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Melena and Silent Mesenteric Ischemia: Portomesenteric Thrombosis without Tenderness

Amirabbas Sheykhalo[1*], Seyed Hamzeh Mousavi[2], Mohsen Khaleghian[3]

1. Department of General Surgery, Iran University of Medical Sciences, Tehran, Iran.
2. Department of Colorectal Surgery, Iran University of Medical Sciences, Tehran, Iran.
3. Department of Vascular Surgery, Iran University of Medical Sciences, Tehran, Iran.

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ABSTRACT

Acute mesenteric venous thrombosis is an uncommon but potentially fatal cause of mesenteric ischemia, whose nonspecific presentation can delay diagnosis. We report a 46-year-old man with no prior thromboembolic, hepatic, or malignant disease who presented with five days of periumbilical pain, vomiting, and melena. Upper endoscopy revealed hematin without an identifiable bleeding source. Computed tomography angiography revealed extensive portal, splenic, and superior mesenteric venous thrombosis with jejunal ischemia. Intravenous heparin was started, and the patient was monitored closely because his abdomen remained soft and non-tender. After approximately 48 hours, he developed sudden hemodynamic collapse; emergency laparotomy revealed 100 cm of necrotic jejunum, which was resected without primary anastomosis. Despite intensive postoperative support, he died of cardiovascular collapse approximately 24 hours later. This case shows that extensive portomesenteric thrombosis can cause severe and fatal intestinal ischemia despite reassuring abdominal findings, underscoring the need for prompt imaging, anticoagulation, and vigilant reassessment.

Introduction

Acute mesenteric ischemia is a life-threatening condition in which delayed recognition may lead to intestinal infarction, systemic deterioration, and death. Mesenteric venous thrombosis (MVT) is an important mechanism of acute mesenteric ischemia and most commonly involves the superior mesenteric vein, with possible extension into the portal and splenic venous systems; modern contrast-enhanced CT has substantially improved its early recognition [1-4]. The clinical presentation of acute MVT is often nonspecific: abdominal pain may be severe, while physical findings remain limited, particularly early in the disease course, and gastrointestinal bleeding may direct the initial evaluation toward a primary luminal source [2, 5]. Systemic anticoagulation is the mainstay of treatment in patients without peritoneal signs, whereas intestinal infarction or clinical deterioration necessitates surgical exploration and resection of

*** Corresponding Author:**
Amirabbas Sheykhalo
Address: *Department of General Surgery, Iran University of Medical Sciences, Tehran, Iran.*
E-mail: *dr.a.sheykhalo@gmail.com*

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nonviable bowel [1, 3, 6].

Here, we report a fatal case of extensive portomesenteric venous thrombosis in a previously healthy 46-year-old man who initially presented with abdominal pain and melena, had no peritoneal signs despite radiologic evidence of intestinal ischemia, and subsequently developed abrupt hemodynamic deterioration requiring emergency laparotomy.

Case Presentation

A 46-year-old man presented with a 5-day history of progressively worsening periumbilical abdominal pain aggravated by oral intake, progressive anorexia, and three episodes of food-containing vomiting. On the night before admission, he developed two episodes of moderate-volume melena, which prompted him to seek care. He had no history of abdominal surgery, thromboembolic events, known coagulation disorders, malignancy, liver disease, or other significant illness; he took no regular medications, reported no relevant family history, and smoked approximately 10 cigarettes per day.

On admission, the vital signs were stable, and the abdomen was soft without tenderness, guarding, or rebound. Melena led to suspicion of an upper gastrointestinal bleeding source. Esophagogastroduodenoscopy showed a normal esophagus and a large amount of hematin in the stomach and duodenum, but no definite bleeding source was identified. The patient was transferred to the intensive care unit for close monitoring. A nasogastric tube yielded coffee-ground material, and a subsequent decline in hemoglobin prompted CT angiography.

