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HEPATIC ARTERY PSEUDOANEURYSM LIGATION AFTER ORTHOTOPIC LIVER TRANSPLANTATION—A REPORT OF 7 CASES[1]

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Pseudoaneurysm (PA) is a rare but life-threatening complication of liver transplantation. The authors present their experience on 7 patients treated by ligation of a post-OLT PA. Hepatic artery ligation or embolization was performed from 10 to 70 days after liver transplantation. Of the seven patients, four survived, one developed a biliary stricture, treated by percutaneous ballon dilatation, two died of a complication not related to treatment, and one died of multiple organ failure.

Vascular complications remain a significant cause of postoperative morbidity and mortality following liver transplantation. Thrombosis of the hepatic artery remains the most common complication (*1–15*). Although ligation of a native hepatic artery is tolerated in the majority of cases (*23*), the integrity of the arterial blood supply is important for graft and patient survival. It is generally accepted that dearterialization, particularly if it occurs early, usually results in gangrene of the allograft, biliary complications—or, in some cases, recurrent sepsis. However, long-term survival after hepatic artery thrombosis has not been rare (*2, 12*). In these cases the vitality of the allograft is thought to be preserved by portal flow and development of arterial collaterals—which have been demonstrated to arise from the superior mesenteric and the splenic arteries, as well as diaphragm, omentum, and bowel (*24*). Pseudoaneurysms (PAs)* are uncommon after orthotopic liver transplantation. They may be asymptomatic and detected during imaging evaluation for other reasons or manifest with

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* Abbreviation: PA, pseudoaneurysm.

gastrointestinal bleeding or hemoperitomeum. PAs usually occur at the arterial anastomosis and are often caused by a combination of infection and/or technical failure. Therapeutic options are excision with ligation, retransplantation, or excision with immediate arterial reconstruction (*10–22*). This article describes the clinical course and the outcome of seven patients treated by ligation of the hepatic artery pseudoaneurysms.

MATERIALS AND METHODS

Case material. Between 1986 and 1991, 2516 patients underwent orthotopic liver transplantation. There were 2003 adults and 513 children. We analyzed the course of 7 patients who underwent ligation of hepatic pseuaneurysm in this period. There were six adults (range 38–53 years old) and one child. Hepatic artery ligation or embolization was performed from 10 to 70 days after liver transplantation. The reasons for ligation were bleeding, either due to pseudoaneurysm rupture into the gastrointestinal (GI) tract (n=5), or hemoperitoneum (n=1) (Table 1). In one case, a pseudoaneurysm was incidentally discovered during angiographic evaluation of hepatic artery patency (case 1).

Case reports. Case 1: A 41-year-old woman who had her first transplant for non A, non B hepatitis was retransplanted four months later as a result of hepatic artery thrombosis and multiple hepatic abscesses. Postoperatively angiographic evaluation to check patency of the hepatic artery demonstrated a 3-cm pseudoaneurysm at the site of the previous celiac axis–hepatic artery anastomosis. The pseudoaneurysm was resected and the celiac axis ligated 70 days after the second transplant. There were no positive cultures from the pseudoaneurysm. Liver function tests did not show any significant elevation after ligation (Fig. 1). The patient is currently alive and well five years after ligation.

Case 2: A 51-year-old man underwent liver transplantation for cryptogenic cirrhosis. Postoperatively he developed severe pancreatitis and a pancreatic fistula requiring laparotomy with pancreatic debridement leaving the abdominal cavity open. One week later, he developed massive abdominal bleeding due to hepatic artery rupture. This necessitated ligation of the hepatic artery 20 days after transplantation. Cultures from the pseudoaneurysm site were negative. Transaminase levels did not show any significant changes after ligation; however, the levels of bilirubin and canalicular enzymes increased progressively and peaked four weeks after ligation and then decreased in the subsequent weeks (Fig. 1). Liver biopsies obtained at the time of peak elevations showed steatosis and cholestasis, consistent with resolving ischemic injury. After a prolonged convalescence and secondary closure of the abdominal wound he is currently doing well three years after ligation.

