Arterial complications after liver transplantation

Abstract From September 1988 through April 1998, 1000 liver transplantsations were performed on 911 patients. During the postoperative control examinations of 837 patients, we found 23 (2.74%) with hepatic artery thromboses, 27 stenoses of the hepatic artery (3.22%), and 6 aneurysms of the graft artery. Seventeen patients underwent retransplantation because of arterial complications. Depending on the clinical symptoms, we treated both the local situation as well as the resulting complications of inadequate arterial graft flow. The aneurysms were primarily treated surgically. The first choice of treatment of stenoses was balloon angioplasty. Early postoperative artery thromboses were also treated surgically by thrombectomy in selected cases. For the resulting biliary and local septic complications we preferred endoscopic and drainage procedures. Our clinical experiences have led us to find pretransplantation angiography recommendable, especially in the case of splanchic artery stenoses, for bypassing from the aorta for arterial perfusion of the graft.

Key words Liver transplantation - Arterial complications

Introduction

The adequate reconstruction of afferent vessels is a precondition for successful liver transplantation. Poor blood inflow and technical problems often lead to early postoperative organ failure and graft loss. Arterial complications prevail with an incidence of 2–25% in patients after liver transplantation of which 50% require retransplantation [4, 11]. Early portal vein occlusion is rare, occurring in 1–3% of transplantsations.

Stenoses, occlusions, steal syndromes, and aneurysms can arise as complications in the arterial reconstruction after liver transplantation. Stenoses are found in the recipient artery, at the anastomosis, or in the graft artery and are situated extra- or intrahepatically. Pre-existing stenoses of the celiac artery are hemodynamically relevant after transplantation and affect graft function. Stenoses at the anastomotic region are usually caused by technical errors. Postanastomotic stenoses result from the twisting and kinking of graft arteries that are too long. Immunologic and local ischemic damage of the vascular wall will be discussed further below. Early occlusions and thrombosis of the graft artery, respectively, are caused by technical error, preservation injury, or occur in marginal organs with high peripheral vascular perfusion resistance [10]. Late arterial occlusions may develop from early stenoses. Aneurysms of the liver artery after transplantation, if extrahepatic, are often mycotic aneurysms caused by a local infection (e.g., partial biliary insufficiency that induces erosion of the vascular wall or anastomotic suture). If aneurysms occur intrahepatically, they are often caused by liver punctures [2, 7, 16]. In the present paper we discuss the incidence, causes, and treatment of arterial complications in our liver transplant patients.
Patients and methods

Between September 1988 and April 1998, 1000 liver transplantations were performed on 911 patients at our institution. Thirty-four transplantations were performed on pediatric patients and were excluded from this study. The preoperative evaluation of transplant recipients included abdominal CT scan, angiography, and duplexsonography of the hepatic artery, vena cava, and portal vein. In a few patients with acute liver failure and/or severe coagulopathy, angiography was omitted. Organs were either harvested by surgeons from our institution, or when simultaneous arterial and portal perfusion (n = 649), or were shipped from other institutions (n = 351).

Liver transplantation in adult patients was done following standard techniques and using a veno-venous bypass. The hepatic vena cava was resected, and an end-to-end interposition of the donor vena cava was performed. Reconstruction of the portal vein was done as end-to-end veno-venous anastomosis. The arterial anastomosis is typically performed on the insertion of the gastroduodenal artery of the recipient hepatic artery. Depending on the preoperative angiography, the anastomoses were modified [13]. Patients with complex vascular reconstructions (reconstruction of accessory arteries, multiple anastomoses) received low-dose heparinization during the early postoperative period. We continued anticoagulation only in patients with hypercoagulation disorders (Budd-Chiari syndrome etc.). Vascular patency was routinely checked by sonographic examination during hospitalization and upon presentation at our liver transplant outpatient clinic. In the case of pathologic findings (modification of systolic waveform and abnormal values for resistive index and systolic acceleration time) with clinical symptoms, angiographic control was ruled out. All patients received immunosuppressive therapy consisting of either cyclosporine- or tacrolimus-based regimens.

Retransplantation (n = 89) became necessary in 79 patients. The reasons for retransplantation were initial non-function of the graft (n = 20), recurrence of viral hepatitis (n = 19), chronic rejection (n = 14), acute hepatic artery thrombosis (n = 13), extrahepatic bile duct destruction (n = 10), acute rejection (n = 6), recurrence of Budd-Chiari syndrome (n = 2), occlusion of vena cava (n = 2), and combinations of the above (n = 3).

Results

Oclusions of the hepatic artery

We examined 837 of our adult transplant patients in the follow-up program for incidence of arterial complications. Occlusions of the hepatic artery were found in 23 patients (2.74%). These patients had undergone transplantation for posthepatic cirrhosis (n = 9), postethylic cirrhosis (n = 5), PSC (n = 2), Budd-Chiari syndrome (n = 1), and other reasons (n = 6). The chronologic occurrence of the occlusion is shown in Fig. 1. Six of the 23 patients (26%) developed early occlusions (till day 30 after transplantation). Thirteen patients (57%) underwent retransplantation because of arterial occlusion (Fig. 1). Five patients died (22%) – two of them after retransplantation. Three deaths were not associated with arterial problems or retransplantation.

All but two patients – receiving transplants for acute liver failure – underwent an angiography of the splenic arteries preoperatively, a routine procedure for patients before transplantation. At that time six stenoses of the celiac trunk were diagnosed (three caused by compression from the arcuate ligament), one aneurysm of the lienal artery, and one of the mesenteric artery.

Arterial reconstruction was performed as standard procedure (at the insertion of the gastroduodenal artery) using the celiac trunk of the donor in 17 patients. As a consequence of preoperative angiography, in one operation this was combined with a resection of the arcuate ligament. In five patients, a graft interposition (iliac artery) to the aorta was performed, of which one was infrarenal and four directly subdiaphragmatic. In another patient, the splenic artery insertion was used for the anastomosis.

Arterial occlusions occurred with an isolated increase of transaminases (n = 1) or in combination with cholestasis (n = 18). Five patients developed cholangitis, and in seven an ischemic destruction of the biliary system was seen. Seven patients developed an abscess in the liver. Two patients showed clinical symptoms of an initially nonfunctioning organ. In one patient, the hepatic artery occlusion was diagnosed as an incidental finding. For all patients, the clinical and duplexsonographic suspicion of arterial occlusion was confirmed by angiography. In three patients, steal syndrome to the lienal artery was also diagnosed, and in one patient, a portal vein occlusion was found.

Apart from retransplantation (n = 13) as treatment of the arterial occlusion in three patients, a hepatico-jejunostomy was performed to aid biliary drainage because of extrahepatic biliary duct destruction. One patient diagnosed with acute postoperative thrombosis was immediately re-operated and thrombectomized with an additional resection of the arcuate ligament and banding of the splenic artery. During retransplantation, four patients underwent standard arterial reconstruction at the insertion of the gastroduodenal artery.

![Fig. 1 Chronologic occurrence of hepatic artery occlusion after transplantation and incidence of retransplantation](image-url)