Gastroenterology Clinics
Volume 32 • Number 4 • December 2003

Review article

Intestinal ischemia

Bryan J. Burns, MD
Lawrence J. Brandt, MD *

Division of Gastroenterology
Department of Medicine
Montefiore Medical Center
Albert Einstein College of Medicine
111 East 210th Street
Bronx, NY 10467, USA

A vascular emergency of the gastrointestinal tract usually results from a disturbance in the arterial blood supply or venous drainage of the bowel with consequential severe ischemic injury. Such severe ischemia manifests as gangrene and perforation or less commonly as stricture formation with intestinal obstruction. Vascular emergencies much more commonly involve the small intestine than the colon. Indeed, most cases of colon ischemia resolve spontaneously without sequelae [1] [2] [3] .

Acute ischemic injury to the small bowel is referred to as “acute mesenteric ischemia” (AMI) and may be arterial or venous; arterial causes of AMI are more common and include superior mesenteric artery embolus (SMAE), superior mesenteric artery thrombosis (SMAT), and nonocclusive mesenteric ischemia (NOMI). Venous forms of AMI tend to result from superior mesenteric venous thrombosis (MVT).

Regardless of the cause of the ischemic insult, the end results are similar: a spectrum of bowel injury that can range from mild alteration in bowel function to vascular emergencies with transmural necrosis and intestinal gangrene. Early recognition, appropriate diagnostic studies, and aggressive treatment of the latter are essential if the usually poor outcome is to be improved. This article discusses the presentation, diagnosis, and treatment of these conditions.

Anatomy of the splanchnic circulation

The celiac axis and superior mesenteric artery (SMA) normally supply all of the blood flow to the small intestine, with most of the blood supply delivered by the SMA. There is great variability in the vascular anatomy from person to person, but consistent patterns have been described [4] .

The celiac axis arises from the anterior aorta and divides into three major branches: (1) the left gastric artery, (2) the common hepatic artery, and (3) the splenic artery. These arteries branch further as they supply blood to the stomach, duodenum, pancreas, and liver. A rich collateral circulation between the branches of the celiac axis and those of the SMA accounts for the rarity of ischemic events to these organs.

The SMA also arises from the anterior aorta, slightly distal to the takeoff of the celiac axis. The SMA gives rise to four major branches ([1] the inferior pancreaticoduodenal artery, [2] middle colic artery, [3] right colic artery, and [4] ileocolic artery) and numerous jejunal and ileal branches. These intestinal branches form a series of arcades, eventuating in a terminal arcade from which numerous straight end arteries enter the intestinal wall. This anatomic pattern renders the intestine especially vulnerable to ischemic insults because the end arteries are without collateral pathways.

The major anastamoses between the celiac axis and the SMA are by the superior pancreaticoduodenal branch (off the common hepatic artery) of the celiac axis and the inferior pancreaticoduodenal branch of the SMA, which join to form the pancreaticoduodenal arcade, supplying blood to the pancreas and duodenum [5] .

AMI: general features

Clinical presentation

Most patients with AMI present with severe abdominal pain. The pain may vary in location and nature but, early in the course of the disease, the degree of pain is classically more impressive than are the physical findings (ie, the pain may be very severe, whereas the abdomen tends to be soft and relatively nontender). As the ischemia progresses and infarction develops, the patient typically develops signs of peritonitis, including a rigid, distended abdomen with a loss of bowel sounds. It is imperative to consider and either diagnose or exclude AMI in patients who present with severe abdominal pain and a paucity of significant abdominal findings. The dangers of a delay in diagnosis outweigh the risk of early invasive studies [6] .

Several clues to the etiology of the ischemia may be present. Acute abdominal pain followed by rapid and forceful bowel evacuation strongly suggests SMAE. A history of chronic abdominal pain (abdominal angina) for several weeks to months followed by an acute event can be present in up to 50% of patients with SMAT [7] . NOMI may be present in patients with underlying hypotension and volume deficits, including congestive heart failure, hypovolemia, sepsis, and cardiac arrhythmias. NOMI may present atypically with unexplained abdominal distention or gastrointestinal bleeding and pain may be absent in up to 25% of cases of NOMI. A history or the presence of a deep vein thrombosis may indicate superior MVT. Superior MVT also tends to manifest less acutely than arterial etiologies, because the thrombotic occlusion tends to evolve over several days.

Aside from abdominal pain, AMI may present with other signs, for which the clinician should be alert. The stool may contain occult blood in up to 75% of the patients. The passage of maroon or red blood per rectum, although characteristic of colon ischemia, may be seen in colon ischemia involving the ascending colon with concurrent or subsequent AMI. This association occurs because the SMA provides blood to both the small intestine and the ascending colon [8] .

Unexplained abdominal distention may be present early in the course of AMI and may often herald intestinal infarction. Mental confusion also has been reported in 30% of cases of AMI in elderly patients [9] .

Overall, the presenting symptoms and signs are relatively nonspecific for mesenteric ischemia and can be seen in a wide variety of gastrointestinal disorders. Early diagnosis of AMI requires a high index of clinical suspicion. Ischemia should always be considered in patients at high risk for embolic or thrombotic events: patients with cardiac disease, arrhythmias, congestive heart failure, or a recent myocardial infarction. AMI may also be precipitated by severe illness that leads to hypotension and hypovolemia (eg, sepsis). NOMI has been increasingly recognized following cardiac surgery and hemodialysis [10] [11] . Other risk factors for AMI include prothrombotic states (eg, factor V Leiden mutation, lupus anticoagulant, carcinomatosis); vasculitis (eg, polyarteritis nodosa, Henoch-Schönlein purpura); or medications and drugs that cause vasoconstriction (eg, digitalis, vasopressin, pseudoephedrine, cocaine, amphetamines) [12] . AMI caused by SMAT has even been reported following routine procedures, such as colonoscopy or laparoscopic cholecystectomy [13] [14] [15] .

Laboratory findings and radiologic studies

Most laboratory findings are associated with late disease and are not helpful in identifying patients sufficiently early that intervention dramatically improves outcome. On admission to the hospital, approximately 75% of patients with AMI have a leukocytosis above 15,000 cells/mm^3 and about 50% have metabolic acidemia. Other late findings include elevation of amylase levels in the serum and peritoneal fluid, serum D-lactate levels, and bacteremia as a result of bacterial translocation across compromised bowel [16] [17] . There is no sensitive or specific marker for the diagnosis of mesenteric ischemia before infarction develops. A small study recently analyzed serum D-dimer levels in patients suspected of having acute bowel ischemia and found them to be elevated in all patients with AMI, regardless of the cause [18] . Further studies are necessary to evaluate whether serum D-dimer levels can be useful as a marker for early ischemia.

