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Case Report

Post-traumatic superior mesenteric venous thrombosis with subsequent extension to the portal vein

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ABSTRACT

Acute superior mesenteric vein thrombosis was first described in 1935 by Warren and Eberhardt. It is a potentially life-threatening condition, as it can lead to bowel ischemia and, ultimately, infarction. Its etiology may be primary or secondary to acquired prothrombotic conditions. Early recognition of mesenteric venous thrombosis is important, but can be challenging due to its nonspecific clinical presentation. Contrast-enhanced computed tomography is currently the gold standard for diagnosis. Systemic anticoagulation and surgical resection of the necrotic segment are the two main treatments. Here, we describe a case of acute post-traumatic superior mesenteric vein thrombosis, which was treated with systemic anticoagulation and resection of the ischemic bowel segment, with subsequent extension of the thrombosis to the portal vein.

Introduction

Acute mesenteric vein thrombosis was first described as a clinical entity by Warren and Eberhardt [1]. It accounts for 5%-15% of all cases of acute mesenteric ischemia, with the superior mesenteric vein being the most frequently affected [2,3].

The widespread use of computed tomography (CT) imaging has made early diagnosis possible by a noninvasive approach. Contrast-enhanced CT, which has approximately 90% accuracy, is now the gold standard for diagnosis [2].

Some of the common risk factors for the development of acute mesenteric vein thrombosis include a prothrombotic state, surgery, inflammatory bowel disease, malignancy pancreatitis, and infection [4]. Cases of acute mesenteric vein thrombosis associated with polytrauma (serious vehicle accidents) or multiple stab wounds to the abdomen have been reported in the literature [5].

Systemic treatment with anticoagulant therapy and selective resection of the necrotic bowel segment are the best treatments to prevent extension of the thrombosis, thereby limiting the necessary resection [6].

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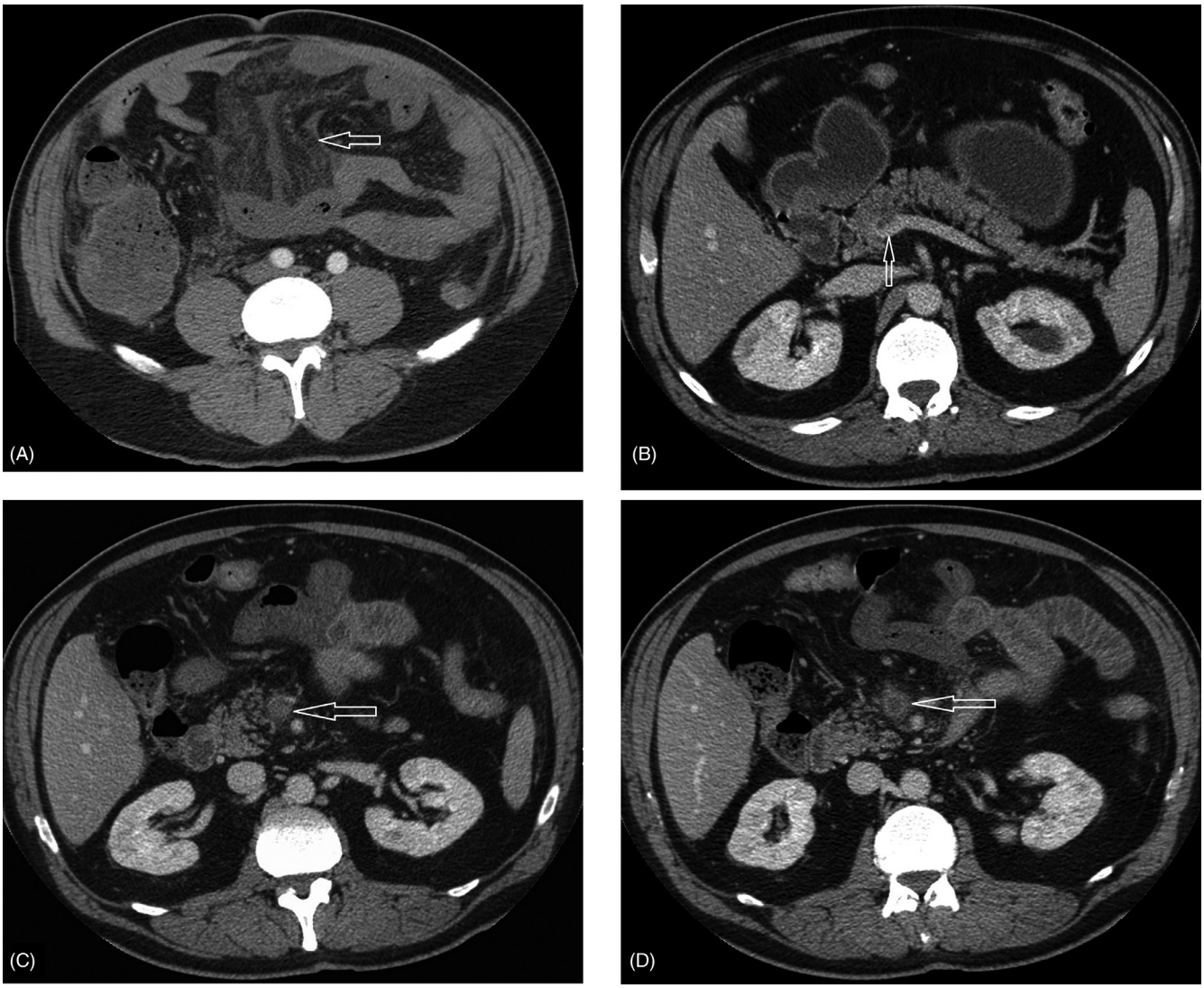

