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Case Report

Combination of Surgical Thrombectomy and Direct Thrombolysis in Acute Abdomen with Portal and Superior Mesenteric Vein Thrombosis

Hyuk Jae Jung and Sang Su Lee

Division of Vascular and EndoVascular, Department of Surgery, Pusan National University Yangsan Hospital, Yangsan, Korea

Portal vein (PV) thrombosis (PVT) is a rare condition with development of thrombosis in the PV and its branches. Further extension to the splenic and superior mesenteric vein (SMV) causes intestinal infarction, with a reported mortality of up to 50%. A variety of treatments for PVT exist including anticoagulation, thrombolysis, surgical thrombectomy, insertion of shunts, bypass surgery, and liver transplantation. We experienced a case of successfully treated by surgical thrombectomy with direct thrombolysis into the thrombosed-PV and SMV. A 31-year-old male presented worsening abdominal pain for one week. Preoperative contrast enhanced computed tomography scan revealed complete PVT extending to splenic vein and SMV. The PV was accessed surgically and opened by thrombectomy; visual inspection confirmed proximal and distal flow. Urokinase was administered directly into the inferior mesenteric vein with successful decrease in thrombus burden. The complete angiography showed complete dissolution of thrombosis in PV and SMV.

Key Words: Portal vein thrombosis, Superior mesenteric vein thrombosis, Acute abdomen, Thrombectomy, Thrombolysis

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Corresponding author: Sang Su Lee
Division of Vascular and EndoVascular, Department of Surgery, Pusan National University Yangsan Hospital, 20 Geumo-ro, Mulgeum-eup, Yangsan 626-770, Korea
Tel: 82-55-360-2124
Fax: 82-55-360-2154
E-mail: phoenixdr@naver.com
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INTRODUCTION

Portal vein (PV) thrombosis (PVT) is a rare condition that usually occurs in the extrahepatic PV and extends into the intrahepatic PV, or distally to the superior mesenteric and splenic veins [1]. With the development of imaging technology, the rate of diagnosis for PVT and superior mesenteric vein (SMV) thrombosis (SMVT) has gradually increased [2]. Although some patients with PVT may be asymptomatic, many manifest various symptoms such as abdominal pain, nausea, anorexia, and weight loss. Thrombosis extended into the SMV can be serious and cause intestinal infarction, with mortality rates of up to 50% [3]. Recently, minimally invasive techniques including endovascular approaches have been introduced to treat PVT and SMVT. However, these methods are limited in cases of suspicious intestinal infarction. We present a case of a 31-year-old male patient with an acute abdomen due to massive PVT and SMVT who was successfully treated by surgical thrombectomy and direct thrombolysis.

CASE

A 31-year-old male patient was transferred from the local medical center to our emergency room complaining of worsening abdominal pain, nausea, and vomiting for one week. He had a history of subtotal gastrectomy Billroth-II surgery due to duodenal ulcer perforation 5 years ago.

On physical examination, his temperature was normal, and mild abdominal tenderness with decreased bowel sound was found. He also complained of diffuse and constant abdominal pain with radiation to the back, however the signs of acute abdomen were not found. His initial blood pressure was maintained at 110/70 mmHg with pulse rate of 70 beats/min. Blood tests revealed normal white cell counts ($6.43\times10^3/\mu L$) with neutrophil counts (43.8%) and mild elevated C-reactive protein (CRP) (1.34 mg/dL). One day after admission, he had increasing and intolerable abdominal pain with tenderness and rebound tenderness. Follow-up lab findings were worse with high white blood cell (WBC) counts ($13.26\times10^3/\mu L$) and neutrophil counts (87.1%), and high CRP (10.1 mg/dL). Compared to the prior contrast enhanced computed tomography (CECT) scan taken 2 days before, a follow-up CT scan revealed complete PVT extending to the splenic and SMVs, and mild edema of the jejunum was found (Fig. 1). Immediately following the second CECT, the patient was transferred emergently to the operating room for an exploratory laparotomy.