Plain films of the abdomen usually are normal early in ischemia. In one series, 6 (26%) of 23 patients with proved AMI had normal abdominal radiographs [19] . As ischemia progresses, patients may develop formless loops of bowel, ileus, or thickening of the bowel wall with “thumbprinting,” suggestive of submucosal edema or hemorrhage. Any

radiologic abnormalities must be considered as relatively late signs of AMI and portend a poor outcome.

Duplex ultrasonography may be useful to assess flow in the SMA and the portal vein and can detect thrombosis and occlusion of these vessels, but is limited in the emergent evaluation of patients with acute abdominal pain [20] . Only the proximal portions of the major vessels can be evaluated consistently; the peripheral branches cannot be well visualized [21] [22] . Furthermore, patients with NOMI may have normal duplex results despite significant vasoconstriction.

Computed tomography (CT) scanning has been helpful in diagnosing AMI. Unfortunately, however, as with other radiographic methods, the early signs on CT scans are relatively nonspecific, and the late signs indicate necrotic bowel [23] . A retrospective study of CT scan results in patients with proved intestinal infarction showed specific findings in 39% of patients and nonspecific abnormalities in 35% of patients [19] . Other studies on the value of CT scanning in suspected AMI have demonstrated sensitivities in the range of 26% to 64% [24] [25] . Early signs include bowel wall thickening and luminal dilation. Highly suggestive, but uncommon, late signs include pneumatosis (gas in the bowel wall) and mesenteric or portal venous gas, both of which typically indicate necrotic bowel [26] [27] .

Contrast-enhanced CT scanning is the procedure of choice for diagnosing acute MVT [28] [29] . Lack of opacification of the mesenteric veins after injection of contrast is indicative of a clot in the mesenteric vein. Other findings include a central lucency in the lumen of the superior mesenteric vein (SMV), thickening of the bowel wall, or dilated collaterals in a thickened mesentery.

Magnetic resonance angiography with gadolinium has been studied in patients with suspected mesenteric ischemia [30] . Like duplex ultrasonography and CT scans, it was found to be useful in evaluating the origins of the celiac axis and SMA, but was limited in its evaluation of the IMA and peripheral branches of the splanchnic vessels. As with the other imaging modalities it also may be falsely negative in NOMI.

Direct imaging of the splanchnic vasculature by mesenteric angiography is the mainstay of diagnosis in both occlusive and nonocclusive forms of AMI [31] [32] [33] [34] [35] . Opponents of routine angiography for patients with suspected AMI note that it is often quite difficult to perform the test on critically ill patients and that it may contribute to delays in surgery. These are legitimate arguments, but several overriding factors exist. First, if an early diagnosis is not made (ie, before intestinal infarction), the mortality rate is usually between 70% and 90%; decreased mortality rate has been demonstrated clearly in series in which routine angiography has been used [33] [34] [36] [37] . Second, angiography has a very high sensitivity (74% to 100%) and specificity (100%) with minimal complications. It can diagnose AMI and differentiate between occlusive and nonocclusive forms of AMI in most patients. Third and possibly most important, treatment by infusion of vasodilators or thrombolytic agents, which have been shown to improve outcome, can be instituted at the same time as the diagnostic study.

All agree that prompt exploratory laparotomy is indicated in patients with suspected AMI in whom angiography is not immediately available. Laparotomy without prior angiography may be indicated in unstable patients with peritoneal signs.

Treatment

All patients with suspected AMI should initially undergo volume resuscitation; correction of hypotension, congestive heart failure, and cardiac arrhythmias; and infusion of broad-spectrum antibiotics. Antibiotics offer theoretic protection against bacterial translocation, which has been shown to occur with the loss of mucosal integrity [38] [39] . Although no controlled studies have been done to provide evidence of clinical benefit, several old experimental studies showed reduction in the severity and extent of bowel damage when antibiotics were given before or during an ischemic event [40] [41] [42] . Plain films or CT scans of the abdomen should be performed to exclude other causes of abdominal pain. If no alternative diagnosis is made on these studies then selective SMA angiography should be performed. Based on the angiographic findings the patient should be treated according to the specific cause of the ischemia [43] .

SMAE

Superior mesenteric artery emboli are responsible for approximately 50% of AMI events. These emboli typically originate from the heart and lodge at points of normal anatomic tapering, usually just distal to the origin of a major branch. Angiography may reveal one or more filling defects with partial or complete obstruction to flow.

Various therapeutic approaches haven been proposed for SMAE depending on whether the embolus is in the SMA above the origin of the ileocolic artery (major embolus) or more distally in the SMA or one of its branches (minor embolus); whether it is completely or only partially occluding; and whether or not the ischemic event is associated with signs of peritonitis (Fig. 1).

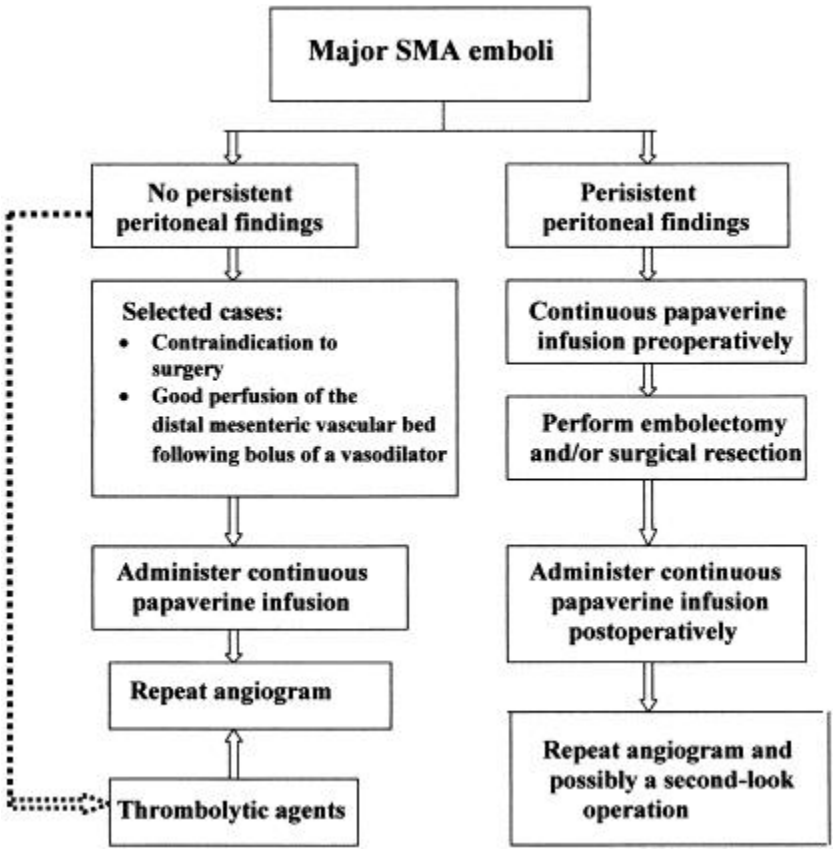

Fig. 1. Algorithm for the management of major SMA emboli. (*From* Brandt LJ, Boley SJ. AGA technical review on intestinal ischemia. Gastroenterology 2000;118:954; with permission.)

