Mesenteric Venous Thrombosis

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Abstract

The prevalence of mesenteric venous thrombosis has increased over the past 2 decades with the routine use of contrast-enhanced computed tomography (CT) in patients presenting with abdominal pain and those with portal hypertension. Concurrent with increasing recognition, routine and frequent use of anticoagulation has reduced the need for surgical intervention and improved outcome in these patients. 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. The presence of collateral circulation and cavernoma around a chronically thrombosed vein differentiates chronic from acute disease. The superior mesenteric vein is often involved, whereas involvement of the inferior mesenteric vein is rare. Associated portal venous thrombosis can be seen if the disease originates in the major veins instead of the small vena rectae. Thrombophilia and local abdominal inflammatory conditions are common causes. Management is aimed at preventing bowel infarction and recurrent thrombosis. Anticoagulation, the mainstay of management, has also been safely used in patients with cirrhosis and portal hypertension. This review discusses the pathogenesis of thrombosis of mesenteric veins, the diagnosis and differentiation from arterial ischemia, the emergence of the JAK2 (Janus kinase 2) sequence variation as a marker of thrombophilia and myelodysplastic neoplasms, and new anticoagulants. Algorithms for the management of acute and chronic mesenteric venous thrombosis are provided to help readers understand and remember the approach to the management of acute and chronic mesenteric venous thrombosis.

Mesenteric venous thrombosis (MVT) may be an incidental finding on abdominal imaging or a cause of abdominal pain. Mesenteric venous thrombosis was originally described by Warren and Eberhard as a distinct entity that is separate from mesenteric arterial thrombosis. Isolated thrombosis of the portal vein is also a distinct entity but can occur in association with MVT. Prothrombotic states or thrombophilia and local intra-abdominal infections are major causes of MVT. Abdominal pain is the most common symptom, especially with acute thrombosis, whereas chronic MVT manifests as portal hypertension and esophageal varices. Over the past approximately 2 decades, the increasing use of computed tomography (CT) for the investigation of abdominal pain and anticoagulation as the first approach to treatment of acute MVT have improved outcome in these patients. Surgery and bowel resection may occasionally be needed for patients with bowel infarction, perforation, and peritonitis. The management of patients with chronic MVT is aimed at reducing complications of portal hypertension.

NORMAL MESENTERIC CIRCULATION

The venous drainage of the intestine follows the same pattern as the arterial circulation (Figure 1). The superior mesenteric vein (SMV) drains the entire bowel from the second portion of the duodenum to approximately the right two-thirds of the transverse colon. The SMV joins the splenic vein posterior to the neck of the pancreas to form the portal vein. The left gastric vein draining the lower part of the esophagus and upper half of the lesser curve of the stomach enters at the point of formation of the portal vein. Short gastric veins draining the fundus of the stomach enter into the splenic vein. The inferior mesenteric vein (IMV) drains the left colon and enters into the splenic vein.

ACUTE MVT

Incidence and Prevalence

Mesenteric venous thrombosis is a rare condition accounting for 1 in 5000 to 15,000 inpatient admissions, 1 in 1000 emergency department admissions, and 6% to 9% of all cases of acute mesenteric ischemia. With the widespread use of abdominal imaging, recognition of MVT
has increased. The recognition of MVT in Sweden has increased from 2.0 per 100,000 patient-years before the year 2000 to 2.7 per 100,000 patient-years after 2000.5

**Pathogenesis**
Prothrombotic states, local vessel wall injury, and venous stasis contribute to MVT (Table 1). Thrombosis may originate in the vena rectae or in the major veins. The latter is usually associated with portal venous thrombosis, whereas the former manifests as isolated thrombosis of mesenteric veins.6 The SMV is more commonly involved, with IMV thrombosis, for unclear reasons, representing only 0% to 11% of cases of MVT.7-9

