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BRIEF REPORT

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Primary permanent arterialization of the portal vein in liver transplantation

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Abstract Permanent total arterialization of the portal vein in liver transplantation has been described as a method of providing portal inflow after insufficient thrombectomy due to chronic occlusion of the portal-vein system. A specific problem is the restriction of the arterial inflow and its long-term adaptation after transplantation. We describe here the surgical techniques and clinical course of three patients who underwent portal-vein arterialization for liver transplantation. Two patients had an uneventful course. In one patient, a flow reduction by means of coil embolization of one arterial inflow branch was performed; thereafter, the patient recuperated well. Analysing the microcirculation of an arterialized graft in comparison with liver grafts with normal non-arterialized portal-vein inflow, we observed an increase in inter-sinusoidal distance and a decrease in sinusoidal red blood cell velocity. From a technical point of view, we recommend permanent portal-vein arterialization by an iliac artery graft interposition from the subdiaphragmatic aorta. The inflow to the portal vein can easily be reduced by the banding of the arterial graft interposition.

Keywords Liver transplantation · Portal vein thrombosis · Arterialization

Introduction

Arterialization of the portal vein is known from porto-systemic shunt surgery [5]. In clinical liver transplantation the arterialization of the portal vein was first described by Sheil [9], who used temporary arterialization in order to shorten warm ischemia time and provide adequate reperfusion. Normal portal-vein graft perfusion, i.e. the drainage of the recipient's portal blood to the portal vein of the graft, may be severely compromised or even impossible, with extensive splanchnic thrombosis or reduced inflow after re-canalization of the portal vein due to thrombotic occlusion. In portal-vein thrombosis, the first therapeutic option is thrombectomy [1, 11]. If adequate flow cannot be achieved, the use of large collaterals or graft interpositions (e.g. iliac vein) to the superior mesenteric vein is required [7]. In the case of insufficient portal inflow, either a caval-to-portal anastomosis [10] or the permanent total arterialization of the portal vein has been described [2]. In this paper we describe our clinical experience with three cases of portal-vein arterialization in orthotopic liver transplantation.

Case reports

Case 1

A 35-year-old woman with Budd–Chiari-Syndrome underwent transplantation for acute decompensation. Pre-operative Doppler sonography and magnetic resonance tomography showed thrombosis of all major portal branches. This finding was confirmed intra-operatively. Thrombectomy resulted only in a low-flow

situation, therefore portal-vein arterialization was indicated. In addition, an inferior vena cava thrombosis was found, which could be thrombectomized after explantation of the liver, prior to the veno-venous bypass being started. Since no donor iliac arteries were available, arterial reconstruction was performed directly to the supra-truncal aorta, and the portal vein was then anastomosed to the common hepatic artery on the insertion of the gastroduodenal artery of the recipient (Fig. 1). After reperfusion (10-h cold ischemia time) of the graft, we adjusted portal-vein flow at 1.000 ml/min (electromagnetic measurement) by mending the portal vein.

Initial graft function was good, but the patient developed progressive hepatomegaly, ascites and renal failure. As clinical symptoms persisted 14 days after transplantation, we measured the pressure of the portal vein (15 mmH_2O)–central vein(13 mmH_2O) gradient and did not find pathological differences (transjugular–transhepatic approach). With the patient showing persistent hepatomegaly, ascites and signs of right-heart decompensation (increased cardiac index and central venous pressure) 3 months after transplantation, we performed an angiographic coil embolization of the common hepatic artery in order to reduce the inflow. As a result, the portal vein was perfused only from the gastroduodenal artery (Fig. 1). The liver volume decreased, and the right ventricular decompensation disappeared. At present, after 3 years of follow-up, the patient's liver function tests are normal (bilirubin, AST and ALT), and she is in excellent clinical condition.

Case 2

A 53-year-old man received a liver graft for HCV-induced cirrhosis with hepatocellular carcinoma. The presence of portal-vein thrombosis was known prior to the transplantation, and the intra-operative thrombectomy was unsuccessful. Portal inflow was realized by means of anastomosis of the portal vein onto the recipient's common hepatic artery. In order to achieve an inflow reduction, we induced stenosis of the artery by a banding procedure (also flow adaptation to 1.000 ml/min measured intra-operatively). Arterial reconstruction of the graft was done by means of anastomosis to the insertion of the recipient's splenic artery. Reperfusion was performed after 12 h of cold ischemia time, and the initial function of the graft was excellent. The postoperative course was uneventful, with a follow-up, meanwhile, of almost 3 years. Liver biopsies have disproved any signs of fibrosis, and the liver function tests are normal.

