

Received: 2018.11.14
Accepted: 2018.12.04
Published: 2019.02.20

Nasogastric Tubes Can Cause Intramural Hematoma of the Esophagus

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 84
Final Diagnosis: Intramural hematoma of esophagus
Symptoms: Chest pain • hematemesis
Medication: —
Clinical Procedure: Esophagogastroduodenoscopy
Specialty: Gastroenterology and Hepatology

Objective: Diagnostic/therapeutic accidents





Background: Intramural hematoma of the esophagus (IHE), a rare manifestation of acute mucosal injuries of the esophagus, can be caused by trauma such as endoscopic surgeries. Coagulation disorders increase the risk of IHE. The most common location of IHE is in the distal esophagus. The characteristic clinical triad of manifestations comprises acute retrosternal pain, odynophagia or dysphagia, and hematemesis. It is important to distinguish IHE from other acute conditions such as acute coronary syndrome, aortic dissection, and pulmonary embolism.

Case Report: An 84-year-old male was scheduled for coil embolization for an endoleak after endovascular aneurysm repair. For this reason, he was taking aspirin and warfarin. A nasogastric tube had been inserted during surgery and subsequently removed without any problems reported. Postoperatively, he experienced chest pain and hematemesis of sudden onset. Urgent esophagogastroduodenoscopy demonstrated a large, dark red, non-pulsatile, submucosal, esophageal mass in the area of the mid-esophagus with a little oozing. He was diagnosed as having an IHE; other possible diagnoses were excluded by contrast-enhanced computed tomography and aortography. He was treated with fasting, a proton pump inhibitor, and cessation of anti-thrombotic drugs; he recovered completely. The bleeding spot in the esophagus was in the area of the mid-esophagus, which was around the second natural constriction site. It was possible that the nasogastric tube had contact with the esophageal wall at this second natural constriction, and caused intramural esophageal bleeding.

Conclusions: Nasogastric tubes are not generally recognized as a cause of IHE. However, they can cause them, especially when a patient is taking anti-thrombotic drugs.

MeSH Keywords: Anticoagulants • Esophagus • Hematoma • Intubation, Gastrointestinal

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/914133>

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Background

Intramural hematoma of the esophagus (IHE), a rare manifestation of acute mucosal injuries of the esophagus, can be caused by trauma such as during endoscopic surgery, or occur spontaneously such as in individuals with coagulation disorders or individuals who are receiving antiplatelet therapy [1]. The characteristic clinical triad of manifestations of IHE comprises acute retrosternal pain, odynophagia or dysphagia, and hematemesis [2]. It is important to distinguish IHE from other acute conditions such as acute coronary syndrome, aortic dissection, and pulmonary embolism. IHE is usually diagnosed by esophagogastroduodenoscopy (EGD) and characteristically manifests as a dark red to purple, smooth, non-pulsatile, submucosal mass. We here report a patient with IHE that manifested after coil embolization under general anesthesia and which might have been caused by a nasogastric tube despite appropriate placement of that tube during surgery.

Case Report

An 84-year-old male was scheduled for coil embolization for endoleak after endovascular aneurysm repair. His medical history also included femorofemoral bypass for abdominal aortic aneurysm, thoracic aortic aneurysm, and hypertension. His medications included aspirin (100 mg daily), warfarin (2.5 mg daily), amlodipine (5 mg daily), losartan (50 mg daily), and lansoprazole (15 mg daily). He smoked 20 cigarettes per day and drank socially. There was no significant family history. On admission, the patient was afebrile with a heart rate of 53 beats per minute, blood pressure 142/52 mmHg, pulse oximetry 97% on room air, hemoglobin 13.7 g/dL, hematocrit 40.9%, platelet count 160 000/ μ L, and international normalized ratio 2.13. He underwent coil embolization under sevoflurane general anesthesia without preoperative stopping of aspirin and warfarin. The procedure was completed without immediate complications. A nasogastric tube (Salem Sump 16Fr; Covidien, Tokyo, Japan) was inserted without difficulty preoperatively. The gastric contents that drained through the nasogastric tube intraoperatively were normal and free of blood. On completion of the surgical procedure, the tube was removed, and oral secretions were cleared by suction, after which the tracheal tube was removed. There was no vomiting or violent coughing. Thirty minutes after the operation was completed, the patient reported chest pain and chest tightness of sudden onset, and then vomited a small amount of fresh blood. His hemoglobin was 13.1 g/dL, international normalized ratio 2.14, cardiac enzyme concentrations normal, and an electrocardiogram and echocardiogram were within the normal range. Contrast-enhanced computed tomography (CT) performed to rule out aortic dissection and aorto-esophageal fistula from thoracic aortic aneurysm demonstrated no changes relating to the known thoracic



Figure 1. Contrast-enhanced computed tomography image of esophagus. Horizontal arrow shows diffuse thickening of esophageal wall and luminal narrowing.