When the underlying etiology cannot be identified, the term primary or idiopathic MVT is used. The proportion of patients with primary MVT varies from 0% to 49%, with a decrease in frequency with more extensive investigation (Table 1).10,11 In many patients, more than one factor may account for the thrombosis.12 Prothrombotic states are the most common cause for patients presenting with isolated MVT. In contrast, local causes may be more frequently associated with combined mesenteric and portal venous thrombosis.6 Myeloproliferative neoplasms and malignancy are the most commonly diagnosed thrombophilic conditions. Abdominal operations, especially splenectomy, may be a cause of MVT.13-15 The prevalence of mesenteric and portal venous thrombosis is higher in cirrhotic patients, probably due to turbulent venous flow and an increased tendency for thrombosis,16 and is reported in approximately 15% of patients awaiting liver transplant.16

Mesenteric venous thrombosis impairs venous return from the bowel, resulting in venous engorgement and ischemia. With rapid and complete occlusion of mesenteric veins, there may be insufficient time for development of a collateral circulation, and transmural bowel infarction may occur. The transition from normal to ischemic bowel is gradual, unlike in arterial ischemia in which this transition is abrupt. Arterial spasm secondary to venous engorgement may occur, with resulting irreversible bowel ischemia despite treatment of the venous thrombus. Thrombosis isolated to mesenteric veins results in earlier and more frequent development of infarction as compared to mesenteric combined with portal venous thrombosis. With transmural infarction, there is loss of integrity of the bowel mucosa, allowing bacterial translocation and potential for occurrence of the fatal consequences of lactic acidosis, sepsis, multiorgan failure, and death.

**Clinical Presentation**
The presentation of MVT is either acute with sudden onset of symptoms, subacute with presentation over days to weeks, or chronic. Acute MVT presents with abdominal pain

![Figure 1. Normal mesenteric circulation.](image-url)
and chronic MVT with complications of portal hypertension such as variceal hemorrhage. Acute disease is more likely to result in bowel infarction compared with subacute MVT.

The mean age at presentation is 40 to 60 years, and the disease is most common in males. Abdominal pain is the dominant symptom and is usually severe, located in the mid abdomen, and out of proportion to physical signs. Nausea, vomiting, diarrhea, or gastrointestinal bleeding may also occur. Other reported symptoms are constipation, anorexia, and fever. Fever at the onset of symptoms suggests pylephlebitis or an infected portal venous thrombosis seen with intestinal infections, usually acute diverticulitis or appendicitis.

About 75% of patients are symptomatic for more than 48 hours before presentation, with the mean duration of symptoms reported to vary from 6 to 14 days. Abdominal signs such as abdominal tenderness, and abdominal distention, and ascites may occur with increasing ischemia. Patients may be dehydrated from volume loss or third-space compartmentalization of fluid. In severe cases, peritoneal signs such as rebound tenderness, guarding, and/or rigidity may be noted because of transmural infarction and bowel gangrene. About 6% to 29% of patients, especially those with severe disease, may be hemodynamically unstable. Peritoneal signs, hemodynamic instability, and fever suggest severe disease with bowel infarction and poor outcome.

Diagnosis

Differentiation between ischemia due to MVT and that due to mesenteric arterial thrombosis is usually possible. The former usually occurs in the setting of thrombophilia, whereas cardiac causes and atrial fibrillation are commonly associated with arterial ischemia. Other causes of acute abdomen such as acute pancreatitis, intestinal obstruction, viscous perforation, acute cholecystitis, and acute appendicitis can be ruled out by clinical presentation and compatible abdominal imaging. A personal or family history of deep venous thrombosis, present in about 17% to 44% of cases, heightens suspicion for a diagnosis of MVT. Similarly, development of ascites in a patient with acute abdominal pain heightens suspicion for MVT.

