Case Report

Duodenal Dieulafoy's lesion- challenges in diagnosis: A Case report

Syeda Nur-E- Jannat,[1] Md. Raisul Arafat Riad,[2] Mohammad Shohidul Islam,[3] Md. Kamal Hossain Mozumdar[4]

Abstract

Dieulafoy's lesion is a rare but potentially life-threatening cause of upper gastrointestinal (GI) bleeding, typically presenting with severe haematemesis, melaena, or haematochezia without prior symptoms, often with hemodynamic instability requiring transfusion of multiple units of packed erythrocytes. Diagnosis is frequently challenging due to its subtle endoscopic appearance and obscure location of the lesion. Early endoscopy during a bleeding episode is essential for an accurate diagnosis and sometimes multiple endoscopies are needed to establish the diagnosis. Here, we present a case of a 51-year-old female with multiple comorbidities, including end-stage renal disease (ESRD) on maintenance hemodialysis and ischemic heart disease, who presented with persistent melaena for one month. After extensive evaluation, she was diagnosed with Dieulafoy's lesion in the duodenum. Prompt endoscopic management stabilized her condition. This case highlights the diagnostic challenges and clinical importance of recognizing Dieulafoy's lesion in patients with complex medical histories.

Authors:
Syeda Nur-E- Jannat
Associate professor (Gastroenterology),
National Gastroliver Institute & Hospital

Md. Raisul Arafat Riad
FCPS Part 2 Trainee, National Gastroliver Institute & Hospital

Mohammad Shohidul Islam
Assistant professor (Gastroenterology),
National Gastroliver Institute & Hospital

Md. Kamal Hossain Mozumdar
FCPS Part 2 Trainee, National Gastroliver Institute & Hospital

Corresponding Author
Syeda Nur-E- Jannat
MBBS (DMC), FCPS (Medicine), MD (Gastroenterology)
Associate professor (Gastroenterology),
National Gastroliver Institute & Hospital,
Email: jannatdmc@yahoo.com, Phone: +8801819442631

Introduction:

Dieulafoy's lesion (DL) was first reported by Gallard in 1884, but was named after a French surgeon, Georges Dieulafoy, who described the lesion more precisely in 1898.1,2 It is defined anatomically as a dilated (1-3 mm), aberrant, submucosal artery that erodes the overlying GI mucosa in the absence of an underlying ulcer, aneurysm, or intrinsic mural abnormality.3 It accounts for 1–2% of acute GI bleeding cases.[4,5] Patients are typically asymptomatic before presenting with acute, profuse GI bleeding, often recurrent, which can manifest as hematemesis, melena or hematochezia with haemodynamic instability.[6,7] About 75% of lesions are located in the stomach, usually within 6 cm of the gastroesophageal junction along the gastric lesser curve.8 The duodenum is the second most common site for a Dieulafoy's lesion (14–18% of cases), and the majority of these lesions are located in the duodenal bulb (53%), followed by the third part of the duodenum (29%) and the junction of the first and second parts of the duodenum (18%).[9,10] The diagnosis and management of bleeding DLs rely primarily on endoscopy. Given its rarity and difficulty in diagnosis - especially in patients with significant comorbidities such as ESRD and ischemic heart disease - early diagnosis and timely endoscopic intervention are crucial.

Case Presentation:

A 51-year-old woman, a housewife from Savar, Dhaka, presented with a history of black, tarry, foul-smelling stools (melaena) 4-5 episodes per day for the last 4 weeks. She also complained of extreme fatigue and breathlessness on exertion for the last 15 days. She had no history of abdominal pain, heartburn, hematemesis, or any other bleeding manifestation like haemoptysis, epistaxis, or easy bruising. No history of jaundice, pedal edema, altered level of consciousness, joint pain, oral ulceration, skin rash, fever, cough, contact with a known TB patient, weight loss, or recent history of taking NSAIDs, steroids, or iron-containing drugs. Her appetite was normal. With all the above complaints, she got admitted herself to the National Gastroliver Institute & Hospital (NGLIH), Dhaka for better management.

She was known to be hypertensive, non-diabetic and diagnosed with CKD of unknown etiology in 1997. She had a renal transplant in 2009, followed by immunosuppressive therapy (steroids, cyclosporine, and azathioprine) and was doing well until 2021. Since then, her renal function deteriorated, requiring hemodialysis (HD) and she discontinued all immunosuppressant medication. Now, she was on maintenance hemodialysis (MHD) three times per week and on erythropoietin once in each cycle of HD. She was a known case of ischemic heart disease (IHD) with PTCA & stenting to the LCX in 2021 and was taking aspirin and clopidogrel. She used to take regular anti-hypertensive medication with good control of BP. She required blood transfusions every 2-3 months interval after starting HD since 2023.

