Mesenteric venous thrombosis in adults

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INTRODUCTION

Acute mesenteric ischemia refers to the sudden onset of intestinal hypoperfusion, one cause of which can be mesenteric venous occlusion. Mesenteric venous thrombosis can present acutely or in a subacute or chronic manner. At one time, acute mesenteric venous thrombosis was thought to be the principal cause of acute mesenteric ischemia; however, with increasing recognition of and differentiation from the occlusive and nonocclusive forms of acute arterial mesenteric ischemia, the proportion of cases attributed to mesenteric venous thrombosis has decreased to approximately 10 percent of all cases of acute mesenteric ischemia [1].

Mesenteric venous thrombosis will be reviewed here. Acute and chronic mesenteric arterial occlusion affecting the small intestine, nonocclusive mesenteric ischemia, and colonic ischemia are discussed separately. (See "Overview of intestinal ischemia in adults" and "Chronic mesenteric ischemia" and "Colonic ischemia" and "Nonocclusive mesenteric ischemia".)

MESENTERIC VENOUS ANATOMY AND PATHOPHYSIOLOGY

The venous drainage parallels the arterial circulation and drains into the portal venous system (figure 1 and figure 2). The anatomy of the intestinal circulation, normal physiology of the intestine, and response to ischemia are discussed in detail elsewhere. (See "Overview of intestinal ischemia in adults", section on 'Collateral circulation' and "Overview of intestinal

ischemia in adults", section on 'Intestinal vascular anatomy' and "Overview of intestinal ischemia in adults", section on 'Physiology and mechanisms of ischemia'.)

Pathophysiology — Venous thrombosis is predominantly a result of stagnation of blood flow, vascular injury, and hypercoagulability (ie, Virchow's triad). Local factors (eg, splenectomy, pancreatitis) appear to be associated with initial thrombus formation in the large veins, whereas systemic hypercoagulable states (eg, protein C deficiency) lead to thrombosis initiated in the intramural venules, vasa recta, and venous arcades [2]. Mesenteric vein thrombosis almost always involves the distal small intestine (superior mesenteric venous drainage) and rarely involves the colon (inferior mesenteric venous drainage) [3]. The anatomic site of involvement in acute mesenteric venous thrombosis is most often ileum (64 to 83 percent) or jejunum (50 to 81 percent), followed by colon (14 percent) and duodenum (4 to 8 percent) [4,5]. The inferior mesenteric venous distribution is less commonly involved for reasons that are poorly understood but possibly related to collateral flow through the internal iliac system, rectal venous plexus, or systemic circulation via the left renal vein, splenic vein, and hemiazygous veins, though this is unproven [6-9].

Acute thrombotic occlusion of one or more mesenteric veins reduces perfusion pressure due to increased resistance in the mesenteric venous bed. As flow stagnates, increased venous pressure leads to efflux of fluid into the tissues, causing profound bowel wall edema, which can lead to submucosal hemorrhage. If the venous arcades and vasa recta are involved and venous return from the bowel wall is completely occluded, bowel infarction will occur [6]. However, not all cases of mesenteric venous thrombosis are associated with intestinal infarction. In animal models, gradual occlusion of the superior mesenteric vein is associated with the development of collateral venous drainage without ischemic damage [10]. Chronic mesenteric venous thrombosis often features dilated venous collaterals, which can bleed, due to elevated venous pressures [11]. Many patients with chronic mesenteric venous thrombosis also exhibit portal vein thrombosis. (See "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management".)

Sequestration of fluid into the bowel lumen combined with massive bowel wall edema results in relative hypovolemia and systemic hypotension. As a consequence, arterial flow is also reduced, which exacerbates ischemia [12]. Arterial vasospasm is another factor thought to be important in the pathogenesis of ischemia and infarction associated with mesenteric venous thrombosis. Under experimental conditions, arterial spasm can occur in the presence of venous occlusion [10,13-15]. Arterial spasm can persist and may be of sufficient degree to cause intestinal infarction or secondary arterial thrombosis, even though the veins have returned to normal caliber after relief of temporary venous obstruction.

INCIDENCE AND RISK FACTORS

In a time cohort study from Sweden, the incidence of mesenteric venous thrombosis increased from 2.0 per 100,000 patient-years between 1970 and 1982 to 2.7 per 100,000 patient-years between 2000 and 2006, which was attributed to increased diagnosis using computed tomography, which was more prevalent in the latter cohort, compared with a surgery or autopsy diagnosis in the earlier cohort [16,17].

The proportion of cases of **acute** mesenteric ischemia attributed to mesenteric venous thrombosis has varied widely in the literature but has decreased over time, likely related to better differentiation from acute arterial occlusion and nonocclusive mesenteric ischemia. In a systematic review of cases from 1966 to 2002 that included 3692 patients, mesenteric venous thrombosis was responsible for 3 percent of cases of acute mesenteric thrombosis [18]. In other reviews, acute mesenteric venous thrombosis accounts for between 2 and 10 percent of cases [4,5,19].

Estimates of the incidence of **chronic** mesenteric venous thrombosis are reported less frequently than those of acute mesenteric venous thrombosis, most likely because a large proportion of these patients do not complain of abdominal pain. In two reviews that included patients with either acute or chronic mesenteric venous thrombosis, chronic mesenteric venous thrombosis accounted for 24 to 40 percent of the total number of cases [20,21].

Risk factors — Mesenteric venous thrombosis is a multifactorial disorder predisposed by certain risk factors, which can be broadly divided into acquired and inherited conditions [22].

- Local intra-abdominal inflammatory processes (eg, pancreatitis, inflammatory bowel disease) or trauma (eg, splenectomy) increase the risk for acquired mesenteric venous thrombosis and tend to affect the larger veins. Mesenteric venous thrombosis has been reported as a postoperative complication of laparoscopic sleeve gastrectomy for obesity. In two retrospective surveys of more than 2900 patients, the incidence of this complication was approximately 0.7 percent [23,24].

- Heritable and acquired thrombophilias (eg, prothrombin G20210A mutation, MPD) and hypercoagulable states related to systemic disorders (eg, nephrotic syndrome, malignancy) are more likely to affect the smaller veins. (See 'Pathophysiology' above.)

  Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (ie, coronavirus disease 2019 [COVID-19]) has been associated with acute mesenteric thrombosis,

including documented cases of mesenteric venous thrombosis related to hypercoagulability [25-28]. (See "COVID-19: Hypercoagulability".)

Common risk factors for mesenteric venous thrombosis are listed below [4,5,9,19,20,29-48]. A comprehensive discussion of the causes of venous thrombosis can be found elsewhere. (See "Overview of the causes of venous thrombosis".)

- Abdominal mass (eg, tumor, pseudocyst) leading to venous compression
- Abdominal inflammatory processes (eg, acute pancreatitis, diverticulitis)
- Myeloproliferative disorders (eg, *JAK-2* V617F mutation)
- Portal hypertension and cirrhosis (increased portal venous pressure)
- Personal or family history of venous thromboembolism
- Acquired thrombophilia (eg, malignancy, oral contraceptives)
- Inflammatory bowel disease
- Mesenteric adenopathy/viral infection (eg, influenza)
- Inherited thrombophilia – Factor V Leiden mutation, prothrombin G20210A mutation, protein S deficiency, protein C deficiency, antithrombin III deficiency, activated protein C resistance, and antiphospholipid syndrome
- Endoscopic sclerotherapy
- Obesity surgery

At least one predisposing risk factor is recognized in most patients who present with mesenteric venous thrombosis [30,49-54]. A previous history of deep vein thrombosis is reported in approximately 20 to 40 percent of patients with mesenteric venous thrombosis. It is unknown whether the superior mesenteric vein thrombosis has any unique associations with a specific heritable thrombophilia, compared with portal or splenic vein thrombosis, or if acute mesenteric venous thrombosis has risk factors separate and distinct from subacute or chronic mesenteric venous thrombosis. Patients without identifiable risk factors are regarded as having idiopathic mesenteric venous thrombosis. The frequency of idiopathic cases in various series ranges from 21 to 49 percent. However, on closer examination, up to one third of these patients have identifiable risk factors and, in modern series, laboratory evidence of a coexistent heritable thrombophilia [32,55].

