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Case Report

Liver retransplantation due to splenic artery steal syndrome in a patient with common hepatic artery arising from the superior mesenteric artery

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ABSTRACT

Splenic artery steal syndrome, (SASS) is a controversial cause of hepatic artery (HA) hypoperfusion attributed either to a decreased HA flow due to deviation to the splenic artery or to HA vasoconstriction in response to the increased portal flow due to the hepatic arterial buffer response (HABR). Herein we report a case of SASS that showed an anatomic variant of a replaced common hepatic artery (CHA), originating exclusively from the superior mesenteric artery (SMA) that was treated successfully with splenic artery ligation. These findings support, at least in this case, that the etiology of graft dysfunction is the increased portal flow rather than the deviation of the hepatic artery flow to the splenic artery.

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Introduction

Splenic artery steal syndrome, (SASS) is a controversial cause of hepatic artery (HA) hypoperfusion, and is a serious complication of liver transplantation, occurring in 0.6–10.1 % of patients [1]. Initially, SASS was defined as decreased HA flow due to deviation to the splenic artery [2]. Subsequently, the decreased hepatic artery flow was attributed to HA vasoconstriction in response to the increased portal flow due to hepatic arterial buffer response (HABR) [3]. Herein we report a case of SASS that showed an anatomic variant of the common hepatic artery (CHA), which originated from the superior mesenteric artery (SMA), supporting the increased portal flow theory.

Case Report

A male 53-year-old patient, suffering from decompensated primary biliary cirrhosis with Model of End Stage Liver Disease (MELD) score of 21, encephalopathy and splenomegaly, underwent a whole liver transplantation in June 2011, with the piggy back technique. The donor was a 62 years old female and the graft had 7 hours of cold ischemia without significant steatosis. The patient had a replaced common hepatic artery, originating from the superior mesenteric artery and an arterial anastomosis was performed with the celiac artery of the hepatic graft.

After a prolonged admission in the ICU, due to an infection with a multidrug resistant *acinetobacter baumannii*, the patient was transferred to the ward on the 8th postoperative day. He also presented with an early cytomegalovirus viremia and renal insufficiency and, finally, left the hospital on 38th postoperative day. The immunosuppressive regimen consisted of cyclosporine, mycophenolate mofetil and methylprednisolone. Postoperatively, the patient presented a good graft function but the hypersplenism worsened. On the 3rd postoperative month, liver function tests showed a persistent elevation aspartate aminotransferase (AST) 188 U/L, alanine aminotransferase (ALT) 208 U/L. A liver biopsy was not diagnostic, and the condition was attributed to cytomegalovirus hepatitis due to the elevation of anti-cytomegalovirus IgM antibodies, in combination with the previous cytomegalovirus viremia, and to the transient improvement with the administration of valganciclovir. The size of the spleen and the hypersplenism syndrome increased and finally a cholestatic pattern was established with stenosis of the common bile duct, sludge and recurrent episodes of cholangitis. A liver biopsy revealed cholestasis and confluent centrilobular necrosis, an endoscopic cholangiopancreatography revealed stenosis of the bile duct anastomosis and the common, the right and left hepatic ducts and two stents were inserted in the left and right hepatic bile duct, but the patient was improved only transiently. A CT angiography demonstrated a replaced common hepatic

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artery (CHA), originating from the superior mesenteric artery (SMA) with delayed arterial flow and no peripheral filling of the hepatic arterial distribution, without stenosis of the arterial anastomosis. In contrast, the splenic artery (SA) was very enlarged and was filling peripherally into the spleen (Figure 1). These findings suggested SASS, but the patient had established end stage liver disease with a total bilirubin (TB) of 23.4 mg/dL, aspartate aminotransferase (AST) 388 U/L, alanine aminotransferase (ALT) 408 U/L, gamma glutamyl transpeptidase (GGT) 587 U/L, and alkaline phosphatase (AP) 457 U/L, with a serum creatinine value of 1.59 mg/dL, an international normalized ratio (INR) 1.49, and a MELD score of 27.

