Hepatic Artery-duodenal Fistula with Gastrointestinal Bleeding after Liver Transplantation

Ruey-Shyang Soong, MD; Kuang-Tse Pan[1], MD; Chen-Fang Lee, MD; Wei-Chen Lee, MD

Gastrointestinal bleeding caused by peptic ulcer or portal hypertension is a serious complication in liver transplant recipients. However, Gastrointestinal bleeding from hepatic artery origin after liver transplantation is rare. We report duodenal massive bleeding secondary to erosion of hepatic artery after liver transplantation in two patients. This was diagnosed by angiography and treated subsequently by surgical intervention with hepatic artery re-anastomosis. We could not salvage one of these patients who died of complications of graft failure. Therefore, early diagnosis and aggressive surgical intervention may be required in order to salvage both recipient and graft. *(Chang Gung Med J 2009;32:579-83)*

Key words: liver transplantation, gastrointestinal bleeding, hepatic artery-duodenal fistula

Gastrointestinal (GI) bleeding is a serious complication for liver transplantation (LT) recipients. The major causes of the bleeding are peptic ulcer, enteritis, portal hypertension, and Roux-en-Y bleeding after choledochojejunostomy reconstruction.[1] The mortality rate of GI bleeding is high if prompt intervention is not initiated. There are only few cases of reported bleeding from the hepatic artery in LT receipts.[2] In retrospect we reviewed 114 LT recipients at our institution for the complication of upper GI bleeding after transplantation between January, 2002 and January, 2006. Herein, we report our experience of two liver transplant recipients who developed severe GI bleeding secondary to hepatic artery-duodenal fistula.

CASE REPORT

Case1

A 53-year-old male with hepatitis B-related decompensated liver cirrhosis had live donor liver transplantation with right lobe graft on the 21st February 2006. Preoperative upper gastrointestinal endoscopy revealed duodenal bulb deformity with deep penetrating ulcer over anterior wall. He received ulcer treatment omperazole (30 mg/day) for 6 weeks. Repeated endoscopy showed a healed ulcer on anterior wall of duodenum.

After transplantation, the postoperative course was uneventful and he was transferred from the intensive care unit to ordinary ward on the 27th February 2006. However, he developed hematemesis complicated with shock on the 7th March 2006. Eight units of packed red blood cells were transfused immediately. Endoscopy was attempted but could not delineate the site of bleeding. Subsequently an angiography was performed. It revealed extravasation of contrast from the recipient's-sided hepatic artery to the duodenum (Fig. 1). Because of ongoing hemorrhage, emergent laparotomy was underdone. Operative finding showed hepatic artery was adhered to the duodenum which had a 2 x 2 cm ulcer with a perforation on the anterior wall of the first portion and the recipient's common hepatic artery was erod-

From the Department of Liver Transplantation; [1]Department of Radiology, Chang Gung Memorial Hospital, Taipei, Chang Gung University College of Medicine, Taoyuan, Taiwan.
Received: Apr. 30, 2008; Accepted: Jun. 26, 2008
Correspondence to: Dr. Wei-Chen Lee, Department of Liver Transplantation, Chang Gung Memorial Hospital. 5, Fusing St., Gueishan Township, Taoyuan County 333, Taiwan (R.O.C.) Tel.: 886-3-3281200 ext. 3366; Fax: 886-3-3285818;
E-mail: raymond@cgmh.org.tw

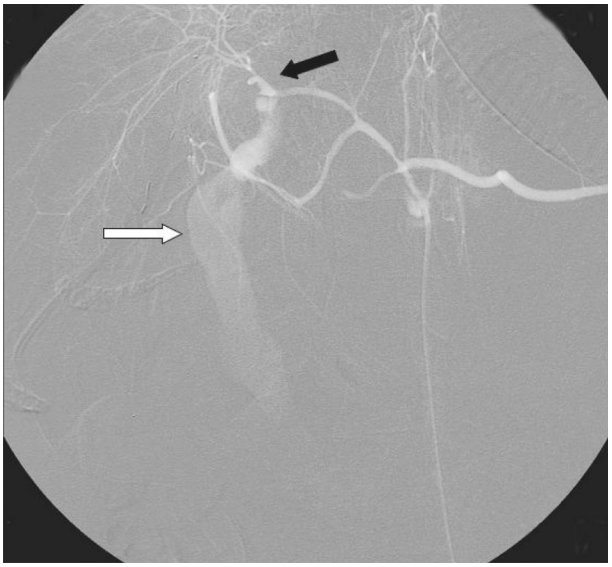

Fig. 1 Case 1 celiac arteriography. Contrast media leaks from recipient's common hepatic artery and donor's right hepatic artery anastomostic site (black arrow) into duodenum (white arrow).