Malignancy is reported in 4 to 16 percent of patients with acute mesenteric venous thrombosis [4,5,9,19,20,40]. No studies have examined differences in the incidence of mesenteric venous thrombosis for the various types of malignancy (eg, solid intra-abdominal, solid extra-abdominal, blood malignancy). Myeloproliferative disorders, which are associated with an increased risk for arterial or venous thrombosis, are identified in 8 to 18 percent of patients with acute or chronic mesenteric venous thrombosis [32]. (See "Risk and prevention of venous thromboembolism in adults with cancer".)

CLINICAL PRESENTATIONS

The clinical features of mesenteric venous thrombosis are determined by the location and timing of thrombus formation within the splanchnic vasculature. Mesenteric venous thrombosis can present acutely, subacutely (which may be related to a delay in seeking medical attention or to delayed diagnosis), or more chronically [11]. There appears to be considerable overlap between the various forms. Patients whose disease process starts in the small veins and in whom the most peripheral venous drainage is affected from the outset appear to be at greatest risk of intestinal infarction [6,20,56]. (See 'Pathophysiology' above.)

The average age at presentation of mesenteric venous thrombosis in various reports is between 45 and 60 years, with a slight male to female predominance [4,5,11,16,19,20,40,57-59]. The age at presentation varies with underlying etiology.

Acute — Acute mesenteric venous thrombosis, like other forms of acute mesenteric ischemia, is suggested by the onset of colicky, periumbilical abdominal pain, present for at least a few hours and out of proportion to the abdominal findings on physical examination, at least initially. However, the onset is often less sudden than other forms of mesenteric ischemia, and the pain is usually more dull. Over 75 percent of patients report at least two days of pain before seeking medical attention; the duration of symptoms ranges from 5 to 14 days. Although patients with objective signs of acute mesenteric venous thrombosis occasionally report pain that has persisted for more than a month, some authors suggest the definition of acute mesenteric venous thrombosis should be limited to symptom duration less than four weeks [4,57]. Approximately one-half of patients have nausea and vomiting [4,5,16,19,20,40,57-59].

The abdominal examination may reveal abdominal distension, and occult blood may be found in the stool. Signs of peritoneal inflammation, such as rebound tenderness and guarding, are typically absent. However, if bowel distention progresses, the bowel can become ischemic. Bowel sounds will become absent, and peritoneal signs will develop. The incidence of peritonitis

reported is variable, but complications leading to peritonitis are uncommon in those who are appropriately anticoagulated. (See 'Anticoagulation' below and 'Abdominal exploration' below.)

Plain abdominal radiographs are relatively nonspecific and may be completely normal in more than 25 percent of patients [12]. Findings suggestive of acute mesenteric ischemia include the presence of an ileus with distended loops of bowel and bowel wall thickening, the latter of which is particularly prominent in patients with acute mesenteric venous thrombosis, and/or pneumatosis intestinalis (image 1). Pneumatosis may be identified in patients with advanced ischemia.

Subacute — The presentation of mesenteric venous thrombosis can be more insidious, with subacute symptoms that can be present for days to weeks before diagnosis [12,60]. Nonspecific abdominal pain may be the only feature.

Whether this presentation represents a distinct group or a subset of acute mesenteric venous thrombosis related to delays in seeking medical attention or delay in diagnosis is not entirely clear.

The subacute form of mesenteric venous thrombosis occurs in the circumstance of venous occlusion sufficient to produce ischemia but adequate compensation through collateral vessels to allow recovery [11]. Nevertheless, some patients with subacute mesenteric venous thrombosis can progress to acute intestinal infarction requiring surgical intervention. Others may develop chronic symptoms. (See 'Abdominal exploration' below.)

Chronic — Patients with chronic mesenteric venous thrombosis are frequently asymptomatic, and the diagnosis has been established incidentally by imaging studies performed for unrelated reasons. In one study of 121 patients, a similar number of patients presented with asymptomatic chronic mesenteric venous thrombosis as those who presented acutely [21]. Symptomatic patients with chronic mesenteric venous thrombosis typically present with complications of portal hypertension (variceal bleeding or ascites), in most cases related to concomitant portal or splenic vein thrombosis. A minority of patients have intermittent abdominal pain after eating due to increased demand for splanchnic blood flow or related to acute thrombus superimposed on chronic thrombosis [21,54].

Some patients with chronic presentation have portal hypertension-related bleeding. (See "Epidemiology and pathogenesis of portal vein thrombosis in adults" and "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management" and "Portal hypertension in adults".)

DIAGNOSIS

There are no clinical features (symptoms, abdominal findings, laboratory tests) that are specific for mesenteric venous thrombosis, and, as with all forms of mesenteric ischemia (algorithm 1), a high index of suspicion is necessary to make an early diagnosis in patients with the acute or subacute forms or identify those with chronic thrombosis. A careful review of the patient's personal and family history may increase suspicion for venous thrombosis in a patient with a clinical presentation that suggests intestinal ischemia. A personal or family history of a deep vein thrombosis or pulmonary embolism is present in approximately one-half of patients with acute mesenteric venous thrombosis [22]. (See 'Risk factors' above.)

Nevertheless, a definitive diagnosis of mesenteric venous thrombosis relies upon the demonstration of thrombosis within the mesenteric veins on imaging studies, which is discussed below. The diagnosis will necessarily be made in the operating room in patients who present with signs of bowel infarction. (See 'Abdominal exploration' below.)

Imaging — A definitive diagnosis of mesenteric venous thrombosis is established with imaging studies that show thrombosis within the mesenteric veins. Mesenteric venous thrombosis is most reliably demonstrated using magnetic resonance (MR) venography, although we recommend computed tomography (CT) with and without oral and intravenous contrast as an initial screening study because of its widespread availability. For patients with a nondiagnostic CT for whom a clinical suspicion for mesenteric venous thrombosis persists (ie, no alternative explanation for symptoms and a known hypercoagulable risk factor or family history of venous thrombosis), we suggest angiography (CT angiography with delayed imaging to capture venous phase, MR angiography, or standard catheter based).

MR angiography is an excellent imaging study for the diagnosis of mesenteric venous thrombosis, but motion artifacts may limit its accuracy. Some studies report 100 percent sensitivity and specificity for the diagnosis of acute or chronic mesenteric venous thrombosis [61-63]. However, CT angiography of the abdomen is reasonably sensitive and specific for mesenteric venous thrombosis, provided there is adequate portal venous phase contrast, and is preferred over MR because of its lower costs and wide availability [64,65]. In addition, as an initial study, CT more reliably demonstrates findings of focal or segmental bowel wall ischemia, in addition to ruling out other causes of acute abdominal pain [66-69]. (See 'Differential diagnosis' below and "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Exclude bowel compromise'.)

In retrospective studies, the accuracy of abdominal CT for the diagnosis of mesenteric venous thrombosis is at least 90 percent [4,22,40,70-73], but most studies do not include comparisons with venography. Isolated mesenteric vein thrombosis may be more difficult to appreciate. In one study, abdominal CT was less frequently diagnostic for patients without compared with those with concomitant portal or splenic vein involvement (67 versus 97 percent) [20].

On CT, the diagnosis of mesenteric venous thrombosis is made by the presence of venous filling defects or absent flow in the mesenteric veins during the venous phase. A central low attenuation within a sharply defined, enhanced venous wall (contrast within the vasa vasorum) defines the defect [40]. In addition, resistance to venous flow may cause reflux of intravenous contrast into the aorta. Other associated findings include enhanced bowel wall and/or mesenteric stranding related to edema and changes associated with bowel obstruction or intestinal infarction (bowel wall thickening >3 mm, intestinal pneumatosis, portal vein gas (image 2), bowel dilation, unexplained ascites) [74]. (See 'Differential diagnosis' below and "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Abdominal CT'.)

For patients in whom the diagnosis of acute mesenteric ischemia is uncertain but a suspicion remains high, catheter-based mesenteric angiography should be performed, although it is uncommonly necessary. In addition to more accurately distinguishing arterial from venous forms of acute mesenteric ischemia, catheter-based arteriography provides access for thrombolytic and other interventional therapies. Findings on angiography consistent with acute mesenteric venous thrombosis include late filling of the superior mesenteric vein, thrombus in the superior mesenteric vein with partial or complete obstruction, failure of arterial arcades to empty normally, reflux of contrast into the arterial system (aorta), arterial spasm, and a prolonged vascular blush [75].