In the absence of peritoneal signs, infusions of vasodilators and thrombolytics have been used with success. There is ample evidence that an embolus in the SMA induces profound vasoconstriction of both the obstructed and unobstructed branches of the SMA. If not corrected promptly such vasoconstriction can become irreversible and persist following removal of the embolus and explains the recurrent bowel necrosis that occurs after surgical embolectomy. Relief of mesenteric vasoconstriction, in the authors' opinion, plays an essential role in the treatment of AMI from mesenteric emboli. Vasodilation usually is accomplished with infusion of papaverine, a potent vasodilator, administered directly through the angiography catheter as a continuous drip at a rate of 30 to 60 mg/h (Fig. 2).

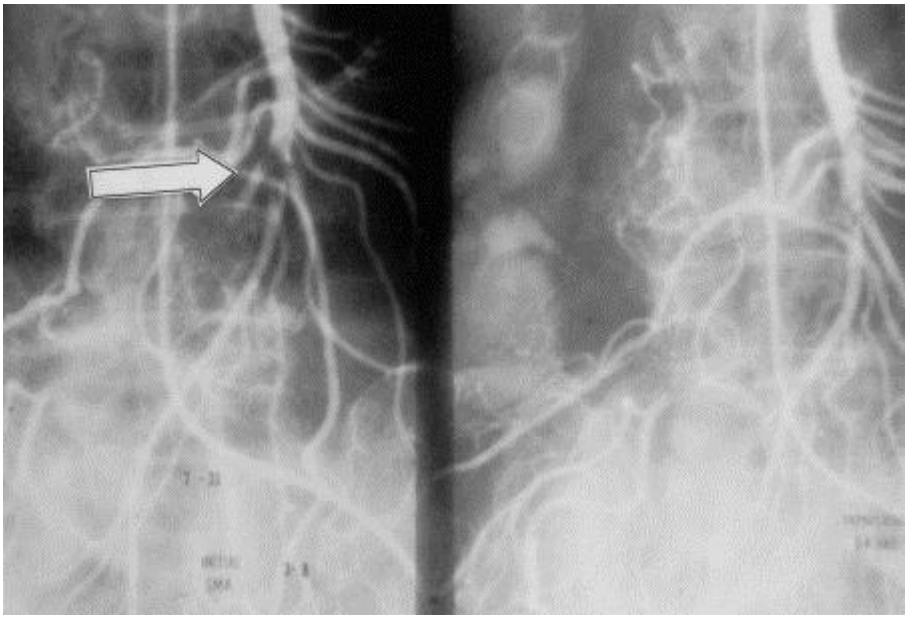

Fig. 2. (*A*) SMA angiogram from a 71-year-old man with abdominal pain shows an embolus occluding the SMA at the level of the origin of the right colic artery (*arrow*). Vasoconstriction is noted distal to the embolus. (*B*) Repeat angiogram 24 hours after SMA embolectomy and preoperative and postoperative papaverine infusions into the SMA. Vasodilation is seen, and all vessels are patent except for a distal jejunal branch. (*From* Brandt LJ, Boley SJ. Intestinal ischemia. In: Feldman M, Friedman LS, Sleisenger MH, editors. Sleisenger and Fordtran's gastrointestinal and liver disease. Philadelphia: WB Saunders, 2002. p. 2321; with permission.)

Although not accepted as standard treatment, infusion of papaverine has been used as the sole therapy in patients with minor emboli [44] and also has been used alone in selected patients with major emboli who are high-risk surgical candidates, do not have peritoneal signs, and have good reperfusion of the vascular bed distal to the obstruction after a bolus of vasodilator. Intra-arterial papaverine is routinely used in the authors' institution as an integral part of the preoperative, intraoperative, and postoperative management of patients with AMI and has been infused for up to 5 days without complication. Thrombolytic agents, including streptokinase, urokinase, and recombinant tissue plasminogen activator, have been used with variable success in selected cases [45] [46] [47] [48] [49] [50] . Thrombolytic therapy is most likely to be successful when the thrombus is a minor one (ie, distal to the origin of the ileocolic artery); when it is only partially occluding; and when it is given within 12 hours of the onset of symptoms.

There is uniform agreement that laparotomy is mandatory when signs of peritonitis are present, with the intent to restore intestinal blood flow and resect necrotic bowel. Restoration of blood flow, either with embolectomy or arterial bypass, precedes evaluation of intestinal viability because bowel that may initially appear infarcted may recover entirely once flow is restored. Only bowel that is clearly necrotic is resected at surgery, and a primary anastomosis is performed. If portions of the bowel are of questionable viability they should be left alone, and a re-exploration, or second-look

performed within 12 to 24 hours [51] . This 12- to 24-hour period is used to maximize medical therapy (eg, blood volume correction, antibiotics, and intra-arterial vasodilation). Recent reports have advocated the use of second-look laparoscopy as a safe alternative to laparotomy in critically ill patients [52] [53] .

NOMI

NOMI is responsible for 20% to 30% of cases of AMI and results from mesenteric vasoconstriction following hypoperfusion of the gut [54] [55] . Hypoperfusion may be precipitated by congestive heart failure; cardiac arrhythmia; shock; or large volume shifts, such as occur during hemodialysis. The ischemia may not manifest itself for several hours or days following the precipitating event, but is believed to be caused by persistent splanchnic vasoconstriction, even after the initial event has resolved (Fig. 3).