Case 3: A 5-year-old girl received a liver transplant for biliary atresia and a failed Kasai lost her first graft to chronic rejection requiring retransplantation two years later. She developed severe pancreatitis after the second transplant necessitating total pancreatectomy and splenectomy, duodenal resection, partial gastrectomy, and transverse colon resection seven days later. The aortic-iliac graft that had been placed at the second transplant was intact. However, severe gastrointestinal tract bleeding secondary to an arterial-gastric fistula occurred, necessitating ligation of the hepatic artery 54 days after the transplant. She grew *Enterococcus* from the peritoneal cavity at the time of ligation. In this case pancreatitis and associated infection were presumably the causes of the pseudoaneurysm. There were no changes in her bilirubin or liver enzyme levels (Fig. 1). She is well two years after ligation.

Case 4: A 38-year-old woman received a liver transplant for non A, non B hepatitis. Bile duct reconstruction was performed with a choledochocholedocostomy. Twenty-five days after transplantation, she developed a bile leak necessitating laparotomy and conversion to a Roux-en-Y hepaticojejunostomy. One month later she suddenly went into shock from massive gastrointestinal tract bleeding and hemoperitoneum. Emergency angiography revealed a pseudoaneurysm at the stump of the ligated donor gastroduodenal artery and she underwent ligation of the hepatic artery above and below the pseudoaneurysm 55 days after transplantation. She developed a stricture of the common hepatic bile duct and recurrent cholangitis five months after ligation of the hepatic artery (Fig. 2). There was a mild elevation of the SGOT and SGPT for several days and peak elevation of her bilirubin to 6.2 during the first week after ligation. Abdominal computed tomography one month after ligation confirmed a small abscess in the left lobe of the liver and liver biopsy confirmed ischemic changes. The biliary stricture responded well to two transhepatic balloon dilatations. She is currently alive and well five years after ligation. Recent transhepatic cholangiography showed no evidence of biliary stricture.

Case 5: A 51-year-old man with the diagnosis of primary sclerosing cholangitis had previous upper abdominal surgery complicated by a biliary-pleural fistula. Transplantation was performed with an aortic-

TABLE 1. Clinical data of 7 patients who underwent occlusion of the hepatic artery after OLTX

Patient number	Age	Sex	Liver disease	Cause of PA*	Presentation	Time/method of occlusion	Outcome
1	41	F	Hepatitis C	Infection	Associated with HAT**	70 days, ligation	Alive and well for 5 years
2	51	M	Cryptogenic cirrhosis	Pancreatitis	Bleeding (hemoperitoneum)	20 days, ligation	Alive and well for 3 years
3	5	F	Biliary atresia	Pancreatitis	Bleeding (gastrointestinal)	54 days, ligation	Alive and well for 2 years
4	38	F	Hepatitis C	Biliary leak	Bleeding (gastrointestinal)	55 days, ligation	Alive and well for 5 years Biliary stricture 5 months after ligation, responded to percutaneous dilation
5	51	M	Sclerosing cholangitis	Biliary leak	Bleeding (gastrointestinal)	60 days, embolization	Death from *Candida* peritonitis 1 month after embolization
6	53	M	Alcoholic cirrhosis	Biliary leak	Bleeding (gastrointestinal)	60 days, ligation	Death from *Candida* peritonitis 3.5 months after ligation
7	58	F	Hepatitis C	Biliary leak	Bleeding (gastrointestinal)	10 days, ligation	Acute hepatic failure—retransplanted, but died of multiorgan failure and sepsis 5 days later

* PA—Pseudoaneurysm.
** HAT—Hepatic Artery Thrombosis.