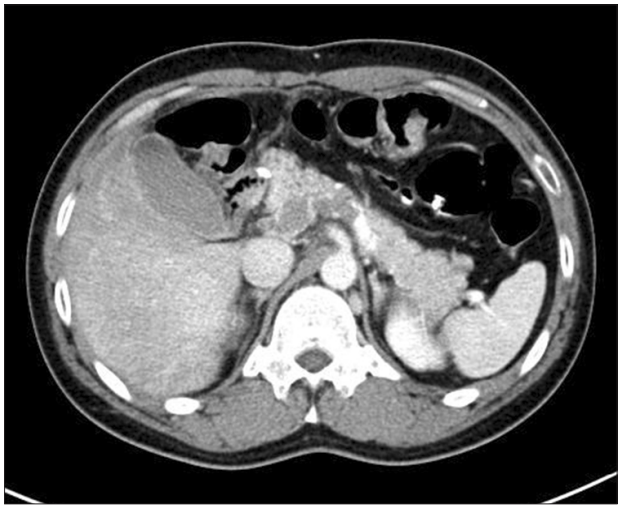

Fig. 1. Preoperative computed tomography scan showed massive thrombosis in the portal vein and superior mesenteric vein.

With the patient under general anesthesia, careful inspection of the intra-abdominal cavity was done. Fortunately, the liver and spleen were unremarkable and both large and small bowels were viable without significant edema. However, upon dissection of the PV and SMV, massive thrombosis of the PV and SMV was grossly noted. After clamping both proximal and distal PV, the thrombosis was removed through a vertical incision of the PV, and intermittent release of the clamps allowed for further removal of the thrombus. Flow through the PV was confirmed both proximally and distally. After surgical thrombectomy, a 6 Fr infusion catheter (multi-pore; Cook, Bloomington, IN, USA) was placed through a small incision of the inferior mesenteric vein in order to deal with the expected residual thrombosis affecting the smaller branches. Angiography was performed intraoperatively under mobile C-arm guidance and revealed remnant thrombosis in PV and SMV. A bolus of 300,000 IU of urokinase (Abbott Labs, North Chicago, IL, USA) was administered directly via the catheter (Fig. 2). Within 10 minutes, completion angiography revealed complete dissolution of thrombosis in the PV and SMV. Postoperatively, therapeutic heparinization was commenced, and a hypercoagulability workup was performed. The hypercoagulable workup, which

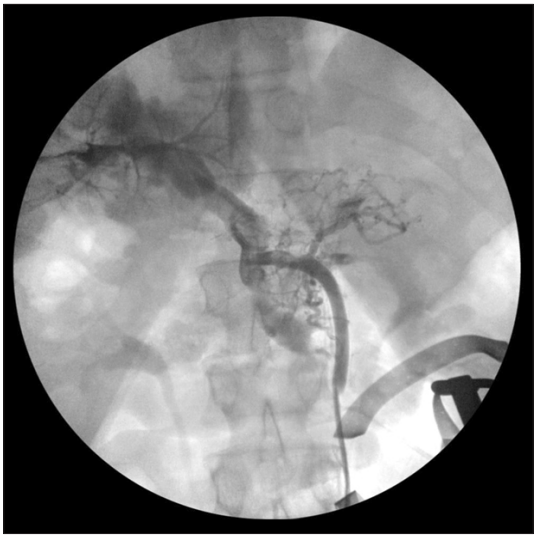

Fig. 2. Urokinase was directly administered via the inferior mesenteric vein.

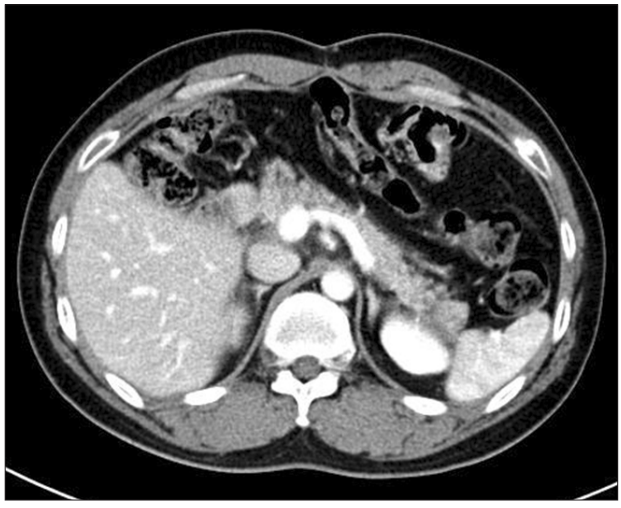

Fig. 3. A 1-year follow-up computed tomography scan showed complete resolution of thrombosis in the portal vein.

