Abstract

Dissection of the common hepatic artery is a rare complication after orthotopic liver transplant. Subsequent thrombosis and occlusion of the transplant artery can result in graft failure requiring retransplant. We describe a case of hepatic artery dissection, occurring on the basis of primary vasculopathy, extending into the celiac trunk, with subtotal occlusion of the vessel through accompanying thrombosis. An attempt of endovascular rescue led to successful recanalization of the vessel and graft survival.

Key words: Dissection, Common hepatic artery, Stent, Vasculopathy

Introduction

Vascular complications of orthotopic liver transplant can result in graft loss and mortality. Thrombosis can affect both the hepatic artery and the portal vein, and it occurs in up to 5% of all liver transplants. Vessel stenosis usually develops in the hepatic artery, at the site of the anastomosis, or close to it, owing to clamping injury and can be treated successfully by percutaneous transluminal angioplasty. Dissection of the hepatic artery is a rare complication in liver transplant surgery with few surgical options. Endovascular management can be a reasonable alternative, as surgical treatment is associated with increased morbidity and mortality. Continuous improvement of the endovascular equipment including guide wires, catheters, and flexible stents that allow interventions in small and tortuous vasculature.

Case Report

A 67-year-old otherwise healthy woman was started oral anticoagulation with phenprocoumon owing to therapy-resistant atrial fibrillation. On the basis of a mild cirrhosis (child A), which was neither evident clinically, or on ultrasound, or with respect to the liver enzymes at the time making the decision for oral anticoagulation, she developed acute liver failure with massive jaundice and progressive hepatic encephalopathy aspartate aminotransferase, 13.12 μkat/L (787 U/L); alanine aminotransferase, 21.99 μkat/L (1317 U/L); gamma glutamyl transpeptidase, 7.38 μkat/L (443 U/L); alkaline phosphatase, 3.11 μkat/L (186 UL); bilirubin, 540.5 μmol/L (31.6 mg/dL); and international normalized ratio, 2.2. Within 2 days, she had reached a MELD score of 40 and was set on high urgency for a liver transplant.

Two days later, the patient received a liver allograft from a 32-year-old deceased donor. The liver transplant was performed in standard Belghiti technique with side-to-side cavo-caval anastomosis, end-to-end portal venous reconstruction, and hepatic artery anastomosis using a gastroduodenal patch. Bile duct reconstruction was performed using an end-to-end choledochotomy procedure and inserting a T tube. Explant histopathology revealed massive...
hemorrhagic necrosis of both liver lobes, severe cholestasis, and inflammation as signs of an acute toxic liver damage. Additionally, mild micronodular cirrhosis was present, which was regarded as the basis for the subsequent phenprocoumon-induced liver failure. The early posttransplant course was uneventful. On the fifth day after the transplant, she developed a single rejection episode, which was treated with a standard corticosteroid regimen. The subsequent postoperative course was unremarkable, and the patient recovered well from the transplant surgery with liver enzymes constantly decreasing: aspartate aminotransferase, 1.05 μkat/L (63 U/L); alanine aminotransferase, 2.45 μkat/L (147 U/L); gamma glutamyl transpeptidase, 4.12 μkat/L (247 U/L); alkaline phosphatase, 1.55 μkat/L (93 U/L); international normalized ratio of 1.1, and bilirubin of 141.96 μmol/L (8.3 mg/dL) on the 18th day after transplant. Three weeks after the transplant, she developed a sudden increase of liver enzymes: aspartate aminotransferase, 1.88 μkat/L (113 U/L); alanine aminotransferase, 7.62 μkat/L (457 U/L); gamma glutamyl transpeptidase, 22.39 μkat/L (1343 U/L); alkaline phosphatase, 7.25 μkat/L (435 U/L); and international normalized ratio of 1.2. Progressive cholestasis was present with bilirubin levels of 220.6 μmol/L (12.9 mg/dL). Duplex ultrasound could not detect patent flow over the hepatic artery.

Subsequent contrast-enhanced computed tomography revealed a homogeneous perfusion of the liver parenchyma in the portal-venous phase with only a circumscribed area of decreased perfusion in segment VII. The arterial phase demonstrated a dissection of the common hepatic artery (CHA) with extension into the celiac trunk (white arrow, panels A and C). The dissection led to a subtotal occlusion of the CHA by a large thrombus in the recipient segment (white arrowheads, panels B and D). Only a slim residual lumen of the donor vessel could be identified on computed tomography, with a remnant arterial perfusion of the transplanted organ (white dotted arrow in panel B).

The patient underwent subsequent arterial angiography for further evaluation via a transfemoral approach and a 5-French equipment. Angiography confirmed the dissection of the recipient’s CHA extending into the celiac trunk and even further into the orifice of the splenic artery. The dissection led to subtotal occlusion and thrombosis of the CHA because of the lack of a re-entry, with only a marginal residual perfusion of the donor artery (white dotted arrow, panel E) predominantly via the gastroduodenal artery.

The dissection started approximately 2 cm proximal of the arterial anastomosis with retrograde extension into the celiac trunk. Angiography of the celiac trunk further revealed vasculitic transformation in the territory of the left gastric artery with “beading” small arteries and several microaneurysms (black arrows, panel F). The initial revascularization attempt under full heparinization via the transfemoral approach (black asterix in panels E and G) remained unsuccessful with repeated dislocation of the endovascular instruments from the celiac trunk because of elongation and tortuosity of the vasculature.