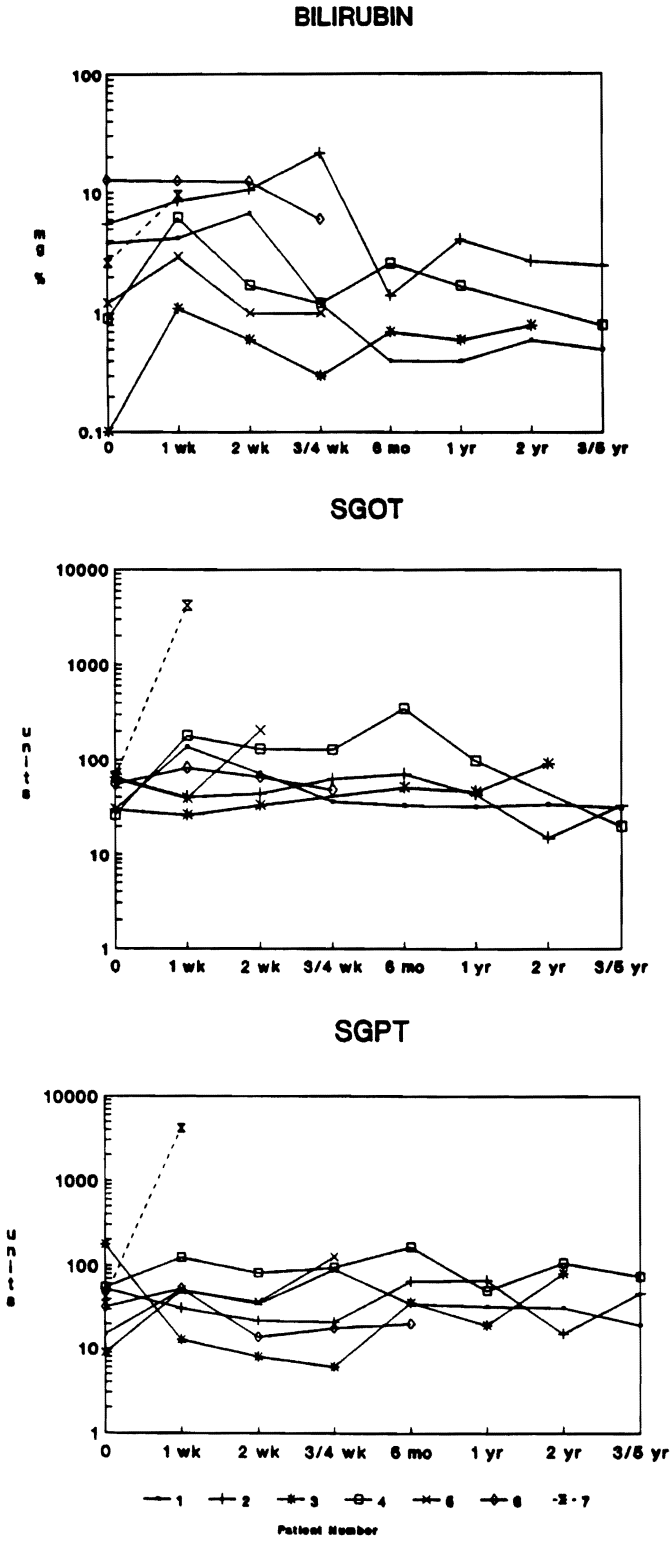

FIGURE 1. Changes in the bilirubin and transaminases in the patients.

FIGURE 2. Marked stricture of the common hepatic duct (arrows) causing high-grade biliary obstruction demonstrated on transhepatic cholangiogram. Early necrosis of the confluence of the right and left hepatic ducts is evident by irregularity and indistinctness of the duct walls and intraluminal filling defects. This area developed into a central biloma cavity over several months.

