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LETTER TO THE EDITOR

Portal vein stenosis following living donor liver transplant: manifestations and management

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Stenosis or occlusion of the portal vein (PV) is infrequent after living donor liver transplants (LDLT) with a reported incidence of 2–3% [1]. When it occurs, it manifests as one or more of the following: oesophageal varices, ascites, splenomegaly, thrombocytopenia or even fulminant allograft failure.

As repeat surgery in this situation may be technically difficult, nonsurgical methods have been used to restore PV patency. These include infusion of thrombolytic agents (e.g. recombinant tissue-type thromboplastin activator or rTPA) [2], angioplasty of the associated PV stenosis (PVS) [3], portocaval shunts [4], mechanical thrombectomy and catheter embolization of competing collaterals. Stenting of delayed PVS has been described after orthotopic liver transplantation [5]. We present our experience of three patients.

From January 2002 to May 2008, 240 LDLTs were performed at our centre. Of these, two patients presented with graft dysfunction, refractory ascites and thrombocytopenia at 3 and 22 months and one with lower gastrointestinal bleed 17 months after LDLT. All three patients received right lobe grafts with inclusion of the middle hepatic vein. Two of them had standard recipient main PV to donor PV anastomosis, whereas one patient's graft had separate anterior and posterior PVs, which were extended on the bench with a Y-shaped cryopreserved vein graft, which was anastomosed to the recipient PV. Anastomoses were performed using 5-0 prolene continuous suture and growth factor was left in all cases. In our entire series, only four recipients in the non-PVS group had ligation of major collaterals.

The PVS was first detected by ultrasound Doppler and confirmed by computerized tomography (CT) portal venography and magnetic resonance venography (Figs 1 and 2). The criteria for the identification of PVS on USG Doppler were 50% or less diameter of the stenosed portion compared with the main PV, an acceleration of flow at the anastomosis or a postanastomotic jet [6].

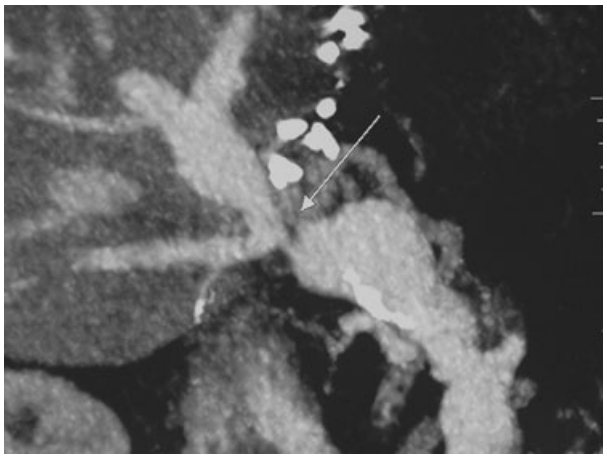

Figure 1 A 56-year-old male developed PV stenosis 22 months after living related liver transplant for cryptogenic chronic liver disease. CT scan shows PV with severe tight stenosis (arrow) with proximal dilatation. Note the venous backflow.

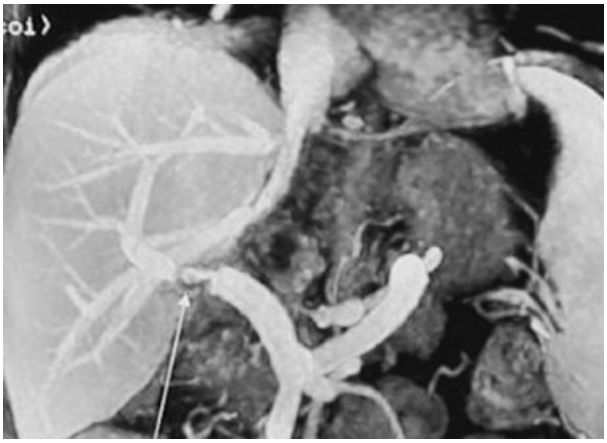

Figure 2 Magnetic resonance venography showing thrombosis and narrowing of venous conduit.

