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TECHNICAL NOTE

Percutaneous Transhepatic Portography for the Treatment of Early Portal Vein Thrombosis After Surgery

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Abstract We treated three cases of early portal vein thrombosis (PVT) by minimally invasive percutaneous transhepatic portography. All patients developed PVT within 30 days of major hepatic surgery (one case each of orthotopic liver transplantation, splenectomy in a previous liver transplant recipient, and right extended hepatectomy with resection and reconstruction of the left branch of the portal vein for tumor infiltration). In all cases minimally invasive percutaneous transhepatic portography was adopted to treat this complication by mechanical fragmentation and pharmacological lysis of the thrombus. A vascular stent was also positioned in the two cases in which the thrombosis was related to a surgical technical problem. Mechanical fragmentation of the thrombus with contemporaneous local urokinase administration resulted in complete removal of the clot and allowed restoration of normal blood flow to the liver after a median follow-up of 37 months. PVT is an uncommon but severe complication after major surgery or liver transplantation. Surgical thrombectomy, with or without reconstruction of the portal vein, and retransplantation are characterized by important surgical morbidity and mortality. Based on our experience, minimally invasive percutaneous transhepatic portography should be considered an option toward successful recanalization of early PVT after major liver surgery including transplantation. Balloon dilatation and placement of a vascular stent could help to decrease the risk of recurrent thrombosis when a defective surgical technique is the reason for the thrombosis.

Keywords Early portal vein thrombosis · Thrombectomy · Percutaneous transhepatic portography · Minimally invasive angiographic approach · Vascular stent

Portal vein thrombosis (PVT) represents an uncommon but serious complication of major surgery, especially after orthotopic liver transplantation (OLT), occurring in up to 2.7% of cases [1, 2]. The development of early PVT can compromise patient survival due to acute liver failure. After OLT, early thrombosis increases the risk of graft loss requiring highly urgent retransplantation. PVT manifests clinically as ascites, variceal bleeding, diffuse abdominal pain, diarrhea, and hepatic dysfunction [3]. The diagnosis is established using clinical criteria, laboratory exams, abdominal color-Doppler ultrasound (US), which can distinguish a fresh thrombus, especially with contrast-enhancing agent, or CT scan. Contrast-enhanced three-dimensional magnetic resonance (MR) angiography has an accuracy for the detection of PVT comparable to that of digital subtraction angiography [4]. Causes of PVT are related to portal flow reduction, due to Budd-Chiari syndrome or local technical problems such as portal vein stenosis, bad positioning or kinking of the portal vein, and variations in standard end-to-end portal vein anastomosis during liver transplantation [5, 6]. Even splenectomy in patients with massive splenomegaly can result in PVT, with an incidence of 6.3% of cases [7, 8], due to hemodynamic alteration and low-flow regimen in the splenoportal axis. Early diagnosis and prompt treatment of PVT are crucial for the restoration of portal venous flow and

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reduction of morbidity and mortality. Different standard strategies for the treatment of PVT have been reported. They depend on the type and extension of PVT and the clinical symptoms (upper variceal bleeding, hepatic liver failure, and multiple organ failure) [9, 10]. In patients with underlying prothrombotic disorder, oral anticoagulation alone has evidenced a favorable benefit-risk ratio [9]. In patients with Budd-Chiari syndrome, transjugular intrahepatic portosystemic shunt (TIPS) placement seems to be effective and can be used as a bridge to liver transplantation; moreover, TIPS should even be recommended when cirrhosis and uncontrollable variceal bleeding are present [6]. Surgical thrombectomy has been the mainstay for the treatment of early PVT [11]. Moreover, early PVT after transplantation can result in graft loss with the need for high-urgent retransplantation. Surgical thrombectomy and retransplantation are hampered by high surgical morbidity and mortality and can also result in waste of highly precious grafts in an era of chronic organ shortage [12]. Interventional radiology by percutaneous approach may represent a valid and less invasive alternative to surgical treatment for decreasing surgically related morbidity and mortality and avoiding retransplantation [13, 14]. Herein we describe three cases of PVT, occurring within 30 days after liver transplantation or major hepatic surgery, that were successfully treated by a minimally invasive transhepatic percutaneous approach.

