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Recurrent Upper Gastrointestinal Bleeding in a Young Ugandan Man with Grade III Esophageal Varices and Peptic Ulcer Disease: A Case Report

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Case Report

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Abstract

Background

Upper gastrointestinal bleeding (UGIB) remains a common medical emergency in sub-Saharan Africa, where esophageal varices related to schistosomal periportal fibrosis and viral hepatitis contribute a much larger share of cases than in high-income settings.

Case presentation:

We describe a 28-year-old Ugandan man, a fuel pump attendant, who presented with a two-day history of hematemesis and melena, epigastric pain, and generalized weakness, on a background of similar symptoms three months earlier that had been attributed to grade III esophageal varices and gastritis on upper gastrointestinal endoscopy. On this admission he had marked conjunctival pallor, mild dehydration, and epigastric tenderness with guarding, but no stigmata of chronic liver disease, organomegaly, or lymphadenopathy. He was managed with intravenous fluids, proton-pump inhibition, empirical antibiotics, tranexamic acid, and blood transfusion, with clinical improvement over three days of follow-up documented by resolving melena and stabilizing vital signs.

Conclusion

This case illustrates the diagnostic overlap between variceal and peptic causes of UGIB in a young patient without classic risk factors for either, and underscores the importance of definitive endoscopic evaluation, secondary variceal prophylaxis, and cautious, evidence-based use of adjunctive pharmacotherapy such as tranexamic acid in recurrent bleeding.

INTRODUCTION

Upper gastrointestinal bleeding (UGIB) is a potentially life-threatening medical emergency that requires prompt recognition, resuscitation, risk assessment, and identification of the bleeding source. It commonly presents with haematemesis, melaena, or both, and major causes include peptic ulcer disease, erosive gastrointestinal lesions, and gastro-oesophageal variceal haemorrhage [1, 2]. Although substantial advances in endoscopic and pharmacological treatment have improved outcomes, UGIB remains associated with significant morbidity and mortality, particularly when bleeding is severe or recurrent [1, 3].

Oesophageal variceal haemorrhage is an important cause of UGIB and occurs in the setting of portal hypertension. Portal hypertension may result from cirrhotic and non-cirrhotic causes, including sinusoidal, presinusoidal, and postsinusoidal disorders [2, 4]. Acute variceal bleeding requires a different therapeutic approach from non-variceal UGIB, with early vasoactive therapy, antibiotic prophylaxis, and

timely endoscopic treatment forming important components of contemporary management [2, 4, 5]. In patients who survive an initial variceal haemorrhage, appropriate secondary prophylaxis is essential because recurrent bleeding is a major clinical concern [4, 5].

Peptic ulcer disease remains one of the principal causes of non-variceal UGIB and may present with haematemesis, melaena, epigastric pain, or a combination of these features [1]. Clinical manifestations alone, however, may not reliably distinguish variceal from non-variceal bleeding. Consequently, upper gastrointestinal endoscopy remains central to establishing the source of bleeding and determining appropriate definitive therapy [1, 5]. Current recommendations emphasize early endoscopic assessment in hospitalized patients with overt UGIB and source-directed endoscopic haemostasis when indicated [1, 5].

The present case is unusual because it involves recurrent overt UGIB in a young Ugandan man who had previously undergone upper gastrointestinal endoscopy demonstrating gastritis and grade III oesophageal varices. Despite the documented varices, the patient had no known chronic medical illness and denied alcohol use or illicit drug use; examination also revealed no jaundice, ascites, peripheral oedema, or other obvious clinical manifestations of chronic liver disease. His recurrent presentation with haematemesis, melaena, significant weakness, and epigastric pain therefore raises important clinical considerations regarding the source of bleeding and the underlying cause of his oesophageal varices. The case highlights the importance of definitive endoscopic reassessment and appropriate evaluation of patients with recurrent UGIB and previously documented varices.

The purpose of reporting this case is to describe the clinical presentation, diagnostic considerations, management, and early outcome of recurrent upper gastrointestinal bleeding in a young patient with previously documented grade III oesophageal varices and gastritis.

CASE PRESENTATION

A 28-year-old Ugandan man presented to a regional referral hospital with a two-day history of haematemesis and generalized body weakness. He reported sudden-onset vomiting of a large volume of blood, estimated at approximately 2 litres over two days, occurring at least twice daily, without an identifiable precipitating or relieving factor. At the onset of the haematemesis, he also passed bloody stool, which subsequently changed to black stool. He developed constant burning upper abdominal pain, predominantly in the epigastric region, which he rated as 8/10 in severity. The pain was associated with reduced appetite and weight loss and had no identified aggravating or relieving factors. He also reported generalized weakness for two days, which significantly limited his ability to perform his usual activities.