CT angiography demonstrated partial portal vein thrombosis extending into the right and left portal branches and the splenic vein at the portosplenic confluence, together with complete thrombosis of the superior mesenteric vein extending into segmental and peripheral branches. Long-segment circumferential thickening of jejunal loops, mesenteric vascular engorgement, fat stranding, and a small-to-moderate amount of free intraperitoneal fluid were also observed, findings highly suggestive of mesenteric ischemia secondary to extensive superior mesenteric venous thrombosis (Figure 1).

Intravenous unfractionated heparin was started as a 5,000-IU bolus followed by a continuous infusion at 1,000 IU/h, with serial activated partial thromboplastin time monitoring targeting 50–70 seconds; heparin was continued before, during, and after surgery. Because the patient had no peritoneal signs, he underwent intensive monitoring, including hourly clinical assessment, serial abdominal examinations, and repeated assessment of nasogastric output. The abdomen remained soft throughout, he remained conscious and alert with preserved urine output, and no metabolic acidosis was detected. The white-

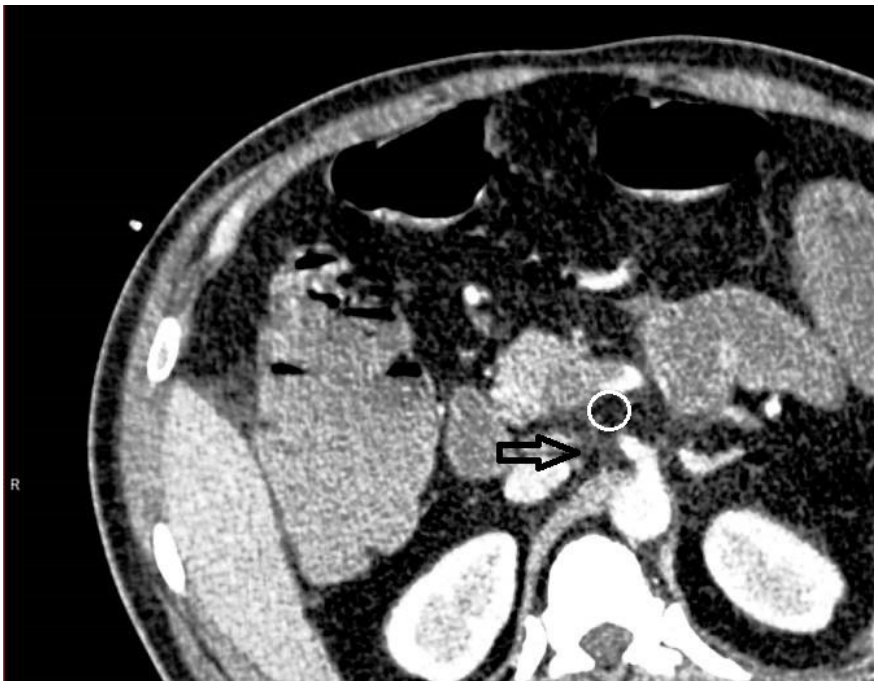


Fig. 1. Contrast-enhanced abdominal CT angiography demonstrating extensive portomesenteric venous thrombosis and radiologic features of mesenteric ischemia. White circle: SMV (superior mesenteric vein) Black arrow: Portal vein



Fig. 2. Intraoperative photograph showing extensive ischemic and necrotic small bowel with marked mesenteric edema and black discoloration. (White arrow: bowel necrosis)

cell count was approximately 12,000/µL and did not clearly increase during observation, and hemoglobin did not decline further.

An evaluation for thrombotic, autoimmune, infectious, and rheumatologic causes was unrevealing: rheumatoid factor was negative, antinuclear antibody was 0.1, anti-cyclic citrullinated peptide antibody was 0.67 by ELISA, and lupus anticoagulant was not detected. Complement levels were C3 195 and C4 23, respectively. Serologic tests for hepatitis B surface antigen, hepatitis C virus, and HIV were negative. The procalcitonin level was 6.63 ng/mL, and the LDH level was 448 U/L at the final assessment. Serum creatinine levels increased from 1.7 mg/dL on admission to 3.1 mg/dL before death. No clear provoking factor for extensive thrombosis was identified.

