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The management of mesenteric vein thrombosis: a single institution's experience

Mezenter ven trombozuna yaklaşım: Tek merkez deneyimi

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BACKGROUND

Mesenteric vein thrombosis occurs rarely and is responsible for approximately 5-15% of all cases of acute mesenteric ischemia. The aim of this report was to discuss the management of mesenteric vein thrombosis based on our experience with 34 patients.

METHODS

In the present study, 34 patients who were admitted to our emergency surgery department between January 2007 and January 2010 with a diagnosis of acute mesenteric vein thrombosis were assessed retrospectively. Patients with peritoneal signs first underwent diagnostic laparoscopy to rule out perforation or bowel gangrene. We performed a second-look laparoscopy within 72 hours of the first operation. All patients were administered 100 mg/kg of the anticoagulant enoxaparin twice daily. In the 6th and 12th months of follow up, CT angiography was performed to evaluate recanalization of the veins.

RESULTS

CT angiography revealed superior mesenteric vein thrombosis in 25 (73%) patients, portal vein thrombosis in 24 (70%) patients, and splenic vein thrombosis in 12 (35%) patients. Eleven patients with peritoneal signs underwent diagnostic laparoscopy; eight of the patients underwent small bowel resection, anastomosis, and trocar insertion. During second-look laparoscopy, small bowel ischemia was found in two patients and re-resection was performed.

CONCLUSION

Early diagnosis with CT angiography, surgical and non-surgical blood flow restoration, proper anticoagulation, and supportive intensive care are the cornerstones of successful treatment of mesenteric vein thrombosis.

Key Words: Algorithm; antithrombotic therapy; mesenter vein; thrombosis.

AMAÇ

Mezenter ven trombozu, akut mezenter iskemi olgularının yaklaşık %5-15'inden sorumlu olan ve nadir görülen bir durumdur. Bu çalışmanın amacı, 34 hastalık tecrübemizi paylaşmak ve mezenter ven trombozuna yaklaşımı tartışmaktır.

GEREÇ VE YÖNTEM

Ocak 2007 ve Ocak 2010 tarihleri arasında acil cerrahi servisimize mezenter iskemi tanısı ile başvuran 34 hasta geriye dönük olarak incelendi. Peritonit bulgusu mevcut olan hastalara, başvurularında tanısal laparokopi uygulandı. Ameliyatın bitirilmesine yakın, karın sol alt kadrana 10 mm laparoskopi trokarı yerleştirildi. Anastomoz yapılan olgularda ameliyat sonrası ilk 72 saatlik dönemde laparoskopik ikincil bakı yapıldı. Tüm hastalar günde iki kez subkutan 100 mg/kg enoksaparin uygulandı. Ven rekanalizasyonu değerlendirilmesi amacıyla tüm hastalara, 6. ve 12. aylarda bilgisayarlı tomografi (BT) anjiyografi görüntüleme yapıldı.

BULGULAR

Bilgisayarlı tomografi anjiyografi ile 25 (%73) hastada superiyor mezenterik ven trombozu, 24 (%70) hastada portal ven trombozu ve 12 (%35) hastada splenik ven trombozu saptandı. Peritonit bulgusu olan 11 hastaya tanısal laparoskopi yapıldı. Bu hastaların 8 tanesine ince bağırsak rezeksiyonu ve anastomozu yapılarak ikincil bakı için trokar yerleştirildi. İkincil bakı yapılan hastalardan 2 tanesinde ince bağırsak iskemisi saptanarak re-rezeksiyon gerçekleştirildi.

SONUÇ

Mezenterik ven trombozunun tedavisinde BT anjiyografi ile erken tanı, cerrahi ya da cerrahi dışı yöntemlerle kan akımının sağlanması, uygun antikoagülan kullanımı ve yoğun bakım destek tedavileri, hastalığın başarılı bir şekilde yönetilmesinde hayati rol oynamaktadırlar.

Anahtar Sözcükler: Algoritma; antitrombotik tedavi; mezenter ven; tromboz.

