LETTER TO THE EDITOR

The significance of complete interruption of large spontaneous portosystemic collaterals in adult living donor liver transplantation as a graft salvage procedure

doi:10.1111/j.1432-2277.2008.00639.x

In adult living-donor liver transplantation (LDLT), maintenance of adequate portal inflow is essential for the regeneration of partial liver graft. If portal flow diverted from partial liver graft through persistent collaterals even after LDLT, post-transplant liver with impaired graft regeneration and eventually graft failure would occur [1–3]. We aims to discuss on the significance of complete interruption of large spontaneous portosystemic collaterals to prevent portal flow steal (PFS) by presentation of following two patients.

Case 1

A 37-year-old female was transplanted for hepatitis B-related acute-on-chronic liver failure by using living donor left lobe graft [440 g, graft-to-recipient-weight ratio (GRWR) 0.98] on 8 May, 2002. Her model of end-stage liver disease (MELD) score was 34. Preoperative computed tomography (CT) scan had revealed large portosystemic collateral vein but we did not recognize its significance. On postoperative day 8, aspartate aminotransferase (AST)/alanine aminotransferase (ALT) abruptly began to rise from 71/110 to 1249/1074 IU/l, but we could not find any abnormal findings. Anti-rejection therapy was attempted with steroid pulse without pathologic confirmation. However, AST/ALT was rapidly elevated to 11253/8139 IU/l at day 10. CT scan revealed severe hypoattenuation of the liver graft, indicating poor parenchymal perfusion of the liver. On Doppler ultrasonogram, portal venous flow was not detectable but hepatic arterial flow was augmented. Indirect portogram revealed near total diversion of portal flow through persistent portosystemic shunt into inferior mesenteric vein, but we missed the finding (Fig. 1). Re-LDLT was performed at day 12 because of graft failure provoked by the PFS.

Case 2

A 40-year-old-male was transplanted for hepatitis B related acute-on-chronic liver failure by using living donor right lobe graft (650 g, GRWR 1.0%) on 30 November, 2003. Preoperative total bilirubin level was 66.1 mg/dl and MELD score was 40 points. The patient had mural thrombus and large coronary vein and splenorenal shunt (SRS) on preoperative CT scan. Eversion thrombectomy was performed and gastric lesser curvature including coronary vein was only ligated, because portal flow gushing from the end of main portal vein looked sufficient without ligation of left renal vein to interrupt SRS [4]. At day 5, portal flow on Doppler ultrasonography was decreased 45–20 cm/s, which was much weaker than usual flow. The recipient's total bilirubin level rose to 34 mg/dl at day 10. Under the suspicion of PFS through the patent SRS, related with persistently increased intrahepatic resistance after occlusion of interposition graft of reconstructed middle hepatic vein branches, we ligated left renal vein after laparotomy to enhance portal inflow to the impaired graft. Regardless of interruption of large SRS, liver function worsened much

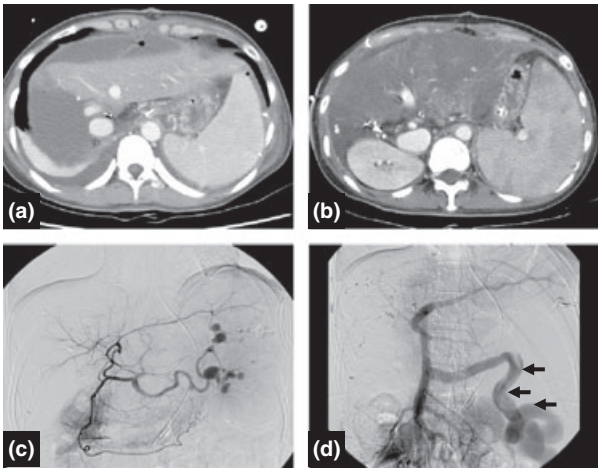

Figure 1 On postoperative day 1, computed tomography (CT) scan showed no specific finding except perihepatic fluid collection (a). On postoperative day 10, CT scan showed total infarction of the graft (b). On the same day, hepatic angiography revealed no thrombosis (c), but large splenomesenteric shunt (black arrows) was noted on indirect portogram (d).