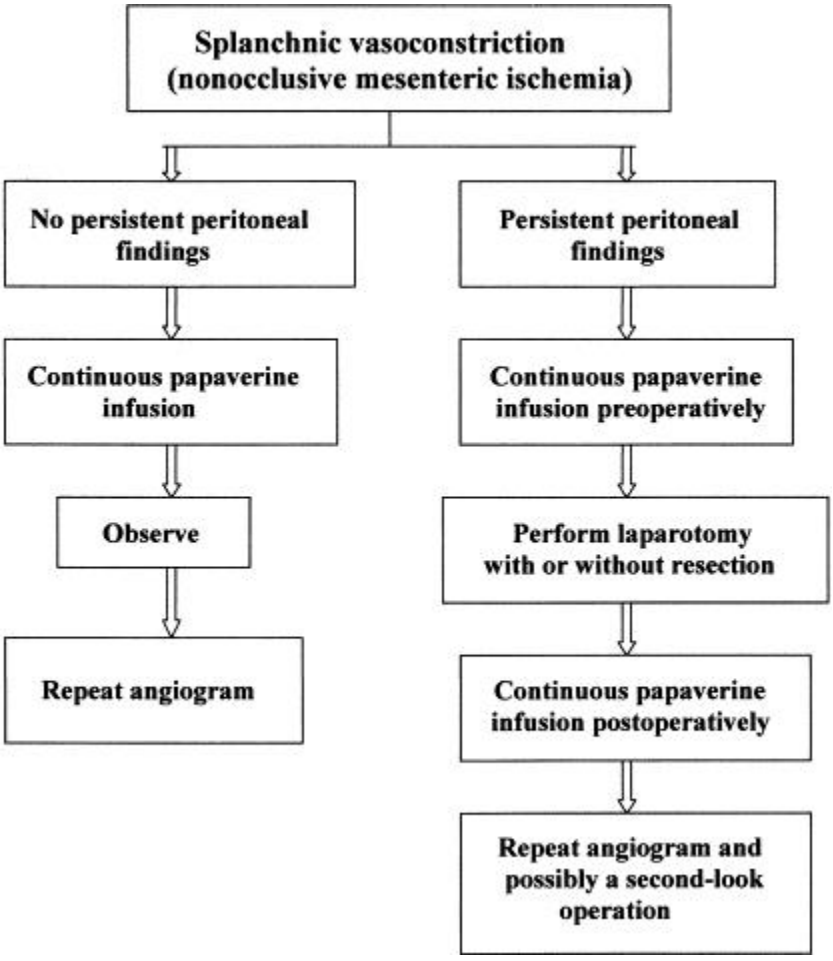

Fig. 3. Algorithm for the management of nonocclusive mesenteric ischemia. (*From* Brandt LJ, Boley SJ. AGA technical review on intestinal ischemia. Gastroenterology 2000;118:954; with permission.)

Angiography is the only way to diagnose NOMI before infarction occurs and there are four criteria used for this purpose: (1) narrowing of the origins of the SMA branches, (2) irregularities in these branches, (3) spasm of the mesenteric arcades, and (4) impaired filling of the intramural vessels [31] [56] .

Once NOMI has been identified, papaverine infusion should be initiated to relieve the vasoconstriction and prevent further ischemic damage (Fig. 4). In series where papaverine was used following angiography the mortality rate was reduced from historical levels of 70% to 90% to 0% to 55% [33] [36] [57] . The infusion should be maintained and angiography should be repeated daily until there is no further evidence, clinically and roentgenographically, of persistent vasoconstriction.

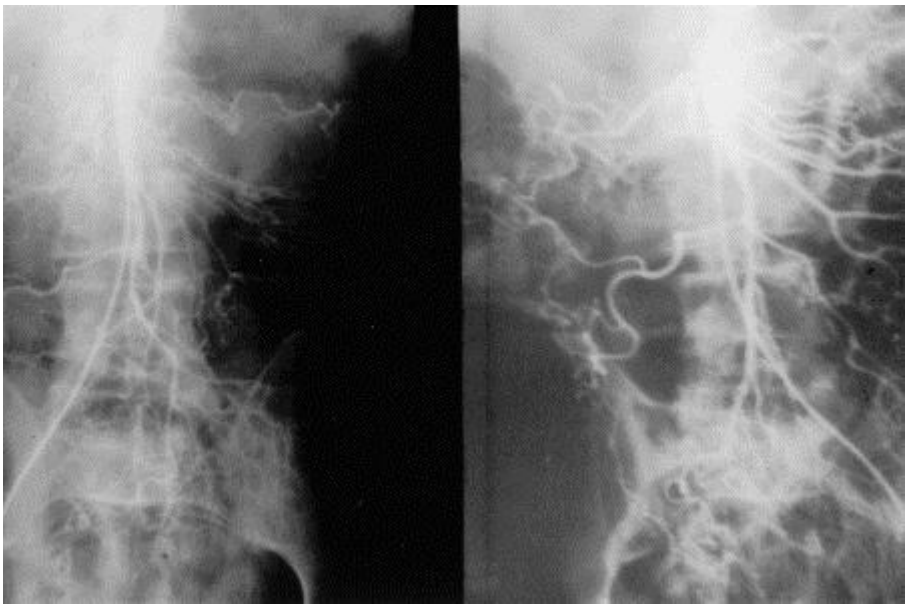

Fig. 4. Patient with nonocclusive mesenteric ischemia following an episode of gastrointestinal hemorrhage and shock. (*A*) Initial SMA angiogram showing diffuse vasoconstriction. (*B*) Repeat angiogram performed after 48 hours of papaverine infusion showing marked vasodilation. (*From* Brandt LJ, Boley SJ. Ischemic intestinal syndromes. Adv Surg 1981;15:1; with permission.)

Patients who present with peritoneal signs should undergo laparotomy with resection of necrotic bowel. Papaverine should continue to be infused before, during, and after surgery. Liberal preservation of questionable bowel with frequent re-explorations should be used, because potentially viable bowel may improve dramatically with papaverine infusion, limiting the need for more extensive intestinal resections. In a series reported from the authors' institution 75% of patients who survived AMI had less than 1 m of bowel resected [32] .

SMAT

Acute SMAT occurs in patients with severe atherosclerosis who often have a history of prior myocardial infarction, cerebrovascular accident, or claudication; most have a history of chronic abdominal angina for weeks to months before the acute ischemic event (Fig. 5).

