

Portal Revascularization in the Setting of Cavernous Transformation Through a Paracholedocal Vein: A Case Report

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ABSTRACT

Diffuse thrombosis of the entire portal system (PVT) and cavernomatous transformation of the portal vein (CTPV) represents a demanding challenge in liver transplantation. We present the case of a patient with nodular regenerative hyperplasia and recurrent episodes of type B hepatic encephalopathy concomitant with PVT as well as CTPV, successfully treated with orthotopic liver transplantation. The portal inflow to the graft was carried out through the confluence of 2 thin paracholedochal varicose veins, obtaining good early graft function and recovery of the encephalopathic episodes. This alternative should be kept in mind as an option to assure hepatopetal splanchnic flow in those cases of diffuse thrombosis and cavernomatous transformation of portal vein.

THE COMPLICATIONS of diffuse portal-splenic and superior mesenteric vein thrombosis (PVT) with subsequent cavernous transformation of the portal vein (CTPV) in noncirrhotic patients represent a clinical challenge. When the medical and interventional procedures fail, liver transplantation (OLT) or combined liver–small bowel transplantation remain the ultimate option.^{1,2} Although several surgical alternatives have been described to overcome PVT and CTPV in clinical series, the absence of portal flow remains a challenge in OLT, for it increases the morbidity and mortality of the procedure.^{3,4} Herein we have presented a case of a successful OLT in a patient with PVT and CTPV. The portal inflow of the graft was achieved through the confluence of two pericholedochal veins (Petren's veins).

CASE REPORT

A 54-year-old man with complete thrombosis of the portal vein and nodular regenerative hyperplasia (NRH) was referred for a transjugular intrahepatic portosystemic shunt (TIPS) procedure. The diagnosis of PVT due to antiphospholipid antibody syndrome had been previously established during 2 episodes of abdominal pain. Doppler ultrasound and computed tomography confirmed the PVT diagnosis. The patient later developed esophageal varices and bled on 3 occasions despite /3-blocker administration, requiring sclerotherapy sessions.

Six months after the TIPS procedure, he suffered several episodes of portosystemic encephalopathy (PSE). The patient was treated with conservative medical therapy, administration of lactulose nonabsorbable antibiotics, and a low-protein diet. However, the encephalopathy symptoms persisted, and he underwent an endovascular shunt reduction without clinical improvement.⁵ OLT was indicated due to the progressively debilitating PSE.

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Transplant Procedure

OLT was performed using an 80-year-old isogroup ABO graft from a cadaveric donor. The complete portal vein thrombosis including a prior stent, extended to the superior mesenteric vein and splenic vein with cavernous transformation of the portal vein. After meticulous dissection of the liver hilum, we identified 2 thin-walled pericholedochal varices above the pancreas, which we formed into a common cloaca with a venoplasty at their confluence (Fig 1).

Despite the piggy-back technique, the native hepatectomy was technically demanding because of the presence of vascular adhesions.⁶ We ligated the recipient portal vein, which was replaced by a fibrotic, thrombosed cord. After completing the suprahepatic anastomosis in the usual fashion, the arterial inflow to the new liver was provided by an end-to-lateral anastomosis between the donor celiac axis and the recipient common hepatic artery. Once the liver was reperfused through the hepatic artery, an end-to-end portal anastomosis was constructed between the donor and common cloaca formed by the confluence of 2 pericholedochal veins, using a running suture of 6/0 Prolene, leaving a large growth factor of 1 cm. At the end of the procedure, arterial and portal flow were 300 and 800 mL/min, respectively (Medi-stim Butterfly Flowmeter. Medi-stim AS).

The patient experienced an uneventful postoperative outcome. Two postoperative Doppler ultrasound tests showed the patency and adequate flow in the hepatic artery and portal vein. The pathology report of the native liver confirmed the diagnosis of NRH with a chronic organized portal thrombosis containing an endothelialized partially patent Wallstent prosthesis. The patient was treated with prophylactic low-molecular-weight heparin and acetylsalicylic acid. He recovered from encephalopathy and has remained asymptomatic for 20 months posttransplant.

