Vasopressors: A Novel Tool for the Provocation and Diagnosis of Dieulafoy Lesions

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ABSTRACT

A Dieulafoy lesion is an uncommon but potentially life-threatening cause of an acute gastrointestinal (GI) bleed. The lack of an associated mucosal ulcer can make locating and treating an actively bleeding Dieulafoy lesion difficult. Historically, a heparin bolus can be used to assist detection by provoking an active upper GI bleed. This case report examines using vasopressors for provocation of a Dieulafoy lesion in a 58-year-old male with persistent upper GI bleeding despite multiple endoscopies, angioembolizations, and abdominal exploration.

BACKGROUND

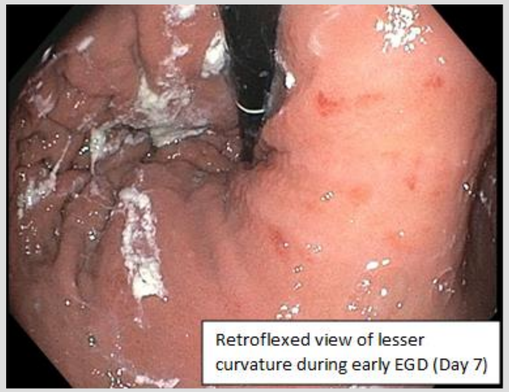
A Dieulafoy lesion is an aberrant dilation of submucosal blood vessels that erode through the overlying epithelial layer without a visible associated mucosal ulcer. It makes up for 1-2%[i] of all gastrointestinal bleeds, and while infrequent, can be life threatening if unnoticed or untreated [ii]. Most commonly, Dieulafoy lesions are found in the stomach, however rare cases have been described throughout the GI tract. The standard for diagnosing a Dieulafoy lesion is via endoscopy[iii]. Current diagnostic criteria for a Dieulafoy lesion via endoscopy includes:

- Active arterial spurting or micropulsatile streaming from a mucosal defect less than 3 millimeters or through normal surrounding mucosa.
- Visualization of protruding vessel with or without bleeding, within a minute, mucosal defect or through a normal surrounding mucosa
- The appearance of fresh, densely adherent clot with a narrow point of attachment to a minute, mucosal defect or next to normal appearing mucosa.

For treatment, surgical resection was historically the first-line choice of treatment. Now with advances in endoscopic procedures, surgical resection is employed rarely in cases refractory to endoscopic or angiographic methods. This often entails some form of a gastrectomy. Diagnosing and treating a Dieulafoy lesion proves a challenge, and this case discusses a possible novel technique for the diagnosis and localization of a Dieulafoy lesion.

