Superior mesenteric venous thrombosis causing intestinal infarction: Report on two clinical cases

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Abstract

Introduction: Superior mesenteric venous thrombosis (SMVT) is a complication of intra-abdominal venous disease that poses a serious risk to patients. It is one of the significant causes of acute intestinal ischemia, accounting for approximately 16% of total cases. Recent research and treatment experiences have improved the diagnosis and treatment of SMVT, particularly due to advancements in imaging technology and enhanced medical knowledge. This report describes two cases of irreversible intestinal ischemia caused by superior mesenteric venous thrombosis from different etiologies, treated with surgical resection of the affected bowel segment combined with anticoagulation therapy. Early diagnosis and combined treatment, priority is given to anticoagulation treatment and monitoring. The surgical indication is for irreversible intestinal ischemia by clinical manifestation and imaging diagnosis.

Keywords: superior mesenteric venous thrombosis, irreversible intestinal ischemia, bowel resection.

Introduction

Superior mesenteric venous thrombosis (SMVT) occurs when a thrombus forms in the superior mesenteric vein, obstructing blood flow and leading to intestinal ischemia. This complication plays a crucial role in cases of acute intestinal ischemia, accounting for 16% of these cases [1]. SMVT has various causes, including deficiencies in anticoagulant factors such as protein C, protein S, antithrombin III, inflammatory conditions, and abdominal surgeries or trauma. Liver cirrhosis and heart failure can also increase the risk of SMVT[2]. The main symptoms of SMVT typically include acute abdominal pain, often accompanied by nausea and vomiting. The most severe complication is acute intestinal ischemia, which can lead to death if not promptly addressed.

Diagnosis primarily relies on the patients history of risk factors (cardiovascular disease, coagulopathy, etc.), CT imaging to detect thrombosis before treatment, and quantifying coagulation factors (protein C, protein S, etc.) to identify deficiencies. Initial treatment usually involves anticoagulation with intravenous heparin or subcutaneous heparin. In severe cases with intestinal necrosis, surgical resection of the affected bowel segment is necessary to save the patients life. The prognosis of SMVT treatment depends on the speed of diagnosis and accurate treatment. Identifying risk factors and early intervention can minimize mortality and future complications[3]. We report two recent clinical cases of patients with superior mesenteric vein thrombosis requiring bowel resection, and from there, review the literature on this condition.

Clinical cases

Case 1: A 24-year-old obese male (BMI 34) presented with left flank pain for 7 days accompanied by nausea. He had a history of femoral vein thrombosis 6 years ago, which was surgically treated, anticoagulated, and involved the placement and later removal of an inferior vena cava filter. The patient had been hospitalized at a lower-level facility for 4 days without improvement and was transferred in a state of: pulse 100 beats/min, blood pressure 100/60 mmHg, SpO2 100%, marked abdominal distension, and left flank tenderness with abdominal guarding. Laboratory tests showed: neutrophil count 20 G/L, PT 67%, CRP 296, D-Dimer 20,949 mcg/L, Protein S 17%, Protein C 68%, Antithrombin III 51%. CT imaging of the abdomen revealed: complete thrombus in the superior mesenteric vein and splenic vein extending into the portal vein, poorly enhanced segments of the small intestine in the left flank, and significant free abdominal fluid. The patient was diagnosed with irreversible intestinal ischemia due to superior mesenteric venous thrombosis and underwent emergency surgery for bowel resection, restoration of blood flow, and enterostomy. Systemic heparin therapy was started immediately post-surgery. The patient resumed oral intake on the 8th day post-surgery and transitioned from systemic heparin to low molecular weight heparin (Lovenox) and finally to Xarelto 20 mg/day. The postoperative recovery was stable, and the patient was transferred to a hematology center for further treatment on the 10th day post-surgery.