Chronic mesenteric venous thrombosis is suggested by the presence of thrombus with well-developed collateralization, often in association with cavernous transformation of the portal vein (collateral development within or around the thrombosed portal vein) and in the absence of signs of intestinal ischemia or infarction. Although CT is reasonably accurate for a diagnosis of chronic mesenteric venous thrombosis, Doppler ultrasound, MR angiography, or transhepatic portography can also be used; however, estimates of sensitivity and specificity are lacking. Doppler ultrasound is widely available, rapidly performed, and noninvasive, but it is not able to detect thrombosis in smaller mesenteric vessels. Although specificity for mesenteric venous thrombosis is excellent (100 percent), sensitivity is less (70 to 90 percent) [61,76].

Identifying associated hypercoagulability — After mesenteric venous thrombosis is diagnosed and treated, we suggest hypercoagulable testing for patients without known

predisposing illnesses for hypercoagulable conditions to help determine the duration of anticoagulation, predict associated complications of the underlying illness, and educate family members of those with heritable conditions about the genetics of their familial disease [11]. (See "Evaluating adult patients with established venous thromboembolism for acquired and inherited risk factors".)

Patients with acute isolated superior mesenteric venous thrombosis may be more likely to harbor an underlying systemic hypercoagulable state compared with patients who have thrombosis of the portal or splenic veins in addition to the superior mesenteric vein [20].

We suggest testing for hypercoagulability in patients with mesenteric venous thrombosis. At least one predisposing risk factor is recognized in most patients who present with mesenteric venous thrombosis, and, among patients with no obvious clinical risk factors at presentation, a heritable thrombophilia is frequently identified. Two or more procoagulant risk factors were present in 17 percent of 60 patients with chronic mesenteric venous thrombosis in one study [54]. Hypercoagulability testing should include protein S, protein C, and antithrombin III, factor V Leiden mutation, prothrombin G20210A mutation, activated protein C resistance, anticardiolipin antibodies, antiphospholipid antibodies, or lupus anticoagulant.

DIFFERENTIAL DIAGNOSIS

Patients with acute mesenteric venous thrombosis most commonly present with abdominal pain, which must be distinguished from other causes of abdominal pain and, importantly, from other causes of acute mesenteric ischemia. (See "Causes of abdominal pain in adults" and "Overview of intestinal ischemia in adults", section on 'Clinical features'.)

Chronic mesenteric venous thrombosis may also present with ascites or upper gastrointestinal bleeding related to portal hypertension, which can be due to portal or splenic vein thrombosis or other etiologies. (See "Acute portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management" and "Evaluation of adults with ascites", section on 'Differential diagnosis' and "Portal hypertension in adults", section on 'Evaluation for an underlying cause'.)

TREATMENT

The treatment of established mesenteric venous thrombosis (acute and subacute) is predominantly conservative, consisting of systemic anticoagulation to minimize extension of thrombus, bowel rest, and careful, serial observation for any signs of clinical deterioration (algorithm 2) [19,57,77-80]. Surgical exploration is limited to those patients with definite

signs of bowel infarction. Adjuncts to anticoagulation may include thrombolytic therapy or other endovascular treatment in selected patients unresponsive to conventional anticoagulation. (See 'Nonoperative management' below and 'Abdominal exploration' below and 'Pharmacologic thrombolysis/mechanical thrombectomy' below.)

It is imperative to identify patients with hypercoagulable conditions and treat them with long-term anticoagulant therapy. (See 'Identifying associated hypercoagulability' above and 'Anticoagulation' below.)

Nonoperative management — Patients with acute or subacute mesenteric venous thrombosis without indications for urgent surgery can be safely observed. In addition to anticoagulation, initial management includes intravenous fluid administration, bowel rest, and bowel decompression, with close monitoring for signs of worsening ischemia [43]. Based largely on animal studies demonstrating a survival benefit, prophylactic antibiotics are given to patients with acute mesenteric venous thrombosis to limit the effects of bacterial translocation [81-83]. Patients with acute isolated superior mesenteric venous thrombosis may be more likely to progress to surgery compared with patients who have thrombosis of the portal or splenic veins in addition to the superior mesenteric vein [20].

Initial management of patients with symptomatic chronic mesenteric venous thrombosis is focused on controlling variceal bleeding. Bleeding most commonly arises from the esophagus or stomach; less commonly, "ectopic" variceal bleeding arises in the small intestine, colon, or rectum. Bleeding from esophageal or gastric varices typically is managed endoscopically, as in the cirrhotic patient, whereas management of ectopic varices, ascites, and related symptoms is individualized. Beta blocker therapy is associated with decreased morbidity and mortality in those with superimposed portal vein thrombosis [54]. Other rarely used treatment options have included aggressive endovascular recanalization, portal reconstruction, and shunting to relieve chronic right-sided colonic ischemia [84,85]. (See "Overview of the management of patients with variceal bleeding" and "Methods to achieve hemostasis in patients with acute variceal hemorrhage" and "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management", section on 'Management'.)

Anticoagulation — We recommend anticoagulation for all patients with acute or subacute mesenteric venous thrombosis without signs of acute abdomen for which surgical intervention is imminent in order to limit propagation of thrombus and allow for recanalization [77,86,87]. Initiation of anticoagulation should not be delayed and remains the cornerstone of treatment in patients with mesenteric venous thrombosis. Treatment of patients with chronic mesenteric venous thrombosis (symptomatic or asymptomatic) is individualized, but anticoagulation is suggested [88]. The risk of bleeding risk appears to be <10 percent [86,89].

Anticoagulation can also be recommended in cirrhotic patients, even those with varices. Patients with bleeding varices should first receive treatment directed at the varices, and when stabilized, can proceed with anticoagulation. In patients with cirrhosis and portal vein thrombosis, anticoagulation increased recanalization rates, reduced progression of thrombosis, and reduced variceal bleeding [90]. (See "Acute portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management", section on 'Efficacy of anticoagulation'.)

Hospitalized patients can be initiated on systemic anticoagulation; typically, either unfractionated heparin or low-molecular-weight heparin (LMWH) may be used initially (or alternative agent as indicated), although LMWH should not be used in patients with renal failure or who require imminent surgery (eg, bowel necrosis) [91-93]. Once the patient's condition has stabilized and no further intervention is planned, the patient can be transitioned to an oral anticoagulant (vitamin K antagonist, novel oral anticoagulant [NOAC]) [94]. The presence of cirrhosis may affect the dosing of NOACs; however, available data are not clear regarding the appropriate dose. Case reports support a standard dose of rivaroxaban without any effect on efficacy and bleeding events [95]. (See "Warfarin and other VKAs: Dosing and adverse effects" and "Direct oral anticoagulants (DOACs) and parenteral direct-acting anticoagulants: Dosing and adverse effects".)

Treatment for at least three to six months is recommended; however, a longer duration may be warranted if a thrombophilic state has been identified [9,77,96]. In one review, a decision to continue treatment in patients with splanchnic vein thrombosis (portal vein, splenic vein, or mesenteric veins) was significantly associated with younger age, symptomatic presentation, multiple vein involvement, and unprovoked thrombosis [97].

In historical reviews, anticoagulant therapy reduced mortality rates from 50 percent for those who did not receive anticoagulants to 0 percent for those who received postoperative anticoagulation (diagnoses made with surgery) [8,77,98,99], a benefit that has been repeatedly demonstrated in later studies [4,89,91]. Anticoagulation also reduced recurrence of symptoms (14 versus 26 percent in one study, 0 versus 19 percent in another) [9,21] and improved survival in patients with acute mesenteric venous thrombosis (22 versus 59 percent) [9]. Following anticoagulation, most thrombosed veins partially or completely recanalize. In one study, vascular recanalization was 80 percent in anticoagulated patients, compared with <10 percent for patients who were not anticoagulated over a mean follow-up of five months [100].

Anticoagulation may also benefit patients with chronic mesenteric venous thrombosis, particularly those with hypercoagulability (see 'Identifying associated hypercoagulability' above). The main goal of anticoagulation in these patients is prevention of new episodes of thrombosis, but any potential benefit must be weighed against the risk of upper

gastrointestinal bleeding from varices. The available data suggest that anticoagulation may improve survival [54,101,102]. Anticoagulation can be suggested for patients with chronic mesenteric venous thrombosis, even those who have bled from varices, provided the patient is reliable and understands the risks and benefits of treatment.

