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CASE REPORT

Successful percutaneous pulse spray thrombolysis of extensive acute portocaval hemitransposition thrombosis

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Summary

We report a case of extensive acute thrombosis of portal vein and cava in a portocaval hemitransposition liver graft that was treated successfully with percutaneous pulse spray thrombolysis through a femoral vein access. The patient subsequently developed descending and sigmoid colon ischemic necrosis because of the venous thrombosis necessitating emergency colon resection. The patient had prolonged postoperative intensive care stay, but was eventually discharged in a good condition with normal liver function. Three month follow up demonstrated persistent normalization of hepatic function and normal duplex ultrasound.

Introduction

Portocaval hemitransposition, first described by Tzakis *et al.* in 1998, was found to be a useful salvage procedure when hepatopetal flow to the liver graft cannot be established by other means in patients with extensive porto-mesenteric thrombosis [1–4].

Patients with portocaval hemitransposition have normal liver function and histology that is indistinguishable from those of conventional liver transplant [1].

One limitation of portocaval hemitransposition is the persistence of portal hypertension with the risk of developing variceal bleeding. The other limitation is the development of ascites which usually disappears in 4 months, but can lead to renal insufficiency [1,5]. Long-term anticoagulation is required because of the higher risk of delayed portal vein, cava, and lower extremity deep vein thrombosis [2].

Acute portal vein thrombosis in the conventional liver transplant is well described and was found to be associated with an increase in liver enzymes and prothrombin time, marked ascites, encephalopathy, enlarging gastro-esophageal varices, and variceal bleeding [5–7]. Acute portal vein thrombosis after portocaval hemitransposition, however, has not been reported in the literature to the best of our knowledge. The diagnosis of portal vein thrombosis can be confirmed using duplex ultrasonography and/or with angiography [8].

There have been successful reports of nonsurgical management of acute post-transplant portal vein thrombosis. Some of them were conservative when the patient and liver function were stable [6,7]. Others have used percutaneous thrombolysis with or without stent placement via transjugular or transhepatic approaches [9–12].

In the case described we took advantage of the unique anatomy of portocaval hemitransposition (Fig. 1) and performed percutaneous pulse spray thrombolysis via femoral vein access.

Case report

The patient is a 58-year-old Caucasian male, who was referred to us for evaluation of cryptogenic liver cirrhosis with extensive porto-mesenteric venous thrombosis diag-

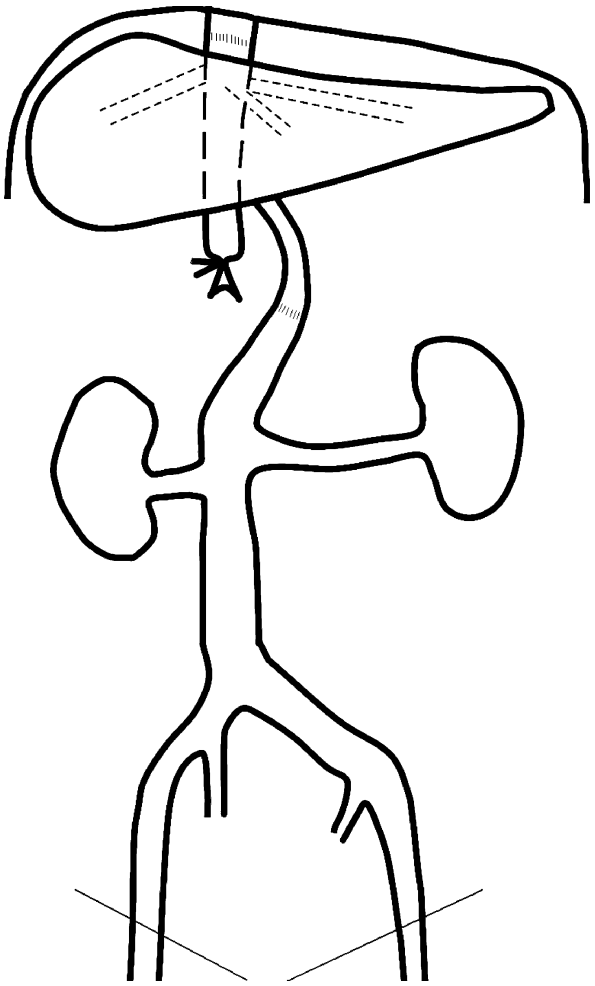

Figure 1 This figure shows the anatomy of portocaval hemitransposition. Suprahepatic cava of the donor is anastomosed end to end to recipient's suprahepatic cava. Recipient's infrahepatic vena cava is anastomosed end to end to donor portal vein. Donor infrahepatic cava is ligated.

nosed by duplex ultrasonography and confirmed by an angiogram. The patient did not have history of encephalopathy, denied gastrointestinal bleeding, but suffered fatigue, muscle wasting, grade 3 esophageal varices, gastric varices, and marked ascites. The patient past medical history was significant for stable pulmonary sarcoidosis diagnosed by lung biopsy 10 years earlier. He was seen by a hematologist and placed on warfarin (Coumadin®, Bristol-Myers Squibb Princeton, NJ, USA) after extensive workup for hypercoagulability of unknown etiology.

