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CASE REPORT

"Spontaneous Intramural Esophageal Hematoma Associated with Uncontrolled Hypertension: Case Report and Review"

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Introduction: Intramural esophageal hematoma (IEH) is a rare entity in the spectrum of esophageal injuries that often present with symptoms, such as acute chest pain, dysphagia and/or hematemesis. Herein, we present an interesting case of spontaneous IEH in a patient with uncontrolled hypertension, in the absence of established predisposing factors.

Case Presentation: 61-years female, with a history of hypertension but no coagulopathy or anticoagulant/antiplatelet use, presented with sudden onset chest pain associated with complete dysphagia for 6 hours. She was found to have uncontrolled hypertension, with a blood pressure of 220/110 mmHg. She underwent computed tomography imaging of the chest followed by upper gastrointestinal endoscopy, which confirmed a diagnosis of spontaneous IEH. Conservative management and aggressive blood pressure control led to complete resolution of IEH within four weeks.

Conclusion: This case highlights the importance of considering spontaneous IEH in the differential diagnosis of acute-onset chest pain with dysphagia in hypertensive patients, particularly in those with atypical presentations.

Keywords: esophageal hematoma, esophageal dissection, uncontrolled hypertension, dysphagia, chest pain

Introduction

Intramural esophageal hematoma (IEH) is a rare condition that ranges from mucosal tear (Mallory Weiss tear) to transmural perforation (Boerhaave syndrome).[1] IEH can occur spontaneously or after trauma due to ingested foreign body and after esophageal instrumentation.[2–4] Spontaneous IEH can occur in patients who have intrinsic coagulopathy or are taking anticoagulant or antiplatelet medications.[5,6] IEH typically presents with abrupt retrosternal chest pain and/or hematemesis. The characteristic triad of chest pain, dysphagia, and hematemesis is reported in about 35% of patients, and two of these three symptoms are seen in nearly 80% of cases.[7,8] Acute retrosternal chest pain is a common symptom of acute myocardial infarction, aortic dissection, and pulmonary embolism and should be ruled out with a careful history, physical examination, and appropriate diagnostic tests, as these conditions are much more common than IEH. The presence of dysphagia, odynophagia, or hematemesis can be useful in differentiating IEH from other critical diseases in conjunction with other diagnostic modalities.[9]

We present a rare case of spontaneous IEH associated with uncontrolled hypertension with no other established predisposing factors.

Case Presentation

A 61 year old female (body mass index- 29 kg/m^2) with a history of hypertension presented to our emergency department with acute onset of severe chest pain associated with complete dysphagia lasting six hours. There had no history of vomiting or hematemesis. She had uncontrolled hypertension due to irregular antihypertensive medication intake over the previous few weeks. The patient was not taking anticoagulants or antiplatelet medications. The patient had no history of

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gastrointestinal, cardiac, pulmonary, or bleeding disorders. On examination, her blood pressure was 220/110 mmHg. Results of cardiac, respiratory, and abdominal examinations were normal. Fundoscopic examination revealed marked arteriolar attenuation and soft exudates with flame-shaped hemorrhages but no papilloedema. Based on her medical history and clinical examination, a differential diagnosis of acute coronary syndrome, aortic dissection, pulmonary embolism, and intramural esophageal hematoma was considered.

Her baseline hemoglobin was 11.8 g/dl with a normal platelet count and coagulation profile. Her liver enzyme, troponin I, electrocardiogram, and chest radiography results were normal.

The patient underwent contrast-enhanced computed tomography (CECT) of the chest with angiography, which showed a longitudinal intramural homogenous hyperdensity consistent with a hematoma extending from the proximal esophagus to the gastro-esophageal (GE) junction [Figure 1]. There was no evidence of esophageal perforation, aortic dissection, or aorto-enteric fistula on the imaging. Upper gastrointestinal (UGI) endoscopy revealed a single longitudinal bluish-colored submucosal column with thin overlying mucosa, causing a significant intraluminal bulge that began immediately below the upper esophageal sphincter and extended to the GE junction [Figure 2]. No mucosal breach was observed at or below the GE junction. There was no visible blood in the esophagus or stomach. Following this work-up, a diagnosis of spontaneous IEH was established.