Monitoring progress — Although prospective studies are lacking, reviews of patients with acute mesenteric venous thrombosis who were initially treated with anticoagulation therapy suggest that subsequent surgical therapy is deemed necessary in approximately one third of patients [57,103]. Thus, we monitor the patient with serial abdominal examination and laboratory studies for signs of clinical worsening that may manifest as peritonitis related to transmural ischemia, infarction or perforation, or development of small bowel obstruction. Abdominal imaging studies are repeated as the clinical situation dictates. We also monitor for the development of abdominal compartment syndrome. The absence of any of these issues and gradual clinical improvement allow initiation of a diet.

We monitor laboratory studies daily (or more frequently when needed), including complete blood count (CBC), white blood cell (WBC) count with differential, serum electrolytes including bicarbonate, and serum lactate levels for signs of bowel ischemia, metabolic acidosis, or sepsis. Follow-up abdominal computed tomography (CT) scan in 24 to 48 hours is reasonable to exclude signs of transmural necrosis. Most of the literature on the subject considers surgical exploration to be indicated if there are signs of peritoneal irritation at presentation. However, with acute mesenteric venous thrombosis, peritoneal signs may not strictly correlate and may underestimate the severity of bowel ischemia. Some authors have suggested that bowel wall thickness and bowel wall enhancement on the arterial phase of CT should be evaluated as criteria to determine whether or not to proceed with abdominal exploration [19]. Small bowel obstruction may develop in an area of irreversible ischemia but needs to be distinguished from ileus due to bowel edema. (See "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Adynamic (paralytic) ileus' and "Management of small bowel obstruction in adults", section on 'Indications for immediate surgery' and "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Diagnosis'.)

ABDOMINAL EXPLORATION

Surgery should not be delayed in patients with overt signs of intestinal necrosis or perforation based upon clinical, radiographic, or laboratory parameters [77].

When abdominal exploration is indicated, we prefer an open approach over laparoscopic exploration. The extent of bowel edema and resulting abdominal distention make a laparoscopic approach difficult, and insufflation of the abdomen can exacerbate mesenteric venous hypertension [14].

Considerable difficulty may be encountered in determining the extent of resection during abdominal exploration. Limited areas of nonviable, grossly infarcted small bowel should be resected and can be primarily anastomosed without significant morbidity. The border between ischemic bowel and viable bowel is often diffuse and indistinct in the acute stage of mesenteric venous thrombosis [104]. If extensive ischemia is present, every attempt should be made to conserve as much bowel as possible.

Because bowel viability is often difficult to determine, some surgeons advocate the use of intraoperative Doppler ultrasound or fluorescein infusion with Wood's lamp examination to aid the assessment of intestinal viability [4,14]. Numerous other techniques for assessing intestinal viability are available [105]; however, we do not feel that they are useful for venous ischemia. Data are derived primarily from cases of arterial ischemia [106]. Further studies are needed to determine their value in the management of mesenteric venous thrombosis.

The presence of thrombi in the cut venous supply of grossly normal segments surrounding frankly gangrenous bowel has led some authors to advocate wider surgical margins and others to advocate "second-look surgery" [3], especially given the high recurrence rate at the anastomotic site. Second-look surgery is necessary in approximately one fourth of patients. There are no absolute indications that dictate whether the abdomen should be left open following laparotomy [107]. But, whenever the viability of the intestine is in question, a second-look operation should be planned and performed as scheduled [4,14,72,103,106,108]. Other indications include a high risk for abdominal compartment syndrome and abdominal sepsis. (See 'Second-look operation' below.)

Although adjunctive measures, such as thrombolytic infusion, have been administered intraoperatively [14,109,110], most studies have reported on transcatheter routes of such therapy. The use of intra-arterial vasodilators, such as papaverine, is also controversial in cases of occlusive mesenteric ischemia [111]. When used to minimize the associated arterial vasospasm, the typical dose of papaverine is 30 to 40 micrograms/kg/minute. (See 'Pharmacologic thrombolysis/mechanical thrombectomy' below.)

PHARMACOLOGIC THROMBOLYSIS/MECHANICAL THROMBECTOMY

Although there are no large or well-controlled studies to guide decision making, catheter-based techniques offer an adjunct to anticoagulation in centers with sufficient technical expertise and experience. Successful venous thrombolysis with streptokinase, urokinase, and tissue plasminogen activator has been reported in case reports and small series [109,112-125]. Transarterial thrombolysis may be an alternative approach for selected mesenteric beds. In specialized centers, transcatheter thrombolysis may be a reasonable therapeutic adjuvant for well-selected patients with severe disease, for whom an inadequate response to anticoagulation is observed, but without signs of bowel necrosis. However, additional studies are needed to demonstrate the safety and efficacy of thrombolysis compared with anticoagulation. At this time, thrombolysis for mesenteric venous thrombosis should be considered experimental.

Trans**venous** thrombolysis and/or thrombectomy, which may be useful for decreasing clot burden in acute large vessel thrombosis [2,77], has been described via percutaneous transhepatic, transfemoral, and transjugular approaches [120,124,126-129]. A transvenous approach was used in two case series that included a total of 28 patients with acute superior mesenteric venous thrombosis [126,130]. The majority of patients (82 percent) achieved partial or complete lysis, and 87 percent of patients had an improvement in their symptoms. No patient required intestinal resection, although two patients died of refractory thrombosis in the setting of sepsis and serious bleeding occurred in several patients. In another experience, a worse outcome occurred when thrombolytic therapy was initiated more than 24 hours after presentation [59]. In a retrospective review of 25 patients with circumscribed peritonitis, patients who received catheter-directed thrombolysis had similar 30 day and one-year mortality rates, compared with a matched, surgically treated group [131]. However, the thrombolysis group had a significantly shorter hospital stay (43 versus 20 days), earlier resumption of enteral or oral nutrition (20 versus 9 days), and lower total treatment costs.

For selected patients, a trans**arterial** route can be used with placement of a catheter into the superior mesenteric artery [125-127,132,133]. Such an approach may be particularly useful for patients with isolated superior mesenteric vein thrombosis who require emergency laparotomy. In a retrospective study of 32 patients, those who received catheter-directed thrombolysis (urokinase/papaverine x 72 hours after a urokinase bolus) into the superior mesenteric artery had significantly better outcomes compared with those who received only postoperative anticoagulation [134]. The rate of complete thrombus resolution was increased (80 versus 20 percent), and the need for second-look laparotomy decreased (20 versus 70 percent). Among those who required follow-up laparotomy, there was less bowel necrosis (6 versus 41 percent), and bowel resection was needed in fewer patients (13 versus 59 percent). Overall, the incidence of short bowel syndrome and the 30 day mortality rates were also lower (7 versus 41 percent), and one-year survival was improved (93 versus 53 percent). However, clinically significant

bleeding was higher among those who received catheter-directed thrombolysis (20 versus 12 percent).

POSTOPERATIVE CARE AND FOLLOW-UP

Patients may remain hospitalized for several weeks or even longer depending on various factors, including pain control, need for surgery, issues related to anticoagulation, and difficulties returning to an oral diet requiring enteral or parental nutrition support. In one review, among patients successfully treated with a nonoperative approach, the average length of hospitalization is 13 to 23 days [19,57].

Once patients have remained clinically stable, with no signs of hemodynamic compromise; absence of abdominal pain, nausea, vomiting, or distention; and have had resolution of ischemic changes on computed tomography (CT) scan, oral nutrition can be instituted and gradually advanced with careful monitoring for signs of recurrent ischemia. For patients who are not meeting nutrition goals (ie, calorie, nutrient, and fluid requirements), enteral or parenteral nutrition support may be required. (See "Clinical assessment and monitoring of nutrition support in adult surgical patients".)

Long-term anticoagulation for a minimum of six months is advocated. Based on the etiology of the thrombosis, indefinite anticoagulation may be indicated. Among patients for whom long-term oral anticoagulation is indicated, the risk of recurrent venous thromboembolism is approximately 15 percent [135]. (See 'Anticoagulation' above.)

The postoperative course for patients requiring bowel resection varies according to the operative findings and extent of resection. (See "Bowel resection techniques" and "Overview of colon resection".)

