Acute mesenteric venous thrombosis: Case for nonoperative management

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Objective: **Initial treatment in the management of acute mesenteric vein thrombosis (MVT) is controversial. Some authors have proposed a surgical approach, whereas others have advocated medical therapy (anticoagulation). In this study, we analyzed and compared the results obtained with surgical and medical treatment to determine the best initial management for this disease.**

Methods: **We retrospectively reviewed the records of patients treated for MVT in a secondary care surgical department from January 1987 to December 1999. Before January 1995, our departmental policy was to perform surgery in patients with suspected MVT. Since January 1995, we have preferred a medical approach when achievable. Each patient in this study was assessed for diagnosis, initial management (laparotomy or anticoagulation), morbidity, mortality, duration of hospitalization, the need for secondary operation, portal hypertension, and survival rates.**

Results: **Twenty-six patients were treated, 14 before January 1995 (group 1) and 12 since January 1995 (group 2). Morbidity, mortality, secondary operation, portal hypertension, and 2-year survival rates were 34.6%, 19.2%, 15.3%, 19.2%, and 76.9%, respectively. No statistical difference was observed between the two groups. The mean duration of hospitalization was 51.6 days in group 1 and 23.2 days in group 2 ($P < .05$). Among the 12 patients treated by means of laparotomy with bowel resection, 10 patients (83%) had mucosal necrosis without transmural necrosis at pathologic study.**

Conclusion: **Nonoperative management for acute MVT is feasible when the initial diagnosis with a computed tomography scan is certain and when the bowel infarction has not led to transmural necrosis and bowel perforation. The morbidity, mortality, and survival rates are similar in cases of surgical and nonoperative management. The length of hospital stay is shorter when patients are treated with a nonoperative approach. A nonoperative approach, when indicated, avoids the resection of macroscopically infarcted small bowel (without transmural necrosis) in cases that are potentially reversible with anticoagulation alone. (J Vasc Surg 2001;34:673-9.)**

Mesenteric venous thrombosis (MVT) is a rare but potentially lethal form of mesenteric ischemia. MVT must be distinguished from arterial and nonocclusive types of mesenteric ischemia, and accounts for 5% to 15% of all cases of mesenteric ischemia. Patients may have evocative signs, such as abdominal pain, nausea, or vomiting. However, a clinical diagnosis is often difficult because abdominal symptoms are nonspecific.[1] Primary MVT accounted for 25% to 55% of cases in early studies, but recent reports show a decline in primary MVT because of improvements in the diagnosis of hypercoagulable states.[2] Advances in new imaging techniques also have enabled early recognition of this disease without or before laparotomy.[3-5] There is no consensus about initial treatment in the management of MVT. Some authors have proposed an aggressive surgical approach,[6] whereas others have advocated medical therapy.[7]

Before January 1995, our department policy was to perform an operation first in patients with suspected MVT for diagnosis and treatment. Since January 1995, we have preferred to use a medical approach for the initial management of MVT, with anticoagulation therapy alone and close follow-up. The aim of this study was to analyze the outcome of patients admitted in our surgical unit for MVT in a 12-year period. The results obtained in each of the two groups in which either surgical or medical therapy was primarily performed during this period were assessed to determine the best management of this uncommon disease.

MATERIALS AND METHODS

We retrospectively reviewed the clinical course of 26 patients treated for acute MVT between January 1, 1987, and December 31, 1999. This group represented 9.2% of the 281 patients treated for mesenteric ischemia, whatever its cause, during the same period in our department. The department is a primary and secondary care surgical department. No patient with acute MVT (as defined) was excluded from the current study. We restricted the diagnosis of acute MVT to patients with symptoms of less than 4 weeks' duration.[4] Patients who incidentally were found to have MVT by means of abdominal computed tomography (CT) scans that had been obtained for other reasons were excluded. Primary MVT was defined as thrombosis of a mesenteric vein that was not associated with any other disease or etiologic factor. The term *secondary MVT* was used for patients with any condition known to predispose them to MVT. These conditions include earlier abdominal surgery, blunt abdominal trauma, oral contraceptive use,

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Table I. Patient characteristics

	All patients	*Group 1*	*Group 2*
No.	26	14	12
Age (y)	55.0 ± 3.6	53.5 ± 4.7	56.7 ± 5.9
Sex			
Men	18	12	6
Women	8	2	6
ASA score > 3 (%)	15	7	25

ASA, American Society of Anesthesiologists.

