

## CASE REPORT

# Primary mesenteric vein thrombosis: a case series

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## Abstract

Mesenteric vein thrombosis (MVT) is a rare condition, often misdiagnosed due to its vague and misleading clinical presentation. It can cause intestinal infarction, peritonitis, and consequently necessitate bowel resection. CT scanning with intravenous contrast enhancement is the gold standard for its diagnosis. Radiologists have an important role in defining the extent of thrombosis and identifying any signs of intestinal infarction influencing the decision whether or not to operate. In patients with no clinical signs of peritonitis or radiological evidence of intestinal infarction, the treatment can be exclusively medical, based on full anticoagulation (initially with low molecular weight heparin, followed by vitamin K antagonists or direct acting oral-anticoagulants). The duration of medical treatment depends on radiological evidence of resolution of thrombosis and the identification of pro-coagulant risk factors.

## CASE 1

I.P., a 48-year-old man, presented with abdominal pain, which had started 3 weeks earlier as a diffuse abdominal discomfort and worsened in the previous 24 hours. Blumberg's sign was positive, serological markers showed mild neutrophilic leukocytosis, with high CRP and normal serum lactate levels.

A CT scan showed a massive thrombosis involving the superior mesenteric vein, the splenic vein and the origin of the portal vein. Small bowel wall edema and a modest amount of pelvic and peri-hepatic free fluid were also detectable.

The patient underwent an urgent laparotomy, during which a 90-cm segment of ischemic small bowel was resected and primary anastomosis was performed. A Meckel diverticulum was incidentally found and resected. Histology of the surgical specimen confirmed the diagnosis of venous intestinal infarction and Meckel diverticulum with an area of gastric metaplasia.

No inherited causes or acquired factors of thrombophilia were identified. The patient continued therapy with low molec-

ular weight heparin (LMWH) for 30 days and was then switched to direct oral anti-coagulant (DOAC).

## CASE 2

M.R.C., a 72-year-old female, presented with biliary vomiting and constipation that had started 4 days prior to admission. Abdominal X-ray showed multiple colic air–fluid levels. Serological markers showed mild neutrophilic leukocytosis, high CRP and normal lactate levels.

The patient had a history of deep vein thrombosis. Her sister had intestinal infarction due to mesenteric and portal vein thrombosis; her granddaughter had pulmonary embolism and her nephew experienced two myocardial infarctions. No thrombophilic gene mutations were ever documented in the patient or her relatives.

A CT scan showed partial occlusion of the pulmonary right artery, thrombosis of the portal, superior and inferior mesenteric veins, dilation of the proximal small bowel, mesenteric edema and a small amount of abdominal free fluid.

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Considering the absence of abdominal pain, surgery was not performed immediately and unfractionated heparin (UH) infusion was started.

Four days later, a repeat CT scan showed persistent of small bowel dilation with signs of intestinal occlusion.

Seven days after admission, the patient underwent laparotomy and resection of a small segment of ischemic jejunum with primary anastomosis.

LMWH was administered for 20 days and then replaced with vitamin K antagonist (VKA).

A CT scan, performed 4 months later, showed complete resolution of the portal vein and pulmonary arterial thrombosis with partial canalization of the mesenteric veins.

### CASE 3

R.B., a 60-year-old male presented with acute abdominal pain that started 8 hours earlier. An abdominal X-ray documented only coprostasis, while ultrasound was suggestive of portal vein thrombosis. Serological markers showed neutrophilic leukocytosis, a mild increase in CRP and normal serum lactates.

A CT scan showed thrombosis of the superior mesenteric and portal veins, mesenteric and small bowel wall edema and free abdominal fluid.

An urgent laparotomy was performed removing an 85 cm segment of ischemic small bowel with primary anastomosis.

Screening for thrombophilic genetic mutations revealed the G20210A prothrombin variant. Anticoagulant therapy with LMWH was administered initially, and after discharge the patient underwent imbrication with VKA.

### DISCUSSION

Acute mesenteric venous thrombosis is a rare but potentially fatal cause of abdominal pain, accounting for 6–9% of all mesenteric ischemia cases [1].

The clinical presentation, vague and misleading, may involve abdominal pain, nausea, vomiting, hematemesis, constipation or diarrhea and melena. If not promptly diagnosed, it can lead to peritonitis with severe abdominal pain, intestinal infarction and death.

Mesenteric vein thrombosis (MVT) is defined as acute if abdominal pain has been present for less than 4 weeks.

The diagnosis is essentially radiological based on CT scanning with intravenous iodine contrast.

Radiological signs of MVT include defective canalization of the superior or inferior mesenteric vein, which can extend to the splenic and portal vein and occasionally reach the intrahepatic portal bifurcation. Signs of intestinal ischemia (Fig. 1) include mesenteric edema, small bowel edema and wall thickening, dilated bowel loops, intestinal pneumatosis and ascites [2].

The presence of radiological evidence of intestinal ischemia associated with severe abdominal pain or peritonitis makes urgent surgical exploration mandatory.

In a Swedish retrospective study on a series of 102 patients affected by MVT, mesenteric edema, small bowel wall edema, small bowel dilation and ascites were associated with higher bowel resection rate. Among these factors, small bowel edema represented an independent risk factor for intestinal resection [3]. Kim et al. identified the extent of thrombosis as another risk factor for bowel resection.



Figure 1: Abdominal CT scan with evidence of portal [1], splenic [2] and superior mesenteric vein [3] thrombosis, along with free fluid in the right iliac fossa [4] and small bowel thickening [5], suggesting of intestinal infarction.

If the patient is weakly symptomatic, vital parameters are stable and radiological signs of intestinal ischemia are not so clear, but mesenteric vein occlusion is documented, treatment may be exclusively medical with full-dose anticoagulants.

The role of hematological parameters in the diagnosis of acute mesenteric ischemia is still under debate. Leukocyte count and neutrophil-to-lymphocyte ratio, mean platelet volume, lactate dehydrogenase, D-dimer, serum lactate, red cell distribution width and transaminases can all have a role in assessing the severity of mesenteric ischemia rather than in its diagnosis [4].

Patients with MVT and no evident acute thrombophilic risk factors should be investigated for inherited or acquired thrombophilic disorders. Inherited thrombophilic disorders include factor V Leiden mutation, prothrombin gene mutation, and protein C, protein S and antithrombin deficiencies. Acquired thrombophilic factors include lupus anticoagulant and cardiolipin antibodies.

MVT may be the first sign of an undiagnosed cancer, such as myeloproliferative neoplasms, which can be better identified by testing for Jak2 V617F mutations), or intra-abdominal solid tumors. In a Swedish retrospective cohort of 120 patients with MVT, 23 (19.2%) had a solid cancer [5].

The duration of anticoagulant therapy depends on any identification of pro-coagulant disorders and on clinical and radiological response to anticoagulant treatment. Pooling of the data from retrospective surveys by Condat et al. showed that 6 months of therapy for patients with MVT is associated with complete recanalization in 50% of cases, partial recanalization in 40% and no recanalization in 10%.

In patients with known transient risk factors, discontinuation of anticoagulant therapy can be proposed after complete splanchnic vein recanalization. Lifelong anticoagulation is recommended; however, for patients with proven, inherited or acquired thrombophilia, as well as for patients with recurrent vein thrombosis or if recurrent thrombosis could have severe clinical consequences [6].

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