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Acute mesenteric vein thrombosis (MVT) causes 5-15% of acute mesenteric ischemia cases.[1,2] Clinical signs of MVT are usually non-specific.[3] The most common presenting symptom is abdominal pain.[1] Of MVT cases, 25-55% are primary MVT cases, and recent reports have suggested that the incidence of primary MVT is declining. The incidence decline has been largely attributed to the progression and incorporation of hypercoagulability screening into daily practice, as well as, an increase in malignancy incidence. Both are crucial co-morbidities which are highly associated with MVT, and both should be sought for by managing MVT.[4,5] The most common causes of MVT are prothrombotic states, due to heritable or acquired disorders of coagulation or to cancer, intraabdominal inflammatory conditions, the postoperative state, cirrhosis, and portal hypertension.[1] Oral contraceptive use accounts for 9 to 18 percent of mesenteric venous thrombosis episodes in young women.[6,7]

Due to recent advances in computed tomography (CT) technology and the introduction of CT angiography, early diagnosis of MVT has become possible. Furthermore, the administration of subsequent anticoagulation has resulted in lower surgical intervention rates.[8,9] No consensus exists regarding optimal treatment for MVT; some authors favor aggressive surgical interventions, while others prefer a more conservative approach.[10,11]

Currently, no absolute consensus on or guide algorithm for treatment of MVT exists. In this report, we present our recent experience with acute MVT and discuss our treatment algorithm.

MATERIALS AND METHODS

Thirty-four patients who presented to our emergency surgery department with acute abdomen and were diagnosed with acute MVT were assessed retrospectively. Presentation, diagnosis, diagnostic modality, and treatment protocols were assessed for each patient. Patient records were analyzed for age, gender, presenting symptoms, diagnostic modalities, thrombus location, treatment, surgical intervention, hospital stay, etiology, and follow-up. Patients presenting with acute abdomen suggesting mesenteric ischemia were evaluated with CT angiography after hemodynamic stabilization. Thrombosis of the superior or inferior mesenteric veins (SMV or IMV), the portal vein (PV), or the splenic vein (SV) was diagnosed as MVT (Figure 1). Patients with peritoneal signs and unstable hemodynamic parameters underwent initial exploration via diagnostic laparoscopy. If necrosis was found, a laparotomy with bowel resection was performed. Surgical options were limited to bowel resection and/or second-look laparoscopy and, in one patient, third-look laparoscopy.

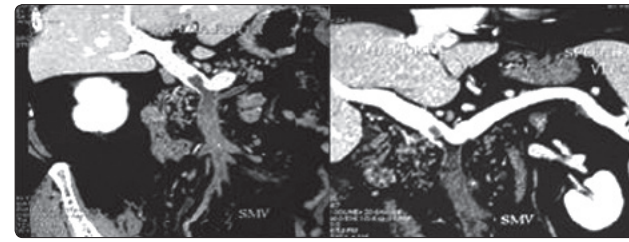

Fig. 1. Mesenteric vein thrombosis.

All patients were treated with enoxaparin, a low molecular weight heparin (LMWH) (100 mg/kg, twice daily), after diagnosis with MVT, until an oral anticoagulant (warfarin) could be administered, if indicated. Once a second surgical intervention seemed unlikely to be necessary, oral anticoagulation therapy was started with the aim of maintaining an international normalized ratio (INR) between 2.0 and 3.0. Anticoagulation treatment was continued throughout each patient's life, and patients were followed up every three months. Patients were assessed for etiology; those without risk factors were classified as having primary MVT, while those with at least one risk factor were classified as having secondary MVT. Thrombophilia screening was performed for all patients with indications. All patients were diagnosed with CT angiography. To assess if the thrombosis cranes to the branches of the portal vein, additional mesenteric venous duplex US, including of the portal vein, was performed in eight patients.

RESULTS

The study included 24 males (70%) and 10 females (30%) with a median age of 45 years (range 18-76 years). There were 19 patients (55%) with primary MVT and 15 patients (45%) with secondary MVT (Table 1). In seven patients, thrombophilia screening was positive, and six out of the seven had combined protein C/S deficiency. Twelve patients presented with abdominal pain only; 18 patients presented abdominal pain, as well as, nausea, vomiting, and distension. Three patients presented with gastrointestinal bleed-

Table 1. Secondary mesenteric vein thrombosis

Etiology	n	%
A. Primary MVT	19	55
B. Secondary MVT	15	45
Prothrombotic factors	7	
Combined Protein C/S deficiency	6	
(2 patients with additional AT-III deficiency)		
Factor V Leiden mutation	1	
Cirrhosis / Portal Hypertension	4	
Necrotizing Pancreatitis	1	
Malignancy (gallbladder cancer, prostate cancer)	2	
Deep vein thrombosis	1	

ing alone, and one patient presented with deep venous thrombosis (DVT). The median time elapsed until reference was three days (range 1-20).

