

Mesenteric and Portal Vein Thrombosis: Treated with Early Initiation of Anticoagulation

J.-H. Joh and D.-I. Kim*

Division of Vascular Surgery, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, South Korea

Objective. Superior mesenteric vein thrombosis (SMVT) is generally difficult to diagnose and can be fatal. Mesenteric and portal vein thrombosis is rare and can be presented as more serious conditions than that of SMVT. We report patients with combined SMVT and portal vein thrombosis (PVT) who were treated successfully with early initiation of anticoagulation.

Methods. The medical records of six patients (five male, one female) who presented with combined SMVT and PVT in our institute between January 1994 and September 2003 were reviewed retrospectively. All of the patients were treated with early initiation of anticoagulation using unfractionated heparin or low molecular weight heparin.

Results. The mean hospital stay was 31 days and the mean follow-up period was 32 months. Three patients had an antithrombin III deficiency. The most common symptom was diffuse abdominal pain and signs included abdominal distension and tenderness. During the follow-up period, there were two patients who developed stricture of the small bowel necessitating resection and anastomosis of the small bowel. There was no case of peritonitis due to bowel necrosis or mortality.

Conclusion. The early initiation of anticoagulation in patients of SMVT combined with PVT could minimise the serious complication such as peritonitis due to bowel necrosis required immediate exploratory laparotomy.

Keywords: Superior mesenteric vein thrombosis; Portal vein thrombosis; Anticoagulation.

Introduction

Superior mesenteric vein thrombosis (SMVT) is an uncommon cause of mesenteric ischaemia. It was first recognised more than 100 years ago by Eliot,¹ who described intestinal gangrene resulting from mesenteric venous occlusion. In 1913, Trotter reported that 41% of patients with mesenteric ischaemia had SMVT.² The subject remained virtually unexplored until 1935 when Warren and Eberhardt reported two personal cases and collected 73 others from published reports.³ At the time of their publication, SMVT became recognised as a distinct clinical entity. They reported a mortality rate of 34%. Additionally, they found that only 5% of patients who did not undergo surgical treatment survived. Unfortunately, this mortality rate still holds despite improvements in therapy.

SMVT combined with portal vein thrombosis (PVT) is a disease that is more rare than SMVT, but can

present acute and serious symptoms and signs that are potentially fatal. Because the symptoms are not specific, the diagnosis and initial treatment are often being delayed, but once SMVT and PVT are suspected, computed tomography (CT) scan should be performed promptly. In cases with signs of peritonitis, transmural bowel infarction, or haemodynamic instability require immediate exploratory laparotomy.⁴ However, there is a few recent reports in which SMVT combined with PVT was successfully treated by anticoagulation therapy alone and close follow-up.^{5,6}

We report six cases of combined thrombosis of the superior mesenteric (SMV) and portal veins (PV) which we treated by early initiation of anticoagulation without surgical or radiological intervention.

Patients

Between January 1994 and September 2003, we treated 15 patients (10 men, and five women) with superior mesenteric thrombosis (SMVT) and/or portal vein thrombosis (PVT). There were one PVT, eight SMVT

* Corresponding author. Dong-Ik Kim, MD, Division of Vascular Surgery, Samsung Medical Center, 50 Irwon-dong, Kangnam-ku, Seoul 135-710, South Korea.
E-mail address: dikim@smc.samsung.co.kr

and six SMVT combined with PVT. All these cases were diagnosed by abdominal CT scanning. A positive CT scan consisted of the enlargement of the thrombosed vein, a central area of low attenuation representing intraluminal thrombus, and the vein wall sharply delineated by a hyperlucent rim of tissue.

One case of PVT occurred in a patient with advanced gastric cancer with multiple liver metastasis. This patient presented with oesophageal variceal bleeding necessitating endoscopic variceal ligation without anticoagulation treatment. Eight cases of SMVT were treated by anticoagulation with heparin followed by warfarin.

In this series, we reviewed six cases of SMVT combined with PVT. There were five men and one woman with an average age of 45 (range: 31–70 years). The patient characteristics, conditions predisposing to thrombosis, symptoms and signs at admission, CT scan findings of abdomen, extent of thrombus, use of heparin, and complications were analysed. Blood sampling for thrombophilic disorders was done prior to anticoagulation therapy.

