Transplant International

LETTER TO THE EDITOR

Percutaneous transfemoral embolization of a spontaneous splenorenal shunt presenting with ischemic graft dysfunction 18 months post-transplant

doi:10.1111/j.1432-2277.2008.00691.x

Sadamori *et al.* [1] reported on outcome of living donor liver transplantation (LT) with prior spontaneous large portasystemic shunts and concluded that despite the complex surgery, the outcome was satisfactory and no intervention pre-LT or during the procedure was required. In contrast, Lee *et al.* [2] preemptively ligated the left renal vein in LT recipients with large splenorenal shunts. This seems to be a rather extensive procedure, which may have several short- and long-term disadvantages. Large splenorenal shunts may cause steal phenomenon and by this significantly may reduce initial perfusion of graft [1–3]. However, in small-for-size grafts such a shunt may prevent hyperperfusion syndrome [4–6]. The hemodynamic changes after LT may cause spontaneous closure of such shunts, however, if they remain patent they can cause ischemic graft dysfunction [7,8]. Development of splenorenal shunt after LT is frequently the result of portal vein thrombosis or venous outflow obstruction and therefore, graft dysfunction is common. Splenorenal shunts can be surgically ligated but endovascular embolization using a transhepatic or transfemoral route is the preferred treatment today [2,9–12].

A 56-year-old African-American female with sclerosing cholangitis (PSC) underwent piggyback LT with Roux-en-Y biliary reconstruction in January 2001 receiving a pediatric graft of 667 grams. A porto-caval shunt was created using the inverted confluence of the iliac veins with one limb anastomosed to the graft portal vein, one to the graft inferior vena cava (IVC) and the IVC segment of the vein graft to the recipient portal vein. The postoperative course was complicated by hepatic encephalopathy, hyperglycemia and renal impairment, however, these conditions resolved within 2 weeks. Initial immunosuppression included tacrolimus (TAC), mycophenolate-mofetil and a steroid taper. The 1-year protocol liver biopsy was unremarkable and the patient remained asymptomatic with liver enzymes only mildly elevated until July 2002 (18 months after LT), when she had an asymptomatic increase in her aminotransferases (Table 1). On Doppler ultrasound normal vascular flow and no biliary dilatation was found. A liver biopsy showed pericentral dropout consistent with ischemia and no signs of rejection or recurrent PSC. Three months later, she developed intermittent itching and progressive fatigue though her liver enzymes showed some improvement (Table 1). A repeat liver biopsy in November 2002 showed worsening pericentral necrosis and inflammation (Fig. 1a). A hepatic venography demonstrated spontaneous closure of the surgical porto-caval shunt. Computed tomographic (CT) scan and magnetic resonance (MR) imaging confirmed venography findings but in addition demonstrated development of a large splenorenal shunt. The graft had decreased in size and the spleen was enlarged measuring $12 \times 5 \times 11$ cm (Fig. 2). Ischemic graft dysfunction secondary to steal effect of the splenorenal shunt was suspected and embolization of the splenorenal shunt was performed in December 2002 (23 months post-LT). A right transfemoral approach was utilized as CT and MR imaging revealed a large communication with the left renal vein. A 5-French Cobra catheter was utilized to select the splenorenal shunt arising from the mid portion of the left renal vein (Fig. 3). A 5-French glide catheter and glidewire was advanced several cm into the tortuous splenorenal shunt. After placing a stiff wire, a 7-French sheath was advanced into the shunt; through a 7-French catheter two 20-mm 0.052 coils were placed within the shunt. Four additional 15-mm 0.052 coils were placed. Following this anchor of coils, multiple tornado and Nester coils were placed to completely occlude the shunt. A total of 14 coils were

Table 1. Laboratory parameters during follow up.

