Transpl Int (1997) 10: 392–394

BRIEF REPORT

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Renal-splenic shunt for infrahepatic caval occlusion after piggy-back liver transplantation

Received: 17 January 1997
Received after revision: 2 May 1997
Accepted: 13 May 1997

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Abstract Inferior vena cava thrombosis after liver transplantation is uncommon. We describe a case of this unusual complication occurring after piggy-back (end-to-side) graft implantation. Renal failure, lower limb edema, and hemodynamic instability were the presenting symptoms requiring immediate surgical correction with a left renal-to-splenic vein shunt over a ringed 2.5-cm prosthesis. The decision to go ahead with the shunt was preceded by an intraoperative confirmation of a 10-cm H_2O pressure gradient between the caval and portal circulations. This gradient, unlike that observed in liver cirrhosis, ultimately turned a splenorenal shunt into a renal-splenic one. Six months after the procedure, the patient is alive and well with normal liver and renal function. The technique described may be useful in the management of other clinical conditions of acute infrahepatic caval hypertension.

Key words Liver transplantation, vena cava thrombosis, splenorenal shunt

Introduction

Vascular complications are among the main causes of morbidity and mortality after liver transplantation. Hepatic artery thrombosis is the most frequent [11], portal vein thrombosis is less common (1.8 %–2 % of cases), and thrombosis of the inferior vena cava (IVC) is observed in only 0.5 % of cases [9].

A broad variety of surgical alternatives have been proposed for the reconstruction of vena cava continuity during liver transplantation [2, 13]. Herein, we describe a procedure commonly used in the correction of portal hypertension – the splenorenal shunt – that has been successfully adapted for the correction of lower vena cava hypertension brought about by a recurrent native cava thrombosis in a patient whose liver had been anastomosed end-to-side, according to the piggy-back technique. This sort of "renal-splenic shunt" may be useful in the management of other clinical conditions of acute infrahepatic caval hypertension.

Case report

A 57-year-old man with a Child B liver failure underwent an identical blood type liver transplantation for a hepatitis C virus-related liver cirrhosis associated with a small hepatocellular carcinoma. During total hepatectomy, the patient had to be placed on venovenous bypass since a large caudate lobe made the dissection of the infrahepatic vena cava difficult. Contrary to expectations, the native vena cava was preserved, and the liver graft was anastomosed end-to-side following the piggy-back technique [4]. There was an apparent discrepancy in graft size since the donor liver weighed 1960 g and the explanted liver 1430 g. Portal and arterial anastomoses were performed end-to-end and biliary continuity was established through a Roux-en-Y choledochojejunostomy.

The early postoperative period was unremarkable, but on the 2nd post-transplant day, renal function started to deteriorate, with serum creatinine and blood urea nitrogen (BUN) increasing in 2 days to 4.2 and 199 mg/dl, respectively. Because of fluid retention and hyperkalemia, the patient was placed on continuous veno-venous hemodialysis while the liver outflow and function continued to be preserved. At this point, native retrohepatic cava thrombosis was suspected and the patient was taken to the operating room. At laparatomy, the retrohepatic IVC was found to be obstructed by a

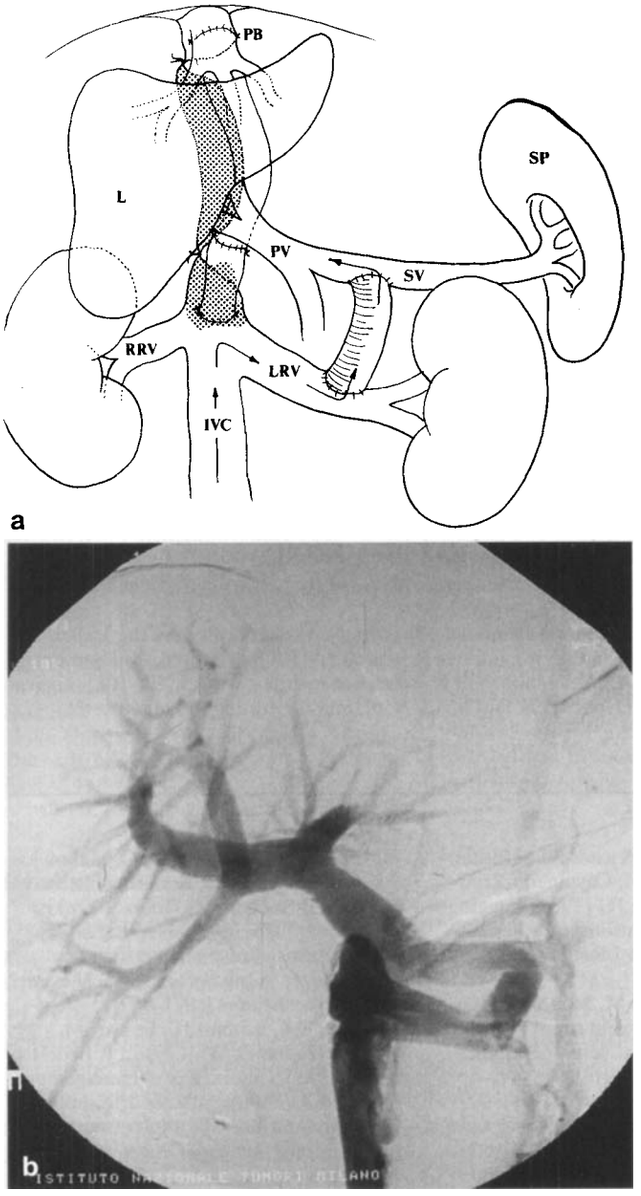

Fig. 1 a, b The renal-splenic shunt: **a** schematic representation of the shunt with flow direction (*arrows*) from inferior vena cava and left renal vein to the splenic and portal vein. Ultimately, both portal and caval flows pass through the liver graft; **b** caval angiogram showing patent renal-splenic shunt 4 months after surgery (*PB* piggy-back (end-to-side) caval anastomoses, *L* liver, *PV* portal vein, *IVC* inferior vena cava, *RRV* right renal vein, *LRV* left renal vein, *SP* spleen, *SV* splenic vein, *shaded areas* post-transplant side of thrombosis of the infrahepatic native vena cava

thrombus 3–4 cm long that originated just behind the piggy-back anastomosis and extended downward to the level of the renal veins. The native retrohepatic IVC was then ligated above and below the thrombus and the caval blood flow was restored through an iliac vein graft placed as a bridge between the recipient and the donor IVC. Urine production resumed and creatinine and BUN normalized within 5 days.