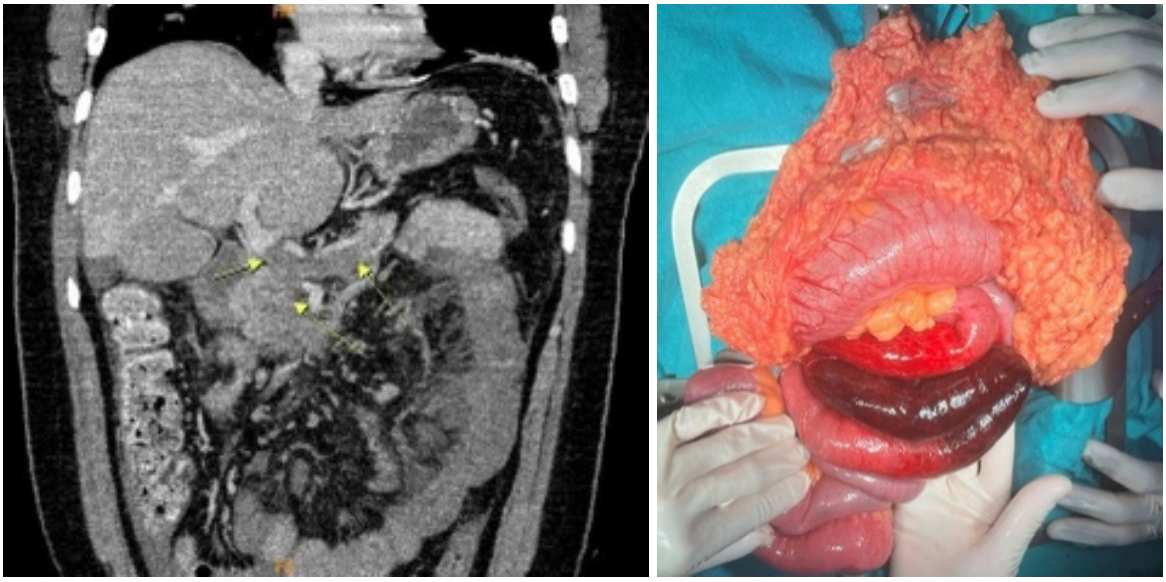

Image 1: CT scan showing complete thrombus in the superior mesenteric vein, part of the splenic vein, and portal vein (yellow arrow), with poorly enhanced bowel loops (blue arrow). The surgical image shows 40 cm of bowel removed due to infarction.

Case 2: A 62-year-old female with a history of splenectomy 10 years ago, partial thyroidectomy 6 years ago, and liver cirrhosis presented with periumbilical pain, constipation, and vomiting for 5 days prior to hospital admission. She was transferred in a state of: pulse 141 beats/min, blood pressure 100/60 mmHg, SpO2 90%, significant abdominal distension, periumbilical tenderness, abdominal guarding, and inability to pass gas. Laboratory tests: leukocyte count 16 G/L, platelet count 135 G/L, D-Dimer 34,639 mcg/L. CT imaging of the abdomen showed: significant free abdominal fluid, complete occlusion of the superior mesenteric vein and portal vein, and poorly enhanced segments of the small intestine. The patient was diagnosed with septic shock and bowel necrosis due to superior mesenteric venous thrombosis and cirrhosis. Prior to surgery, the patient received systemic heparin and discontinued it 4 hours before surgery. The intraoperative findings included 2 meters of nonviable small intestine with irreversible ischemia 1.8 meters from the Treitz angle and 40 cm from the cecum. The patient underwent resection of the necrotic bowel segment and formation of a stoma. Systemic heparin was reintroduced immediately after surgery. The patient resumed oral intake 7 days post-surgery and transitioned to Xarelto 20 mg/day. The postoperative recovery was stable, and the patient was discharged on the 13th day post-surgery.

Discussion

Causes and Risk Factors

SMVT occurs when a thrombus forms within the superior mesenteric vein, obstructing blood flow from the intestines and potentially leading to intestinal ischemia and infarction. The development of SMVT is often multifactorial, involving both inherited and acquired conditions that predispose to hypercoagulability or venous stasis.

Inherited Thrombophilias

Protein S deficiency: Protein S is a natural anticoagulant that works with protein C to deactivate factors Va and VIIIa. A deficiency, whether congenital or acquired, disrupts this pathway, increasing the risk of venous thrombosis, including SMVT[4].

Protein C deficiency: Similar to protein S, a deficiency in protein C increases the risk of thrombus formation due to impaired inhibition of clotting factors[5].

Factor V Leiden mutation: This genetic mutation leads to resistance to activated protein C, resulting in an increased risk of venous thrombosis[6].

Prothrombin gene mutation (G20210A): This mutation increases the production of prothrombin, raising the likelihood of thrombosis[7].

Acquired Risk Factors

Malignancies: Abdominal and gastrointestinal cancers are associated with a hypercoagulable state, which can predispose patients to mesenteric vein thrombosis[8].

Inflammatory diseases: Conditions such as inflammatory bowel disease (e.g., Crohn's disease, ulcerative colitis) have been linked to SMVT due to chronic inflammation and endothelial damage[9].

Intra-abdominal infections: Localized infections such as diverticulitis, pancreatitis, or appendicitis can lead to thrombosis in the superior mesenteric vein due to local inflammation and venous stasis[10].

Post-surgical status: Major abdominal surgeries, particularly involving bowel resections, can trigger SMVT due to direct trauma to the vessels, immobility, or postoperative hypercoagulability[11].

Liver cirrhosis and portal hypertension: These conditions impair venous return from the intestines, leading to venous stasis and an increased risk of thrombus formation[12].

Obesity: Obesity (defined by a body mass index [BMI] ≥ 30) has been shown to increase the risk of venous thromboembolism. Increased intra-abdominal pressure in obese individuals may contribute to venous stasis in the mesenteric veins, increasing the risk of thrombosis [13].

