

# Superior Mesenteric Vein Thrombosis in an Adult Patient

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Mesenteric venous thrombosis (MVT) refers to acute, subacute, or chronic thrombosis and occlusion of the mesenteric veins. MVT is an uncommon disorder, with an estimated incidence of 2.7 per 100,000 patient-years. It accounts for 1 in 5000 to 15,000 inpatient admissions [1,2].

Venous thrombosis mainly results from Rudolf Virchow's classic triad of blood flow stagnation, vascular epithelial injury, and hypercoagulability. Location, dimensions, and rate of thrombosis are important factors that affect the extent of intestinal tissue damage. Inadequate venous flow and increased resistance due to acute thrombotic occlusion lead to infiltration of fluid into the tissues, leading to hypoxia of the affected tissues, which can result in the severe form, in ischemia and ultimately infarction of the bowel tissue.

Risk factors for MVT are commonly divided into acquired hypercoagulable states (e.g., nephrotic syndrome, pregnancy), inherited disorders (e.g., thrombophilias), and intra-abdominal traumatic or inflammatory conditions (e.g., pancreatitis, postoperative).

Considering the non-specific and variable presenting symptoms of MVT, a high index of suspicion is needed to diagnose this condition. Physical assessment, laboratory testing, and abdominal X-ray

are of limited use and lack sufficient sensitivity or specificity to reliably diagnose or exclude the presence of MVT. Considering its accuracy and relative availability, contrast-enhanced computed tomography (CT) angiography is the diagnostic imaging modality of choice for MVT [3].

Following diagnosis, the main objective of successful treatment is to prevent or minimize intestinal infarction, prevent recurrences, and identify predisposing hypercoagulable conditions. Treatment for acute patients is based on intravenous fluids, prophylactic antibiotics, bowel rest, and decompression. Anticoagulation remains the mainstay of therapy in patients with MVT, which is used for unfractionated heparin or low molecular weight heparin for the inpatient, and oral anticoagulant (e.g., vitamin K antagonist, novel oral anticoagulant [NOAC]) in out-patient settings. Interventional therapeutic options include fibrinolysis, thrombectomy, or abdominal surgery, often reserved for hemodynamically unstable patients [4]. This case report describes the progression of an acute MVT of the superior mesenteric vein.

## PATIENT DESCRIPTION

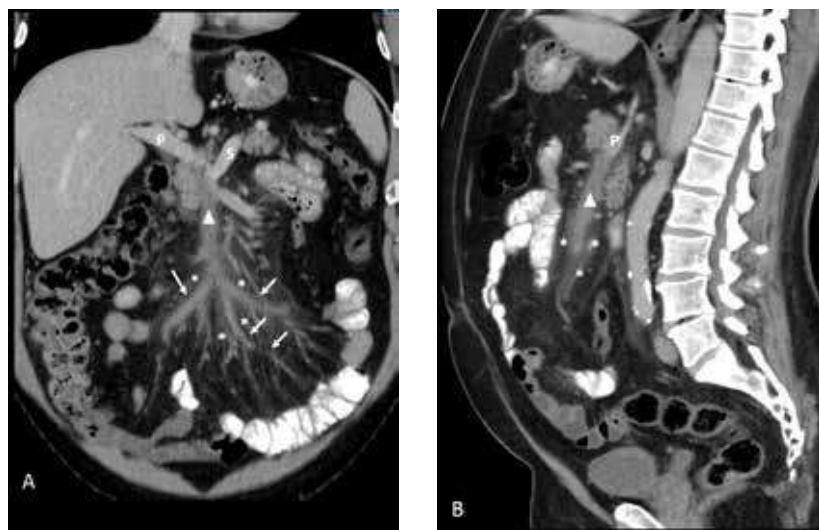
A 56-year-old male patient presented to the emergency department (ED) complaining of abdominal pain. His medical history included past smoking, essential hypertension, and elective cholecystectomy 3 years prior.