Date	APh	ALT	GGT	AST	Albumen	TB	Cr	INR	Plt
3/5/2001	250	23	55	27	3.2	1.1	1	1.2	171
4/3/2002	155	39	15	44	4.4	1.1	0.8	1.2	122
7/1/2002	183	384	25	239	3.9	0.5	0.9	1.2	114
12/5/2002	147	170		132	4.2	0.8	0.8	1.0	120
12/16/2002	182	87	23	75	4.6	0.9	0.7	1.0	163
7/21/2003	84	107	18	79	4.3	0.6	0.8	1.1	155
12/5/2003	85	38	13	41	4.4	0.6	0.9	1.0	144
1/22/2007	64	26		27	4.5	0.3	1.0	1.1	156

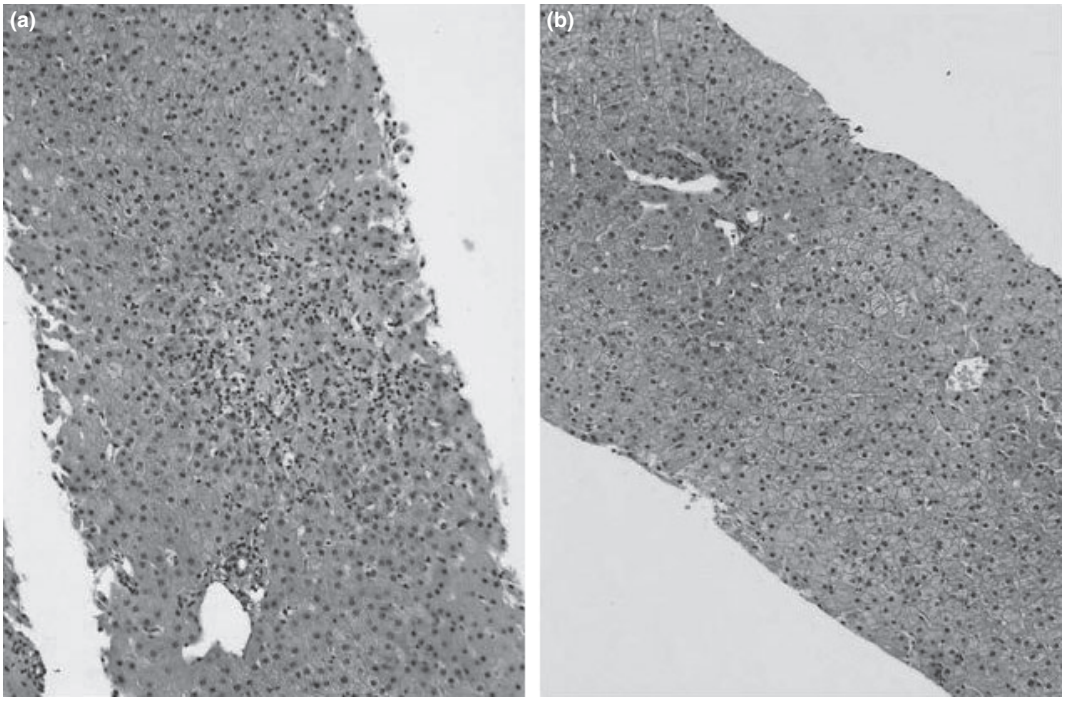

Figure 1 (a) Biopsy from 2002 showing pericentral necrosis consistent with ischemia (H&E stain, magnification ×200). (b) Biopsy from 2007 showing absence of necrosis (H&E stain, magnification ×200).

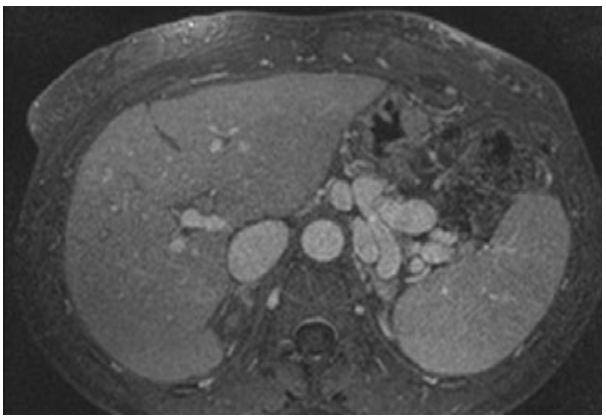

Figure 2 Magnetic resonance imaging. Spenomegaly and formation of large splenorenal shunt.

utilized (Fig. 3a–d). As a result, liver enzymes rapidly improved and normalized by the end of 2003 (Table 1). The patients remained well; follow-up liver biopsies (last in January 2007) revealed minimal focal portal inflammation with no other pathological findings (Fig. 1b) and liver enzymes are within normal range. The graft has significantly increased in size as determined by ultrasound and CT-scan.

