Left Hepatic Artery Pseudoaneurysm Caused by Acute Pancreatitis

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Introduction

Hepatic artery pseudoaneurysm (HAP) is a rare complication of acute or chronic surgical injury to the hepatic artery. Sethi et al. [1] reported that 19% of pseudoaneurysms due to pancreatitis occur in the hepatic artery. The HAP usually occurs in the common or proper hepatic arteries rather than the peripheral arterial branches. As a result, most HAPs occur in the extrahepatic region [1, 2]. While there are some data regarding the prevalence and treatment of pseudoaneurysms in chronic pancreatitis, very little has been reported in acute pancreatitis [3, 4]. Right HAP caused by acute pancreatitis has been reported sporadically [4, 5], but left HAP caused by acute pancreatitis has not been reported. Hence, we hereby report the successful treatment of a left HAP with coil embolization.

Keywords
Pseudoaneurysm · Left hepatic artery · Acute pancreatitis · Endoscopic papillectomy

Abstract
Objective: The aim of this work was to report a case of left hepatic artery pseudoaneurysm due to acute pancreatitis following endoscopic papillectomy. Clinical Presentation and Intervention: A 74-year-old female with an ampullary adenoma underwent papillectomy, which was complicated by acute pancreatitis. Computed tomography showed aneurysmal dilatation of the proximal left hepatic artery. An angiography with coil embolization was performed and was successful. The patient was doing well at the 1-year follow-up. Conclusion: This patient with left hepatic artery pseudoaneurysm following severe acute pancreatitis was successfully treated with coil embolization.

Significance of the Study
• This rare case of left hepatic artery pseudoaneurysm, which occurred following severe acute pancreatitis, was successfully treated with coil embolization.
• Awareness of this clinical manifestation could be important for early diagnosis and treatment.
Case Report

A 74-year-old female with a history of hypertension was admitted for an endoscopic papillectomy to remove an ampullary adenoma with low-grade dysplasia which was proven by previous biopsy. Her vital signs were stable and a physical examination revealed unremarkable findings. An abdominal computed tomography (CT) scan upon admission day showed unremarkable findings (Fig. 1a). The blood test results upon admission were white blood cell count (WBC) 5,640/μL, hemoglobin 13.2 g/dL, and platelet count 299,000/μL. Liver function tests were all within normal limits (aspartate aminotransferase 40 IU/L, alanine aminotransferase 36 IU/L) and so was the normal serum total bilirubin level (0.4 mg/dL). Serology was negative for hepatitis B and hepatitis C, and serum amylase and lipase, the renal function test, and clotting profiles were also within normal limits. C-reactive protein was 0.07 mg/dL and the serum level of CA 19-9 was below 2 U/mL (normal 0–37 U/mL). We performed endoscopic papillectomy on the day after admission, with endoscopic retrograde cholangiopancreatography (ERCP) showing a slight bulging of the ampulla of Vater and normal bile duct figures. After performing a papillectomy, the pancreatic duct was accessed and the injection of contrast showed good drainage, based on which we did not insert a pancreatic duct stent. During the ERCP, we did not use a guidewire or catheter that might injure the left hepatic artery. On the day following the endoscopic papillectomy, epigastric and periumbilical abdominal pain developed and WBC (10,960/μL) and serum amylase (1,868 IU/L) and lipase (3,775 U/L; normal 0–60) were increased. The patient was diagnosed with acute pancreatitis on the basis of her clinical findings and laboratory data. An abdominal CT scan 3 days after the papillectomy revealed acute pancreatitis with peripancreatic fat infiltration and fluid collections. The symptoms of acute pancreatitis and elevated WBC and serum amylase and lipase lasted for several days. The abdominal CT scan was checked 9 days after the papillectomy, revealing severe acute pancreatitis with necrosis and fat infiltration and a newly developed aneurysmal dilatation (1.5 × 1.0 cm) of the proximal portion of the left hepatic artery in the fissure for ligamentum venosum (Fig. 1b). On day 6 after the detection of the aneurysm by abdominal CT scan (15 days after the endoscopic papillectomy), we performed angiography and embolization using a sandwich method with microcoils for a fusiform aneurysm on the left hepatic artery (Fig. 2). After embolization, the abdominal CT scan showed well-embolization state of aneurysmal dilatation of the left hepatic artery. One week later, embolization, serum amylase and lipase levels were further decreased, and almost all of the subjective symptoms had subsided. The patient received continued supportive care for acute pancreatitis and was discharged on the 39th hospital day. She had an uneventful course during 1 year of follow-up.

Discussion

This case of HAP was a result of acute pancreatitis that occurred following endoscopic papillectomy and involved an intrahepatic branch of the left hepatic artery, which is an unusual site. Embolization with angi-
ography was successfully performed just after the detection of the HAP.

HAP rupture is common and occurs in up to 76% [6]. Thus, early diagnosis and treatment are considered to be essential. Intrahepatic HAPs account for only about 20% of all HAPs and most of them arise from a complication of percutaneous procedures such as transhepatic cholangiography, transhepatic drainage catheter placement, or liver biopsy [7]. Therefore, acute pancreatitis could be considered a rare cause of intrahepatic pseudoaneurysm. According to the analysis of 7 cases of HAP reported by Finley et al. [5], 4 developed in the common hepatic artery in the extrahepatic region, 1 was in the right hepatic artery in the extrahepatic region, and 2 cases were developed in the intrahepatic branch of right hepatic artery. A recent report of HAP by Kazue et al. [4] also reported a pseudoaneurysm that involved the right hepatic artery as a complication of acute pancreatitis. Our case of HAP was a result of acute pancreatitis, but it occurred in the early phase following papillectomy, which is very rare, and the involvement of an intrahepatic branch of the left hepatic artery is also unusual. The most sensitive test for detecting HAP is selective angiography. A pseudoaneurysm usually develops 3–5 weeks after the onset of acute pancreatitis, but pseudoaneurysm hemorrhage may occur from a few days to several years after the onset of pancreatitis [1, 6]. In our patient, the pseudoaneurysm of the left hepatic artery may have occurred during the early phase of inflammation and was detected only 9 days after the onset of acute pancreatitis. Pancreatic duct stents and/or postprocedure rectal nonsteroidal anti-inflammatory drug suppositories should be utilized to lower the risk of severe post-ERCP pancreatitis (PEP) in high-risk patients [8]. Some meta-analyses report that placement of prophylactic pancreatic stents may lower the overall incidence of PEP in high-risk patients, but not the incidence of severe PEP [9]. Furthermore, some debate still exists regarding the use of indomethacin [10]. In this case, we did not use rectal indomethacin and a pancreatic duct stent, which might not alter the process of severe inflammation in acute pancreatitis. It is our opinion that severe acute pancreatitis could be a rapid development of pseudoaneurysm at an unusual remote site, such as the left intrahepatic region. For the management of an arterial pseudoaneurysm arising in the setting of pancreatitis, coil embolization should be initially attempted if the patient is hemodynamically stable [2].

Conclusion

This patient with a left HAP occurring following severe acute pancreatitis was successfully treated with a coil embolization.

References