Bile Peritonitis due to Spontaneous Perforation of the Left Hepatic Duct: A case report

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Abstract

This case report concerns a patient with bile peritonitis due to spontaneous perforation of the intrahepatic bile duct. A 67-year-old woman underwent an emergency laparotomy for acute abdomen with a tentative diagnosis of acute cholangitis with a calculus in the common bile duct. Intraoperatively, however, bile peritonitis due to perforation of the peripheral left hepatic duct was found. After cholecystectomy and common bile duct exploration, intraoperative cholangiography was performed, and the perforation site was suture ligated. She was discharged from the surgical service 31 days after surgery with complete recovery.

Key words Bile peritonitis, Spontaneous perforation, Intrahepatic bile duct

Introduction

Spontaneous perforation of the intrahepatic bile duct is an extremely rare event in adults. This rare form of bile peritonitis results in a 30 to 50% mortality in spite of adequate surgical therapy and postoperative intensive care.1 We report on a patient with bile peritonitis due to spontaneous perforation of the left hepatic duct and review the literature.

Case Report

A 67-year-old female, who had undergone ventriculo-peritoneal shunt operation 3 years previously, complained of abdominal discomfort with nausea and two episodes of nonbilious vomiting. She had neither prior episodes of abdominal pain nor history of trauma. She was initially admitted to the department of internal medicine in our hospital with a clinical diagnosis of entero-colitis and was given intravenous antibiotics. On the third day of admission, she started to complain of diffuse abdominal pain. She underwent computed tomography (CT) and ultrasonography (US), which was compatible with cholecystitis and choledocholithiasis.

On referral to our surgical service, her vital signs were as follows: body temperature 36.9°C, blood pressure 147/90 mmHg, heart rate 76/min. Physical examination revealed a thin female in severe distress due to abdominal pain. Her bowel sounds were diminished, and diffuse abdominal tenderness was noted with maximum rebound tenderness in the upper abdomen. A knock pain in the right upper quadrant of the abdomen was significant and Murphy’s sign was positive. Significant laboratory data were as follows: white blood cell count 15,200/μL, hemoglobin 12.9 g/dL, platelets 199,000/μL, total bilirubin 2.0 mg/dL, direct bilirubin 1.5 mg/dL, alkaline phosphatase 1,105 units/L, serum amylase 37 units/L. Chest radiograph revealed no free intraperitoneal air...
and no acute pulmonary process. No dilated bowel loops were seen in a flat abdominal film. An upright abdominal film demonstrated a non-specific bowel gas pattern without air-fluid levels in the intestines. The abdominal US showed cholecystitis with a small gallstone and sludge associated with wall thickening of the gallbladder (Fig. 1a). The US also identified the dilated intrahepatic bile duct (Fig. 1b). The CT of the abdomen and pelvis confirmed the gallbladder wall thickening (Fig. 2a). A calculus in the common bile duct and moderate ascites were also noted (Fig. 2b). No free air was identified in accordance with the chest radiograph. The preoperative diagnosis was acute abdomen possibly due to gangrenous cholecystitis with a concomitant choledocholithiasis.

At operation, the gallbladder was edematous and severely inflamed, and bilious peritoneal fluid and diffuse fibro-purulent exudates covered the visceral peritoneum. In addition, dark-colored ascites was found in the left subhepatic area and the left subphrenic fossa. We therefore performed full abdominal exploration (Fig. 3a). The left lobe of the liver revealed a minor bile leak emanating from the peripheral biliary tree (Fig. 3b). Cholecystectomy was performed, and an intraoperative cholangiogram through the cystic duct...
duct stump showed the presence of a perforation in the anterior aspect of the lateral segment of the liver indicated by the leakage of the contrast material from the perforation site. The cholangiogram also identified a stone incarcerated in the common bile duct. A 3 mm plastic tube was successfully inserted through the perforated hole, and we confirmed that a perforation occurred at a terminal branch of the left hepatic duct by the tube cholangiography (Fig. 4). The perforation site was suture-obliterated with interrupted 4-0 Vicryl, which was followed by choledocholithotomy and T-tube drainage.

The postoperative course was uneventful. No retained stone was found on a T-tube cholangiogram at 3 weeks after surgery, when T-tube was removed. The patient was discharged 31 days after surgery. She was well with regard to the hepatobiliary system at 6 months postoperatively, and she remains well except for a fracture of the left hip by trauma.

**Discussion**

Rupture of the hepatic duct in the absence of operative injury or severe trauma is an extremely rare cause of bile peritonitis in adults. Since the first description by Freeland in 1882, 22 cases of spontaneous perforation of the intrahepatic bile duct have been reported in the English literatures. McWilliams, in 1912, reviewed 108 cases of bile peritonitis in which hepatic duct perforation accounted for only one case. Perforation in the hepatic duct was also the rarest cause of bile peritonitis in a review by Nomura et al.

Several possible mechanisms have been advocated for spontaneous perforation of the biliary system, which include: (a) increased intraductal pressure due to either mechanical blockade by stones or reflex spasm of the sphincter of Oddi, or both; (b) intramural infection which weakens the duct wall and lowers its resistance to intraductal pressure increase; (c) thrombosis of a mural ves-
sel leading to necrosis of the affected part of the bile duct wall; (d) reflux pancreatic secretions resulting in autodigestion; (e) diverticulum. In the present case, the putative cause seems to be incarceration of a calculus in the distal common bile duct which increased intraductal pressure and resulted in the perforation of the periphery of the intrahepatic bile duct. Other reasons, such as acute infection, might have contributed to the development of the perforation.

US is a noninvasive and rapid examination which is recommended as a first choice to evaluate abnormalities in the biliary system. CT should also be performed to detect calculi in the common bile duct or abnormal fluid collection. In our case, US showed severe cholecystitis and a mildly dilated intrahepatic bile duct, and CT revealed cholecystitis and ascites also, which led us to a diagnosis of peritonitis due to severe acute cholangitis and cholecystitis. Retrospectively, we might have been able to suspect bile peritonitis due to perforation in the biliary system based on the findings of dilated intrahepatic bile duct and fluid collection in the abdominal cavity.

In the present case, intraoperative cholangiography was a very useful diagnostic technique to identify the site of perforation. Nobusawa et al. reported the usefulness of intraoperative cholangiography using indigo carmine. Fortunately, the perforation site in our case could be detected easily by retrograde cholangiography through the perforated hole in an aberrant hepatic duct which is located at the end of the left hepatic triangular ligament.

References