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BRIEF REPORT

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Arterialization of the portal vein in pediatric liver transplantation

A Report of two cases

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Abstract Portal vein arterialization (PVA) is an acquired concept in shunt surgery for portal hypertension. This technique, recently described as both a temporary and permanent procedure in adult liver transplantation, is reported by the authors in two cases of pediatric transplantation. The indication was low portal blood flow after reperfusion with poor graft function due to persistence of spontaneous retroperitoneal venous shunts. In both cases described, PVA allowed for satisfactory macroscopic liver reperfusion. The increase in portal blood flow from 150 to 500 ml/min in the second patient enabled the liver to be reperfused correctly and led to successful transplantation. The graft function in both cases improved in the 1st postoperative week, but thrombosis of the PVA occurred in the 1st patient 2 months after transplantation. Signs of hepatic hyperarterialization occurred in the second patient and this necessitated a dearterialization of the portal vein 2 weeks later. Although the benefit of this procedure appears to be beyond doubt in the immediate postoperative period, we have no data on long-term arterialization. We do think that PVA can be performed in pediatric liver transplantation, but it may need to be done only in special, individual situations when no valid alternative can be proposed, such as in the absence of a mesenteric vein and/or the presence of spontaneous retroperitoneal venous shunts.

Key words Liver transplantation, portal vein arterialization · Portal vein arterialization, liver transplantation

Introduction

Portal vein arterialization (PVA) is an acquired concept in shunt surgery for portal hypertension [1]. It may be considered an alternative procedure whose purpose is to preserve liver perfusion and, at the same time, to minimize the risk of hepatic failure and encephalopathy. Since 1952, when Hunt first reported its application in four patients [5], many reports have been published to investigate the long-term effects of PVA. However, results have not always been satisfactory, with primarily anastomotic thromboses and complications related to the excessive liver arterialization [7–9]. Up until now, there have been reports on both temporary and permanent portal arterialization in liver transplantation (LT). Sheil et al. [12] first reported on temporary PVA in adult LT. The procedure was intended to shorten the warm ischemic time, to provide stable reperfusion, and to facilitate safe completion of all anastomoses. Pichlmayr et al. [10] described a permanent arterialization to increase the portal perfusion in cases of low flow, and recently, Erhard et al. [4] reported long-term clinical results in three cases of successful, total, permanent PVA.

Indications for PVA in LT include (1) low splanchnic perfusion, (2) extended splanchnic vein thrombosis that does not allow for successful thrombectomy, and (3) anatomical variations (e.g., absence of the portal and mesenteric veins) [8, 10]. Up until now, there have been no

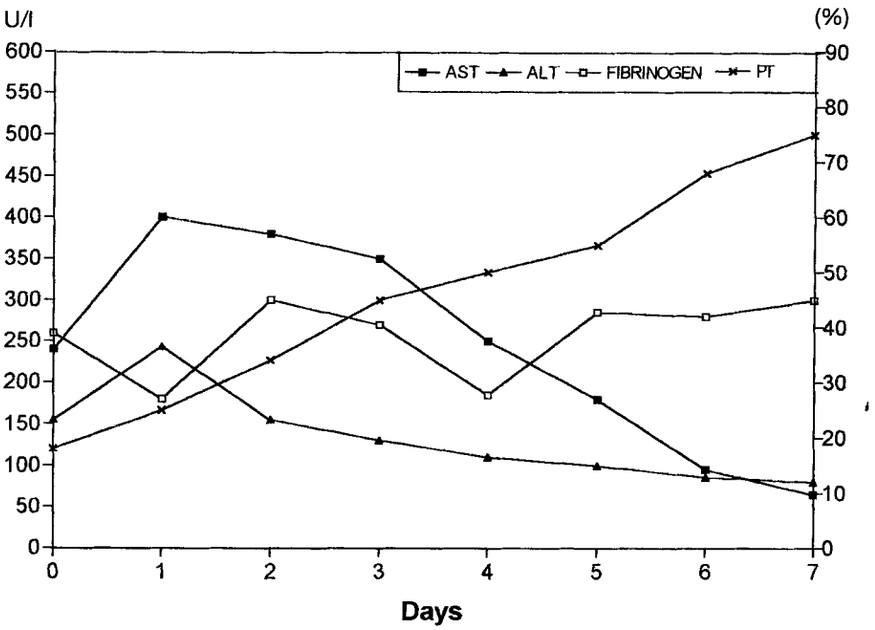


Fig. 1 Patient 1. Serum transaminase activity, fibrinogen level and prothrombin time of the patient after the PVA procedure

