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BRIEF REPORT

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Hepatic artery interruption followed by portal vein thrombosis in an adult liver transplant

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Abstract Vascular complications following liver transplantation result in significant morbidity and mortality. We report a 28-year-old man who, because of a mycotic false aneurysm, underwent ligation of the hepatic artery 4.5 weeks post-transplantation and who, 4.5 months later, suffered a portal-mesenteric vein thrombosis. Adverse hepatic sequelae did not follow these events, demonstrating the capacity of the collateral circulation to perfuse the transplanted liver.

Key words Liver transplantation, mycotic aneurysm, portal vein thrombosis · Hepatic artery ligation, liver transplantation · Portal vein thrombosis, liver transplantation

Introduction

Vascular complications after liver transplantation include hepatic artery thrombosis, portal vein thrombosis and mycotic endovascular infections. These complications compromise the vascular supply of the transplanted liver, resulting in significant morbidity and mortality. Survival is dependent upon the development of compensatory collateral circulation. Following interruption of the hepatic artery, thrombosis of the portal vein is usually devastating. We report a 28-year-old man who survived hepatic artery ligation 4.5 weeks after liver transplantation and who developed a portal-mesenteric vein thrombosis 4.5 months later, yet maintained normal liver function.

Case history

Our patient developed portal hypertension in early childhood on the basis of congenital hepatic fibrosis (CHF). At age 7 he required portocaval shunting for upper gastrointestinal (GI) bleeding. His adolescence and early adulthood were punctuated by recurring episodes of variceal bleeding. By age 27, he was suffering recurrent hepatic encephalopathy and ascites. Other medical problems included polycystic kidney disease with renal tubular ectasia, mild chronic renal failure, nephrocalcinosis and renal stones.

Liver transplantation was undertaken. The donor was a motor vehicle accident victim with no significant medical history. The donor allograft had a normal appearance and perfusion characteristics; cold ischemia time was 18 h. Intraoperatively, the recipient's native spleen and liver were inseparable and so were removed together. Packing of the abdomen was necessary to achieve hemostasis and the bile duct was drained externally. Follow-up laparotomy

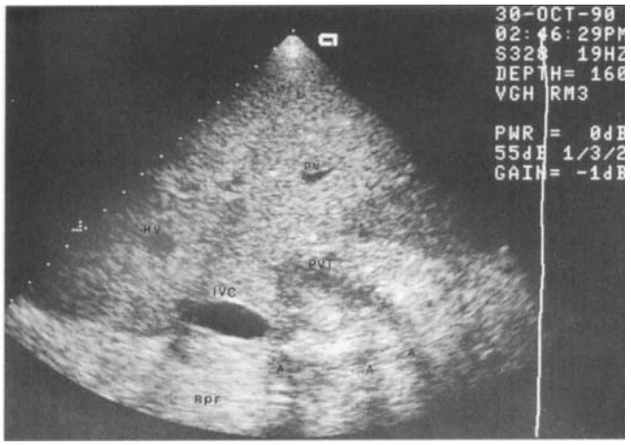

Fig. 1 Hepatic ultrasound demonstrating a portal vein thrombosis *(PVT)*. Note the increased echogenicity of clot within the portal vein *(PV)* compared to the inferior vena cava *(IVC)*. (*L* liver, *HV* hepatic vein, *RPF* retroperitoneal fat, *A* artifact)

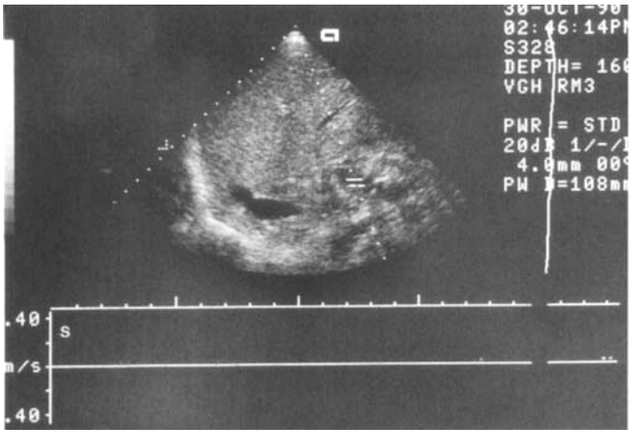

Fig. 2 Doppler flow ultrasound demonstrating lack of flow within the portal vein because of the portal vein thrombosis *(PVT)*. Note the Doppler flow gate within the *PVT (two horizontal hash lines)* and the absence of Doppler signal within the scale *(S)*, indicating absence of flow

was undertaken 48 h later to remove packing and reconstruct the biliary tree with a Roux-en-Y anastomosis.

