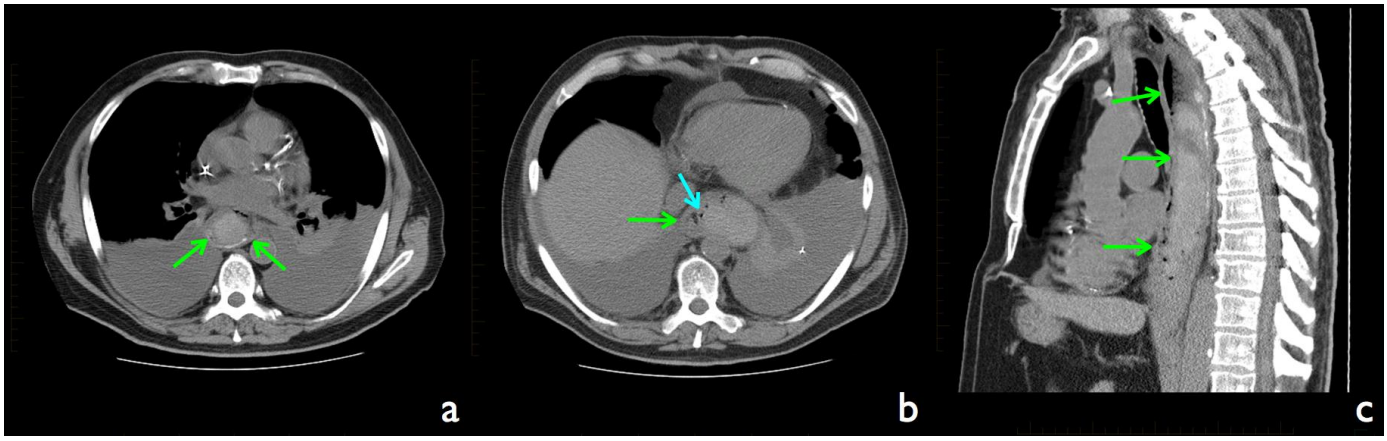

Figure 4: 73-year-old man with resolving intramural esophageal hematoma. (a) Axial non-contrast CT of the chest demonstrates a high attenuation mass measuring 3.5 x 4.8 x 19.2 cm (AP x TR x CC) in the proximal esophagus consistent with an esophageal hematoma (green arrows). (b) Axial non-contrast CT of the chest demonstrates a high attenuation mass in the distal esophagus (green arrow). Air fluid levels within the esophageal lumen suggest an intramural location of the hematoma (blue arrow). Note the decrease in size in comparison to Figure 3. (c) Para-sagittal non-contrast CT of the chest demonstrates the extension of the high attenuation mass along the mid to distal esophagus (green arrows). Note the decrease in affected length and presence of air fluid levels compared to Figure 3. (Protocol: 436 mA, 120 kVp, 5.0 mm thickness)