reports on PVA in pediatric LT, where the risk of portal vein thrombosis is higher. When it occurs early, the risk of graft failure increases [2]. In the following two cases, temporary PVA in pediatric orthotopic liver transplantation (OLT) will be described with regard to the indication and the technique.

Case reports

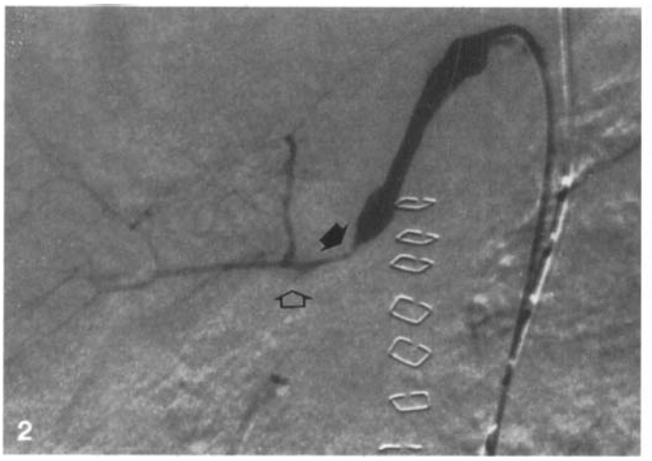
Patient 1

A 6-year-old boy underwent OLT because of terminal liver failure due to secondary biliary cirrhosis 4 years after resection of a neuroblastoma of the coeliac trunk with partial hepatectomy. Preoperative angiography showed a thrombosis of the hepatic artery with a complex arterial collateral circulation. During the venous phase, there was good visualization of the superior mesenteric vein (SMV) and less so of the portal vein (PV), with preferential drainage into the splenogastric and retroperitoneal collaterals. The transplantation technique consisted of a piggy-back implantation of a full-size pediatric liver. Arterial anastomotic reconstruction was done with an interposed iliac arterial graft between the recipient aorta and the common hepatic artery of the donor liver. Portal venous reperfusion was carried out via an anastomosis of the donor porta on the confluence of the vena lienalis and the SMV. Despite a wide patent anastomosis, liver reperfusion remained borderline, presumably due to the persistence of multiple retroperitoneal collaterals. Postoperatively, Doppler ultrasound (US) showed a portal blood flow velocity of 10 cm/s (V_{max}). Twenty hours later, the patient required reoperation because of delayed graft function. At re-exploration, portal blood flow remained low, despite negative Fogarty exploration of the donor porta and recipient SMV. Assuming that retroperitoneal shunts were the cause of the reduced flow, and considering the difficulty of precisely identifying and ligating them, preference was given to increasing the portal flow via an arteriovenous shunt. A PVA was created by laterally anastomosing the donor gastroduodenal stump on the vena porta. Liver perfusion and liver tests improved during the following days, as did the patient's general condition (Fig. 1). Postoperatively, Doppler US confirmed a portal flow velocity of 45 cm/s (V_{max}). Nevertheless, two months later the patient developed a clinical sepsis with progressive deterioration of liver function due to a liver abscess that was documented on a CT scan. Because portal and arterial thrombosis were suspected on US evaluation, angiography was performed. The latter indeed showed thrombosis of the portal vein and hepatic artery stenosis with no opacification of the arterial shunt (Fig. 2). The diagnosis of PVA thrombosis was confirmed at re-exploration. Thrombectomy of the portal vein was unsuccessful, and the child was scheduled for urgent retransplantation. The child died, however, 24 h later as no donor organ was available.

