Endoscopic Treatment of a Gastric Dieulafoy's Lesion

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Summary

Gastrointestinal (GI) bleeding is associated with significant mortality and a prompt search and treatment of the etiology is important. Upper GI endoscopy is the gold standard for diagnosis and treatment after initial resuscitation of the patient. In a majority of cases, the cause will be easily identified during endoscopy. Dieulafoy's lesion, a caliber persistent artery in the submucosa, is a rare but important cause of intermittent painless massive GI bleeding. Due to its intermittent nature, it can easily

be missed and often requires multiple endoscopic evaluations. We present a case of one such patient with subsequent endoscopic findings and successful treatment along with a review of the literature.

Keywords: Endoscopy; Hematemesis; Dieulafoy's lesion; Stomach

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Introduction

Upper gastrointestinal (GI) bleeding is a medical emergency that warrants prompt identification of cause and appropriate treatment. Although etiologies differ with the age of the patient, nature of the bleed, and preceding history; the majority of causes of upper GI bleeding can be attributed to sequela of peptic ulcer disease, variceal disease, and malignancy. Endoscopy remains the gold standard for the diagnosis and treatment of upper GI bleeding. In a majority of cases, the apparent cause of the bleed is easily identified during the endoscopic evaluation. However, at times, patients can present as diagnostic enigmas with repeat negative endoscopy findings despite evidence of repeated episodes of hematemesis, melena, or a dropping hematocrit.

Case report

A 74-year-old woman, with controlled hypertension and diabetes, was referred to the endoscopy unit at Tenwek Hospital with a history of five episodes of vomiting frank blood. The initial episode was reported

to have been approximately 150mL and subsequent ones were approximately 75 to 100mL each. She described general body weakness but denied any preceding vomiting or abdominal pain. She denied any prior history of vomiting blood, early satiety, or change in stool color prior to the first episode. She also denied any previous use of alcohol or report of liver disease. Her medications were atenolol, nifedipine, losartan, metformin, and mixtard insulin. She denied using aspirin, clopidogrel, or any nonsteroidal anti-inflammatory drugs (NSAIDS). On presentation, she was breathing with ease, appeared pale and had no stigmata of liver disease. Her blood pressure was 143/59 mmHg, temperature 36.7°C and pulse rate 80beats per minute. Her abdomen was slightly distended, soft and with no organomegally. Her hemoglobin level was 11.9g/dl, platelets count of 167,000, international nationalized ratio (INR) of 1.28, sodium 137mmol/L and elevated potassium 5.6mmol/L.

The patient was resuscitated with two litres of normal saline and intravenous omeprazole 40mg at the

emergency department. Patient was prepared for an emergent endoscopy which revealed a small vascular bleb on the greater curvature of the gastric body at 49cm from upper incisors. Although the lesion was not actively bleeding, it was identified as the apparent cause of bleeding. No surrounding inflammation and no blood in the stomach was found. The area was marked with a tattoo for easy identification in case of rebleeding. The rest of the stomach had mild diffuse erythema. The esophagus and duodenum up to its third portion were all grossly normal. The patient was started empirically on Helicobacter pylori kit for 14 days and planned to undergo repeat hemoglobin checks after 1 and 4 weeks. She was also advised to avoid use of NSAIDs.

Ten days later, the patient had another episode of vomiting fresh blood of reported approximately 500mL and her hemoglobin dropped to 8.0g/dL. She underwent repeat endoscopy which revealed the small vascular bleb on greater curvature of gastric body at 49 cm which was not actively bleeding but had an adherent clot (Figure 1a). Three hemostatic Resolution Clips® were placed successfully without any complications (Figure 1b & c). She experienced no further episodes of hematemesis nor were meleanotic stools reported.

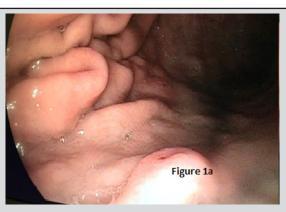






Figure 1a: Endoscopic View of Vascular Bleb Figure 1b: Hemostatic Clips on Lesion Figure 1c: Retroflexed View after Clip Application

Discussion

Dieulafoy's lesions, exulceratio simplex, are a rare but challenging cause of upper GI bleeding due to their intermittent nature. Upper GI bleeding carries a significant mortality depending on the etiology and the patient's co-morbid conditions. Dieulafoy's lesions contribute approximately 6% of non-variceal upper GI bleeds and 1-2% of all GI bleeding (1-5). These lesions, though first described by Gallard in 1884, have been attributed to Dieulafoy - a French surgeon who described three cases in 1898 (6). These are aberrant large, tortuous, submucosal vessels that can erode through and cause massive, painless and

intermittent GI bleeding (7, 8). Histologically, the lesions are thick walled large caliber-persistent artery in the submucosa with subintimal fibrosis without surrounding inflammation but can be ulcerated (3,9). The large submucosal vessel pulsations together with mechanical or chemical injury result in erosion of the overlying mucosa and resultant hemorrhage from the vessel. There is still controversy regarding if this is a congenital or acquired age related defect.

