A case of severe biliary stenosis after cholecystectomy and hepatoduodenal ligament lymph node dissection for early gallbladder cancer

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Abstract

We report a patient who developed severe biliary stenosis after undergoing cholecystectomy and hepatoduodenal ligament lymph node dissection for early gallbladder cancer. A 43-year-old man underwent cholecystectomy for gallbladder cancer, developed postoperative biliary stenosis, and again underwent surgery involving bile duct resection. The pathological diagnosis for the bile duct wall was a fibrous scar with no evidence of malignancy; therefore, the biliary stenosis was presumably secondary to disruption of the bile duct blood supply caused by lymph node dissection.

Key Words: biliary stenosis, cholecystectomy, lymph node dissection, gallbladder cancer

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Introduction

Biliary stenosis is divided into two broad categories, benign and malignant1). Benign biliary stenosis is often associated with surgical procedures, particularly cholecystectomy1, 2). We report a patient who developed severe biliary stenosis after undergoing cholecystectomy and hepatoduodenal ligament lymph node dissection for gallbladder cancer, and discuss the causes and preventive measures for biliary stenosis.

Case Report

A 43-year-old man with no medical or family history of note was found to have a gallbladder polyp on abdominal ultrasound during a medical checkup, and was admitted to our hospital for treatment. Abdominal ultrasound showed a 21-mm, elevated lesion with an irregular border in the gallbladder neck. Open cholecystectomy was performed with the suspicion of gallbladder cancer. Since the resected specimen was diagnosed as primary gallbladder cancer on intraoperative frozen-section examination, the operation was completed with dissection of the lymph nodes in the hepatoduodenal ligament and on the posterior surface of the pancreatic head. The final pathological diagnosis was tubular adenocarcinoma, T1, N1, Stage I. His postoperative course was uneventful, and he was discharged on the eighth postoperative day with symptomatic improvement.

On the 19th postoperative day, the bulbar conjunctivae were stained yellow, and he was re-admitted to our hospital. On admission, his consciousness was clear, blood pressure was 118/62 mmHg, pulse was 84 beats/min and regular, the palpebral conjunctiva was not anemic, and bulbar conjunctiva was icteric. The abdomen was flat, and the liver was not palpable.

The laboratory data on admission were as follows: T-Bil, 11.1 mg/dL; D-Bil, 7.0 mg/dL; GOT, 255 IU/L; GPT, 945 IU/L; γ-GTP, 769 IU/L; LAP, 381 IU/L; and ALP, 1,387 IU/L (indicating an abnormal liver function and elevated biliary enzyme levels).

Abdominal CT showed mild intrahepatic bile duct dilatation. ERCP revealed intrahepatic bile duct dilatation and a circumferential, irregular stenosis of the bile duct over a length of 4 cm (Fig. 1a). On PTC, the intrahepatic bile ducts were dilated, the bile duct from the bifurcation of the left and right hepatic ducts to the upper border of the pancreas was markedly stenosed, and so a drain was placed for jaundice reduction (Fig. 1b). The cytology of bile collected from a PTCD tube and brushing cytology of the bile duct were class I, indicating no malignancy. Since the bile duct stenosis did not improve during a one-month follow-up under PTCD placement, re-surgery was performed. At the time of the operation, the bile duct wall from the porta hepatis to the upper border of the pancreas was markedly thickened and surrounded by fibrous tissue. These findings led to a diagnosis of biliary
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stenosis due to inflammatory cicatricial contraction. The left and right intrahepatic bile ducts were transected at the porta hepatis, and the bile duct down to the upper border of the pancreas was resected along with the surrounding scar tissue. Hilar bile duct-jejunum Roux-en-Y anastomosis was performed for biliary reconstruction, and the surgery was completed by placing two drains in the left and right bile ducts for biliary decompression.

Histopathological examination of the bile duct showed marked fibrous scarring of the bile duct wall with no evidence of malignancy, leading to a diagnosis of benign biliary stenosis due to hepatoduodenal ligament lymph node dissection (Fig. 2).

His postoperative course was uneventful, and he was discharged on the 21st postoperative day, with biliary drains in place. The drains were removed in the outpatient clinic on the 56th postoperative day. At present, about 3 years after surgery, he is doing well and has not developed biliary stenosis.

Fig. 1 (a) ERCP revealed intrahepatic bile duct dilatation and a circumferential, irregular stenosis of the bile duct over a length of 4 cm.
(b) On PTC, the intrahepatic bile ducts were dilated, the bile duct from the bifurcation of the left and right hepatic ducts to the upper border of the pancreas was markedly stenosed, and so a drain was placed for jaundice reduction.

Fig. 2 Histopathological examination of the bile duct showed marked fibrous scarring of the bile duct wall with no evidence of malignancy, leading to a diagnosis of benign biliary stenosis due to hepatoduodenal ligament lymph node dissection.
Discussion

Researchers have reported that the most common causes of benign biliary stenosis are inflammatory disease and postoperative scarring of the bile duct. The possible causes of postoperative biliary stenosis include bile duct injury, bile duct wall ischemia, and suppurative biliary inflammation. We speculate that, in the present patient, the frequent use of the electric knife for hemostasis and use of clips for lymphorrhea prevention in the lymph node dissection for gallbladder cancer blocked blood flow in the bile duct wall, resulting in marked biliary stenosis due to cicatricial contraction. The biliary tract is supplied by the proper hepatic artery, left and right hepatic arteries, cystic artery, and anterior and posterior superior pancreaticoduodenal arteries, and the hepatic ducts and upper common bile duct are supplied by the posterior superior pancreaticoduodenal artery, left and right hepatic arteries, and proper hepatic artery (Fig. 3). These arteries are distributed to the bile duct wall through the epicholedochal plexus, which forms a reticular network. In particular, the posterior superior pancreaticoduodenal artery sends two branches, the marginal and paracholedochal arteries, accounting for 60% of the blood supply to the common bile duct at the level of the upper duodenum. Injury to these arteries is prone to cause ischemia and stenosis of the common bile duct.

When performing hepatoduodenal ligament lymph node dissection, it is important to preserve the epicholedochal plexus. Specifically, it is vitally important to perform patient-friendly surgical procedures: (1) try to perform careful hemostasis and avoid the excessive use of electrocoagulation hemostasis, and (2) try not to choose sharp-pointed surgical instruments (tweezers, scissors, etc.).

References