Routine laboratory evaluation is usually not helpful for the diagnosis of MVT. Patients with severe disease and dehydration may show evidence of hemoconcentration. Nonspecific leukocytosis may be present. Patients with small-vessel disease and isolated MVT may have higher platelet counts compared to patients with combined portal and mesenteric vein involvement. This may be due to associated thrombophilic conditions in those with small-vessel disease and isolated MVT. Amylase levels may be elevated with bowel ischemia, but levels higher than 1000 U/L suggest acute pancreatitis. Liver

<table>
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<tr>
<th>Causes of Mesenteric Venous Thrombosis</th>
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<tr>
<td><strong>Thrombophilia</strong></td>
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<td>Protein C or S deficiency</td>
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<td>Factor V Leiden deficiency</td>
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<td>Antithrombin deficiency</td>
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<td>Prothrombin gene sequence variation (G20210A)</td>
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<td>Methylene tetrahydrofolate reductase gene sequence variation</td>
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<td>Acquired</td>
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<td>Hematologic conditions</td>
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<td>Myelofibrosis</td>
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<td>Thrombocytopenia</td>
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<td>JAK2 gene sequence variation</td>
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<td>Antiphospholipid antibodies</td>
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<td>Oral contraceptive pills</td>
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<td>Nephrotic syndrome</td>
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<td>Intra-abdominal surgery</td>
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<td><strong>Stasis</strong></td>
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<td>Congestive splenomegaly</td>
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<td>Cirrhosis</td>
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<td>Congestive heart failure</td>
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<td><strong>Idiopathic</strong></td>
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*Either one of these is included in the diagnosis of myeloproliferative neoplasms. Sequence variation analysis for the Janus kinase gene (JAK2V617F) is a diagnostic criterion and identifies latent cases of myeloproliferative neoplasms.

*Intra-abdominal cancers in most patients, with pancreatic cancer being the most common.

*The most common surgery is splenectomy.
enzymes may be elevated to a modest degree, especially in patients with portal vein involvement. Hypoxemia and lactic acidosis are late findings and predict poor outcome. Blood cultures should be obtained in patients with high fever, peritoneal signs, hemodynamic instability, and perforation. Paracentesis and cultures may be helpful because hemorrhagic ascites may develop in some patients.

**Abdominal Imaging.** With routine use of abdominal imaging, the diagnosis of MVT is usually made noninvasively. Plain abdominal radiography shows abnormalities in 50% to 75% of cases, with mostly nonspecific findings such as dilated bowel loops, ileus, and thumbprinting from mucosal edema. Specific findings of bowel ischemia are seen in fewer than 5% of cases. The presence of gas within the portal vein or intestinal wall and free air within the abdomen suggest bowel gangrene and bowel perforation, respectively. Computed tomography with contrast medium is the diagnostic modality of choice. The diagnostic finding for MVT is the presence of thrombus within the vein seen as focal translucency (Figure 2). Other findings are expansion of the vein with a sharply defined wall. The accuracy of CT is about 90% for the diagnosis of MVT. Findings of intestinal ischemia such as bowel wall thickening of greater than 3 mm, thickened mesentery, indistinct bowel margins, and ascites may also be noted. Homogeneous bowel enhancement along with bowel wall thickening of 10 mm or more has about 90% accuracy for identification of transmural infarction.

Abdominal Doppler ultrasonography can detect thrombosis of larger veins but is limited by its inability to visualize small vena rectae. Magnetic resonance angiography is an excellent technique, but experience with its use is limited. Further, motion artifacts may affect its accuracy. Nuclear scintigraphy is relatively easily performed, but the technique is limited by a lower accuracy of 75% and the lack of wide availability. Mesenteric angiography is seldom required for the diagnosis of MVT.

**Identifying the Cause of Mesenteric Thrombosis.** Local causes such as an abdominal malignant neoplasm, surgery, or inflammatory conditions are usually obvious on history, physical examination, and abdominal imaging. Before starting anticoagulants, blood should be withdrawn for a thrombophilia screen, looking especially for a myeloproliferative neoplasm (MPN)—namely, polycythemia vera, essential thrombocythemia (ET), and myelofibrosis. The diagnosis of MPN may be difficult in the presence of portal hypertension and splenomegaly because hemodilution and hypersplenism may result in lower blood counts.

The JAK2V617F sequence variation with gain of function resulting in growth factor–independent proliferation of blood cell lines has been reported in 5% to 46% of cases without overt MPN. Further, the presence of the JAK2 sequence variation can differentiate reactive thrombocytosis from ET and primary from secondary polycythemia with reported positivity of the JAK2 sequence variation in nearly all cases of polycythemia vera and in 50% of cases of ET and myelofibrosis. Hence, detection of the JAK2 sequence variation has replaced bone marrow examination as the first test to screen for MPNs (Table 2).