Fig. 1 – Contrast-enhanced computed tomography (CT) of a 55-year-old male patient with superior mesenteric vein thrombosis. (A) Contrast-enhanced CT showing an ischemic ileal loop with wall thickening, initial pneumatosis of the wall, and adjacent free liquid (white arrow). (B) Extension of the thrombosis to the confluence of the superior mesenteric vein with the splenic and portal veins (white arrow). (C and D) Thrombosis of the superior mesenteric vein with a filling defect during venous phase contrast-enhanced CT (white arrow).

Here, we describe a case of acute superior mesenteric vein thrombosis associated with blunt trauma to the abdomen.

Case report

A 55-year-old male with a history of non–insulin-dependent diabetes mellitus, arterial hypertension, and previous cholecystectomy presented to the emergency unit of our institution with continuous and slowly increasing epigastric and mesogastric abdominal pain, which had begun approximately 48 hours prior, accompanied by nausea but not vomiting or fever. During discussion with the patient, he reported having suffered blunt trauma to the abdomen 48 hours prior during a soccer match that caused him strong pain, forcing him to quit the match. The pain had diminished during the day but had not disappeared. The patient reported that a continuous increase in pain led to him present to the emergency department.

On admission, clinical examination revealed diffuse abdominal pain upon palpation of the epigastric and mesogastric regions, diminished peristalsis and abdominal sounds, negative Murphy sign, and positive Blumberg sign. Laboratory tests showed an increased white blood cell count (13830/mm^3; 91% neutrophils), hemoglobin level of 14.6 mg/dL, platelet count of 248,000 mm^3, erythrocyte sedimentation rate of 26.5 mm/h, C-reactive protein level of 29.1 mg/L, a high D-dimer level of 1564 μg/dL, and lactate level of 3.7 mmol/L. The abdominal x-ray showed no specific findings.