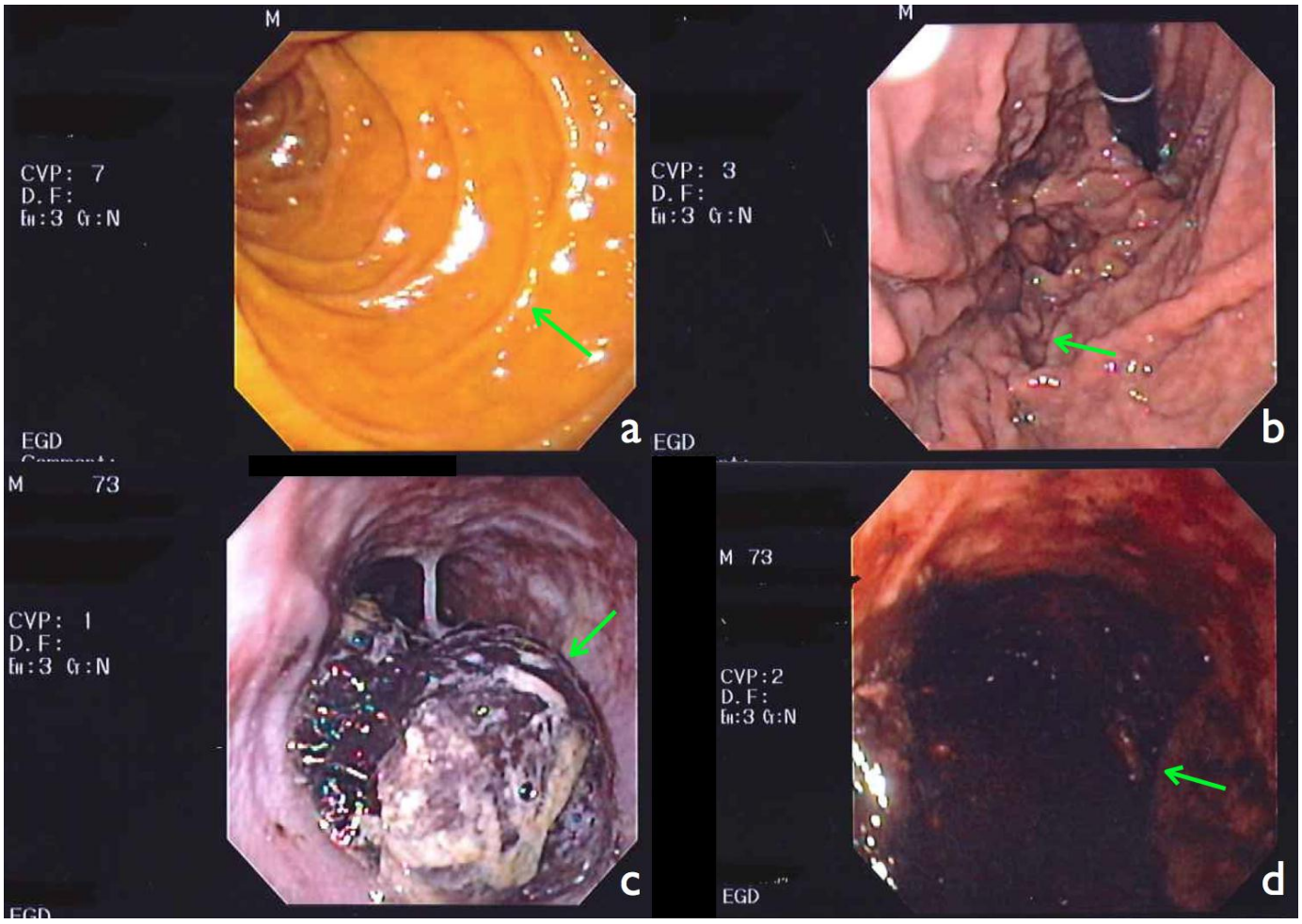

Figure 5: 73-year-old man with persistent chest pain and dysphagia. Upper gastrointestinal endoscopy demonstrates (a) areas of normal esophagus (b) circumferential erosion and ulceration of the esophagus (c) an organized clot in the esophagus (d) a clot resulting in the obliteration of the esophageal lumen.