**Treatment**

The goals of treatment of MVT are to prevent extension of the thrombus and intestinal infarction in the short term and to prevent recurrence of thrombosis in the long term.
Medical Management. Medical management includes general measures and anticoagulation. Pain control, bowel rest, and replacement of fluids and electrolytes should be initiated for patients with an acute presentation. Red blood cell transfusion may be needed for gastrointestinal bleeding and nasogastric aspiration for patients with abdominal distention, ileus, and severe intractable nausea or vomiting. Broad-spectrum antibiotics are used for patients with pylephlebitis or septic thrombophlebitis of the mesenteric vein and in patients with superimposed sepsis due to bacterial translocation from bowel infarction.7,33

Anticoagulation is the initial approach to management, and heparin should be administered as soon as the diagnosis is made (Figure 3).2,3,7,13,17 The degree of gastrointestinal bleeding rarely is a contraindication to initiation of anticoagulation. Unfractionated heparin requires monitoring with activated partial thromboplastin time; monitoring is not required with low-molecular-weight heparin. The advantages of using unfractionated heparin are safety in the presence of renal failure and in patients with invasive procedures are planned. In contrast, low-molecular-weight heparin is cleared through the kidneys with a half-life of 6 to 12 hours and cannot be used in the presence of renal failure. Once improvement is noted and invasive procedures are no longer likely, warfarin therapy is initiated. When the international normalized ratio reaches the target range of 2 to 3, heparin is discontinued and warfarin alone is continued. The duration of anticoagulation is about 6 months for patients with known reversible conditions but lifelong in patients with prothrombotic states and without any identifiable etiology.

Warfarin has the disadvantages of required regular monitoring of the international normalized ratio, drug-drug interactions, and interaction with food due to its metabolism via the cytochrome P450 enzyme pathway. In this respect, newer oral anticoagulants such as direct thrombin inhibitors (ximelagatran, dabigatran, and AZD0837) and direct inhibitors of factor Xa (rivaroxaban, apixaban, edoxaban, otamixaban, LY517717, TAK-442, and fondaparinux) have fewer problems. However, because the dose is fixed on the basis of the patient’s body weight, lower doses of these newer agents may be required in the presence of extensive liver and renal disease. Further, in case of overdosing, the action cannot be reversed because of the unavailability of an antidote.34

Anticoagulation helps recanalize the thrombosed vein.35 Among patients awaiting liver transplant, recanalization of the portal vein occurs in 50% of those receiving anticoagulation but in none who are not given anticoagulants. Associated splenic vein occlusion, ascites, and the extent of thrombus predict failure to recanalize the vein.36 In a retrospective study, 13 patients seen before 1995 who did not receive...
Anticoagulants were compared with 28 patients seen after 1995 who were given anticoagulants. The patients in the later group had a shorter mean duration of hospital stay (13 vs 26 days), reduced hospital mortality (11% vs 39%), less need for surgery (33% vs 85%), and lower risk of short bowel syndrome.13

**Surgical Management.** Patients with impending transmural infarction and peritoneal signs are treated with surgery (Figure 3). Bowel viability at surgery is determined by visual inspection, Doppler ultrasonography, or fluorescein infusion, with fluorescein infusion being the most accurate means for detecting bowel viability.19 Resection and anastomosis is the standard procedure, with the goal of conserving as much bowel as possible. Among various series, the average length of bowel resected is 50 to 60 cm.5,15 Surgery and/or resection is often needed in patients with disease of small veins and thrombosis limited to the mesenteric vein as compared with combined portal and mesenteric veins.6 After initial resection, second-look operations may be performed within 24 to 48 hours to determine the need for additional resection.2

