Superior Mesenteric Venous Thrombosis Presenting with Hematemesis: A Case Report

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Summary

The superior mesenteric vein (SMV) is one of the two tributaries to the portal vein, which is the main pre-hepatic drainage channel of the splanchnic circulation. Venous thrombosis in the SMV is one of the rare causes of splanchnic ischemic syndrome. Clinical presentation is often vague abdominal symptoms. It seldom presents with hematemesis, which may further confound diagnosis, thereby increasing the risk of poor outcomes. This case highlights an otherwise healthy 31-year-old woman, who presenting at a tertiary hospital in Nairobi with a 2-day history of epigastric pain and copious hematemesis. She had been treated for *Helicobacter pylori* gastritis 2 months prior. Common differentials were considered. However, with normal esophagogastroduodenoscopy findings and dark-colored fluid sequestered in the upper gastrointestinal tract, mesenteric venous congestion with associated small bowel ischemia was suspected. Three-phase abdominal computed tomography angiogram confirmed proximal superior mesenteric venous thrombosis extending into the portal vein confluence. Associated small bowel necrosis was suspected, and surgery involving resection and primary ileo-jejunal anastomosis was performed. Post-operative 2-week parenteral nutrition and anticoagulation were administered. The patient was discharged after 3 weeks of hospitalization and remains in excellent condition.

Keywords: Hematemesis, Intestinal thickening, Mesenteric thrombosis, Ischemia and necrosis, Portal vein

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Introduction

The superior mesenteric vein (SMV) drains the small intestines and proximal half of the large intestines into the portal vein. Primary and secondary factors maycause thrombus formation in this vessel, leading to acute or chronic superior mesenteric venous thrombosis (SMVT). This is a relatively rare cause of mesenteric ischemia (1). In the acute type, the most common presenting symptom is vague abdominal pain, nausea, vomiting, and rarely hematemesis (2). Diagnostic challenges associated with acute SMVT are well known.

However, with improved diagnostic capacities and early multispecialty involvement, delays in appropriate care, which otherwise are common with this condition, have been ameliorated in recent times, and the associated high mortality thereby reduced (3). This is a report of a case of a 31-year-old woman who had a unique presentation of mesenteric ischemia and was managed at a tertiary hospital in Nairobi, Kenya.

Case presentation

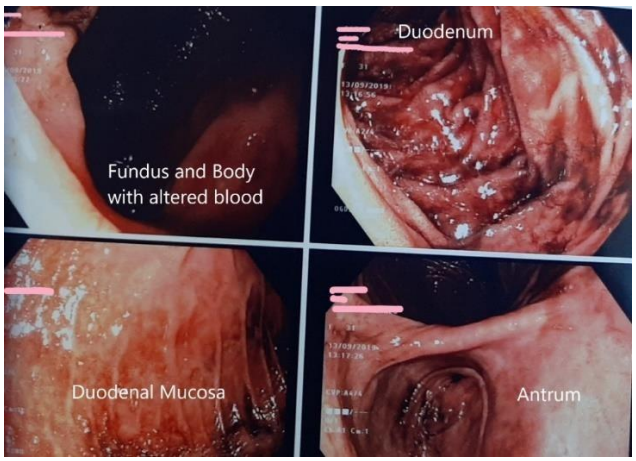
Clinical presentation

A 31-year-old Kenyan female presented to the accident and emergency department with a 2-day history of dull epigastric pain that was increasing in severity. The pain was aggravated by movements and not relieved by over-the-counter analgesics or antacids. Within a few hours, vomiting started, which became copious. The vomitus was coffee ground and non-bilious. Stool and flatus were passed normally and had no melena. Two months prior to this presentation, she had been treated for *Helicobacter pylori* gastritis. She got some relief until this presentation. Three years prior to this presentation, she experienced on and off burning pain at the epigastrium. It was exacerbated by eating particular foods and relieved by antacids. Otherwise, she was healthy and had no change in lifestyle. She had three previous deliveries, the last one being via cesarean section due to twin breech pregnancy 4 years prior. Subsequently, she used combined oral contraceptive pills. At presentation, she was in fair general condition and not in obvious pain. Her body mass index was 29 kg/m^2. She had dry mouth, pulse rate of 110 beats per minute, blood pressure of 125/77 mmHg, and temperature of 36.0^{o}C. There was epigastric and supraumbilical tenderness and mild distension at the upper abdomen. The results of digital rectal examination and other abdominal examination were normal. Systemic examination was also unremarkable.