Second-look operation — Any planned "second-look" operation should be undertaken in the first 12 to 48 postoperative hours to reassess any intestinal segments of questionable viability [4,14,106,108]. In the largest experience reported with reoperation for mesenteric venous thrombosis, second laparotomy was performed 24 hours after the initial resection because of concerns regarding intestinal viability in 14 of 31 patients; in all cases, gangrene was found and further resection was required [4].

Management of open abdomen — For patients in whom it was elected to leave the abdomen open following initial or second-look laparotomy following exploration for acute mesenteric venous thrombosis, the open abdomen can be managed using a variety of techniques. Methods

for temporary abdominal closure and timing of definitive closure are discussed separately. (See "Management of the open abdomen in adults".)

MORBIDITY AND MORTALITY

Acute mesenteric venous thrombosis has a better prognosis than other forms of acute mesenteric ischemia. In a large systematic review of almost 3700 cases of acute mesenteric ischemia, the overall mortality rate of patients with mesenteric venous thrombosis was 44 percent, compared with 66 to 89 percent for patients with arterial occlusive or nonocclusive ischemia [18]. Morbidity and mortality related to mesenteric venous thrombosis have improved due to better recognition and early treatment [4,9,16,18,46,57,79,100,103]. With prompt diagnosis and anticoagulation, mortality rates for acute mesenteric venous thrombosis in modern studies are between 10 and 20 percent [16,19,20,40,136,137].

Morbidity, mortality, and survival rates are similar in surgical and nonsurgical groups, with a shorter length of hospital stay in patients who do not require surgery [19,57]. Mortality rates are high for those with intestinal infarction at more than 75 percent [70].

The prognosis for patients with chronic mesenteric venous thrombosis is related to the severity of the underlying illness (eg, malignancy is associated with shorter survival). Overall survival rates as high as 78 to 83 percent over one to five years have been reported [4,54]. Among patients with a history of mesenteric venous thrombosis not associated with cirrhosis or malignancy, the three-year survival rate may be as high as 93 percent [21]. Beta blockade (to prevent variceal bleeding) and long-term anticoagulation may be associated with longer survival. In a review of 60 patients with chronic portal, splenic, and/or mesenteric thrombosis, survival was 82 percent at one year and 78 percent at five years; 10 percent of patients had cirrhosis [54].

Recurrent symptoms — Though the reported mesenteric venous thrombosis recurrence rate seems to be low while patients are receiving anticoagulation, recurrence is still possible [89,135]. Most cases of acute mesenteric venous thrombosis evolve toward the chronic form, with vein stenosis or occlusion and development of collateral veins [138]. For patients with symptomatic recurrent thrombosis, the need to reoperate can lead to catastrophic sequelae, such as short bowel syndrome, if additional segments of bowel require resection. (See "Chronic complications of the short bowel syndrome in adults" and "Management of short bowel syndrome in adults".)

Intestinal stricture — After patients recover from the acute stage of mesenteric venous thrombosis, the development of a small bowel stricture is among the possible complications during the chronic stage [47,91].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Intestinal ischemia".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Ischemic bowel disease (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Mesenteric venous thrombosis** – Acute mesenteric ischemia refers to the sudden onset of intestinal hypoperfusion, which can be due to mesenteric venous occlusion. The proportion of cases of acute mesenteric ischemia attributed to mesenteric venous thrombosis has decreased over time, likely related to better differentiation of mesenteric venous thrombosis from the occlusive and nonocclusive forms of acute arterial mesenteric ischemia. (See 'Introduction' above.)

- **Pathophysiology** – Acute thrombotic occlusion of one or more mesenteric veins (figure 1 and figure 2) reduces perfusion pressure due to increased resistance in the

mesenteric venous bed. Mesenteric vein thrombosis almost always involves the distal small intestine (superior mesenteric venous drainage) and rarely involves the colon (inferior mesenteric venous drainage), possibly related to difference in collateral flow. Increased venous pressure leads to efflux of fluid into the tissues, causing profound bowel wall edema, which combined with sequestration of fluid into the bowel lumen results in relative hypovolemia and systemic hypotension. (See 'Mesenteric venous anatomy and pathophysiology' above.)

- **Risk factors** – Mesenteric venous thrombosis is a multifactorial disorder predisposed by certain risk factors, which can be broadly divided into acquired (eg, pancreatitis, malignancy) and inherited conditions (eg, prothrombin G20210A mutation). Risk factors are listed above. At least one predisposing risk factor is recognized in most patients who present with mesenteric venous thrombosis. (See 'Incidence and risk factors' above.)

- **Clinical presentations** – Clinical features of mesenteric venous thrombosis are determined by the location and timing of thrombus formation within the splanchnic vasculature. Mesenteric venous thrombosis can present acutely, subacutely, or more chronically. Patients with acute or subacute mesenteric venous thrombosis have variable degrees of abdominal pain with or without peritoneal signs related to ischemic complications. Patients with chronic mesenteric venous thrombosis can be asymptomatic or symptomatic and typically present with complications of portal hypertension (variceal bleeding or ascites), related in most cases to concomitant portal or splenic vein thrombosis. (See 'Clinical presentations' above.)

- **Diagnosis** – A high index of suspicion based upon history and clinical findings is necessary to make an early diagnosis. Definitive diagnosis of mesenteric venous thrombosis relies upon the demonstration of thrombus within the mesenteric veins on imaging studies. Although magnetic resonance (MR) venography is overall the most accurate imaging study for the diagnosis of mesenteric venous thrombosis, we suggest computed tomography (CT) of the abdomen as an initial screening study for acute or subacute mesenteric venous thrombosis. Abdominal CT more reliably demonstrates findings of focal or segmental bowel wall ischemia, in addition to excluding other causes of acute abdominal pain, and is inexpensive and widely available. For patients in whom the diagnosis of acute mesenteric ischemia is uncertain but a suspicion remains high, angiography (CT angiography, MR angiography, or catheter based) should be performed, though it is uncommonly necessary. The diagnosis will necessarily be made in the operating room in patients who present with signs of bowel infarction. (See 'Diagnosis' above and 'Abdominal exploration' above.)

- **Management** – The management of established mesenteric venous thrombosis (acute and subacute) is predominantly conservative, consisting of systemic anticoagulation to minimize extension of thrombus, bowel rest, and careful, serial observation for any signs of clinical deterioration (algorithm 2). Surgical exploration is limited to those patients with definite signs of bowel infarction.

  - **Anticoagulation** – For patients with **acute** or **subacute** mesenteric venous thrombosis without signs of acute abdomen for which surgical intervention is imminent, we initiate systemic anticoagulation to minimize extension of thrombus, rather than expectant management alone (includes bowel rest and decompression, fluid therapy, and serial abdominal examination). An adjunct to anticoagulation may include thrombolytic therapy or other endovascular treatment. We also initiate prophylactic antibiotics to minimize bacterial translocation. (See 'Treatment' above and 'Anticoagulation' above and 'Pharmacologic thrombolysis/mechanical thrombectomy' above.)

    For patients with **chronic** mesenteric venous thrombosis, anticoagulation is individualized and depends upon symptoms, location and extent of thrombosis, and risk for bleeding.

    Our recommendations for duration of therapy are as follows:

    - For patients with no identifiable risk factors, or transient or correctable acquired risk factors (eg, pancreatitis), we anticoagulate for six months of anticoagulation, rather than long-term anticoagulation.

    - For patients with risk factors that cannot be corrected (malignancy, inherited condition), we anticoagulate long term, rather than six months of anticoagulation.

  - **Monitoring** – We serially monitor the patient for signs of clinical worsening (physical examination, laboratory testing, repeat imaging). The absence of any of these issues and serial clinical improvement in symptoms allow initiation of a diet. (See 'Monitoring progress' above and 'Postoperative care and follow-up' above.)

  - **Surgery** – Patients with clinical signs of bowel infarction require surgical intervention. We use an open approach rather than laparoscopic exploration. The extent of bowel edema and resulting abdominal distention make a laparoscopic approach difficult, and insufflation of the abdomen can exacerbate mesenteric venous hypertension. We have a low threshold for leaving the abdomen open to facilitate second-look operation. Whenever the viability of the intestine is in question, a second-look operation should be

planned and performed as scheduled. (See 'Abdominal exploration' above and 'Second-look operation' above.)