included protein C and S activity, protein C and S Ag, anticardiolipin immunoglobulin A (IgA), IgM, and IgG, tissue plasminogen activator, homocysteine, and factor V Leiden revealed protein C and S deficiency in the patient. The postoperative course was uneventful, and he was discharged on oral anticoagulation. A 1-year follow up CT scan showed no residual thrombosis in the PV and SMV (Fig. 3).

DISCUSSION

PVT is the development of thrombus in the PV and causes blockage of blood flow to the liver. Further extension to the SMV and mesenteric venous arch can cause intestinal infarction which needs surgical emergency operation [3]. The diagnosis usually relies on imaging study when patients have unexplained abdominal pain and distension. Various etiologies have been reported for PVT. Approximately, 6%-11% of patients with PVT have underlying liver cirrhosis, and 35% are related with hepatocellular carcinoma [4]. In one study of 184 patients without cirrhosis, 34% reported previous history of abdominal surgery, 14% had pancreaticobiliary disease, and 9% had alimentary tract disease [5]. These factors may be related with PV injury during surgery or stasis of blood flow. Other causes are genetic factors such as protein C deficiency, protein S deficiency, antithrombin III deficiency, hyperfibrinogenemia or acquired factors such as antiphospholipid antibody or anticardiolipin antibody syndromes, collagen vascular disease, or dysfibrinogenemia [6,7]. The patient of our case presented with protein C and protein S deficiency in hypercoagulability workup and had a history of abdominal surgery because of gastric ulcer perforation.

Clinically, acute PVT has symptoms such as abdominal pain, nausea, emesis, and fever. These conditions may be more severe when mesenteric infarction causes intestinal necrosis. Nonspecific symptoms including diarrhea, anorexia, weight loss, and abdominal distention may develop. Our patient presented with severe abdominal pain, nausea, and emesis when he arrived into the emergency room, which tally with the ensuing diagnosis. Color Doppler ultrasound, CECT scan, and magnetic resonance angiography are useful for diagnosis of PVT. Recently, contrast enhanced abdominal CT scan has been shown to accurately detect PV and mesenteric vein thrombosis [8].

Treatment should be based on the individual patient's clinical symptom and situation, because there are few controlled data on which to base clinical decisions in patients with PVT and SMVT. In the patient without signs of acute abdomen, or biochemical upset (rising lactate, increasing CRP/WBC etc.), anticoagulation should be started initially. Pharmacological and mechanical thrombolysis, transjugular intrahepatic portosystemic stent-shunt, and endovascular treatment may be effective options in thrombus removal or dissolution [9]. However, thromboses of both portal and mesenteric veins have a higher chance of developing an acute abdomen and are associated with more congestion and fewer collateral vessels as with our case. Once surgical abdomen is suspected, exploratory laparotomy should be indicated. Therefore, we suggest that a combined treatment of surgical thrombectomy and direct thrombolysis using a thrombolytic agent may be favored in these circumstances. For extensive thrombosis of the portal and mesenteric veins, surgical thrombectomy can initially debulk the thrombus, and direct thrombolysis can probably lyse any residual thrombosis after surgical thrombectomy. Thrombolysis through transjugular and transhepatic routes have been reported in the literature [10], however, retrograde access through the inferior mesenteric vein or omental veins gives easy and safe access when the patient has already undergone laparotomy for surgical abdomen.

Our case expands from earlier reports illustrating the general surgeons' point of view. Surgical thrombectomy combined with direct thrombolysis may be an effective treatment if the patient needs surgical exploration due to acute abdomen. We believe this method can be effective in the removal of massive thrombus burden in patients with acute abdomen resulting from PV and SMV thrombosis.

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