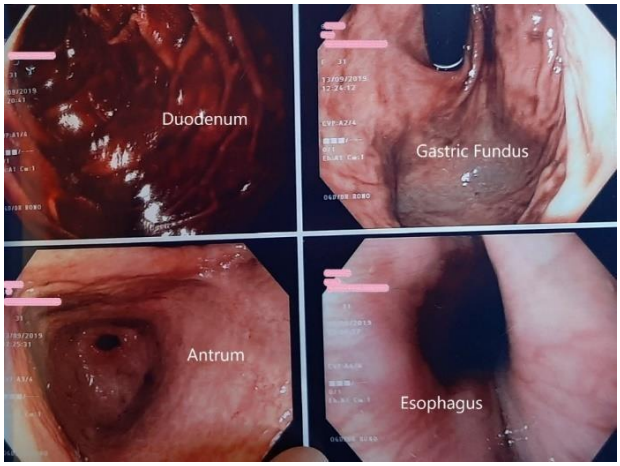
Management

Laboratory blood works showed leukocytosis (15.45×10^9/L) with neutrophilia (86%). Hemoglobin was 155 g/L. Liver enzymes, electrolytes, blood urea nitrogen, serum amylase, and lipase were normal. Broad-spectrum antibiotics, high-dose proton-pump inhibitors, intravenous fluid, analgesics, and anti-emetics were started, and the patient was not given anything by mouth.

Esophagogastroduodenoscopy (EGD) revealed a large-volume dark-colored fluid in the stomach and duodenum that was apparently flowing back from the jejunum. The mucosa appeared normal; neither ulcers nor active bleeding were observed in the scoped areas (Figure 1A and B).



(A)



(B)

Figure 1. Esophagogastroduodenoscopy (A) before suctioning showing dark colored bloody gastric contents and (B) after suctioning showing normal mucosa.

With a negative EGD result, the cause was suspected to be beyond the duodenum. The likelihood of splanchnic vasocongestion involving the mesentery was

entertained. A triple-phase abdominal computed tomography (CT) angiogram revealed a filling defect at the proximal SMV extending to the confluence of the portal vein. The stomach was markedly distended, the jejunum was dilated and had thickened walls but negative for pneumatosis intestinalis. Some ascites was present. There was no pneumoperitoneum. Other viscera were normal. A diagnosis of SMVT with extension into the portal vein was made with associated ischemic small bowel (Figure 2).

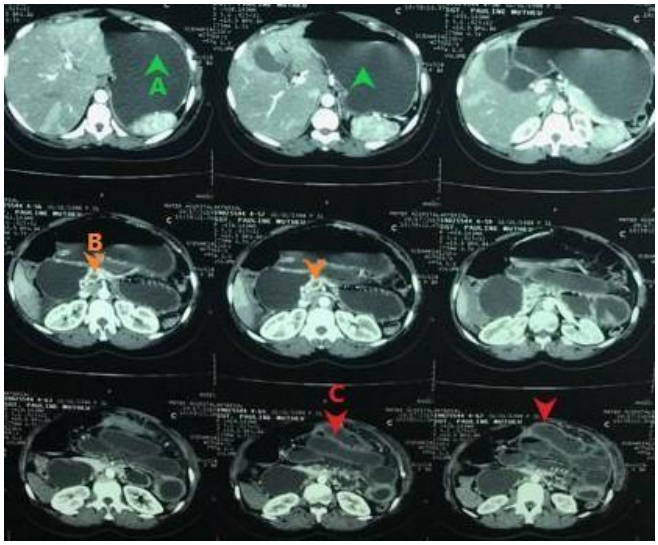


Figure 2. Computed tomography angiographic images of the abdomen showing salient features of superior mesenteric venous thrombosis. (A) Green arrows showing distended fluid-filled stomach. (B) Orange arrows showing filling defect in SMV. (C) Red arrows showing dilated and thick-walled jejunal loops

Laparotomy was indicated, which was supported further by a rapidly increasing white cell count that reached 33.93×10^9/L and decreasing hemoglobin (92 g/L) within 48 hours. Nonetheless, no peritoneal signs had developed.

