Transpl Int (1995) 8: 330–332

LETTER TO THE EDITOR

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Detection of suprahepatic caval stenosis following liver transplantation and treatment via balloon-expandable intravascular stent

Received: 8 December 1993
Received after revision: 27 January 1995
Accepted: 15 February 1995

Sir: In the following report, we would like to comment upon the detection and nonsurgical treatment of a severe stenosis to the suprahepatic caval anastomosis (SHCA) following orthotopic liver transplantation (OLT) for hepatitis B-induced cirrhosis with newly detected hepatocellular carcinoma of the right lobe. Emphasis will be placed on the central role played by color-flow Doppler imaging (CFDI), routinely performed by a member of the Department of Surgery, in both the detection and treatment of the high-grade stenosis.

Following technically difficult OLT (operating time 9 h, blood loss 32 units RBC and 60 FFP, both significantly higher than average), CFDI (Vingmed CH 700, Diasonics) was first performed 10 h postoperatively. Organ perfusion was unusual for the early post-transplant phase, revealing an average flow velocity of less than 10 cm/s in the portal vein, as opposed to normal post-OLT values of up to 30 cm/s. Concomitantly, arterial blood flow was elevated to an average velocity of 24 cm/s and there was a noticeably altered flow curve, representing decreased resistance in the arterial blood-stream (Table 1). Hepatic vein flow was barely detectable, revealing a discontinuous flow signal in all veins.

Due to delayed organ recovery associated with multiorgan failure (MOF), CFDI was routinely performed on the following days, with specific regard to the assumed liver outflow obstruction. The latter was confirmed by a repeat liver biopsy on the 8th post-OLT day that revealed increasing centrilobular necrosis in association with distinct vascular congestion.

Since a gradual decrease in transient stenosis due to reactive edema at the site of vascular anastomosis has been described, as well as the development of sufficient collateral blood flow following stenosis of the SHCA, a reserved attitude towards reintervention had been assumed up until this time. However, renewed relapse of organ function (Table 2) and a CFDI-confirmed decrease in blood flow led to the decision to perform transfemoral cavography in combination with eventual angioplasty with surgical stand-by on the 11th post-OLT day. (Primary surgical intervention was regarded as extremely hazardous at this time.)

Radiography confirmed the CFDI diagnosis of a high-grade stenosis of the SHCA with a strong collateral flow via the azygos vein system. The stenosis was first treated by balloon dilatation, but the result was not sufficient, due to elastic recoil of the stenosis. Given the patient's condition, the decision was then made to attempt definitive correction by positioning a balloon-expandable stent across the stenosis. The stent could, in fact, be easily expanded using less than one atmosphere of pressure, indicating that a tight suture could not have been the cause of the stenosis.

Following placement of the stent (Palmaz-Stent, Johnson and Johnson Interventional Systems, Warren, N. Y., U. S. A.), organ perfusion was again checked via CFDI. Angle-corrected velocity measurements revealed a dramatic increase in portal perfusion, coinciding with both a net reduction in arterial blood flow and normalization of the earlier "low-impedance type" flow in the arterial system. This had been interpreted as an expression of intrahepatic arterioportal shunting due to the increased resistance that mainly harmed the portal system, as demonstrated by the velocity measurements prior to successful stenting of the stenosis. Venous blood flow in the liver was now detectable as a continuous centripetal signal for the first time since OLT, as was renal

Table 1 Vessel diameter and flow velocity values

a Vessel diameter[a] in millimeters

	Stent:							
Date	Day 1 18.10.	Day 4 21.10.	Day 8 25.10.	Day 11 28.10.	Day 12 29.10.	Day 14 31.10.	Day 17 3.11.	Day 34 20.11.
IVC	31	37	37	38	26	26	28	24
Portal vein	12	14	15	14	11	11	11	11
Hepatic vein	10	13	13	13	10	10	10	10

[a] Average of end-expiratory values

b Average flow velocity values (cm/s)