Case Reports

Case 1

A 52-year-old male affected by hepatitis C virus-related cirrhosis underwent ''piggyback'' OLT. The immediate postoperative course was characterized by normalization of liver function tests. On the 16th postoperative day, after severe diarrhea, abdominal pain, and ascites, an ultrasound Color-doppler scan revealed a complete thrombosis of the extrahepatic portal vein. Symptoms were associated with mild hyperbilirubineima (3 mg/dl) and low cholinesterase level (<2000 U/L). Continuous heparin infusion was immediately started at a dose of 20 IU/kg/h to maintain a partial thromboplastin time >60 s. On portal vein angiography the extent of PVT was localized to the common trunk of portal vein without affecting the intrahepatic branches. The thrombus was removed by mechanical fragmentation associated with simultaneous thrombolysis. Due to discrepancy between the ''donors'' and ''recipient'' portal vein caliber, we decided to insert a vascular stent to keep the portal vein patent. At the end of the procedure optimal portal blood flow to the liver was restored. Heparin therapy was continued until discharge on the 26th postoperative day, when the patient was started on oral anticoagulant treatment, achieving an international normalized ratio (INR) of between 2 and 2.5. After 5 years of follow-up, the patient is in good clinical condition, with normal graft function and a patent portal vein at color-Doppler follow-up.

Case 2

A 45-year-old female affected by hepatitis B cirrhosis underwent OLT using a piggyback technique. The recipient portal vein was patent, allowing an end-to-end anastomosis with the portal vein of the graft. Four months after OLT the patient presented with ascites. US examination revealed patent vascular anastomosis without dilation of the biliary tract. A selective arterial angiogram showed a markedly enlarged splenic artery, with diversion of the blood flow through it, and hypoperfusion of the graft. Partial occlusion of the splenic artery showed good restoration of flow to the graft, demonstrating a splenic artery steal syndrome. Thereafter the patient underwent splenectomy, was put on antiaggregation therapy, and was discharged 10 days after the operation with normal liver function tests. One month after splenectomy the patient presented with ascites associated with an increased in liver function tests and cholestasis. US examination revealed complete PVT. Continuous heparin infusion was immediately started at a dose of 20 IU/kg per hour to maintain a partial thromboplastin time of 60 to 80 s. Selective percutaneous portal vein angiography was performed, and the thrombus was removed by mechanical fragmentation with concomitant infusion of thrombolytic therapy. At the end of the procedure, optimal portal blood flow to the liver was restored. The clinical course was uneventful and no complications related to the procedure occurred; liver function tests return to normal values within 10 days. Heparin therapy was switched to oral anticoagulant therapy 6 days after the procedure, achieving an INR of between 2 and 2.5. After 4 years of follow-up the patient is in good clinical condition, with normal graft function and a patent portal vein.

Case 3

A 58-year-old male, affected by Klatskin tumor (type 3B), underwent extended right hepatectomy (I segment-V-VI-VII-VIII). Resection of a tract of the left branch of the portal vein was necessary due to tumor infiltration, and reconstruction was performed by end-to-end anastomosis. The immediate postoperative course was uneventful, with normal color-Doppler ultrasound and a patent left portal vein. On postoperative day 9, the patients developed liver function test deterioration, diffuse abdominal pain, and ascites. A CT scan of the abdomen evidenced thrombosis of the left branch of the portal vein. Continuous heparin

infusion was immediately started at a dose of 20 IU/kg per hour to maintain a partial thromboplastin time >60 s. Selective portography showed extension of the thrombosis up to the splenomesenteric portal axis (Fig. 1). The thrombus was removed by mechanical fragmentation associated with thrombolysis. A discrepancy of the portal vein caliber at the level of surgical reconstruction was evidenced; therefore a vascular stent was inserted to keep the portal vein patent and reduce the risk of relapse of the thrombosis. At the end of the procedure normal portal blood flow to the liver was restored (Fig. 2). No complications related to the procedure occurred; liver function tests returned to normal values within 10 days. Heparin therapy was switched to oral anticoagulation 3 days after the procedure, achieving an INR of 2–2.5. After 3 months of follow-up the patient is alive, with satisfactory liver function and a patent portal vein (Fig. 3).