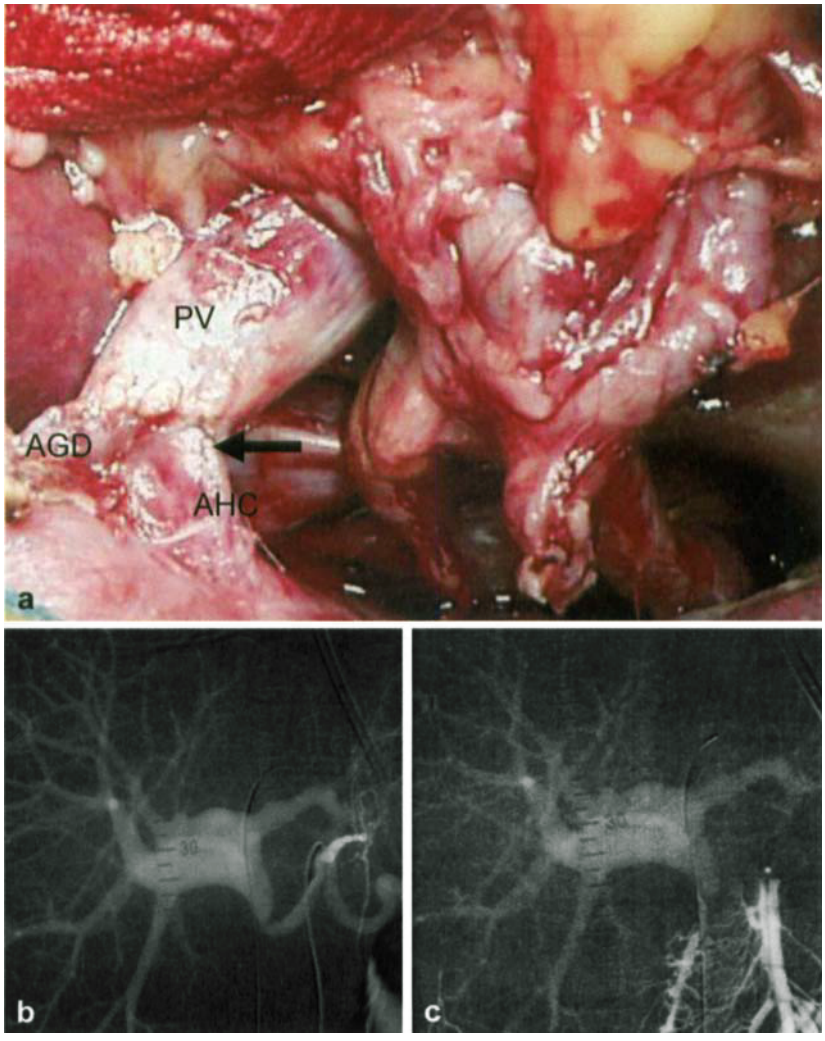

Fig. 1 **a** Intra-operative aspect of portal vein arterialization. The portal vein (*PV*) is inserted onto the bifurcation of the common hepatic (*AHC*) and gastroduodenal artery (*AGD*). After anastomosis, the vein was tapered (patient 1). **b** Postoperative angiography via the coeliac trunk, showing the portal inflow from the common hepatic-artery branch, which was embolized to treat hyperarterialization and normalize the blood flow. **c** Postoperative angiography via the superior mesenteric artery to the portal-vein which remained after embolization of the common hepatic artery (*see above*)

Case 3

A 55-year-old male patient underwent re-transplantation for recurrent hepatitis B-virus-related cirrhosis. Nine months beforehand, he had received a distal spleno-renal shunt (Warren) due to the development of a portal-vein thrombosis, portal hypertension, and repeated gastro-oesophageal bleeding from varices. Intra-operatively, the shunt was functioning well. For hepatic graft perfusion an iliac artery was inserted to the subdiaphragmal aorta, and one branch was anastomosed in end-to-end fashion to the donor's hepatic artery, and the other to the portal vein. The iliac arterial branch to the portal vein was then banded to ensure flow restriction. Reperfusion after 9 h of cold ischemia time was good, and the postoperative course was uneventful. Four months after transplantation, the patient had to undergo re-operation for a bile duct stenosis. Thereafter, the clinical course continued without any problems. In annual routine liver biopsies, no signs of fibrosis were found. During the above-mentioned re-operation the sinusoidal perfusion of the graft was visualized microscopically by OPS imaging [3]. The microvascular perfusion showed a homogeneous sinusoidal perfusion pattern. However, compared with the physiological hepatic microcirculation assessed in healthy donors for living related-donor liver transplantation (manuscript submitted), both an increase in intersinusoidal distance and a decrease in sinusoidal red blood cell velocity were observed (Fig. 2). The further postoperative course was uneventful for nearly 2.5 years.