Intraoperatively, 1 L of dark-colored, non-foul-smelling ascitic fluid was observed. The omentum had gathered and snugly covered most of the jejunal bowel loops. Upon separation of this entanglement, approximately 170 cm of necrotic jejuno-ileal segment with clear demarcations was found starting from 18 cm distal to the duodeno-jejunal junction to 210 cm proximal to the ileo-cecal junction. The 18-cm jejunal stump was edematous but well perfused. The mesentery was also edematous (Figure 3). Jejunal and ileal arcades were normal, but vasa arcades and jejunal veins were mildly engorged. The visualized superior mesenteric vessels appeared normal. No thrombosis was evident up to the ligament of Treitz and was not explored beyond this ligament.

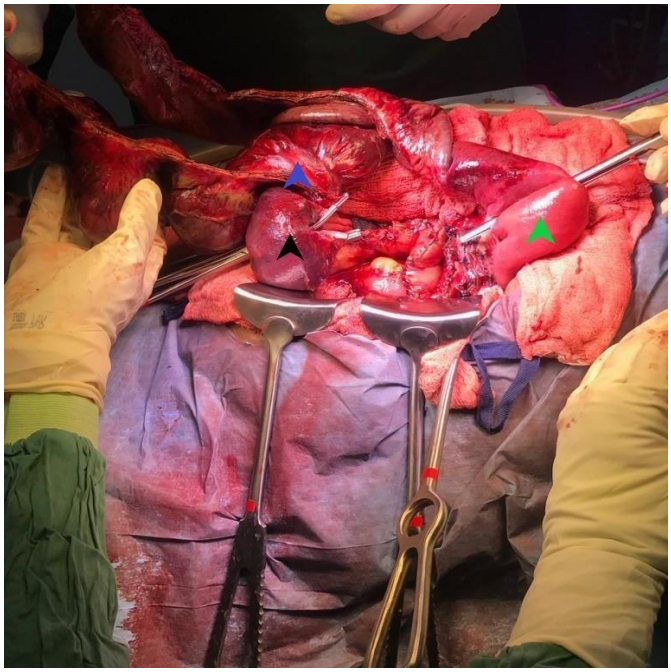


Figure 3. Key intraoperative findings. Blue arrow, edematous mesentery; black arrow, necrotic small bowel loop; green arrow, viable proximal jejunum

The necrotic segment was resected, and a secure hand-sewn primary jejuno-ileal anastomosis was performed. The rectus was closed, but the skin was left open. The patient was managed in the critical care unit for the first 24 hours under a low threshold for relook surgery, but she did not require reoperation. Her vital signs and condition quickly improved. She was transfused, and antibiotics were continued. Low-molecular-weight heparin was instituted 24 hours post-operatively. The nasogastric tube drained minimally at 48 hours and was removed. The bowel was rested, and total parenteral nutrition was administered for 2 weeks. Delayed primary closure of the skin was performed on post-operative day 5.

Outcome and follow-up

The post-operative period was uneventful. Enteral intake was resumed on post-operative day 14. Heparin was changed to rivaroxaban, and the patient was discharged on post-operative day 17. Connective tissue disorder screening showed normal results. Screening for familial thrombophilia was negative. Repeat abdominal CT angiogram performed 2 weeks post-operatively showed some interval resolution of the mesenteric thrombus. No other abnormality was noted. A follow-up CT angiogram performed 3 months later showed no residual thrombus in the SMV or the portal vein. Non-hormonal contraception according to the patient's choice was instituted. Anticoagulation was continued indefinitely. At 24 months, the patient was in excellent condition with no abdominal symptoms whatsoever.

Discussion

Etiology and pathophysiology

SMVT is one of the rare causes of a broad spectrum of ischemic syndromes of the splanchnic viscera and peritoneal structures (3). SMVT accounts for 6–9% of mesenteric ischemia. It can be acute or chronic, primary or secondary. SMVT without identifiable cause accounts for 21–49% of cases. Secondary SMVT is commonly caused by prothrombic states, hematologic malignancies, myeloproliferative diseases, pancreatitis, surgery, intra-abdominal infections, and bowel inflammatory diseases. Oral contraceptives have been incriminated in 9–18% of SMVT among young women (4). This was the only probable risk factor identified in our patient.