ed and resulted in artery anastomosis disruption. The recipient's common hepatic artery was ligated. Hepatic artery flow was reconstructed from the recipient's right gastric artery (1 mm) to graft's right hepatic artery (2 mm) by microscopic technique with 9-Nylon. Duodenal perforation was repaired primarily and covered with an omental patch. Liver graft was pink and soft immediately after the hepatic artery was re-vascularized. The liver function returned normal after one week and patient was discharge from the hospital on th 15th of April 2006.

Case 2

A 51-year-old male was admitted due to alcoholic liver cirrhosis with failure on the 5th March 2006. He had an orthotopic liver transplantation with a reduced-size right lobe graft on the 17th March 2006. Postoperatively, he had a complication of sepsis with blood culture and ascites culture positive for *A. baumannii*. Antibiotics were shifted from Ampicillin (800 mg every 8 hours) plus ceftriaxone (1 gm every 12 hours) to Imepenem (500 mg every 12 hours). However, progressive epigastralgia was noted. He had melena with hemoglobulin level which dropped from 10 to 8 mg/dl without hemodynamic change on 2nd April 2006. Famotidine was replaced by Omeprazole (30 mg per day). Unexpectedly, he had hematochezia which leaded to hemodynamic instability on 8th August 2006. Endoscopy revealed bleeding from a 1.5 x 1.5 cm ulcer on the superior wall of the 1st portion of the duodenum. Hemoclip was applied and diluted adrenaline was injected locally. The local treatment failed and bleeding persisted. The patient was then referred for angiography. The angiography revealed extravasation of contrast from the receipt's common hepatic artery and graft's celiac artery anastomostic site into the duodenum. (Fig. 2) Embolization with n-butyl cyanoacrylate (NBCA) and lipidol was performed for temporary control. As bleeding persisted and patient's hemodynamic state was unstable despite 12 units of packed bed blood cells and 12 fresh frozen plasma transfusion, the patient then underwent an emergency operation. Operative findings revealed that the hepatic artery anastomostic site had attached to the duodenum. A 1 x 1 cm ulcer was found on the superior wall of first part of the duodenum which had eroded the anastomosis of the graft's celiac artery and recipient's common hepatic artery. A thrombosis in the recipient's common hepatic artery was also noted. After debriding, microvascular reanastomosis was done between the recipient's com-

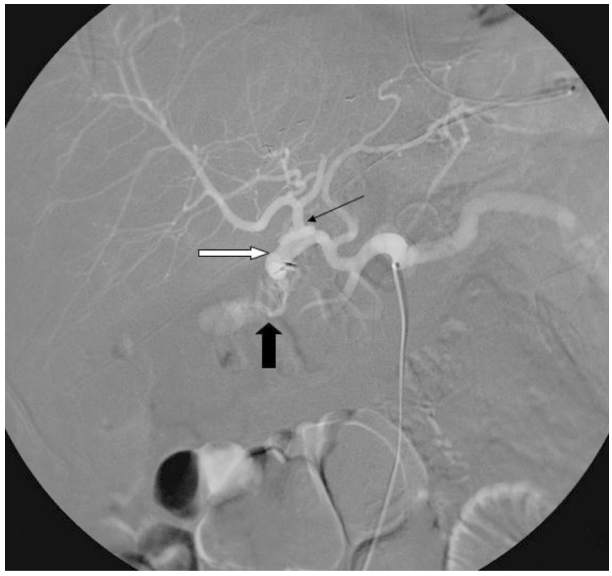

Fig. 2 Case 2 celiac arteriography. The white arrow shows the recipient's common hepatic artery and the thin black arrow reveals graft's celiac artery. The thick black arrow indicates a contrast leakage into duodenum.

mon hepatic artery (3 mm) and graft's celiac artery (3 mm) with 9-0 Nylon. The liver remained congested even after revascularization. The recipient died of graft failure on the second day after the surgery.