The patient was conservatively managed and kept nil per oral for 24 hours, followed by a clear liquid diet with high-dose esomeprazole infusion (8 mg/h) for 48 hours. She was started on an intravenous nitroglycerine (NTG) infusion of 5 µg/min and titrated to 20 µg/min to control uncontrolled hypertension. After 48 h, the NTG infusion was tapered, and oral nifedipine 10 mg three times daily was started. The patient recovered uneventfully as her chest pain gradually subsided, and blood pressure normalized. Five days later, a second UGI endoscopy was performed, which showed ulcerated mucosa over the IEH in the proximal esophagus, with areas of partial resolution[Figure 3].

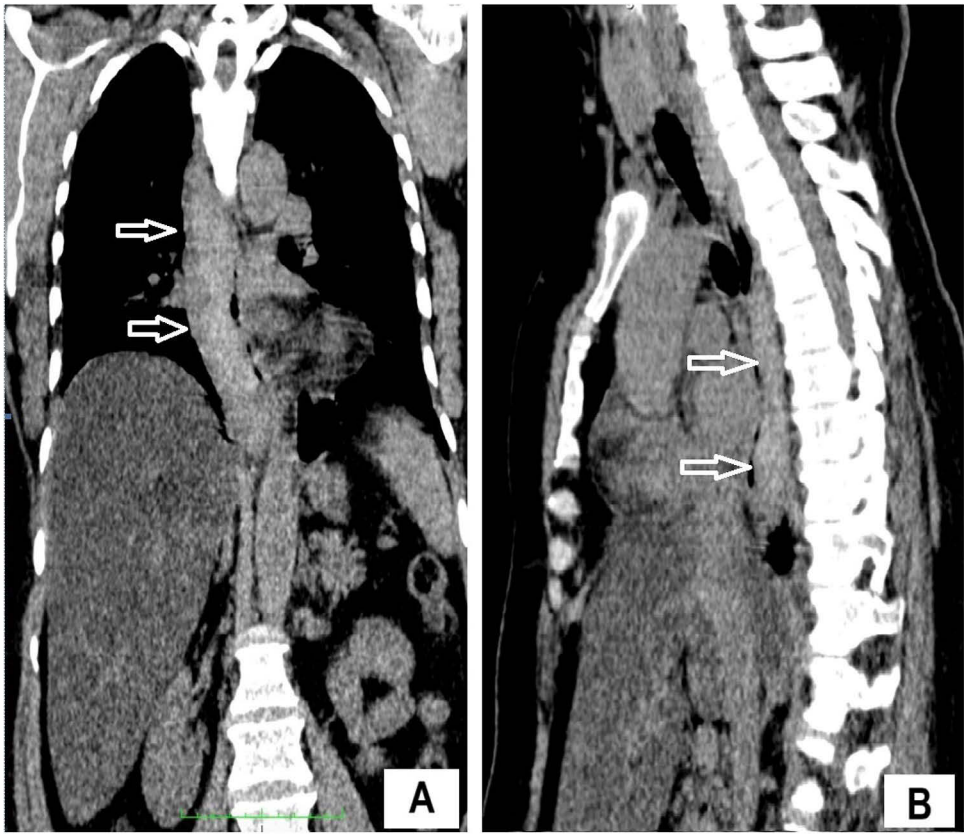

Figure 1 Computed tomography coronal section (**A**) and sagittal section (**B**) image showing longitudinal intramural homogenous hyperdensity (white arrows) suggestive of hematoma seen extending from proximal esophagus to gastroesophageal junction.