The most common complaint is abdominal pain, which was reported in 91–100% of patients. Anorexia, nausea, vomiting, and diarrhea are common. Patients may rarely present with hypotension (3, 5, 6). Together with melena and hematochezia, hematemesis is a rare presentation of acute SMVT. It is present only in 15% of cases (2). Abdominal examination is often less remarkable than the symptoms (5s). The unique presentation with hematemesis such as our patient's may be explained by the occlusion of the mesenteric vein impeding venous return from the affected bowel. The veins and venous arcades become engorged, increasing local hydrostatic pressure. These cause stasis ischemia, bowel edema, and egression of the fluid into the lumen. Mucosal sloughing occurs, leading to intraluminal bleeding. The resulting dark-colored fluid sequestered in the affected intestinal tract causes gut dilatation and may be vomited or passed as diarrhea or melena. Hypovolemia may ensue, causing splanchnic hypoperfusion. Autoregulatory vasoconstriction risks complete shutdown of local circulation. Being an acute incident, no collaterals are available; hence, bowel necrosis is likely to occur within 6–8 hours (1, 3, 4, 7-9).

Diagnosis and investigation

The rarity of acute SMVT presenting with hematemesis and vague abdominal pain presents a diagnostic challenge especially in a low-resource clinical setting (8). Obvious differential diagnoses of upper gastrointestinal bleeding, such as bleeding peptic ulcer, may delay consideration of the actual diagnosis, thus delaying appropriate care, which has been reported at a median of 48 hours (6). EGD is indicated in nearly all cases of upper gastrointestinal (GI) bleeding (8). A normal EGD helps rule out common causes of upper GI bleeding. In our case, the copious dark-colored gastrointestinal fluid with a normal EGD findings hinted at the possibility of a vascular cause beyond the ligament of Treitz. Triple-phase abdominal CT angiography can reliably investigate splanchnic vessels, with a sensitivity of 90%. Besides, it can reveal other diagnoses and any complication if present (3, 4). In our case, the CT angiogram confirmed the SMV/portal venous thrombosis and showed small-bowel dilatation and wall thickening. An alternative imaging technique is magnetic resonance angiography, which possesses a higher sensitivity and specificity of 100% and 98% respectively (10). Digital subtraction angiography, which has both diagnostic and therapeutic utilities, is a good option. As a standard of care, it can accurately diagnose the cause of splanchnic ischemia and allows for necessary revascularization interventions. Doppler ultrasound ca.n also be a useful tool, with diagnostic accuracies ranging from 80% to 90% in experienced hands (3). Plain abdominal radiograph may show

changes, such as gut dilatation, wall thickening, pneumatosis intestinalis, or pneumoperitoneum in 70% of cases that occur late in the disease process. It can also help to exclude other diagnoses such as intestinal obstruction (4).

Blood works such as the hemogram, liver function tests, electrolytes, serum amylase, and lipase help in the assessment of patient physiology and may give insight to the disease process. In our patient, the increasing white cell count, decreasing hemoglobin, and normal serum amylase and lipase clarified the need for surgery.

Treatment and outcomes

Treatment of acute SMVT should be individualized. An algorithm exists in approaching suspected SMVT (4). The possibility of intestinal bacteremia should be anticipated and broad-spectrum antibioticsadministered. Surgery is required in approximately 64% of patients, 90% of whom need bowel resection (6). If feasible and safe, surgical thrombectomy may be done during laparotomy. Measures to avoid short bowel syndrome should be taken as far as practicable.Extensive or complete thrombosis without emergentcompromise to the bowel may require angiographic thrombectomy. Otherwise, anticoagulation suffices for the majority of those who do not reach the threshold for surgery. Anticoagulation improves mortality outcomes whether or not surgery was required and should becontinued for at least 6 months (4, 6, 8). With anticoagulation, the thrombus re-canalizes within 6 months in more than 90% of cases. However, it recurs in36% of cases within 5 years in the same or different splanchnic vein (6). Long-term or lifelong anticoagulation reduces the risk of recurrence tobetween 0% and 14%, more so when the causative factoris untreatable or unknown (6).

With improved awareness and diagnostic capacities across various clinical setups, better outcomes of acute mesenteric venous ischemia, which has a mortality rate as high as 44% (4), is possible

Conclusion

Early presentation to hospital and early multispecialty involvement provide definitive care in a timely manner, which, thus, improves outcomes. In a patient with vague abdominal symptoms, coffee ground vomitus, and normal EGD findings, the possibility of superior venous mesenteric ischemia should be considered, especially if there is dilated and thickened fluid-filled small bowel. With a rapidly increasing white cell count, severe bowel compromise and necrosis should be suspected, and emergent surgical exploration should be considered.

Ethical consideration

Institutional approval was granted for the publication of this case report. Consent was obtained from the patient for the case presentation and use of images and pictures with concealed identity

Author contributions

EM led in the conceptualization and writing of the first draft. All other authors contributed equally to reviewing and editing the original draft.

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