Dieulafoy's lesions can occur in patients of any age, with a slight male predominance and no familial tendency. They have been reported in almost every part of the gastrointestinal tract. The majority have been reported in the lesser curvature of the stomach where 80-95% are located approximately 6cm distal to the gastro-esophageal junction with blood flow directly from the left gastric artery (4, 10, 11). Other locations in the GI tract where they have also been reported include the duodenum, colon, jejunum and ileum, rectum and anal canal (10, 12-14). Lesions outside of the gastrointestinal tract have also been reported in the bronchus, which can present with episodes of massive hemoptysis and are discovered on bronchoscopy (15). Mortality rate from Dieulafoy's lesions range is variable and is due to massive bleeding with different studies reporting rates as high as 35% but with advancement and more access to endoscopy, mortality has been reducing with a rate of 8.6% compared to historical mortality rates of 80% (16, 17). Dieulafoy's lesions typically have an intermittent nature of bleeding and on average, its takes three or more upper GI endoscopies to diagnose (4). Occasionally one would find a spurting arterial bleeder during repeat evaluation or more frequently a nonbleeding vascular bleb with or without an adherent clot. However, due to the lesion's previously mentioned intermittent nature of bleeding, initial endoscopy can be non-revealing and only after repeat careful evaluations would the apparent cause be identified. Another challenge in the identification can also be due to the massive amount of blood in the stomach obscuring it. In the medical literature, patients with bleeding Dieulafov's lesions would frequently return after an apparently normal endoscopy with another bout of hematemesis in a median duration of 4 days from the initial evaluation (3).

Treatment of Dieulafoy's lesions depends on the location of the lesion. Historically, surgery was the cornerstone of arresting all bleeding Dieulafoy's lesions. Currently, endoscopy is the gold standard for the treatment of gastric lesions with low reported recurrence rates of less than 10% (18, 19). Several endoscopic techniques are currently instituted including injection of vasoconstrictors like epinephrine, application of hemostatic clips, and banding. The choice of the modality used depends on their availability and the endoscopist's skills and preference. Use of more than one modality in the treatment has been shown to have a lesser risk of recurrence when compared with monotherapy of either of the modalities (20). Mechanical modalities, hemostatic clips and banding, have been proposed to be the initial treatment of choice arguing they would have better control as it results in a smaller lesion compared with injection therapy or thermocoagulation. Small intestine lesions can be diagnosed with capsule endoscopy and surgery is the most common option for treatment, where small bowel enteroscopy is not available.

In the case of our patient, she was not using aspirin, clopidogrel, or any other anticoagulants. She did not have any warning signs in her history to suggest any malignancy and had no signs of liver disease. Our patient had a greater curvature lesion, unlike the typical location in the reported majority in the lesser curvature. Arterial hypertension, which was present in our patient and is a co-morbidity in over 90% of the patients with bleeding Dieulafoy's lesion, was not found to be a risk factor for re-bleeding in Dieulafoy's lesions (3,8). During the initial endoscopy, no features suggestive of ongoing bleeding and therefore no intervention done then. After the application of the hemostatic clips, the patient did well. At follow-up, she has not had any more episodes of hemorrhage.

Conclusion

Although uncommon, Dieulafoy's lesions should always be considered as a differential in any patient with massive painless GI bleeding. Due to its intermittent nature, initial evaluation may not identify the lesions thus requiring repeat exams. Endoscopy is the modality of choice for the identification and treatment of gastric Dieulafoy's lesions. A hope of this case presentation and literature review is to encourage providers to remember this potential cause and to facilitate its management when dealing with such dilemmas of chronic anemia, gastrointestinal bleeding, and ongoing hematemesis. This case presentation represents a successfully managed patient whose typical course characterizes the difficulty in identifying Dieulafoy's as the cause of ongoing bleeding.

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