Computed tomography angiography was performed on all patients. Additional mesenteric venous duplex US, including the portal vein, was performed on eight patients, and there were no cases of thrombosis in the intrahepatic branches of the portal vein. The most common thrombus localizations were the superior mesenteric vein (25 patients) and the portal vein (24 patients). In three patients, an inferior vena cava thrombus was ascertained together with thromboses in the other three veins (Table 2).

All patients received subcutaneous enoxaparin (100 mg/kg, twice daily) immediately following diagnosis with MVT. Serial abdominal exams were performed,

Table 2. Computed tomography angiography findings

Thrombus location	Patients	
	n	%
Superior mesenteric vein	25	73
SMV	11	
SMV + PV	8	
SMV + PV + SV	3	
SMV + PV + SV + IVC	3	
Portal vein	24	70
PV	6	
PV + SMV	8	
PV + SV	4	
PV + SMV + SV	3	
PV + SMV + SV + IVC	3	
Splenic vein	12	35
SV	2	
SV + PV	4	
SV + SMV + PV	3	
SV + SMV + PV + IVC	3	
Inferior vena cava	3	8
IVC + SMV + PV + SV	3	

SMV: Superior or inferior mesenteric; PV: Portal vein; SV: Splenic vein; IVC: Inferior vena cava.

Table 3. Treatment results

Treatments	Patients	
	n	%
Medical treatment	23	68
Surgical treatment	11	32
Diagnostic laparoscopy	3	9
Second look	2	6
Third look	1	3
Diagnostic laparoscopy+small bowel resection	8	24
Second look	2	6

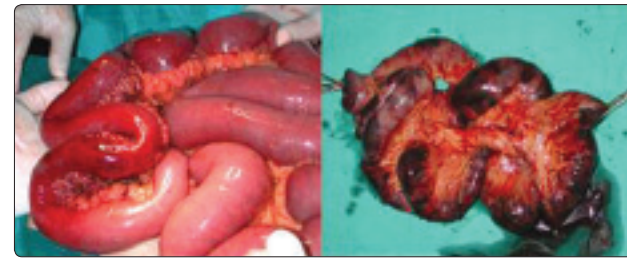

Fig. 2. Laparotomy images of mesenteric vein thrombosis.

(Color figur can be viewed in the online issue, which is available at www.tjtes.org).

and leukocyte counts and CRP levels were assessed. The absence of peritoneal signs excluded surgical intervention, and 23 patients (68%) were placed on oral anticoagulation (warfarin, 5 mg daily) therapy aimed at maintaining an INR between 2.0 and 3.0 (Table 3). After adequate INR levels were reached, administration of subcutaneous enoxaparin was stopped. Patients were then discharged and received follow up every three months, as well as, life-long oral anticoagulation.

Eleven patients (32%) with peritoneal signs underwent surgical intervention. Diagnostic laparoscopy was performed initially to assess bowel viability, and bowel necrosis or perforation necessitated laparotomy. Eight of the patients underwent small bowel resection (Figure 2); in two patients, a port was left in-situ for second-look laparoscopy to assess the progression of low-flow state, bowel edema, and ecchymosis. Second-look laparoscopy was performed 24 hours after the resection, and resolutions to suspicious findings were noted for these patients. In three patients diagnostic laparoscopy was performed without a need for subsequent resection; two patients required second-look laparoscopy, and one of the two required third-look laparoscopy (after 48 hours) which revealed resolution of ischemia on suspicious bowel segments (Table 3).

Mortality occurred in three patients (8%), all of whom had been treated surgically. In two patients, subtotal bowel resection with anastomosis was performed, and in one patient, laparotomy revealed total small bowel, stomach, and colon ischemia, which was considered inoperable. One patient succumbed due to sepsis, one patient due to associated mesenteric arterial and celiac arterial thrombosis, and the remaining patient due to pulmonary failure. There were no late mortalities related to MVT, although two patients died because of malignancy.