Description of the Endovascular Technique

Selective portal vein angiography was carried out by percutaneous puncture (8 French × 24 cm; Arrow International Inc., Reading, PA) of an intrahepatic right branch or left branch in the case of right hepatectomy. The thrombus was removed by mechanical fragmentation using a Trerotola device (Arrow International Inc) and by contemporaneous thrombolysis with 200,000/300,000 IU of urokinase (A.I.C. CRINOS, Como, Italy) delivered locally through the percutaneously placed portal vein angiographic catheter, for 20 min. In two cases, hepatic resection and liver transplantation, a portal vein stenosis due to technical problems was evident; thereafter we decided to insert a vascular stent of 60- and 30-mm length, respectively (Saxx-BARD Inc., Covington, GA, USA) to keep the portal vein patent after angioplasty balloon dilatation to 10 mm. Stent positioning was not necessary in the patient with PVT occurring after splenectomy because no technical defects were responsible for the development of the thrombosis. At the end of the procedures optimal portal blood flow to the liver was restored in all cases. The percutaneous hepatic access (8 French) was sealed by two cylinders of collagen (Vaso Seal Vascular Hemostasis Device; Datascope Corp., Montvale, NJ, USA) in order to avoid bleeding or peritoneal bile leakage. In all cases heparin therapy was switched to oral anticoagulation between the 3rd and the 26th day after the procedure, achieving an INR of 2—2.5. Follow-up was performed with laboratory exams and color-Doppler ultrasound every day during the first week after the procedure and, subsequently, twice a week for the first month. Longer follow-up has always been done with color-Doppler ultrasound.

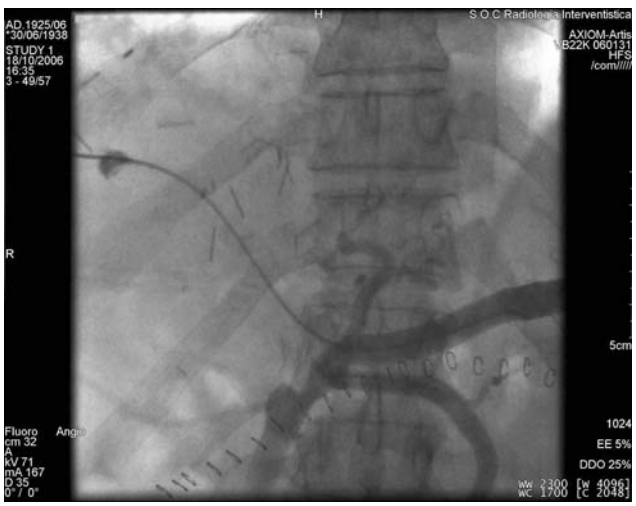

Fig. 1 Percutaneous transhepatic portography shows portal vein thrombosis extending to intrahepatic tree

Discussion

PVT is an uncommon but serious vascular complication related to major liver and splenic surgery, and also after liver transplantation, resulting in organ dysfunction and, ultimately, graft loss if not promptly diagnosed and treated. Causes of PVT are related to portal flow reduction, technical problems (stenosis, vessel malrotation, or kinking), and variations in standard end-to-end portal vein anastomosis during liver transplantation. Splenectomy in patients with splenomegaly is a risk factor for PVT, with an incidence of 6.3% of cases [2, 8, 15], due to alteration in the pattern of blood flow through the splenoportal axis. Early initiation of anticoagulation therapy for acute PVT is associated with complete and partial success in 40%–50% of patients [16]. However, recanalization of the portal vein with anticoagulation alone may not be consistent or appropriate in highly symptomatic patients. Surgical thrombectomy and, eventually, retransplantation are the two principal therapeutic options; however, both are hampered by significant morbidity and mortality [17]. Recently, several authors have reported the use of minimally invasive transhepatic approach to treat early PVT [18–21]. New therapeutic alternatives for the management of PVT include percutaneous transhepatic infusion of fibrinolytic agents, balloon dilatation, and stenting. Direct transhepatic portography allows precise determination of the degree of stenosis and extension of the thrombus within the portal vein, as well as pressure measurements [22]. Simple thrombotic occlusions in the absence of a surgical defect can be managed by mechanical fragmentation associated with pharmacologic thrombolysis [13–16]. Portal vein occlusions due to organized or refractory

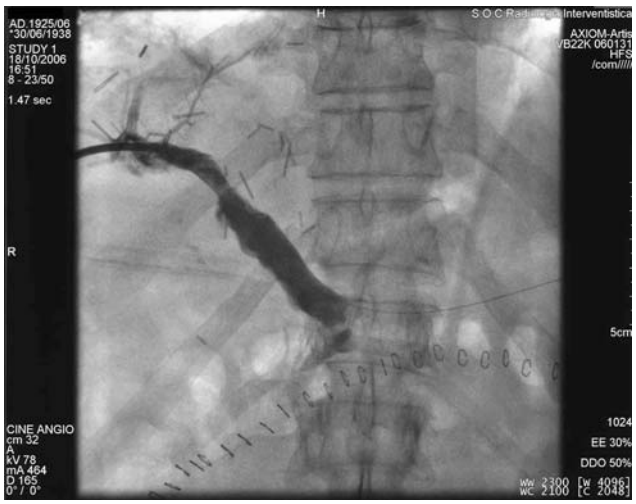

Fig. 2 Restoration of portal flow after thrombus removal performed with mechanical fragmentation and thrombolysis