**Interventional Radiologic Options.** Interventional radiologic options are considered for patients at risk for bowel infarction but without peritonitis. These are typically patients who have worsening abdominal pain despite 48 to 72 hours of anticoagulation therapy. If the mesenteric vein is patent, vasodilator therapy with papaverine infusion with or without thrombolytics (tissue plasminogen activator or streptokinase) may be considered (Figure 3). The mesenteric vein can be approached by the transhepatic or transjugular route, the latter preferred for patients with ascites.37 These procedures are not a substitute for anticoagulation, which should continue after a successful procedure. Data on the use of thrombolytic therapy are limited to case reports, and small case series have suggested a high rate of bleeding with thrombolysis. In the largest retrospective study to date that included 20 patients with MVT treated with thrombolysis, 75% had partial or complete clot resolution, 85% had resolution of symptoms, and 60% experienced a major complication; bleeding was the most common complication, and death from bleeding occurred in one patient.37

A transjugular intrahepatic portosystemic shunt (TIPS) may be used in patients with acute MVT with or without portal venous thrombosis in whom anticoagulation is unsuccessful and whose clinical condition worsens. A TIPS is placed with the goal of recanalizing the occluded portal vein segment and maintaining blood flow across the occluded segment. To achieve this, the shunt may need to be extended into the main portal vein. Contraindications to TIPS placement and complications after the procedure are similar to those that occur in other patients with cirrhosis.38 In one study, the use of TIPS resulted in successful recanalization with immediate symptomatic improvement in 20 of 24 patients (83%).39

**Outcome**

With the widespread availability of imaging techniques such as CT and the change to early use of anticoagulation, mortality rates in patients with MVT have decreased to 0% to 23% among various series.5,14,17,40 The most common cause of death is sepsis with multiorgan failure. Other causes include recurrent thrombosis, short bowel syndrome, and pulmonary embolism. The most important factor predicting the short-term (within 30 days) outcome is the presence of bowel infarction. Other factors that have been reported in various series are age, the lack of use of anticoagulation, treatment on a nonsurgical ward, and the presence of colonic ischemia.5,9,11,17 The long-term outcome of patients depends on the following factors.

**Underlying Prothrombotic State.** Patients with MPNs have the potential for clonal evolution to acute leukemia and myelodysplasia. This risk can be reduced considerably with the use of cytoreductive therapy and hydroxyurea.41 These patients also have poor quality of life including fatigue, bone pain, pruritus, and weight loss.42 Bleeding, usually mild, can occur because of thrombocytopenia or platelet dysfunction.

**Short Bowel Syndrome.** Short bowel syndrome occurs in patients with extensive resection and should be avoided by preserving as much of the bowel as possible at laparotomy.

**Recurrent Thrombosis.** Recurrent thrombosis at any site has been reported in 3% to 40% of patients and within the mesenteric venous
circulation in 0% to 25%. Most recurrences occur within the first 30 days after presentation. Recurrence rates may be as low as 0% to 3% in patients who continue to receive anticoagulation treatment. Underlying thrombophilia and oral contraceptive therapy predict recurrent disease.

Bleeding From Anticoagulation. Bleeding rates among patients treated with anticoagulation is low at less than 10%, with gastrointestinal bleeding being the most common. Varices predict bleeding and if present should be managed as in patients with cirrhosis. Such management includes the use of pharmacological therapy with β-blockers or endoscopic ligation of varices. Pharmacological therapy is preferred because variceal ligation may be associated with ligation ulcers and risk of bleeding in patients who are receiving anticoagulants. Unless associated with intracranial bleeding, hemorrhage is rarely the cause of death in patients treated with anticoagulation.

