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BRIEF REPORT

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Intraoperative measurement of graft blood flow – a necessity in liver transplantation

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Abstract Portal venous and hepatic arterial flow was measured intraoperatively in the 70 most recent patients undergoing liver transplantation in our institution. Impaired graft flow due to vascular abnormalities was detected in six patients. One patient suffered from arterial steal due to stenosis of the recipient celiac trunk with blood shunting from the hepatic to the splenic artery. Ligation of the recipient hepatic artery restored the arterial graft flow. In two patients we found reduced portal venous flow due to large portosystemic collaterals. The collaterals accountable for the impaired portal flow were identified and ligated, which restored portal venous graft flow. Excessive sensitivity of the portal venous flow to the position of the graft was found in a 6-month-old boy. Portal venous flow varied considerably, depending upon the position of the graft, and intraoperative flow measurement allowed the best position of the graft to be identified. Two patients developed arterial thrombosis in the early postoperative course. Immediate laparatomy with thrombectomy resulted in good, palpable pulsation in the graft artery in both patients. Intraoperative flow measurement demonstrated satisfactory arterial flow in one patient, whereas there was no net flow in the other patient's graft artery. Pulsation in this patient was caused by blood oscillating in and out of the liver. In conclusion, we find that causes of primary graft dysfunction due to technically flawed reperfusion of the graft can be identified and alleviated by intraoperative measurement of the flow in the graft vessels.

Key words Liver transplantation, intraoperative blood flow · Blood flow, intraoperative, liver transplantation

Introduction

Adequate perfusion of the graft is crucial in liver transplantation. Although the success rate has improved in recent years, primary graft dysfunction is still a significant problem, occurring in 10 %–15 % of all liver transplant recipients [10, 11]. It is a serious complication in the early postoperative period that requires prompt identification. Reasons for early graft dysfunction include ischemic injury, technically imperfect revascularization of the graft, hyperacute rejection, and disease in the donor liver [12]. The latter two are rare causes [13].

In our 70 most recent patients, we have routinely used ultrasound transit time flow probes and flowmeter (Medistim, Norway) to intraoperatively measure the flow in the graft portal vein and hepatic artery. This method has enabled us not only to demonstrate defective flow, but also to identify exact causes of the abnormal flow and to take steps to correct it. We illustrate this in six case reports.

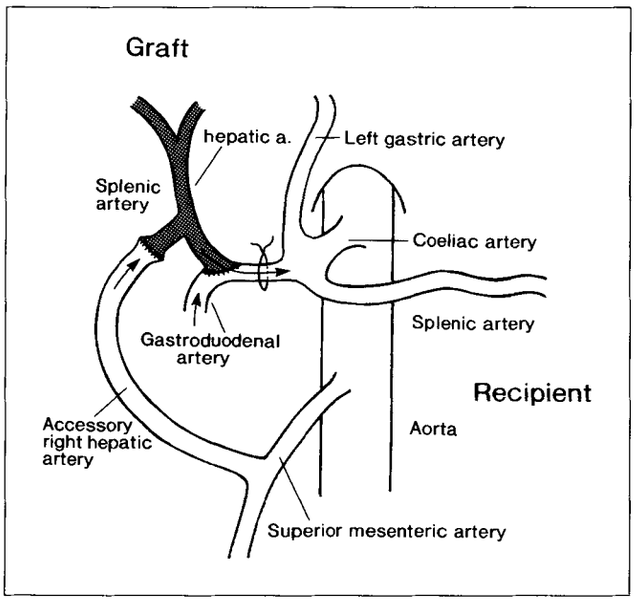

Fig. 1 Arterial reconstruction. Sequential clamping of the three recipient arteries involved in the reconstruction revealed the flow directions indicated by *arrows*. Ligation of recipient common hepatic artery restored graft arterial flow

Case reports

Case 1

A 48-year-old woman underwent transplantation due to cryptogenic cirrhosis. The anatomy of the donor graft was normal and the recipient had an accessory right hepatic artery from the superior mesenteric artery (SMA). Arterial reconstruction was achieved with an end-to-side anastomosis between donor celiac trunk with a Carrel patch of the aorta and the common hepatic artery and the gastroduodenal artery of the recipient. After declamping, the arterial flow in the graft artery was inadequate – 128 ml/min – and the recipient right accessory artery of the SMA was anastomosed end-to-end to the stump of the donor splenic artery (Fig. 1). Arterial flow in the graft artery was still low – 144 ml/min – but the flow in the recipient hepatic artery, gastroduodenal artery, and right accessory artery was 276 ml/min, 219 ml/min, and 358 ml/min, respectively. The only possible explanation was retrograde flow in one of the recipient arteries (Fig. 1). Selective clamping of the recipient arteries demonstrated that arterial flow in the graft was normal – 525 ml/min – when the recipient common hepatic artery was clamped. The recipient common hepatic artery was tied and the operation was concluded. The postoperative course was unremarkable.