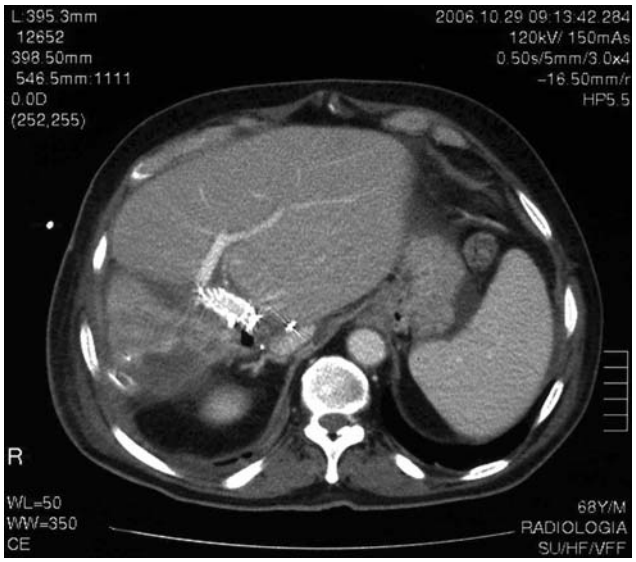

Fig. 3 CT scan 1 month after the procedure shows normal portal blood flow and the stent previously positioned

thrombus or associated with postsurgical stenosis are best corrected by balloon angioplasty and stent placement [21, 22]. Cherukuri et al. [23] reported two cases of PVT early after liver transplantation that were successfully treated by percutaneous thrombolysis and stent placement. In 1990, Olcott et al. [24] described a series of five cases of portal anastomotic stenosis treated with angioplasty, one of which included thrombolysis and metal stent placement. A series of pediatric liver transplant recipients with stenotic portal vein treated with angioplasty was reported by Funaki et al. [25]; metallic stents were placed in cases of elastic or recurrent stenoses and the stents remained patent at a mean follow-up of 15 months. Percutaneous thrombolysis can be performed in the case of early posttransplantation PVT as demonstrated also by Durham et al. [26], who successfully treated three cases at 19, 22, and 39 days after liver transplantation. In our experience, we treated three cases of early PVT after right hepatectomy with portal vein resection, splenectomy for massive splenomegaly, and transplantation with a minimally invasive angiographic approach, in order to reduce the morbidity and mortality due to surgical thrombectomy or retransplantation. All of our cases needed immediate treatment due to acute and severe deterioration of liver function, with a high risk of hepatic failure in the case of a conservative approach. Our patients developed PVT despite receiving prophylactic subcutaneous heparin postoperatively. Advantages of mechanical fragmentation of an early thrombus include the potential to rapidly remove the thrombus without the need for prolonged thrombolytic infusions and reduction of the potential life-threatening complications of thrombolytic therapy. The adoption of the described technique also allows avoidance of long-term (24- to 28-h) fibrinolysis, reducing the risk of bleeding. Balloon dilatation and placement of a vascular stent could be an important help to decrease the risk of recurrent thrombosis, especially in cases of discrepancy of the portal vein caliber or when a technical defect in the anastomosis was the reason for the thrombosis. The decision to perform portography trough a percutaneous and not a transjugular access was based on the operator's experience, avoiding the risk of creating an unnecessary communication between the hepatic vein and the portal vein system and is also technically less demanding in the case of PVT. Moreover, in our experience, the use of cylinders of collagen at the end of the procedure assures secure sealing of the percutaneous hepatic access, reducing the risk of peritoneal bleeding in patients with abnormal coagulation tests. In our patients the minimally invasive approach restored normal portal vein flow, with progressive normalization of hepatic function. No complications related to the procedure occurred; the absence of transaminase elevation immediately after the procedure suggested that fragmentation of the thrombus did not cause multiple small emboli in the peripheral portal vein. In conclusion, the cases reported herein confirmed the possibility of successfully recanalization of early PVT after both major liver surgery and transplantation by a minimally invasive transhepatic percutaneous angiographic approach.

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