DISCUSSION

GI bleeding is a serious complication in liver transplantation recipients. In respect to GI bleeding both patient and graft survival rate is relatively low. In 1997, Tabasco-Minguillan et al. reported that the cumulative incidence of GI bleeding of liver transplantation was 8.9%. The most common causes are ulcer (27%), following enteritis (26%), portal hypertension lesion (15%), Roux-en-Y bleeds and other (30%).[1] However, there were only a few case reports of GI bleeding from the hepatic artery. We report two cases of the GI bleeding from the hepatic artery.

Incidence of peptic ulcer is 15% after liver transplantation.[3] Liver transplant patients should be treated as being at high risk for developing of ulcers and further complications, because these patients receive corticosteroids, require major surgery, or may have associated infection or organ failure. Association of preoperative *H. Pylori* infection and occurrence of peptic ulcer has also been demonstrated after liver transplantation.[3,4] Ulcer bleeds are not uncommon events, and are usually secondary to erosion of the gastroduodenal artery or superior pancreaticoduodenal artery. There are very few reported cases of peptic ulcer bleeding secondary to hepatic artery erosion. Gastroduodenal artery is a branch of the common hepatic artery, thereafter it continues as the hepatic artery proper, which further divides into right and left hepatic artery which runs upwards towards the liver. Therefore, the hepatic artery is too high up for direct erosion by a duodenal ulcer. However, for LT patients, such as that in case 1, because of dissection in the supra-colic compartment, there is alteration to the normal anatomy, which may subsequently lead to erosion of hepatic artery by an ulcer.

Intra-abdominal vascular aneurysm inducing vascular-enteric fistula is rare in proportion to GI bleeding and a hepatic artery aneurysm is a rare phenomenon in LT. The clinical presentation of these cases could present a non-symptomatic, abdominal pain with pulsating mass, hemoperitoneum, hemobilia and GI bleeding. Extra-hepatic pseudoaneurym are common with infection origin, genrally expected to develop in the first two months after LT. The cause of infection is often systemic or associated with subhepatic infected fluid acumination. Pseudoaneurysm arising from the hepatic artery has the reported incidence of 0.3~2%.[5] Hepatic pseudoaneurysm eroding into the duodenum resulting in hepatic artery-duodenal fistula is reported in blunt abdominal trauma,[6] polyarteritis nodosa,[7] or hepatic artery access port.[8] There was only one such case reported in a living donor liver transplantation recipient.[2] Early diagnosis requires a high level of suspicion, as presentation may be non-specific with hemobilia, unexplained fever, graft dysfunction, or a falling hemoglobilin level. But pseudoaneurysm remains unrecognized until it presents with profound shock and rupture into the peritoneum or GI tract which requires emergency laparotomy. Hence 50% risk of pseudoaneurysm rupture at onset is present in the transplantation group, differing from a 14% risk of rupture in non-transplantation patients.[5] Persistent sub-hepatic fluid collection is a key etiologic factor for pseudoaneurysm. Mutiple-phasic contrast enhanced helical computer tomography may show intrahepatic pseudoaneurysm, but extrahepatic pseudoaneurysm may be misdiagnosed.[9]

Angiography still plays a definitive role in the investigation to demonstrate the source of bleeding and is also a resource for determining an intervention. Embolization to diminish acute hemorrhage is used as a temporary measure allowing re-transplantation. However, in Taiwan, due to the shortage of orthotopic liver graft, re-transplantation after hepatic artery emboization is almost impossible. Immediate revascularization is the standard treatment even in infected field, being the factor enhancing survival compared to ligation of the hepatic artery.[10] Both cases underwent revascularation by microsurgery. In one case the right gastric artery of the recipient was used to replace the common hepatic artery. Alternatively a covered coronary stent graft was reported to successfully treat the hepatic artery pseudoaneurysm as a treatment choice.[11]

Bleeding due to a hepatic artery–duodenal fistula is a rare phenomena and is associated with a high mortality rate in LT. A duodenal ulcer eroding the extra-hepatic artery could be due to post-operative adhesion associated with hepatic artery adhesion to duodenal wall. Sub-hepatic local infection is another

factor which may cause a hepatic pseudoaneurysm, furthermore massive GI bleeding originates from the ruptured hepatic pseudoaneurysm into the duodenum. Aggressive post-LT ulcer prophylaxis and early recognition and diagnosis of a pseudoaneurysm may reduce the mortality rate. Embolization is a temporary method for hemorrhage control. Exploratory laparotomy with hepatic artery revascularization is necessary in orthotopic liver graft shortage area.