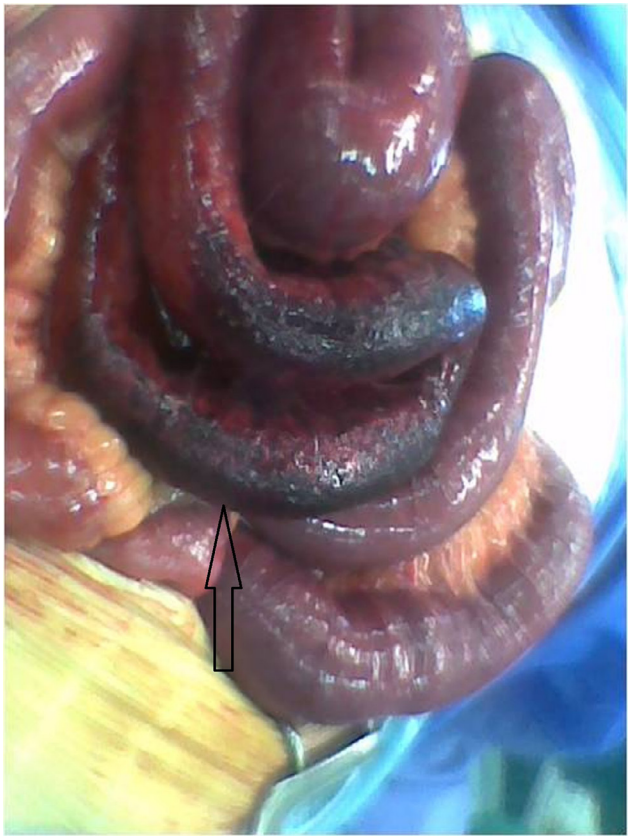

Fig. 2 – Ischemic ileum loop discovered during laparotomy, which was subsequently resected (black arrow).

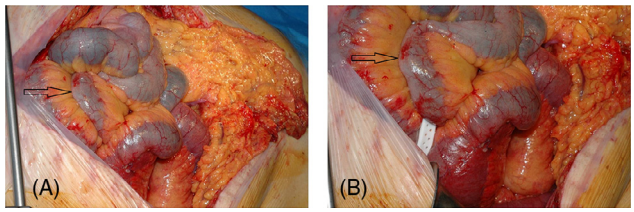

Fig. 3 – Second-look surgery. (A and B) Normal vascularization and trophy of the ileal loops, with no extension of necrosis (black arrows).

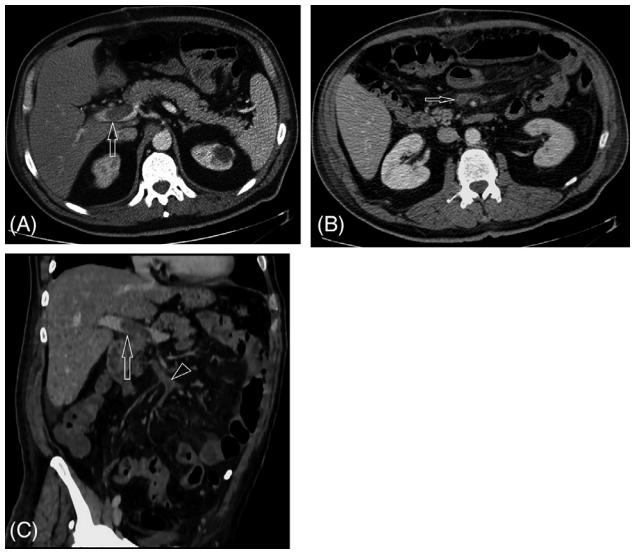

Fig. 4 – Contrast-enhanced computed tomography (CT) on day 5 postoperative. (A) Extension of the thrombosis to the portal vein with a filling defect during venous phase contrast-enhanced CT (white arrow). (B) Presence of superior mesenteric vein thrombosis with a filling defect during venous phase contrast-enhanced CT (white arrow). (C) Oblique reconstructed image showing thrombosis with a filling defect in the superior mesenteric vein (white arrow head) and its extension to the portal vein (white arrow).

