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BRIEF REPORT

Gidon Almogy
Alan Bloom
Anthony Verstandig
Ahmed Eid

Hepatic artery pseudoaneurysm after liver transplantation

A result of transhepatic biliary drainage for primary sclerosing cholangitis

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G. Almogy (✉) · A. Eid
Department of Transplantation,
Hadassah-Hebrew University Hospital,
Ein-Kerem, POB 12000,
Jerusalem 91120, Israel
E-mail: galmogy@hotmail.com
Tel.: +972-2-6777111
Fax: +972-2-6417997

A. Bloom · A. Verstandig
Department of Interventional Radiology,
Hadassah-Hebrew University Hospital,
Ein-Kerem, POB 12000,
Jerusalem 91120, Israel

G. Almogy
Department of Surgery,
Hadassah-Hebrew University Hospital,
POB 12000, Jerusalem 91120, Israel

Abstract Hepatic artery pseudoaneurysm (HAP) is a rare but often life-threatening complication of liver transplantation. Treatment is usually ligation, revascularization, or retransplantation. We report a patient suffering from primary sclerosing cholangitis who required transhepatic percutaneous biliary drainage. Following orthotopic liver transplantion (OLT), he developed HAP, which was successfully embolized angiographically. We discuss the association between transhepatic biliary drainage and the development of HAP after OLT, and the possibilities for angiographic intervention in these cases.

Keywords Hepatic artery pseudoaneurysm · Liver transplantation · Primary sclerosing cholangitis · Percutaneous transhepatic biliary drainage

Introduction

Primary sclerosing cholangitis (PSC) is an idiopathic disorder characterized by inflammatory destruction of large and small bile ducts, resulting in fibrosis, strictures, and biliary stasis. It is usually an indolent, but progressive disease, characterized by remissions and exacerbations, and terminating in liver failure. Medical treatment has shown few results in preventing recurrent episodes of cholangitis and progression to liver failure. Various percutaneous and endoscopic drainage techniques have been utilized in the prevention of biliary sepsis [1, 4]. We report a patient who developed an infected hepatic artery pseudoaneurysm (HAP) secondary to percutaneous transhepatic cholangiographic drainage, which was performed prior to orthotopic liver transplantion (OLT).

Case report

A 45 year-old male patient was diagnosed with PSC 2 years prior to admission. Due to recurrent episodes of ascending cholangitis, insertion of a drain by endoscopic retrograde cholangiopancreatography (ERCP) was attempted. This failed owing to technical reasons. Therefore, a percutaneous transhepatic biliary drain was inserted. The patient was offered liver transplantation owing the increasing frequency of episodes of cholangitis.

Five days after another episode of ascending cholangitis, a compatible liver was available. The patient underwent an orthotopic liver transplantation with choledochojejunostomy. The standard immunosuppressive protocol (FK-506, and intravenous

then oral steroids) was commenced. On postoperative day 5, the patient developed a wound infection at the site of the former transhepatic drain. Cultures grew *Citrobacter freundi*, and the patient was treated with intravenous antibiotics, which obtained a good response. On postoperative day 10, cholestatic and hepatocellular enzyme levels rose sharply. A liver ultrasound (US)-duplex showed good flow through the hepatic artery, and a liver biopsy was performed. Pathology revealed acute rejection. The patient was successfully treated by pulse steroids and was discharged.

Five days after discharge (31 days postoperatively) the patient presented with melanotic stools and severe weakness at another hospital . Upon arrival in the emergency room, he was pale, his blood pressure was 80/60 mmHg, and his pulse was 120/min. He was resuscitated and taken to the operating room. Endoscopy revealed hemobilia, and a laparotomy was performed. There was a large amount of blood in the distended small bowel, and there appeared to be a pseudoaneurysm of the hepatic artery. Cultures taken from debris surrounding the hepatic artery grew *Citrobacter freundi*, identical in characteristics to the organism that grew in the infected wound. The hepatic artery was allegedly ligated and the biliary anastomosis reconstructed. The patient received a total of 24 units of blood.

He was transferred to our hospital, intubated and stable. He developed adult respiratory distress syndrome (ARDS) and severe pneumonia, but eventually recovered. Surprisingly, US-duplex of the hepatic artery revealed impaired arterial flow. Elective celiac angiography, performed to evaluate flow in the hepatic artery in view of the US report, demonstrated focal narrowing and irregularity of the hepatic artery, without clear evidence of HAP. Therefore, embolization of the artery was not performed. Seven days later, the patient became hypotensive secondary to another episode of massive bleeding. He was resuscitated and taken immediately to the angiographic suite. Selective celiac angiography revealed an 8×11 mm pseudoaneurysm of the hepatic artery and a patent portal vein with normal hepatopetal flow (Fig. 1). Using a coaxial microcatheter, the artery was successfully embolized with 8-mm platinum-tufted coils (Cook, Bloomington, Ind.). There was no further evidence of bleeding and the patient did not require a further blood transfusion. Liver enzymes remained within normal range and the patient was discharged. Ten months postembolization, the patient had slightly elevated γ-glutamyltranspeptidase (GGTP) levels but was otherwise doing well.