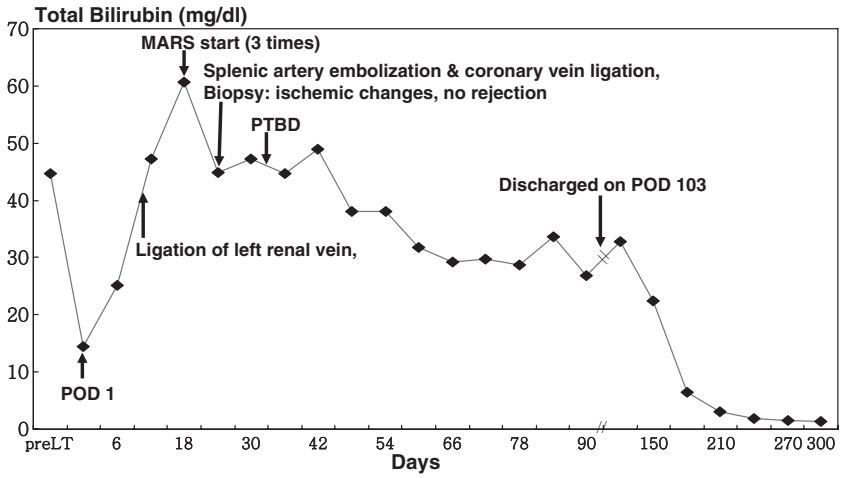

Figure 2 The changes of total bilirubin level before and after living donor liver transplantation in Case 2. POD, postoperative day.

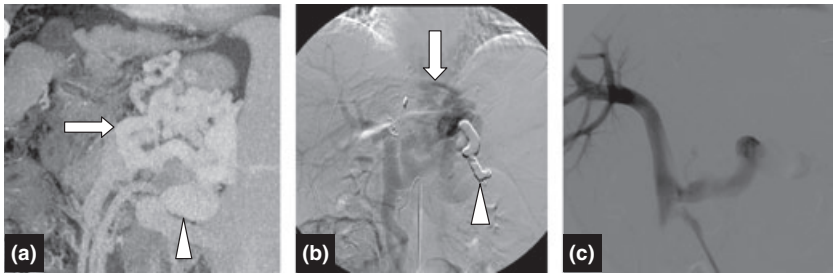

Figure 3 Preoperative multi-detector computed tomography (CT) scan showed enlarged coronary vein (white arrow) and splenorenal shunt (arrow head) (a). Indirect portogram during splenic artery embolization(arrowhead) on postoperative day 22 revealed persistent portosystemic shunt through coronary veins (white arrow) (b). During exploration on the postoperative day 24, the shunt was completely interrupted on the intraoperative cine-portography (c).

more (Fig. 2). At day 22, splenic artery embolization was performed to avoid possibility of portal hyperperfusion after interruption of shunt. We found large portosystemic collaterals through the coronary vein on indirect portogram, which is not included previous ligation. The large collaterals were interrupted at suprapancreatic area under the guidance of intraoperative cine-portogram at day 24 (Fig. 3) [5]. Wedge liver biopsy revealed ischaemic damage. Thereafter, the patient's liver function recovered and discharged at day 103. He is doing well to date with normal liver function.

After deceased donor whole-liver transplantation, interruption of portosystemic collaterals or not is usually not a critical problem [6]. In adult LDLT, however, PFS is not a rare problem, because the partial liver grafts have much smaller volume to accommodate hyperdynamic portal blood flow than in deceased donor whole-liver transplantation, and rapid regeneration of the liver graft to meet the metabolic demands of recipient transiently increase intrahepatic resistance to the portal inflow [7]. Our two cases also suffered from PFS, occurring after adult LDLT. Therefore, it should be kept in mind about the possibility of PFS during postoperative management of adult LDLT patients.

The experience of Case 1 taught us how important the interruption of large portosystemic shunt is in adult LDLT**.** There are a few similar reports from Japan [7–9]. Since the experience of Case 1, we made it a rule to ligate large postosystemic collaterals of it caliber 10 mm and above, and also ligate <10 mm calibered collaterals associated with portal vein stenosis on the preoperative CT scan. Coronary collaterals are interrupted by ligation of coronary vein only, or whole contents of lesser curvature side of stomach. SRS are interrupted directly or by ligation of left renal vein [4].

However, if the large portosystemic shunts showed multiple, labyrinthine-like and deeply located retroperitoneal courses, the intra-operatively complete interruption of collaterals by using our conventional ligation methods were often very difficult and inaccurate. Case 2 indicate the importance of complete interruption of large portosystemic shunt, even though portal flow was good enough

on direct visual examination and also intraoperative Doppler ultrasonography. After this life-threatening event, we additionally perform intraoperative cine-portography to monitor and confirm whether the interruption is performed completely when recipient has complicated multiple large portosystemic collaterals [5].

In conclusion, complete interruption of large collateral vessels might be needed as a part of adult LDLT procedure to avoid devastating PFS, even though GRWR reaches near to 1.0%.

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