He experienced dizziness during the bleeding episode and reported a mild frontal headache, blurred vision, and awareness of his heartbeat. He denied cough, chest pain, dyspnoea, orthopnoea, fever, convulsions, syncope, loss of consciousness, urinary frequency, dysuria, haematuria, or abnormal urethral discharge. He had no history of joint pain or joint swelling.

This was his second episode of similar symptoms and his third hospital admission. Approximately three months earlier, he had presented with haematemesis, abdominal pain, and generalized weakness and was admitted to another hospital, where upper gastrointestinal endoscopy demonstrated gastritis and grade III oesophageal varices. He received two units of blood during that admission. He reported no known chronic medical illness and no known drug or food allergies. Three years earlier, he had undergone abdominal surgery for a hernia, with no reported history of trauma or accidents. He was not taking any documented long-term medication. He denied tobacco, alcohol, and illicit drug use. There was no reported family history of diabetes mellitus, hypertension, asthma, or sickle cell disease. He reported no history of swimming, fishing, or other involvement in water bodies.

Before referral, he had been managed at a private medical facility with intravenous fluids and antibiotics for three days. He was subsequently referred to the regional referral hospital for blood transfusion, where he received two units of blood on admission.

On examination, he was a young adult in fair general condition and not in respiratory distress. He was alert and oriented, with a Glasgow Coma Scale score of 15/15. He was afebrile, with a temperature of 36.7°C, but had marked conjunctival pallor and mild dehydration. There was no jaundice, lymphadenopathy, lower-limb oedema, finger clubbing, or central or peripheral cyanosis. Capillary refill time was greater than three seconds, and there was no palmar erythema. His blood pressure was 127/66 mmHg, pulse rate 78 beats/min, respiratory rate 19 breaths/min, and oxygen saturation 96% on room air.

Abdominal examination revealed a symmetrical abdomen of normal fullness with a periumbilical surgical scar. There were no visible dilated abdominal veins or visible peristalsis. Bowel sounds were present, with three bowel sounds heard within one minute. There were no abdominal aortic or renal artery bruits. Superficial palpation revealed epigastric tenderness associated with guarding and rigidity, without palpable abdominal masses or organomegaly. The liver span was approximately 12.4 cm, and the spleen was not palpable. Percussion was tympanic, with no shifting dullness.

Cardiovascular examination revealed an apex beat in the left fifth intercostal space at the mid-clavicular line. There were no thrills or bruits. S1 and S2 were audible, with a humming quality reported on examination. There was no raised jugular venous pressure. Respiratory examination showed a symmetrical chest with normal expansion, equal bilateral air entry, and vesicular breath sounds without added sounds. Neurological examination showed an alert and fully oriented patient with no focal neurological deficit.

The initial clinical impression was peptic ulcer disease, based on the history of burning epigastric pain associated with haematemesis and a previous similar episode. The differential diagnoses included bleeding oesophageal varices, gastro-oesophageal reflux disease, and Mallory-Weiss tear. His previous upper gastrointestinal endoscopy had demonstrated grade III oesophageal varices and gastritis, making recurrent variceal haemorrhage an important consideration.

Investigations requested included a complete blood count, blood grouping, cross-matching, Helicobacter pylori stool antigen testing, and repeat upper gastrointestinal endoscopy. No laboratory or repeat endoscopy results were documented in the available case information.

The patient was managed as an acute upper gastrointestinal bleed. He received intravenous normal saline 500 mL every three hours, intravenous paracetamol 1 g three times daily, intravenous omeprazole 80 mg twice daily, intravenous ampicillin-cloxacillin 500 mg three times daily for three days, intravenous metronidazole 200 mg twice daily for three days, and blood transfusion. He was kept nil per oral.

On the second day of admission, he remained alert and was not in respiratory distress. The stool had changed from bloody to black, while the epigastric pain had reduced slightly. However, marked fatigue, dizziness, and mild headache persisted. He remained afebrile, with marked conjunctival pallor and mild dehydration. His blood pressure was 128/86 mmHg, pulse rate 85 beats/min, respiratory rate 17 breaths/min, and oxygen saturation 96%. Abdominal examination continued to demonstrate epigastric tenderness without organomegaly or palpable masses. The liver span was approximately 12.3 cm, and bowel sounds were present. Cardiovascular, respiratory, and neurological examinations remained essentially unchanged.

The management plan on the second day included a post-transfusion complete blood count, intravenous normal saline 500 mL every four hours, intravenous omeprazole 40 mg twice daily, intravenous ampicillin-cloxacillin 500 mg three times daily, intravenous metronidazole 200 mg three times daily, intravenous tranexamic acid 1 g once daily, and regular monitoring of vital signs.

