Loss of Distal Native Bile Duct in Liver Transplant Recipient with Necrotizing Pancreatitis

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Abstract
Acute pancreatitis in acute liver failure carries significant morbidity and mortality. We review a case of severe acute pancreatitis recognized during liver transplant that evolved into necrotizing pancreatitis and subsequent loss of the native intrapancreatic common bile duct. The treatment and management of pancreatitis in liver transplant patients is poorly defined; the dissolution of native distal common bile duct has not been reported. This manuscript outlines our management and multidisciplinary approach to this complex surgical issue.

Keywords: Liver transplantation; Acute pancreatitis; Necrotizing pancreatitis

Introduction
Acute pancreatitis has been a recognized complication of acute liver failure (ALF) since the 1970s [1]. In the general population, acute pancreatitis typically has a mild, self-limited course [2]. However, liver transplant patients tend to have a higher prevalence than the general population, with reported incidence of acute pancreatitis ranging from 3% to 8% [3]. A 1998 study by Kuo et al. showed the prevalence of acute pancreatitis in ALF was 33% and occurred in the setting of severe hepatic encephalopathy, renal insufficiency, and overall physiologic dysfunction. The results indicated that acute pancreatitis in the setting of ALF was associated with dysfunction of multiple organ systems and significantly increased mortality, up to 60% [1].

In nontransplant general patients, 15% to 25% of the patients with acute pancreatitis have necrotizing pancreatitis [2]. Clinical diagnosis of pancreatitis in ALF patients is challenging, as encephalopathy can mask the symptoms of pancreatitis [4], and interpretation of raised serum amylase can be obscured by systemic inflammation and extra-pancreatic production [5]. More often the diagnosis is made postmortem because of the absence of symptoms [6].

In this report we review the case of a patient with ALF who developed necrotizing pancreatitis that was diagnosed during liver transplant and was successfully managed. The patient survived seven years and is currently alive but lost the native common bile duct from enzymatic dissolution during the necrosis of the pancreas.

Case Presentation
A 31-year old patient with a history of epilepsy and no past history of liver disease was airlifted from an outside facility to our center. Previously, the patient had been in a motor vehicle accident resulting in pelvic fractures. Consequent chronic pain required long-term use of narcotic pain medication, in addition to non-steroidal anti-inflammatories.

The patient became lethargic at home and could not be aroused. At the time of local hospital presentation the patient was determined to be in ALF and started on N-acetyl cysteine prior to being urgently airlifted to our center. Upon arrival, the patient was in deep coma with Glasgow coma score of 3; severe hepatic encephalopathy with cerebral edema was present. The brain edema was treated with external ventricular drainage (EVD) for intracranial pressure (ICP) relief; the initial opening ICP was 24 mmHg which decreased to 5 mmHg following EVD. The patient was listed for liver transplant.

During transplantation, the exploration of the abdomen showed diffuse reddish calcification deposits within the large and small intestinal mesenteries, omentum, and throughout the surface of the body of the pancreas. Ectopic calcifications and retroperitoneal hemorrhage were observed around the pancreas. There was concern for necrotizing pancreatitis. However, the pancreas was

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firm without clinical evidence of necrosis. A full consultation with our transplant critical care service, anesthesiology, and transplant hepatology teams was undertaken, and the consensus was to proceed with the lifesaving procedure of liver transplantation. The native liver was removed and the liver transplant was completed with piggyback technique, primary hepatic artery reconstruction, and duct-to-duct biliary reconstruction with a placement of transcytotic biliary tube. The patient was maintained with hypothermic protocol maintaining core body temperatures 32°C - 34°C. As the new liver was successfully implanted, the patient became hemodynamically stabilized. ICP varied between 5 mmHg to 9 mmHg during the transplant. Continuous renal replacement therapy was maintained throughout the procedure. Patient was returned to intensive care unit.