It seems unusual that the initial surgically created porto-caval shunt had closed and a spontaneous splenorenal shunt developed 18 months after LT in the absence of pre- or posthepatic portal hypertension; additionally, liver biopsy showed no evidence of rejection, recurrent PSC or portal fibrosis. Spontaneous portasystemic shunts result from portal hypertension and are associated with hepatic encephalopathy and deterioration of hepatic function. Treatment of portal hypertension with surgically created shunts has been largely replaced by endovascular techniques (TIPS) and endoscopic therapy (esophageal varices). LT represents the best treatment for cirrhotic patients with splenorenal shunts, however, high shunt volumes may cause poor initial graft perfusion potentially leading to graft dysfunction/failure [1,2,13–15]. Sadamori emphasized that outcome of LT is good and no intervention seems necessary, which is in contrast to the data of Lee *et al.* [1,2]. LT with renoportal anastomosis after distal splenorenal shunt was performed with good results; also intraoperative ultrasound has been utilized [16–19]. Ligation of the renal vein during LT is a radical and permanent approach and the questions remains, in how many patients these shunts would have spontaneously closed. As renal failure represents one of the most common long-term complications of LT, any additional injury to the kidneys should be avoided. Ligation of the shunt would be an alternative option during LT, which could be tailored to the shunt volume. In patients who receive very small grafts it would be even a protective