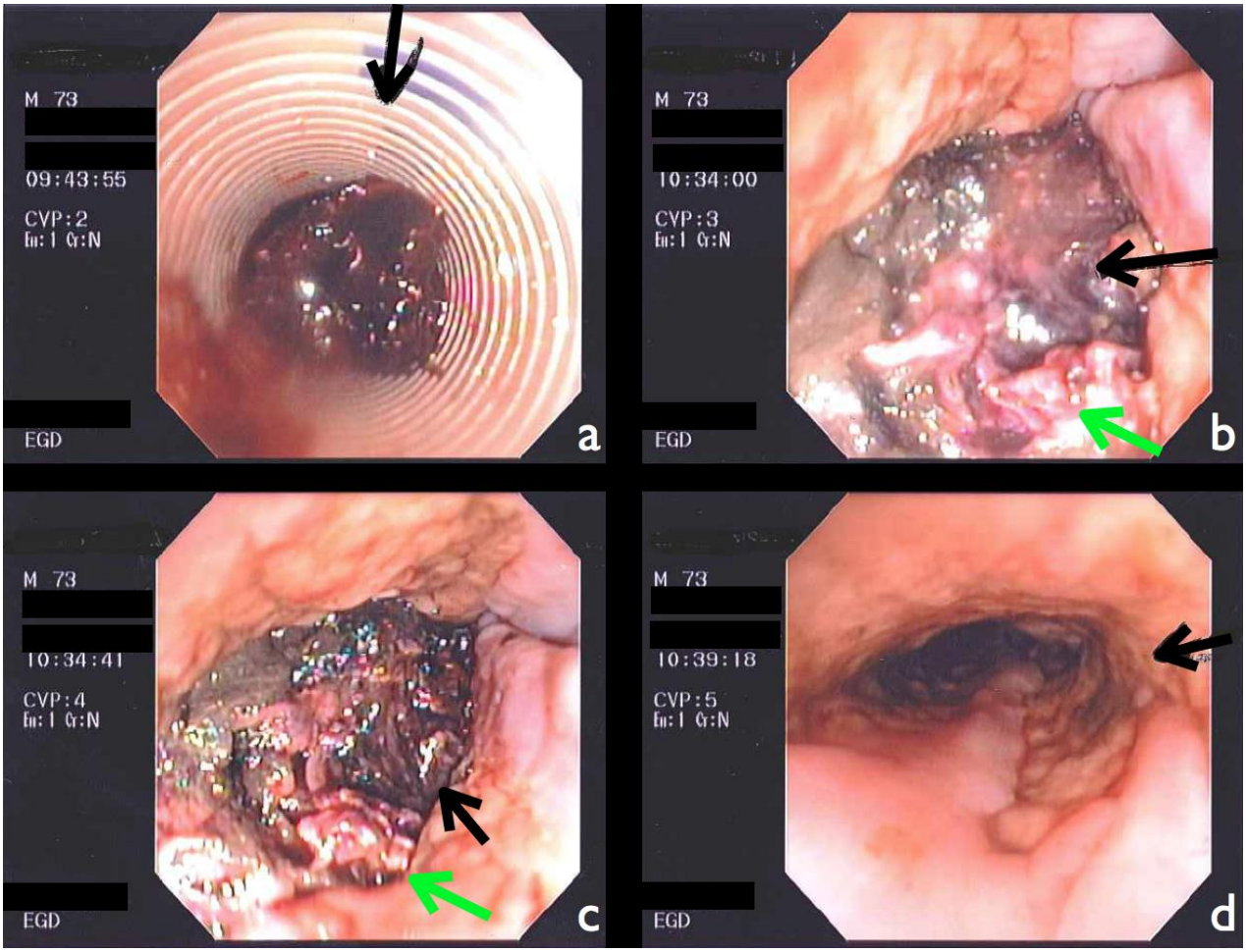

Figure 6: 73-year-old man with partial removal of a clot from the esophagus. Upper gastrointestinal endoscopy demonstrates (a) the removal of a clot (arrow) with the scope and (b and c) the remaining clot burrowed in the distal submucosa (black arrow) surrounded by circumferential ulceration of the esophagus (green arrow). (d) Circumferential erosion and ulceration of the esophagus can also be seen along the length of the esophagus proximal to the clot (black arrow).