Table II. Conditions associated with MVT

	All patients	*Group 1*	*Group 2*
Primary (%)	9 (34.6)	8 (57.1)	1 (8.3)
Secondary (%)	17 (65.4)	6 (42.9)	11 (91.7)*
Earlier abdominal surgery	5	1	4
Leiden factor V	3	2	1
Deep venous thrombosis	2	—	2
Oral contraceptive use	2	1	1
Alcohol abuse	1	1	—
Malignant tumor	1	—	1
Anticardiolipin antibodies	1	—	1
Protein S deficiency	1	1	—
Protien C deficiency	1	—	1

*$P < .05$.

inflammation, portal hypertension, or hypercoagulable states.[5]

The diagnosis of acute MVT was established either if there was a positive CT scan result or when the patient underwent a laparotomy. A CT scan was considered to be positive for MVT when a thrombus in the superior mesenteric vein or an abnormal thickening of ischemic bowel wall with streaky mesentery was demonstrated.[4,5] Laparotomy permitted diagnosis of MVT either by means of macroscopic findings after dissection of the superior mesenteric vein (thrombosis) or by means of macroscopic and microscopic pathologic studies of resected small bowel segments. Hematoxylin-eosin–stained sections of each specimen of resected small bowel were reviewed by one of the authors (L.A.) to assess the extent of intramural or transmural bowel necrosis.

Our departmental policy concerning the treatment of patients with clinical suspicion of MVT changed during the period of this study. Before January 1995, patients with the clinical suspicion of MVT underwent an operation as a means of confirming diagnosis and performing treatment (group 1). During this period, when a CT scan was performed at admission and was used as a means of confirming the diagnosis of MVT without peritoneal signs (guarding), patients were treated with anticoagulation alone. Eighty-six percent of the patients in this group were treated surgically. After January 1995, all patients underwent a CT scan at the time of their admission to the hospital (group 2). Patients who had a confirmed diagnosis of MVT by means of the initial CT scan and did not have peritoneal signs (guarding) were treated with anticoagulation alone. Ninety-two percent of the patients in this group were treated with anticoagulation.

In all patients, treatment at admission included intravenous fluid administration and prophylactic antibiotic therapy. Intravenous heparin was given at the time of diagnosis when we decided to use nonsurgical management. Systemic administration of heparin was guided by the activated partial thromboplastin time (APTT), and a minimum of two times the normal APTT value was preferred. Close clinical and hemodynamic follow-up of these patients was undertaken in an intensive care unit. When surgical management was chosen, a laparotomy was performed within 6 hours after the patient's admission. In these cases, intravenous heparin was given immediately at the end of the operation, with the same parameters used in management with anticoagulation alone.[5] After initial treatment (medical or surgical), the systemic administration of heparin was followed in all cases by anti–vitamin K therapy. The presence of coagulation abnormalities was investigated at 3 months. When present, anti–vitamin K therapy was maintained permanently. If no coagulation abnormality was found, anti–vitamin K therapy was maintained for 6 months. The duration of hospitalization was evaluated for both the initial length of hospital stay and the total number of hospitalizations related to MVT (stoma closure, incisional hernia, reoperation). Sepsis was defined as any septic event leading to death. Morbidity,