Two and a half weeks post-transplantation, the patient began spiking fevers. *Candida* was cultured from several sites and treatment with amphotericin B was instituted. Four weeks post-transplantation, the patient suffered a massive GI bleed. Ultrasonography (US) suggested a hepatic artery aneurysm; angiography revealed a large false aneurysm. Emergency surgery was undertaken. A false aneurysm in excess of 10 cm in diameter was discovered communicating with the distal end of the previously oversewn bilde duct. The hepatic artery was ligated proximal to the anastomosis.

Postoperatively the patient had a seizure and was treated with phenytoin. Cytomegalovirus (CMV) encephalitis was suspected and a course of ganciclovir administered. Liver function remained unremarkable. A transient elevation in serum transaminases to 800 U/l returned to near-normal levels within days. Immunosuppression consisted of cyclosporin A and prednisone. There was no evidence of rejection. At discharge the serum AST was 60 U/l (< 40 U/l), ALT 48 U/l (< 60 U/l), and bilirubin 28 μmol/l (< 23 μmol/l). US revealed normal portal flow.

Four and a half months later, the patient presented with abdominal pain and vomiting. Liver enzymes were: serum AST 20 U/l, ALT 84 U/l, alkaline phosphatase 200 U/l (< 105 U/l), total bilirubin 14 μmol/l and prothrombin time (PT) 13.2 s (12–13 s). A doppler flow US (Figs. 1, 2) revealed no flow in the main and right portal veins, questionable flow in the left portal vein, and normal flow in the inferior vena cava. The findings were consistent with portal vein thrombosis. The liver otherwise appeared unremarkable; ascites was present. An abdominal aortogram and celiac and superior mesenteric arteriography were performed; they revealed no mesenteric or portal flow despite injection of high-dose nitroglycerin, acting as a vasodilator, into the superior mesenteric artery. Arterial collateral flow was seen filling the hepatic artery distal to the ligation via small vessels originating from the gastroduodenal artery inferiorly and from numerous vessels off the celiac axis at the porta hepatis (Fig. 3). The proximal gastroduodenal artery was occluded but filled retrograde via the inferior pancreaticoduodenal and dorsal pancreatic arteries. No portal collateral vessels were noted.

Laparotomy was undertaken and confirmed a portal-mesenteric vein thrombosis. Fifteen centimeters of infarcted small bowel was resected. A liver biopsy revealed mild chronic rejection only. Postoperatively the patient's liver enzymes remained unchanged. The patient signed out against medical advice but remains well more than a year later without obvious gastroesophageal bleeding.

Discussion

This patient underwent hepatic artery (HA) ligation for the management of a mycotic false aneurysm, an uncommon [6, 8, 9] and often fatal [6, 8] vascular complication of transplantation, without suffering significant sequelae. Mycotic involvement of the HA is thought to be the result of contamination during the creation of the choledochojejunostomy [9]. Surgical options include ligating the HA [12] or attempting a vascular reconstruction in an infected field [8, 9]. Houssin et al. ligated two patients with mycotic HA aneurysms. Unlike our patient, their first patient developed a delayed bile leak but eventually recovered; their second patient died of septic shock [8].

The outcome of HA ligation after liver transplantation is similar to that of acute hepatic artery thrombosis (HAT). In the nontransplant patient, interruption of the HA is usually not associated with significant hepatic sequelae. HA collateral flow is rapidly established [1, 11] and there is a compensatory increase in oxygen uptake from the portal venous circulation [11]. The patient with a transplanted liver, however, represents a different situation because of both disruption of pre-existing collateral channels and ongoing allograft rejection [17]. The well-described consequences of acute post-transplant HAT include (1) fulminant liver failure; (2) delayed biliary leak, as the distal common bile duct is completely dependent on the HA; and (3) relapsing bacteremia [17].