- **Further evaluation** – For patients diagnosed with mesenteric venous thrombosis who have no known predisposing risk factors, we suggest hypercoagulable testing to help determine the duration of anticoagulation, predict associated complications of the underlying illness, and educate family members. Patients with acute isolated superior mesenteric venous thrombosis may be more likely to harbor an underlying systemic hypercoagulable state, compared with patients who have thrombosis of the portal or splenic veins in addition to the superior mesenteric vein. (See 'Identifying associated hypercoagulability' above and "Screening for inherited thrombophilia in asymptomatic adults".)

- **Outcomes** – Acute mesenteric venous thrombosis has a better prognosis than acute arterial mesenteric ischemia. Morbidity and mortality related to mesenteric venous thrombosis have improved due to better recognition and early treatment. With prompt diagnosis and anticoagulation, mortality rates for acute mesenteric venous thrombosis in modern studies are between 10 and 20 percent, but mortality rates remain as high as 75 percent for those with intestinal infarction. The prognosis for patients with chronic mesenteric venous thrombosis is related to the severity of the underlying illness (eg, malignancy). (See 'Morbidity and mortality' above.)

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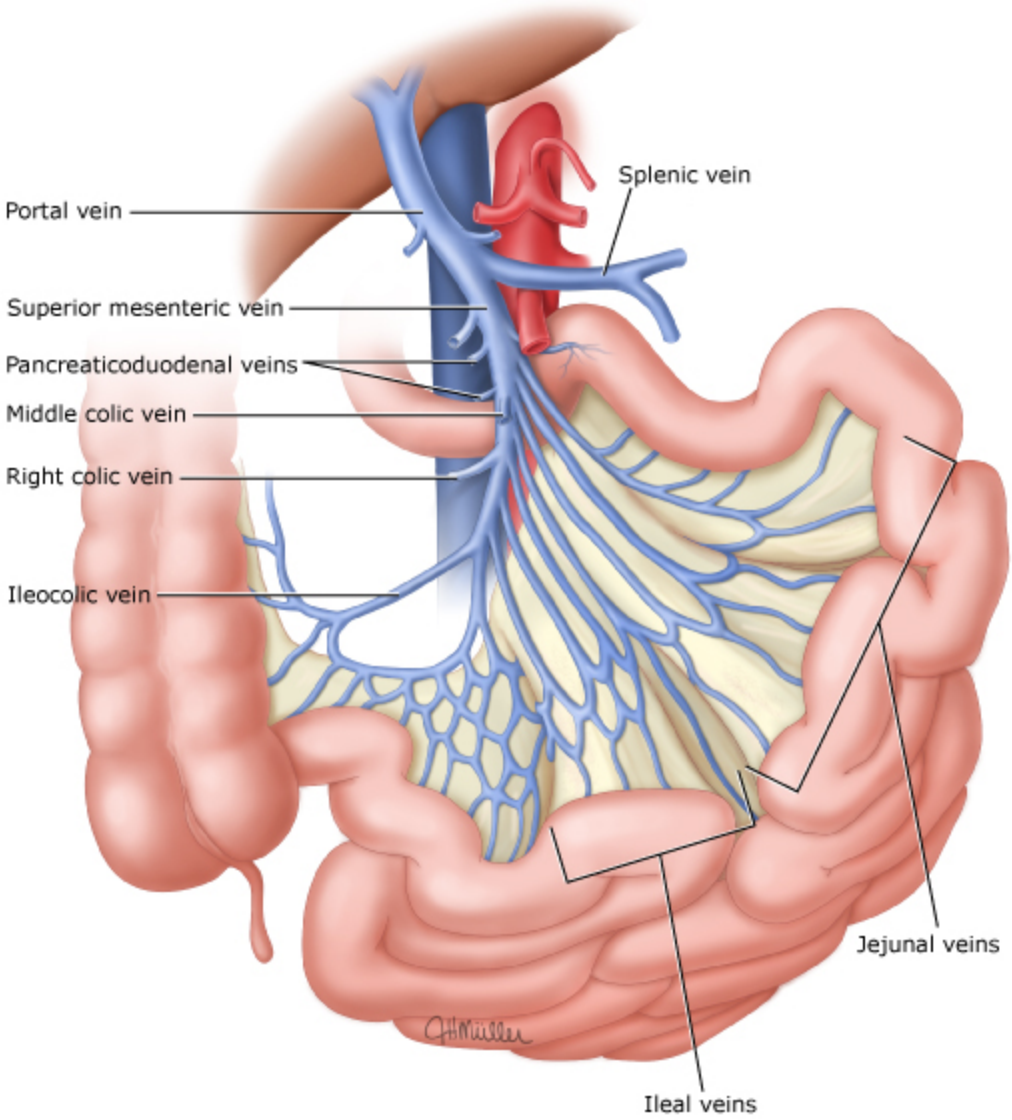
Topic 91220 Version 16.0

GRAPHICS

Venous drainage of the small intestine

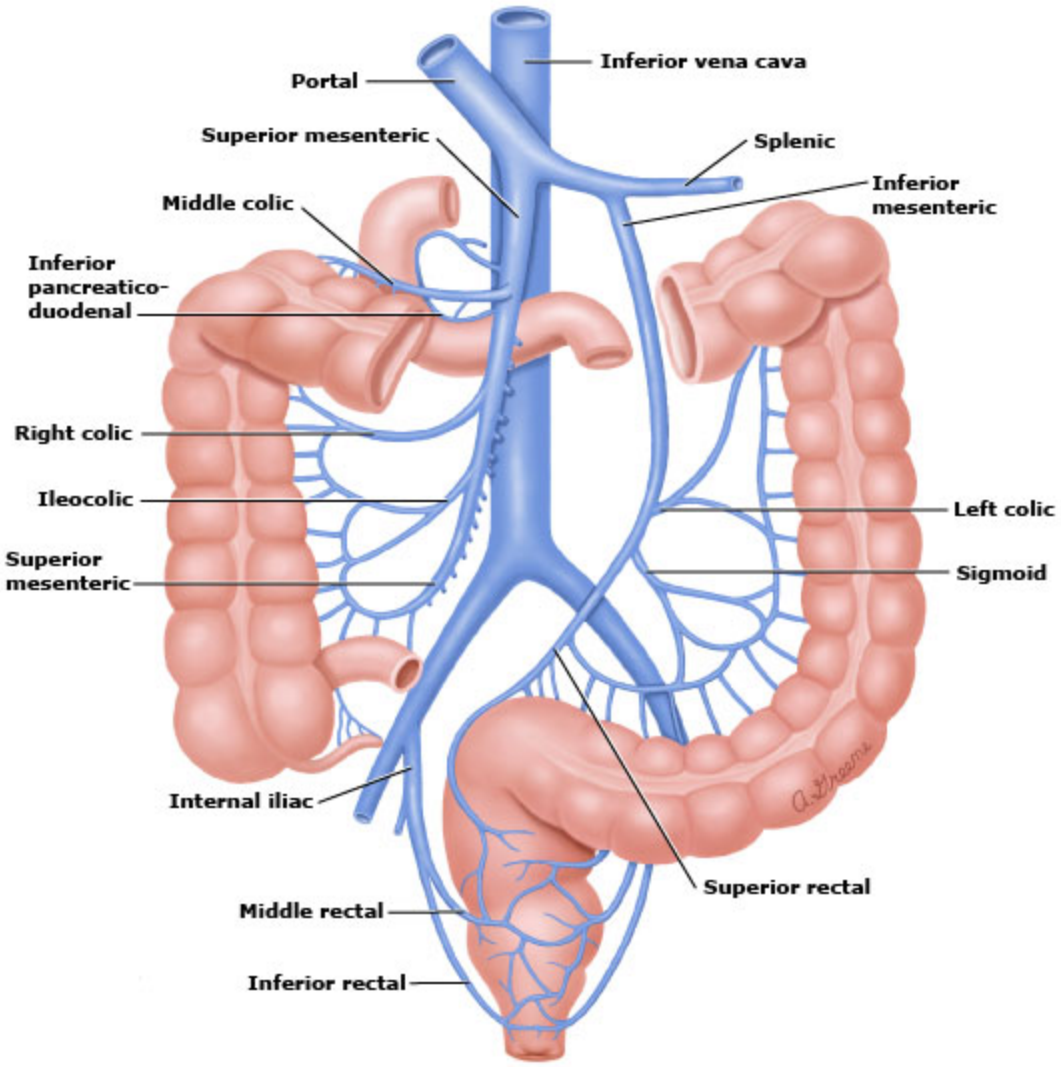


The mesenteric veins parallel their corresponding arteries. The SMV drains the small intestine, cecum, ascending, and transverse colon via the jejunal, ileal, ileocolic, right colic, and middle colic veins. The SMV joins the splenic vein to drain into the portal vein.