Trauma: Direct abdominal trauma, including from accidents or blunt force, can damage the mesenteric vessels, precipitating thrombosis[14].

Other Hematologic Disorders

Polycythemia vera: An elevated red blood cell mass increases blood viscosity, contributing to thrombosis in various vascular beds, including the mesenteric vein[15].

Paroxysmal nocturnal hemoglobinuria (PNH): A rare hematologic disorder that leads to venous thrombosis, particularly in the abdominal veins.

In our study, the 24-year-old male patient had a history of femoral vein thrombosis, surgical thrombus removal, and anticoagulation but had the inferior vena cava filter removed. He had a deficiency in protein S (17%), which increases the risk of venous thrombosis, particularly in prone areas like the superior mesenteric vein and splenic vein. Protein S deficiency may be hereditary or due to insufficient production. Obesity (BMI 34) may also affect protein S function. Identifying and treating protein S deficiency is crucial for managing this patient to reduce future thrombotic risks and optimize treatment outcomes. According to a study by A.W. Broekmans et al[16], protein S deficiency is a rare cause, but when plasma protein S levels fall below 50% of normal, it can lead to mesenteric venous thrombosis. In this patient, there are three risk factors may have contributed to the formation of superior mesenteric vein thrombosis: low blood levels of protein S, obesity, and a history of previous surgery.

The second patient had a history of splenectomy, partial thyroidectomy, and cirrhosis. These conditions, along with the surgeries, maybe create structural vascular changes and affect the body's natural coagulation system, increasing thrombotic risk [2][11][12].

Diagnosis and Treatment

Diagnosis: Acute thrombosis often presents with abdominal pain, whereas chronic disease manifests either as an incidental finding on CT or with features of portal hypertension. Contrast-enhanced CT diagnoses about 90% of cases. CT angiography is the gold standard for diagnosing SMVT. Doppler ultrasound can also be used but is less sensitive. Protein S/C levels, D-dimer, and coagulation profiles may assist in identifying underlying causes like thrombophilia [17].

Fever and abdominal guarding suggest progressing intestinal ischemia or infarction. Some studies report risk factors for bowel resection in SMVT patients, including elevated leukocytes [18], peritoneal inflammation, increased lactate levels, and bowel wall thickening on CT [19].

Both of our patients were diagnosed with intestinal ischemia due to superior mesenteric vein thrombosis, in which the second patient thought the cause was protein S deficiency.

Treatment: Most patients with SMVT without peritoneal irritation signs receive initial anticoagulation therapy: intravenous heparin for severe cases and low molecular weight heparin for milder symptoms. When clinical symptoms improve, oral anticoagulants (DOACs) or vitamin K antagonists (VKAs) may be considered. However, 10%-20% of patients do not respond to anticoagulation and require bowel resection [3]. Anticoagulation is the primary treatment for most SMVT patients. For those with identified risk factors, anticoagulation should be maintained for 6 months, while patients with hidden coagulopathies or unidentified risk factors may need lifelong anticoagulation. The goal is to prevent intestinal ischemia, portal hypertension, and recurrence, as recurrence significantly increases mortality. Anticoagulation should start as early as possible [3].

Endovascular interventions may be effective for some patients resistant to or unresponsive to anticoagulation therapy, including thrombectomy or local thrombolysis via endovascular intervention [20]. However, the efficacy of these methods remains unclear due to limited studies and lack of control group comparisons for irreversible intestinal ischemia cases. Some studies suggest that 55% of patients undergoing endovascular intervention may

still require bowel resection due to bowel narrowing or progressing necrosis[19].

According to a study by Andraska et al[20], approximately 85% of patients with superior mesenteric vein thrombosis can be treated with anticoagulation, 5% may undergo endovascular intervention to avoid bowel resection, and 10% require surgical bowel resection.

Surgical intervention is necessary in many cases with signs of irreversible bowel ischemia, although there are no clear criteria for bowel resection. Bowel infarction should be resected, and early resection of infarcted bowel segments is advisable[21]. Decisions on performing an intestinal anastomosis depend on the remaining bowel and associated risk factors. In our two cases, one underwent immediate reestablishment of intestinal flow because the remaining upper segment of the intestine is short and the other had a stoma. Intravenous heparin should be used early post-surgery. Identified risk factors should be managed accordingly. In our first case, after identifying protein S and Antithrombin III deficiencies, treatment was combined by both internal medicine and surgically. The patient was transferred to a hematology center for further management once postoperative conditions stabilized.

Conclusion

Superior mesenteric venous thrombosis is a rare surgical pathology. From the two cases requiring bowel resection that we encountered had risk factors including protein S deficiency, cirrhosis, and post-multiple surgeries. Initial treatment involves anticoagulation therapy, with surgery indicated when there are signs of irreversible bowel ischemia. Follow-up and anticoagulation treatment after surgery for patients with high-risk factors are crucial in preventing disease recurrence.

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