Shortly after, the patient reported an increase in abdominal pain. During a repeat clinical evaluation, we observed an abdominal defense reaction and an absence of bowel movements and sound. A contrast-enhanced CT scan was performed, which revealed a complete thrombosis of the upper mesenteric vein that extended until the confluence with the portal and splenic veins, as well as an ischemic ileum segment with edematous thickening, initial pneumatosis, decreased contrast enhancement of the wall, and free fluid in the abdominal and Douglas pouch. Some mesenteric lymphatic nodes in the peritoneum showed increased dimensions (Fig. 1).

The patient was immediately heparinized and sent to the operating room for explorative laparotomy. During the surgery, a necrotic ileum segment, almost 100 cm in length (Fig. 2), was resected, then laterolateral functional ileoileal anastomosis was performed. Hemoperitoneum was detected during the surgery.

On postoperative day 2, the patient underwent a scheduled second-look surgery, which showed no extension of bowel ischemia and anastomotic integrity (Fig. 3).

The patient was placed on anticoagulation therapy with low-molecular-weight heparin (1 mg/kg twice daily, administered as a subcutaneous injection of enoxaparin) and antibiotic therapy with amoxicillin and clavulanic acid (1-g Augmentin twice daily). The postoperative course was clinically uneventful.

On the fifth day postoperative, an increase in transaminase levels was detected (aspartate transferase (AST) 53 U/L and alanina transferasi (ALT) 65 U/L). A control contrast-enhanced CT scan was performed, which showed extension of the thrombosis to the portal vein (Fig. 4).

A vitamin K antagonist (warfarin) was subsequently added to the therapy regimen. The patient's clinical condition began to improve, and the patient was discharged on postoperative day 9.

One month later, the patient underwent a scheduled control contrast-enhanced CT scan, which showed reduced thrombosis in the portal vein and permanence of the superior mesenteric vein thrombosis (Fig. 5). The patient continued with the same therapy regimen. A follow-up contrast-enhanced CT at 2 months showed complete resolution of the thrombus in the vena porta and superior mesenteric vein

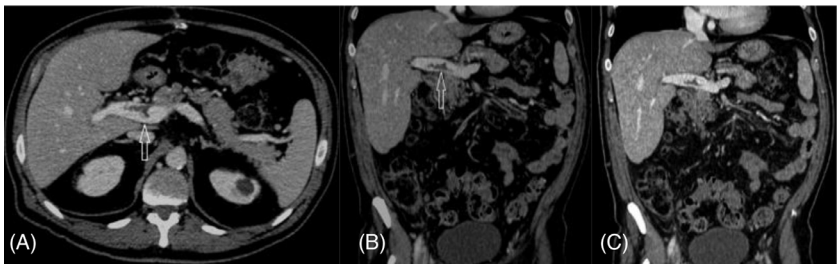

Fig. 5 – Follow-up contrast-enhanced computed tomography after 1 month. (A) Axial image showing a reduction in portal vein thrombosis (white arrow). (B and C) Coronal images showing a reduction in portal vein thrombosis (white arrow), with no evidence of superior mesenteric vein thrombosis.

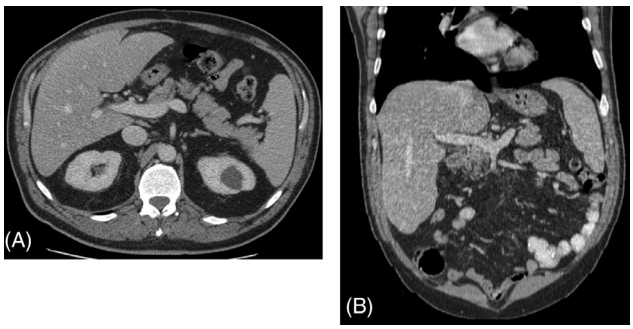

Fig. 6 – Follow-up contrast-enhanced computed tomography after 2 months. (A) Axial and (B) coronal images showing complete resolution of the thrombosis in the portal and superior mesenteric veins.