Table III. Symptoms and findings at admission

	All patients (%)	*Group 1 (%)*	*Group 2 (%)*
Abdominal pain	26 (100)	14 (100)	12 (100)
Diffuse abdominal pain	19 (73)	10 (71.4)	9 (75)
Localized abdominal pain	7 (27)	4 (28.6)	3 (25)
Vomiting	7 (27)	4 (28.6)	3 (25)
Diarrhea	6 (23)	3 (21.4)	3 (25)
Constipation	6 (23)	2 (14.3)	4 (33.3)
Symptoms duration (d)	4.7 ± 0.8	4.8 ± 1.2	4.4 ± 0.8
Symptoms duration ≤ 48 h	9 (34.6)	5 (35.7)	4 (33.3)
Peritoneal signs	8 (30.8)	5 (35.7)	3 (25)
Abdominal tenderness	4 (15)	1 (7)	3 (25)
Guarding	4 (15)	4 (28.6)	0
Abdominal distension	9 (34.6)	6 (42.9)	3 (25)
Blood (digital rectal examination)	8 (30.8)	4 (28.6)	4 (33.3)
Temperature (°C)	37.7 ± 0.1	37.7 ± 0.8	37.8 ± 0.3
Temperature ≥ 38°C	11 (42.3)	6 (42.9)	5 (41.7)
Pulse rate (bpm)	104 ± 3	109 ± 5	98 ± 4
WBC (10^9/L)	18.1 ± 1.3	19.4 ± 6.6	16.7 ± 6.9
Amylases (IU/L)*	61.4 ± 9.3	81.5 ± 15.8	41.2 ± 5.6
Hypotension (< 90 mm Hg)	5 (19.2)	2 (14.3)	3 (25)

*Reference range < 98 IU/L.
WBC, White blood cell count.

mortality, the need for secondary operations, portal hypertension, and survival rates were assessed.

Statistical analysis was performed with Statview software (Abacus Concepts, Berkeley, Calif). The Yates corrected χ^2 test was used as a means of evaluating differences in categoric variables, and the Mann-Whitney *U* test was used for continuous variables. Kaplan-Meier estimates were used as a means of assessing survival, and the log-rank test was used as a means of testing for differences in survival between groups. All values were expressed as a mean ± SE. Statistical significance was accepted when the *P* value was less than .05.

RESULTS

Acute mesenteric vein thrombosis was diagnosed in 18 men and eight women. Their mean age was 55 ± 3.6 years. Fourteen patients (54%) were treated in the first period (group 1), and 12 patients (46%) were treated in the last period (group 2). The mean age, the sex ratio, and the American Society of Anesthesiologists score at admission were similar in the two groups (Table I). Seventeen patients (65.4%) were found to have conditions currently known to predispose them to MVT. Nine patients (34.6%) were considered to have primary MVT. Predisposing conditions associated with secondary mesenteric venous thrombosis were more frequently found ($P < .05$) in group 2 (Table II).

The most common presenting symptom was abdominal pain (100%). Other symptoms included vomiting (27%), diarrhea (23%), and constipation (23%)(Table III). The mean duration of presenting symptoms before admission was 4.7 ± 0.8 days. Nineteen patients (73%) had diffuse abdominal pain. When pain was localized, it was located in the upper quadrants in four patients and in the lower quadrants in three patients. Physical findings at admission were abdominal distention in nine patients (34.6%) and peritoneal signs in eight patients (30.8%). The mean temperature of patients at admission was 37.7 ± 0.1°C. The mean white blood cell count and amylase level were 18.1 ± 1.3 10^9/L and 61.4 ± 9.3 IU/L, respectively. Systolic hypotension (defined as < 90 mm Hg) at admission was present in five patients. There was no difference in these parameters between the two groups.

A CT scan was performed at admission in 18 patients (69.2%), including six patients(42.8%) from group 1 and 12 patients (100%) from group 2 ($P < .01$). A thrombus in the superior mesenteric vein (SMV) was demonstrated by means of a CT scan in 16 patients (88.9%). Thrombosis extended to the portal vein in 10 patients (62.5%) and to the splenic vein in one patient. Bowel wall thickening and peritoneal effusion were found in 16 patients (88.9%). There was no difference in the incidence of these findings between the two groups (Table IV). In group 1, the diagnosis of MVT was established by means of CT scanning in five patients (35.7%) and by means of laparotomy in nine patients (64.3%). In group 2, the diagnosis was established by means of CT scanning in 11 patients (91.6%) and by means of laparotomy in one patient (8.4%).

