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• *CASE REPORTS* •

Bile peritonitis due to intra-hepatic bile duct rupture

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Abstract

Generalized biliary peritonitis is a serious intra-abdominal emergency. Most of them occur due to duodenal ulcer perforation and rapidly evolve into bacterial peritonitis due to contamination by gut organisms and food. In this situation, recognition of the pathology and its treatment is straightforward and is usually associated with a good outcome. There are a few unusual causes of biliary peritonitis, of which rupture of the biliary tree is one. We describe a rare case of biliary peritonitis due to rupture of an intra-hepatic biliary radical. Unusual causes of peritonitis do interrupt our daily routine emergency surgical experience. Rapid recognition of the presence of peritonitis, adequate resuscitation, recognition of operative findings, establishment of biliary anatomy, and performance of a meticulous surgical procedure resulted in a good outcome.

Key words: Biliary peritonitis; Intra-hepatic biliary radicle; Rupture

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INTRODUCTION

Rupture of an intra-hepatic biliary duct leading to biliary peritonitis is a rare occurrence, with only few cases reported in the literature[1-4]. This case report reinforces the necessity of complete and meticulous operative assessment of the biliary system in every case of bile peritonitis.

CASE REPORT

A 78-year-old male with severe spondyloarthropathy presented with a 12-h history of upper abdominal pain, nausea and vomiting. He was shocked (BP 80/40, pulse 120/min) and clinical examination revealed guarding, tenderness with rebound in the upper abdomen and absence of bowel sounds. A diagnosis of peritonitis was made and he was appropriately resuscitated. He responded well to the treatment and became hemodynamically stable. His hematological blood profile as well as his urea and electrolytes were unremarkable. He had conjugated hyperbilirubinemia with raised alkaline phosphatase and alanine transaminase. Plain radiology (erect CXR and abdominal films) did not demonstrate any pneumoperitoneum.

When the patient was optimized, he underwent surgery. At laparotomy, there was no evidence of gastrointestinal perforation; however, free intra-peritoneal bile was found. The extra hepatic biliary system was grossly dilated due to obvious obstruction at the distal common bile duct by an impacted calculus. The gall bladder contained calculi and was thick walled. There was a perforated superficial biliary radicle in the left lobe of the liver, which was the source of the free bile in the peritoneal cavity.

A cholecystectomy was performed and the CBD was explored. The calculus in the distal CBD needed to be extracted through a trans-duodenal sphincterotomy. Post exploratory choledochoscopy and on-table cholangiography confirmed clearance of the bile ducts. The common bile duct was drained with a T-tube and at the end of the operation the ruptured biliary radicle stopped leaking bile. It was however reinforced with two interrupted 3/0 prolene sutures. The right sub-hepatic space was drained and the abdomen was closed as per standard.

The patient was transferred to the high dependency unit for immediate care. He made a slow, but steady post-operative recovery. T-tube cholangiogram performed on day 14 (post-op) confirmed free passage of contrast into the duodenum and absence of residual CBD stones; thus allowing its removal. The patient was discharged 34 days following surgery and he is still healthy with normal liver function tests when last reviewed 2 years following surgery.

DISCUSSION

Peritonitis requiring surgical intervention is caused by perforated peptic ulcer in about 40% cases (duodenum: gastric: 3:1), appendicitis in 20%, gangrene of the small bowel or gall bladder in 15%, post-operative complications in 10% and miscellaneous causes in 15% cases[5]. Most commonly, peritonitis in the clinical setting is due to microorganisms, though the initial insult is usually chemical as in peptic ulcer perforation where bile, pancreatic

enzymes, blood, etc., gain access into the peritoneal cavity.

The peritoneum can be contaminated with bile through a number of routes. The commonest is post-cholecystectomy. This is usually due to the division of small bile channels between the gall bladder and liver, imperfect clipping of the cystic duct, residual CBD stones causing raised intrabiliary pressure and inadvertent division of an accessory hepatic duct. The latter is potentially serious usually requiring biliary reconstruction. Other causes include post liver transplant biliary peritonitis, spontaneous hepatic rupture in pregnancy and trauma to the extra-hepatic biliary system such as that following minimal access renal surgery[6].

Perforation of the biliary tract secondary to rupture of the gall bladder (empyema/gangrene) is well documented. However, spontaneous rupture of the CBD is exceedingly rare and here the etiologies are increased intra-ductal pressure, calculus erosion and necrosis of the duct wall secondary to thrombosis[7]. Spontaneous perforation of extra hepatic ducts is also a very rare cause of jaundice in infancy[8]. The commonest site is the confluence of the cystic and common hepatic ducts[9]. Biliary peritonitis secondary to intra-hepatic duct rupture is rarely reported in the literature; the causes are calculus disease of the biliary ducts (as in this case), stenosis of the papilla and Caroli's disease[10].

The clinical picture associated with biliary peritonitis varies and the correct pre-operative diagnosis is difficult. This combined with the associated comorbidity of the patient population (mainly elderly) significantly contributes to a mortality rate of 30–50%[11]. Though the initial insult by bile is chemical, secondary bacterial infection is the usual sequelae. Furthermore, it has been clearly shown that, in the presence of bacteria, bile further impairs local host defense mechanism through its detergent lytic effects[12]. Paralytic ileus is also a frequent complication. Laboratory findings are usually non-contributory but biliary peritonitis should always be suspected in any patient with unexplained abdominal symptoms.

The aim of treatment is to prevent sepsis in the abdominal cavity and thus prompt recognition of the condition and control of source of the contamination with appropriate drainage/reconstruction of the biliary system is of paramount importance. The type of surgery is dependent upon the general condition of the patient as well as on biliary anatomy. Regardless, biliary peritonitis requires some form of drainage, either externally via the percutaneous route or internally via the endoscopic/open surgery route. In our patient, the intra-hepatic duct rupture was presumably due to a very high pressure in the biliary system secondary to calculus obstruction in the distal common bile duct. Transduodenal sphincterotomy and extraction of the impacted calculus along with 'T' tube drainage resulted in an uneventful resolution. Prompt recognition of this condition before biliary/systemic sepsis supervened played a major role in the positive outcome for this patient. In conclusion, prompt laparotomy in a well-resuscitated patient and an adequately tailored operation depending on the operative findings are the mainstay to avoid local and systemic sepsis and long-term morbidity in these cases of peritonitis.

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