Results

Conditions associated with thrombosis

Three patients had an antithrombin III deficiency. One out of three patients had combined protein C and protein S deficiency. This patient also had liver cirrhosis of Child B classification. There was one patient with inflammation and swelling of the vermiform appendix. There was one patient with diverticulitis of the colon.

Symptoms and signs at admission

All of the patients were admitted through the emergency department with diffuse abdominal pain, severe abdominal distension, and tenderness. Three patients showed rebound tenderness. One patient had ascites detected clinically with a fluid wave on physical examination and three patients had ascites detected by CT scanning.

Diagnostic evaluation

SMVT and PVT was diagnosed by abdominal CT scanning. All patients showed typical positive findings of CT scan (Fig. 1). The patient was followed up with CT scan for evaluating the disease progression

(Figs. 2 and 3). The stricture of the small bowel was diagnosed primarily on the basis of clinical findings and definitely on the basis of the small bowel series (Fig. 4).

Extent of thrombus

Among the six patients with combined SMVT and PVT, one patient showed combined splenic vein thrombosis, one iliac vein thrombosis, one splenic and iliac vein thrombosis.

Patient management

All patients were treated with anticoagulation, antibiotics, and total parenteral nutrition (TPN) as soon as they were diagnosed with SMVT combined with PVT. Nadroparin calcium 2850 IU was given 12 hourly in two patients, enoxaparin sodium 40 mg was given 12 hourly in two patients, and unfractionated heparin maintaining the activated partial thromboplastin time (aPTT) between 60 and 85 s was used in two patients. TPN was initiated via a central line. Triple antibiotics comprising of second-generation cephalosporins, aminoglycoside, and metronidazole were used. Our divisional policy is that immediate exploratory laparotomy is indicated where signs of peritonitis are present. This diagnosis is based on radiological findings such as a transmural gas shadow within the bowel wall irrespective of the leucocyte count. Laparotomy was not indicated in any patient in this series. All of the SMVT and PVT recanalised regaining at least 50% lysis of the thrombus on CT scanning during follow-up period. All patients were advised to continue life-long anticoagulation with warfarin.

Complications

Cavernous transformation of the portal vein developed in four patients (Fig. 5). During the follow-up period, two patients presented with symptoms of small bowel obstruction after 1.5 and 4 months following initial anticoagulant therapy. Contrast radiology with an upper gastrointestinal swallow and small bowel study showed a stricture of the small bowel. The location of the stricture in one patient was at the jejunum 50 cm distal to the Treitz ligament, 3 cm in length, and 0.8 cm in external diameter. In the other patient, the stricture was in the jejunum 70 cm distal to the Treitz ligament, 3.5 cm in length. We resected the strictures with anastomosis of the small bowel. Histological examination showed segmental stenosis

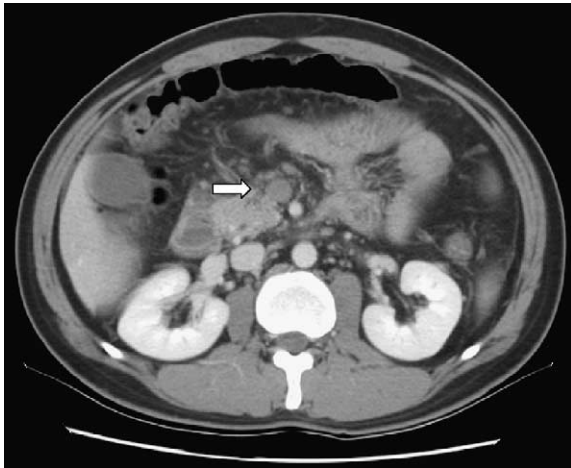


Fig. 1. CT scan shows the enlargement of superior mesenteric vein with central area of low attenuation representing intraluminal thrombus (white arrow).

with features suggestive of ischaemic enteritis combined with venous thrombosis. There was a pleural effusion in two patients and oesophageal varices in two patients.

Clinical outcome

To date, there has been no mortality. The mean follow-up period is 32 months (range: 1–79 months). The mean duration of hospitalisation was 31 days (range: 13–63 days).

