

Percutaneous Transhepatic Catheter-Directed Thrombolysis for Extensive Portal Vein System Thrombosis in Patients with Autoimmune Liver Cirrhosis After Splenectomy: A Case Report

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1. Abstract

Although portal vein system thrombosis is a common complication after splenectomy, the extensive portal vein system thrombosis (PVST) is rare. The experience of management and treatment of multiple portal vein thrombosis is worth sharing. We report a 23-year-old female patient with autoimmune liver cirrhosis, failed to give effective anti-coagulation in time to prevent thrombosis, and appeared serious extensive PVST. After the failure of anti-coagulation with heparin, the patient was successfully treated by percutaneous trans hepatic catheter-directed thrombolysis. As far as we know, there is no English case report of extensive portal vein thrombosis in the context of splenectomy for autoimmune liver cirrhosis.

2. Introduction

Portal vein system thrombosis (PVST) refers to thrombosis occurs in the splenic vein, portal vein, and superior mesenteric vein or

intrahepatic portal vein branches [1], which is potentially fatal for patients if the diagnosis is not timely or the treatment is not proper. The risk factors of PVST after splenectomy include surgery option [2], liver cirrhosis [3], a wider preoperative portal vein and splenic vein diameter [4-6], age [5], and lower preoperative platelet counts [6], increased platelet reactivity [3].

The management of acute portal vein thrombosis remains controversial according to its severity, location, background disease or time of discovering [7]. As recommended by the European Association for the Study of Liver, the current mainstay of therapy for acute PVST is anti-coagulation in the absence of contraindication [8]. In addition to anti-coagulation therapy, there are a variety of treatments, including percutaneous transhepatic or transjugular approaches with the use of mechanical thrombectomy, balloon dilatation, stenting, and pharmacologic thrombolysis and open thrombectomy [9-13].

To our knowledge, cases of postoperative thrombosis in decompensated patients with autoimmune liver cirrhosis after splenectomy are rare. Therefore, we take the lead to report a 23-years-old women patient with extensive portal vein thrombosis after splenectomy for autoimmune liver cirrhosis treated by percutaneous transhepatic portal vein catheterization for direct thrombolysis restoring the portal blood flow.

3. Case Report

A 23-years-old woman who was admitted to our hospital on December 9, 2019, had been diagnosed with decompensation stage of autoimmune liver cirrhosis and presented splenomegaly and hypersplenism. Computed tomography (CT) showed liver cirrhosis, splenomegaly, portal hypertension (Figure 1a). The liver function was normal and the Child-Pugh stage was A. The blood routine test showed platelet was $29 \times 10^9 /L$ and white blood cell was $3.03 \times 10^9/L$. In order to reduce hypersplenism, the patient underwent splenectomy on December 18, 2019. During the operation, because of the large spleen and poor visual field, the original laparoscopic splenectomy was changed to open, no other obvious abnormality.

After operation, platelet increased progressively (Figure 1b). Because of the bleeding of the wound (200-300 ml per day) and slight fever after operation, the patient was administered kalosulfonate sodium, Baimei snake venom to stop bleeding, and antibiotic for anti-infection. On the third day after operation, the bleeding stopped and 4000IU qd heparin anticoagulant therapy was given continuously. However, on December 23, the patient complained upper abdominal intense pain. Emergent CT showed that thrombosis occurred in the portal vein, intrahepatic portal vein branches

and superior mesenteric vein, and the liver function deteriorated (Figure 2a-d). Intravenous low molecular heparin at a dose of 4000IU every 12 hours was administered to anti-coagulation. However, 2 days later, CT showed more severe intrahepatic portal vein thrombosis and focal hepatic infarction (Figure 3a-d), and liver function test reported AST and ALT increased to 382U/L and 493U/L, respectively.

On December 27, 2019, after a discussion with clinicians of multiple disciplines, the emergency percutaneous trans hepatic portal vein catheterization was performed for direct thrombolysis. With patient under local anesthesia, the posterior branch of right portal vein was punctured under ultrasound guidance using a 7F vascular sheath with subsequent placement of angiography catheter to superior mesenteric vein. Venography showed no blood flow in portal vein (Figure 4a-b). Then the catheter for thrombolysis was placed in superior mesenteric vein (Figure 4c). Urokinase at a dose of 200 000 IU was infused into the thrombus with continuous micro-pumping every 8 hours. CT examination on the first day after thrombolysis showed partial resolution of the thrombus in the portal vein and recirculation of portal blood flow to liver parenchyma (Figure 5a-c). On December 30, 2019, the catheter implanted at the superior mesenteric vein was retrieved, and a new catheter was put in the left branch of portal vein to further thrombolysis. 11 days after thrombolysis, CT scan showed that the infarction disappeared and thrombus significantly decreased (Figure 5d-f). 14 days after thrombolysis, the catheter was removed and there was no procedure-related bleeding. The patient was discharged after all clinical symptoms disappeared, Liver function returned to normal, and no recurrence of portal vein system thrombosis developed during the follow-up period.