On the third day after admission, the patient reported only a mild headache, and the stool colour had changed from black to yellow. He remained in fair general condition, alert, and without respiratory distress. He was afebrile, with moderate but improving conjunctival pallor and mild dehydration. His blood pressure was 124/78 mmHg, pulse rate 89 beats/min, respiratory rate 21 breaths/min, and oxygen saturation 97% on room air. Abdominal examination continued to demonstrate epigastric tenderness without palpable masses or organomegaly; the liver span remained approximately 12.3 cm, with bowel sounds present and no shifting dullness. Cardiovascular, respiratory, and neurological examinations remained stable. The documented plan was to continue intravenous normal saline 500 mL every four hours, intravenous omeprazole 40 mg twice daily, intravenous ampicillin-cloxacillin 500 mg three times daily, intravenous metronidazole 200 mg three times daily, and intravenous tranexamic acid 1 g once daily.

DISCUSSION

This case describes a 28-year-old Ugandan man who presented with recurrent overt upper gastrointestinal bleeding characterized by approximately two days of haematemesis, melaena, epigastric pain, generalized weakness, dizziness and palpitations. He had experienced a similar episode approximately three months earlier, during which upper gastrointestinal endoscopy demonstrated gastritis and grade III oesophageal varices. On the current admission, he had marked conjunctival pallor,

mild dehydration and epigastric tenderness with guarding and rigidity, but no jaundice, peripheral oedema, ascites, palpable splenomegaly or other documented clinical features of chronic liver disease. He received intravenous fluids, blood transfusion, proton-pump inhibitor therapy and antibiotics, with subsequent clinical improvement over the first three days.

The presentation is consistent with clinically significant UGIB, given the combination of haematemesis and melaena with symptoms suggestive of substantial blood loss. Current guidelines emphasize early assessment of haemodynamic status, risk stratification, appropriate blood transfusion, and timely endoscopic evaluation in patients admitted with overt UGIB [1, 2]. The patient's marked conjunctival pallor and delayed capillary refill were compatible with significant blood loss and/or hypovolaemia, although a haemoglobin concentration was not available in the documented investigations. The decision to transfuse two units of blood should therefore be interpreted in the context of the patient's clinical condition and the absence of a documented admission haemoglobin value. Contemporary guidance generally recommends a restrictive red-cell transfusion strategy in UGIB, with a threshold around 7 g/dL in most hospitalized patients, while recognizing that individual clinical circumstances may modify this approach [1].

A particularly important feature of this case is the previously documented grade III oesophageal varices in a young patient without a known chronic illness or reported alcohol use. Variceal bleeding is strongly associated with clinically significant portal hypertension, but portal hypertension may have cirrhotic or non-cirrhotic causes [3]. Therefore, the absence of overt stigmata of chronic liver disease in this patient does not by itself establish or exclude an underlying hepatic or vascular disorder. The case record does not contain liver function tests, viral hepatitis investigations, abdominal ultrasonography with portal and splenic assessment, elastography, or other investigations capable of determining the aetiology of the varices. Consequently, a specific cause of portal hypertension cannot be assigned from the available findings.

The coexistence of previously documented grade III oesophageal varices and gastritis also creates an important diagnostic challenge. The current presentation included burning epigastric pain and epigastric guarding, which led the treating team to consider peptic ulcer disease as the leading diagnosis. However, these clinical features cannot reliably establish a non-variceal source of bleeding, particularly in a patient with known oesophageal varices. Current guidelines emphasize endoscopy as the definitive investigation for identifying the bleeding source and providing endoscopic treatment when indicated [1, 2, 4]. In this case, repeat upper gastrointestinal endoscopy was requested, but its result was not available in the documented record. Therefore, it is not possible to conclude whether the recurrent haemorrhage was caused by oesophageal varices, gastritis, peptic ulcer disease, or another lesion.

The management documented in this case partially overlaps with recommended supportive management of UGIB, particularly intravenous fluid administration, blood transfusion and acid suppression while awaiting definitive evaluation. For non-variceal UGIB, proton-pump inhibitor therapy is an established component of management, while endoscopy within 24 hours is recommended for

hospitalized patients [1, 2]. However, if acute variceal bleeding is suspected, contemporary guidance recommends early vasoactive therapy with agents such as terlipressin, octreotide or somatostatin, together with antibiotic prophylaxis and urgent endoscopic therapy, including endoscopic band ligation for oesophageal variceal bleeding [3, 4]. In the present case, antibiotics were administered, but no vasoactive agent or endoscopic haemostatic intervention is documented during the available three-day follow-up. Because the bleeding source was not confirmed during the documented admission, it is difficult to determine retrospectively whether the patient received optimal source-directed treatment.