Initially, she was admitted to a local hospital, where she was resuscitated with IV fluid, injection ceftriaxone 1 gm twice a day, 9 units of blood transfusion for melaena, sucralfate (1 gm every 6 hours), and injection proton pump inhibitor (8 mg/hour) infusion for the last 7 days without any significant improvement in bleeding. She was started on heparin-free HD and dual antiplatelet therapy was also stopped for the last 1 month.

She had no family history of liver disease, renal disease, cardiac disease, or coagulation disorder. She denied any history of tobacco, alcohol or betel nut intake. She had three abortions, and her menstrual cycle was normal. She was vaccinated for HBV and COVID-19.

On examination, she appeared ill, moderately anemic, non-icteric, had a cushingoid appearance, generalized hyperpigmentation, and leukonychia. There was also a left-arm arteriovenous fistula, but no stigmata of chronic liver disease (CLD), no edema, no lymphadenopathy, and JVP was not raised. Vitals were within normal limits.
Abdominal examination revealed a scar mark in the right iliac fossa (RIF) and hypogastric region, mild hepatomegaly and a transplanted kidney in the RIF. No splenomegaly, no ascites. Cardiovascular examination detected a pansystolic murmur all over the precordium. Other systemic examinations revealed normal findings.

Investigations:

Her hemoglobin level was repeatedly low, ranging from 6.4 to 7.4 gm/dL with blood transfusion indicating ongoing bleeding; the total platelet count was also low, ranging from 90,000/µL to 77,000/µL. Serum creatinine ranged between 4.8 to 8.8 mg/dL with MHD. Liver function tests, prothrombin time with INR, APTT, FDP and D-Dimer were within normal limit. Iron profile was consistent with anemia of chronic disease. Viral markers, HBsAg and anti-HBc (total) were negative, but anti-HCV was positive with HCV RNA negative.USG of the whole abdomen revealed a graft kidney in the right iliac fossa, with normal liver echotexture, no intra-abdominal lymphadenopathy and no ascites. Doppler USG of the hepatobiliary-pancreatic vessels revealed normal vascular flow patterns. Chest X-ray showed mild cardiomegaly, and echocardiogram revealed a good ejection fraction (70%).

She underwent an upper GI endoscopy outside the hospital before admission, which showed antral polypoid swelling and clotted blood in the stomach, with fresh oozing of blood in the bulb, postbulbar area and second part of the duodenum onward, without any specific bleeding source in the duodenum or stomach. After admission to NGLIH, we repeated upper GI endoscopy twice with adequate water jet irrigation, flushing and suction, with the same findings of fresh and altered blood in the stomach and duodenum, without identifying any specific point of active bleeding.

As bleeding continued, after resuscitation and adequate counseling, under anesthesia, we attempted a 4th upper GI endoscopy. After rinsing with water and aspiration, a specific source of oozing blood from the floor close to the lateral wall near the second part of the duodenum could be identified (Figure 1). Due to peristalsis and pulsation, it was difficult to exactly localize the bleeding point. So, with the aim of better visualization of the lateral wall and floor of the second part of the duodenum, we introduced a pediatric colonoscope, with adequate suction and water insufflation. Micro pulsatile bleeding from a bleeding vessel without any ulceration could be identified at the floor of the junction of the first and second part of the duodenum. Adrenaline (1:10,000 dilution) was injected at the base, followed by argon plasma coagulation (APC) applied to the bleeding vessel (Figure 2). After two days, her melaena stopped, her vital status remained stable, and her hemoglobin also rose to 10 gm/dL. No surgical or angiographic intervention was required. Subsequently, the patient was discharged from the hospital to home.

Thus, our final diagnosis was actively bleeding Dieulafoy's lesion at the junction of the first and second parts of the duodenum.

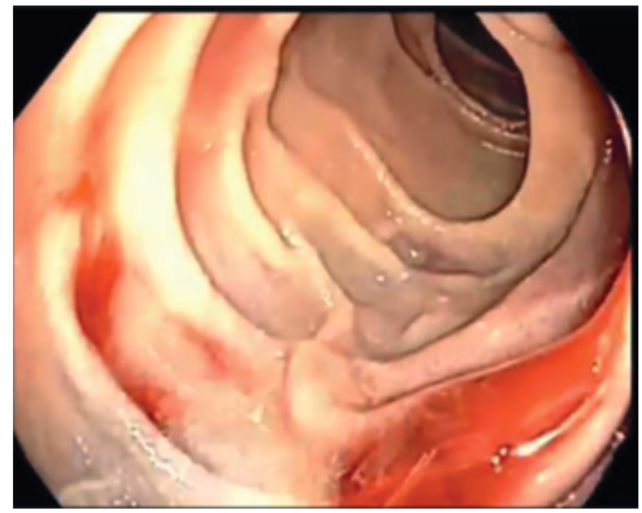

Figure I: Shows fresh blood in infero-lateral wall of duodenum.