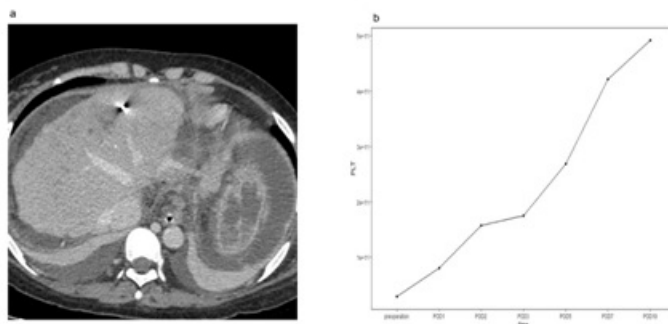


Figure 1: a. CT before splenectomy. b. Platelet changes after splenectomy. POD: postoperative day.

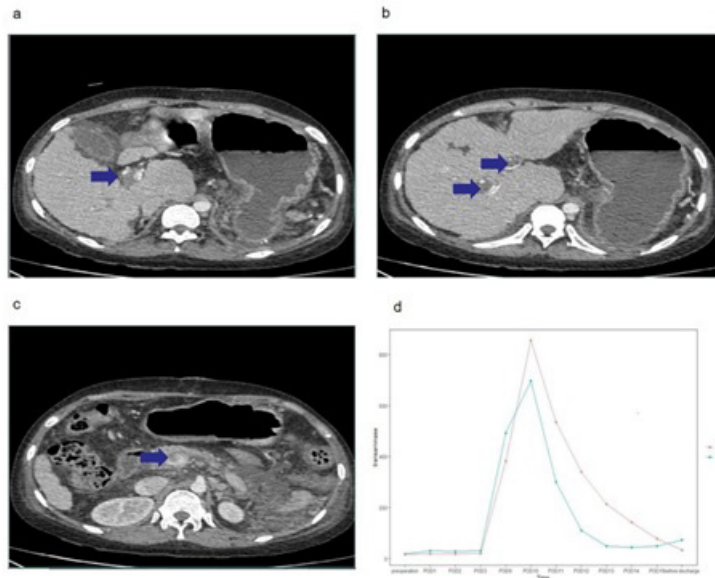


Figure 2: a. thrombosis of Main portal vein. b. Thrombosis of left and right branches of portal vein. c. thrombosis of Superior mesenteric vein. d. AST and ALT changes after splenectomy. POD: postoperative day.

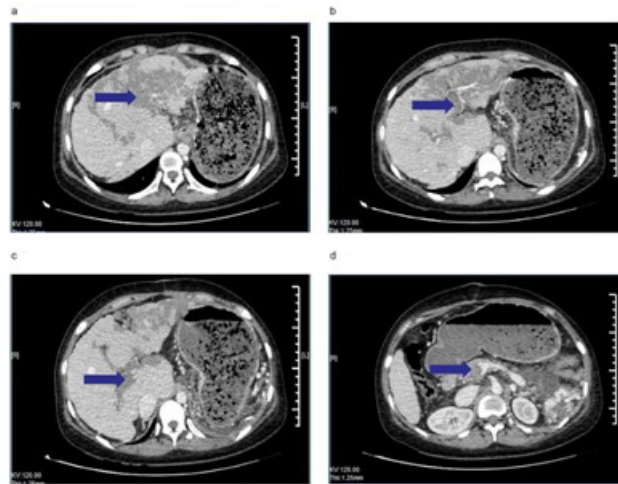


Figure 3: a. Large area hepatic infarction. b. Thrombosis of left branch of portal vein. c. Thrombosis of right branch of portal vein. d. Superior mesenteric vein thrombosis.

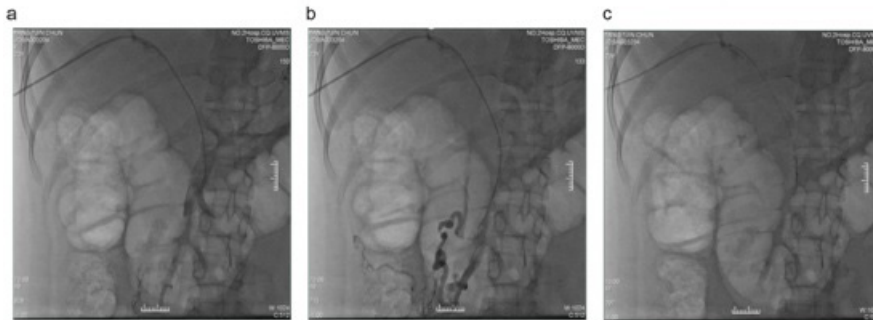


Figure 4: a. Superior mesenteric filling defect. b. Lateral branches of superior mesenteric vein cava open, portal vein blocked. c. Catheter thrombolysis location.