The initial management in this study consisted of anticoagulation in 13 patients (50%) and laparotomy in 13 patients (50%). In group 1, 12 patients underwent laparotomy. Of these, four patients underwent operations after a CT scan (3 with peritoneal signs and 1 with an uncertain diagnosis despite the CT scan). Two patients (14%) in group 1 were treated with anticoagulation alone and thus received treatment that was identical to that of most patients in group 2. In group 2, 11 patients were treated with anticoagulation alone, and one patient was treated

Table IV. CT scan findings

	All patients (%)	*Group 1 (%)*	*Group 2 (%)*
CT scan	18	6 (42.8)	12 (100)*
MVT diagnosis	16 (88.9)	5 (83.3)	11 (91.6)
Thrombus SMV	16 (88.9)	5 (83.3)	11 (91.6)
Wall thickening	16 (88.9)	6 (100)	10 (83.3)
Peritoneal effusion	16 (88.9)	6 (100)	10 (83.3)
Streaky mesentery	12 (66.6)	4 (67)	8 (67)
Thrombus portal vein	10 (62.5)	3 (60)	7 (63.6)
Thrombus splenic vein	1 (6)	–	1 (9)

*$P < .01$.

Table V. Morbidity, mortality, and outcome

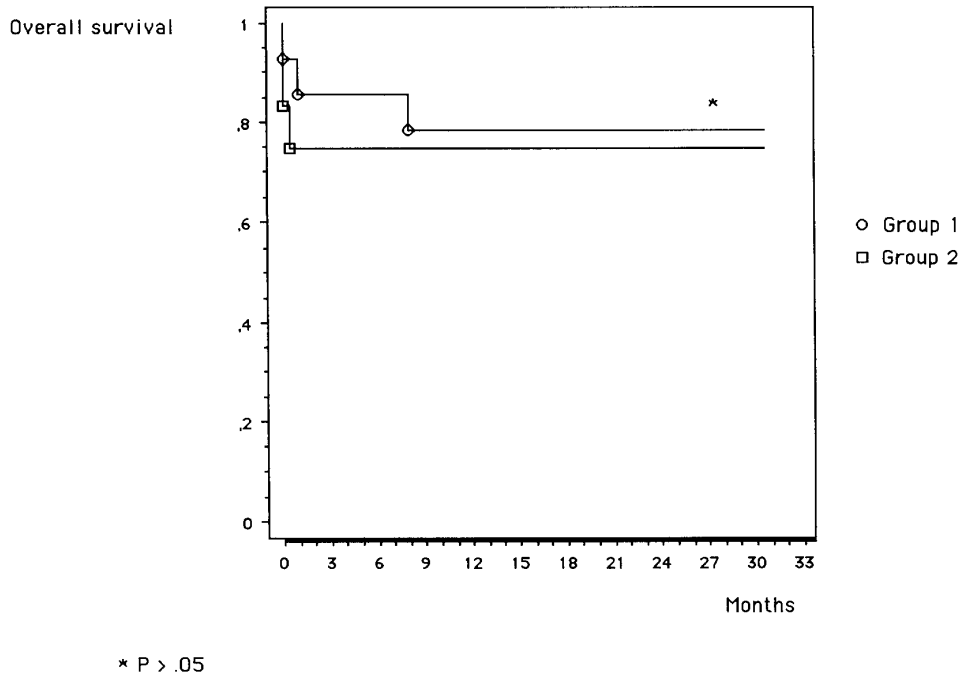
	All patients (%)	*Group 1 (%)*	*Group 2 (%)*
Morbidity	9 (34.6)	7 (50)	2 (16.6)
Pneumonia	5 (19.2)	3 (21.4)	2 (16.6)
Wound infection	3 (11.5)	3 (21.4)	0
Renal failure	2 (7.6)	2 (14.2)	0
Sepsis	2 (7.6)	1 (7.1)	1 (8.3)
Short bowel syndrome	1 (3.8)	1 (7.1)	0
Cavernous transformation of the portal vein	5 (19.2)	2 (14.3)	3 (25)
Secondary operation	4 (15.3)	1 (7.1)	3 (25)
Bowel perforation	2	—	2 (16.6)
Bowel stricture	1	1 (7.1)	—
Portal hypertension	1	—	1 (8.3)
Mortality (≤ 24 h)	3 (11.5)	1 (7.1)	2 (16.6)
Mortality (≤ 30 d)	5 (19.2)	2 (14.3)	3 (25)
Mean follow-up (mo)	51.3 ± 9.1	74.2 ± 13.8	24.6 ± 5.3
Two-year survival (%)	76.9	78.6	75

surgically ($P < .001$). Bowel wall thickening was shown by means of a preoperative CT scan in this patient, but the diagnosis of MVT was not confirmed.