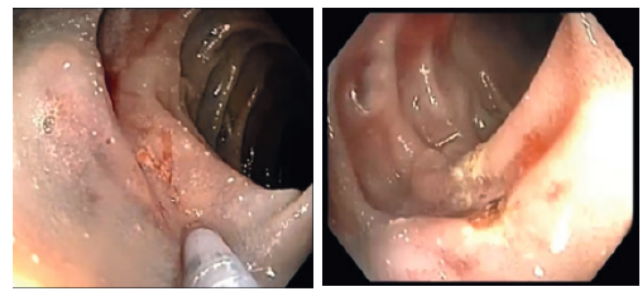

Figure II: haemostasis with injection adrenalin (1:10,000 dilution) followed by Argon Plasma Coagulation (APC)

Discussion:

Dieulafoy's lesion (DL) is an uncommon but important cause of recurrent GI bleeding. The condition is often underdiagnosed due to its small mucosal defect, obscure location of the lesion, and intermittent bleeding.[5,11] We diagnosed duodenal DL as a cause of recurrent melaena in a woman in her sixth decade of life. DL can occur at any age but is most common in the elderly, typically in the fifth or sixth decade of life.[5,12,13] GI bleeding from DL is frequently linked with comorbid conditions, including cardiovascular disease, chronic kidney disease, hypertension, peptic ulcer disease, diabetes mellitus and long-term use of medications such as nonsteroidal anti-inflammatory drugs and anticoagulants.[14,15] In this patient, ESRD with hemodialysis and prior dual antiplatelet therapy likely contributed to her bleeding risk.

Our patient required endoscopy four times to establish a diagnosis of duodenal DL, although initial endoscopy is diagnostic in approximately 70% of cases, with about 6% of patients requiring three or more attempts. Initial endoscopy may be compromised by factors such as excessive blood, subtle lesions obscured by folds or gastric contents, surrounding normal mucosa, blood clots adhering to the site or pooled blood from significant hemorrhage.[5,8] Moreover, DL located in the periampullary area and second part of the duodenum is more difficult to diagnose, as they cannot be seen or treated by a forward-viewing endoscope.[16,17]

Lateral-view endoscopy can visualize the periampullary region better. We used a pediatric colonoscope, as it has additional insertion length and a favorable 5- to 6-o'clock orientation of the working channel, which provided an advantage for better visualization of the lateral and posterior walls and distal duodenum to identify the bleeding point source.[18] Endoscopies performed within the first 12 hours have a high success rate for diagnosing DLs because of their capability to pinpoint the bleed location.[19]

Endoscopic hemostasis by different procedures, like regional injection, thermal techniques, and mechanical methods, is the main modality of treatment, with surgery reserved for refractory cases in patients with uncontrollable bleeding, alongside endoscopic and angiographic embolization methods.[5,20,21] The choice of therapeutic technique will depend on the clinical presentation, lesion site, and available surgical and endoscopic expertise. Endoscopic injection therapy using vasoconstrictors like adrenaline, sclerosants (ethanol, polidocanol) or N-butyl 2-cyanoacrylate is a simple and cost-effective method but has a high risk of rebleeding when used alone. Thermal therapy includes contact methods (bipolar, heater probe), which are effective but carry a risk of transmural injury, and non-contact argon plasma coagulation (APC), which is safer but mainly useful for superficial lesions. Mechanical therapies, such as hemoclips and band ligation, are preferred for achieving hemostasis but can be challenging due to duodenal angulation. Band ligation has a lower risk of perforation but may cause rebleeding ulcers.[13,19,22] Combining endoscopic therapies, such as injection therapy followed by thermal or mechanical interventions, has proven to be superior to single-modal approaches, with permanent hemostasis achieved in 95% of patients.3 In our case, a combination of APC with adrenaline injection was used to successfully secure hemostasis.
The risk of rebleeding from DL ranges from 9% to 40%,[5,21] for which regular follow-up for at least 6 months is recommended. Mortality from bleeding due to this lesion is 9%–13%.[8]

Conclusion:

Although rare, Dieulafoy's lesion should be considered an important etiology of unexplained, life-threatening upper GI bleeding, particularly in an elderly patient with multiple comorbidities. The localization of Dieulafoy's lesion in the duodenum can be more difficult. Increased awareness and careful, early endoscopic evaluation following the bleeding episode are key to accurate diagnosis and prompt therapeutic intervention. Endoscopic hemostasis is crucial for successful management, a favorable outcome and reducing the need for invasive surgical interventions.

