Case Report

Journal of Emergency Medicine Case Reports

Surgical Treatment of a Refractory Gastroesophageal Junction Dieulafoy Lesion Using Intraoperative Endoscopic Tattooing

Aşkın Kadir Perçem[1], Ramazan Topcu[2], Orhan Aslan[2], Mahmut Arif Yüksek[2], İsmail Sezikli[1]
[1]Hitit University Erol Olçok Training and Tesearch Hospital, Department of Gastroenterology Surgery, Çorum, Türkiye
[2]Hitit University Faculty of Medicine, Department of Surgery, Çorum, Türkiye

Abstract

Dieulafoy's lesion (DL) is a rare but potentially life-threatening cause of upper gastrointestinal bleeding. Endoscopic therapy is the first-line treatment; however, recurrent bleeding may necessitate surgical intervention. A 55-year-old male presented to the emergency department with hematemesis and hemodynamic instability. Initial endoscopy revealed a Dieulafoy's lesion in the gastric fundus with active bleeding. Hemostasis was achieved with epinephrine injection, thermal coagulation, and hemoclip placement, and temporary stabilization was obtained after blood transfusion. On the third hospital day, recurrent bleeding required repeat endoscopy and an additional hemoclip application, but the patient continued to experience symptoms. On day eight, intraoperative endoscopic tattooing with methylene blue facilitated accurate localization of the lesion, and wedge resection was performed. The postoperative course was uneventful, and histopathological examination confirmed the diagnosis of DL. This case demonstrates that intraoperative endoscopic marking is a valuable adjunct in refractory Dieulafoy's lesions, enabling precise localization, avoiding unnecessary radical surgery, and allowing successful limited resection.

Keywords: Dieulafoy's lesion, endoscopic hemostasis, intraoperative endoscopy, recurrent bleeding, wedge resection

Introduction

Dieulafoy's lesion (DL) is a rare cause of upper gastrointestinal bleeding, most commonly originating from a congenital vascular malformation along the lesser curvature of the stomach. Hemorrhage occurs due to erosion of the gastric mucosa overlying a large-caliber submucosal vessel (1). The lesion was first described in 1884 by Gallard as an aneurysm resulting from congenital malformations associated with arteriovenous (AV) shunting and submucosal abnormalities. Later, in 1898, the French surgeon Georges Dieulafoy termed it "exulceratio simplex," considering it an early stage of ulceration characterized by a visible artery responsible for bleeding (2).

DL can occur in any part of the gastrointestinal tract but is predominantly localized at the gastroesophageal junction and along the stomach's lesser curvature. It accounts for approximately 2–5% of intermittent upper gastrointestinal hemorrhages. The majority of lesions are located within 6 cm of the gastroesophageal junction, though rare cases have been reported in the bronchus and other regions of the gastrointestinal system (3). Bleeding is usually occult, but in rare cases, massive hemorrhage may occur when a large artery is involved.

Historically, in the absence of endoscopic techniques, surgery was the mainstay of treatment for DL bleeding. Radical procedures such as subtotal or total gastrectomy were associated with considerable morbidity and mortality. With the advent of endoscopic hemostatic methods, surgical intervention has been largely replaced and is now required in fewer than 10% of cases (4). Currently, endoscopy is considered the gold standard for both diagnosis and treatment, achieving success rates of 80–100%, with enhanced efficacy when combined with thermal therapy. Nevertheless, surgical intervention remains the final option in cases of uncontrolled or massive bleeding due to DL (5).

Case Report

A 55-year-old male presented to the emergency department with hematemesis, generalized fatigue, and dizziness. His medical history was notable for hypertension. He denied alcohol use, tobacco consumption, and nonsteroidal anti-inflammatory drug (NSAID) use. Three years prior, an upper gastrointestinal (GI) endoscopy had revealed only mild gastritis.