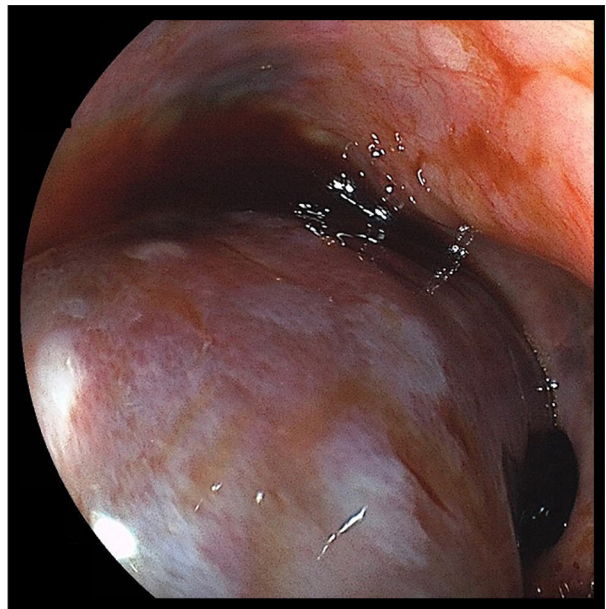


Figure 2. Endoscopic image of the intramural hematoma in the esophagus shows a large, dark red, non-pulsatile, submucosal mass that extended from dental arch 35 cm to 20 cm.

aortic aneurysm, but did show diffuse thickening of the esophageal wall with luminal narrowing (Figure 1). There was no evidence of other causes of his symptoms, such as pulmonary embolism. EGD demonstrated a large, dark red, non-pulsatile, submucosal mass extending from dental arch (DA) 35 cm to DA 20 cm with a little oozing from its surface (DA 22 cm) (Figure 2). An aortography was also performed to exclude an

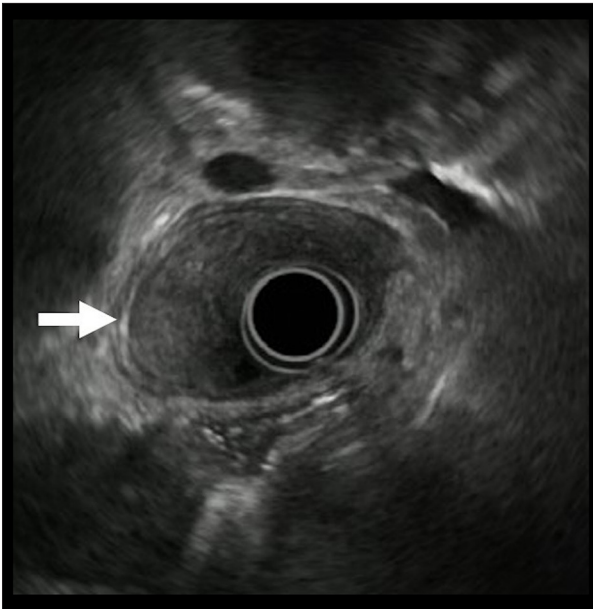


Figure 3. Endoscopic ultrasonography image of the esophagus. The arrow shows a homogenous hypoechoic lesion in the submucosal layer that was diagnosed as an intramural hematoma.

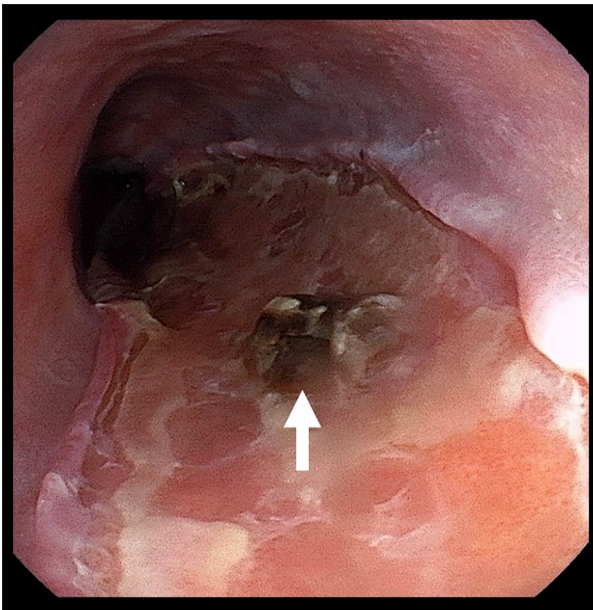


Figure 4. Image from endoscopy performed 1 week after development of the intramural hematoma of the esophagus. It shows the submucosal mass has ruptured, exposing submucosal tissue. The vertical arrow indicates a small dark spot in the area of the mid-esophagus (28 cm from dental arch) that may have been the bleeding point; however, it is not actively bleeding.

aortoesophageal fistula from the known thoracic aortic aneurysm; no such fistula was identified. He was therefore diagnosed as having an IHE and treated with fasting, a proton pump inhibitor, and cessation of aspirin and warfarin therapy.

Endoscopic ultrasonography performed 2 days later demonstrated a homogenous hypoechoic lesion in the submucosal layer (Figure 3). No branch vessels were identified as penetrating the hematoma by Doppler imaging. EGD performed again 1 week later showed that the IHE had ruptured, exposing submucosal tissue. A small dark spot in the area of DA 28 cm that may have been the bleeding point, but was not actively bleeding, was noted (Figure 4). Thereafter, he was permitted to eat and was discharged on the 12th day after the development of the IHE. He was treated with a proton pump inhibitor for 3 months after his discharge from the hospital. Cessation of aspirin and warfarin was continued. He did not have any symptoms after discharge. EGD performed 3 months later showed complete healing. Therefore, he resumed aspirin and warfarin therapy. He has not had any recurrence of IHE.