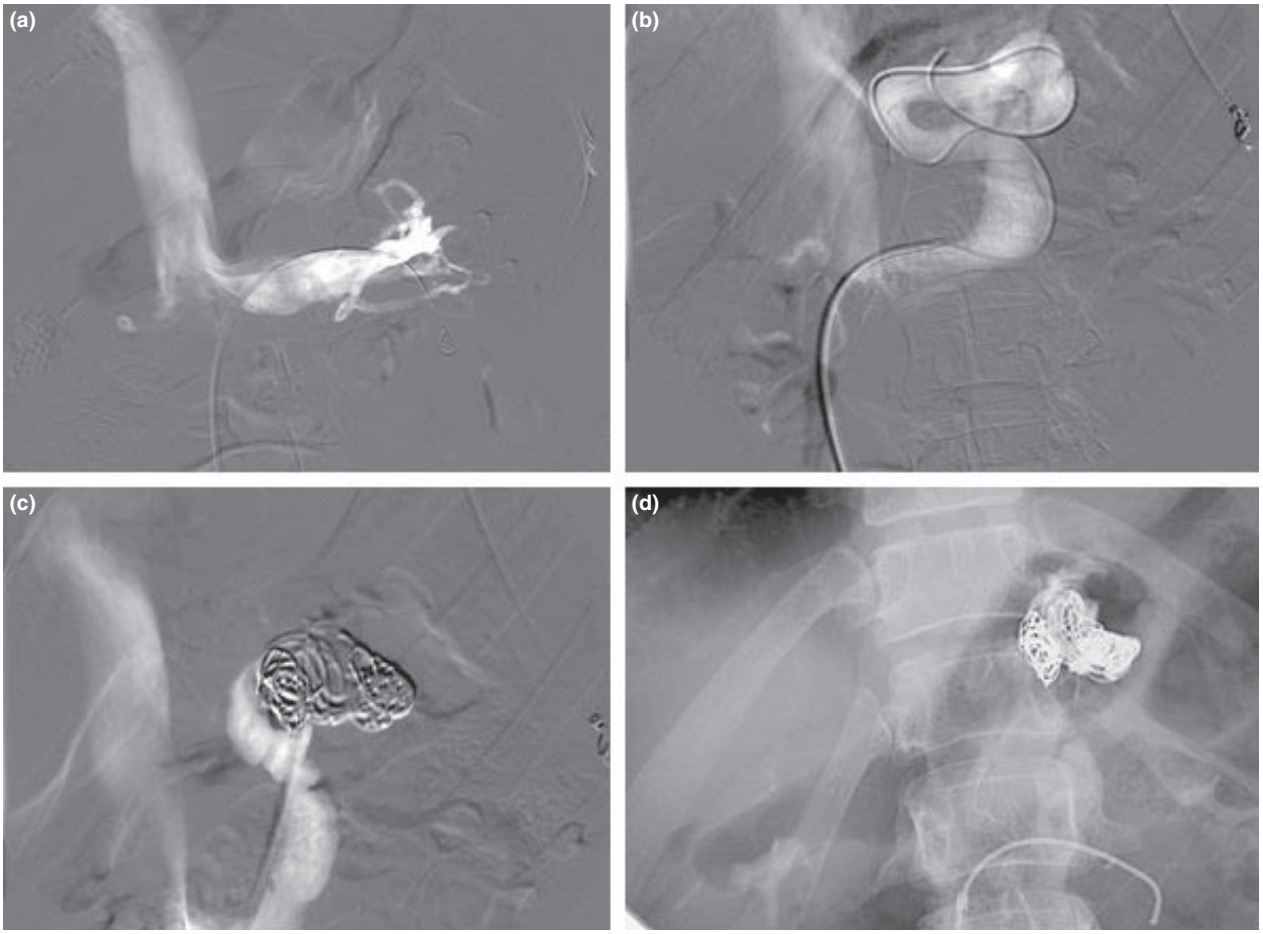

Figure 3 Angiography. (a) Transfemoral access, imaging of renal vein; (b) imaging of splenorenal shunt; (c) placement of coils into the shunt; (d) occlusion of the shunt.

diversion of portal blood preventing graft hyperperfusion. Very large shunts could be embolized prior to LT or early post-LT, if necessary. In many cases, these shunts are combined with splenomegaly and ligation of the splenic artery may improve arterial blood flow to the graft and correct thrombocytopenia. Portosystemic shunts may develop in the case of increased intrahepatic vascular resistance from ischemic injury, acute rejection, fibrosis, volume overload, recurrent cirrhosis and commonly in the presence of small-for-size grafts. Ligation of the shunt has been shown to restore normal flow. In our patient, a pediatric graft was utilized and in order to prevent hyperperfusion syndrome, we created a temporary portosystemic shunt, which obliterated by time. The resolution of the biochemical and histological abnormalities after embolization of the spontaneous splenorenal shunt supports diagnosis of graft dysfunction secondary to the steal effect. Vascular interventional radiology plays an essential role and has high success rate [20]. This is the first reported LT recipient in whom a splenorenal shunt was embolized via a transfemoral access, which is less frequently used than the transhepatic approach [21]. This approach should be considered if direct access via the left renal vein can be demonstrated on preinterventional imaging.

Ali Al hajjaj,[1] Hugo Bonatti,[2] Murli Krishna,[3]
Rolland Dickson,[1] McKinney J. Mark,[4]
Justin Nguyen,[5] Jeffery Steers[6] and
Jaime Aranda-Michel[1]
1 Department of Hepatology and Transplantation,
Mayo Foundation Jacksonville,
St' Luke's Hospital,
Jacksonville, FL, USA
2 Department of Surgery,
University of Virginia Health Services,
Charlottesville, VA, USA
3 Department of Pathology,
Mayo Foundation Jacksonville,
St' Luke's Hospital,
Jacksonville, FL, USA

4 Department of Radiology,
Mayo Foundation Jacksonville,
St' Luke's Hospital,
Jacksonville, FL, USA
5 Department of Surgery,
Mayo Foundation Jacksonville,
St' Luke's Hospital,
Jacksonville, FL, USA
6 Department of Surgery,
Aurora Health Care,
Milwaukee, WI, USA