iliac graft. One month later, he developed massive gastrointestinal tract bleeding due to an arterial enteric fistula from a mycotic pseudoaneurysm of the hepatic artery. At exploration bleeding was encountered from an arterial branch in the hilum and it was ligated. However, the pseudoaneurysm could not be identified from the dense scar tissue. A bile leak was also noted at the choledochojejunostomy, which was controlled with a drain. He was discharged from the hospital with a low output biliary fistula. Ten days later he was readmitted for rectal bleeding. Emergency angiography (Fig. 3A) and embolization of the pseudoaneurysm successfully stopped the bleeding. Subsequent abdominal CT two weeks after embolization showed a focal infarct of the right hepatic lobe (Fig. 3B). There were no significant elevations of the bilirubin or the transaminases (Fig. 1). He recovered after embolization having only a persistent small biliary leak well controlled by the drain and the patient was discharged. One month later he was readmitted with severe jaundice. The biliary fistula had already closed and transhepatic cholangiography was normal. Liver biopsy showed severe chronic rejection that could not be reversed by increasing immunosuppression and he received a retransplant. During the course of the hepatectomy dense inflamed tissues were encountered. The abdomen was left partially open because of sepsis. He succumbed to *Candida* peritonitis one month later.

Case 6: A 53-year-old man received a liver transplant for alcoholic cirrhosis. Arterial reconstruction was performed with an aortic-iliac graft. Postoperatively he had two episodes of biliary leak and failed biliary revisions at four and six weeks. Two months later he bled from a mycotic aneurysm and required ligation of the hepatic artery graft with resection of the aneurysm. *Candida* was cultured from the perivascular soft tissues of the hepatic artery aneurysm. Ten days later he developed an aortic gastric fistula, and again a new biliary leak was confirmed by transhepatic cholangiography two months postligation. This could not be repaired surgically and only could be controlled with external drainage. The bilirubin and transaminase levels showed no significant changes after ligation (Fig. 1).

Abdominal CT done at this time confirmed an infarct in the left hepatic lobe. Three months after ligation liver biopsy showed central lobular ischemic changes. He never recovered from sepsis and eventually died from *Candida* peritonitis, 3½ months after ligation.

Case 7: A 58-year-old woman with a diagnosis of non A, non B hepatitis had two previous liver transplants. The first graft had failed