Of the 13 patients who underwent operations, one patient in group 1 had peritoneal signs (guarding) and was found to have an extensive infarction of the small bowel and the right colon at laparotomy. In this patient, the SMV was exposed to confirm its thrombosis, but no bowel resection was performed. This patient died 6 hours postoperatively. In the other 12 patients, macroscopic involvement of bowel segments, featuring dark red coloration with marked wall thickening, was revealed by means of the surgical exploration. An infarcted small bowel lesion without macroscopic perforation was found in 10 patients (83%), and sealed perforations of infarcted small bowel without generalized peritonitis were found in two patients (17%). All macroscopic bowel lesions were resected, and no thrombectomy or thrombolytic therapy was performed. The mean length of resected bowel was 100 ± 18 cm. Immediate bowel anastomosis was performed in six cases, and ileostomy was performed in another six cases. Histologic features of ischemia, including submucosal congestion, hemorrhage, and edema of the intestinal wall, were observed in all 12 patients. Early epithelial damage restricted to the mucosa without ischemic involvement of the entire thickness of the bowel wall was observed in 10 patients (83%). Complete coagulative necrosis (transmural necrosis) of the bowel wall was found in two patients (17%). Peritoneal signs (abdominal tenderness in two patients and guarding in one patient) were present before laparotomy in three (30%) of the patients whose microscopic features showed only mucosal necrosis. Both patients with transmural necrosis had peritoneal signs (guarding) before laparotomy.

In-hospital morbidity occurred in 34.6% of patients, with no significant difference between the two groups (Table V). Cavernous transformation of the portal vein (CTPV) was observed in five patients (19.2%). This complication occurred in two patients in group 1 who were treated surgically, and in three patients in group 2 who were treated with anticoagulation alone. Three of these patients had no symptoms, whereas two patients had episodes of variceal bleeding that were documented by means of endoscopy.

A secondary operation after initial treatment was needed in four patients (15.3%; Table V). One patient in group 1 had chronic intestinal obstruction 10 weeks after initial treatment with anticoagulation. This patient under-



Kaplan-Meier estimates of 2-year survival for patients in group 1 and group 2. There was no difference in short-term survival between the 2 groups (log-rank test).

went surgery for intestinal stricture caused by MVT, and 18 cm of jejunum were resected with immediate reanastomosis. Two patients in group 2 who did not initially have peritoneal signs underwent operations for secondary small bowel perforations. Both patients had peritoneal signs that occurred 24 days and 25 days after their initial treatment with anticoagulation. Resections of 20 cm and 25 cm of small bowel were performed with immediate anastomosis in one patient and with ileostomy in the other. The third patient in group 2 returned with extrahepatic portal hypertension and cavernous transformation of the portal vein 10 months after the initial treatment with anticoagulation. Because the treatment of recurrent bleeding gastroesophageal varices with endoscopic sclerotherapy was unsuccessful, this patient underwent esophagogastric devascularization and esophageal transection.

The mean duration of hospitalization for initial treatment was 51.6 ± 14.9 days for patients in group 1 and 23.2 ± 8.4 days for patients in group 2 ($P < .05$). The total hospital stay, including readmissions, was 62.4 ± 16.3 days for patients in group 1 and 28.5 ± 11 days for patients in group 2 ($P < .025$). The 24-hour mortality rate was 11.5%; one patient in group 1 and two patients in group 2 died of MVT. The 30-day mortality rate was 19.2%; two patients died because of comorbid conditions (pneumonia with sepsis) at day 13 in group 2 and at day 28 in group 1 (Table V). The mean follow-up period was 51.3 ± 9.1 months. One patient in group 1 died of a comorbid condition at 6 months (pulmonary edema). The overall 2-year survival was 76.9%, and no difference ($P < .8$) was found between the two groups (Figure).