SMV: superior mesenteric vein.

Graphic 88949 Version 4.0

Venous drainage of the colon and rectum

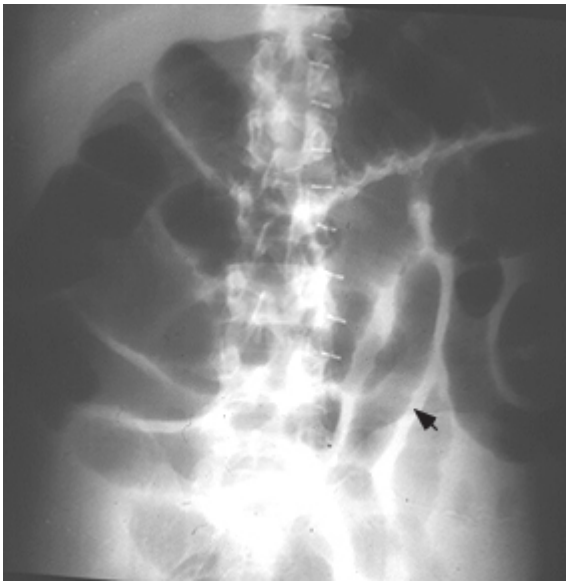


The mesenteric veins parallel their corresponding arteries. The SMV drains the small intestine, cecum, and ascending and transverse colon via the jejunal, ileal, ileocolic, right colic, and middle colic veins. The IMV drains the descending colon through the left colic, the sigmoid through the sigmoid vein, and the rectum through the superior rectal vein. The IMV fuses with the splenic vein, which then joins the SMV to form the portal vein.

SMV: superior mesenteric vein; IMV: inferior mesenteric vein.

Graphic 81960 Version 4.0

Small bowel ischemia on plain abdominal film

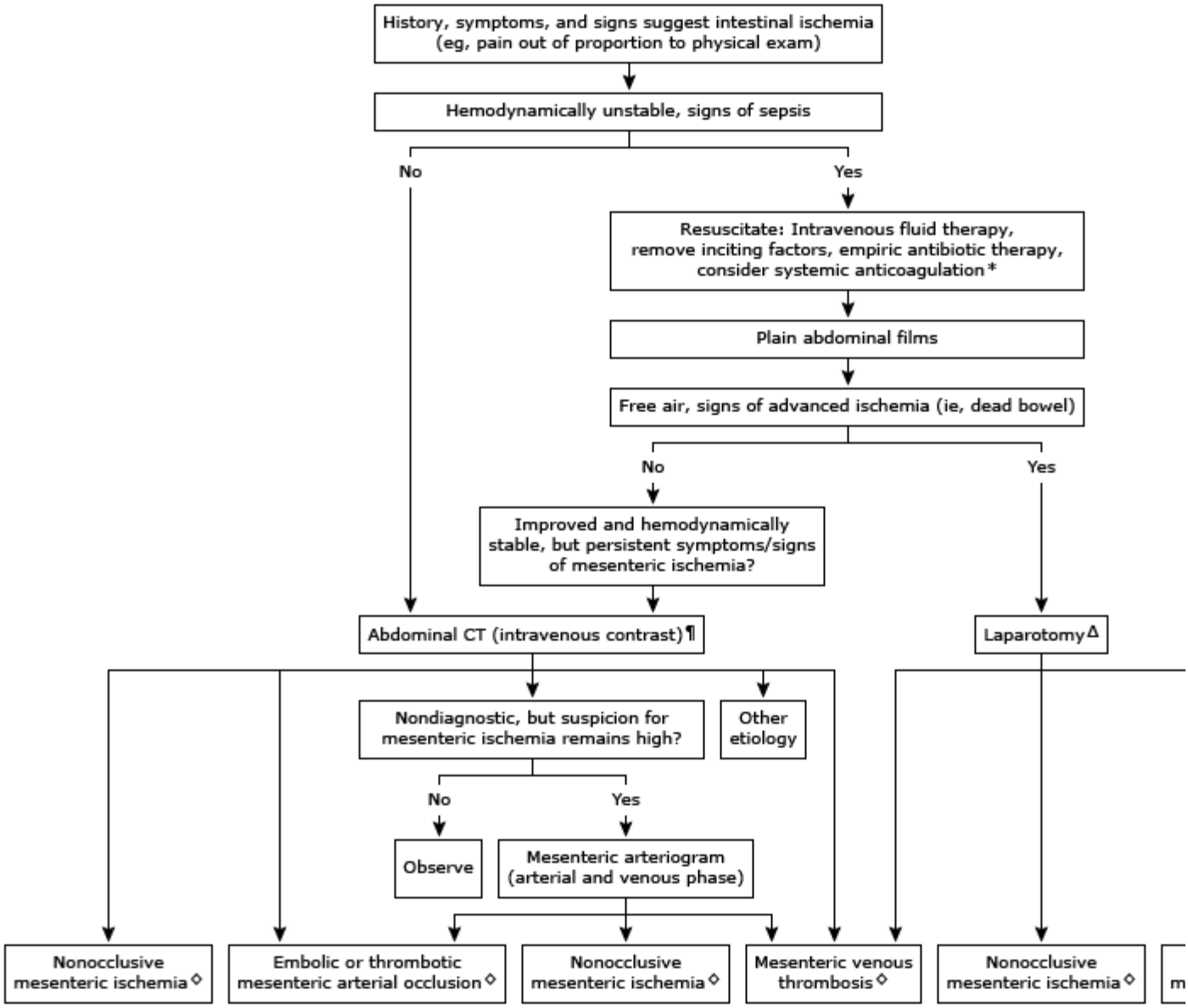


A plain radiograph of the abdomen demonstrates distended featureless loops of small bowel with wall thickening (arrow) and separation of the bowel loops. These findings are consistent with hemorrhage into the bowel wall secondary to ischemia.

Courtesy of Jonathan B Kruskal, MD, PhD.

Graphic 74055 Version 3.0

Diagnosis and initial management of intestinal ischemia



CT: computed tomography.

* Patients ultimately identified with nonocclusive mesenteric ischemia will not benefit from anticoagulation, be discontinued.