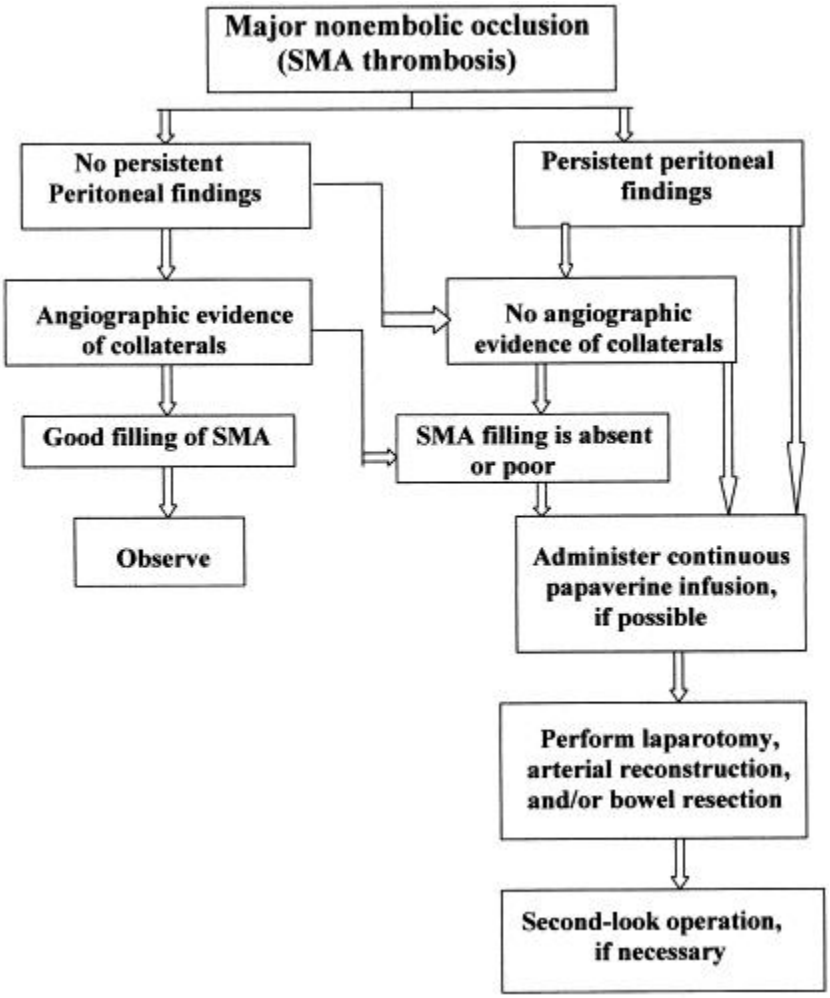

Fig. 5. Algorithm for the management of SMA thrombosis. (*From* Brandt LJ, Boley SJ. AGA technical review on intestinal ischemia. Gastroenterology 2000;118:954; with permission.)

SMAT is best demonstrated on aortography, which usually shows an occlusion of the SMA 1 to 2 cm from its origin. Branches distal and proximal to the thrombosis may demonstrate vasoconstriction. Some distal filling of the SMA by collaterals commonly is seen. It is important to determine whether the occlusion is acute or chronic; the absence of collaterals points to an acute SMAT and necessitates immediate intervention. Although papaverine infusion may help decrease acute vasoconstriction, surgical revascularization generally is recommended.

Papaverine infusion should be started immediately and emergency surgical revascularization should be attempted. Thrombolytic therapy, percutaneous angioplasty, and the use of intravascular stents have been attempted in selected cases. Because the angle of the origin of the SMA from the aorta varies, placement of the angioplasty catheter and stent deployment may be technically difficult. Restenosis rates range from 25% to 50% in a few limited series [58] [59] .

The use of anticoagulation in patients with AMI remains controversial; although heparin generally is regarded as a beneficial intervention there is debate as to the timing of its initiation. Although the authors have recommended a delay of 48 hours because of the risk of intraluminal bleeding from damaged bowel, other investigators believe that immediate heparinization may offset that danger [60] . Some authorities recommend immediate anticoagulation if no infarction is present, but delay anticoagulation if intestinal infarction is present [61] . No good data exist to support any of these approaches.

Acute MVT

MVT is an infrequent condition, occurring in 5% to 10% of patients with AMI [62] [63] , although with the advent and frequent use of CT scanning is the most increasingly diagnosed cause of intestinal ischemia. It has long been regarded as an imitator of other abdominal disorders, in part because its symptoms may be so nonspecific. In the past, a cause of MVT was found in fewer than half of the cases but multiple predisposing conditions now have been identified and a cause can be determined in 80% to 90% of cases (Box 1) [64] .

Box 1 Conditions associated with MVT

- Hematologic and hypercoagulable states

  Sickle cell anemia
  Polycythemia vera
  Thrombocytosis
  Antithrombin III deficiency
  Protein C or S deficiency
  Factor V Leiden mutation (activated protein C resistance)
  Lupus anticoagulant
  Factor II 20210A mutation
  Neoplasms or carcinomatosis
  Migratory thrombophlebitis
  Peripheral deep vein thrombosis
  Pregnancy
  Local venous congestion and stasis
  Hepatic cirrhosis
  Congestive splenomegaly
  Compression of portal venous radicals by tumor

- Intra-abdominal inflammation and sepsis

  Cholangitis
  Pancreatitis
  Diverticulitis
  Appendicitis
  Peritonitis
  Inflammatory bowel disease
  Pelvic or intra-abdominal abscess

- Parasitic infection

  Ascaris lumbricoides

- Blunt abdominal trauma
- Decompression sickness
- Iatrogenic

  Abdominal operations (especially splenectomy and pancreatectomy)
  Sclerotherapy of esophageal varices followed by vasopressin infusion
  Estrogens (oral contraceptives)

From Greenwald DA, Brandt LJ, Reinus JF. Ischemic bowel disease in the elderly. Gastroenterol Clin North Am 2001; 30:445; with permission.

The location of a thrombus within the mesenteric venous circulation is closely related to its cause. Thrombosis from hypercoagulable states tends to originate in the smaller venous branches and progresses into the major trunks. Thrombosis secondary to cirrhosis, neoplasm, or operative injury begins at the site of obstruction and extends peripherally.

Abdominal pain is a cardinal feature of acute MVT, occurring in 90% of patients. The pain often can be tolerated by the patient and may be present for a mean of 5 to 14 days before the patient seeks medical attention; 25% of patients may have pain for more than 30 days [65] . Other symptoms include nausea and vomiting in 60% to 70% of patients and an alteration in bowel habits (diarrhea or constipation) in 30%. Fecal occult blood is found in more than 50% of patients with MVT. The presentations of patients with MVT are often more variable than those of patients with arterial causes of AMI.