Patient 2

A 2-year-old girl underwent OLT for secondary biliary cirrhosis due to biliar atresia and complex polysplenia syndrome with situs inversus and an absent inferior vena cava. The child had undergone several previous operations for duodenal atresia, but attempts at biliary drainage were unsuccessful. Three weeks before OLT, no portal or mesenteric vein could be identified on Doppler sonography and angiography. Indirect mesenteric portography only showed small proximal mesenteric vessels drained retroperitoneally into parietal collaterals and intercostal veins; a variceal coronary vein was the only residual anatomically recognizable splanchnic vein (Fig. 3). This child had been refused for transplantation in most transplant centers but was accepted into our program as a possible candidate for PVA.

A full-size liver graft was implanted 1 month later with an end-to-end caval anastomosis direct caudal to the right atrium and an end-to-side portal anastomosis on a varicose retroperitoneal vein. Because of macroscopic signs of insufficient liver perfusion and a



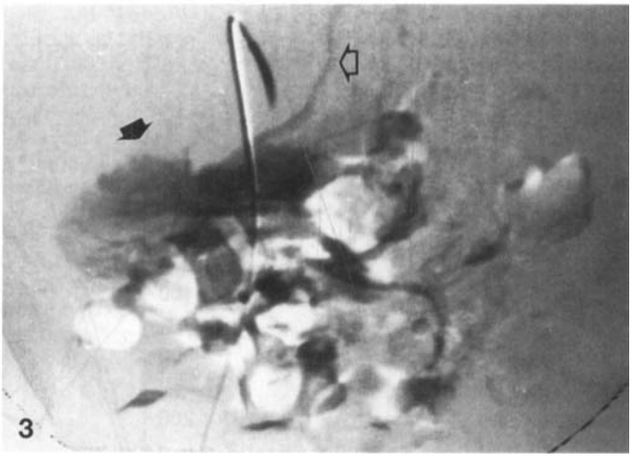


Fig. 2 Patient 1. Angiogram of the abdominal aorta. There is an anastomotic stenosis of the interposed iliac artery graft (*solid arrow*) that provides the arterial blood supply for the liver only. There is no opacification of the portal vein due to thrombosis of the PVA at the site of gastroduodenal artery (*open arrow*)

Fig. 3 Patient 2. Angiogram of the abdominal aorta. There is drainage of the mesenterial blood by parietal collateral and intercostal veins on indirect mesenteric portography (*solid arrow*). Note the absence of portal and mesenteric vein opacification and the presence of a small collateral to the caval vein (*open arrow*)

peroperative further increase in blood lactate, we decided to add a PVA by connecting the donor splenic artery stump to the donor portal vein. Before the procedure, the portal flow was electromagnetically measured at 150 ml/min; this increased to 500 ml/min after the PVA. This portal flow remained stable until closure of the abdomen in the presence of a stable cardiac output. Given the very dense small bowel adhesions, which carried the risk of profuse peroperative bleeding in the case of further dissection, the choice was made to delay the Roux-en-Y loop for biliary reconstruction until there was normalization of the coagulation tests.

Liver function tests increased rapidly after reperfusion and during the following postoperative days (Fig. 4). US evaluation postoperatively showed a portal blood flow velocity of about 60 cm/s (V_{max}). Four days later, the biliary anastomosis was easily performed with a Roux-en-Y loop without marked splanchnic venous hypertension. Fifteen days later a small bowel perforation occurred that required a laparotomy. During the operation, the liver was macroscopically found to be congested with ascites, as seen in cases of hyperarterialization. A liver biopsy confirmed parenchymal congestion on frozen section, and arterialization was abdished. US examination showed that the portal blood flow velocity had decreased (V_{max} = 40 cm/s), but it remained stable at subsequent follow-ups. No deterioration in liver function occurred after dearterialization of the portal vein. Eighteen months after transplantation, the patient's condition remains very good with normal hepatic tests and without signs of parenchymal alterations on liver biopsy.