REFERENCES

1. Tabasco-Minguillan J, Jain A, Naik M, Irish W, Fung JJ, Rakela J, Starzl TE. Gastrointestinal bleeding after liver transplantation. Transplantation 1997;63:60-7.
2. Slater K, Ludkowski M, Mehling J, Forman L, Pieramici E, Wachs M, Kam I, Bak T. Hepatic artery pseudoaneurysm - duodenal fistula after living donor liver transplant. Clin Transplant 2004;18:734-6.
3. Hosotani Y, Kawanami C, Hasegawa K, Watanabe T, Ito T, Oike F, Kaihara S, Okazaki K, Tanaka K, Chiba T. A role of Helicobacter pylori infection in the development of duodenal ulcer after adult living-related liver transplantation. Transplantation 2003;76:702-4.
4. Klompmaker IJ, Slooff MJ, de Bruijn KM, Sluiter WJ, Bams JL. Prophylaxis with ranitidine against peptic ulcer disease after liver transplantation. Transpl Int 1988;1:209-12.
5. Fistouris J, Herlenius G, Backman L, Olausson M, Rizell M, Mjornstedt L, Friman S. Pseudoaneurysm of the hepatic artery following liver transplantation. Transplant Proc 2006;38:2679-82.
6. Aboujaoude M, Noel B, Beaudoin M, Ghattas G, Lalonde L, The Bao, Bui, Oliva VL. Pseudoaneurysm of the proper hepatic artery with duodenal fistula appearing as a late complication of blunt abdominal trauma. J Trauma 1996;40:123-5.
7. Shuster TA, Almeida J, Coats R, Kalra A, Silver D. Gastrointestinal bleeding as the initial manifestation of a polyarteritis nodosa-associated hepatic artery aneurysm-duodenal fistula--a case report. Vasc Endovascular Surg 2004;38:563-8.
8. Reiher F, Ridwelski K, Pross M, Lippert H. Acute upper gastrointestinal hemorrhage as a complication of hepatic artery port catheter. Zentralbl Chir 2000;125:174-7.
9. Marshall MM, Muiesan P, Srinivasan P, Kane PA, Rela M, Heaton ND, Karani JB, Sidhu PS. Hepatic artery pseudoaneurysms following liver transplantation: incidence, presenting features and management. [erratum appears in Clin Radiol 2001 Sep;56:785]. Clin Radiol 2001;56:579-87.
10. Bonham CA, Kapur S, Geller D, Fung JJ, Pinna A. Excision and immediate revascularization for hepatic artery pseudoaneurysm following liver transplantation. Transplant Proc 1999;31:443.
11. Muraoka N, Uematsu H, Kinoshita K, Takeda T, Morita N, Matsunami H, Itoh H. Covered coronary stent graft in the treatment of hepatic artery pseudoaneurysm after liver transplantation. J Vasc Interv Radiol 2005;16:300-2.

肝動脈十二指腸瘻管在肝臟移植術後

宋睿祥 潘廣澤[1] 李正方 李威震

因消化性潰瘍或門脈高壓所引起的出血在肝臟移植是一個嚴重的併發症。但由肝動脈所引起的出血卻是罕見的病例報告。兩個案例在肝臟移植術後因肝動脈接合處浸潤侵蝕，進而形成十二指腸瘻管出血。在血管攝影區分出血來源後，進而施行外科治療，重新進行肝臟動脈接合，以維持移植肝臟的功能。其中一個案例先使用肝動脈栓塞失敗後才進行手術，在術後死於肝臟衰竭的併發症。對於這樣的少見的術後併發症應即早診斷及積極外科介入以保存肝臟功能及病人性命。(長庚醫誌 2009;32:579-83)

關鍵詞：肝臟移植，消化道出血，肝動脈十二指腸瘻管

長庚紀念醫院 台北院區 肝臟移植中心，[1]放射診斷科；長庚大學 醫學院
受文日期：民國97年4月30日；接受刊載：民國97年6月26日
通訊作者：李威震醫師，長庚紀念醫院 肝臟暨移植外科。桃園縣333龜山鄉復興街5號。Tel.: (03)3281200轉3366; Fax: (03)3285818; E-mail: raymond@cgmh.org.tw