The absence of reliable specific symptoms, signs, or laboratory studies in patients with MVT makes the diagnosis difficult. An abdominal CT scan with intravenous contrast is the diagnostic test of choice, enabling the diagnosis in more than 90% of patients [66] [67] . CT scan may demonstrate a central lucency in the lumen of the SMV, enlargement of the SMV, thickening of the bowel wall, or dilated collaterals in a thickened mesentery. When MVT is diagnosed on CT scan, angiography may not be necessary. In selected patients it better delineates thrombosed veins and provides access for intra-arterial vasodilators. Angiographic findings include thrombus in the SMV with partial or complete occlusion, failure to visualize the SMV or portal vein, slow or absent filling of the mesenteric veins, failure of the arterial arcades to empty, and prolonged blush in the involved segment [36] [68] .

In symptomatic patients in whom the diagnosis of MVT has been made by CT scan or angiography, treatment depends on the presence or absence of peritoneal signs (Fig. 6). In patients with signs of peritonitis, an emergent laparotomy is mandated with resection of necrotic bowel [69] [70] . If bowel viability is questionable, papaverine may be infused into the SMA to relieve any associated arterial spasm that may be contributing to the ischemic injury. Mesenteric venous thrombectomy may also have a role in selected cases. A second-look operation should then be performed 12 to 24 hours after the first operation [71] .

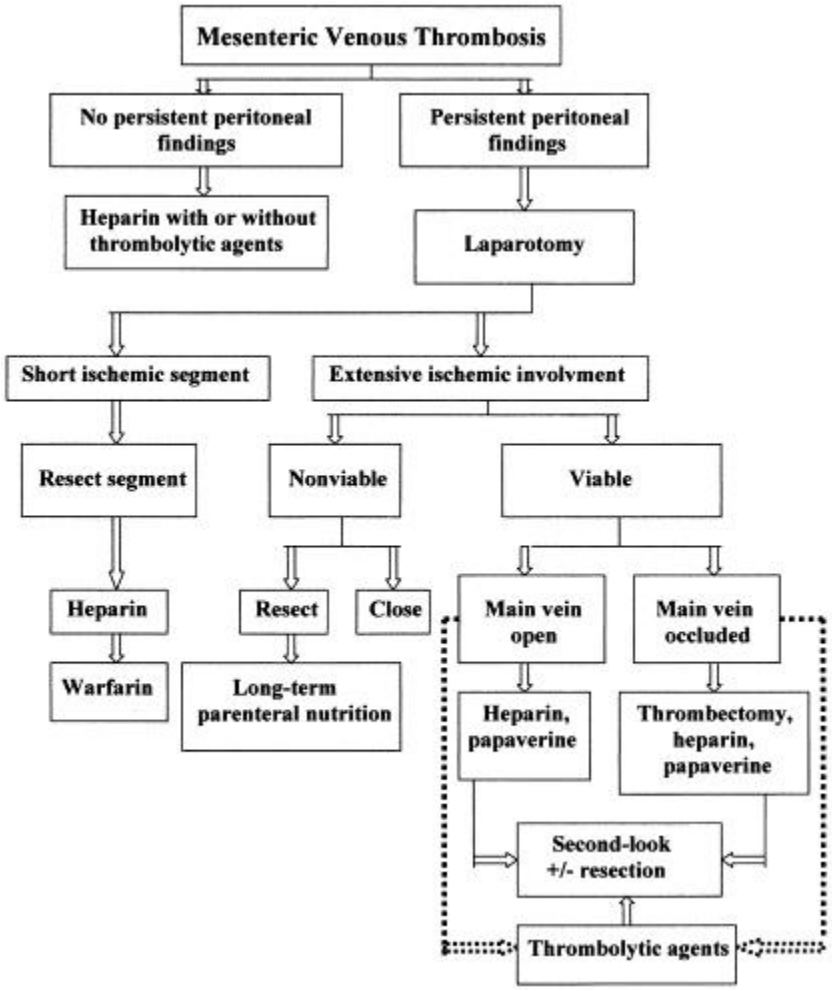

Fig. 6. Algorithm for the management of mesenteric venous thrombosis. (*From* Brandt LJ, Boley SJ. AGA technical review on intestinal ischemia. Gastroenterology 2000;118:954; with permission.)

Acute MVT is the one form of mesenteric ischemia in which anticoagulation should be used routinely following surgery [72] . Immediate heparinization for 7 to 10 days has been shown to decrease thrombus recurrence and progression and to improve survival. In patients who receive heparin the recurrence rate is lowered from 25% to 13% and mortality is reduced from 50% to 13% [73] . Warfarin should then be administered for 3 to 6 months.

In patients with abdominal pain but without peritoneal signs, who have evidence of an SMV thrombus on CT scan, immediate anticoagulation with heparin followed by a 3- to 6-month course of warfarin may be all that is necessary [70] . These patients do need close clinical observation, and surgery is indicated if signs of peritonitis develop.

Occasionally, an SVT thrombus is discovered incidentally in asymptomatic patients who undergo a CT scan for another reason besides abdominal pain. A 3- to 6-month course of

warfarin may be reasonable in these patients, especially if a predisposing condition can be identified (eg, hypercoagulable state or deep vein thrombosis) that might warrant anticoagulation.

Summary

Ischemic injury to the gastrointestinal tract can threaten bowel viability with potential catastrophic consequences, including intestinal necrosis and gangrene. The presenting symptoms and signs are relatively nonspecific and diagnosis requires a high index of clinical suspicion. AMI often results from an embolus or thrombus within the SMA, although a low-flow state through an area of profound atherosclerosis may also induce severe ischemia. Because most laboratory and radiologic studies are nonspecific in early ischemia an aggressive approach to diagnosis with imaging of the splanchnic vasculature by mesenteric angiography is advocated. Various therapeutic approaches, including the infusion of vasodilators and thrombolytics, may then be used. Proper diagnosis and management of patients with AMI requires vigilance and a readiness to pursue an aggressive course of action.

References

[1]. Brandt LJ, Boley SJ, Goldberg L, et al. Colitis in the elderly: a reappraisal. Am J Gastroenterol 1981;76:239. Abstract

[2]. Brandt LJ, Boley SJ. Colonic ischemia. Surg Clin North Am 1992;72:203. Abstract

[3]. Brandt LJ, Boley SJ. Colonic ischemia. In: BrandtLJ, editors. Clinical practice of gastroenterology Philadelphia: Current Medicine; 1999. p. 696.