The patient had an extended post-transplant course of 104 days. ICP remained normal and EVD was removed on postoperative day (POD) 10. Postoperative complications included: necrotizing pancreatitis; renal failure; respiratory failure; steroid-induced hyperglycemia; ischemic necrosis of all fingers and toes due to prolonged use of vasopressors requiring distal phalangeal amputations; acute cellular rejection; C. difficile colitis; malnutrition and multiple interventions for pancreatic complications that are described below.

The acute pancreatitis progressed into pancreatic necrosis, acute necrotic pancreatic and peripancreatic collections, infected necrosis, and later walled-off necrosis. These infected pancreatic and peripancreatic necroses were intensely managed with interventional radiology using multiple peripancreatic drains. On POD 21, the postoperative cholangiogram showed the presence of allograft and recipient distal common bile duct. However, by POD 125, very little native distal bile duct was recognizable on MRI; the native distal bile duct was probably digested by pancreatic enzyme during the necrotic processes. The allograft bile duct remained intact but there was biliary and pancreatic leakage at the junction bile duct and pancreatic margin; these leakages were confirmed and controlled with a percutaneous transhepatic stent. The internal/external transhepatic biliary stent was extended into duodenum creating a track coursing through the necrotic pancreatic head. This biliary stent is capped and flushed daily functioning as an indwelling biliary drain in the vicinity of the transplanted allograft with the total bilirubin level 1.6 mg/dl or less (Figure 1). After nine months of intense drainages and management, the pancreatic necrotic collections resolved and all the drains were removed except for the biliary tube.

Medical comorbidities prolonging the hospital stay, aside from the aforementioned disruption of the native common bile duct and necrotizing pancreatitis, improved and were mostly resolved prior to hospital discharge. Although the patient required multiple hospital admissions for fevers, pain, and complications relating to the biliary drain in the first year post-liver transplant, admission has not been required since that time. The patient is now seven years out from liver transplant and living a relatively normal life, independently engaging in activities of daily life. The transhepatic biliary stent remains in place at the time of this publication, and the patient continues to undergo biliary tube exchange every six weeks.

Discussion

Acute pancreatitis in the setting of ALF is associated with poor prognosis [6]. In fact under severe conditions, including necrotizing pancreatitis, it is a contraindication for transplant patients. At the time of liver transplant, we believed this patient might indeed have acute pancreatitis, potentially even moderately severe. However, liver transplantation was the only hope to have a chance at reversing the multi-organ failure including brain, kidney, and lungs. The patient’s recovery after liver transplantation supports this. Although management of necrotizing pancreatitis is challenging, advances in interventional radiology and medical therapy have made it possible to control the patient’s necrotizing pancreatitis and its complications. The patient’s diabetes is also manageable, a mobile glucose monitoring system has been approved to provide continuous glucose monitoring and assist in improved diabetes control.

Literature outlining the management of post-liver transplant patients with necrotizing pancreatitis is limited. Recent studies by da Costa et al. and Kokosis et al. support a shift from open surgical procedures to minimally invasive step-up strategies [7] completed by a multidisciplinary team [8]. However, no reports were found describing the dissolution of the native distal common bile duct, presumably by digestive pancreatic enzymes, and management of such complication in liver transplant patients.

The patient has chosen to continue conservative tube management instead of other options eliminating the internal/external tube. One possibility is to place a covered biliary stent extending from the transplant common duct to the duodenum and endoscopic replacements as needed, and another is a laparotomy and Roux-en-Y choledochojunostomy. Liver MRI done five years post-liver transplant, and after multiple interval biliary tube exchanges, showed parenchymal loss and peripheral duct dilatation in the vicinity, likely due to ductal exclusion caused by the drain. There was little discernible pancreatic tissue seen, except in the head of the pancreas. Magnetic resonance elastography values were consistent with stage 2-3 liver fibrosis. Roux-en-Y choledochojunostomy becomes an important consideration if the chronically indwelling drain is contributing to the liver fibrosis.

References


