Spontaneous Common Bile Duct Perforation In Adult: A Case Report

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Citation

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Abstract

A 35 year old male was admitted to the orthopedics department and operated for ununited fracture shaft of the left femur. On the 3rd post operative day, the patient developed pain abdomen which did not improved on conservative management. On the 14th post operative day, features of frank peritonitis appeared with acute renal failure. The patient underwent explorative laparotomy. Intro-operatively common bile duct (CBD) perforation was found. Thorough peritoneal lavage was done and aT-tube was inserted within the CBD and the abdomen closed with one sub-hepatic drain. Post-operative recovery was uneventful. The patient was discharged in satisfactory condition.

INTRODUCTION

Spontaneous perforation of the extrahepatic bile duct is an exceedingly rare and usually fatal cause of bile peritonitis in adults. Freeland reported the first case of spontaneous hepatic duct perforation in 1982, which was diagnosed at autopsy1. Since then, very few cases of extrahepatic bile duct perforationhave been reported in literature. We recently managed a 35-year old man with spontaneous perforation of the common bile duct, present herein clinical details of the case as well as review of the pertinent literature.

CASE REPORT

A 35 yr old male operated 5 months back for fracture shaft left femur by open reduction and internal fixation, presented in Orthopaedic Department of our institution for ununited fracture the left shaft femur. Implant removal and exchanged intramedullary nailing with bone grafting was done. A bone graft was procured from the left iliac crest. On the 3rd postoperative day, he developed pain abdomen, which was continuous mainly in upper abdomen not associated with fever, vomiting or any other complaint. Bowel habits were normal. On examination the patient was hemodynamically stable, per abdominal examination was unremarkable with no signs of peritonism. Till the 13th postoperative day, the patient was seen several times by the surgical team for the same complaint. The patient was managed conservatively and did not show any worsening of symptoms or general condition.

On the 14th postoperative day, features of frank peritonitis developed (generalised, intense, continuous abdominal pain with nonpassage of flatus and faeces and per abdominal examination showing generalised tenderness, guarding). General condition of the patient worsened with tachycardia of 106/min and blood pressure of 90/60. The patient went in acute renal failure and also developed jaundice. On investigation X-ray abdomen (erect) showed - no gas under diaphragm, USG revealed free fluid in peritoneal cavity with normal liver, gall bladder, common bile duct (no stones). Liver function tests (LFT) were deranged (total bil. 5.4 mg/dl, direct bil. 2.6 mg/dl, indirect bil. 2.8 mg/dl, SGOT 196, SGPT 210, Alk. Phosphatase 276 IU/l). Kidney function test were deranged - Blood urea 181mg% and Serum creatinine 5.2.mg%. Abdominal paracentasis revealed a bilious collection. Exploratory laparotomy was planned with presumptive diagnosis of iatrogenic colonic perforation during bone graft harvesting.

Exploratory laparotomy was done. Operative findings included one litre of bile-stained purulent fluid in peritoneum with multiple pus flakes present, and a 0.75 cm x 0.75 cm perforation was present in anterior surface of common bile duct about 1.5 cm distal to cystic duct and common hepatic duct confluence. Gall bladder was normal and no stones or diverticulum were found in gall bladder & common bile duct.Thorough peritoneal lavage was done. A T-tube was inserted into common bile duct through perforation site and the abdomen closed with one subhepatic

drain.

Patient was prescribed inj Fortum (ceftazidime) 1 gm iv bd and inj metrogyl 500 mg tds for a week. The patient was given required fluids (normal saline and 5% dextrose) with careful serum electrolyte monitoring. The patient was hemodynamically stable postoperatively and did not require any inotropic support. The patient was regularly dialysed till 2 weeks until renal function recovered. Jaundice also resolved gradually.

T-tube cholangiogram was performed on the 14th postoperative day and revealed T-tube in normal position and distal path was patent. Magnetic resonance pancreaticocholangiography (MRCP) was done which was suggestive of normal intra and extrahepatic biliary tree. T-tube was removed on 14th postoperative day. Patient recovered well and discharged subsequently in satisfactory condition.

DISCUSSION

Perforation of the biliary system occurs most frequently in the gallbladder. According to Roslyn and Busuttil2 it is seen in 3-10% of patients with acute cholecystitis and is more common in elderly male presenting as acute cholecystitis, severe atherosclerotic cardiovascular disease, or malignancy.

Perforation of the extrahepatic bile duct on the other hand is far less common and is described mainly in infants due to congenital anomalies of common bile duct3. Very few cases of bile duct perforation have been reported in adults. The pathogenesis of nontraumatic perforation of the biliary tract system is poorly understood, but the following mechanisms are generally accepted4-9 [a] Calculous perforation at the site of impaction or erosion without evidence of mechanical disproportion of the ductal system **increased intracanalicular pressure due to obstruction by tumor, impacted stone, or spasm of the sphincter of Oddi, [c] intramural infection [d] intramural infarction due to thrombosis of the mural vessel [e] rupture of a diverticulum or a cyst of the biliary tract; [f] regurgitation of activated pancreatic juice through a common outlet of the common bile duct and the pancreatic duct; [g] traction from neighbouring organs and friability of biliary tract due to previous operation. A combination of factors is probably responsible for perforation of the common bile duct by a gallstone. Erosion of a calculus through the wall of the duct is usually a slow process and is frequently associated with fistula formation. Complete distal obstruction results in an elevation of the intraductal pressure and in most cases this will present as obstructive jaundice. However, if the wall of the bile duct has been weakened, the raised pressure may cause a perforation. Infection plays an important role in the etiology of spontaneous perforation, and combined with the presence of calculi and/or a diverticulum, is probably the important factor in causing weakness of the duct wall. Thrombosis of the intramural vessels probably follows infection, although the mechanism itself may be obscure. A spontaneous bile duct perforation may thus be due to single or multiple factors.**

Presentation of such patients is either insidious or acute. In acute cases, fulminant bile peritonitis with pain, vomiting, fever and distension abdomen are seen, while patients presenting insidiously painless abdominal distension, increasing jaundice, clay coloured stools are generally seen.

Ultrasound is useful for evaluating patients with suspected peritonitis because it provides rapid, safe, low cost evaluation of liver, gallbladder, pancreas, appendix and other viscera. It also detects and characterizes distribution of intra-abdominal fluid collection. CT scan has further provided definitive improvements in diagnostic accuracy in evaluating patients with acute abdominal pain10. But even after careful examination and investigation diagnostic uncertainty can remain. Some patients may have equivocal physical findings. Laparoscopy has emerged as an accurate modality for diagnosis and treatment of patients with acute abdominal pain where the diagnosis cannot be clearly made by physical examination and noninvasive methods. The diagnostic accuracy of laparoscopy varied from 93% to 100%. Also laparoscopic technique accomplish definitive treatment of the underlying disease in 44% to 73% of cases11,12.

Treatment recommended is cholecystectomy with CBD exploration & T-tube drainage. Patients may require even Roux-en-Y ductal reconstruction if the ductal disruption is severe.

Our patient presented with confusing features of postoperative ileus, viral hepatitis, stress induced peptic ulcer perforation, and Iatrogenic colonic perforation peritonitis. On exploration, no calculus or diverticulum was found in CBD. Localized ischemia of the bile duct wall can't be ruled out. We presume that a localized inflammatory event of the CBD wall must have resulted in CBD perforation, possibly with increased biliary pressure due to sphincter spasm.

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