[4]. Michels N. Blood supply and anatomy of the upper abdominal organs, with a descriptive atlas. Philadelphia: JB Lippincott; 1955.

[5]. Kornblith PL, Boley SJ, Whitehouse BS. Anatomy of the splanchnic circulation. Surg Clin North Am 1992;72:1. Abstract

[6]. Kaleya R, Sammartano R, Boley SJ. An aggressive approach to acute mesenteric ischemia. Surg Clin North Am 1992;72:157. Abstract

[7]. Smithline AE, Brandt LJ, Boley SJ. Acute and chronic mesenteric ischemia. In: BrandtLJ, editors. Clinical practice of gastroenterology Philadelphia: Current Medicine; 1999. p. 574.

[8]. Brandt LJ, Boley SJ. Intestinal ischemia. In: FeldmanM, FriedmanLS, SleisengerMH, editors. Sleisenger and Fordtran's gastrointestinal and liver disease Philadelphia: WB Saunders; 2002. p. 2321.

[9]. Finucani PM, Arunachalam T, O'Dowd J, et al. Acute mesenteric infarction in elderly patients. J Am Geriatr Soc 1989;37:355. Abstract

[10]. Gennaro M, Ascer E, Matano R, et al. Acute mesenteric ischemia after cardiopulmonary bypass. Am J Surg 1993;166:2321. Abstract

[11]. Han SY, Kwon YJ, Shin JH. Nonocclusive mesenteric ischemia in a patient on maintenance hemodialysis. Korean J Intern Med 2000;15:81. Abstract

[12]. Osorio J, Farreras N, Ortiz De Zarate L, et al. Cocaine-induced mesenteric ischemia. Dig Surg 2000;17:648. Abstract

[13]. Rice E, DiBaise JK, Quigley EM. Superior mesenteric artery thrombosis after colonoscopy. Gastrointest Endosc 1999;50:706. Full Text

[14]. McGovern RP, Franco RA. Acute mesenteric ischemia after colonoscopy. Am J Gastroenterol 1995;90:170. Full Text

[15]. Andrei VE, Schein M, Wise L. Small bowel ischemia following laparoscopic cholecystectomy. Dig Surg 1999;16:522. Abstract

[16]. Kurland B, Brandt LJ, Delany H. Diagnostic tests for intestinal ischemia. Surg Clin North Am 1992;72:85. Abstract
[17]. Murray M, Gonze M, Nowak L, et al. Serum D(-) lactate levels as an aid to diagnosing acute intestinal ischemia. Am J Surg 1994;72:85.
[18]. Acosta S, Nilsson TK, Bjork M. Preliminary study of D-dimer as a possible marker of acute bowel ischaemia. Br J Surg 2001;88:385. Abstract
[19]. Smerud M, Johnson C, Stephens D. Diagnosis of bowel infarction: a comparison of plain films and CT scans in 23 cases. AJR Am J Roentgenol 1990;154:99. Abstract
[20]. Moneta GL, Yeager RA, Dalman R, et al. Duplex ultrasound criteria for the diagnosis of splanchnic artery stenosis or occlusion. J Vasc Surg 1991;14:511. Abstract
[21]. Harward T, Smith S, Seeger J. Detection of celiac axis and superior mesenteric artery occlusive disease with use of abdominal duplex scanning. J Vasc Surg 1993;17:738. Abstract
[22]. Bowersox JC, Zwolak RM, Walsh DB, et al. Duplex ultrasonography in the diagnosis of celiac and mesenteric artery occlusive disease. J Vasc Surg 1991;14:780. Abstract
[23]. Bartnicke B, Balfe D. CT appearance of intestinal ischemia and intramural hemorrhage. Radiol Clin North Am 1994;32:845. Abstract
[24]. Alpern MB, Glazer GM, Francis IR. Ischemic or infarcted bowel: CT findings. Radiology 1988;166:149. Abstract
[25]. Taourel PG, Deneuville M, Pradel JA, et al. Acute mesenteric ischemia: diagnosis with contrast-enhanced CT. Radiology 1996;199:632. Abstract
[26]. Hashimoto A, Fuke H, Shimizu A, et al. Hepatic portal venous gas caused by non-obstructive mesenteric ischemia. J Hepatol 2002;37:870. Citation
[27]. van den Hauwe L, Degryse H, Coene L. Portomesenteric vein gas in mesenteric infarction. JBR-BTR 2002;85:162. Citation
[28]. Rosen A, Korobkin M, Silverman P, et al. Mesenteric vein thrombosis: CT identification. AJR Am J Roentgenol 1984;143:83. Abstract
[29]. Boley SJ, Kaleya RN, Brandt LJ. Mesenteric venous thrombosis. Surg Clin North Am 1992;72:183. Abstract
[30]. Meaney JF, Prince MR, Nostrant TT, et al. Gadolinium-enhanced MR angiography of visceral vessels in patients with suspected chronic mesenteric ischemia. J Magn Reson Imaging 1997;7:171. Abstract
[31]. Bakal C, Sprayregen S, Wolf E. Radiology in intestinal ischemia: angiographic diagnosis and management. Surg Clin North Am 1992;72:125. Abstract
[32]. Boley SJ, Sprayregen S, Veith FJ, et al. An aggressive roentgenologic and surgical approach to acute mesenteric ischemia. In: NyhusLM, editors. Surgery annual New York: Appleton-Century-Crofts; 1973. p. 355.
[33]. Boley SJ, Sprayregen S, Siegelman SS, Veith FJ. Initial results from an aggressive approach to acute mesenteric ischemia. Surgery 1977;82:848. Abstract
[34]. Boos S. Angiography of the mesenteric artery 1976 to 1991: a change in the indications during mesenteric circulatory disorders. Radiologe 1992;32:154. Abstract
[35]. Bradbury AW, Brittenden J, McBride K, et al. Mesenteric ischemia: a multidisciplinary approach. Br J Surg 1995;82:1446. Abstract
[36]. Clark RA, Gallant TE. Acute mesenteric ischemia: angiographic spectrum. Am J Radiol 1984;142:555.

[37]. Czerny M, Trubel W, Claeys L, et al. Die acute mesenteriale ischamie. Zentralbl Chir 1997;122:538. Abstract
[38]. Bennion RS, Williams RA. Early portal anaerobic bacteremia in mesenteric ischemia. Arch Surg 1984;119:151. Abstract
[39]. Redan JA, Rush BF, Lysz TW, et al. Organ distribution of gut-derived bacteria caused by bowel manipulation or ischemia. Am J Surg 1990;159:85. Abstract
[40]. Sarnoff SJ, Fine J. Effect of chemotherapy on ileum subjected to vascular injury. Ann Surg 1945;121:74.