References

1. Sadamori H, Yagi T, Matsukawa H, *et al.* The outcome of living donor liver transplantation with prior spontaneous large portasystemic shunts. *Transpl Int* 2008; **21**: 156.
2. Lee SG, Moon DB, Ahn CS. Ligation of left renal vein for large spontaneous splenorenal shunt to prevent portal flow steal in adult living donor liver transplantation. *Transpl Int* 2007; **20**: 45.
3. De Carlis L, Del Favero E, Rondinara G. The role of spontaneous portosystemic shunts in the course of orthotopic liver transplantation. *Transpl Int* 1992; **5**: 9.
4. Demetris AJ, Kelly DM, Eghtesad B, *et al.* Pathophysiologic observations and histopathologic recognition of the portal hyperperfusion or small-for-size syndrome. *Am J Surg Pathol* 2006; **30**: 986.
5. Troisi R, Ricciardi S, Smeets P, *et al.* Effects of hemi-portocaval shunts for inflow modulation on the outcome of small-for-size grafts in living donor liver transplantation. *Am J Transplant* 2005; **5**: 1397.
6. Cheng YF, Huang TL, Chen TY, *et al.* Liver graft-to-recipient spleen size ratio as a novel predictor of portal hyperperfusion syndrome in living donor liver transplantation. *Am J Transplant* 2006; **6**: 2994.
7. Tissières P, Pariente D, Chardot C. Postshunt encephalopathy in liver transplanted children with portal vein thrombosis. *Transplantation* 2000; **70**: 1536.
8. Cescon M, Sugawara Y, Kaneko J. Restoration of portal vein flow by splenorenal shunt ligation and splenectomy after living-related liver transplantation. *Hepatogastroenterology* 2001; **48**: 1453.
9. Semiz-Oysu A, Keussen I, Cwikiel W. Interventional radiological management of prehepatic obstruction the splanchnic venous system. *Cardiovasc Intervent Radiol* 2007; **30**: 688.
10. Nakai M, Sato M, Sahara S. Transhepatic catheter-directed thrombolysis for portal vein thrombosis after partial splenic embolization in combination with balloon-occluded retrograde transvenous obliteration of splenorenal shunt. *World J Gastroenterol* 2006; **12**: 5071.
11. Bilbao JI, Arias M, Herrero JI. Percutaneous transhepatic treatment of a posttransplant portal vein thrombosis and a preexisting spontaneous splenorenal shunt. *Cardiovasc Intervent Radiol* 1995; **18**: 323.
12. Durham JD, LaBerge JM, Altman S. Portal vein thrombolysis and closure of competitive shunts following liver transplantation. *J Vasc Interv Radiol* 1994; **5**: 611.
13. Shioyama Y, Matsueda K, Horihata K. Post-TIPS hepatic encephalopathy treated by occlusion balloon-assisted retrograde embolization of a coexisting spontaneous splenorenal shunt. *Cardiovasc Intervent Radiol* 1996; **19**: 53.
14. Boillot O, Houssin D, Santoni P. Liver transplantation in patients with a surgical portasystemic shunt. *Gastroenterol Clin Biol* 1991; **15**: 876.
15. Mazzaferro V, Todo S, Tzakis AG. Liver transplantation in patients with previous portasystemic shunt. *Am J Surg* 1990; **160**: 111.
16. Kato T, Levi DM, DeFaria W. Liver transplantation with renoportal anastomosis after distal splenorenal shunt. *Arch Surg* 2000; **135**: 1401.
17. Margarit C, Lázaro JL, Charco R. Liver transplantation in patients with splenorenal shunts: intraoperative flow measurements to indicate shunt occlusion. *Liver Transpl Surg* 1999; **5**: 35.
18. Cheng YF, Huang TL, Chen CL. Intraoperative Doppler ultrasound in liver transplantation. *Clin Transplant* 1998; **12**: 292.
19. Cheng YF, Chen YS, Huang TL. Interventional radiologic procedures in liver transplantation. *Transpl Int* 2001; **14**: 223.
20. Kessler J, Trerotola SO. Use of the Amplatzer Vascular Plug for embolization of a large retroperitoneal shunt during transjugular intrahepatic portosystemic shunt creation for gastric variceal bleeding. *J Vasc Interv Radiol* 2006; **17**: 135.
21. Matake K, Tajima T, Yoshimitsu K, *et al.* Hepatic encephalopathy from dual splenorenal shunts treated with balloon-occluded retrograde transvenous obliteration by using a double-balloon technique. *J Vasc Interv Radiol* 2007; **18**: 1436.