Discussion

The effects of PVA and its influence on hepatic function are not yet completely understood. Although hemodynamic parameters and changes in pressure seem to be involved in the pathogenesis of hepatocyte damage [3, 13], it has been reported that hepatic function and architecture are preserved when portal vein flow and pressure are kept within physiological values [6, 7, 9]. In adult LT, Sheil et al. reported interesting results of regulating temporary arterial shunts in the porta with a flow of up to 1000 ml/min [12]. The benefit is claimed to be due to a reduction in the duration of the anhepatic phase and stable portal reperfusion. Erhard et al. [4] showed good results using a permanent PVA procedure in which the portal blood flow was tapered to 1500–1800 ml/min, permitting not only recovery of graft function but also long-term adequate liver function. Performed in life-threatening, clinical situations, this represented the first time that permanent PVA was done without deterioration in liver function or the development of neurological complications.

No data are thus far available on the PVA procedure in pediatric LT, where it is well known that low portal flow or portal vein thrombosis can produce devastating complications. Our experience demonstrates that temporary PVA may be useful in specific situations, such as low portal flow due to multiple, spontaneous, retroperitoneal venous shunts or in the absence of a SMV. Ploeg and others [11] concluded in a recent study that the presence of portosystemic shunts after LT has a significant, detrimental effect on liver graft function and survival. In our first patient, PVA allowed for satisfactory liver reperfusion after a first unsuccessful attempt by classical portal blood flow reperfusion. In patient 2, no SMV was found because of a totally abnormal splanchnic vascular network; the only way to provide sufficient liver perfusion was to perform a PVA. In this case, the portal blood flow rose to 500 ml/min, allowing for stable

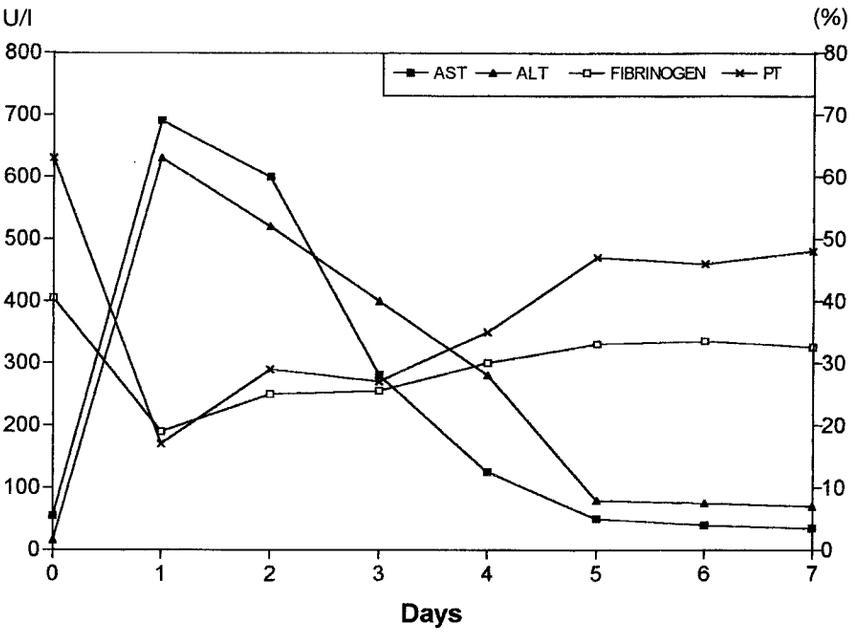


Fig. 4 Patient 2. Serum transaminase activity, fibrinogen level, and prothrombin time of the patient after the PVA procedure

reperfusion and successful transplantation. Graft function in both cases improved in the 1st postoperative week.

Although there is no doubt about its benefit in the immediate postoperative period, we have no data on long-term arterialization in children. If, in the case of portal thrombosis but a patent mesenteric vein, a jump graft between this vein and the liver is the best solution, arterialization is the only choice when no splanchnic veins can be found or when retroperitoneal venous shunts cannot be safely ligated. Arterialization through the gastroduodenal artery is probably insufficient, but through a splenic artery it is excessive. Some degree of banding of this arterial shunt should be considered if permanent arterialization is intended. Thus far, one has only been able to speculate about the minimum and maximum portal flow needed when performing transplantation on small children in order to avoid the risk of primary nonfunction or delayed graft function, on the one hand, and hyperarterialization on the other. For now, the option of temporary PVA and interruption of the shunt whenever hyperarterialization is suspected may be the preferred option when splanchnic veins cannot be found.

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