	Stent:							
Date	Day 1 18.10.	Day 4 21.10.	Day 8 25.10.	Day 11 28.10.	Day 12 29.10.	Day 14 31.10.	Day 17 3.11.	Day 34 20.11.
Portal vein	9	12	10	11	21	21	23	21
Hepatic artery	24	28	22	25	12.5	25	26	19

Table 2 Laboratory values

	Stent:						
Date	Day 1 18.10.	Day 4 21.10.	Day 8 25.10.	Day 11 28.10.	Day 12 29.10.	Day 14 31.10.	Day 17 3.11.
Bilirubin (mg/dl)	3.7	3.4	4.1	3.4	2.7	2.5	3.1
ALT (U/l)	565	410	106	192	95	97	18
Faktor V. (%)	50	82	–	46	47	79	80
Glutamate dehydrogenase (U/l)	52	441	46	45	14	13	3.3
Lactate (mg/dl)	4.9	3.03	3.4	–	2.98	1.57	0.96

vein flow, coinciding with the beginning of diuresis.

Normalization of the vascular diameter and, more important, of flow values (Table 1) in the following days was accompanied by a gradual improvement in both organ function (Table 2) and the patient's general condition. Routine CFDI could detect no signs of partial thrombosis within the stent, whose position remained absolutely stationary despite its precarious subdiaphragmal position.

Unfortunately, recurrent infectious complications associated with possible hepatitis reinfection led to rapid renewed deterioration of graft function 3 weeks after angioplasty, and the decision was made to perform retransplantation (re-OLT) in this special case. The patient succumbed to MOF 3 days after re-OLT, which was performed 26 days after angioplasty.

During retransplantation, the stent had been removed without difficulty, revealing no macroscopic signs of thrombosis. Histological examination revealed partial coverage with immature endothelial cells and fibroblast proliferation along the struts of the stent. The explanted graft revealed tissue necrosis with anticipated fibrosclerotic thickening of the intima of the hepatic vein system, indicating prolonged venous congestion of the graft.

Although predominantly arterial complications are diagnosed in the early post-OLT phase, angiography and duplex sonography have also detected a significant number of portal and venous obstructions to graft flow [2, 5]. CFDI has mainly been used to check morphologic disturbances, such as high-grade stenoses or thromboses of major vessels [8]. The importance of altered intrahepatic blood flow, despite inconspicuous anastomosis and/or course of the hepatic artery, especially in the case of primary nonfunction, has recently been described [10].

Whereas immediate correction of pathologic, angiographic, or duplex sonographic findings in cases of arterial or portal impairment has always been the rule in transplantation surgery, a more conservative attitude has often been described with regard to disturbances of the caval anastomosis or to venous drainage from the graft, despite the fact that uncorrected, continuous obstruction will ultimately lead to graft loss [1, 4]. One reason for this discrepancy may be that a significant number of suprahepatic caval stenoses result from extraordinarily difficult anatomic situations that demand special attention when creating the primary anastomosis and lead one to fear that corrective surgery may not bring about the required improvement in vascular patency.

Interventional radiology has made use of refined materials and techniques in the past decade, and less precarious methods are now available to correct natural or iatrogenic vascular stenoses [6]. As balloon dilatation alone has repeatedly led to restenosis in this region [3, 7, 9], especially in cases of elastic recoil, a novel technique was employed for the definite correction of the SHCA stenosis in this case, using a 30-mm long, 12-mm caliber stent. The final graft loss, even after successful angioplasty, 11 days after OLT must be seen in the context of the primary impairment of organ perfusion, despite CFDI findings of compensatory increased arterial flow in this vulnerable phase. Histologic examination of the explanted graft confirmed permanent damage, sustained by the 11-day phase of posthepatic hypertension due to stenosis to the SHCA.

CFDI appears to be the method of choice for morphologic and functional evaluation of graft perfusion under the condition of impaired outflow. Early, preferably radiologic, reintervention with surgical standby is, therefore, recommended in the case of high-grade stenosis to the SHCA if permanent damage and eventual loss of the graft are to be avoided. Lack of a spontaneous increase in perfusion within 2–3 days may be regarded as a safe margin for reintervention in such cases.

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