[41]. Poth EJ, McClure JN. Intestinal obstruction: protective action of sulfasuxidine and sulfathalidine to ileum following vascular damage. Ann Surg 1950;131:159.
[42]. Cohen I, Floyd CE, Dresden CF, et al. Strangulation obstruction in germ-free animals. Ann Surg 1962;156:692.
[43]. Brandt LJ, Boley SJ. AGA technical review on intestinal ischemia. American Gastrointestinal Association. Gastroenterology 2000;118:954. Full Text
[44]. Boley SJ, Feinstein FR, Sammartano R, et al. New concepts in the management of superior mesenteric artery embolus. Surg Gynecol Obstet 1981;153:561. Abstract
[45]. Badiola CM, Scoppetta DJ. Rapid revascularization of an embolic superior mesenteric artery occlusion using pulse-spray pharmacomechanical thrombolysis with urokinase. AJR Am J Roentgenol 1997;169:55. Citation
[46]. Kwauk ST, Bartlett JH, Hayes P, et al. Intra-arterial fibrinolytic treatment for mesenteric arterial embolus: a case report. Can J Surg 1996;39:163. Abstract
[47]. Regan F, Karlstad RR, Magnusan TH. Minimally invasive management of acute superior mesenteric artery occlusion: combined urokinase and laparoscopic therapy. Am J Gastroenterol 1996;91:1019. Full Text
[48]. Gallego AM, Ramirez P, Rodriguez JM, et al. Role of urokinase in the superior mesenteric artery embolism. Surgery 1996;120:111. Citation
[49]. Simo G, Echenaguisia AJ, Camunez F, et al. Superior mesenteric arterial embolism: local fibrinolytic treatment with urokinase. Radiology 1997;20:775.
[50]. McBride KD, Gaines PA. Thrombolysis of a partially occluding superior mesenteric artery thromboembolus by infusion of streptokinase. Cardiovasc Intervent Radiol 1994;17:164. Abstract
[51]. Shaw RS. The “second-look” after superior mesenteric arterial embolectomy or reconstruction for mesenteric infarction. In: EllisonEH, FriesenJR, KulhollandJH, editors. Current surgical management Philadelphia: WB Saunders; 1965. p. 509.
[52]. Seshadri PA, Poulin EC, Mamazza J, et al. Simplified laparoscopic approach to “second-look” laparotomy: a review. Surg Laparosc Endosc Percutan Tech 1999;9:286. Abstract
[53]. Zamir G, Reissman P. Diagnostic laparoscopy in mesenteric ischemia. Surg Endosc 1998;12:390. Citation
[54]. Trompeter M, Brazda T, Remy CT, et al. Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy. Eur Radiol 2002;12:1179. Abstract
[55]. Anantharaju A, Van Thiel DH. Nonocclusive mesenteric ischemia: reality. J Gastroenterol 2002;37:876. Citation
[56]. Siegelmann SS, Sprayregen S, Boley SJ, et al. Angiographic diagnosis of mesenteric arterial vasoconstriction. Radiology 1974;122:533. Citation
[57]. Ward D, Vernava AM, Kaminski DL, et al. Improved outcome by identification of high-risk nonocclusive mesenteric ischemia, aggressive re-exploration, and delayed anastomosis. Am J Surg 1995;170:5777. Abstract
[58]. Hallisey M, Deschaine J, Illescas F, et al. Angioplasty for the treatment of visceral ischemia. J Vasc Intervent Radiol 1995;6:785.
[59]. VanDeinse W, Zawacki J, Phillips D. Treatment of acute mesenteric ischemia by percutaneous transluminal angioplasty. Gastroenterology 1986;91:475. Abstract
[60]. Bergan JJ. Acute mesenteric ischemia. In: HaimoviciH, editors. Vascular emergencies New York: Appleton-Century Crofts; 1982. p. 553.
[61]. Batellier J, Kieny R. Superior mesenteric artery embolism: eighty-two cases. Ann Vasc Surg 1990;4:112. Abstract
[62]. Greenwald DA, Brandt LJ, Reinus JF. Ischemic bowel disease in the elderly. Gastroenterol Clin North Am 2001;30:445. Full Text
[63]. Harward RTS, Green D, Bergan JJ, et al. Mesenteric venous thrombosis. J Vasc Surg 1989;9:328. Abstract
[64]. Rhee RY, Globviczki P, Mendonca CT, et al. Mesenteric venous thrombosis: still a lethal disease in the

1990s. J Vasc Surg 1994;20:688. Abstract
[65]. Sack J, Aldrete JS. Primary mesenteric venous thrombosis. Surg Gynecol Obstet 1982;154:205. Abstract
[66]. Matos C, Van Gansbeke D, Zaleman M, et al. Mesenteric venous thrombosis: early CT and ultrasound diagnosis and conservative management. Gastrointest Radiol 1986;11:322. Abstract
[67]. Clavien PA, Huber O, Mirescu D, et al. Contrast-enhanced CT scan as a diagnostic procedure in mesenteric ischemia due to mesenteric venous thrombosis. Br J Surg 1989;76:93. Citation
[68]. Zemen RK, Siverman PM, Vieco PT, et al. CT angiography. AJR Am J Roentgenol 1995;165:1079. Abstract
[69]. Kaleya RN, Boley SJ. Mesenteric venous thrombosis. In: NajarianJS, DelaneyJP, editors. Progress in gastrointestinal surgery Chicago: Year Book; 1989. p. 417.
[70]. Chong AK, So JB, Ti TK. Use of laparoscopy in the management of mesenteric venous thrombosis. Surg Endosc 2001;15:1042. Abstract
[71]. Levy P, Krausz MM, Manny J. The role of second-look procedure in improving survival time for patients with mesenteric venous thrombosis. Surg Gynecol Obstet 1990;170:287. Abstract
[72]. Petitti DB, Strom BL, Melmon KL. Duration of warfarin anticoagulant therapy and the probabilities of recurrent thromboembolism and hemorrhage. Am J Med 1986;81:255. Abstract
[73]. Grieshop RJ, Dalsing MC, Cikrit DF, et al. Acute mesenteric venous thrombosis: revisited in a time of diagnostic clarity. Am Surg 1991;57:573. Abstract