Eight of the 10 patients who had thrombus extension into the portal vein at admission had complications: secondary CTPV developed in 5 patients, 2 patients had secondary small bowel perforations, and 1 patient had a secondary intestinal stricture (Table V). Restoration of venous flow without thrombus in the SMV was revealed by means of CT scans in five patients with CTPV, but not in the two patients with secondary small bowel perforations or in the patient with secondary intestinal stricture.

DISCUSSION

Acute MVT is defined as a thrombus of the mesenteric vein with bowel infarction, regardless of its severity.[4,8] This study suggests first, that nonoperative management of acute MVT is feasible when the diagnosis made by means of the initial CT scan is certain and when bowel infarction has not led to transmural necrosis and bowel perforation. Second, initial surgical and nonoperative approaches are similar in morbidity, mortality, and survival rates. Third, the hospital stay is shorter when patients are treated nonoperatively. Finally, a nonoperative approach, when achievable, avoids the resection of macroscopically infarcted small bowel without transmural necrosis, which potentially is reversible with anticoagulation alone.

In a review of the literature from 1911 to 1984, Abdu et al[8] showed that the use of anticoagulants as an adjunct to bowel resection significantly decreases the mortality

rate (22% vs 59%) and improves the survival of patients (77% vs 65%). The results of surgery associated with anticoagulation are similar to those in group 1 in our study. Currently, the consensus for MVT management is that patients should receive anticoagulants once the diagnosis of MVT is established either by means of a diagnostic test or intraoperatively.[1,9,10] Recently, nonoperative treatment has been proposed in patients with MVT.[4,7,11-13] We changed our preferred treatment from surgical to nonoperative management in consideration of the favorable outcome with anticoagulation alone in two patients in group 1. Our study confirms that nonoperative management is feasible, but only when the diagnosis of MVT is confirmed with certainty by means of noninvasive abdominal imaging at admission. Currently, this can be established by means of ultrasound scanning, CT scanning, or magnetic resonance imaging.[5] Rhee et al[4] showed that CT scanning was the most sensitive tool for detecting acute MVT. Currently, high-quality abdominal CT scans are used as a means of detecting MVT in more than 90% of cases.[5,12,14,15] This is substantiated by the current study.

The second criterion for nonoperative management is the absence of transmural bowel necrosis at admission. Evaluation of the actual severity of bowel ischemia at admission is difficult. When peritoneal signs initially are present, an immediate operation is probably indicated.[4,5] We agree with Chen et al[3] that guarding indicates the need for laparotomy because of the risk of bowel perforation as a consequence of transmural infarction of the intestine. However, we encountered mucosal necrosis without transmural necrosis or bowel perforation in three patients with peritoneal signs (abdominal tenderness in two patients and guarding in one patient). This suggests that peritoneal signs may not strictly correlate to the severity of bowel ischemia, thus accounting for the difficulty in establishing a clinical diagnosis of intestinal infarction.[16] Greater accuracy in the evaluation of bowel ischemia may be attainable in the future with gastric intramural pH measurement[17] or with the evaluation of bowel wall thickening by means of contrast-enhanced CT scanning.[15] These examinations may help the surgeon decide more accurately when nonoperative management is appropriate. Overall, features allowing nonoperative management have been found in approximately 36% of the patients who have been reported in the literature since 1990.[4,9,12,18-21] In our study (group 2), this proportion increased to 80% of patients when CT scans were rapidly performed at admission.

In-hospital complications occurred in 55% of the patients in the Mayo series.[4] In the recent literature, mortality rates have ranged from 29% to 38%[4,6,19,20] for surgical treatment and from 13% to 19%[9,12] for nonoperative management, with 1-year survival of 61% to 77%[4,8] and 56%,[4] respectively. Because the morbidity, mortality, and survival rates in our series are consistent with earlier reports, we think that our patients did not represent a favorable selection bias and that our conclusions are valid. Furthermore, patients in group 2 cannot be considered as having MVT that is more susceptible to cure with anticoagulants, because the incidence of hypercoagulable states was similar in both of our treatment groups.