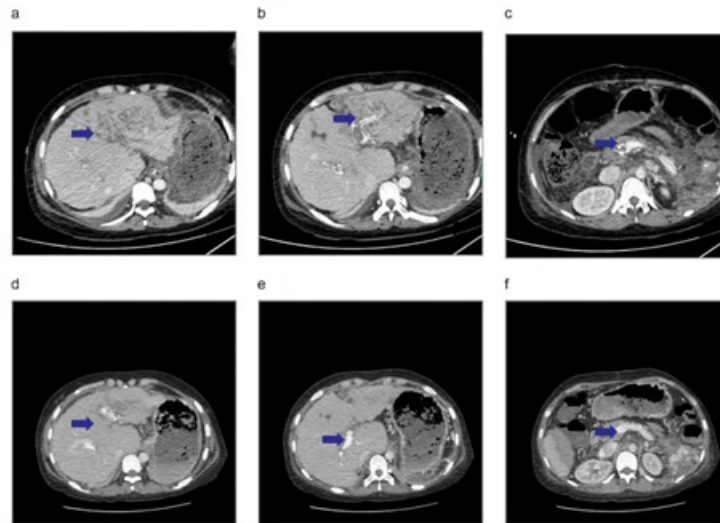


Figure 5: a. Reduction of hepatic infarction. b. Increased blood flow in left branch of portal vein. c. Reduction of mesenteric thrombus. d. Liver infarction almost disappeared. e. Increased blood flow in right branch of portal vein. f. Further reduction of mesenteric thrombus.

4. Discussion

PVST is a common complication of splenectomy. Pathophysiological characteristics of PVST include that reduced blood flow, damage of the vascular endothelium, and imbalance between anti-coagulation and coagulation after splenectomy are contribute to the thrombosis. The risk factors of PVST after splenectomy including the surgical approach, liver cirrhosis, increased platelet reactivity as mentioned above. Although PVST is more common in the context of malignancy, Dmitri B's analysis demonstrated a clear association between autoimmune causes of end stage liver disease and perioperative thrombotic complications [14]. In our reported case, the autoimmune liver cirrhosis, the platelet change, the vascular injury may be contributed to the PVST.

The symptoms of PVST are unspecific and unnoticed. The clinical manifestations of portal vein system thrombosis rang from asymptomatic to severe complications including fever, abdominal pain, nausea, vomiting, hepatic coma or even fatal intestinal necrosis. Fever not only is a symptom of inflammation or infection after splenectomy, but also can be a unspecific manifestation of PVST after splenectomy [15]. PVST should be suspected in patients with fever after splenectomy [15, 16]. Our patient presented fever on the second day after operation, and we considered it was an infectious manifestation and adjusted the antibiotics. But maybe the fever was a hint of PVST, and we neglected it. So clinical workers should attach importance to the fever and the other unspecific presentations to avoid the lethal PVST.

The PVST can be diagnosed with the use of enhanced CT or Doppler ultrasonography. The PVST should be treated timely. Once the liver function has deteriorated, treatment of acute PVT becomes limited and the risk of mortality greatly increases. According to the guideline of the European Association for the Study of Liver,

the anti-coagulation therapy was recommended [8]. But Systemic anticoagulant is of limited value in extensive PVST, as it has low efficacy and is time consuming [17]. So when anti-coagulation was failed to recanalize the portal vein in our patient, we took the next step for thrombolysis.

It was reported thrombectomy is a legitimate strategy in PVST. The main advantage of thrombectomy is complete clearance of the PVST under direct inspection, whereas the disadvantage is huge amounts of bleeding due to vigorous collateral vessels with portal hypertension [18], and it's highly invasive and risky. There would be a high risk of bleeding in our postoperative patient if thrombectomy treatment was given. Furthermore, endovascular interventional treatments have been reported to dissolve thrombus successfully. Endovascular interventional treatments include indirect access via the superior mesenteric artery, direct access to the portal vein by a trans jugular or trans hepatic route. Indirect therapy may result in thrombolytic agents diverting through collateral, leading to an increased risk of bleeding and is not suitable for extensive PVST. Although trans jugular approach does not traverse the hepatic capsule, and thus would avoid the risk of sub capsular hemorrhage, it's technically relative difficult and risky. Trans jugular intrahepatic portosystemic shunt (TIPS) was been reported to treat extensive PVST. However, the occurrence of TIPS dysfunction in cirrhotic patients and hepatic encephalopathy was high [19], and significant intraperitoneal portal vein bleeding is also a potential serious complication [20]. In contrast, percutaneous trans hepatic access is technically relatively easy, and suitable for the removal of larger clots within the trunk of the portal vein and superior mesenteric vein [17]. Weighing the advantages and disadvantages of these treatments during our discussion with clinicians of multiple disciplines, the emergency percutaneous trans hepatic portal vein

catheterization was performed for direct thrombolysis. Technic success was achieved in our patient, and no complications, such as intraperitoneal or sub capsular hepatic hemorrhage or contrast extravasation were observed during the procedure.

Even though that patients with acute PVST are treated with catheter-directed thrombolysis have been reported by many clinical workers, postoperative extensive thrombosis of portal vein system in the patient with decompensation stage of autoimmune liver cirrhosis after splenectomy has not been described. Therefore, our case demonstrates that the percutaneous trans hepatic catheter-directed thrombolysis is an effective means of treating extensive portal vein thrombosis. And we think this treatment has advantages over traditional therapy strategies such as systematic thrombolysis, surgical thrombectomy and TIPS in our case.

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