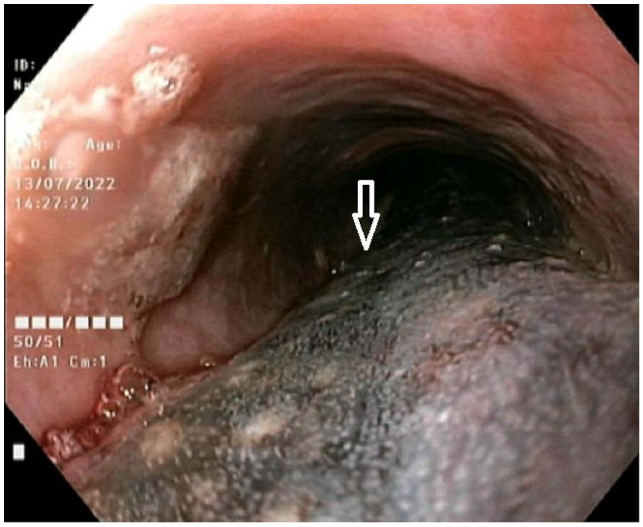

Figure 2 Initial endoscopy (day 0) showing an intramural esophageal hematoma with a bluish-tinged (white arrow) longitudinal column.

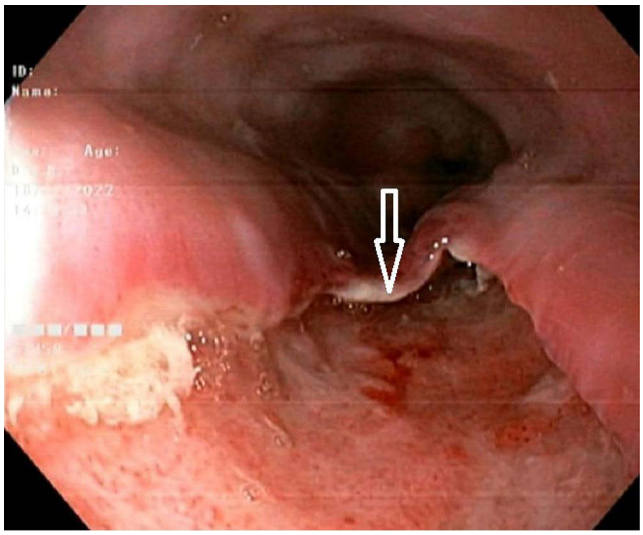

Figure 3 Repeat endoscopy (day 5) showing a mucosal defect with exposed submucosa (white arrow) in the proximal esophagus.

She was discharged on a full-liquid diet on the 6th day with good control of blood pressure during oral therapy. Four weeks later, repeat endoscopy revealed a normal esophagus with complete resolution of the intramural hematoma [Figure 4]. There were no adverse or unanticipated events.

Discussion

Intramural esophageal hematoma is a rare entity that occurs due to bleeding and hematoma formation within the esophageal wall. IEH was originally described as an intramural rupture of the esophagus by Marks and Keet in 1968.[10] It is also known as dissecting intramural hematoma, intramural esophageal dissection, esophageal apoplexy, or submucosal hematoma.[11]

IEH is associated with multiple etiologies and can generally be classified as spontaneous or secondary. Secondary IEH can be due to traumatic injury caused by sharp foreign body ingestion, impaired swallowing of a large, bulky bolus, or ingestion of toxic substances. Esophageal instrumentation due to deep endoscopic biopsies, esophageal dilation, endotracheal intubation, nasogastric tube insertion, endoscopic retrograde cholangiopancreatography, endoscopic ultrasound-guided mediastinal fine-needle aspiration/biopsy, and transesophageal echocardiography have been shown in case reported to cause traumatic IEH.[12–17]

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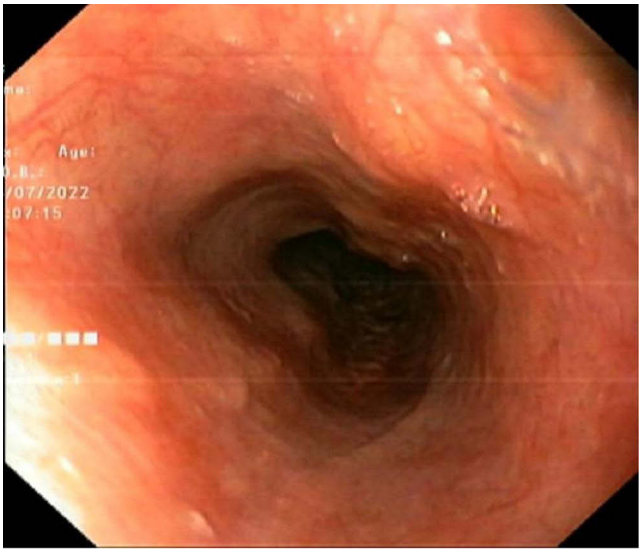

Figure 4 Repeat endoscopy (at 4 weeks) showed complete resolution of the esophageal lesion with no residual scarring.