Approximately 48 hours after diagnosis, the patient developed abrupt hemodynamic deterioration with sudden hypotension and tachycardia exceeding 140 beats/min. He remained conscious, and his abdomen showed no tenderness or peritoneal signs, with no clear worsening of leukocytosis or metabolic acidosis. Given the sudden cardiovascular deterioration in the setting of extensive portomesenteric thrombosis and CT evidence of intestinal ischemia, emergency exploratory laparotomy was performed.

At laparotomy, approximately 200 mL of bloody intraperitoneal fluid was evacuated. The jejunum and its mesentery showed extensive ischemic and necrotic changes; the mesentery was markedly edematous and black, and no palpable arterial pulse was detected in the superior mesenteric artery or the affected mesenteric vessels. The involved bowel showed complete loss of peristalsis and dark-black discoloration, and bled from the resection margins during transection. Edematous bowel that retained peristaltic activity and lacked frank necrosis was preserved; only grossly nonviable, black, aperistaltic bowel was resected. Approximately 100 cm of small bowel was resected from approximately 15 cm to 120 cm distal to the ligament of Treitz (Figure 2). Because of persistent hemodynamic instability, primary anastomosis was deferred, the abdomen was closed, and a second-look laparotomy was planned for 48 hours later.

Postoperatively, the patient required mechanical

Table 1. Timeline of the patient's clinical presentation, diagnostic evaluation, treatment, surgery, and outcome

Time point	Clinical events
Day -5	Progressive periumbilical abdominal pain began and was aggravated by oral intake.
Days -5 to -1	The patient experienced progressive pain, anorexia, and three episodes of food-containing vomiting.
Day -1	Two episodes of moderate-volume melena were observed.
Day 0 - Admission	The patient was hemodynamically stable, and the abdomen was soft without tenderness, guarding, or rebound.
Day 0 - Endoscopy	Normal esophagus; hematin in the stomach and duodenum; no definite bleeding source.
Day 0 - ICU	The coffee-ground nasogastric aspirate and hemoglobin levels subsequently declined.
Day 0 - CT angiography	Extensive portomesenteric thrombosis with complete SMV thrombosis and jejunal ischemia.
Day 0 - Anticoagulation	UFH 5,000 IU IV bolus, then 1,000 IU/h infusion; aPTT target 50-70 s.
Next ~48 h	Hourly assessment revealed that the abdomen remained soft, urine output was preserved, and there was no initial acidosis. The patient developed sudden hypotension and tachycardia >140/min; emergency exploratory laparotomy.
Intraoperative	~200 mL bloody fluid; ~100 cm black, aperistaltic necrotic jejunum resected; no palpable SMA pulse; primary anastomosis deferred; second look planned.
Postoperative 0-24 h	Mechanical ventilation, norepinephrine up to 50 µg/min plus vasopressin, and creatinine level rose from 1.7 to 3.1 mg/dL.
~24 h after surgery	Final assessment BP 60/30 mmHg; pulse 100/min; CVP 4 mmHg; VBG pH 7.25, pCO2 60, HCO3- 26.3, BE -1.8; LDH 448 U/L. Terminal bradycardia and loss of blood pressure; death from cardiovascular collapse. Second-look laparotomy was not performed.

Abbreviations: SMV, superior mesenteric vein; UFH, unfractionated heparin; aPTT, activated partial thromboplastin time; SMA, superior mesenteric artery; BP, blood pressure; CVP, central venous pressure; VBG, venous blood gas; LDH, lactate dehydrogenase.

ventilation, norepinephrine escalating to 50 µg/min, and vasopressin. He developed persistent hypotension, metabolic acidosis, and leukocytosis despite intensive support and substantial fluid resuscitation, although urine output remained preserved and the wound was satisfactory. Approximately 24 hours after surgery, his pulse was 100/min, blood pressure was 60/30 mmHg, and central venous pressure was 4 mmHg; venous blood gas showed pH 7.25, pCO2 60 mmHg, $HCO3^{-}$ 26.3 mmol/L, and base excess −1.8. He developed terminal bradycardia followed by loss of measurable blood pressure and died approximately 24 hours after surgery from cardiovascular collapse; the planned second-look laparotomy could not be performed.

