ABSTRACT

Spontaneous extrahepatic bile duct perforation (SEBDP) is generally seen in infants. Although rarely seen in adults, it may be seen with fatal bile peritonitis. Therefore, for a patient presenting with acute abdominal symptoms, differential diagnosis must be made with radiological imaging such as abdominal ultrasonography or computed tomography, without any loss of time. In these imaging tests, in cases of gallstone disease together with perihepatic free fluid or choledocus which can not be monitored, it should be considered in the differential diagnosis. An emergency surgical intervention should be planned to avoid serious complications. The aim of this paper was to present the rare cause of acute abdomen which developed associated with spontaneous common hepatic canal perforation in an adult.

Keywords: Acute abdomen; common hepatic duct; spontaneous perforation; surgery.

INTRODUCTION

Spontaneous extrahepatic bile duct perforation (SEBDP) is an extremely rarely seen cause of acute abdomen in adults.[1] Preoperative diagnosis is very difficult, and diagnosis is often made during surgery.[2] However, with the use of abdominal ultrasonography (US) together with multidetector computed tomography (CT), preoperative diagnosis can be made. Although the most common location is the choledocus, hepatic canal damage may also be seen.[3] In cases of gallstone disease, simple peritoneal drainage, cholecystectomy and T-tube drainage are effective and safe treatment choices.[2] In these patients, fatal complications can be avoided with early diagnosis and treatment.

CASE REPORT

A 36-year-old female had been admitted to our clinic 15 days previously with a diagnosis of biliary oedematous pancreatitis (Fig. 1a), and with clinical recovery after medical treatment, she was discharged after 8 days. She presented again with complaints of sudden onset back and abdominal pain, nausea and bilious vomiting. On physical examination, the abdomen was distended with widespread sensitivity, defence and rebound. Murphy’s sign was positive. Intestinal sounds were reduced with auscultation. Vital signs included body temperature of 38.5°C, pulse of 110/minute, and an arterial blood pressure of 100/60 mmHg. Laboratory test results were determined high with leukocytes 16x10³ /µL (5.2–12.4), CRP 132 mg/l (0–10), amylase 230 U/L (28–100), lipase 205 U/L (21–67), alkaline phosphatase 674 U/L (30–120), LDH 268 U/L (0–247), total bilirubin 1.38 mg/dL (0.3–1.2) and direct bilirubin 0.51 mg/dL (0–0.2). On abdominal US, intrahepatic bile ducts were evident in the centre, the gallbladder wall thickness had increased. The bile duct perforation was thought to have occurred as the choledocus could not be monitored because of the adjacent fluid. In this patient who had previously experienced pancreatitis, an area was determined within

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The patient was admitted for surgery with an initial diagnosis of bile duct perforation. In the exploration, there was widespread bile within the abdomen. Following dissection of adhesions in the subhepatic area, it was seen that the hydropic and wall thickness of the gallbladder had increased. Millimetric stones were palpated in the gallbladder neck. In the continuation of the exploration, a 2 cm ischaemic area was determined in the anterior of the common hepatic canal and it was here that the perforation had occurred (Fig. 1c). There was slight oedema of the head of the pancreas, but no haemorrhagic or necrotising appearance in the pancreas. After cleaning within the abdomen, and cholecystectomy, the distal choledocus and proximal bile ducts were checked with choledocus coils. As no additional pathology, such as choledocus stone, papillary stenosis or choledocal cyst was determined, a T-tube drain was placed from the perforation area to the common hepatic canal and the operation was terminated. On the postoperative twenty-first day, the passage to the duodenum was seen to be normal on cholangiography applied from the T-tube, and the T-tube was removed. Pathological examination of cholecystectomy determined ulcerous cholesistitis and cholelithiasis. During a one-year follow-up period, no complications were seen.

DISCUSSION

In the majority of SEBDP cases, perforation location is determined at the conjunction of the cystic canal with choledocus. Very few cases have been reported in the common hepatic canal, especially in adults. As it is rarely seen, prevalence, morbidity and mortality rates are not certain. However, when diagnosis is delayed, fatal bile peritonitis may develop. While 80% of cases with SEBDP are seen to be clinically subacute, acute abdominal findings occur in the remainder. The case presented here had findings of acute abdomen.

In adults, the etiological factors held responsible are viral infections, stone and/or erosion caused by a stone, biliary sludge, congenital weakness of the wall because of a connective tissue defect, bile canal ischaemia, intramural infection, cyst or diverticulum and pancreas fluid reflux. In the current case, there were many millimetric stones within the gallbladder lumen. However, they were not of a size to create pressure on the extrahepatic bile ducts. In the extrahepatic bile ducts, no gallstones, sludge or sphincter which would increase pressure in the lumen were determined. However, the wall of the common hepatic canal was extremely thin and weak in structure. As the patient had recently had pancreatitis, the etiological reasons could be considered to be pancreatic fluid reflux, wall weakness and stones in the gallbladder lumen determined in the operation and necrosis of the common hepatic canal.

Advanced methods such as scintigraphy and choledoscopy can be useful in preoperative diagnosis but are difficult to apply under emergency conditions. Even though it is said in the literature that generally radiological imaging methods such as abdominal US and CT are insufficient for preoperative diagnosis, after careful examination of these tests, bile duct perforation can be determined. Therefore, in patients presenting with acute abdomen, SEBDP should be considered in the differential diagnosis in cases of gallstone disease determined together with perihepatic free fluid or peritonitis or when the choledocus can not be monitored.

In the treatment of SEBDP, there are choices such as symptomatic, medical, interventional, and surgical methods. From surgical treatment choices, the most frequently recommended ones are simple peritoneal drainage when it is seen that there is no obstruction in the distal, and T-tube drainage applied to the perforation area together with cholecystectomy. The T-tube should be removed after remaining in place for free drainage for up to two-three weeks. In the case presented here, no problems were experienced during drainage or after removing the T-tube.

Possible complications of SEBDP are cholangitis, portal vein thrombosis and bile leakage. However, with early diagnosis and appropriate surgical intervention as in the current case, the frequency of complications is reduced.
In conclusion, in a patient with a recent history of pancreatoc-lobiliary pathology presenting with acute abdomen, abdomi- nal US and multidetector CT must be taken without losing any time. In cases of gallstone pathology and determination of pericholecystic, perihepatic free fluid and when the cho- ledocus can not be monitored on these imaging tests, extrahepatic bile duct perforation should be considered in the differential diagnosis. Emergency surgery must be planned in the shortest time possible to reduce and protect against complications.

Conflict of interest: None declared.

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Nadir bir akut karın nedeni: Spontan ortak hepatik kanal perforasyonu

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