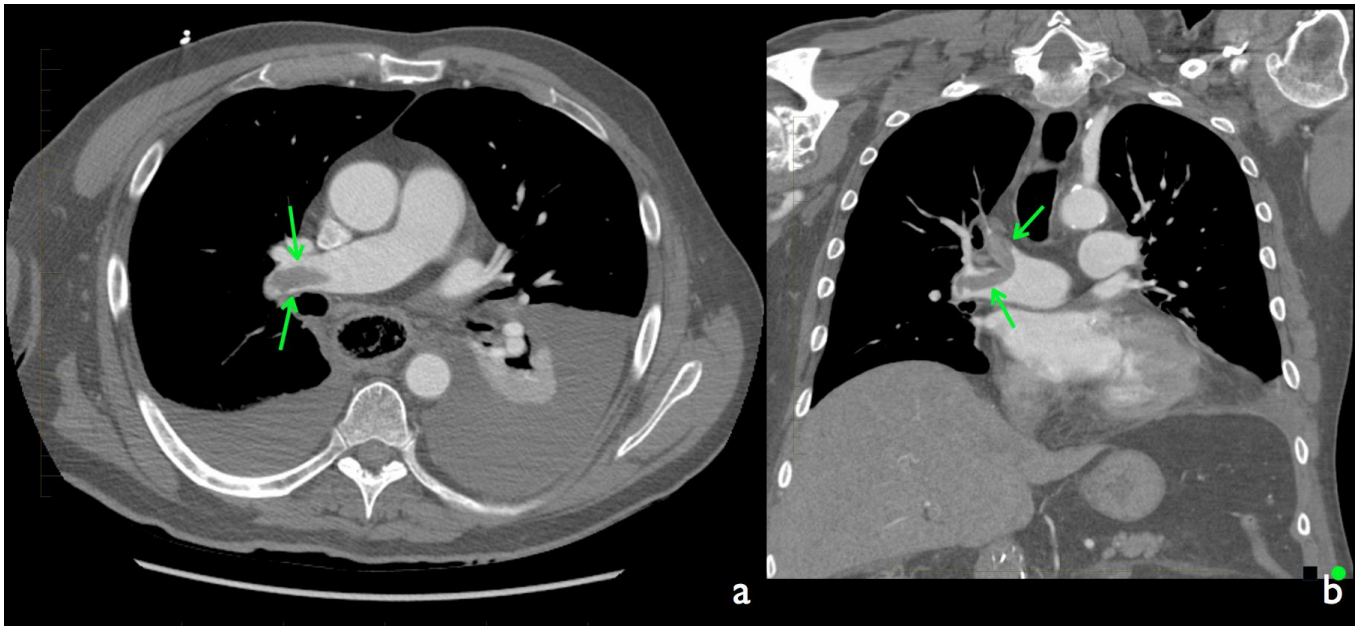

Figure 7: 73-year-old man with a recurrent pulmonary embolism. (a) Axial contrast enhanced CTA of the pulmonary arteries in the pulmonary arterial phase demonstrates filling defects in the right pulmonary artery (arrows). (b) Axial contrast enhanced CTA of the pulmonary arteries in the pulmonary arterial phase demonstrates filling defects in the right arteries supplying the upper, middle and lower lobes (arrows). (Protocol: 348 mA/573 mA respectively, 120 kVp, 5.0 mm thickness, 120cc of Isovue 370)