On examination, the patient was hypotensive (BP 80/50 mmHg) and tachycardic (HR 110 bpm). Rectal examination

Corresponding Author: Ramazan Topcu
e-mail: carrotbanana@hotmail.com
Received: 03.09.2025 • **Revision:** 09.01.2026 • **Accepted:** 12.01.2026
DOI: 10.33706/jemcr.1777562

Cite this article as: Perçem AK, Topcu R, Aslan O, Yüksek MA. Surgical Treatment of a Refractory Gastroesophageal Junction Dieulafoy Lesion Using Intraoperative Endoscopic Tattooing. Journal of Emergency Medicine Case Reports. 2026;17(1): 37-40

DOI: 10.33706/jemcr.1777562
Journal of Emergency Medicine Case Reports. 2026;17(1): 37-40

revealed melena. Laboratory tests showed anemia with hemoglobin of 8.1 g/dL. Given suspicion for upper GI bleeding, emergent esophagogastroduodenoscopy (EGD) was performed. Endoscopy identified active bleeding from the gastric fundus without mucosal ulceration. A protruding submucosal vessel consistent with a Dieulafoy lesion was visualized. Initial hemostasis was attempted with epinephrine injection and thermal coagulation (Figure-1-2). The patient received three units of packed red blood cells, raising hemoglobin to 10.5 g/dL.

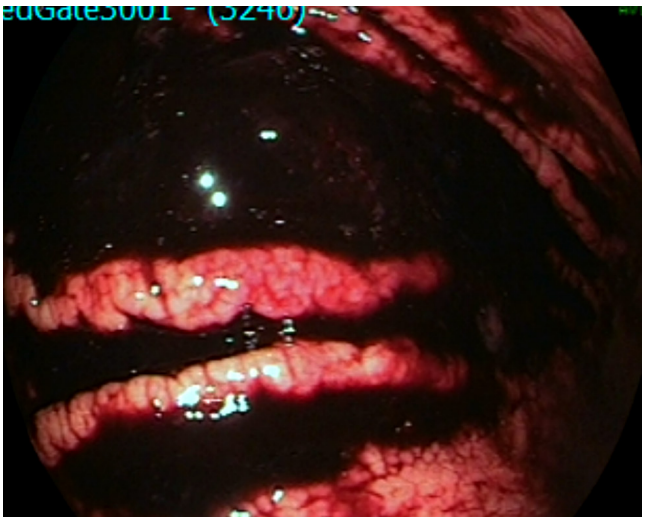

Figure 1. The stomach is full with blood.

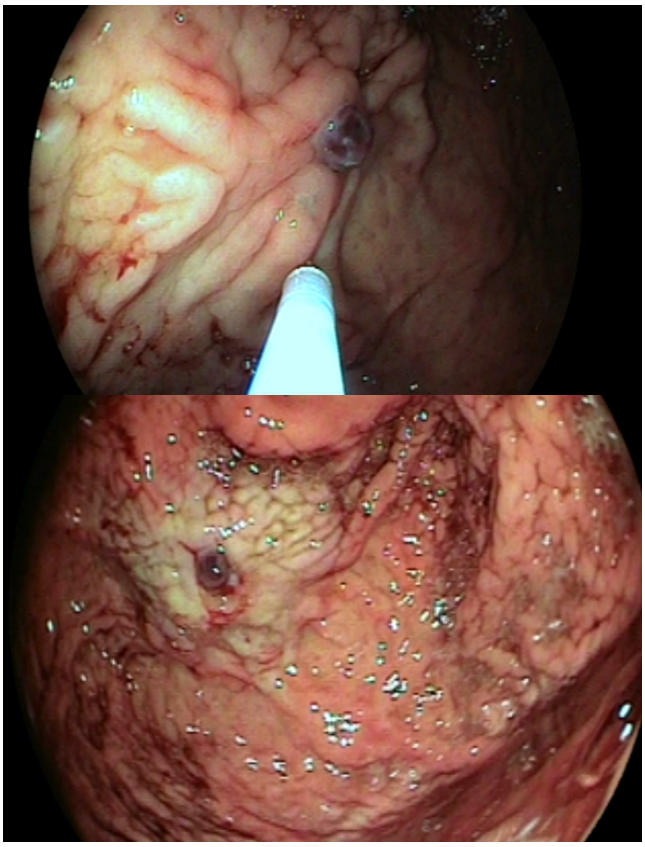

Figure 2. Adrenalin injection is made after irrigation with tap water and after sclerotherapy

On hospital day three, recurrent hypotension and hemoglobin decline (7.5 g/dL) prompted repeat EGD, confirming persistent bleeding. Mechanical hemostasis was achieved with a hemoclip, and three additional units of erythrocyte suspension were transfused, increasing hemoglobin to 9.5 g/dL. Follow-up endoscopy showed the hemoclip in place and no active bleeding.