The clinical courses of the patients are summarized in Table 1.

Discussion

This case illustrates several important features of extensive acute portomesenteric venous thrombosis. First, the severity of intestinal injury may be disproportionate to the physical examination: venous outflow obstruction causes intestinal congestion, bowel wall edema, and hemorrhage; therefore, substantial ischemia may occur while the abdomen remains soft and lacks peritoneal signs, particularly in earlier stages [1-4]. Second, gastrointestinal bleeding can be an important and potentially misleading presentation; in this patient, melena led to upper endoscopy, which showed extensive hematin but no bleeding source. Gastrointestinal bleeding has been described in acute MVT and can contribute to diagnostic delay [5], and when abdominal pain is accompanied by GI bleeding without an adequate endoscopic source, mesenteric venous disease should remain in the differential diagnosis. Third, contrast-enhanced CT is central to diagnosis; in this patient, CTA demonstrated thrombosis of the portal, splenic, and superior mesenteric venous systems, together with jejunal abnormalities compatible with ischemia, consistent with the high diagnostic value of CT in acute mesenteric ischemia [3, 7].

Systemic anticoagulation is the cornerstone of the treatment of acute MVT. Contemporary guidelines support anticoagulation in patients without peritoneal signs, whereas surgery is indicated when infarction is suspected or clinical deterioration occurs [1, 3, 6, 8]. In this case, unfractionated heparin was initiated promptly, and the patient was monitored intensively because he initially lacked peritoneal signs. The timing of surgery in MVT with suspected ischemia remains challenging: delayed or limited resection may preserve bowel when ischemia is potentially reversible, but clearly nonviable bowel requires resection, and when viability is uncertain or the patient is unstable, delayed anastomosis with a planned second-look procedure may be considered [6, 9]. Leukocytosis and a higher severity-of-illness score

have been identified as independent predictors of irreversible intestinal ischemia requiring resection in patients with acute MVT [10]; in this patient, the white-cell count remained relatively stable until the abrupt hemodynamic collapse, which limited its usefulness as an early warning sign in this particular case.

The operative findings demonstrated advanced injury, with resection of approximately 100 cm of black, aperistaltic bowel, while viable, peristaltic bowel was preserved. The absent SMA pulse should not be interpreted in isolation as evidence of primary arterial thrombosis, since preoperative CTA had already demonstrated extensive venous thrombosis; bleeding from the resection margins provided an additional intraoperative indicator of perfusion at the chosen transection sites. The patient's rapid postoperative deterioration and death despite resection and intensive hemodynamic support further emphasize the systemic consequences of advanced mesenteric ischemia; the rise in creatinine from 1.7 to 3.1 mg/dL and the terminal hemodynamic and blood-gas abnormalities occurred in the setting of refractory cardiovascular instability, and the planned second-look laparotomy could not be performed before death.

The principal limitations of this report are the absence of a second-look laparotomy because of the patient's death, the absence of a documented pathology report in the available records, and the limited availability of a complete thrombophilia workup; these limitations are acknowledged to avoid overinterpretation of the etiology of the thrombosis.

Conclusion

Extensive portomesenteric venous thrombosis can cause severe and fatal intestinal ischemia despite an initially soft, non-tender abdomen. Gastrointestinal bleeding may obscure the diagnosis when endoscopy does not identify a definite source. Prompt CT angiography, early systemic anticoagulation, repeated clinical reassessment, and timely operative intervention when irreversible ischemia is suspected are essential. This case highlights the potentially deceptive clinical course of acute MVT and the need for continued vigilance, even when early findings appear reassuring.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this article.

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Conflict of Interests

The authors have no conflict of interest to declare.

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