References:

1. Gallard MT. Aneurysme miliaires de l'estomach, donnant lieu a des hematemese mortelles. *Bull Soc Med Hop Paris* 1884; 1: 84-91

2. Dieulafoy G. Exulceratio simplex. L'intervention chirurgicale dans les hematemeses foudroyantes consecutives a l'exulceration simple de l'estomac. *Bull Acad Med* 1898; 49: 49-84

3.Lee YT, Walmsley RS, Leong RW, Sung JJ. Dieulafoy's lesion. *Gastrointest Endosc* 2003; 58: 236-243 [PMID: 12872092] https:// doi. org/ 10. 1067/ mge. 2003. 328.

4. Malik T. F. and Anjum F., "Dieulafoy's Lesion Causing Gastrointestinal Bleeding," in StatPearls *(StatPearls Publishing, 2023).*

5. Baxter M, Aly EH. Dieulafoy's lesion: current trends in diagnosis and management. *Ann R Coll Surg Engl.* 2010;92(7):548–554. https:// doi. org/ 10. 1308/ 00358 8410X 12699 66390 5311.

6. Luis LF, Sreenarasimhaiah J, Jiang Tang S. Localization, efficacy of therapy, and outcomes of Dieulafoy lesions of the GI tract – The UT Southwestern GI Bleed Team experience. *Gastrointest Endosc.* 2008; 67: AB 87

7. Lopez-Arce G, Zepeda-Gomez S, Chavez-Tapia NC, et al. Upper gastrointestinal Dieulafoy's lesions and endoscopic treatment: first report from a Mexican centre. *Therap Adv Gastroenterol.* 2008;1(2):97–101

8. Nojkov B, Cappell MS. Gastrointestinal bleeding from Dieulafoy's lesion: Clinical presentation, endoscopic findings, and endoscopic therapy. *World J Gastrointest Endosc* 2015; 7(4): 295-307. https:// doi. org/ 10. 4253/ wjge. v7. i4. 295.

9. Chaer RA, Helton W.S. Dieulafoy's disease *J. Am. Coll. Surg.*2003 Feb1;96 (2):290-296.

10. Rana SS, Bhasin DK, Gupta R, Yadav TD, Gupta V, Singh K. Periampullary Dieulafoy's lesion: an unusual cause of gastrointestinal bleeding. *JOP.*2010;5 (11) :266-269.

11. Schmulewitz N, Baillie J. Dieulafoy lesions: a review of 6 years of experience at a tertiary referral center. *Am J Gastroenterol.* 2001;96(6):1688–1694.

12. Shewaye AB, Berhane KA, Fanta AD, Adem A. Bleeding duodenal Dieulafoy's lesion in a pediatric patient from Ethiopia: A Case Report. Clinical Case Reports, 2025 Aug; 13(8):e70743 https://doi.org/10.1002/ccr3.70743

13. Di Nardo G, Esposito G, Mauro A, et al. Dieulafoy lesion: two pediatric case reports. *Italian Journal of Pediatrics.*2020;46(1) doi:10.1186/s13052-020-0814-8.

14. Saleh R, Lucerna A, Espinosa J, Scali V. Dieulafoy lesion: the little known sleeping giant of gastrointestinal bleeds. *American journal of Emergency Medicine*.2016 Dec1; 34(12):2464.e3–5

15. Kasapidis P, Georgopoulos P, Delis V, Balatsos V, Konstantinidis A, Skandalis N. Endoscopic management and long term follow- up of Dieulafoy's lesions in the upper GI tract. *Gastrointestinal Endoscopy.* 2002 Apr;55(4): 527–531.

16. Lee WS, Cho SB, Park SY, et al. Succesful side-viewing endoscopic hemoclipping for Dieulafoy-like lesion at the brim of a periampullary diverticulum. *BMC Gastroenterol.* 2010;10:24–29.

17. Pollack R, Lipsky H, Goldberg RI. Duodenal Dieulafoy's lesion. *Gas trointest Endosc.* 1993;39(6):820–822.

18. Michael J. Bourke,1,2 Simon K. Lo,3 Ross C. D. Buerlein,4 and Koushik K. Das AGA Clinical Practice Update on Nonampullary Duodenal Lesions: Expert Review. *Gastroenterology* 2025;168:169–175.

19. Yılmaz T, Kozan R. Duodenal and jejunal Dieulafoy's Le sions: optimal management, *Clinical and Experimental Gastroenterology*.2017;10: 275–283.

20. López JDM, Hernández CAT, Gómez DO, Návar AD, Ortiz CAB, Reséndiz BF, et al. Duodenal dieulafoy injury: case report. *Int Surg J.*2024;11:104-106.

21. Jeon HK, Kim GH. Endoscopic management of Dieulafoy's Lesion. *Clinical Endoscopy.* 2015;48(2):112–120.

22. Inayat F, Amjad W, Hussain Q, Hurairah A. Dieulafoy's Lesion of the Duodenum: a comparative review of 37 cases. *BMJ* Case Reports. 2018 Feb22; bcr-2017-223246