Despite initial control, on day eight, the patient experienced another episode of hematemesis with hypotension (BP 90/60 mmHg, HR 110 bpm). Due to recurrent hemorrhage and failure of endoscopic management, a third EGD was performed in the operating room for lesion localization via methylene blue tattooing. Active bleeding from the previously clipped site was confirmed.

A laparotomy was performed, and the tattooed lesion was identified (Figure-3). The lesion was excised via wedge resection (Figure-4). The postoperative course was uneventful, and the patient resumed oral intake on postoperative day three. Histopathology confirmed a Dieulafoy lesion, characterized by a dilated submucosal artery protruding through otherwise intact mucosa.

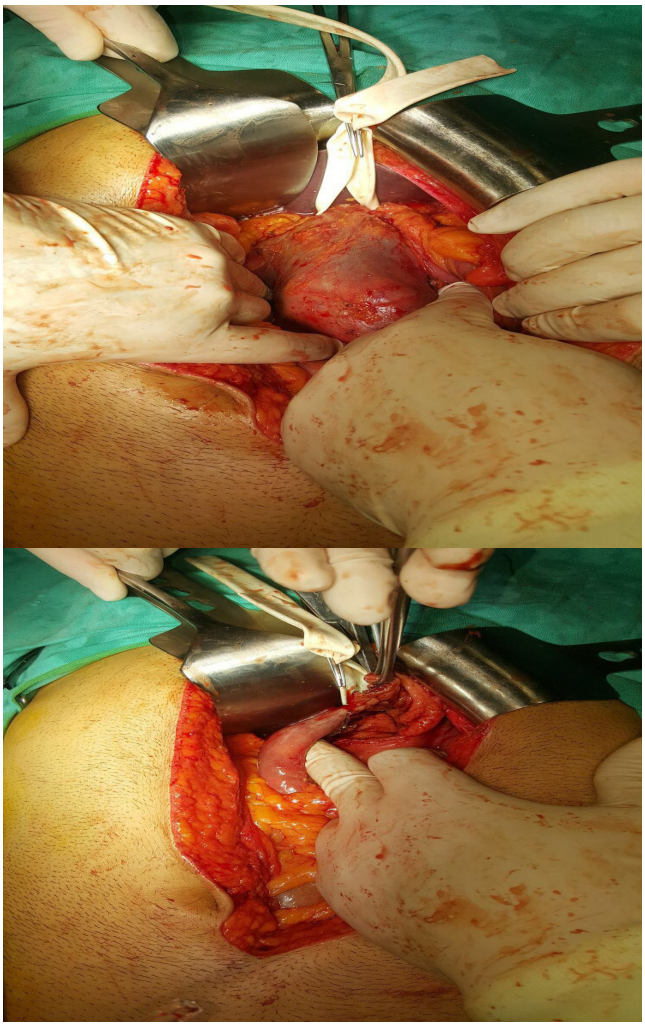

Figure 3. The lesion is stained with Methylene Blue Dye and anterior stained portion is opened and the lesion is found