Case 2

A 48-year-old man underwent combined double lung and liver transplantation due to chronic obstructive lung disease and cryptogenic cirrhosis. The lungs were transplanted first. At the time the liver graft was reperfused, the patient was stable with good respiratory and cardiac functions. The liver graft looked well-perfused and normal, and the portal vein looked and felt normal. Portal flow, however, was very poor – 145 ml/min. Ligation of huge portosystemic collaterals along the lesser curvature of the stomach boosted the portal flow to ± 700 ml/min. However, this flow was still too low in relation to cardiac output. Another collateral of considerable dimension was found to the azygos system. After ligation of this collateral, portal flow increased to 1.5 l/min. Postoperative graft function was good.

Case 3

A 42-year-old woman underwent transplantation due to a hemangioendothelioma. Portal flow after reperfusion was low, ± 350 ml/min. Collaterals along the lesser curvature and a collateral between the portal vein at the site of the confluence and the caval vein were ligated, and the portal flow increased to ± 2 l/min. The postoperative course was uneventful.

Case 4

A 6-month-old boy underwent transplantation due to biliary atresia after an unsuccessful Kasai operation at the age of 8 weeks. The left lobe from a 7-year-old donor was used. The weight match was 5.6 kg/22 kg. Portal venous reconstruction was accomplished between donor portal vein and the confluence of the superior mesenteric and splenic veins of the recipient. The initial flow in the portal vein of the graft was satisfying – 175 ml/min – but after reconstruction of the arterial system, it was reduced to ± 20 ml/min. Portal flow was found to be very sensitive to the position of the graft; it was restored to "normal" after a change was made in the position of the graft. The postoperative course was uncomplicated.

Case 5

A 12-year-old boy underwent transplantation for acute liver failure due to Wilson's disease. A full size liver graft from a 32-year-old donor was used. The transplant procedure was uneventful. The boy was extubated with good graft function 24 h post-transplantation. On day 4, there was a significant increase in S-ALT to 2450 and S-LDH to 16500. Ultrasonic Doppler investigation demonstrated good flow in the portal and hepatic veins, whereas no arterial signal was found. An emergency laparotomy revealed no flow in the graft artery due to thrombosis. A thrombectomy was carried out and the graft artery was flushed with heparinized saline. After thrombectomy, a good pulsation could be felt in the graft artery; flow measurement, however, showed that blood was oscillating in and out of the graft artery. Thus, the net flow was zero. Several attempts were made to flush the intrahepatic arterial system, and although a very good pulsation could be felt, flow measurement still revealed no net flow. The boy was listed for an emergency retransplantation, which was performed 2 days later. The course after the retransplantation was uneventful.

Case 6

An 18-months-old girl developed fulminant hepatic failure. Liver biopsy revealed changes consistent with hepatitis, but all viral markers were negative. An emergency liver transplantation was performed using a left lateral segment from a 51-year-old donor. The immediate postoperative course was uneventful. The girl had a good recovery and was extubated after 48 h. On the 4th day post-transplantation, no arterial signal was found at Doppler inves-

Table 1 Initial abnormal flow reading after graft reperfusion. Problem identified from the abnormal flow reading, action taken, and final flow reading

Case	Initial flow	Problem identified	Action	Outcome
1	Low arterial graft flow	Arterial steal phenomenon	Ligation of recipient common hepatic artery	Normalization of arterial graft flow
2	Low portal venous graft flow	Open large portosystemic collaterals	Identification and ligation of collaterals	Normalization of portal venous graft flow
3	Low portal venous graft flow	Open large portosystemic collaterals	Identification and ligation of collaterals	Normalization of portal venous graft flow
4	Low portal venous graft flow	Extreme sensitivity of graft portal venous flow to position of graft	Optimal position identified	Normalization of portal venous graft flow
5	No arterial flow	Arterial thrombosis	Thrombectomy	No net flow despite good palpable pulse in graft artery → retransplantation
6	No arterial flow	Arterial thrombosis	Thrombectomy	Good arterial graft flow

tigation (although it had been normal the day before), and ALT and LDH were three times higher than on the previous days. An emergency laparatomy was performed, and a fresh clot was removed from the graft artery. The graft was flushed with heparinized saline. A good pulse was felt in the graft artery and an adequate arterial flow was measured. The flow curve was normal with normal acceleration, indicating that there were no flow-limiting factors proximal to the site of flow measurement. The flow in the portal vein was unremarkable. The girl had a good recovery, her biochemical parameters were normalized, and daily Doppler investigations revealed a normal arterial signal. The girl is now 6 months post-transplantation with normal arterial graft flow.