His Model for End Stage Liver Disease score was calculated to be 20 (serum creatinine: 1.57 mg/dl, total bilirubin: 3.6 mg/dl, International Normalized Ratio (INR): 1.57). He had an MRI of the abdomen that demonstrated a shrunken, nodular liver with multiple varicosities and thrombosed portal, superior mesenteric and splenic veins. He had a normal stress echo and was felt to be a candidate for liver transplantation.

The patient received a liver from a 34-year-old deceased donor after a difficult transplant. The portal vein was very small and chronically occluded, and no identifiable varix large enough for anastomosis to the donor portal vein was seen. A portocaval hemitransposition was performed with the suprahepatic donor cava anastomosed end to end to suprahepatic recipient cava and infrahepatic vena cava anastomosed end to end to the donor portal vein (Fig. 1). Arterial reconstruction was performed by anastomosing a patch of hepatic artery at the level of donor splenic artery to a patch at the level of recipient gastroduodenal artery. The biliary reconstruction was performed with a Roux-en-Y hepatico-jejunostomy. The patient received alemtuzumab (Campath®, Genzyme, Cambridge, MA, USA) for induction and was on tacrolimus (Prograf®, Astellas Pharma US, Inc., Deerfield, IL, USA) as maintenance immunosuppression therapy.

Postoperatively, the patient developed prerenal nonoliguric acute renal failure because of marked third spacing and ascites formation. This was treated with fluid and albumin replacement. On postoperative day 7 the patient was noted to have mild elevation of the liver enzymes, hematuria and a significant increase in ascites drainage from 1.5 to 5 l/day. Urgent duplex ultrasonography demonstrated portal vein and bilateral renal vein thrombosis.

Immediate arrangements were made, the patient was brought to the angiography suite and venogram, performed through the right femoral vein, demonstrated complete infrarenal occlusion of the inferior vena cava (IVC), consequently there was involvement of both renal veins (Fig. 2). We elected to proceed with the radiological intervention.

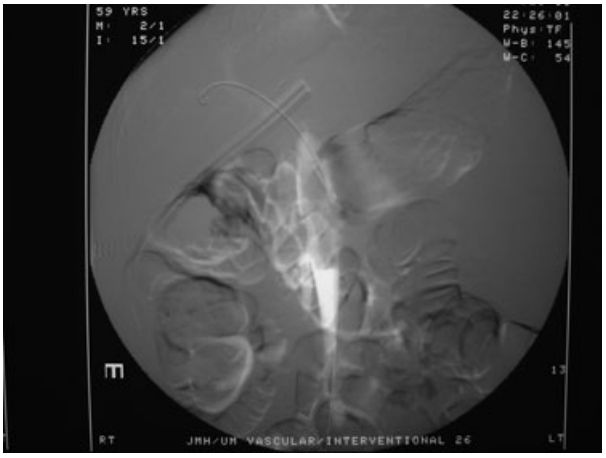

Figure 2 Venogram of the inferior vena cava (IVC) via right femoral vein catheter demonstrates complete occlusion of the infrarenal IVC.

As a result of the recent surgery, thrombectomy using a mechanical thrombectomy device (AngioJet® Thrombectomy system; Possis Medical, Inc., Minneapolis, MN, USA) was attempted. This was only partially successful so we elected to add the tissue plasminogen activator alteplase (Activase®; Genentech, Inc., Oceanside, CA, USA) and perform pulse spray thrombolysis. A total dose of 10 mg of alteplase resulted in marked resolution of thrombus in the IVC, renal veins, main portal vein and most of the 2nd and 3rd order intrahepatic portal venous radicals. An indwelling catheter was left for continuous thrombolysis at a rate of 0.25 mg/h overnight. Repeat venogram performed next day showed minimal improvement with persistent filling defects in multiple intrahepatic portal branches.