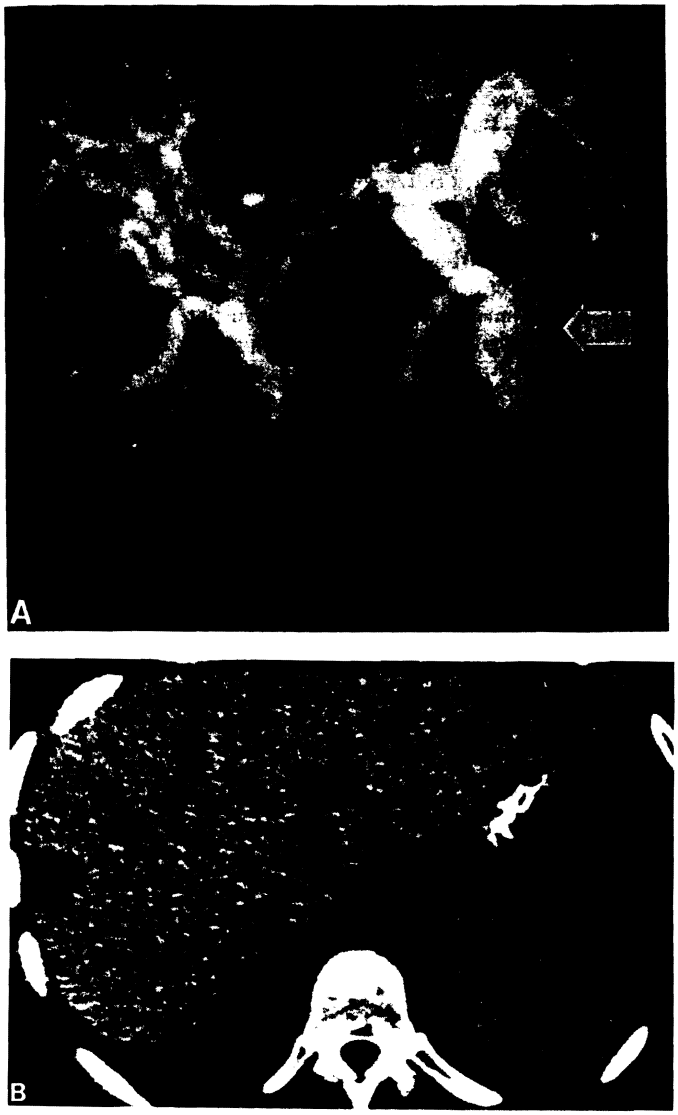

FIGURE 3. (A) Large pseudoaneurysm (arrow) at the hepatic artery anastomosis demonstrated on selective hepatic arteriogram. (B) Area of decreased attenuation (arrow) in periphery of right lobe representing a hepatic infarct demonstrated on CT scan.

as a result of thrombosis of the portal vein, and following the second graft she was discharged with normal bilirubin and transaminase levels.

Severe acute rectal bleeding prompted her readmission ten days later. The source of bleeding could not be demonstrated by endoscopy. Emergency angiography demonstrated an arterial-enteric fistula from the hepatic artery into a loop of small bowel, most likely secondary to an anastomotic pseudoaneurysm (Fig. 4). The patient was taken to the operating room where the presence of a pseudoaneurysm of the right branch of the hepatic artery was confirmed.

A biliary leak had formed an abscess posterior to the choledochojejunostomy that caused erosion into the right hepatic artery, necessitating ligation of the hepatic artery and repair of the biliary leak. Postoperatively the bilirubin and transaminase levels had an acute rise (Fig. 1).

Tissue cultures grew heavy microaerophilic streptococci in the wall of the aneurysm. Her condition was critical; a new graft became available and she received her third liver graft. The operative findings disclosed a severely mottled and ischemic liver. An aortic-iliac graft to the hepatic artery had to be placed. The allograft showed evidence of hepatic necrosis. Her condition continued to deteriorate and she succumbed to sepsis and multiorgan failure five days later.

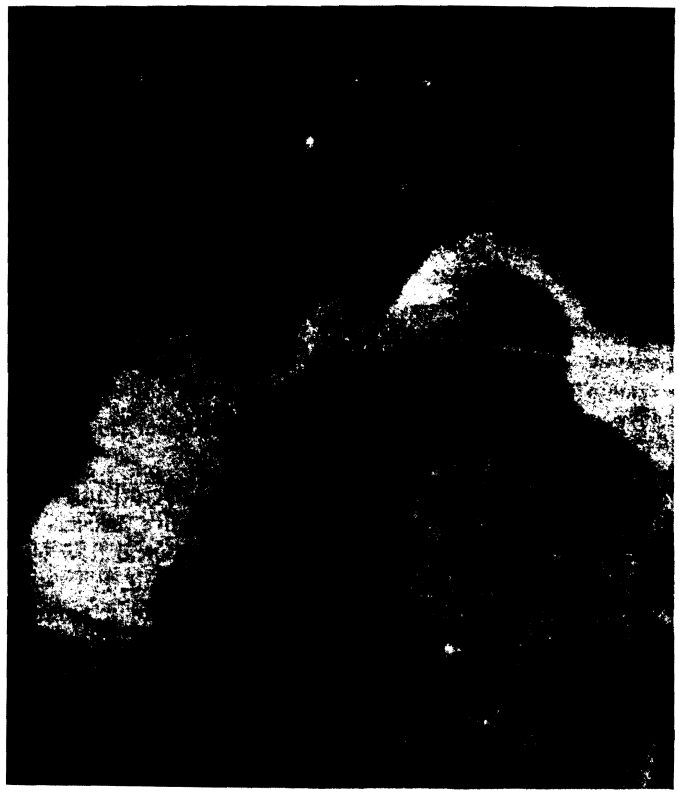

FIGURE 4. Hepatic artery–jejunal fistula demonstrated on selective hepatic arteriogram. There is massive contrast extravasation into the jejunum from the allograft hepatic arterial anastomosis. Note the markedly diminished size of the allograft intrahepatic arterial tree.