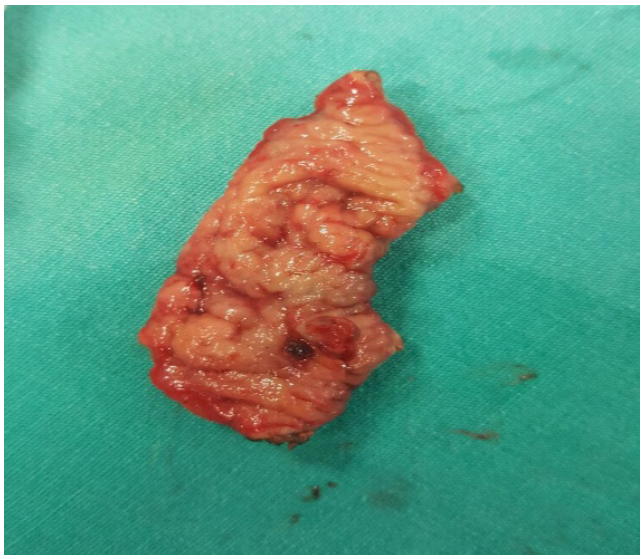

Figure 4. The lesion is seen in pathologic specimen

Discussion

Dieulafoy lesions (DL) are uncommon but potentially life-threatening causes of acute upper gastrointestinal bleeding, representing approximately 1–2% of cases (6). They are characterized by a large-caliber submucosal artery, typically 1–3 mm in diameter, that protrudes through a small mucosal defect, often covered with an adherent clot. The pathogenesis involves thinning of the overlying mucosa, which predisposes the vessel to erosion and intraluminal hemorrhage. Lesions are most frequently located within 4–6 cm of the gastroesophageal junction along the lesser curvature of the stomach, although they may occasionally occur in other gastrointestinal sites (7). In our patient, the lesion was located approximately 6 cm distal to the gastroesophageal junction on the lesser curvature, consistent with the most common location reported in the literature.

Although the exact etiopathogenesis of DL remains incompletely understood, it is considered a multifactorial process. The predominant mechanism is believed to be a congenital submucosal vascular malformation in which a dilated, large-caliber artery is covered by a thin mucosal layer that may erode over time and bleed. Several systemic and environmental factors have been proposed to modulate this process, including advanced age, which may reduce mucosal resilience and microvascular integrity, as well as chronic comorbidities such as cardiovascular disease, hypertension, cirrhosis, renal insufficiency, and diabetes, all of which may impair tissue perfusion, coagulation, or mucosal healing (8,9,10). Lifestyle factors such as smoking and alcohol consumption may contribute to chronic inflammation and ischemia, while the role of diet and genetic predisposition remains largely speculative. Overall, these factors suggest that DL develops on a background of vascular anatomy but may be unmasked by systemic or environmental stressors.

Medication-related mucosal injury, particularly from aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs), is known to increase the risk of DL rupture. Baettig et al. reported that up to 39% of patients with DL had a history of NSAID or aspirin use. Such agents can compromise the mucosal barrier, exposing the submucosal vessel and predisposing it to necrosis and rupture. In contrast, our patient had no history of NSAID or aspirin use, and endoscopy revealed normal gastric mucosa without ulceration. This underscores that DL can present even in the absence of identifiable mucosal injury (11).

Clinically, DL often presents with sudden, massive, or recurrent upper gastrointestinal bleeding, which may be pulsatile or intermittent. Endoscopy is both diagnostic and therapeutic, allowing direct visualization of the lesion and intervention. Endoscopic hemostatic methods include injection therapy (epinephrine or sclerosants), thermal coagulation, and mechanical therapy with hemoclips or band ligation (12). Combination therapy has been associated with lower rebleeding rates compared with monotherapy, with reported recurrence rates ranging from 9% to 40%. Although generally safe, endoscopic therapy carries a small risk of complications (13).

Surgical intervention is reserved for cases in which endoscopic management fails or bleeding recurs. Options include local excision, wedge resection, ligation via gastrotomy, or, rarely, total gastrectomy. Precise localization of the lesion is often challenging intraoperatively due to its small size and variable location, particularly for posterior or proximal lesions near the gastroesophageal junction. Intraoperative endoscopic localization and marking with dyes, such as methylene blue, can facilitate targeted resection, reduce operative time, and limit the extent of surgery. In our case, intraoperative endoscopic tattooing allowed accurate identification of the lesion, enabling a small anterior wedge resection with confirmation of complete excision on palpation of the specimen (14).

This case highlights several important points. First, DL should be considered in patients presenting with massive upper gastrointestinal bleeding, even in the absence of NSAID or aspirin use or visible mucosal ulceration. Second, endoscopic therapy remains the first-line treatment, but clinicians should remain vigilant for rebleeding. Third, intraoperative endoscopic localization can optimize surgical management and prevent unnecessarily extensive procedures, particularly in unstable patients or when the lesion is difficult to locate. Finally, anteriorly located lesions may be more readily identified and resected, whereas posterior lesions pose additional challenges.

Conclusion

Gastric Dieulafoy lesions are rare and diagnostically challenging sources of upper gastrointestinal bleeding.

DOI: 10.33706/jemcr.1777562
Journal of Emergency Medicine Case Reports. 2026;17(1): 37-40

Endoscopy is the cornerstone of both diagnosis and treatment, with combination therapy reducing the risk of rebleeding. Surgery is indicated for recurrent or uncontrolled bleeding, and intraoperative endoscopic localization and marking facilitate precise, limited resections while minimizing operative morbidity. Awareness of lesion location, patient risk factors, and potential challenges in surgical management is essential for optimal outcomes.

