Percutaneous transhepatic thrombolysis in the treatment of acute portal venous thrombosis

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ABSTRACT

Acute portal vein thrombosis (PVT) is a rare clinical condition that can cause portal hypertension and bowel infarction. Early diagnosis and treatment of PVT is crucial for the restoration of portal venous flow and reduction of morbidity and mortality. We report a successful treatment of acute PVT which was seen following splenectomy, utilizing catheter directed transhepatic thrombolysis. No complication was encountered related to the procedure. Thrombolytic therapy via transhepatic route proved to be a safe and effective method in the treatment of PVT.

Key words: • portal vein • thrombosis • thrombolytic therapy • splenectomy

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Portal venous thrombosis (PVT) is a rare clinical entity and necessitates early diagnosis and treatment for the prevention of mesenteric ischemia. Myeloproliferative diseases are the most common underlying conditions that result in PVT. Septicemia, abdominal surgery, intraabdominal infections, cirrhosis and primary or secondary liver tumors are other conditions which can cause PVT. Treatment options for PVT range from conservative anticoagulant therapy to surgical thrombectomy.

Endovascular thrombolytic therapy is a new alternative technique for the treatment of PVT (1-4). Presented here is a case of PVT which occurred after splenectomy and successfully treated with catheter directed transhepatic thrombolysis.

Case report

70 year-old female patient with a history of visceral leishmaniasis underwent to splenectomy 20 days ago and admitted to our emergency department with abdominal distention and pain. Thrombus formation was demonstrated in superior mesenteric vein (SMV), main and intrahepatic portal vein (PV) branches with color Doppler ultrasonography (CDUS) and computed tomography (CT). Since the patient was symptomatic and the thrombosis was extensive and there was no sign of peritoneal irritation which necessitates early surgical intervention, percutaneous endovascular intervention appeared to be best alternative for the treatment. According to patient's history, there was no contraindication for thrombolytic therapy. Prior to the procedure, her platelet count was 157000/mm³. Prothrombin time and hemoglobine level were 12 seconds and 11 mg/dl respectively. Patient did not have a coagulation disorder so that percutaneous transhepatic access was preferred.

Under ultrasonographic guidance 6F vascular sheet was placed into the right PV with using Accustic Intervention System (Boston Scientific, Watertown, MA, USA). Diagnostic angiography catheter was then placed into SMV and venograms were obtained. Extensive filling defects in SMV, main PV, right and left PV due to thrombus formation were present (Figure 1). The distal portion of SMV was patent but proximal portions of SMV and PV were occluded. The first 15 cm portion of the infusion catheter that has side holes (Mewissen, Boston Scientific, Watertown, MA, USA) was placed into confluence of the main PV and proximal part of SMV. Pharmacomechanical thrombolysis was performed within the first 3 hours utilizing "pulse spray" method. Since there was no available pump for pharmacomechanical thrombolysis, manual technique was used. After the procedure, the patient was transferred to another room and monitorized. Five milligrams of recombinant tissue plasminogen activator (rd-PA, Actilyse, Boehringer Ingelheim, Germany) diluted with 25 ml saline solution was injected through the infusion catheter



Figure 1. Transhepatic venogram shows filling defects due to thrombus in SMV, main PV, right and left PV branches (SMV: superior mesenteric vein, PV: portal vein).



Figure 2. Control venography following balloon angioplasty and rd-PA infusion shows 90% reduction of thrombosis in SMV and main PV, though there is still prominent thrombosis left in left PV branch (SMV: superior mesenteric vein, PV: portal vein).

with a rate of 0.5 ml /min. Since the thrombus formation was extensive. pharmacomechanical thrombolysis was performed for 3 hours and a total of 15 mg rd-PA was infused. After 3 hours, control venograms showed partial and insufficient lysis of thrombus. Percutaneous transluminal angioplasty (PTA) was then performed with a size of 10x40 mm balloon (Smash, Boston Scientific, Watertown, MA, USA) to PV and SMV. After PTA, same infusion catheter placed into SMV and PV was used to perform rd-PA infusion for 12 hours with a rate of 1 mg/hour.

A day after the procedure, although luminal patency was achieved in PV and SMV, since there were still filling defects in SMV, main PV and left PV branches, rd-PA was injected for an other 1 hour period a with a rate of 5 mg/hour to PV and SMV. After a total of 30 mg rd-PA infused, 90% lysis of thrombus in portal and mesenteric vein was achieved (Figure 2). Procedure was ended after restoring normal flow in PV. Vascular sheet was taken out from the liver parenchyma and to prevent possible bleeding, gelatin sponge (Spongostan, Johnson&Johnson, Skipton, England) was placed into the entry site of the catheter.