Discussion

The clinical diagnosis of SMVT has evaded clinicians

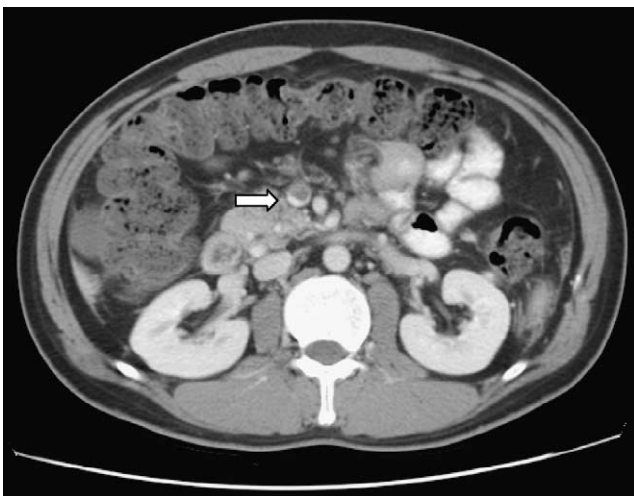


Fig. 2. CT scan after 10 days: partially recanalised superior mesenteric vein (white arrow).

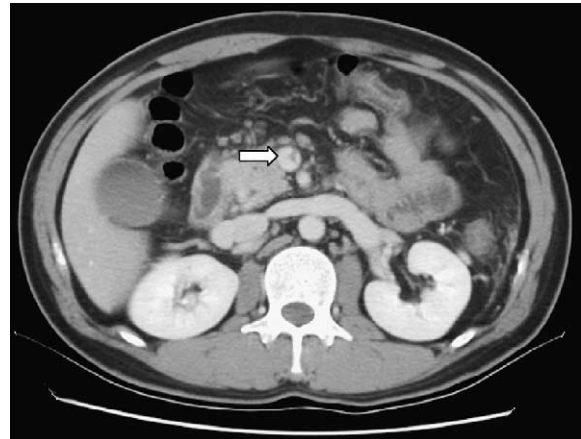


Fig. 3. CT scan after 30 days: more recanalised superior mesenteric vein (white arrow).

for decades; consequently, the true incidence of this condition is unknown. In autopsy studies, SMVT is found in approximately 1.5% of the population.⁷ Traditionally, MVT is grouped under the category of mesenteric ischaemia. Ottinger and Austen found venous thrombus in 0.06% of the patients with mesenteric ischaemia at the Massachusetts General Hospital.⁸ Risk factors for venous thrombosis include hypercoagulable states, portal hypertension, trauma, post-splenectomy, malignancy, inflammatory states, pregnancy, and oral contraceptive use.^{9–12} In young

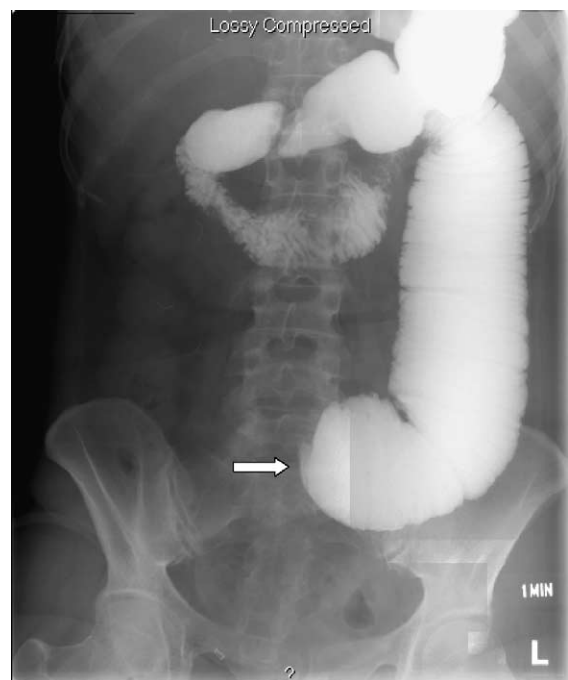


Fig. 4. Small bowel series showing stricture of the small bowel, severe dilatation of proximal portion and no passage of barium to the distal portion (white arrow).