Discussion

Successful liver transplantation is usually dependent on arterial and portal-vein perfusion [10]. Today, portal-vein thrombosis is not a contra-indication for liver transplantation. Neither the absence of a significant splanchnic venous branch at angiography, nor unsuccessful thrombectomy, may be considered an absolute contra-indication for transplantation. In most cases, an intra-operative portal-vein thrombectomy is feasible, thus achieving adequate portal-vein flow. Alternatively, one can use interposition grafts to the mesenteric vein or an atypical collateral in order to restore the portal inflow. A caval-to-portal anastomosis is another alternative using systemic venous blood for portal perfusion and accepting persistent splanchnic hypertension. With arterialization similarly, splanchnic hypertension persists, and portal blood circulates systemically before it reaches the liver. There have been speculations on the impact of continuous portal blood shunting on cerebral function (encephalopathy) and other metabolic effects. None of our three patients has developed any neurological signs or metabolic abnormalities or any other clinical problems (bleeding from gastro-oesophageal varices, high ammonia level, etc.).

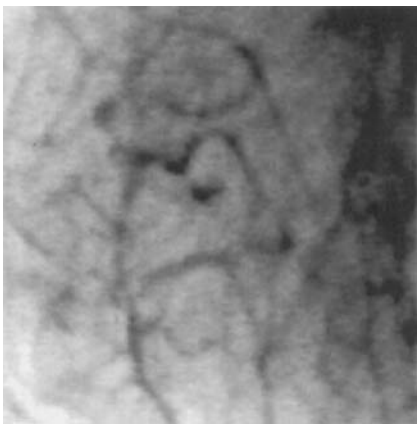

Fig. 2 Hepatic sinusoidal microcirculation in patient 3 recorded during re-operation for bile duct kinking (OPS imaging, Cytoscan A/R, magnification ×400). The homogeneous sinusoidal perfusion following arterialization of the portal vein is of note

Arterialization of the portal vein was described by Erhard et al. [2] as a permanent procedure in three patients. The authors used an iliac artery interposition graft from the infra-renal aorta for arterial and portal perfusion. One of the three patients received a heterotopically positioned auxiliary transplant. Good results were also achieved in paediatric and living related-donor liver transplantations in a few patients [6, 8].

The use of iliac bifurcation grafts to the recipient's aorta is technically easy. This should be the first choice in cadaveric organ transplantation and complicated vascular situations. Arterialization of the portal vein can also be used as augmentation of portal flow for insufficient flow after thrombectomy [6].

Arterial hyperperfusion of the portal vein resulting in right-heart decompensation and graft fibrosis [2] must be prevented by flow adaptation. In experimental models in pigs, auto-regulation at the sinusoidal level has been described, preventing portal hyperperfusion [4]. We could demonstrate a homogeneous sinusoidal perfusion not exceeding the level of physiological hepatic microcirculation in patient 3, suggesting the existence of a similar mechanism in this patient. The altered microcirculation in comparison with normally perfused liver grafts remains unexplained, and we can only speculate on the role of cholestasis and arterialization. Erhard et al. [2] adjusted portal perfusion to 1.500–1.800 ml/min. Having measured normally vascularized grafts after transplantation (unpublished data) we consider an initial portal flow of 1.000 ml/min. to be adequate. To diminish the flow further would increase the risk of thrombosis [8]. In analogy to arterio-venous fistulae for hemodialysis access, the flow may increase gradually over some time and cause hepatic fibrosis in the later course. In our experience, hyper-arterialization did not occur, except for in the first patient, in whom the excess flow was corrected by occlusion of the common hepatic artery. Since this patient is receiving long-term anticoagulation treatment we did not perform a liver biopsy because all clinical and laboratory parameters are normal. In the other patients we did not observe the development of fibrosis.

In conclusion, portal-vein thrombosis is not to be considered an absolute contra-indication to liver

transplantation. If the result of thrombectomy is unsatisfactory and the portal flow remains insufficient, arterialization of the portal vein is a good alternative in assuring portal inflow. Intra-operative flow adaptation is important. The best technical realization is a donor iliac artery interposition graft from the subdiaphragmatic aorta for arterialization of the portal vein. The inflow must be reduced by the artificially stenosis of the graft. Of course, normalization of portal hypertension is not achieved, and the risk of gastrointestinal bleeding remains. Further clinical studies and a longer follow-up time are necessary, for the evaluation of portal inflow dimensioning and hemodynamic changes and for the prevention of hyper-arterialization-induced side effects.

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