DISCUSSION

Complete thrombosis of the portal and splenomesenteric veins combined with cavernous transformation of the portal vein represents a surgically demanding challenge in the setting of OLT.⁴ Portal reconstruction in this setting needs to decrease portal hypertension, provide adequate “portal” flow to the liver and, as a consequence, reestablish hepatic metabolism of ammonia produced in the gut. Various alternatives have been reported to overcome PVT, including cavoportal hemitransposition (CPHT), renoportal anastomosis (RPA), and portal arterialization.^{3,4,7} Other authors have reported combined liver-intestinal transplantation with a 3-year survival rate around 60%.²

In our case, we decided to perform an end-to-end portal anastomosis to the confluence of 2 thin-walled paracholedochal collateral veins, which provided adequate early portal inflow (800 mL/min).

The formation of a fine reticular epicholedochal venous plexus in intimate contact with the paracholedochal veins (known as Petren’s veins) of the common bile duct has previously been reported in the setting of complete PVT.^{8,9}

In 1986, Hiatt et al⁹ described for the first time the use of a thin-walled varix as the inflow to the donor portal vein. The varix was identified inferolateral to the common bile duct just above the pancreas. The anastomosis remained patent after an 8-year follow-up.

The technique described herein for reconstruction of graft portal flow, (when possible,) offers advantages over the other previously mentioned options to reconstruct portal flow with systemic blood flow. In subjects undergoing CPHT and RPA, persistent portal hypertension has been described with the ongoing risks of bleeding, which occurred in 37% of reported cases, as well as of hepatic encephalopathy. Long-term effects of systemic venous flow to the graft are still unknown.

In contrast, hepatotrophic factors that originate in the intestines and pancreas are present in the

portal inflow to the liver is very well established, suggesting the benefit of maintaining the splanchnic drainage through the liver to avoid neuropathologic consequences of a portosystemic shunt, as occurred in our patient secondary to the TIPS procedure before transplantation.¹⁰

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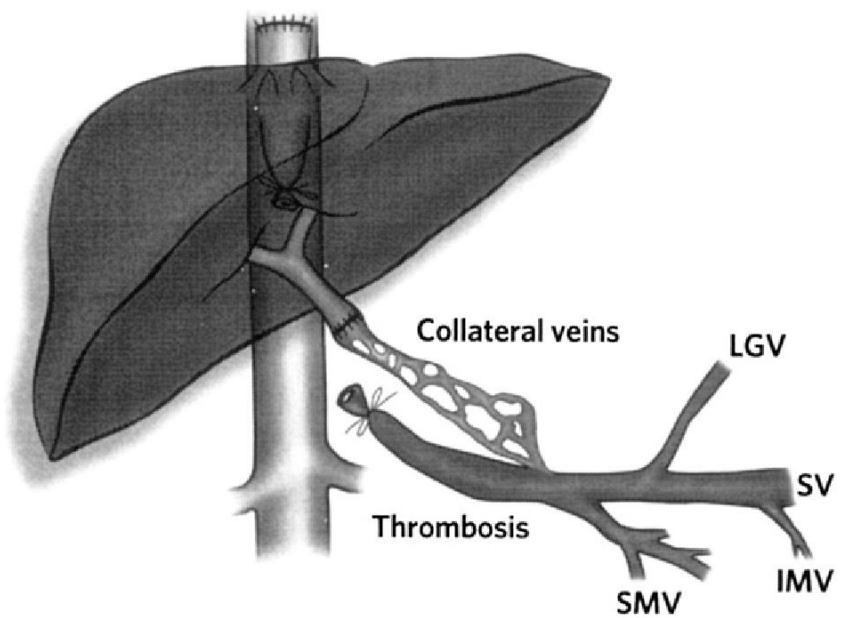


Fig 1. Schematic of CTPV found at surgery and paracholedochal varices and donor portal anastomosis. SMV, superior mesenteric vein; IMV, inferior mesenteric vein; SV, splenic vein; LGF, left gastric vein.