Mechanical and pulse spray thrombolysis using another 4 mg of alteplase, at the level of the intrahepatic portal branches was successful in further improving portal flow. Final angiogram demonstrated near complete resolution of the thrombus with some mild pruning of intrahepatic branches and the catheter was removed (Fig. 3). Postprocedure ultrasonography confirmed hepatopetal flow, and absence of thrombus in both renal veins. The patient was anticoagulated with heparin.

The following day, the patient developed hypotension, became anuric with marked metabolic acidosis, and rising lactic acid level. The abdomen was soft, but there was a change in the ascitic fluid color from serous to brownish reddish. An emergency laparotomy was performed and revealed necrosis of the descending and sigmoid colon, with no perforation. The gangrenous part of the colon was resected. The rectum was left as a Hartmann's pouch and the transverse colon was brought out as a terminal colostomy. Histology of the specimen revealed necrosis of the bowel with transmural edema and marked venous congestion supporting a venous thrombosis etiology.

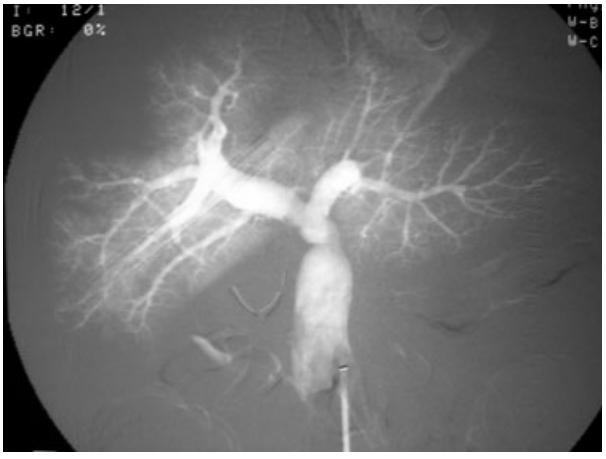

Figure 3 Post pulse spray mechanical thrombolysis and thrombolytic infusion, there is resolution of the inferior vena cava (IVC) thrombus, the IVC portal vein anastomosis is widely patent and majority of intrahepatic portal branches are perfused.

Subsequently, the patient after that had a complex postoperative course. Ascitic fluid sent at the time of exploration grew *Escherichia coli* (*E. coli*), for which he was placed on appropriate antibiotic coverage. He was in septic shock requiring vasopressors for about 2 weeks. He then developed *E. coli* pneumonia and was on the ventilator for another 45 days, because of repeated ventilator associated pneumonias and weakness. He eventually left the hospital in good condition, normal liver function and duplex ultrasound, and no clinically detectable ascites 3 months after transplant on Coumadin anticoagulation.

Discussion

The patient in our case developed early thrombosis of the portocaval reconstruction because of a combination of hypovolemia, from third spacing and ascites, and baseline hypercoagulability status.

The lesson to be learned in this case is the importance of starting proper anticoagulation postoperatively as early as feasible. Our patient unfortunately had subtherapeutic anticoagulation with heparin. The portal vein thrombosis was suspected by the sudden marked increase in the amount of ascitic fluid drainage and was confirmed by duplex sonography of the liver. The ultrasound also confirmed presence of bilateral renal vein thrombosis explaining the sudden onset hematuria. Portocaval hemitransposition has the unique anatomy of direct communication between the infrahepatic vena cava and the portal vein. Therefore allowing access via a femoral vein approach and avoiding the more invasive transhepatic or intrahepatic manipulation by transjugular approach and their complications.

Percutaneous mechanical fragmentation and pulse spray thrombolysis of the acute thrombosis were successful in our case and undoubtedly more advantageous (avoidance of general anesthesia, risk of bleeding, longer postoperative recovery, and postoperative morbidities) than surgical thrombectomy and redo of the portal vein anastomosis. The use of thrombolytic agents in combination with a mechanical thrombectomy device can be successful in treating portal vein thrombosis and limiting the duration of exposure to lytic agents thereby reducing the hemorrhagic complications, which are a concern in the recently postoperative patient.

Despite prompt diagnosis and treatment of the venous thrombosis, the patient developed colonic necrosis likely from venous congestion. Patients with extensive portomesenteric thrombosis have venous collaterals draining the bowel into the systemic veins, in this case probably

the iliacs, and thrombosis of which consequently has a high risk of bowel infarction from venous congestion. Certainly, something to remember in patients with portocaval hemitransposition.

Portocaval hemitransposition, unfortunately, does not resolve the portal hypertension problem. Our patient still needs medical treatment of the portal hypertension and frequent surveillance of esophageal varices.

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