Case Report

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Angiodysplasia simulating variceal bleeding: a challenging case report of diagnosis and intervention

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Introduction and importance: Angiodysplasia, a prevalent vascular anomaly in the gastrointestinal tract, often presents with upper gastrointestinal bleeding, sharing symptoms with gastric varices. The diagnostic challenge arises due to overlapping clinical features. This case report highlights the importance of considering angiodysplasia in the differential diagnosis, especially when variceal bleeding is less likely, and emphasizes the role of various diagnostic modalities in accurate identification.

Case presentation: A 52-year-old male presented with severe hematemesis and melena, mimicking variceal bleeding. Despite initial management, bleeding persisted. Contrast-enhanced computed tomography revealed dilated vascular channels, raising suspicion for both gastric varices and angiodysplasia. Endoscopy confirmed an angiomatous lesion, inadvertently disrupted during the procedure, necessitating angiography. The angiographic findings supported the diagnosis of angiodysplasia, and successful interventions included temporary glue embolization and argon laser coagulation during endoscopy. The patient was discharged with stable hemoglobin; a 2-year follow-up showed no recurrence.

Clinical discussion: The case discusses the challenges in differentiating angiodysplasia from varices, emphasizing the role of imaging and endoscopic modalities. It highlights the need for a tailored approach to treatment, including argon plasma coagulation, and underscores the significance of meticulous follow-up for recurrence.

Conclusion: This case report elucidates the diagnostic and therapeutic journey in managing a patient with angiodysplasia masquerading as variceal bleeding. It emphasizes the importance of considering vascular anomalies without typical signs and the significance of individualized interventions for optimal patient outcomes. The 2-year follow-up without recurrence signifies the successful management of the case.

Keywords: angiodysplasia, case report, diagnostic dilemma, upper gastrointestinal bleeding, variceal bleeding

Introduction

Angiodysplasia, the most prevalent vascular anomaly in the gastrointestinal (GI) tract, particularly in the stomach and duodenum, contributes to bleeding in 4–7% of patients with GI bleeding[1,2]. This can present as hidden or apparent bleeding. The condition shares symptoms with gastric varices, including hematemesis and melena, leading to potential diagnostic confusion[3]. Endoscopic evaluation is the primary diagnostic method, although angiography provides superior visualization for diagnosing and assessing the extent of angiodysplasia[4]. Management strategies vary based on the severity of the condition. Endoscopic treatment options include argon plasma coagulation (APC), electrocoagulation, mechanical hemostasis, and radiofrequency ablation[5–7]. Careful consideration is essential due to the overlapping symptoms with gastric varices. This case report uniquely navigates the diagnostic challenges of angiodysplasia mimicking as variceal bleeding. This case has been reported in line with CARE (CAse REport) guidelines[8].

HIGHLIGHTS

- This study illuminates the diagnostic intricacies surrounding angiodysplasia, often misinterpreted as variceal bleeding.
- Utilizing advanced imaging techniques, including contrast-enhanced computed tomography (CT) and angiography, proved instrumental in accurate diagnosis.
- Tailored interventions, including glue embolization and argon plasma coagulation, successfully managed angiodysplasia, evident in a recurrence-free 2-year follow-up, affirming clinical significance.

Case details

A 52-year-old male presented with severe hematemesis and melena persisting over the course of 5 days. The blood he vomited was notably bright red. Alongside this, he experienced dizziness

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Table 1

Laboratory results of the patient

Investigation	Result	Reference range
Hemoglobin	11	11.5–15 mg/dl
Leukocyte count	10 000	4000–11 000
Platelet count	4 lakh/mm^3	1.5-4.5 lakh/mm^3
Eosinophil	2	1–6%
Bilirubin total	0.8	0.4–1 mg/dl
Bilirubin direct	0.2	0–0.4 mg/dl
ALT	40	5–37 U/l
AST	38	5–37 U/l
ALP	35	0–41 U/l
Total protein	6.5	6.6–8.7 g/dl
Albumin	4	3.8–5.1 g/dl
Prothrombin time	12	10–13 s

ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase.

accompanied by cold, clammy skin. Similar episodes had occurred a month prior, but at that time, he did not undergo any further investigation. He denied any history of alcohol consumption, cigarette smoking, or use of antiplatelet or non-steroidal anti-inflammatory drugs. Furthermore, there was no significant family history present.