DISCUSSION

Pseudoaneurysms, although not frequent after liver transplantation, can be a devastating and often fatal complication. In our cases (Table 1), the pseudoaneurysms were associated with infection and pancreatitis (n=2), sepsis secondary to biliary leak (n=4), or a suspected infection (n=1). Even though no specific risk factors have been identified in other reports, a combination of technical imperfections and infection can be found in the majority of cases. Other possible risk factors found in this series are retransplantation (n=3) and the use of arterial grafts (n=3).

Since PAs may be asymptomatic, they are often detected on radiologic studies performed for other reasons. A high index of suspicion is required. In a previous report comparing the different diagnostic modalities, angiography was found to be the most sensitive test. In 6 patients the initial manifestation was severe bleeding. Angiography was diagnostic in 4 patients and in the others (n=3) the PAs were discovered at the time of emergency laparotomy for bleeding.

The treatment of PAs is controversial. In the absence of rupture or infection, a primary repair by resection and reanastomosis, or by reconstruction with a new aortoiliac graft has, in our experience, occasionally been possible. However direct reconstruction is not feasible in an infected field or when rupture has damaged the artery beyond repair. All the patients but one in this study were treated with excision and ligation. Figure 5 shows the alternative treatments of PAs reported by other authors (*13*, *14*).

The general outcome of our patients was somewhat unexpected. Long-term survival was achived in 4 patients. In case

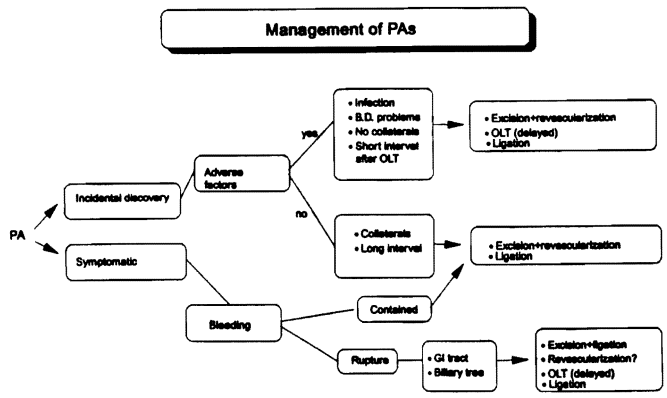

FIGURE 5. Alternative treatments for pseudoaneurysms.

3, the patient had total pancreatectomy for necrotizing pancreatitis. Her recovery was even more impressive, because in addition to having a dearterialized liver, her portal flow was deprived of the splenic and pancreatic venous input.

Only one patient developed a biliary stricture 5 months postligation, which was responsive to percutaneous balloon dilatation. Two patients died (1–3.5 months) after ligation, both from *Candida* peritonitis. The first one had a liver infarct (Fig. 3B) following embolization (case 5). The timing of occlusion also appears to be important, since in a third case (case 7), in which ligation had to be done within 10 days after OLT, acute hepatic failure followed. In the allograft massive hepatic necrosis was present. The patient succumbed to multiple organ failure 5 days after a retransplant.

The possibility of a ruptured hepatic artery pseudoaneurysm should be entertained in the presence of severe gastrointestinal tract or abdominal bleeding in the posttransplant liver patient. Prompt diagnosis by angiography (*21*) followed by embolization or ligation of the pseudoaneurysm can be life-saving.

Occlusion of the hepatic artery pseudoaneurysm will not necessarily require retransplantation in every case. Patients in which biliary leaks are the identifiable cause of the pseudoaneurysm are particularly at risk. In this series, all of the patients in this category died except one. When the presentation of the pseudoaneurysm is in the form of bleeding, without associated bile leak, excision and revascularization, embolization, or ligation of the pseudoaneurysm can be done with a very careful follow-up. When the occlusion needs to be done shortly after transplantation the risk of hepatic failure and need for retransplantation is higher.

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