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ORIGINAL ARTICLE

Gastroduodenal artery steal syndrome during liver transplantation: intraoperative diagnosis with Doppler ultrasound and management

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Summary

Arterial steal syndrome (ASS) after liver transplantation has been reported. ASS causes arterial hypo-perfusion of the graft liver and devastating consequences. However, the diagnosis tends to be delayed. We present the recognized case of a gastroduodenal artery (GDA) steal syndrome that was diagnosed with intraoperative Doppler ultrasound and treated with GDA ligation during the liver transplantation. The patient had variation of hepatic artery anatomy (low bifurcation of the hepatic artery). Graft liver had the common hepatic artery and aberrant left hepatic artery. Doppler ultrasound of the liver was performed after the arterial reconstruction between the donor common hepatic artery and recipient right hepatic artery. It showed low hepatic arterial flow. There is no backflow bleeding from the donor aberrant left hepatic artery stump. After ligating big GDA, hepatic arterial waveform inside the liver drastically improved and strong backflow bleeding was recognized from the donor left aberrant hepatic artery stump. The current case should show the efficacy of intraoperative Doppler ultrasound of the liver on ASS and alert clinician to ligate GDA to prevent ASS if hepatic arterial flows are suboptimal.

Introduction

Arterial steal syndrome (ASS) after liver transplantation has been reported in case reports or limited series [1–8]. Patients usually present with elevated liver enzyme levels, impaired graft function, cholestasis, or hepatic arterial thrombosis after liver transplantation [1–8]. Most of the cases are related to the splenic artery (lienalis steal syndrome). However, some cases of gastroduodenal artery (GDA) steal syndrome have also been reported [3,4,7]. Some factors, including the stenosis of the graft artery, rejection, or flow pattern changes with increased portal blood flow and decreased hepatic arterial blood flow after liver transplantation have been suspected [6–9]. Diagnosis, treatment, and prophylaxis are still not well established. Doppler ultrasound of the liver is an accurate and reliable method to evaluate the hepatic arterial flow [2]. Currently, it has begun to be used widely after liver transplantation. We have been using intraoperative Doppler ultrasound of the liver to evaluate the vascular anastomosis during liver transplantation [10]. We report a 71-year-old male who developed GDA steal syndrome during orthotopic liver transplantation. He was diagnosed with intraoperative Doppler ultrasound of the liver and subsequently treated with ligation of GDA. Effect of intraoperative Doppler ultrasound of the liver on ASS and ligation of GDA are described.

Case report

A 71-year-old man with end-stage liver disease, secondary to chronic hepatitis C virus infection and hepatocellular carcinoma (HCC), was suffering from deteriorating condition. In addition, he had an organized partial portal

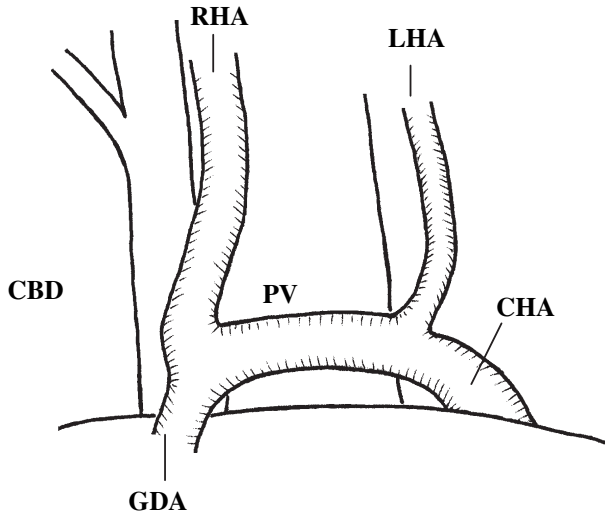

Figure 1 Recipient hepatic artery anatomy before anastomosis. RHA, right hepatic artery; LHA, left hepatic artery; CHA, common hepatic artery; GDA, gastroduodenal artery; CBD, common bile duct; PV, portal vein.