Discussion

Hepatic artery complications after liver transplantation are major causes of post-transplant morbidity and mortality [7]. The liver graft relies entirely on blood supplied by the anastomosed artery, as all arterial collaterals are divided during hepatectomy and develop slowly after transplantation [2]. The most common causes of arterial flow problems are technical anastomotic strictures, kinking, and coiling, yet problems arising from kinking or coiling of the artery often become evident only after the abdomen is closed. By leaving the flow probes on the graft vessels and pulling the abdominal skin flaps together before final closure, these causes of arterial flow problems can be seen and corrected. A technical stenosis of the anastomosis should not occur if a surgical technique has been carried out competently; however, a low arterial graft flow can be tested for anastomotic cause by clamping of the portal vein. If the anastomosis is not the flow-limiting factor, a rise in arterial graft flow will be seen due to the hepatic arterial buffer response [5], which has been shown to be present in the transplanted liver [3, 9]. The shape of the flow curve will most often reveal an anastomotic stricture.

More unusual causes of arterial graft hypoperfusion include arterial steal, due to stenosis of the celiac trunk or the celiac compression syndrome. Two cases of arterial steal due to stenosis of the celiac trunk were described by Manner et al. [6]. The diagnosis was made by arteriography due to rapidly increasing enzymes following transplantation. A simple ligation of the common hepatic artery corrected the arterial graft flow. The use of intraoperative flow measurement permits instant diagnosis and correction of this problem, as demonstrated in case 1 of this study. Celiac compression syndrome has been diagnosed in 10 % of patients undergoing liver transplantation [4] and is suspected when arterial graft flow is low or absent in the expiratory phase of the respiratory cycle. Arterial graft flow may be corrected in these patients by dividing the muscular and fibrous bands on top of the supraceliac aorta. This syndrome can only be diagnosed by routine measurement of graft arterial flow. The clinical importance of the syndrome is unknown.

Arterial thrombosis requires instant intervention and correction of the arterial graft flow. If adequate flow is not obtained following thrombectomy, retransplantation is necessary. Two patients developed arterial thrombosis since we started routine intraoperative flow measurement. Both had normal arterial flow after reperfusion of the graft. Emergency laparatomy was done in each patient, and it was possible to obtain good palpable pulsation of the graft artery after thrombectomy and flushing of the graft with heparinized saline. In one patient, however, intraoperative measurement revealed no net flow. The satisfactory, palpable pulsation of the graft artery was found to be due to blood oscillating in and out of the graft artery. In the other patient, intraoperative flow measurement demonstrated that arterial graft flow had been restored to normal values with an intact arterial buffer response after clamping of the portal vein. The first patient continued to deteriorate until he was retransplanted 2 days later. The second patient has a well-perfused graft now, 6 month later. We therefore feel that intraoperative measurement gives invaluable information following thrombectomy after graft arterial thrombosis.

There are very few reports demonstrating the ideal portal venous flow after transplantation but, from the

limited information available, portal venous flow equal to or higher than normal should be expected in the newly transplanted liver [3, 8]. This is also our experince. In two patients we found a portal flow following reperfusion that was much lower than what is normally seen. Identification and ligation of portosystemic collaterals responsible for most of the portal circulation increased the portal venous graft flow more than fivefold. It has been demonstrated that large, open portosystemic shunts after liver transplantation are associated with higher AST levels in the first 2 weeks, indicating a possible detrimental effect of spontaneous portosystemic shunts on graft perfusion [1]. Especially during acute rejection, De Carlis et al. [1] found large, reopened shunts causing ischemic damage to the graft due to a "steal phenomenon". Peroperative flow measurement is an effective way to identify patients who still have significant open portosystemic shunts after reperfusion of the graft.

The extensive development of portosystemic collaterals poses a special problem in small children with portal hypertension. A minor outflow obstruction due to the position of the graft and kinking of the top caval anastomosis will produce an increase in hepatic venous pressure. Extensive portosystemic collaterals prevent stasis of the portal venous system and, thus, a new hepatic portosystemic pressure gradient will not be built up. An outflow problem in these patients may consequently cause permanent impairment of hepatic portal flow, with a subsequent risk of primary graft dysfunction or graft infarction from portal thrombosis. Resection of the recipient portal vein and portal anastomosis to the confluence will break many, but not all, collaterals. Arteriography during a late venous phase may demonstrate major collaterals that can be closed before reperfusion of the graft. The top caval anastomosis should be fashioned with the graft and recipient vessels as short as possible, but even with these precautions, hepatic portal flow may be very dependent on the position of the graft. Intraoperative flow measurement demonstrates the best position of the graft, and the position can be tested with the flow probes in situ when the skin flaps are pulled together before closure.

From our experience with intraoperative measurement of absolute flow in the graft vessels, we believe that the causes of technically imperfect perfusion of the graft can be identified and instantly corrected so that primary graft dysfunction or graft infarction can be avoided.

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