*Brief reports*

TRANSPLANT International


Transplant Int (1991) 4: 122–124

# Arterial steal: an unusual cause for hepatic hypoperfusion after liver transplantation

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Received August 7, 1990/Received after revision November 13, 1990/Accepted November 19, 1990

**Abstract.** Case reports of two patients with an unusual cause for a rapid increase in transaminases following liver transplantation are described. In the postoperative course, angiography revealed an arterial hypoperfusion of the liver due to a steal phenomenon with blood shunting from the hepatic to the splenic artery. In one case, the underlying pathophysiology was a pre-existing filiform stenosis of the celiac trunk with insufficient recruitment of arterial blood from the superior mesenteric artery via the pancreatic arcade. Adequate liver perfusion was restored by simple ligation of the common hepatic artery. In the other case, angiography showed an arteriovenous fistula formation of the splenic vessels and minimal blood flow through the hepatic vessels. This was successfully corrected by angiographic embolization of the splenic artery with metal coils. After therapeutic intervention, both patients rapidly recovered with excellent liver function.

**Key words:** Arterial steal, in liver transplantation – Liver transplantation, arterial steal – Ischemia, in liver transplantation

Acute occlusion of the hepatic artery following liver transplantation is usually caused by arterial thrombosis and in most cases leads to acute graft loss with the need for urgent retransplantation [11]. The mortality from this complication can reach 50% [9]. Incomplete ischemia due to hypoperfusion of the liver is caused by pre-existing anatomical, postinflammatory, or surgically induced, arterial stenosis [4]. Arterial hypoperfusion after transplantation can be followed by similar damage to the grafted liver. However, graft loss may evolve less rapidly compared to acute and complete arterial occlusion.

We report on two patients with an unusual cause for hepatic hypoperfusion after liver transplantation.

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## Case reports

### *Case 1*

A 36-year-old male patient underwent orthotopic liver transplantation for primary biliary cirrhosis. The patient suffered from progressive liver disease with bilirubin serum levels exceeding 50 mg%, portal hypertension with considerable splenomegaly ($20 \times 12$ cm$^2$) and bleeding from esophageal varices. Following recipient hepatectomy, the new organ was implanted using standard techniques: the common hepatic artery of the graft was anastomosed in an end-to-end fashion to the proper hepatic artery of the recipient using 7–0 prolene sutures. The cold ischemia time of the liver after preservation with UW solution [1] was 7 h.

Immediately after transplantation, an extremely high level of ALAT and ASAT (up to 1000 IU/l) became evident without a tendency for normalization. Furthermore, serum bilirubin remained persistently elevated. Under the clinical suspicion of vascular thrombosis, an angiography was performed which revealed a previously undetected stenosis of the recipient's celiac trunk. In addition, a considerable arterial steal was noticed by the enlarged spleen with retrograde blood flow in the common hepatic artery. Most of this flow was

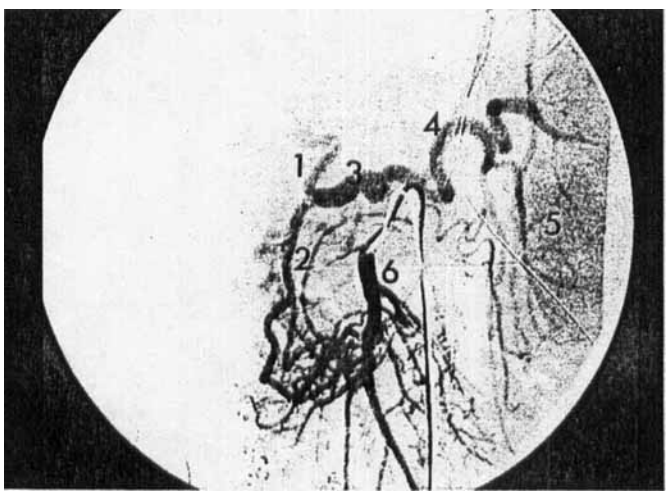


**Fig. 1.** *Case 1.* Postoperative angiography. The catheter is situated in the superior mesenteric artery. *1* Proper hepatic artery; *2* gastroduodenal artery; *3* common hepatic artery; *4* splenic artery; *5* enlarged spleen; *6* superior mesenteric artery