The patient was heparinized during and until 3 days after the intervention. Oral warfarine-Na was given due to maintain optimal INR (international normalized ratio) value of 2-3. No complication was encountered during or after the procedure. After the end of the procedure abdominal pain has disappeared and abdominal distention was significantly decreased. On followup CDUS performed 3 months after the procedure, SMV and portal venous system were patent (Figure 3).

Discussion

Acute PVT is a rare clinical condition that may cause mesenteric ischemia and bowel infarct so that early diagnosis and treatment is crucial. Myeloproliferative diseases, intrabdominal infections or inflammations, septicemia, cirrhosis, intraabdominal surgery, trauma, hypercoagulable states (antithrombin III, protein C, protein S insufficiency and etc.), oral contraceptives, primary or secondary liver tumors are the most common reasons for PVT.

PVT is usually seen after splenectomy. Thrombosis ratio after splenectomy especially for myeloproliferative disease and cirrhosis, is about 13-18% (5,6). Hypercoagulation and blood stasis in the splenic remnant are the two major factors resulting in development of PVT after splenectomy (7).

Surgical thrombectomy is not preferred as treatment method for PVT since it is invasive, technically difficult and can cause hepatic encephalopathy. Surgical treatment is not considered as an alternative treatment in patients with poor general condition (2). If peritoneal irritation is present which is suggestive for bowel infarction, surgical thrombectomy with bowel resection should be performed.

Endovascular treatment for PVT has been started to be used in last few years and its effectiveness has been demonstrated only in small series (1-4). There is no place for endovascular thrombolysis in treatment of chronic PVT. Depending on patient's status, there are different ways for endovascular therapy of acute PVT. Thrombolysis can be achieved using pharmacological and/or mechanical methods. Thrombolytic agents might be given either directly into the PV or indirectly into the superior me-



Figure 3. Three months after the thrombolytic therapy, control CDUS image shows the restored patency in main PV and SMV (PV: portal vein, SMV: superior mesenteric vein).

senteric artery (SMA). Authors supporting thrombolytic therapy via SMA claim that thrombi in small veins can be disintegrated using this method (8,9). But other authors reported that resolving thrombus formation in the small veins is not easy because of collateral vascular circulation and can lead to prolongation of total infusion time via SMA (10). Increased infusion time of thrombolytic agents results in increased risk of bleeding. Antoch et al. reported two cases of PVT that they have restored the portal flow within 8 days using indirect SMA infusion technique. In another case, without restoring complete recanalization, procedure was ended on fifth day because of the femoral hematoma at access site (11-14). In our case, thrombolysis with pharmacomechanical (pulse spray) method was performed directly through the PV and after 3 hours partial flow restoration was achieved. Tissue plasminogen activator was utilized to enhance thrombolytic effect of pharmacomechanical thrombolysis. A day after the procedure, although significant amount of thrombus reduction was achieved, pharmacomechanical method again was performed for the residual thrombus and nearly normal flow was maintained in the PV within 24 hours. No complication was encountered during or after the procedure.

Direct intervention of PV thrombus has advantages compared to indirect method. Due to direct injection of thrombolytic agents, procedure time is shorter. It is known that pharmacomechanic method is more effective and less time consuming than normal thrombolytic infusion (14).

Another advantage of direct intervention of PVT is having the opportunity to perform thromboaspiration, mechanic thrombectomy, baloon angioplasty and stenting (3,10,12). Direct administration of thrombolytic agent in the occluded vessel decreases the systemic dose and related complications. In our case in addition to pharmacomechanical and pharmacological thrombolysis, baloon angioplasty was performed to disintegrate thrombus and to enhance the effect of rd-PA.

Another access for portal venous system is performing transjuguler intrahepatic portosystemic shunt. This procedure is generally preferred in patients of PVT with cirrhosis or portal hypertension. Transhepatic procedure is easier but the risk of hemorrhage is increased in patients with ascite and receiving anticoagulant therapy. Ultrasound guided approach with fine needles (21-22 G) and occlusion of access with gelatin sponge can decrease the risk of bleeding complication (13).

As a result, percutaneous transhepatic thrombolysis is a safe and effective method for the treatment of acute symptomatic PVT. Percutaneous transhepatic approach gives opportunity to pharmacomechanical thrombolysis and percutaneous transluminal angioplasty for recanalization and increases the effectiveness of treatment.

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