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Post-liver Transplantation Pancreatic Pseudocyst without Pancreatitis

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Abstract

Pancreatic pseudocysts rarely complicate acute pancreatitis after liver transplantation. We describe a case of post-transplant pancreatic pseudocyst occurring in the absence of pancreatitis.

A 53-year-old post-hepatitis C cirrhotic man with no history of pancreatic or biliary abnormalities underwent liver transplantation. However, at the time of surgery, a T-tube cholangiogram revealed a mild dilation of the native bile duct and, two months later, a large pancreatic pseudocyst was diagnosed and surgically resected. After two years, biochemical cholestasis developed and a liver biopsy showed biliary damage. ERCP revealed a stenosis of Vater's papilla, which was successfully treated by sphincterotomy.

Pancreatic pseudocyst may arise as a complication of OLT in the absence of postoperative acute pancreatitis or a history of chronic pancreatitis. In our patient, it is suspected that ampullary dysfunction associated with ischemic pancreatic injury and bacterial infection played a role in generating both the early pancreatic pseudocyst and the late cholestasis.

Keywords: Pancreatic pseudocyst; Liver transplantation; Cholangitis; Pancreatitis

Abbreviations

ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; ERCP: Endoscopic Retrograde Cholangio-Pancreatography; GGT: Gamma-Glutamyltranspeptidase; nv: normal values; WBC: White Blood Cells

Introduction

Pancreatic pseudocyst is a known complication of chronic and acute pancreatitis, and has been occasionally described as a complication of post-liver transplantation acute pancreatitis [1,2]. Acute pancreatitis may occur early after liver transplantation in the form of clinical pancreatitis with an incidence of 3%, or as biochemical pancreatitis (hyperamylasemia) in 13% of cases [3].

We here describe a case of pancreatic pseudocyst diagnosed two months after liver transplantation in a patient with no clinical or biochemical signs of acute pancreatitis, and who was not suffering from chronic pancreatitis. It is believed that a pre-existent subclinical Vater's papilla dysfunction was the predisposing factor leading to this uncommon complication.

Case Report

A 53-year-old Caucasian man with post-hepatitis C cirrhosis (Child-Pugh class C1O) underwent orthotopic liver transplantation in April 2012. The pre-transplant radiological evaluation did not reveal any pancreatic or biliary abnormalities other than cholelithiasis. He had a high bilirubin level (total bilirubin 5.0 mg/dl; nv <1.4: direct bilirubin 1.3 mg/dl; nv <0.4), but normal levels of gamma glutamyltranspeptidase (GGT) (34 U/L; nv 11-50) and alkaline phosphatase (ALP) (278 U/L; nv 98-279).

A whole liver transplant was performed using a standard technique. The surgical dissection of the hepatic hilum was free of complications. A right hepatic artery arising from the superior mesenteric artery was tied and arterial anastomosis was performed using the recipient's confluence between the left hepatic and gastroduodenal arteries sutured to the graft's common hepatic artery. Biliary reconstruction consisted of an end-to-end T-tube choledocho-choledochostomy.

The immunosuppressive regimen included cyclosporine and standard steroids with a 1g intraoperative bolus followed by a postoperative recycling schedule and then 20 mg/day of prednisone. The steroids were progressively tapered and finally withdrawn at the end of the third month.

Cholangiography performed on postoperative day 7 before T-tube clamping revealed mild dilation of the recipient's common bile duct, with a normal flow into the duodenum. No stenosis of the biliary anastomosis was observed. The pancreatic duct was also visible, and had a diameter of 3-4 mm for a short tract (2-3 cm) in the cephalic portion of the pancreas. On the basis of the absence of any significant clinical or biochemical signs of cholestasis, no further diagnostic procedures were undertaken. The postoperative course was generally uneventful, and the patient was discharged 11 days after surgery.

Two months after surgery (June 2012), the patient reported acute abdominal and dorsal pain. Ultrasonography revealed a large fluid collection (diameter: 9 cm) between the left hepatic lobe and the pancreas. The results of laboratory blood tests were normal, including liver enzymes, amylase and lipase, and white blood cells (WBC) count. The collection was percutaneously drained, and the subsequently positioned "pig-tail" drain led to an output of 1500 ml/day of purulent liquid during the first two days, after which it dislocated. Biochemical evaluation of the fluid revealed high concentrations of amylase (25891 U/L) and lipase (>10000 U/L), and abdominal computed tomography confirmed the diagnosis of a pancreatic pseudocyst. The day after the CT-scan, the

patient had fever and the culture of the drained fluid showed the presence of Staphylococcus Aureus, which also grew in the bile obtained from the T-tube. The pseudocyst was eventually resected and the postoperative course was uneventful. The T-tube was successfully removed in December 2012 after appropriate induction therapy against Staphylococcus aureus, which was again found in the bile culture.

In November 2012 and April 2013, there were slight increases in liver enzyme levels (aspartate aminotransferase, AST 59 U/L; nv 3-37: alanine aminotransferase, ALT 63 U/L; nv 3-40: GGT 60 U/L, ALP 319 U/L and total bilirubin 0.9 mg/di), and percutaneous liver biopsies revealed the recurrence of hepatitis C with lshak's index values of 7 and 11.

Antiviral alpha-interferon and ribavirin treatment was started in November 2013 and has led to negative serum HCV-RNA detections since March 2014, although mild alterations in hepatic enzyme levels persisted. The main side effects of alpha-interferon were general malaise and fever, and a marked reduction in the WBC count to about 2000/ul (nv 4000-10000), with a neutrophil count of about 800/uL (nv 1800-7700).