CHRONIC MVT
Chronic MVT accounts for about 20% to 40% of all cases of MVT, may be detected incidentally on abdominal imaging, and is differentiated from acute MVT by the presence of an extensive collateral circulation. The diagnosis of chronic MVT is usually made on CT (Figure 4). Patients with portal venous thrombosis present with features of portal hypertension such as splenomegaly, variceal hemorrhage, and thrombocytopenia. If the thrombosis is distal to the junction of the left gastric vein and portal vein, both esophageal and gastric varices occur. However, if the thrombosis is proximal to this junction, gastric varices are predominant. Extensive thrombosis involving the portal vein, SMV, splenic vein, and IMV results in extensive bowel edema, with sequelae being persistent abdominal pain, small-bowel strictures, and inability to meet nutritional needs via the gastrointestinal tract. Some patients may require long-term home parenteral nutrition. Chronic MVT may also be associated with edema and extensive collateral vessels around the bile ducts, giving rise to portal hypertensive cholangiopathy mimicking primary sclerosing cholangitis.
on cholangiography (Figure 5). This entity presents with biochemical cholestasis, biliary strictures, biliary stones, and cholangitis.

Diagnosis
As stated previously, chronic MVT is usually diagnosed on CT. However, magnetic resonance cholangiopancreatography is preferred whenever this diagnosis is suspected (Figure 5).43,44 Endoscopic retrograde cholangiopancreatography may also be used but may be complicated by hemorrhage from puncture of the biliary varices by the biliary catheter used to inject radiologic contrast medium for delineation of the bile ducts.

Management
**Esophageal and/or Gastric Varices.** Esophageal and/or gastric varices are managed as in patients with cirrhosis. Management includes prevention of a first variceal bleed for patients with varices but no history of bleeding, control of variceal hemorrhage, and prevention of recurrent bleeding (Figure 6). If feasible, TIPS may be recommended for patients who continue to experience bleeding despite combined endoscopic and pharmacological therapy.43 Patients without cirrhosis, especially in the pediatric age group, are managed by creation of a surgical shunt between the left portal vein and SMV (Rex bypass).43 Patients with isolated gastric varices due to splenic venous thrombosis are amenable to cure by splenectomy.43

**Portal Hypertensive Cholangiopathy.** Portal hypertensive cholangiopathy requires treatment if patients are symptomatic from cholangitis or biliary obstruction. Stones within the common duct are removed at endoscopic retrograde cholangiopancreatography. Caution is required during the procedure because the presence of portal hypertension makes bleeding more likely. Strictures in the bile ducts are treated with endoscopic stenting and in selected patients by portosystemic shunt surgery. Rarely, hepaticojejunostomy may be required.45

**Anticoagulation.** Anticoagulation should be considered for patients with chronic portal venous thrombosis, especially those with prothrombotic states. However, data are limited on the use of anticoagulants in patients with chronic MVT. In one retrospective study, 18 of 60 patients with chronic portal venous thrombosis underwent long-term anticoagulation. Five patients who had bleeding before anticoagulation had eradication of varices before starting anticoagulation. Of the other patients, 4

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**FIGURE 6.** Algorithm for management of chronic mesenteric venous thrombosis. TIPS = transjugular intrahepatic portosystemic shunt.
had bleeding on follow-up; good control of bleeding was achieved in 3, and the fourth patient died. 46 These data suggest that varices should be adequately treated and obliterated before initiating anticoagulation.

For patients with chronic MVT, 5-year survival rates are 78% to 83%, with outcome usually related to the severity and nature of the underlying disorder. 2,46

CONCLUSION
Acute MVT is a rare but important cause of intestinal ischemia. Early diagnosis requires a high index of suspicion. Anticoagulation should be started as soon as the diagnosis is confirmed and is associated with improved outcome. Patients with underlying thrombophilia should undergo lifelong anticoagulation. Surgery is reserved for patients who have bowel infarction with peritonitis or perforation. The goal of surgery should be to preserve as much bowel as possible in order to avoid short bowel syndrome. Patients with chronic MVT may be asymptomatic or present with complications of portal hypertension. In selected patients, anticoagulation is safe and effective provided therapy is initiated to prevent variceal bleeding. The long-term outcome of patients depends both on the underlying cause of the thrombosis and the efficacy of long-term anticoagulation.

Abbreviations and Acronyms: CT = computed tomography; ET = essential thrombocytemia; IMV = inferior mesenteric vein; MPN = myeloproliferative neoplasm; MVT = mesenteric venous thrombosis; SMV = superior mesenteric vein; TIPS = transjugular intrahepatic portosystemic shunt.

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REFERENCES