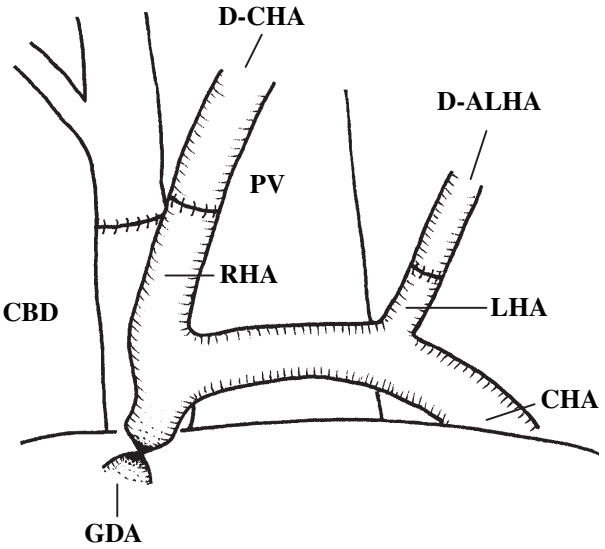

Figure 2 Recipient hepatic artery anatomy after anastomosis. RHA, right hepatic artery; LHA, left hepatic artery; CHA, common hepatic artery; GDA, gastroduodenal artery; CBD, common bile duct; PV, portal vein; D-CHA, donor common hepatic artery; D-ALHA, donor aberrant left hepatic artery.

vein thrombosis and marginal renal function. Computed tomography scan and magnetic resonance imaging showed 3.5 cm HCC in the right lobe (segment 8), splenomegaly, and ascites. The patient was thus listed for liver transplantation with a model for end-stage liver disease score of 24 points.

An organ became available from a 54-year-old male donor who died from an intracranial bleed. The patient was prepared for liver transplantation. There was severe portal hypertension and the hepatectomy was performed without veno-venous bypass. Caval reconstruction was carried out using the piggyback technique with three hepatic vein cuffs. Thrombectomy of the portal vein was performed. The graft liver was flushed through the lower cava of the donor with cold lactated ringer solution. The patient had variation of hepatic artery anatomy (low bifurcation of the hepatic artery, Fig. 1). The right hepatic artery was big (7 mm in diameter) and had strong pulse on palpation. It was located on the right side in the hepatico-duodenal ligament. The right hepatic artery seemed to arise from GDA (7 mm in diameter). The left hepatic artery was smaller (5 mm in diameter) and arose from the common hepatic artery (3 cm proximal from GDA). Graft liver had the common hepatic artery (6 mm in diameter) and aberrant left hepatic artery (4 mm in diameter, Fig. 2). Hepatic arterial reconstruction was performed with 7-0 polypropylene-interrupted sutures between the donor common hepatic artery and the recipient right hepatic artery (Fig. 2). The donor common hepatic artery had pulse, but it was weak on palpation. Doppler ultrasound of the liver (SSD 1700; Aloka, Tokyo, Japan) was performed after the arterial reconstruction. It showed low hepatic arterial flow. Peak systolic flow at the right hepatic artery in the graft was 13.0 cm/s, diastolic flow was 0.0 cm/s and resistive index was 1.0 (type-3 waveform according to García-Criado's classification) [11]. There was no backflow bleeding from the donor aberrant left hepatic artery stump. Anterior sutures of the anastomosis were removed and checked for thrombus or intimal dissection.

There was neither thrombus nor intimal dissection. There was a good arterial flow through the anterior wall suture line. Anterior wall was resutured. Then, GDA was ligated. After ligating big GDA, hepatic arterial waveform inside the liver improved drastically and strong backflow bleeding was recognized from the donor aberrant left hepatic artery stump. Peak systolic flow at the right hepatic artery in the graft was 78.8 cm/s, diastolic flow was 22.8 cm/s and resistive index was 0.71 (type-1 waveform according to García-Criado's classification) [11]. After ligation of GDA, peak systolic flow at the right hepatic artery became 6 times higher than that of before ligation and also diastolic flow became higher. Waveform of the hepatic artery changed from spiky arterial waveform (type 3) to normal arterial waveform (type 1). Prior to the ligation, a very low hepatic arterial flow in the graft liver was suggested by Doppler ultrasound. After ligation of the GDA, an excellent hepatic arterial flow in the graft liver was suggested by Doppler ultrasound. There was very

strong pulse on palpation. Subsequently another hepatic arterial anastomosis was performed between the donor aberrant left hepatic artery and recipient left hepatic artery (Fig. 2). Strong arterial bleeding was recognized from the cystic artery stump during the donor cholecystectomy. Duct-to-duct biliary reconstruction was performed thereafter. After hemostasis was completed, the abdomen was closed. The patient was transferred to the intensive care unit in stable condition.