The history of a previous episode associated with grade III oesophageal varices has important implications for secondary prevention. Current variceal-bleeding guidance recommends secondary prophylaxis after successful control of an oesophageal variceal haemorrhage, generally involving non-selective beta-blocker therapy in combination with repeated endoscopic variceal ligation where appropriate [3, 4]. The case record does not document whether such secondary prophylaxis was initiated following the first episode. The recurrence therefore highlights the importance of establishing a documented follow-up plan after a first variceal bleeding episode. It would be inappropriate, however, to conclude that the recurrence resulted from failure of prophylaxis because the previous treatment and follow-up arrangements are not documented.

Another important issue is the use of tranexamic acid. The patient received intravenous tranexamic acid 1 g once daily from the second day of admission. This treatment is relevant because the large HALT-IT randomized controlled trial involving 12,009 patients with significant gastrointestinal bleeding found that tranexamic acid did not reduce death due to bleeding and was associated with an increased risk of venous thromboembolic events [5]. The investigators concluded that tranexamic acid should not be used routinely for acute gastrointestinal bleeding outside a randomized trial [5]. Thus, the use of tranexamic acid in this case is not supported by contemporary evidence and represents an important opportunity for clinical practice review. The patient's subsequent clinical improvement cannot be attributed to tranexamic acid because the case involved several simultaneous interventions and there was no control or objective assessment of its individual effect.

The case also demonstrates the importance of completing the diagnostic evaluation after stabilization. The requested investigations included complete blood count, blood grouping and cross-matching, *Helicobacter pylori* stool antigen testing and repeat upper gastrointestinal endoscopy, but the results were not available in the case record. This limits interpretation of the severity of anaemia, confirmation of *H. pylori* infection, identification of the active bleeding lesion, and assessment of the current status of the previously documented varices. In particular, without repeat endoscopy, the clinical improvement and change in stool colour cannot be taken as evidence of definitive haemostasis or confirmation of the bleeding source.

The main strength of this case is the clear documentation of recurrent symptoms, a previous objective endoscopic finding of grade III oesophageal varices and gastritis, clinical examination findings, treatment administered, and short-term clinical evolution. It also illustrates a clinically relevant

diagnostic dilemma between variceal and non-variceal UGIB in a young patient. The major limitations are the absence of documented laboratory results, absence of the repeat endoscopy findings, lack of investigation establishing the underlying cause of the oesophageal varices, and limited follow-up beyond the first three days. These limitations prevent definitive attribution of the current bleeding episode to either variceal haemorrhage or peptic/erosive disease and limit assessment of long-term recurrence prevention.

Overall, this case emphasizes that recurrent haematemesis and melaena in a young patient with previously documented oesophageal varices should be managed as potentially serious UGIB requiring prompt stabilization and definitive endoscopic assessment. The absence of obvious stigmata of chronic liver disease should not lead clinicians to dismiss previously demonstrated varices, while clinical symptoms alone should not be used to determine whether bleeding is variceal or non-variceal. The key lesson is that **early stabilization must be followed by confirmation of the bleeding source, evaluation of the underlying cause of portal hypertension where varices are present, and appropriate secondary prevention; routine tranexamic acid should not be used for acute GI bleeding in light of current evidence** [1, 3–5].

CONCLUSION

This case highlights recurrent upper gastrointestinal bleeding in a young man with previously documented grade III oesophageal varices and gastritis, presenting with haematemesis, melaena, epigastric pain and features of significant blood loss. Despite the absence of known chronic illness or overt clinical stigmata of chronic liver disease, the history of documented oesophageal varices emphasizes the need to consider variceal bleeding alongside non-variceal causes of UGIB. The case demonstrates the importance of prompt stabilization, appropriate transfusion, definitive upper gastrointestinal endoscopic evaluation to establish the bleeding source, and assessment for the underlying cause of portal hypertension and strategies to prevent recurrent variceal bleeding.

Declarations

ETHICAL APPROVAL

Ethical approval for this case report was obtained from **Kayunga Regional Referral Hospital**.

Consent for Publication.

Written informed consent for publication of this case report and its accompanying clinical information was obtained from the patient. The patient was informed about the purpose of the case report, the clinical information to be presented, and the potential for publication in a scientific journal. He was assured that reasonable measures would be taken to protect his identity and confidentiality. No personally identifiable information is included in the manuscript.

COMPETING INTERESTS

The authors declare that they have no competing interests.

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