Upon physical examination, the patient displayed low blood pressure (90/60 mmHg), a rapid and weak pulse (120/min), a respiratory rate of 22/min, and a temperature of 37°C. No physical signs indicative of chronic liver failure, such as jaundice, palmar erythema, or abdominal distention, were evident. Additionally, there were no signs of hepatosplenomegaly. The remainder of the systemic examination was unremarkable, and all biochemical studies yielded results within normal ranges (Table 1). Notably, there were no signs of chronic renal failure.

In the emergency department, the patient underwent resuscitation with fluid replacement, blood grouping, and cross-matching. Despite receiving injections of tranexamic acid and pantoprazole, the hematemesis persisted. Consequently, emergency contrast-enhanced computed tomography (CT) of the abdomen was scheduled to identify the source of upper gastrointestinal (UGI) bleeding. The CT scan revealed multiple dilated vascular channels in the fundic part of the stomach, indicative of isolated fundic varices (Fig. 1). Subsequently, UGI endoscopy was performed, revealing an angiomatous nodular lesion in the fundic part of the stomach (Fig. 2). No ulceration was observed in either the stomach or duodenum. Unfortunately, the lesion was inadvertently disrupted during the procedure, leading to further bleeding.

The patient was then transferred to the Intervention room, where angiography was conducted via catheterization of the left gastric artery. The angiography confirmed the diagnosis of angiodysplasia of the stomach, revealing blushing with multiple ecstatic vessels and early venous enhancement (Fig. 3A). Using glue as a temporary embolizing agent, the bleeding was successfully halted. Subsequent angiography demonstrated cessation of flow in the ecstatic vessels, effectively stopping the bleeding (Fig. 3B). The procedure was completed without any adverse effects. The patient was admitted, and the lesion was effectively treated with argon laser coagulation. Upon discharge, his hemoglobin level was measured at 9 gm/dl. Over a 2-year follow-up period, there were no instances of recurrent bleeding.

Discussion

Angiodysplasia remains a potential differential diagnosis in both overt and occult UGI bleeding[1]. The common diagnosis for UGI bleeding is variceal bleeding, which presents with similar symptoms[3]. The similarities in both presentations create a diagnostic dilemma. However, accompanying features with both diagnoses can provide valuable clues. Angiodysplasia is usually associated with renal insufficiency[1]. Gastric varices are invariably present with portal hypertension. In this particular case, the patient had no other associated conditions.