Bowel infarction without transmural necrosis is potentially reversible with anticoagulation.[12] This has been shown by means of the favorable outcome of postoperative anticoagulation in patients with infarction of the entire small bowel at laparotomy, precluding any bowel resection.[22] When surgical management is routinely used, the surgeon usually excises all affected bowel at laparotomy because of the impressive macroscopic bowel appearance that is caused by acute MVT.[3,5,7] In our series (group 1), this strategy led to bowel resection for infarction without transmural necrosis of about 100-cm length in 82% of patients who underwent resection. When nonoperative management is achievable, this major bowel resection is avoided.[11,12]

Our experience with patients who have peritoneal signs without transmural bowel necrosis indicates that peritoneal signs may not strictly correlate with the severity of ischemia. Even if clinical presentation is relevant in determining which patients can be observed versus those patients who require surgery, this criterion should not be the only one evaluated at admission. New criteria, such as bowel wall thickness and bowel wall enhancement on the arterial phase of a CT scan, need further evaluation. Even with anticoagulation, MVT can cause arteriolar spasm leading to transmural infarction.[3,7,8] This complication occurs in about 18% of patients who are treated nonoperatively.[12] The two patients in whom secondary bowel perforation developed in our series had no peritoneal signs at admission. Therefore, clinical findings are not an accurate means of predicting which patients will have a secondary bowel perforation that requires a subsequent operation.[9] Close follow-up of these patients is mandatory to establish the diagnosis of secondary bowel perforation.[7,12] Secondary intestinal stricture as a consequence of intestinal ischemia has been described in case reports,[21,23] but this complication has occurred in less than 10% of patients receiving nonoperative treatment. Like other authors, we have found that even when a secondary bowel resection is necessary for delayed perforation or stricture, it can frequently be of limited length.[12,24]

Extrahepatic portal hypertension occurs in 25% of patients with acute MVT.[12] We have demonstrated that this complication was not specific to patients who were treated nonoperatively. Moreover, thrombus extension into the portal vein should be evaluated by means of CT scanning at admission. When present, it justifies close monitoring, because it increases the risk of CTPV and secondary bowel perforation or stricture. Thrombolytic therapy, administered either peripherally or locally, has been reported.[3,25-27] No patient in these reports had CTPV, which suggests that this therapy might be more effective than systemic anticoagulants. Because the experience is limited, however, the role of this therapeutic option requires further evaluation. Controlled trials will be necessary as a means of prospectively assessing thrombolytic therapy and nonoperative management.

In summary, our experience with surgical and nonoperative initial treatment of MVT suggests that nonoperative management for acute MVT is feasible when the initial diagnosis made by means of CT scan is certain and when bowel infarction has not led to transmural necrosis and bowel perforation. When a nonoperative approach is possible, it avoids resection of macroscopically infarcted small bowel without transmural necrosis in cases that are potentially reversible with anticoagulation alone. In addition, the length of hospital stay is shorter. Initial surgical management and nonoperative approaches are similar in morbidity, mortality, and survival rates. Because complications, such as bowel stenosis or perforation, can occur in patients who are treated nonoperatively, close follow-up must be performed.