The intraoperative results were as follows: operative time 12 h and 51 min, cold ischemia time 7 h and 45 min, warm ischemia time 58 min, red blood cell transfusion 14 units, fresh frozen plasma 17 units, platelet 16 units, cryoprecipitate 20 units, and cell saver blood 250 ml. The postoperative course was uneventful and graft function returned to normal levels. Abdominal ultrasound with Doppler ultrasound was performed daily for 7 days after the transplant. They showed patent hepatic artery, portal vein and hepatic vein. He was extubated on postoperative day 2.

He was transferred to a general medical floor on postoperative day 3 and discharged home in stable condition on postoperative day 7. Currently, the patient is well, and has normal liver function without any sign of liver ischemia.

Discussion

This report shows clinical scenario strongly suggestive of GDA steal syndrome during liver transplantation, which was noticed with intraoperative Doppler ultrasound and subsequently treated with GDA ligation.

Arterial steal syndrome after liver transplantation is a rare but devastating complication that may result in ischemic damage to the liver and biliary complication. Most of the ASS in liver transplantation was diagnosed late after liver transplantation. Patients usually present with elevated liver enzyme levels, impaired graft function, cholestasis or hepatic arterial thrombosis after liver transplantation [1–8]. ASS may be the cause of many of the hepatic artery thrombosis [12]. Incidences of ASS, approximately 3.2–6%, in the liver transplantation are reported [6,7,12]. However, the concept of ASS is not widely recognized and the actual impact of ASS in liver transplant remains unclear [9]. There are limited numbers of reports [1–8,12]. Diagnosis is usually based on clinical findings and angiographic findings that show hepatic arterial flows steal to either the splenic artery or GDA. Most of the cases are related to the splenic artery (lienalis steal syndrome).

Doppler ultrasound of the liver is an accurate and reliable method to evaluate the hepatic arterial flow [2,10]. Currently, it has begun to be used widely after liver transplantation. We have been using intraoperative Doppler ultrasound of the liver to evaluate the vascular anastomosis during liver transplantation. Possible roles of Doppler ultrasound of the liver on ASS are reported [2,9]. However, hepatic arterial flow pattern in ASS with Doppler ultrasound is not well studied yet. In the present case, the hepatic artery had anatomical variation. Initially, it seemed to be the right replaced hepatic artery from the superior mesenteric artery. However, after further dissection of this artery, we found that the patient had low bifurcation of the hepatic artery (Fig. 1). The right hepatic artery was big and had strong pulse on palpation. The right hepatic artery seemed to arise from GDA. The left hepatic artery was smaller and arose from the common hepatic artery (3 cm proximal from GDA). Doppler ultrasound study was performed after the arterial reconstruction. Suboptimal hepatic arterial flow was observed. After ligating GDA, hepatic arterial flow drastically improved. Intraoperative Doppler ultrasound studies were useful and reliable to evaluate the hepatic arterial flow [2]. In the history of cadaveric liver transplantation, GDA has been ligated in order to increase the flow toward the hepatic artery [13]. Ligation of GDA depends on the surgeon's experience and anatomical milieu. In some center, GDA is usually tied during cadaveric liver transplantation. In the other, GDA is not tied (especially live donor liver transplantation). If hepatic arterial flow is suboptimal, GDA should be tied to prevent ASS. Aortic implantation of the graft hepatic artery also may be the alternative procedure in case of suboptimal hepatic arterial inflow.

Mechanisms of ASS have not yet been clearly confirmed. However, some factors including the stenosis of the graft artery, increased intra-hepatic arterial resistance because of rejection or preservation injury, or flow pattern changes with increased portal blood flow and decreased hepatic arterial blood flow after liver transplantation have been suspected [6–9]. Although hepatic artery stenosis should be excluded before diagnosing ASS, stenosis of the graft hepatic artery may augment the shift of blood flow into GDA [7].