Spontaneous IEH, on the other hand, is caused by the rapid alteration of intrathoracic and intraesophageal pressure precipitated by vomiting, retching, or coughing in the absence of known secondary causes. It can also present without any preceding predisposing events; thus, the term spontaneous IEH is used.[18] Female sex, intrinsic coagulopathy and use of anticoagulant or antiplatelet medications have been described as risk factors for spontaneous IEH.[19,20] Uncontrolled hypertension, leading to spontaneous epistaxis and intracranial bleeding, is well known; however, uncontrolled hypertension presenting as spontaneous IEH is unusual. The present case is possibly a rare case of spontaneous IEH caused by uncontrolled/accelerated hypertension, without any coexisting bleeding diathesis or use of anticoagulant or antiplatelet medications. Lu et al[21] reported a similar case of spontaneous IEH associated with uncontrolled hypertension and antiplatelet medication use, masquerading as aorto-esophageal fistula.

Uncontrolled hypertension contributes to IEH primarily by causing vessel fragility and exacerbating bleeding from a small initial injury. The high blood pressure acts as a predisposing or aggravating factor rather than a direct, sole cause. The pathophysiology involves chronic, uncontrolled hypertension which leads to damage and increased fragility of the small blood vessels (capillaries and arterioles) within the submucosal layer of the esophageal wall. It is followed by a precipitating event with a sudden, sharp increase in intraesophageal or intrathoracic pressure which is usually the immediate trigger for a small initial bleed. Any forceful activity like vomiting, retching, severe coughing, sneezing or straining can induce the trauma. The uncontrolled high arterial pressure can exacerbate the initial bleeding from a ruptured vessel, causing it to dissect along the submucosal plane and form a hematoma. As the hematoma expands, it can separate the mucosal layer from the underlying muscle, leading to characteristic symptoms of sudden-onset chest pain, dysphagia, and potentially hematemesis if the overlying mucosa eventually tears. This process is often also associated with the use of anticoagulant or antiplatelet medications, which further impair the body's ability to stop the bleeding.

Whereas intramural hematoma is characterized by separation of the esophageal wall layers with preservation of the mucosa, an esophageal tear involves mucosal rupture of variable depth. Such tears may be spontaneous—often precipitated by forceful vomiting, retching, or straining—or may occur secondary to medical instrumentation or foreign body injury. The clinical presentation of mucosal tear varies from mild chest discomfort to severe pain and hematemesis. UGI endoscopy plays a diagnostic and therapeutic role in this group of patients. A complete, full-thickness perforation, as in Boerhaave syndrome, represents a surgical emergency and carries significant morbidity and mortality.

The most common presenting symptoms of IEH are sudden onset of retrosternal chest pain and/or hematemesis. In 35% of cases, classic triad of chest pain, dysphagia, and hematemesis is reported while in 80% of cases, two out of three symptoms are present.[7,8] In the present case, the patient presented with acute onset of severe chest pain and complete dysphagia. Vomiting or hematemesis was not observed.

The diagnostic modality of choice in IEH is generally a CECT scan of the chest along with angiography, which commonly shows an intra-esophageal mass or filling defect resembling a double barrel/dual-lumen esophagus. It can also delineate the anatomical relationship between the esophagus, aorta, and mediastinal structures and rule out any esophageal perforation, aortic dissection, or aorto-enteric fistulae. Administration of oral contrast and imaging should be performed in instances where perforation is suspected.[22,23]

UGI endoscopy shows a bluish or purplish mass with or without a mucosal breach that partially or completely obstructs the esophageal lumen, indicating the submucosal location of the hematoma.[24] A classification system for the severity of luminal involvement in IEH has been proposed, depicting stage I, when there is an isolated hematoma; stage II, when the hematoma is surrounded by tissue edema; stage III, compression of the esophageal lumen; and stage IV, obliteration of the esophageal lumen by the hematoma.[25] Our patient had stage III esophageal hematoma.