(Fig. 6). Anticoagulant therapy was continued for 6 months, then switched to aspirin at a dose of 75 mg/d for life.

Discussion

Acute mesenteric vein thrombosis was first described as a clinical entity by Warren and Eberhardt [1].

It is a rare but potentially life-threatening emergency, and accounts for 5%-15% of all cases of acute mesenteric ischemia. The superior mesenteric vein is most commonly affected [2,3,7,8]. It most often involves the ileum (64%-83%) and jejunum (50%-81%), followed by the colon (14%), and duodenum (4%-8%) [5].

The mean age of patients is between 45 and 65 years, with a slight male predominance [4]. The complete occlusion of the vein leads to bowel ischemia, and even necrosis in some cases. The duration of symptomatology and time to diagnosis are important predictive factors for the outcome and the need for surgery, and patients with symptoms for up to 3 days more frequently require laparotomy [9].

The primary etiologies that cause the thrombosis include both inherited and acquired prothrombotic states, such as protein C and S deficiencies, antithrombin III deficiency, factor V Leiden mutation, prothrombin G20210A mutation, hyperhomocysteinemia, activated protein C resistance, lupus anticoagulants, and antiphospholipid antibodies. The most frequently acquired prothrombotic state results from intra-abdominal neoplasia, inflammatory bowel disease, pregnancy, major trauma, postoperative conditions (especially splenectomy), cirrhosis, and portal hypertension. Oral contraceptive use is responsible for 9%-18% of mesenteric vein thrombosis (MVTs) in young women [3,5,10–13]. When the underlying etiology cannot be identified, the thrombosis is defined as primary or idiopathic [2].

The duration of symptoms varies from patient to patient, lasting from 5 to 14 days, with >75% of patients presenting after >48 hours of symptomatology [3]. The most common symptom is abdominal pain, which occurs in 91%-100% of cases, and other common symptoms include nausea, vomiting, and melena. The extent of abdominal pain is usually not consistent with the findings of the physical examination. Melena, hematemesis, or hematochezia occurs in about 15% of cases, and occult blood is present in 50% of cases. Fever and peritoneal signs are suggestive of progression of the infarction, and hypotension (systolic blood pressure of less than 90 mmHg) together with the formation of ascites is associated with poor prognosis. Leukocytosis and hemoconcentration are commonly observed. Serum lactate levels may increase, but do not always correlate with the severity of the thrombosis [2,5].

The diagnostic method of choice is contrast-enhanced CT with venous phase, which is now considered the gold standard. This noninvasive method has over 90% accuracy, and has notably reduced the time to diagnosis in addition to the mortality rate, which had previously been 20%-50%. The most common signs of ischemia of the bowel are wall thickening, due to edema and hemorrhage, with abnormal or no enhancement, the halo sign and pneumatosis of the wall. The venous phase should depict a venous filling defect with vein enlargement and engorgement of the mesenteric veins. Mesenteric fat edema is frequently observed, and ascites may also be present [14–16].

The treatment consists of anticoagulation and surgical resection of the necrotic segment.

Systemic anticoagulation with heparin should be started as soon as the diagnosis is made in order to decrease the proportion of patients requiring surgery, thereby improving the outcome and reducing the risk of recurrence. Some studies have demonstrated that anticoagulation therapy increases

vein permeability in nearly 80% of cases. In patients with reversible causes, such as trauma, it is recommended that anticoagulation therapy be continued for 3-6 months [4,17]. Surgical treatment should be decided upon clinical evaluation. If patients develop general or local peritonitis, resection with anastomosis should be performed. The goal of surgery should be to conserve as much bowel as possible. A second-look surgery should be performed 24 hours later to evaluate bowel vitality and extension of the necrosis [2,10,17,18].

This case highlights that abdominal blunt trauma, even if it is not associated with a major traumatic event, should not be underestimated. In these cases, it is important to monitor laboratory findings and perform clinical evaluation during post-operative follow-up.

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