The treatment was performed in order to prevent the steal and keep the optimal hepatic arterial flow, which included splenectomy, coil embolization, banding, the use of the splenic artery for reconstruction of the hepatic arterial flow, or aortic implantation of the graft artery [1–8,12]. Most of the cases were treated after liver transplantation as a result of the clinical symptom and angiographic findings. If suboptimal hepatic arterial flows are noticed during liver transplantation, ASS should be the differential diagnosis. If the steal artery is recognized, ligation of the steal artery is the treatment of choice. However, if any doubt of imperfection of the hepatic arterial flow, converting hepatic arterial inflow using

arterial conduits from the infrarenal aorta or supra-celiac aorta is another recommendable treatment of choice. This procedure is technically more demanding and may be associated with a higher complication rate.

In summary, we report a case of liver transplant recipient who developed GDA steal syndrome during liver transplantation. GDA steal syndrome was noticed with intraoperative Doppler ultrasound and subsequently treated with ligation of GDA. This event appears to be related to hepatic arterial variation. Although not widely accepted, transplant team's surgeon should be aware of the possibility of this complication when performing hepatic arterial reconstruction. Early diagnosis and treatment, depending on intraoperative Doppler ultrasound, are critical in order to prevent ASS and ischemic damage of the graft liver.

References

1. Manner M, Otto G, Senninger N, Kraus T, Goerich J, Herfarth C. Arterial steal: an unusual cause for hepatic hypoperfusion after liver transplantation. *Transpl Int* 1991; **4**: 122.
2. Rasmussen A, Hjortrup A, Kirkegaard P. Intraoperative measurement of graft blood flow—a necessity in liver transplantation. *Transpl Int* 1997; **10**: 74.
3. Langer R, Langer M, Scholz A, Felix R, Neuhaus P, Keck H. The splenic steal syndrome and the gastroduodenal steal syndrome in patients before and after liver transplantation. *Aktuelle Radiol* 1992; **2**: 55.
4. Vogl TJ, Pegios W, Balzer JO, Lobo M, Neuhaus P. Arterial steal syndrome in patients after liver transplantation: transarterial embolization of the splenic and gastroduodenal arteries. *Rofo Fortschr Geb Rontgenstr Neuen Bildgeb Verfahr* 2001; **173**: 908.
5. Geissler I, Lamesch P, Witzigmann H, Jost U, Hauss J, Fangmann J. Splenohepatic arterial steal syndrome in liver transplantation: clinical features and management. *Transpl Int* 2002; **15**: 139.
6. De Carlis L, Sansalone CV, Rondinara GF, *et al.* Splenic artery steal syndrome after orthotopic liver transplantation: diagnosis and treatment. *Transplant Proc* 1993; **25**: 2594.
7. Nüssler NC, Settmacher U, Haase R, Stange B, Heise M, Neuhaus P. Diagnosis and treatment of arterial steal syndromes in liver transplant recipients. *Liver Transpl* 2003; **9**: 596.
8. Rogers J, Chavin KD, Kratz JM, *et al.* Use of autologous radial artery for revascularization of hepatic artery thrombosis after orthotopic liver transplantation: case report and review of indications and options for urgent hepatic artery reconstruction. *Liver Transpl* 2001; **7**: 913.
9. Farges O, Belghiti J. Editorial on ''diagnosis and treatment of arterial steal syndromes in liver transplantation recipients''. *Liver Transpl* 2003; **9**: 603.
10. Nishida S, Kato T, Levi D, *et al.* Effect of protocol Doppler ultrasonography and urgent revascularization on early hepatic artery thrombosis after pediatric liver transplantation. *Arch Surg* 2002; **137**: 1279.
11. García-Criado A, Gilabert R, Salmerón JM, *et al.* Significance of and contributing factors for a high resistive index on Doppler sonography of the hepatic artery immediately after surgery: prognostic implications for liver transplant patients. *AJR Am J Roentgenol* 2003; **181**: 831.
12. Uflacker R, Selby JB, Chavin K, Rogers J, Baliga P. Transcatheter splenic artery occlusion for treatment of splenic artery steal syndrome after orthotopic liver transplantation. *Cardiovasc Intervent Radiol* 2002; **25**: 300.
13. Shaw BW, Iwatsuki S, Starzl TE. Alternative methods of arterialization of the hepatic graft. *Surg Gynecol Obstet* 1984; **159**: 490.