Endoscopic ultrasonography (EUS) is a valuable tool for the diagnosis and management of esophageal submucosal hematomas. It can help to visualize a hematoma, determine its size and extent, and assess its characteristics, which can guide further treatment. EUS can also be used to differentiate hematomas from other submucosal lesions, such as tumors, and to guide interventions such as drainage if necessary. It can also assess any communication with aortic adventitia in suspected aorto-esophageal fistula.[21,26]

IEH generally has a benign course, and a conservative approach is appropriate because spontaneous luminal rupture followed by simple mucosal healing commonly occurs within 2–4 weeks. The initial management of the patient involved withholding oral intake, intravenous fluids, correction of associated coagulopathy, or administration of acid-suppressive medications along with blood pressure control (if indicated). Oral feeding was gradually allowed as the symptoms improved. In most cases, medical and conservative treatment results in full recovery. Recurrent bleeding or increased difficulty in swallowing should raise suspicion of hematoma leakage or rupture in the esophageal lumen and expanding hematoma, respectively, and should be treated as an acute emergency with airway protection and hemodynamic resuscitation.[27]

Therapeutic angiography may be necessary in cases of massive recurrent hematemesis to stop bleeding and hematoma expansion with transarterial embolization.[22] Surgery is usually associated with poor outcomes, but can become necessary for those who do not respond to conservative therapy or have massive hemorrhage that leads to hemodynamic instability or associated severe esophageal obstruction or perforation.[28]

Recently, there have been few case reports describing the use of endoscopic needle-knife incision therapy or iatrogenic mucosotomy in severely symptomatic cases of unruptured IEH. This is usually followed by clot and hematoma evacuation, helping in the early resolution of clinical symptoms and avoiding secondary complications such as super-imposed infection.[29,30]

The patient described in this report was conservatively treated. By 4 weeks, there was no residual hematoma on repeat UGI endoscopy, which showed a normal esophagus (Figure 4). Since accelerated hypertension is associated with epistaxis and/or spontaneous intracranial hemorrhage and spontaneous IEH itself is rare, it needs to be considered as a possibility in the setting of acute-onset dysphagia and chest pain with underlying uncontrolled hypertension, as in the index case. Although this case was thoroughly evaluated, it is acknowledged that EUS was not performed. Furthermore, the relationship between intramural esophageal hematoma (IEH) and accelerated hypertension remains speculative, underscoring the need for additional case reports and series to establish a more robust evidence base for this association.

Conclusion

In conclusion, IEH is a rare cause of submucosal esophageal injury that can present with acute-onset chest pain, dysphagia, or hematemesis, mimicking other conditions, particularly cardiopulmonary emergencies. This can result in delayed or even missed diagnosis. Therefore, a high index of suspicion is required for diagnosis and appropriate management. We report a rare case of spontaneous IEH possibly due to uncontrolled hypertension. This case highlights that in a patient with uncontrolled hypertension, presenting with sudden onset chest pain with dysphagia, one should keep in mind the possibility of spontaneous IEH even in the absence of other predisposing risk factors.

Data Sharing Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

Statement of Ethics

Ethical approval was not required for this study in accordance with the local or national guidelines.

Patient Consent

Written informed consent was obtained from the patient for publication of details of their medical case and any accompanying images.

Acknowledgment

The CARE Checklist has been completed by the authors for this case report.

Patient Perspective: "I came to this institute with my complaints of acute chest pain and difficulty swallowing thinking that I have acute MI. But thanks to AGI team, who correctly diagnosed me as having bleeding within the food pipe due to my uncontrolled hypertension. I was treated well and cured from my problem within few days".

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflict of interest with respect to the publication of this article.

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