Discussion

Acute mucosal injury of the esophagus can manifest as Mallory-Weiss syndrome, Boerhaave syndrome, or IHE, with IHE being the rarest of these conditions [2]. IHE is classified as traumatic IHE or spontaneous IHE [1]. Most traumatic IHEs occur following esophageal instrumentation such as endoscopic surgeries or biopsies [1,3]. Spontaneous causes include increased intra-abdominal pressure caused by vomiting, coagulation disorders, and renal failure. Antiplatelet therapy is also thought to increase the risk of IHE [1–5]. Symptoms of IHE include acute retrosternal pain, odynophagia or dysphagia, hematemesis, and heartburn [2,4], the characteristic clinical triad being acute retrosternal pain, odynophagia or dysphagia, and hematemesis. Only 35% of patients with IHE have all 3 of these symptoms, whereas 99% have at least 1 of these symptoms [2]. The amount of hematemesis tends to be smaller than what is typically associated with Mallory-Weiss syndrome [3,6]. The important differential diagnoses are acute coronary artery disease, pulmonary embolism, varix rupture, and aortoesophageal fistula caused by aortic dissection [2,3,7]. EGD, the most widely-used modality for diagnosing IHE [4], characteristically demonstrates a dark red to purple, smooth, non-pulsatile, submucosal mass that bleeds easily on contact [3,4]. The risk of perforation should be considered when EGD is performed [3,8]. Endoscopic ultrasonography can demonstrate a homogenous hypoechoic tumor in the submucosal layer [3,4,9] and is useful for distinguishing IHE from malignant tumors or aortic pathology [3]. Chest radiographs and electrocardiograms are usually normal [3]. Contrast-enhanced CT is useful for identifying other serious conditions such as aortic dissection and

pulmonary embolism, demonstrating thickening of the esophageal wall, or an isodense mass with luminal narrowing in patients with IHE [2,4]. Most patients recover with conservative treatment such as diet restriction, fluid administration, acid suppression, and correction of any coagulation abnormality. Long-term complications are rare [2–4].

Our case of IHE presented suddenly after coil embolization under general anesthesia. The patient had never undergone EGD, and we assumed the IHE developed postoperatively because the patient had no symptoms before surgery and he reported chest pain and hematemesis of sudden onset after returning to the ward from the operating room. However, IHE is seldom a complication of surgery [1]. There were 2 possible causes of IHE in our patient. One was abnormal coagulation associated with anticoagulation therapy, which would place the IHE in the spontaneous category, and the other cause was associated with the nasogastric tube, and thus would be categorized as a traumatic cause. Regarding the first possible cause, our patient had been taking low dose aspirin and warfarin since undergoing a femorofemoral bypass. However, his level of anticoagulation was within the therapeutic range. Low-dose aspirin, aspirin plus dipyridamole, and clopidogrel have been reported as risk factors for spontaneous IHE [2]. As for the second possible cause, reported traumatic causes of IHE include endoscopic surgery, biopsy, Mallory-Weiss syndrome, achalasia, and foreign body ingestion [1,3]. There have been some reports of nasogastric tubes causing esophageal perforations because of inappropriate use, including long-term placements or an anatomical abnormality such as esophageal diverticula [10,11]. However, a nasogastric tube has not otherwise been a recognized cause of IHE. To our knowledge, there has been only 1 published report of a nasogastric tube possibly causing an IHE [1]. In that reported case, the authors considered that a nasogastric tube could be the cause of an IHE in a patient receiving anticoagulation therapy if the tube was suctioned while its tip was in the esophagus [1]. In our patient, the nasogastric tube was

placed smoothly into the stomach, there were no anatomical abnormalities of the esophagus, and suction was applied while its tip was in the stomach, not while it was in the esophagus. The most common location of IHE is in the distal esophagus (83%), as this area is not supported by enough surrounding structures [12,13]. The bleeding spot in our case was in the area of the mid-esophagus (28 cm from dental arch) which was around the second natural constriction site. It was possible that the nasogastric tube might have had contact with the esophageal wall at the second natural constriction, and might have caused intramural esophageal bleeding, as the patient was receiving 2 anti-thrombotic drugs.

Conclusions

Nasogastric tubes are not generally recognized as a cause of IHE. However, they can cause them. Therefore, if a patient has chest pain, dysphagia, or hematemesis after nasogastric tube insertion, attending physicians should consider IHE as one of the differential diagnosis, especially when a patient is on anti-thrombotic drugs.

Acknowledgements

We wish to acknowledge the assistance of Hideki Onodera, Jun Kataoka, Shun Watanabe, Takanori Uehara, Takashi Kojima, Takehiro Miyazaki, Yoshihisa Fujimoto, and Yasuhiro Norisue.

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Conflict of interest

None.

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