Informed Consent: The patient signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed. Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: A conflict of interest has not been declared by the author.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

References

1. Nojkov B, Cappell MS. Gastrointestinal bleeding from Dieulafoy's lesion: Clinical presentation, endoscopic findings, and endoscopic therapy. World J Gastrointest Endosc. 2015 Apr 16;7(4):295–307. doi: 10.4253/wjge.v7.i4.295
2. Gallard Lee, Y. T., Walmsley, R. S., Leong, R. W. L., & Sung, J. J. Y. (2003). Dieulafoy's lesion. Gastrointestinal Endoscopy, 58(2), 236–243. doi:10.1067/mge.2003.328
3. Chen W, Chen P, Li X, Gao X, Li J. Clinical characteristics and treatments for bronchial Dieulafoy's disease. Respir Med Case Rep. 2019 Jan 7;26:229-235. doi: 10.1016/j.rmcr.2019.01.004. PMID: 30740302; PMCID: PMC6357209
4. Veldhuyzen van Zanten, S. J., Bartelsman, J. F., Schipper, M. E., & Tytgat, G. N. (1986). Recurrent massive haematemesis from Dieulafoy vascular malformations--a review of 101 cases. Gut, 27(2), 213–222. doi:10.1136/gut.27.2.213
5. Jeon HK, Kim GH. Endoscopic Management of Dieulafoy's Lesion. Clin Endosc. 2015 Mar;48(2):112-20. doi: 10.5946/ce.2015.48.2.112. Epub 2015 Mar 27. PMID: 25844338; PMCID: PMC4381137.
6. Baxter M, Aly EH. Dieulafoy's lesion: current trends in diagnosis and management. Ann R Coll Surg Engl. 2010 Oct;92(7):548-54. doi: 10.1308/003588410X12699663905311. PMID: 20883603; PMCID: PMC3229341.
7. Norton, I. D., Petersen, B. T., Sorbi, D., Balm, R. K., Alexander, G. L., & Gostout, C. J. (1999). Management and long-term prognosis of Dieulafoy lesion. Gastrointestinal Endoscopy, 50(6), 762–767. doi:10.1016/s0016-5107(99)70155-0
8. Saleh R, Lucerna A, Espinosa J, Scali V. Dieulafoy lesion: the little known sleeping giant of gastrointestinal bleeds. Am J Emerg Med. 2016 Dec;34(12):2464.e3-2464.e5.
9. Shin HJ, Ju JS, Kim KD, Kim SW, Kang SH, Kang SH, Moon HS, Sung JK, Jeong HY. Risk Factors for Dieulafoy Lesions in the Upper Gastrointestinal Tract. Clin Endosc. 2015 May;48(3):228-33. doi: 10.5946/ce.2015.48.3.228. Epub 2015 May 29.
10. Ding YJ, Zhao L, Liu J, Luo HS. Clinical and endoscopic analysis of gastric Dieulafoy's lesion. World J Gastroenterol. 2010 Feb 7;16(5):631-5. doi: 10.3748/wjg.v16.i5.631.
11. Baettig B, Haecki W, Lammer F, Jost R. Dieulafoy's disease: endoscopic treatment and follow up. Gut. 1993 Oct;34(10):1418-21. doi: 10.1136/gut.34.10.1418. PMID: 8244112; PMCID: PMC1374553
12. Aabdi B, Kharrasse G, Zazour A, Koulali H, Elmqaddem O, Zahi I. Clinical, endoscopic and therapeutic features of bleeding Dieulafoy's lesions: case series and literature review. BMJ Open Gastroenterol. 2024 May 24;11(1):e001299. doi: 10.1136/bmjgast-2023-001299. PMID: 38789268; PMCID: PMC11129027.
13. Levy AR, Broad S, Loomis Iii JR, Thomas JA. Diagnosis and Treatment of a Recurrent Bleeding Dieulafoy's Lesion: A Case Report. Cureus. 2022 Nov 30;14(11):e32051. doi: 10.7759/cureus.32051. PMID: 36600857; PMCID: PMC9802011.
14. Zhiwei, Zhang, and Chen Junqiang. "Laparoscopy and methylene blue staining angiography for precise synchronous resection of small intestinal vascular malformations: A case report." *Frontiers in Medicine* 12 (2025): 1621874.