Discussion

Vascular complications following OLT cause significant morbidity and mortality. Hepatic artery thrombosis is most common, complicating 6–10% of transplants in the adult [6]. HAP occurs in less than 1% of patients after OLT, but is often a devastating complication. HAP is associated with infection, biliary leaks, and choledochojejunostomies [5]. Cultures of the disrupted artery are often positive for gram-negative rods, gram-positive cocci, and *Candida* spp. [3]. In this case, cultures taken from debris surrounding the hepatic artery at the time of laparotomy, and cultures from the infected wound taken 4 weeks earlier, grew identical organisms. We assume the source of infection was biliary, leading to infection at the entry port of the drain and to an infected surgical field. Although ERCP is the preferred approach to biliary drainage, it was unsuccessful in this case. Internalization of the transhepatic drain and preoperative removal possibly could have prevented this rare complication.

Therapeutic options for HAP include ligation, embolization, excision, revascularization with or without a prosthetic conduit, and retransplantion [3]. The longer the time from transplant, the lower the risk of hepatic failure following ligation [5]. However, there is little data in the literature regarding embolization of HAP following OLT. Of the three reported cases of embolization of HAP after OLT, one patient died of *Candida* sepsis, one patient died of unrelated causes, and the third patient had resection of the pseudoaneurysm following successful embolization [2, 5].

Typically in the presence of hemobilia, selective angiography does not demonstrate arterial-biliary extravasation of contrast material. A focal arterial abnormality such as HAP identifies the location of bleeding. It is therefore necessary to embolize the artery proximal and distal to this point, in order to achieve hemostasis. Because our patient already had a failed attempt at ligation, the surgical field was infected, and

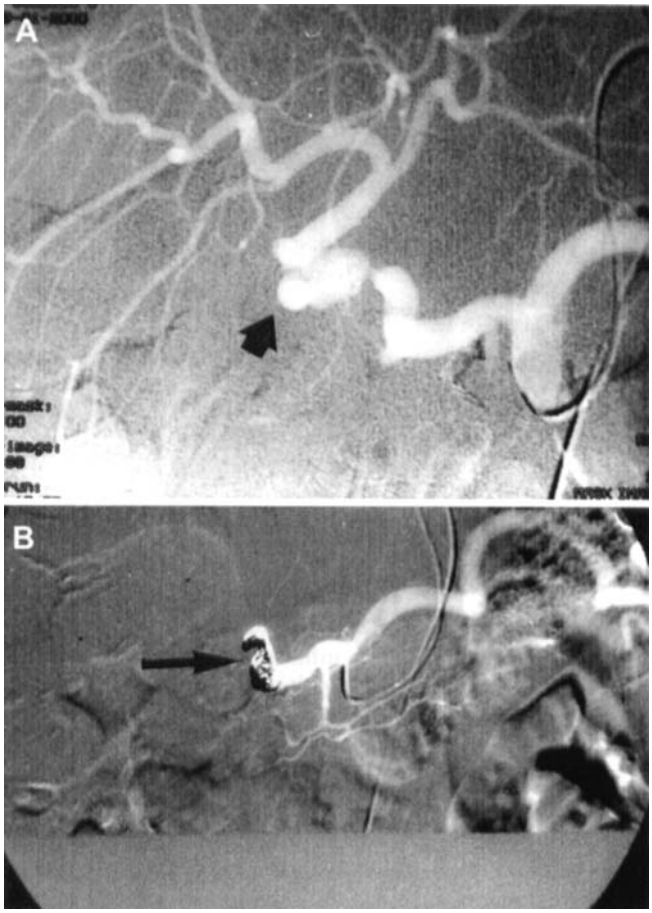

Fig. 1 **A** Celiac angiography showing hepatic artery pseudoaneurysm (*short black arrow*). **B** Postembolization films showing obstruction of flow in the hepatic artery (*long black arrow*)

the option of retransplantion in our setting was almost nonexistent, we chose to embolize the hepatic artery. Hepatic function was not compromised and the patient did not require resection of the pseudoaneurysm.

In summary, we believe every effort should be made to internalize biliary drains in patients with PSC, and perhaps even remove them prior to surgery. Embolization of the hepatic artery should be attempted providing a long enough period of time has elapsed from transplant to detection of the pseudoaneurysm. The treating physician should always be aware of the possibility of the development of biliary strictures secondary to bile duct ischemia in the future.

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