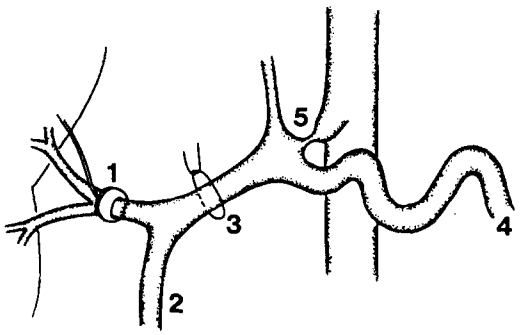


**Fig. 2.** *Case 1.* Intraoperative aspect at relaparotomy. *1* Proper hepatic artery with flowmeter; *2* gastroduodenal artery; *3* common hepatic artery; *4* splenic artery; *5* celiac trunk with severe stenosis

recruited from the superior mesenteric artery via the pancreaticoduodenal arcade (Fig. 1). As a consequence, the nutritive perfusion of the liver was significantly reduced.

Immediate relaparotomy was performed and the arterial steal confirmed by intraoperative electromagnetic flowmetry of the proper hepatic artery (Fig. 2). Hepatic flow was 120 ml/min before, and 280 ml/min after, clamping of the common hepatic artery. The splenic steal was corrected by ligation of the common hepatic artery and liver perfusion restored. Liver function improved rapidly following this procedure with normalization of routine biochemical parameters within 3 days. Currently the patient is alive and well 12 months after transplantation.

*Case 2*

A 27-year-old male patient with hemophilia A had developed liver cirrhosis due to chronic non-A non-B hepatitis with acute liver failure. Ten years previously, splenectomy had been performed for thrombocytopenia. Orthotopic liver transplantation was carried out urgently as described for Case 1. The cold ischemia time was 6 h. The early postoperative course was uneventful. In the third week after transplantation, a massive increase in the transaminases (ALAT 1180 IU/l and ASAT 1320 IU/l) occurred within 24 h.

Angiography was rapidly performed, revealing an aneurysm and arteriovenous fistula of the splenic vessels with a consecutive arterial steal from the hepatic artery (Fig. 3). The splenic artery was embolized and the fistula occluded using metal coils (Fig. 4). Following embolization, angiography demonstrated improved arterial perfusion of the liver. Liver enzymes and function returned to normal within 5 days after this procedure. The patient recovered without complications and is currently well 3 months after transplantation.

## Discussion

Hepatic artery complications after liver transplantation are an important cause of morbidity and mortality [2, 6, 10]. Adequate arterial perfusion of the transplanted liver is of critical importance, since the liver is exposed to considerable perfusion and reperfusion damage after preservation. Furthermore, all arterial colaterals of the liver are interrupted following graft hepatectomy and cannot develop as rapidly as in non-transplant patients [3]. Hypoperfusion of the transplanted liver leads to hepatic cell damage with increased transaminase levels. Reduced arterial blood flow is usually caused by kinking, coiling, technical anastomotic stenosis ('maiden's waist') or by arterial thrombosis [12].

A pre-existing asymptomatic stenosis of the celiac trunk is rare in otherwise healthy individuals. Stenosis of the proximal celiac trunk, associated with dilation of the gastroduodenal artery has occasionally been found in patients with severe portal hypertension [5]. It is known that the diagnosis of celiac trunk stenosis is difficult and can be missed if a lateral abdominal angiography is not performed [7, 8].