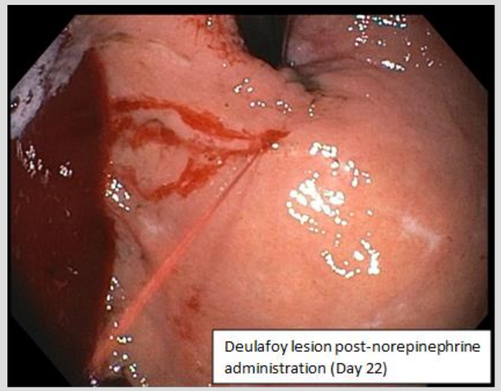
HOSPITAL COURSE

Hospital Day	Summary of Events
0	3U RBC, EGD (no active bleed)
1	2U RBC
2	4U RBC, 1U FFP, EGD (duodenal ulcer w/ adherent clot), IR angioembolization of gastroduodenal, R gastroepiploic arteries
3 - 6	5U RBC
7	1U RBC, EGD (no active bleeding)
9	7U RBC, 3U FFP, 1U Plt, exploratory laparotomy (negative), EGD (no active bleed, re-cauterization of duodenal ulcer with Gold probe)
10	2U RBC
12	IR IVC filter placement
13	2U RBC, Tagged RBC scan (negative)
14	Colonoscopy (negative)
15	2U RBC, Capsule endoscopy (negative)
16	8U RBC, 6U FFP, 3U CPP, EGD w/ heparin bolus 2000U x2 (negative) IR angioembolization of L gastric artery
17	2U RBC, EGD w/ heparin bolus 5000U x2 (negative)
19	2U RBC, IR angioembolization with heparin and t-PA provocation of right gastric, short gastric and left gastrohepatic trunk arteries x2
20-21	4U RBC
22	8U RBC, 8U FFP, 1U Plt, 1U CP, EGD w/ vasopressor provocation, clip and inj of Dieulafoy lesion
24	1U RBC



HOSPITAL DAY 22

On HD 22, the patient became hypotensive and anemic status post 8U RBCs, 8U FFP, 1U platelets, 1U CPP. Another EGD was performed, through which an attempt to provoke the bleeding vessel was attempted with norepinephrine. The EGD advanced to the second part of the duodenum. No active bleeding was noted at this time. A norepinephrine drip was begun, titrating by 2 mcg/min, and the mean arterial pressure was gradually elevated to > 100 with the systolic blood pressure remaining between 140 and 160. A max systolic blood pressure of 173 was recorded via arterial line. With the increased arterial blood pressure, a bleeding lesion was revealed on the lesser curvature of the stomach close to the gastroesophageal junction. This was viewed on retroflexion of the EGD. The lesion was subsequently banded and injected with epinephrine with resolution of the bleed. The norepinephrine drip was weaned, and the patient tolerated the procedure well without any cardiopulmonary complications. After HD 22, the patient required only one additional unit of RBCs and he was transferred to the floor. The patient was deemed medically stable for discharge to a skilled nursing facility on HD 28. During his hospital stay, a total of 53U RBCs, 18U FFP, 2U platelets, and 4U CPP were given. On follow up visit to the general surgery clinic two weeks later, it was noted that the patient reported complete resolution of his fatigue and he reported no further episodes of hematemesis or melanotic stools.



DISCUSSION

The diagnosis of a Dieulafoy lesion is difficult as often it requires multiple EGDs in order to visualize the site of bleeding[iv]. Methods such as heparinization as described by *Wright et al.* have been used to attempt to provoke these otherwise hidden submucosal ulcers.[v] Another confounding factor is that when undergoing endoscopy, patients are typically sedated, and many sedating agents also result in relative hypotension.

For this patient specifically, the resultant hypotension may have masked the bleeding Dieulafoy lesion as there was not enough pressure to adequately perfuse the submucosal ulceration. Consequently, many of the patient's episodes of hematemesis began after transitioning to an oral diet. It can be theorized that the increased perfusion and blood flow to the stomach with oral intake may have been the inciting factor for his episodes of hematemesis. Therefore, artificially creating an increased perfused state of the stomach mimics a state of ingestion. Hemodynamic agents involved in vasoconstriction may aid in counteracting the vasodilatory effects of anesthesia. Alpha-1 agonists such as phenylephrine have been historically used for the hypoperfusion state associated with septic and spinal shock and can be considered when attempting to increase systemic vascular resistance. More recently, adrenergic agonists like norepinephrine[vi] [vii] have been used as first line agents in septic shock to maintain perfusion pressures to organs. We would recommend against the use of vasopressin[viii], as historically it has been used to promote splanchnic vasoconstriction and prevent bleeding, thereby being counterproductive in provoking a GI bleed in the stomach. It should be noted that this method is not tested and should be done in a controlled setting, such as the ICU or operating room. For concerns of ischemic necrosis, having phentolamine as a counteractive agent to vasoconstriction may be useful.

In summary, the use of vasopressors should be further explored in the context of upper GI bleeds of unknown etiology as this method can achieve a higher-pressure state that offers a novel method of provocation in GI bleeds.

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