In the third patient who presented with melaena 17 months after transplantation, upper gastrointestinal endoscopy showed grade 1 and 2 oesophageal varices with a gastric varix and large tortuous collaterals. USG

[Correction added after online publication on 9th December 2008: In the author affiliations, the following error was published on page 3: **Arvinder Singh Soin[2]**. The text was incorrect and should have read **Arvinder Singh Soin[1]**]

Doppler showed narrowing of the PV with pre- and post-stenotic velocities of 28.4 and 56.2 cm/s respectively.

Percutaneous transhepatic angioplasty was attempted in all these patients. After obtaining informed consent, the procedure was performed under local anaesthesia. Under ultrasound guidance, a lateral percutaneous transhepatic puncture of a branch of the PV was carried out using a 22 G Chiba needle and a direct portovenogram performed with pressure measurements across the stenosed segment to confirm functionally relevant stenosis. A nitinol guidewire (0.37 mm, 180 cm length) was passed across the stenosis with an 8 Fr introducer with a balloon 10% larger in diameter (Fig. 3a) than the nonstenotic PV in sheath. The pre- and post-angioplasty portal pressure gradients were recorded. Angioplasty was performed in two cases and angioplasty and a Palmaz stent of 10–12 mm diameter was deployed in one patient (Fig. 3b). The stent size was chosen on the basis of the measured diameter of the proximal PV.

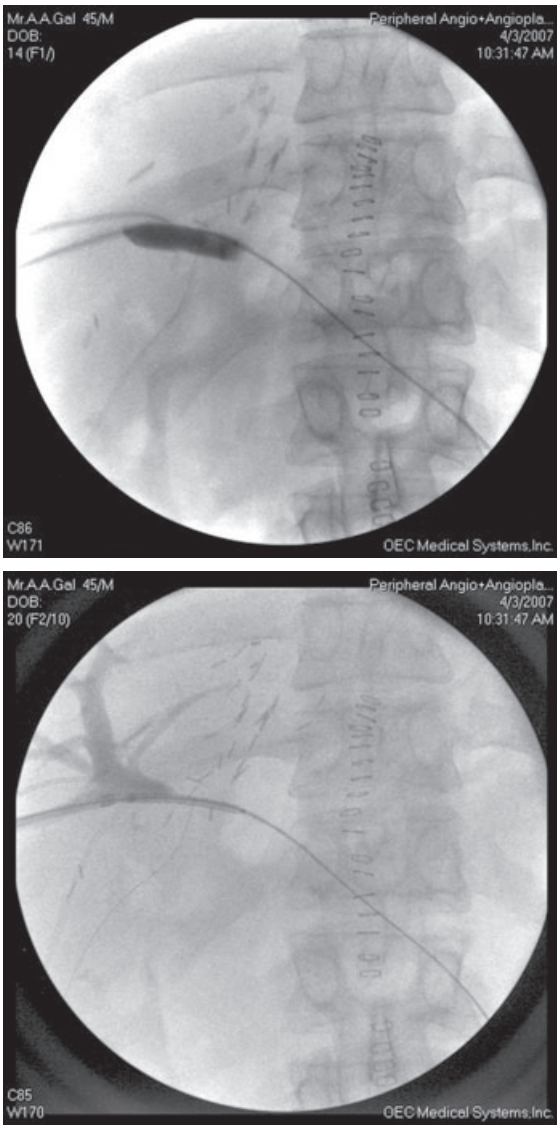

Figure 3 A 45-year-old male developed PV stenosis 3 months after living related liver transplant for HBV with HDV related chronic liver disease and underwent portovenography. (a) Fourteen millimetre balloon angioplasty was performed. (b) Palmaz stent deployed across the stenosis.

Antiplatelet agents were given following the procedure and the results were monitored by follow-up of clinical symptoms, liver function tests, USG Doppler and a check CT venogram at 3 months.

Our technical success rate was 100%. The pre- and post-procedure trans-anastomotic mean pressure gradients in the three patients were 10 and 1 mmHg, 18 and 2 mmHg and 11 and 2 mmHg respectively. The ascites and thrombocytopenia showed a dramatic improvement over 1–8 weeks with a patent stent and repeat endoscopy in the third patient showed regression of the varices. There were no complications after the procedures. All three patients continue to be well with no recurrence of PVS on serial imaging at 16, 13 and 11 months respectively.