On day 12 renal function deteriorated again, and anuria developed rapidly with progressive edema of the limbs, perineum, and flanks, requiring further dialysis and ICU support. Caval angiogram showed a recurrent thrombotic obstruction of the recipient IVC and absence of flow through a stenotic iliac graft-caval anastomosis.

During surgery the approach to the hepatic hilum and the site of vein graft thrombosis was extremely difficult due to the severe edema and tissue swelling. However, dissection away from the hepatic hilum was possible, and we were able to measure a consistent pressure gradient between the lower cava at the level of the renal veins (25 cm H_2O) and the portal vein (15 cm H_2O). We therefore made a "reverse flow" splenorenal shunt using a 1-cm-wide ringed Goretex segment placed between the left renal vein and the splenic vein (Fig. 1 a). At the end of surgery, a flow of about 1800 ml/min was passing through the 2.5-cm-long segment in a caval-to-portal direction (intraoperative Doppler measurement).

Postoperative dialysis was continued for 3 more days while renal function progressively returned to normal, and slight ascites resolved within a week. On day 33, serum creatinine and BUN were 1.3 and 73 mg/dl, respectively, and a ^{99m}Tc-DTPA renal scan showed a bilateral delayed excretion compatible with resolving acute tubular necrosis. Nutritional and rehabilitation support were necessary for 2 more weeks. The patient was able to leave the hospital 48 days after transplantation under cyclosporin and steroid immunosuppression.

Six months later, the patient is alive with normal liver and kidney function. There are no signs of esophageal varices or rejection, and a caval angiogram showed the shunt to be patent with optimal blood flow (Fig. 1 b).

Discussion

Maintenance of the venous return to the heart during the anhepatic phase of liver transplantation can be achieved through an external, pump-driven venovenous bypass. As an alternative, preservation of the retrohepatic vena cava has been recommended, with venous outflow from the graft restored through an end-to-side anastomosis [13] (the so-called piggy-back technique) or through a side-to-side connection [2].

In the case presented herein, an unforeseen surgical risk was run by our insistence on doing a piggy-back anastomosis even though the patient was on external bypass. In retrospect, even if the end-to-side anastomosis looked easy, it lacked the major advantages of the piggy-back procedure, i.e., preservation of the caval flow and avoidance of venovenous bypass. However, caval thrombosis is a complication of the early post-transplant period that has previously been described, possibly associated with other vascular complications [11], but at times without explanation. In this patient, thrombosis occurred in the native cava, while the donor cava and the cava-cava anastomosis remained patent. We were unable to find an explanation for this occurrence, although the difficult dissection of the hypertrophic caudate lobe during the hepatectomy and the do-

nor/recipient graft size discrepancy might have been contributing factors.

Infrahepatic caval occlusion can be asymptomatic when the venous stasis passes down through retroperitoneal and azygos collaterals, but in cases of sudden occlusion, renal failure with limb edema and hemodynamic instability can develop. Of the 21 cases of infrahepatic vena cava thrombosis reported in the literature over the last 10 years, mortality was about 50 % despite antithrombotic therapy or various attempts at surgical revision [1, 3–8, 11, 12, 15]. In the case described herein, the thrombosed tract of the cava was initially tied off in order to avoid pulmonary embolism, but due to recurrent thrombosis, a left renal-to-splenic vein shunt had to be performed for the successful reversal of both renal failure and severe edema in the lower half of the body. The decision to go ahead with the shunt was preceded by an intraoperative confirmation of a 10-cm H_2O pressure gradient measured between the caval and portal circulations. To the best of our knowledge, this is the first description of such a procedure being performed after liver transplantation for acute, life-threatening lower cava hypertension.

A similar successful renal-splenic shunt has previously been described for a left renal vein and caval blockade in a child with a large Wilms tumor originating in the right kidney [7]. In that case, the left renal vein was connected end-to-side with the splenic vein, since the infrarenal cava had been irreparably removed; in our case, the shunt was made through a prosthesis, draining both renal and lower cava flows into the portal vein (Fig. 1). Interestingly, the grafted liver did not seem to suffer any overflow damage, confirming that hepatic parenchyma easily metabolizes venous blood from the kidneys [7].

Splenorenal shunting is a well-known procedure for the treatment of portal hypertension [14] and can be performed for similar indications even after liver replacement [10, 12]. In our case, the procedure was applied to a condition of venous hypertension unlike the one observed in cirrhosis, which ultimately turned a splenorenal shunt into a renal-splenic one. If a similar pressure gradient from caval to portal districts is observed, as in other conditions of acute infrahepatic vena cava occlusion (i.e., caval resection following trauma or tumor ablation), the proposed alternative to a renal-splenic shunt should be considered, especially when the operative approach to the cava and hepatic hilum is difficult.

Acknowledgements This study was supported by the Italian Association for Cancer Research (AIRC). We thank B. Damascelli, A. Marchianò, and C. Spreafico for their work in the Radiological Department and Ms. S. Benjamin for editing the manuscript.

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