He reported sharp pain in the left upper abdominal region that started 3 days prior to his admission. Furthermore, the

pain was not relieved by over-the-counter oral analgesics. He denied any other gastrointestinal, urinary, or respiratory symptoms. Body temperature was 37.5°C measured orally. Other vital signs were within the normal range. The patient appeared alert and oriented on physical examination, without pathological findings on cardiac and respiratory assessment. Abdominal examination revealed a soft abdomen with marked tenderness of the epigastric and right upper quadrant regions. Laboratory test results were unremarkable apart from an elevated C-reactive protein of 214 mg/l (normal range < 5 mg/l). Initial imaging studies included a plain abdominal X-ray and abdominal ultrasonography both of which were unremarkable. Due to persisting abdominal pain, a CT of the abdomen was performed and revealed pyelophlebitis of the superior mesenteric vein (SMV) with thrombosis extending to the portal vein. No acute etiology was detected. Mesenteric venous thrombosis was diagnosed [Figures 1A and 1B].

The patient was admitted to the internal medicine department. He was monitored with successive abdominal examinations and laboratory tests for any signs of clinical worsening, such as peritonitis related to intestinal ischemia, infarction, or small bowel obstruction. He received anticoagulation treatment with subcutaneous injections of low molecular weight heparin. A combination of ceftriaxone and metronidazole was prescribed as prophylactic antibiotics. To identify the cause of mesenteric thrombosis, blood samples were drawn for hypercoagulable states, looking especially for signs of a

**Figure 1.** Thrombosed superior mesenteric vein (SMV). Filling defects are seen in the SMV (arrowhead) as well as the draining jejunal and ileal veins (arrows). Significant fat standing is seen adjacent to these vessels (asterisk)



CT = computed tomography, P = enhanced portal vein, S = enhanced splenic vein

myeloproliferative neoplasm, *JAK2* sequence variation, protein S/C deficiency, factor V Leiden mutation, and antiphospholipid antibodies. After 3 days of hospitalization with no signs of worsening or progression of the mesenteric vein thrombosis, the patient was discharged with no abdominal pain or other symptoms. Subsequent treatment comprised short-term, prophylactic antibiotics and long-term anticoagulation with 5 mg apixaban twice daily.

## COMMENT

We presented a report of an acute, unprovoked life-threatening MVT of a previously healthy 56-year-old man. Since described in 1935, mortality associated with acute MVT has decreased due to early diagnosis. But regardless of anti-thromboembolic treatment advances and wide availability of CT scans, MVT is still associated with an overall high mortality rate of up to 44% in severe cas-

es [4]. Short-term mortality is dependent on whether infarction is present. Earlier diagnosis and adequate therapy improve the prognosis considerably. Mortality is less than 10% in rapidly diagnosed and treated patients as in our case [5]. Considering the non-specific nature of MVT clinical signs and symptoms as well as laboratory assessment, a high index of suspicion is required for performing an accurate diagnosis. Improvements in diagnostic testing increased awareness of the disorder, and the wide availability of contrast-enhanced CT in the ED shortened the time interval considerably from presentation to MVT diagnosis in the past 50 years [3].

Predisposing risk factors for MVT development are found in most patients, although idiopathic cases (originally termed primary MVT) in multiple studies range from 21–49% [2]. Abdominal risk factors for developing MVT, such as an abdominal neoplasm, surgery, or inflammatory bowel disease, are usually appar-

ent on medical history, physical examination, and abdominal imaging. However, hypercoagulable state disorders are generally less pronounced. Considering that many of the predisposing risk factors for MVT development are associated with increased mortality and morbidity and subsequent recurrent thrombotic events [5], Identifying those risk factors is imperative for the patient's well-being and prognosis.

## CONCLUSIONS

In our case, a diagnosis of MVT was made using abdominal CT in the ED due to subjective abdominal pain symptoms and scant laboratory abnormalities. The patient received adequate therapy and was discharged after a short hospitalization. A high index of suspicion along with a low threshold for the utilization of contrast-enhanced abdominal CT improved this patient outcome and prognosis considerably.

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**A wise man fights to win, but he is twice a fool who has no plan for possible defeat.**

Louis L'Amour (1908–1988), American novelist and short story writer