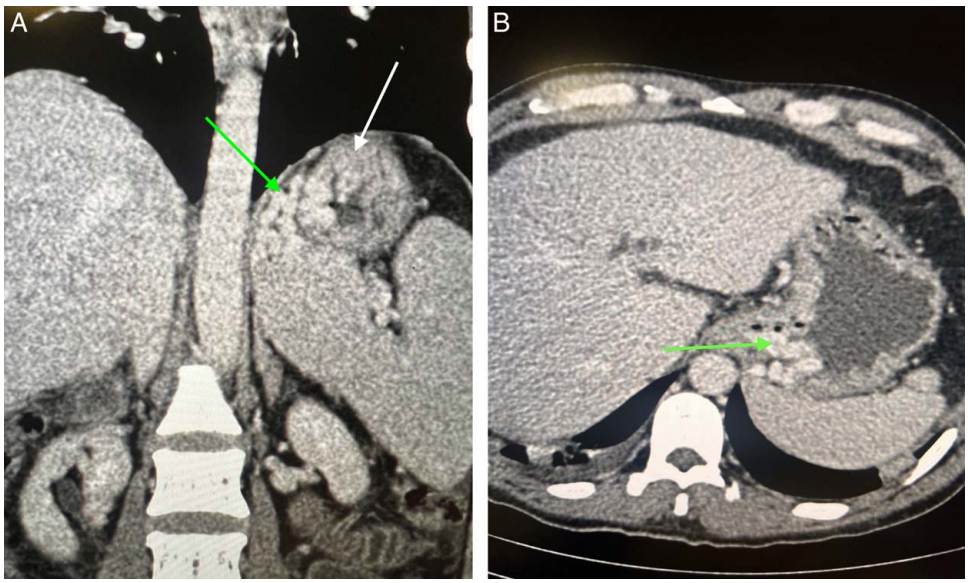

Figure 1. Contrast-enhanced CT images of the abdomen in coronal (A) and axial (B) views showing multiple dilated vessels (green arrow) at the fundic part of the stomach (white arrow) with a normal outline of liver parenchyma.

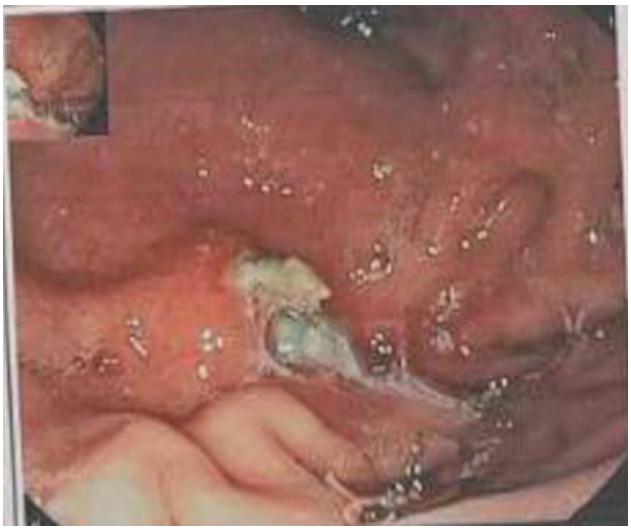

Figure 2. Upper gastrointestinal endoscopy images showing angiomatous nodular lesion in the fundic part of the stomach.

The provisional diagnosis of gastric varices was assumed during the patient's management. Varices are present in ~50% of patients with cirrhosis, and variceal hemorrhage occurs at an annual rate of 5–15%[9]. In contrast, the patient had no symptoms or signs indicative of chronic liver disease. Therefore, the diagnosis of variceal hemorrhage remained unlikely. Proton pump inhibitors (PPIs) before endoscopy have a favorable prognosis in the management of UGI bleeding[10]. Even after tranexamic acid and PPI, the bleeding in our patient was not contained. Atypical cases of UGI bleeding require additional investigation, such as CT, traditional angiography, or video capsule endoscopy[11]. Along with endoscopy, these methods help identify the source of bleeding. The CT scan in our patient revealed multiple dilated vascular channels in the fundus of the stomach, a finding associated with both gastric varices and angiodysplasia. The portal and splenic veins were normal, with no changes supporting signs of chronic liver disease.

Endoscopy as a diagnostic modality was performed[11]. The presence of angiomatous bright lesions with pulsation in the fundic part led to the diagnosis of angiodysplasia in the stomach. Clinical symptoms of blood loss and even the CT scan of the abdomen were unable to differentiate between gastric varices and angiodysplasia. Endoscopic findings can not only be diagnostic but also therapeutic in the management of UGI bleed. An immediate focus during the treatment of UGI bleed should be on stopping the source of bleeding. In our case, temporary glue helped in this regard. Angiographic intervention with a catheter via the left gastric artery, showing multiple ecstatic vessels with early venous enhancement, further confirmed the case. The treatment of angiodysplasia has been individualized based on the location, accessibility of the lesion, experience of the professional, and availability of resources. Cautery remains the most common mode of treatment. APC uses high-frequency energy transmitted to tissue by ionized gas and is one of the definitive and effective treatments for angiodysplasia[12]. The patient successfully underwent APC and was discharged in stable condition. Although one-third of patients with angiodysplasia usually experience a recurrence of bleeding after a mean age of 22 months, there was no recurrence in our patient during a 2-year follow-up period[13]. Patients should be kept on follow-up for further durations to monitor for recurrences.