In June 2014, the patient remained negative for HCV-RNA, but his liver enzyme levels increased, particularly those concerning cholestasis indices (GGT 618 U/L, ALP 847 U/L, total bilirubin 1.5 mg/dl, AST 109 U/L, ALP 100 U/L). A further percutaneous liver biopsy did not reveal any signs of rejection or hepatitis C, but the presence of cholangiolar proliferation suggested biliary damage despite the absence of ultrasonography findings of biliary dilation. An endoscopic retrograde cholangio-pancreatography (ERCP) revealed findings of a normal hepatic biliary tree, a normally patent anastomosis site, and mild dilation of the distal common bile duct leading to delayed contrast medium drainage. However, as it also revealed a stenosis of Vater's papilla, a wide sphincterotomy was performed that normalised the flow into the duodenum. The interferon dose was first reduced and then withdrawn in November 2014. At the last follow-up visit a slow decrease in the biochemical indices of cholestasis was observed (AST 44 U/L, ALT 47 U/L, GGT 295 U/L, ALP 707 U/L, total bilirubin 1.1 mg/dl), and the WBC count was restored (5500/ul, neutrophils 2700).

Discussion

Pancreatic pseudocyst is a well-known complication of both acute and chronic pancreatitis, and has also been described after liver transplantation [1,2]. Acute post-liver transplantation pancreatitis is a rare and potentially severe complication [1,3,4]. lt has been suggested that various factors may contribute to the pathogenesis of acute post-transplant pancreatitis [1], including extensive surgical dissection around the pancreas, prolonged veno-venous by-pass time leading to ischemic damage of the pancreas [5], and hepatitis B infection [1,6]. Furthermore, some drugs may also play a role in the pathogenesis of pancreatitis, including large intraoperative doses of furosemide, steroids or calcium chloride [3].

After undergoing liver transplantation, our patient showed no signs of acute pancreatitis and hyperamylasemia was never observed. However, although the operation was uneventful and characterised by a plain surgical dissection of the hepatic pedicle, the fact that the gastroduodenal artery was divided may have led to ischemic damage of the pancreas. In relation to the other possible contributing factors of post-transplant pancreatitis described in the literature, there were no differences between this patient and the majority of our other liver transplant patients: he received the standard 1g intraoperative bolus steroid dose and underwent the standard postoperative recycling schedule postoperatively, without high doses of furosemide or calcium chloride.

It has been observed that chronic pancreatitis may cause sub-clinical common bile duct stenosis after liver transplantation in patients with alcoholic cirrhosis [7], and that such patients should therefore be carefully investigated intraoperatively in order to detect any pre-existing biliary stenosis. Our patient did not report a history of abdominal pain or alcohol abuse, and our preoperative radiological evaluations did not reveal the presence of biliary tract stenosis or dilation, pancreatic abnormalities such as chronic pancreatitis, or any pre-existent pancreatic cystic lesions. However, the biochemical diagnosis of mechanical cholestasis in cirrhotic patients may be difficult because of the limited accuracy of liver function tests: for example, our patient had high bilirubin levels but normal functional cholestasis index values.

Our surgical evaluation of the pancreas did not reveal any abnormality and the intraoperative cholangiogram only showed a mild dilation of the native common bile duct. However, the patient was affected by stones occluding Hartmann's pouch, and it is known that a functional enlargement of the common bile duct may be caused by chronic gallbladder dysfunction.

The final etiology of the cholestasis observed in our case is probably related to a dysfunction in Vater's papilla that rapidly progressed after transplantation [8]. The ERCP performed two years after transplantation showed a narrowed peri-ampullary tract and delayed contrast medium drainage from the common bile duct. The post-transplantation induction of an immunodeficient status and the presence of a T-tube inside the biliary system can lead to cholangitis [9], which may have been rapidly precipitated by the pre-existent ampullary dysfunction. The pathogenesis of the large pancreatic pseudocyst detected very early after the liver transplantation can therefore be attributed to a surgically induced ischemic pancreatic injury associated with bacterial infection and pancreatic excretion impairment. This last was probably due to a Vater's papilla dysfunction that persisted after the treatment of the pseudocyst and subsequently worsened because of recurrent subclinical biliary infections. The cholestatic clinical presentation was finally explained by the recurrent cholangitis, which is a common late post-transplant complication that frequently has some specific histological picture [10]. We believe that the cholangitis was secondary to the combination of immunosuppression and interferon-related leukopenia in the presence of impaired bile outflow due to Vater's papilla dysfunction.

In conclusion, pancreatic pseudocysts may arise as a complication of liver transplantation even in the absence of postoperative acute pancreatitis or a history of chronic pancreatitis. In our case, it is suspected that a Vater's papilla dysfunction played a role in the etiology of both the early pancreatic pseudocyst and the late cholestasis.

Authorship

FC collected data, analyzed data and wrote the paper, MN and AC collected data, GR and LC designed study and performed study

Conflict of Interest: The authors declare that no conflict of interest exists.

Compliance with Ethical Requirements

1. For Conflict of Interest statements

   Francesco Caruso declares that he has no conflict of interest.

   Marco Nencioni declares that he has no conflict of interest.

   Andrea Chierici declares that he has no conflict of interest.

   Giorgio Rossi declares that he has no conflict of interest.

   Lucio Caccamo declares that he has no conflict of interest.

2. All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008. Informed consent was obtained from all patients for being included in the study.

Additional informed consent was obtained from all patients for which identifying information is included in this article.

3. All institutional and national guidelines for the care and use of laboratory animals were followed.

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