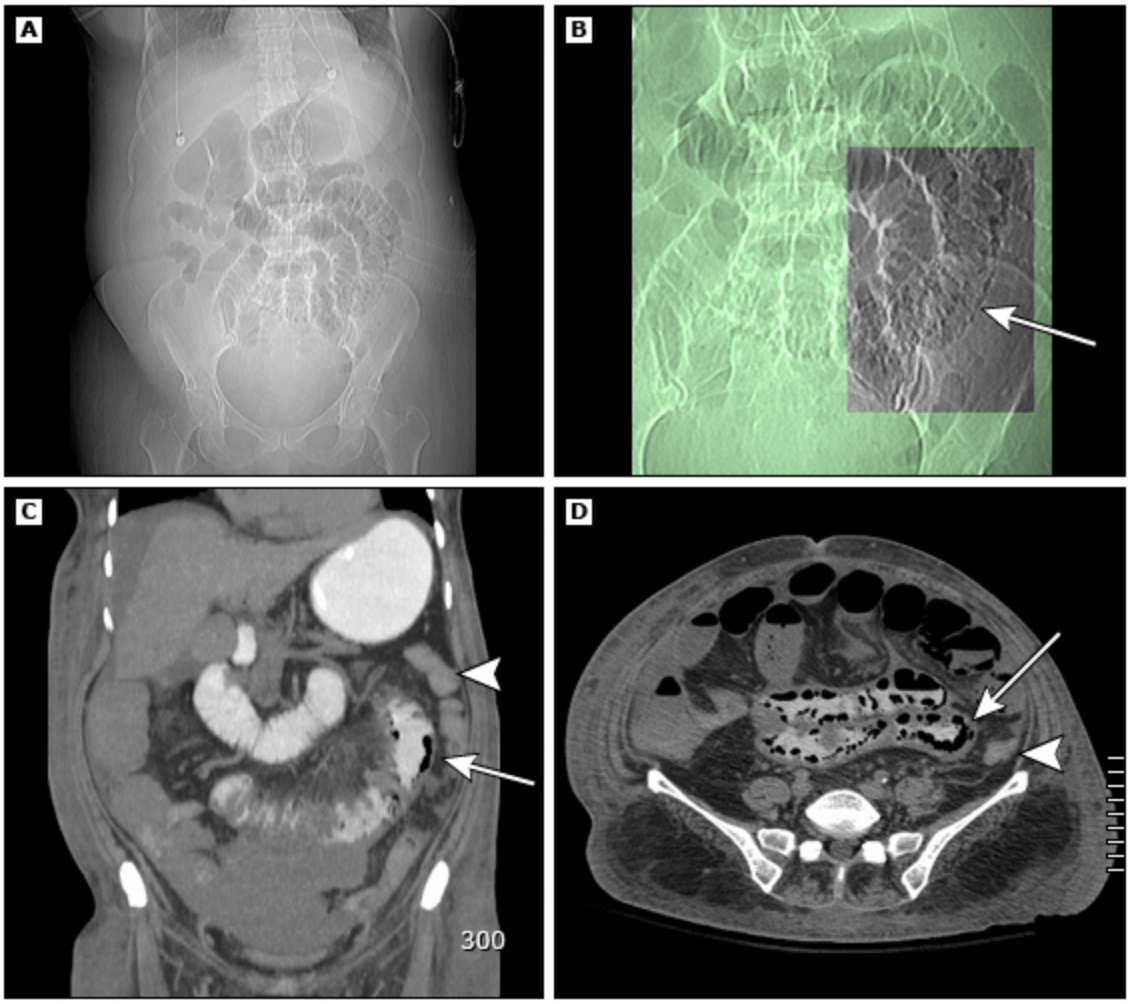
¶ Imaging signs associated with mesenteric ischemia include focal or segmental bowel wall thickening, inte portal vein gas, portomesenteric thrombosis, mesenteric arterial calcification, and mesenteric artery occlusi

Δ Medically fit patients.

◇ Refer to associated UpToDate algorithms on mesenteric ischemia (acute or chronic, occlusive or nonocclu venous).

Graphic 62760 Version 6.0

CT scan of pneumatosis coli

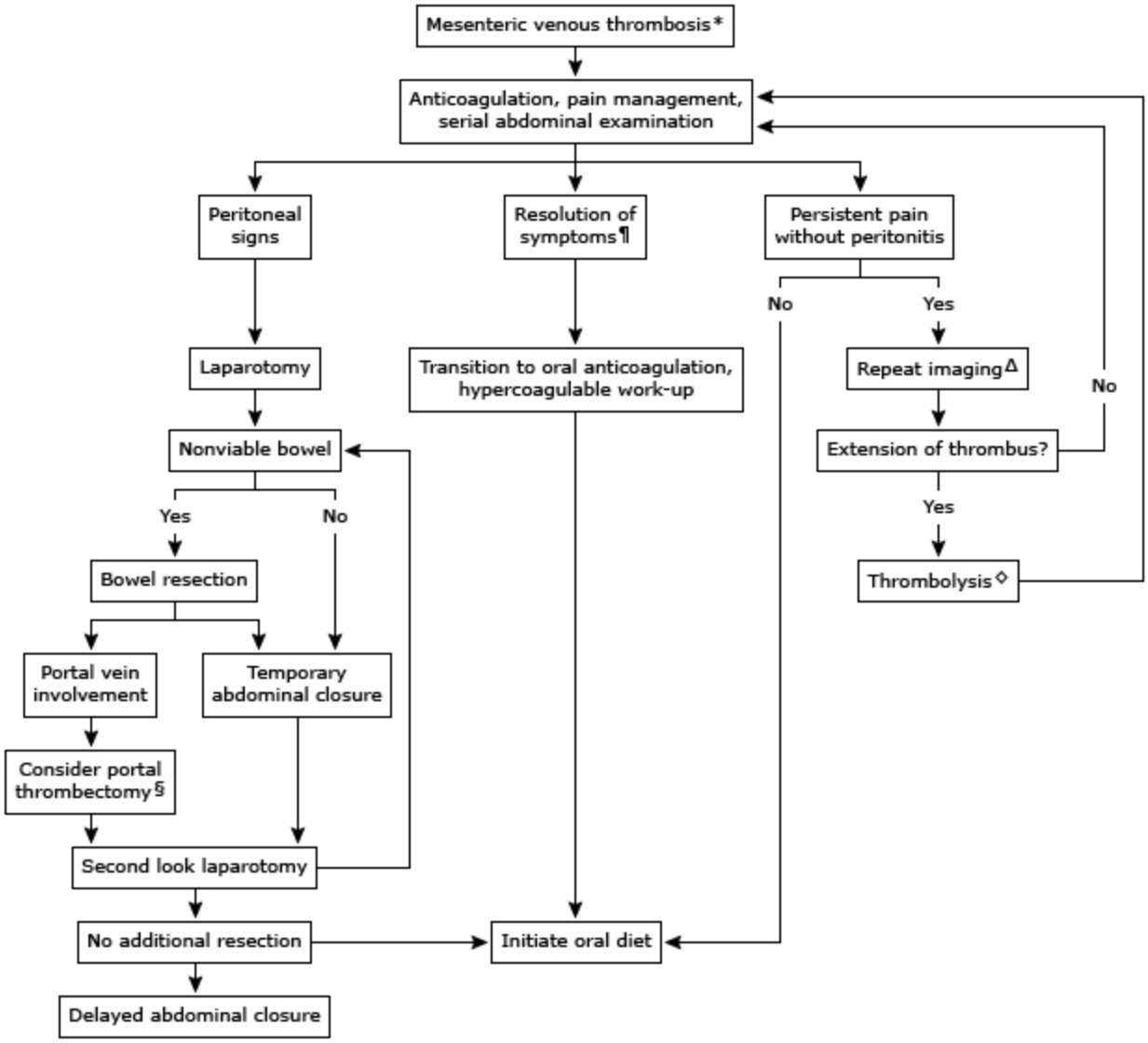


Computed tomography (CT) imaging of the abdomen is from a 55-year-old female who presented with acute abdominal pain and distension. Image A is a scout film of the abdomen and image B is a magnified view of the small bowel from the scout film. The images reveal air within the wall of the dilated loops of small-bowel, characteristic of pneumatosis intestinalis. The paucity of gas in the colon and rectum suggests SBO or severe ileus. Image C is coronal reformat through the abdomen confirming the accumulation of bubbles of air within the wall of the small bowel (white arrow). Image D is an axial image through the affected small bowel loop that shows more extensive accumulation of air within the wall of the thickened small bowel wall. Associated findings include decompressed large bowel (arrowheads), suggesting small bowel obstruction or severe ileus. In the appropriate clinical setting, the findings are highly suggestive of acute ischemia of the small bowel causing a severe ileus and functional obstruction. Less likely but also possible is small bowel obstruction with secondary ischemia.

CT: computed tomography; SBO: small bowel obstruction.

Graphic 58309 Version 3.0

Management of mesenteric venous thrombosis



CT: computed tomography.

* Diagnosis is typically made on CT angiography.

¶ In uncomplicated cases, symptoms typically resolve steadily over time.

Δ Imaging may include magnetic resonance venography.

◇ If thrombolysis is contraindicated, continue anticoagulation.

§ Portal embolectomy is uncommonly performed.

Graphic 76406 Version 4.0

Contributor Disclosures

David A Tendler, MD No relevant financial relationship(s) with ineligible companies to disclose. **J Thomas Lamont, MD** Equity Ownership/Stock Options: Allurion [Weight loss]. Consultant/Advisory Boards: Teledoc [Gastrointestinal diseases]. All of the relevant financial relationships listed have been mitigated. **Peter Grubel, MD** No relevant financial relationship(s) with ineligible companies to disclose. **John F Eidt, MD** Grant/Research/Clinical Trial Support: Syntactx [Clinical events and data/safety monitoring for medical device trials]. All of the relevant financial relationships listed have been mitigated. **Joseph L Mills, Sr, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Kathryn A Collins, MD, PhD, FACS** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

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