Case Report of Multiple Bilobar Hepatic Arterio-Portal Fistulas Post–Liver Transplantation Managed Conservatively

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ABSTRACT

Multiple intrahepatic arterio-portal fistulas are rare. The majority are isolated and occur secondary to liver trauma including iatrogenic interventions such as liver biopsy. Post–liver transplantation 18 cases have been reported, all secondary to an interventional radiological procedure. We report multiple bi-lobar arterio-portal fistulas in a liver transplant recipient recognized 1 year after transplantation. The donor died due to intracerebral bleeding following blunt head and abdominal trauma. In the present case, the etiology is not very clear. The patient was managed conservatively and to date has not required intervention.

ARTERIO-PORTAL fistulas (APFs) are a condition characterized by abnormal communications between the hepatic artery and the portal vein that occur most commonly secondary to liver surgery, trauma, transhepatic intervention or biopsy. The majority are asymptomatic and diagnosed incidentally on imaging. However, APF can be responsible for life-threatening hemobilia [1].

APF are usually divided into three types: (1) small peripheral intrahepatic (type 1); (2) large central (type 2); and (3) diffuse congenital intrahepatic (type 3) [2]. Differentiating between small peripheral intrahepatic APFs and large central APFs is helpful in that the former usually resolve spontaneously, whereas the latter may cause portal hypertension and hepatic parenchymal changes. Congenital APFs (type 3) are intrahepatic and can be difficult to manage. Iatrogenic APFs are reported most commonly after liver biopsy, and less commonly following percutaneous trans-hepatic cholangiogram (PTC) and trans-jugular intra-hepatic portosystemic shunt (TIPS) and rarely from trans-arterial chemoembolization (TACE) for hepatocellular carcinoma [2].

During the process of liver transplantation, livers undergo a significant number of potential interventions, including liver biopsy. The appearance of APF in the post-transplantation setting is not common. The precise incidence is unknown but in a retrospective series reported by Saad et al APFs were identified in 5.4% of angiograms performed in post–liver transplantation patients [3]. Only solitary APFs have been described in this report.

CASE REPORT

A 59-year-old white male was listed for liver transplantation for end-stage liver disease due to hepatitis B, having previously presented with variceal bleeding and sub-acute bacterial peritonitis. He had undergone splenectomy at 5 years of age for hereditary spherocytosis. At listing his Model for End-stage Liver Disease (MELD) score was 27.

He received a liver transplant from a 70-year-old donation after cardiac death (DCD) donor who had suffered an intracerebral hemorrhage following blunt abdominal and head trauma after an assault. Warm ischemic time was 22 minutess and the cold ischaemic time was 7 hours. Computed tomography (CT) scan of the abdomen before retrieval was reported as unremarkable. At multi-organ retrieval, it was noted that there was a significant hematoma within the small bowel mesentery with an injury to the liver at the entry point of the falciform ligament. There were no other visible injuries and the liver appeared macroscopically normal. Two biopsy specimens prereperfusion and postreperfusion were taken from the left lobe. The prereperfusion biopsy specimen showed mild fibrous expansion of portal tracts with no steatosis or cholestasis. The postperfusion biopsy specimen showed features compatible with a preservation/reperfusion injury.

The liver reperfused normally. Post-transplantation serum aspartate aminotransferase (AST) peaked at 2686 IU/L (normal serum AST 10–50 IU/L). He was discharged home at 2 weeks on tacrolimus-based immunosuppression with normal liver function tests.

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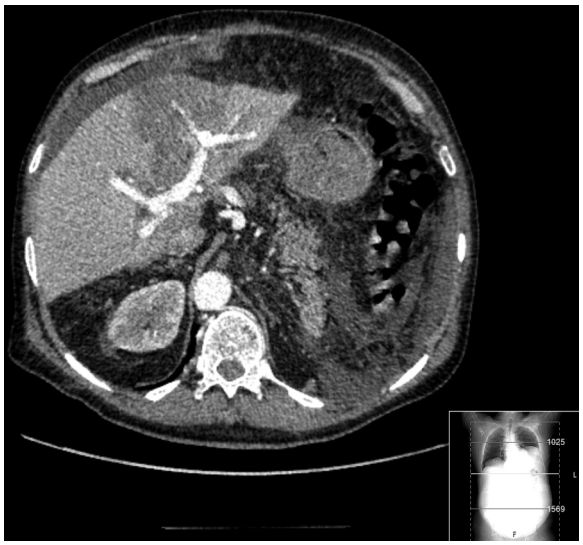

Fig 1. CT 14 months post-transplantation showing portal vein opacification in arterial phase with marked steatosis on left hemi-liver with ascites.