Our experience, albeit small, has shown that symptomatic PV anastomotic stenosis, is an uncommon complication following LDLT. The incidence in whole liver transplants has been reported to be 2.7% [7]. PVS has been ascribed to technical difficulties in PV reconstruction preoperative PV thrombosis, thrombus formation from the portal venous bypass cannula, excessive vessel redundancy/size discrepancy, allograft oedema or hypercoagulability [8]. It may also be because of portal flow steal by previously existing portal hypertensive collaterals. In two of our three cases with normal graft PV anatomy, PVS may have been caused by the persistence of collaterals and low flow as it was easily reversed by simple dilatation, which made the hepatopetal route the path of least resistance for the portal flow. In the third patient, it was the cryopreserved venous conduit that stenosed. This has also been previously observed by others [8]. The latter patient required a Palmaz endoluminal prosthesis, which is usually inserted when angioplasty is not successful either by anatomic or haemodynamic considerations (when pressure gradient continues to be more than 5 mmHg) or when there is immediate recoiling of the stenotic segment after dilatation [5].

The traditional management of PVS has included resection and reconstruction of the anastomosis, thrombectomy, portocaval shunting or retransplantation [9]. Angioplasty and stenting performed either by a minilaparotomy or using a percutaneous transhepatic approach, have an excellent outcome with significantly less morbidity compared with surgical intervention approach the percutaneous transhepatic approach which is associated with

Table 1. Results of postliver transplant portal vein stenosis and management techniques.

Case series	*N*	Technique	Technical success (%)	Complications	Follow up (months)
Raby *et al.* [12]	3	PTA	2 (66.7)	–	12
Olliff *et al.* [3]	2	PTA	2 (100)	–	18;16
Godoy *et al.* [5]	2	PTA and/or stent	2 (100)	–	2–20
Funaki *et al.* [8]	25	PTA and/or stent	19 (76)	–	5–61
Glanemann *et al.* [10]	1	PTA	1 (100)	–	6
Kwang Bo Park [15]	6	PTA	1 (100)	1 – Puncture tract bleed	3–64
Ko *et al.* [11]	9	Stent	7 (77.8)	1 – Pseudoaneurysm, 2 – haemoperitoneum	66.6 ± 16.1
Present series	3	PTA and/or stent	3 (100)	–	11–16

PTA, percutaneous balloon angioplasty.

a risk of intra abdominal bleeding in 2.5% of cases [10]. The major complications that have been described with the transhepatic route include haemoperitoneum and intrahepatic pseudoaneurysm [11]. We used the percutaneous transhepatic approach in all our patients without any complication.

Previous studies have shown that a pressure gradient of more than 5 mmHg across the PV anastomosis is significant [12,13]. Portal angioplasty is successful in cases with symptoms primarily related to stenosis and not graft dysfunction. In our study, the pre- and postprocedure trans-anastomotic mean pressure gradients decreased (from a mean of 13–1.7 mmHg) with subsidence of symptoms and abnormal blood cell counts in all patients.

Encouraging results have been reported earlier using transhepatic portal venoplasty for PVS especially in children who have reduced size liver transplant and develop PVS [14]. The reported recurrence rate following angioplasty is about 28.6–36.8% [6]. The results of PV recanalization techniques for PVS following liver transplant have been shown in Table 1. Clinical or radiological recurrence has not occurred in any of our patients so far. Portal vein stenosis is a rare complication of LDLT, which can be safely and effectively managed by percutaneous angioplasty with occasional need for endoluminal stenting.

Disha Sood,[1] Vinay Kumaran,[1] Rahul Kakodkar,[1] Sundeep J. Punamia,[2] Sanjiv Saigal,[3] Samiran Nundy[1] and Arvinder Singh Soin[1]

1 Department of Surgical Gastroenterology and Liver Transplantation, Sir Ganga Ram Hospital, New Delhi, India

2 Department of Vascular and Interventional Radiology, Bombay Hospital, Bombay, India

3 Department of Medical Gastroenterology and Liver Transplantation, Sir Ganga Ram Hospital, New Delhi, India

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