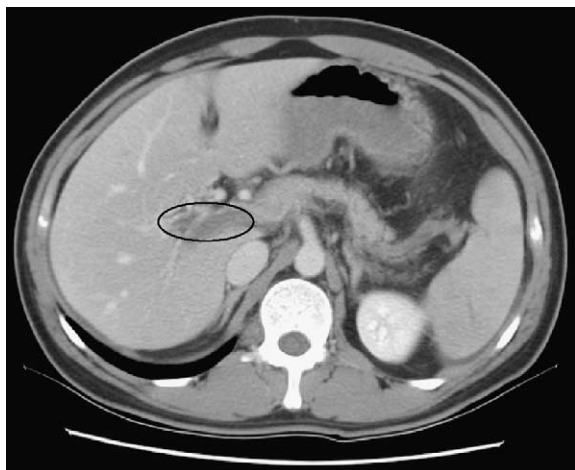


Fig. 5. Cavernous transformation of hepatic hilum (shown by the ellipse).

patients who do not have known risk factors for thrombosis, a thorough workup for primary hypercoagulable states is warranted. Overall, the most common cause is myeloproliferative disease. Other causes include genetic factors such as: factor V Leiden gene mutation (activated protein C resistance); hyperhomocysteinemia; hyperfibrinogenemia; paroxysmal nocturnal haemoglobinuria; antithrombin III deficiency; protein C deficiency; protein S deficiency; or acquired risk factors such as antiphospholipid antibody or anticardiolipin antibody syndromes, collagen vascular disease, or dysfibrinogenemia.⁹⁻¹² In our series, the most common condition predisposing to combined SMVT and PVT was antithrombin III deficiency.

In the past, an objective diagnosis of SMVT has been difficult to establish. The use of the venous phase of a selective mesenteric arteriogram may help¹³ but often it is difficult to detect the actual filling defect. Therefore, the diagnosis has depended on indirect findings such as incomplete venous filling. Recently, contrast-enhanced abdominal CT scan has been shown accurately to detect portal vein, ovarian vein,¹⁴ and mesenteric vein thrombosis.¹⁵ In our study, all the SMVT and PVT were confirmed by abdominal CT scanning. An immediate exploratory laparotomy was indicated if peritonitis associated with bowel infarction was suspected. No patient in this series required laparotomy. Regardless of whether surgical treatment is needed, anticoagulation with heparin and treatment with broad-spectrum antibiotics should begin immediately because they decrease the risk of rethrombosis and ultimately improve survival.⁴ In patients with symptomatic MVT, endovascular treatment has been reported with encouraging initial

results. The route of treatment was indirect intra-arterial infusion of thrombolytic drugs through the superior mesenteric artery.¹⁶⁻¹⁸ Indirect intra-arterial infusion of thrombolytics is less technically demanding and has the theoretic advantage of lysis of the small venules of the mesentery. Some authors believe, however, that indirect lysis is more unpredictable because of preferential flow into collaterals. This technique is generally associated with longer infusion times.¹⁹

The portal vein may be accessed via transjugular or transhepatic^{19,20} routes for pharmacological or mechanical thrombolysis have the advantage of a direct approach to the system. The transhepatic route is technically simpler than the transjugular approach. The main disadvantage of the transhepatic route is the risk of bleeding, especially in patients receiving anticoagulation therapy and in patients with ascites. Opitz *et al.* reported the use of a transjugular intrahepatic portosystemic stent-shunt (TIPS).²¹ TIPS initially served as a diagnostic tool allowing direct angiography of the portal vein, the superior mesenteric vein and the splenic vein. The insertion of the TIPS and subsequent thrombus fragmentation achieved hepatic decompression and full recanalisation of the portal vein.

Surgical thrombectomy has been proposed,^{22,23} especially in patients at high risk of complications from thrombolytic therapy. In patients with extensive thrombosis of the portal vein and mesenteric vein, thrombectomy could initially debulk the thrombus. Pharmacological thrombolysis would probably still be necessary to treat residual thrombosis after surgical thrombectomy. Since, mesenteric and portal vein thrombosis is a rare condition and the number of cases reported is limited, it is hard to compare the results of different approaches to treatment. Further work is required to evaluate these possible therapies.

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