Differential Diagnoses/ Imaging Modalities	Chest Radiograph (CXR)/ Ultrasound	Fluoroscopic Studies	Computed Tomography (CT)
Intramural Esophageal Hematoma	Chest Radiograph • Mediastinal widening and hyperlucent mass at the expected location of the esophagus may be suggestive	Water-Soluble Contrast-Enhanced Esophagography: • Well-defined filling defects • 'Double-barreled' esophagus: creation of a false and true lumen as a result of intramural dissection of the esophagus	Non-Contrast CT*: • Concentric or eccentric mural thickening of the esophageal wall with a high-attenuated esophageal mass that is non-enhancing • Mass may extend along the esophageal wall causing varying degrees of luminal obliteration Contrast CT: • Extravasation of gas and contrast remain within esophageal wall
Aorto - esophageal Fistula	• Mediastinal widening	Barium Esophagography: • Esophageal narrowing associated with extravasation of contrast material	Non-Contrast CT*: • Enhancing submucosal esophageal mass with a fistulous tract between the aorta and the esophagus is definitive • Hyperattenuating enhancing esophageal mass with an associated aneurysm of the adjacent aorta may be indicative
Boerhaave Syndrome	Chest Radiograph • Mediastinal widening • Left-sided pleural effusions or hydro-pneumothorax • Radiolucent streaks of gas along the aorta or in the neck • Subcutaneous emphysema	Esophagography with Nonionic, Water-Soluble Contrast Agent*: • Extravasation of contrast material, submucosal collection of contrast material and esophagopleural fistula (most commonly on the left side)	Helical Contrast-Enhanced CT: • Extra-luminal gas and/or oral contrast medium in the lower mediastinum and possible the upper abdomen • Periesophageal, pleural, pericardial fluid collections • Rarely, may depict a tear in the esophageal wall • Presence of supradiaphragmatic periesophageal air collection in the lower 1/3rd of the esophagus is characteristic
Achalasia	Chest Radiograph • Mediastinal widening with double contour of mediastinal borders • Anterior tracheal bowing • Air-fluid levels in mediastinum, small or absent gastric air bubble	Barium Esophagography*: • Markedly dilated esophagus • Absent primary peristalsis • 'Bird-beak' deformity: V-shaped, conical and smooth with symmetrically tapered narrowing of distal esophagus extending to the gastroesophageal junction (GEJ)	Helical CT with Sagittal Reconstructions • Moderate to marked dilatation of esophagus with diameter > 4cm • Decreased or normal wall thickness • Air-fluid level within dilated esophagus • Abrupt, smooth narrowing of distal esophageal segment near GEJ
Foregut Duplication Cysts	Grayscale Ultrasound* • pathognomonic 3 layers of the bowel wall: echogenic mucosa, hypoechoic muscular layer and echogenic serosa • Debris within the cyst is common		Contrast-Enhanced CT: • Homogeneous mass with low attenuation and a smooth border • Ovoid or rounded cystic lesion with relatively thick, enhancing wall • Fluid-debris-blood levels may be seen • Tubular duplication cysts: may show two esophageal lumina with a thin enhancing rim in the esophageal wall, respresenting mucosal linings
Esophageal Diverticulum		Barium Esophagography*: • Pulsion diverticulum: barium-filled sac with wide, rounded contour • Traction diverticulum: barium-filled tented or triangular-shaped outpouching	

Table 1: Differential Table for Intramural Esophageal Hematoma
* Diagnostic Imaging of Choice

Etiology	Traumatic (endoscopic manipulation), emetogenic (intermediate stages between Mallory Weiss syndrome or Boerhaave syndrome), abnormal hemostasis-related (hemophilia), aorta-related (aortoesophageal fistula) and spontaneous (no identifiable cause)
Incidence	Rare
Gender ratio	Slight female predominance
Age predilection	Middle-aged
Risk Factors	Vomiting and straining, endoscopic manipulation, bleeding disorders
Treatment	Conservative medical management; stop any causative drugs, nothing per oral, parenteral nutrition, treat underlying cause
Prognosis	Good
Findings on images	Chest Radiograph • Mediastinal widening and hyperlucent mass at the expected location of the esophagus may be suggestive Water-soluble Contrast-Enhanced Esophagography – well-defined filling defect: • Well-defined filling defects • 'Double-barreled' esophagus: creation of a false and true lumen as a result of intramural dissection of the esophagus Non-Contrast Computed Tomography: • Concentric or eccentric mural thickening of the esophageal wall with a high attenuation esophageal mass that is non-enhancing • Mass may extend along the esophageal wall causing varying degrees of luminal obliteration

Table 2: Summary Table for Intramural Esophageal Hematoma

ABBREVIATIONS

AE = Aortoesophageal fistula
CT = Computed Tomography
CTA = Computed Tomography Angiogram
DV = Deep Vein Thrombosis
IE = Intramural Esophageal Hematoma
IN = International Normalized Ratio
IV = Inferior Vena Cava
PE = Pulmonary Embolism
PT = Partial Thromboplastin Time
SV = Superficial Vein Thrombosis

KEYWORDS

Esophageal hematoma; intramural; spontaneous; anticoagulation; thrombolysis

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