Our first patient had routine preoperative angiography, but the lesion of the celiac trunk was not detected before surgery. In this case, hepatic blood flow following transplantation was derived from the superior mesenteric artery via the inferior and superior pancreaticoduodenal arteries. However, despite a large caliber common hepatic artery, most of the blood flow was shunted from the liver to the enlarged spleen. This resulted in hypoperfusion of the transplanted liver. We speculate that reperfusion edema with subsequently increased intrahepatic resistance was an additional pathogenetic factor. This steal effect was successfully treated by simple ligation of the common hepatic artery.

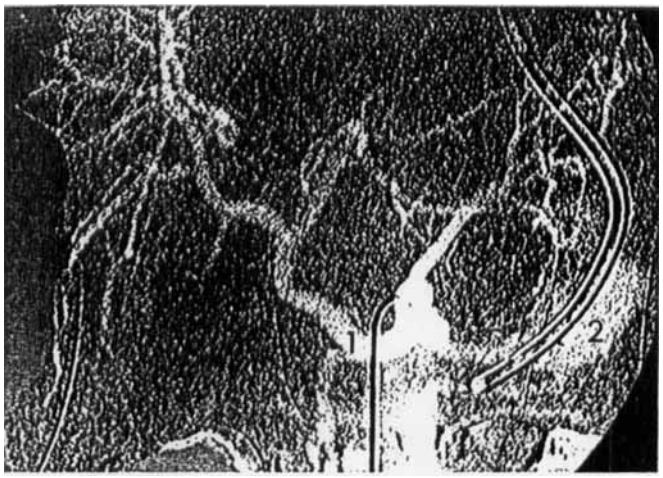


**Fig. 3.** *Case 2.* Postoperative angiography. The catheter is situated in the celiac trunk. *1* Hepatic artery; *2* splenic artery with aneurysm

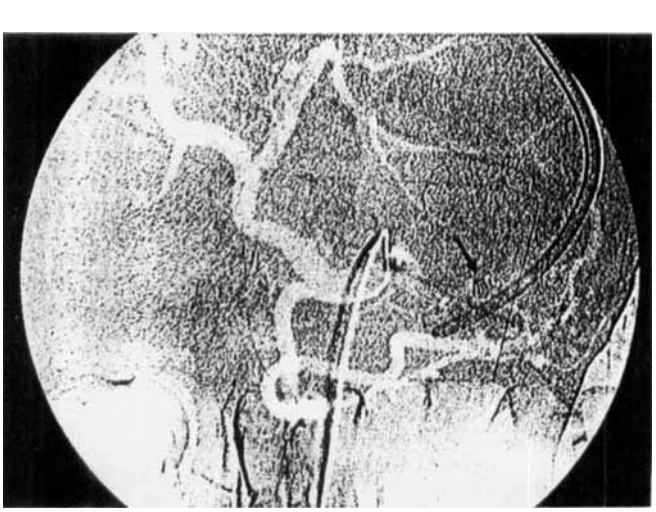

**Fig. 4.** *Case 2.* Angiography after embolization of the splenic artery with metal coils. The splenic artery is now occluded and hepatic perfusion is improved

In the second case, hepatic blood flow was reduced due to an arteriovenous fistula of the splenic vessels after splenectomy many years previously. The splenic artery was occluded by angiographic embolization which restored a sufficient liver blood supply. The origin and date of the fistula formation remained unclear, because no preoperative angiography had been performed in the emergency situation of fulminant hepatic failure. However, since the fistula became hemodynamically evident by rapid increase in transaminases as late as 3 weeks after transplantation, the fistula most likely developed in the late postoperative period.

We believe that the early diagnosis and correction of the hepatic hypoperfusion saved the liver grafts in both cases. This underlines the crucial role of the arterial liver perfusion after transplantation. Our cases emphasize the importance of detailed preoperative angiography for diagnosis of preformed vascular abnormalities. Furthermore, the value of postoperative angiography for early detection of vascular complications is stressed in order to allow specific therapeutic intervention.

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