The mean hospital stay length was 13 days (range: 7-39 days). The mean follow-up period after discharge from the hospital was 24 months. In the 6th and 12th months of follow up, CT angiography was performed to determine recanalization of the veins. Of the patients, 26 (76%) had total recanalization and 8

Table 4. Patients with partial or no recanalization in the 6th month and the 12th month

Diagnosis	Patients (n)	6th month	12th month
Cirrhosis/Portal HT	3	Partial	Partial (n=2) Total (n=1)
Malignancy	2	No	(Exitus) (n=2)
Prothrombotic (protein C/S deficiency)	1	Partial	Total (n=1)
Primary	2	Partial	Partial (n=1) Total (n=1)

patients (24%) had partial or no recanalization in the 6th month. Within the latter group, three patients were diagnosed with cirrhosis/portal hypertension, two with malignancy, two with primary MVT, and one with combined protein C/S deficiency. In the 12th month of follow up, two patients died because of malignancy. Of the remaining six patients (18%), three patients (9%; one with cirrhosis/portal hypertension, one with primary MVT, and one with protein C/S deficiency) showed total recanalization and three (9%; two with cirrhosis/portal hypertension and one with primary MVT) showed partial recanalization (Table 4). Small bowel syndrome occurred in one patient (3%) who underwent bowel resection of 200 cm.

DISCUSSION

Mesenteric venous thrombosis was first described by Warren and Eberhardt in 1935.[2] Up until then, it had been a challenge to determine the underlying causes of MVT. To successfully diagnose MVT, physicians must first be aware of MVT and consider it in differential diagnosis. Because the onset of clinic is slower than arterial occlusions of mesenteric vessels, early diagnosis differs mortality and morbidity much more salutary. MVT presentation can be acute, subacute, or chronic.[1] Acute thrombosis is associated with a bowel infarction in one-third of patients.[12] The primary aim of treatment must be to avoid the pathological process that leads to necrosis, and early diagnosis is required to avoid transmural gangrene, perforation, and peritonitis.[13]

Following advances in radiologic imaging, as well as, increasing awareness of mesenteric venous thrombosis, early detection and medical treatment obviating surgical intervention became feasible. MVT diagnosis via ultrasound is often limited by overlying bowel gas. CT and MRI scans should be considered the primary diagnostic techniques for patients who may have MVT.[14] Venous phase CT angiography is the most accurate imaging modality (sensitivity 90%) for diagnosing mesenteric venous thrombosis.[15] The causes of MVT in young patients are generally thrombophilia and the use of oral contraceptives, whereas, in elderly patients, malignancies must be ruled out. Moreover, autopsy studies have determined that abdominal cancer is present in 22% of cases and hepatic cirrhosis is present in 17%.[12,16]

Anticoagulant therapy for venous thromboembolism was first demonstrated by Barritt and Jordan in 1960.[17] Their randomized study reported that if patients do not receive anticoagulant therapy, approximately 25% experience fatal recurrences and another 25% experience non-fatal recurrences.[17] Some studies have suggested that anticoagulant therapy increases venous recanalization up to 80%.[6,18] In the early phases of MVT, immediate administration of heparin, even intraoperatively, clearly increases survival and