One-month post-transplantation he was re-admitted because of deranged liver function with a serum AST of 2070 IU/L. Liver ultrasound showed attenuated portal venous flow. CT angiography demonstrated normal vasculature with dilated intrahepatic biliary radicals. Magnetic resonance cholangiopancreatography (MRCP) revealed an anastomotic stricture that was dilated and stented by endoscopic retrograde cholangiopancreatography (ERCP). He was discharged 2 days later.

After a further 2 weeks, the serum AST went up from 27 IU/L to 174 IU/L. Percutaneous liver biopsy was performed from the right lobe, which demonstrated mild portal inflammation and was indeterminate for acute cellular rejection. There was also focal cholangiolitic change with no evidence of cholestasis.

The patient's liver function tests settled and he was well for 12 months before re-presenting with ascites. Ultrasound confirmed the presence of ascites and reversed portal flow with dilated intrahepatic biliary radicals. Repeat magnetic resonance imaging (MRI)

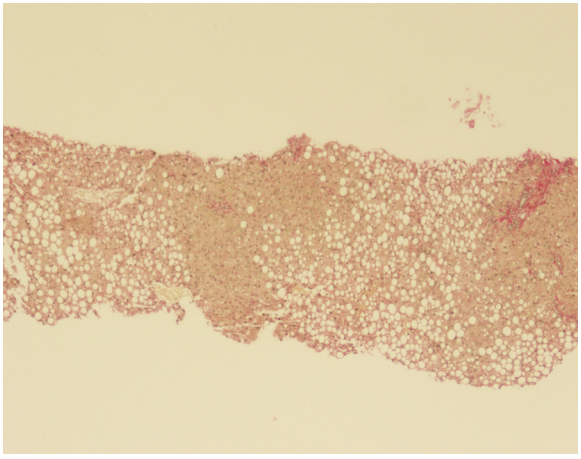

Fig 3. Sirius red staining of left lobe showing no fibrosis and significant steatosis.

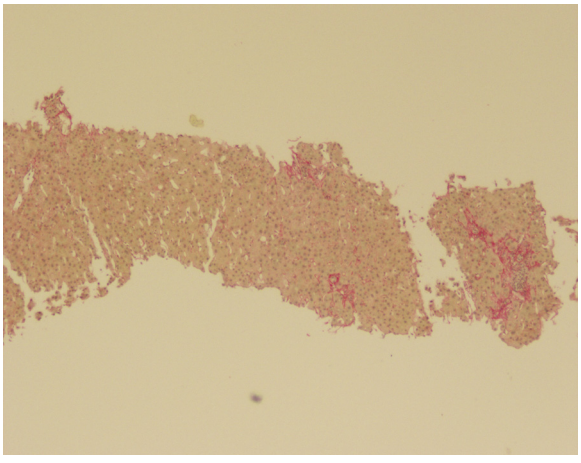

Fig 4. Sirius red staining of right lobe showing no steatosis and no fibrosis.

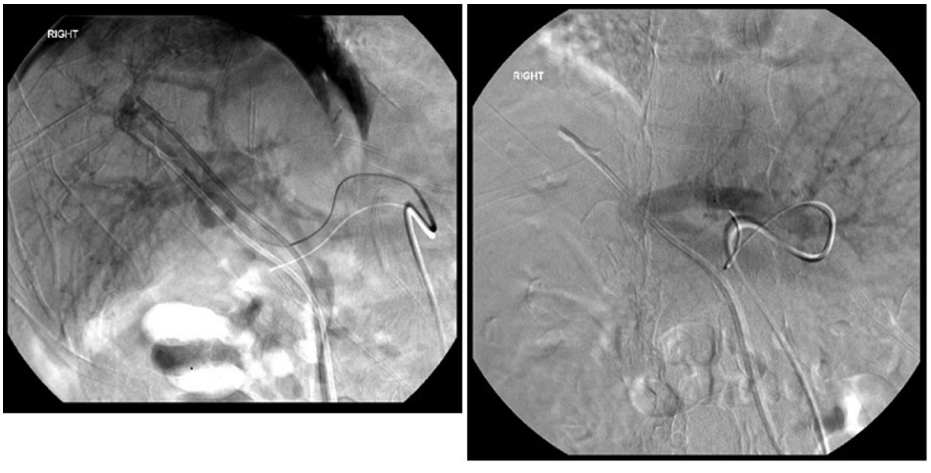

Fig 2. Angiography of right and left hepatic artery showing multiple peripheral shunts to portal vein bilaterally.