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REFERENCES

1. Rhee RY, Gloviczki P. Mesenteric venous thrombosis. Surg Clin North Am 1997;77:327-38.
2. Bergenfeldt M, Svensson PJ, Borgstrom A. Mesenteric vein thrombosis due to factor V Leiden gene mutation. Br J Surg 1999;86:1059-62.
3. Chen MC, Brown MC, Willson RA, Nicholls S, Surawicz CM. Mesenteric vein thrombosis. Dig Dis 1996;14:382-9.
4. Rhee RY, Gloviczki P, Mendonca CT, Petterson TM, Serry RD, Sarr MG, et al. Mesenteric venous thrombosis: still a lethal disease in the 1990s. J Vasc Surg 1994;20:688-97.
5. Hassan HA, Raufman JP. Mesenteric venous thrombosis. South Med J 1999;92:558-62.
6. Klempnauer J, Grothues F, Bektas H, Pichlmayr R. Results of portal thrombectomy and splanchnic thrombolysis for the surgical management of acute mesentericoportal thrombosis. Br J Surg 1997;84:129-32.
7. Boley SJ, Kaleya RN, Brandt LJ. Mesenteric venous thrombosis. Surg Clin North Am 1992;72:183-201.
8. Abdu RA, Zakhour BJ, Dallis DJ. Mesenteric venous thrombosis—1911 to 1984. Surgery 1987;101:383-8.
9. Grieshop RJ, Dalsing MC, Cikrit DF, Lalka SG, Sawchuk A. Acute mesenteric venous thrombosis. Revisited in a time of diagnosis clarity. Am Surg 1991;57:573-7.
10. Bradbury AW, Brittenden J, McBride K, Ruckley CV. Mesenteric ischemia: a multidisciplinary approach. Br J Surg 1995;82:1446-59.
11. Verbanck JJ, Rutgeerts LJ, Haerens MH, Tytgat JH, Segaert MF, Afschritt MB. Partial splenoportal and superior mesenteric venous thrombosis. Early sonographic diagnosis and successful conservative management. Gastroenterology 1984;86:949-52.
12. Harward TR, Green D, Bergan JJ, Rizzo RJ, Yao JST. Mesenteric venous thrombosis. J Vasc Surg 1989;9:328-33.
13. Matos C, Van Gansbeke D, Zalcman M, Ansay J, Delcour C, Engelmholm L, et al. Mesenteric vein thrombosis: early CT and US diagnosis and conservative management. Gastrointest Radiol 1986;11:322-5.
14. Clavien PA, Huber O, Mirescu D, Rohner A. Contrasted enhanced CT scan as a diagnostic procedure in mesenteric ischemia due to mesenteric venous thrombosis. Br J Surg 1989;76:93-4.
15. Taourel PG, Deneuville M, Pradel JA, Regent D, Bruel JM. Acute mesenteric ischemia: diagnosis with contrast-enhanced CT. Radiology 1996;199:632-6.
16. French Universitary Association of Surgical Research. Semeiology of intestinal infarction: a prospective study. Gastroenterol Clin Biol 1989;13:260-4.
17. Haglund U. Gut ischemia. Gut 1994;35(1 Suppl):S73-6.
18. Gertsch P, Matthews J, Lerut J, Luder P, Blumgart LH. Acute thrombosis of the splanchnic veins. Arch Surg 1993;128:341-5.
19. Levy PJ, Krausz MM, Manny J. Acute mesenteric ischemia: improved results—a retrospective analysis of ninety-two patients. Surgery 1990;107:372-80.
20. Inderbitzi R, Wagner HE, Seiler C, Stirnemann P, Gertsch P. Acute mesenteric ischemia. Eur J Surg 1992;158:123-6.
21. Cardot F, Borg JY, Guedon C, Lerebours E, Colin R. Venous mesenteric ischemia: infarction and transient ischemia. Gastroenterol Clin Biol 1992;16:644-8.
22. Lefrancois C, Derlon A, Le Querrec AL, Justum AM, Gautier P, Maurel J, et al. Mesenteric venous thrombosis. Case-study of 18 cases. Ann Fr Anesth Reanim 1994;13:182-94.
23. Eugene C, Valla D, Wesenfelder L, Fingerhut A, Bergue A, Merrer J, et al. Small intestine stricture complicating superior mesenteric vein thrombosis: a study of three cases. Gut 1995;37:292-5.
24. Clavien PA, Durig M, Harder F. Venous mesenteric infarction: a particular entity. Br J Surg 1988;75:252-5.
25. Poplausky MR, Kaufman JA, Geller SC, Waltman AC. Mesenteric venous thrombosis treated with urokinase via the superior mesenteric artery. Gastroenterology 1996;110:1633-5.
26. Ludwig DJ, Hauptmann E, Rosoff L, Neuzil D. Mesenteric and portal vein thrombosis in a young patient with protein S deficiency treated with urokinase via the superior mesenteric artery. J Vasc Surg 1999;30:551-4.
27. al Karawi MA, Quaiz M, Clark D, Hilali A, Mohamed AE, Jawdat M. Mesenteric venous thrombosis, non-invasive diagnosis and follow up and non-invasive therapy by streptokinase and anticoagulants. Hepatogastroenterology 1990;37:507-9.

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