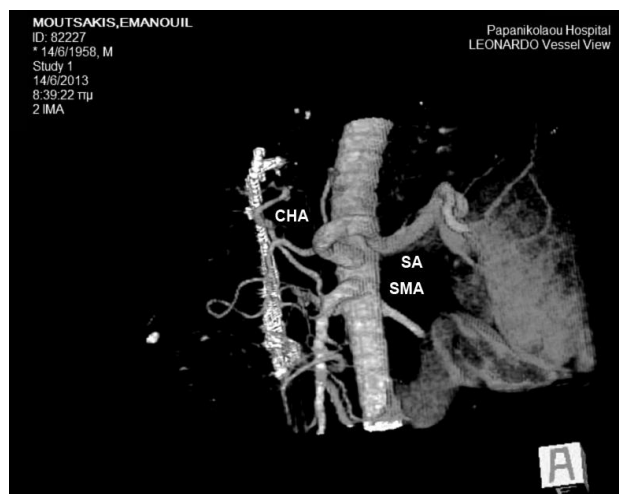


Figure 1: CT angiography demonstrating a replaced common hepatic artery (CHA), originating from the superior mesenteric artery (SMA) with delayed arterial flow and no peripheral filling of the hepatic arterial distribution, without stenosis of the arterial anastomosis. In contrast, the splenic artery (SA) is very enlarged and perfuses the splenic parenchyma abundantly.

The patient was retransplanted on August 2013 with the piggy back technique. An arterial anastomosis was performed between the celiac artery of the graft and the replaced common hepatic artery (CHA), originating from the SMA of the recipient. Graft dysfunction was confirmed on the 1st postoperative day. Color and duplex doppler ultrasound (General Electric Logiq P6) assessed the peak systolic velocity (PSV) and resistivity index (RI) of the hepatic artery and maximal portal vein velocity (PVV). It revealed a pathological flow signal of the hepatic artery with an elevated RI of 1.0. The portal vein showed significant hyper perfusion with a maximum systolic velocity of 115 cm/sec. The 2nd postoperative day the patient was reoperated and the HA was found patent but with weak pulsation which increased considerably after the temporary clamping of the splenic artery. Therefore, the HA hypoperfusion was attributed to SASS despite the fact that the replaced CHA originated from the SMA. Ligation of the splenic

artery was selected as treatment, due to the severe condition of the patient. The intraoperative duplex doppler ultrasound assessment confirmed significant reduction of the maximal portal vein velocity and augmentation of the peak systolic velocity of the hepatic artery. The graft function improved and the 4th postoperative day the duplex ultrasound depicted a regular flow profile of the hepatic artery (RI = 0.65). The portal vein showed a normalized flow signal with a mean velocity of 30 cm/sec. The following days the graft function improved, and the patient was extubated.

Discussion

There are numerous causes of reduced arterial flow to a liver graft with a patent hepatic artery. These include deviation of the hepatic arterial flow to an enlarged splenic artery (splenic artery steal syndrome), functional reduction of hepatic arterial flow in response to hyperdynamic portal flow (through the hepatic artery buffer response, HABF), small hepatic graft relative to normal portal flow (relative increase of portal flow) and increased peripheral resistance in the hepatic arterial bed. Hepatic artery hypoperfusion presents nonspecifically in the early posttransplant period as graft dysfunction with abnormal liver function tests, biliary anastomotic stricture, ascites and, if overlooked, may lead to graft failure [1, 4].

This case demonstrated that the ligation of the splenic artery decreased the flow of the portal vein and increased the flow in the hepatic artery of the graft, despite the fact that the origin of its flow was exclusively from the replaced CHA, originating from the SMA of the recipient. These findings support, at least in this case, that the etiology of graft dysfunction is the increased portal flow and not the deviation of the hepatic artery flow to splenic artery. In addition, SASS can be considered as the overlooked etiology of the deterioration of the first graft which led to retransplantation, due to biliary ischemic lesions and hepatic failure.

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