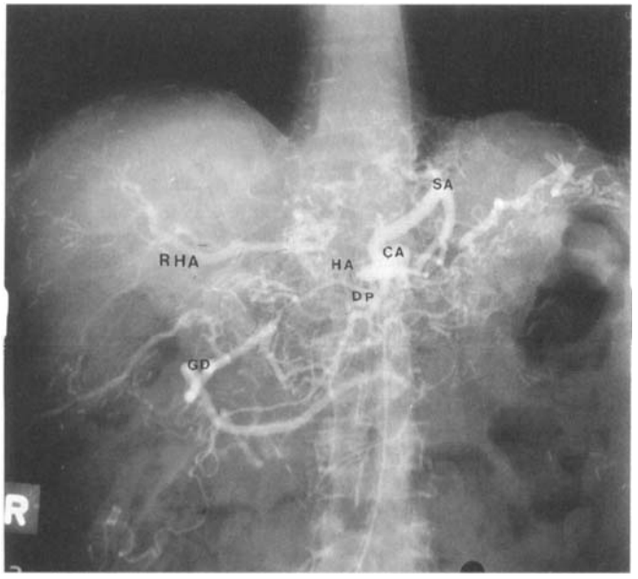

Fig. 3 Celiac angiogram 4.5 months after hepatic artery ligation revealing proximal interruption of the hepatic artery *(HA)* with distal filling of the right hepatic artery *(RHA)* via numerous small vessels from the celiac axis *(CA)* and inferiorly via the gastroduodenal artery *(GD)*. (*SA* splenic artery, *DP* dorsal pancreatic artery)

Our patient did not suffer any of these consequences. His outcome was similar to those of asymptomatic patients with HAT, presumably occurring gradually, found incidently at screening US [7]. This benign course is attributed to the establishment of HA collaterals.

Although the establishment of HA collateral flow occurs within hours to days in the case of nontransplant HA ligation [1, 11], the exact time frame in which adequate collateral circulation is established in liver allografts is unknown. In pediatric allograft recipients with HAT, collateral circulation has been documented angiographically within 2 weeks of transplantation [18]. This has not been demonstrated in adults [18].

Our patient suffered a portal-mesenteric vein thrombosis 4.5 months after transplantation. Angiography demonstrated the establishment of HA collateral flow that was sufficient enough to compensate and allow perfusion of his allograft. Portal vein thrombosis (PVT) is an uncommon complication of liver transplantation compared to HAT. Langas et al. cited an incidence of 1% compared to 6% for HAT [9]. Wozney et al. described five cases in 104 semi-emergent and emergent post-transplant angiograms [18]. In these series most cases occurred in pediatric patients. The reasons cited for PVT after transplantation include acute and chronic rejection [14], surgical technique, previous portal vein surgery, thrombus formation from the portal venous bypass cannula and disorders of hypercoagulability [18]. Both pretransplant portosystemic surgical shunting and splenectomy have been suggested as predisposing factors [10], although Langnas et al. described only one case of acute PVT in 18 patients with previous portal-systemic surgical shunts [9]. Our patient with a previous portocaval shunt, created in childhood, presented with a late PVT. Liver biopsy at the time of PVT revealed only mild chronic rejection. Although protein C, protein S and antithrombin III levels were not assayed, the donor had no history of recurrent thromboses. It should be noted that protein C, protein S, and antithrombin III levels can decline in the post-transplant period but should return to normal by the 7th postoperative day [4, 15]. PVT is occasionally diagnosed incidentally, although patients can present with liver failure, intestinal swelling or complications of portal hypertension, usually bleeding [18]. Our patient presented with abdominal pain secondary to a mesenteric venous small bowel infarct. Management of PVT, depending on the clinical circumstances, includes observation [2], thrombectomy in acute PVT, with or without vein grafting [9], retransplantation [9, 18] and treatment of portal hypertensive complications alone (usually sclerotherapy or portosystemic shunting) [9, 14, 16, 18]. Without specific management of the PVT, hepatopetal and hepatofugal collaterals will eventually develop [3]. Helling has reported one case of portal vein recanalization, however, portal hypertension persisted [5].

Our patient suffered interruption of both the HA and later the portal vein; graft survival was possible due to the development of adequate collateral circulation. Reed et al. reported two similar cases in his series. In both cases, HAT was the cause of HA interruption [13]. Their two cases likewise demonstrated adequate HA collateral flow, however, surgical portosystemic shunting was required.

In conclusion, our patient's remarkably benign course after interruption of the hepatic arterial circulation 4.5 weeks after liver transplantation demonstrates that this event is not always followed by morbid consequences. The continued maintenance of good liver function following later portal vein thrombosis in this patient is evidence of the compensatory capacity of the collateral circulation to perfuse the post-transplant liver.

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