To achieve a more-stable position of the endovascular equipment—adapted to the arterial anatomy of the celiac trunk—an additional transbrachial approach via the left brachial artery was performed, and a 5-French sheath placed a few centimeters above the celiac orifice (white asterix in panel G). The correct lumen was successfully fathomed with a 0.014-inch guide wire from the celiac trunk to the anastomosis. Percutaneous transluminal angioplasty with a 4-mm and 5-mm balloon led to a significant increase of the perfusedarterial lumen. Subsequent stent angioplasty (6 × 40 mm self-expanding Nitinol Stent; Sinus Superflex, Ettingen, Germany) of the CHA was performed with the proximal stent covering the entry of the dissection in the celiac trunk.

The patient continued regular immuno-suppression with tacrolimus and dual therapy with clopidogrel 75 mg/d and aspirin 100 mg/d for 6 weeks to prevent early rethrombosis. The patient recovered well from the endovascular intervention and duplex ultrasound on discharge confirmed a patent celiac trunk and hepatic artery. Six months after the procedure, the patient was re-evaluated with persistent regular flow over the stent, and the hepatic artery on Duplex ultrasound (resistance index, 0.83; peak velocity, 97.8 cm/s; panel H) and regular liver function studies aspartate aminotransferase, 0.30 μkat/L (18 U/L); alanine aminotransferase, 0.55 μkat/L (33 U/L); gamma glutamyl transpeptidase, 0.52, μkat/L (31 U/L); bilirubin, 17.1 μmol/L (1.0 mg/dL); cholinesterase,
89.4 μkat/L (5363 U/L); alkaline phosphatase, 0.82 μkat/L (49 U/L); and an international normalized ratio of 1.1.

Discussion

Hepatic artery thrombosis occurs in approximately 5% of orthotopic liver transplant, severely limiting graft survival.¹ Dissection of the hepatic artery as a complication after a liver transplant is rare. Intimal dissection of the hepatic artery is encountered after transarterial embolization as a bridging therapy to transplant.² Intraoperative intimal dissection of the recipient’s hepatic artery, sometimes extending into the celiac trunk, has been described.³,⁴ An insufficient recipient’s hepatic artery may require anastomosis, that is, to the splenic artery directly,⁴ or by using an autologous interpolate (eg, a segment of the inferior mesenteric artery).⁵ If this is not technically feasible, the donor artery can be anastomosed to intestinal branches, such as a jejunal arcade of a Roux-en-Y limb.³ Hepatic artery dissection occurring postoperatively after an orthotopic liver transplant is rare, and only several cases have been described worldwide.⁵-⁷ Kim and associates reported the first series of hepatic artery dissection in a living-donor liver transplant.⁷ Adami and associates described the development of a hepatic artery dissection from a pseudoaneurysm, extending into the graft bifurcation requiring retransplant.⁶

We describe a case of recipient’s CHA dissection extending into the celiac trunk, most likely in a retrograde manner. The dissection led to a subtotal occlusion of the CHA by a large thrombus. The reduced perfusion of the transplant with residual perfusion via the remnant gastroduodenal artery led to an increase of liver enzymes and bilirubin levels and clinical deterioration of the patient. Computed tomography angiography demonstrated a sustained graft perfusion through the patent portal vein and only a marginal perfusion of the donor hepatic artery.

An interdisciplinary consensus was made for an attempt for endovascular rescue of the CHA. A transbrachial approach via the left brachial artery allowed successful probing of the true lumen, pneumatic dilatation, and subsequent stent placement. Endovascular recanalization of the hepatic artery after a liver transplant is an acceptable technique for treating hepatic artery stenosis to enhance tissue perfusion and to prolong graft survival. Reports of small patient series have described percutaneous transluminal angioplasty as a safe method for endovascular treatment for hepatic artery stenosis after liver transplant.⁸-¹⁰ Hepatic

Figure 1. Dissection of the Common Hepatic Artery

![Figure 1](image-url)

Dissection of the Common Hepatic Artery

Figure 1. Transverse (A and B) and coronal (C and D) CT-angiography images depict the dissection of the common hepatic artery (arrow in A) extending into the celiac trunk. The dissection led to a subtotal occlusion of the CHA by a large thrombus in the recipient segment (white arrow heads, panels B and D). Only a slim residual lumen of the donor vessel could be identified on CT, with a remnant arterial perfusion of the transplant organ (white dotted arrow in panel B). Computed tomography-angiography findings were confirmed by intraarterial angiography (E), which further showed vasculitic transformation in the territory of the left gastric artery with ‘beading’ small arteries and several micro-aneurysms (black arrowheads, panel F). Endovascular repair was only achieved via a transbrachial approach (white arrow in G), while the transfemoral could not enable a stable position of the instruments. Follow-up evaluation by duplex ultrasound 6 months after endovascular repair (H).
artery stenosis can develop any time, depending on the surgical technique, as a result of vascular clamp injury, because of microvascular lesions associated with preservation solutions, or during the course of allograft rejection. The dissection of the CHA, in our case, was most likely the result of pre-existing vasculopathy of the abdominal vasculature. Although the vasculitis serology was negative, underlying vasculitis was considered the most likely cause of the dissection, especially when reviewing the angiogram of the celiac trunk (panel F). Surgical manipulation of the hepatic artery, which is usually tolerated by the healthy recipient artery, most likely then led to dissection extending from the recipient’s CHA, retrogradely into the celiac trunk. Besides endovascular repair, therapeutic options for this vessel pathology included surgical revision of the CHA together with the anastomosis or if retransplant of the dissection extends into the graft. Both are associated with significant morbidity and mortality. A combined surgical-intervention approach was discussed as another alternative: limited relaparotomy with exposure of the hepatic artery and subsequent endovascular access and stent placement in the operating room would have been the other option to avoid retransplant.

References