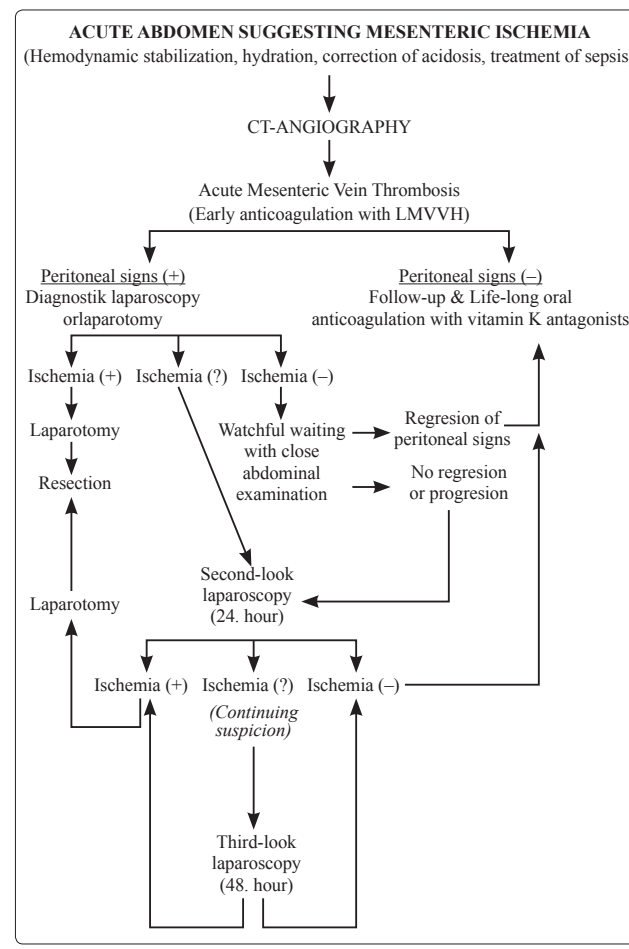

Fig. 3. Diagnosis and treatment algorithm for mesenteric vein thrombosis.

significantly decreases the risk of recurrence.[19] In our series, total recanalization rates were 76% within six months of follow-up and 85% (91% if we exclude deaths caused by malignancies) within 12 months of follow-up.

In a systematic review of management of acute non-cirrhotic and non-malignant portal vein thrombosis by Hall et al.,[20] 29 articles with 315 patients were included, and treatments and outcomes were analyzed. They described the treatment modalities as conservative management, anticoagulation, and thrombolysis and thrombectomy. They concluded that early anticoagulation with subcutaneous LMWH or intravenous heparin is important. They also determined that at least six months of oral anticoagulation is effective in reducing long-term morbidity and mortality in cases of portal vein thrombosis occurring concurrently with SMV or SV.[20] Correspondingly, in cases with prothrombotic risk factors, long-term or life-long anticoagulant treatment could be considered, as stated in recent published consensus statements.[7,20,21]

After diagnosis with MVT, anticoagulation should be started promptly with the administration of enoxaparin 100 mg/kg twice daily. The reported overall mortality rate of 50% in the literature is mainly attributed to difficulties in diagnosis and subsequent delay of necessary therapeutic intervention.[15,22] The first step in treatment is hemodynamic stabilization accompanied by hydration, correction of acidosis, and treatment of sepsis using broad spectrum antibiotics (Figure 3). During the acute phase, serial abdominal exams are necessary to detect peritoneal signs. If there are no peritoneal signs and conservative treatment resolved, oral anticoagulation therapy (warfarin 5 mg daily), aimed at maintaining an INR of 2.0-3.0, must be started immediately. Peritoneal signs should be evaluated via laparoscopy, and appropriate intervention should be undertaken.[13] The optimal duration of anticoagulation therapy is still obscure. Some authors suggest six months of anticoagulation[13] and some recommend life-long anticoagulation.[7,21] If a bowel resection is mandatory, the aim must be conserving as much bowel segments as possible. However, if vascularization is suspected, ostomy or a port for second-look laparoscopy 24 hours later are indicated.[23,24] The aim of second-look and third-look laparoscopy is to conserve as much bowel segment as possible.

Mesenteric vein thrombosis has a high rate of recurrence, and recurrences are most common within 30 days after presentation.[25] We recommend life-long anticoagulation through serious problems related with MVT. A non-operative approach to anticoagulation can be successful in more than 90% of patients.[26] In some studies, thrombolysis and endovascular treatments were attempted in patients diagnosed with MVT and clinical success was reported.[27-32] However, there have been no randomized control trials to provide more certain evidence of the effectiveness of these procedures.

Overall mortality due to MVT is approximately 50%.[15,22] whereas, in our series the mortality rate was 8% (three patients). One patient had total small bowel, stomach, and colon ischemia, which was considered inoperable, and died at postoperative day 3. One patient succumbed from sepsis due to intestinal perforation causing severe intraabdominal contamination. The third patient, who had previous history of pulmonary problems, died due to pulmonary failure after spending 10 days in the intensive care unit.

In conclusion, early diagnosis of MVT, urgent treatment, selective surgical intervention, and proper anticoagulation are the cornerstones of successful treatment, resulting in lower morbidity and mortality. Although we present our clinical experience and treatment algorithm which resulted in successful outcomes, more studies with large series and systematic reviews are needed to clarify the management of MVT.

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