Varices, although a common cause of UGI bleed, can be a less common but equally dangerous cause like angiodysplasia. Similarities in their presentation can make it difficult to differentiate them early. Vascular lesions causing UGI bleeding should be considered in the differential for patients even without signs of renal insufficiency. Although the management of acute UGI bleed is similar, focusing on stabilizing the patient, the definitive

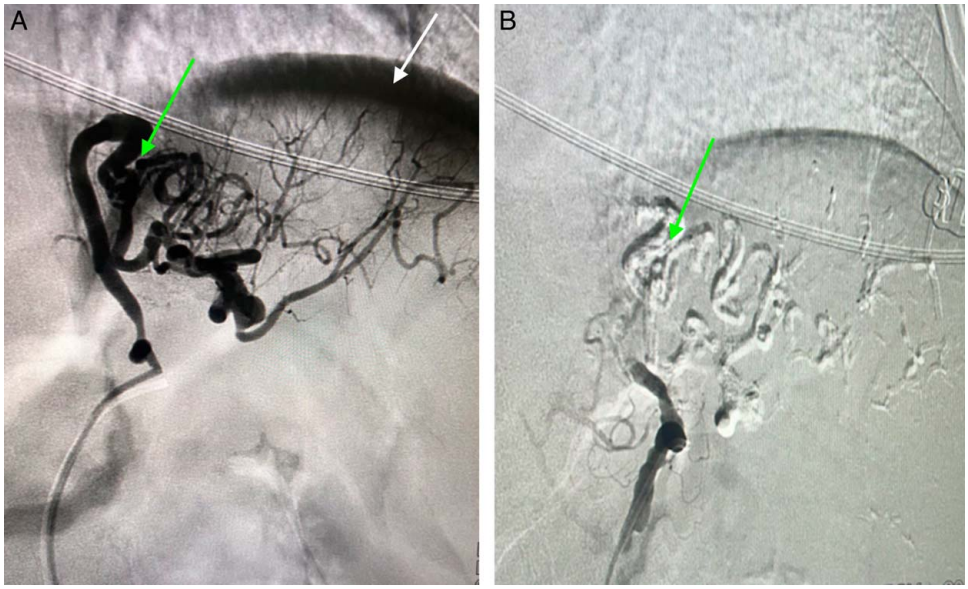

Figure 3. (A) Fluoroscopy-guided angiography with the tip of the catheter placed on the left gastric artery showing multiple ecstatic vessels (green arrow) with blushing at the wall of the fundic part of the stomach (white arrow). (B) Post-embolization imaging with glue demonstrated the absence of flow in the ecstatic vessels (green arrow), accompanied by the cessation of blushing in the wall of the fundic part of the stomach.

treatment remains different. Early diagnosis can aid in evaluating and treating the cause of bleeding. A follow-up protocol and preventative measures also remain entirely different from a case of gastric varices. Meticulous detail can help in identifying vascular lesions like angiodysplasia early.

Conclusion

This case highlights the diagnostic challenges of angiodysplasia, emphasizing the importance of considering vascular anomalies in UGI bleeding. Individualized interventions, including APC, proved effective, ensuring successful management with no recurrence over a 2-year follow-up.

Ethical approval

This case report did not require review by the Institutional Review Committee, Institute of Medicine.

Consent

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-chief of this journal on request.

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None.

Author contribution

D.C.: conceptualization, as mentor and reviewer for this case report and for data interpretation; S.K.: contributed in conceptualization and reviewer; S.S.: contributed in performing literature review and editing; A.T.: contributed in performing literature review and editing; B.B.: contributed in writing the paper and reviewer for this case. All authors have read and approved the manuscript.

Conflicts of interest disclosure

All the authors declare that they have no conflicts of interest.

Research registration unique identifying number (UIN)

The case report at hand is not a first-in-man case report of a novel technology or surgical technique, therefore a registration of these case reports according to Declaration of Helsinki 2013 is not required.

Guarantor

Dinesh Chataut.

Data availability statement

Data sharing is available upon reasonable request.

Provenance and peer review

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