

aspirated to yield a similar picture on direct microscopy and grew *C. albicans*. He was diagnosed to have Candidial liver abscess and was prescribed oral fluconazole 200 mg BD for 6 weeks as the organism was found to be sensitive to the drug. His pain subsided with treatment and an abdominal ultrasound done two months later showed complete resolution of the abscess (**Figure 2**).

Discussion

Our patient was a young male with a large solitary liver abscess. Amoebic liver abscess would have been the expected clinical diagnosis with this presentation. However amoebic serology was negative and the cultures from the abscess cavity repeatedly grew *Candida albicans* without any sign of bacterial growth, thus leading us to the diagnosis of a Candidial liver abscess. The gastrointestinal tract appears to be the probable source of the *Candida* infection in this patient.

To the best of our knowledge, this is only the second instance of a fungal liver abscess reported in an immunocompetent host. In an earlier report *Candida glabrata* liver abscess was discovered in a 64-year-old female, but that patient had chronic obstructive pulmonary disease and had received multiple antibiotics as well as short duration steroid therapy.¹ In contrast our patient did not have any long standing illness and had never been on any immunosuppressant medication. Most hepatic fungal abscesses occur in patients with hematological malignancies and are caused by *Candida albicans*.² Marcus et al, observed that 19 of 20 fungal abscesses in cancer patients were caused by *Candida albicans*. Fifteen of them had hematologic malignancies.³

The patient responded to fluconazole. The current IDSA clinical practice guidelines on the management of candidiasis also recommend fluconazole as the initial therapy for invasive candidiasis caused by *C. albicans* in non-neutropenic adults.⁴

The fact that the patient was immunocompetent may have some bearing on the unusual clinical presentation and excellent response to treatment that was seen in this patient. Our report suggests that it will be prudent to exclude fungal infections in case the bacterial cultures are sterile from a pyogenic abscess as attempts to treat can be quite rewarding.

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Left hepatectomy for oriental cholangiohepatitis

Introduction

Oriental cholangiohepatitis is endemic in the Asia-Pacific region. We are presenting two cases of oriental cholangiohepatitis managed by hepatic resection.

Case reports

Case 1

A 40-year-old woman presented with recurrent pain abdomen and fever with chills since last 2 years. There was no history of jaundice. The haemogram showed a haemoglobin of 10.3 gm%, leucocyte count of 10,290/cumm, and an ESR of 50 mm/hr. Liver function tests showed bilirubin 0.3 mg/dl, ALP 221 IU/L, SGPT 13 IU/L, and GGTP 27 IU. USG abdomen showed left lobe atrophy with dilatation of intrahepatic radicals with intraductal calculi seen in proximal segment of the left hepatic duct. The common bile duct was mildly dilated. CT findings were



Figure 1: CT scan showing grossly dilated intrahepatic biliary radicals, left hepatic duct stricture with intraductal calculi suggestive of oriental cholangiohepatitis with stricture of the left hepatic duct with dilatation of intrahepatic biliary radicals, intraductal calculi and atrophy of the left lobe of liver (**Figure 1**). Cholecystectomy and left hepatectomy were performed for the condition. The post-operative period remained uneventful. The intra-operative bile culture showed growth of *E. coli*. Histopathology findings were consistent with chronic cholangiohepatitis and subacute cholecystitis.

Case 2

A 45-year-old woman presented with generalised weakness and low grade intermittent fever since two years. Her liver function tests were normal. USG abdomen showed left liver lobe atrophy. MRCP showed intrahepatic calculi, left hepatic duct stricture and left lobe atrophy (**Figure 2**). Left hepatectomy and cholecystectomy was done, and the patient made an uneventful recovery.

Discussion

Oriental cholangiohepatitis (OCH) with intrahepatic stones is commonly seen in Asia, particularly in China, Hong Kong, Korea and Japan. The principles of hepatolithiasis management include clearance of stones, correction of strictures, removal of lesions and restoration of biliary drainage.¹ Most patients present with acute cholangitis which is managed by intravenous fluids, antibiotics and analgesics. Urgent biliary decompression is requisite to prevent the patient from going into shock.



Figure 2: MRCP showing left hepatic duct stricture with intraductal calculi

Decompression can be achieved by endoscopic, radiologic or operative approaches. Endoscopy may be a successful treatment option for migrating stones in the absence of bile duct strictures. Interventional radiology by percutaneous approach may help treat intrahepatic lithiasis secondary to the stricture of bilioenteric anastomoses. Intracorporeal or extracorporeal shock-wave lithotripsy can be useful when combined with endoscopic or percutaneous procedures.²

Surgical options include cholecystectomy, exploration of the common duct and choledochoscopy with or without hepaticojejunostomy, hepatico-cutaneous jejunostomy, and partial hepatectomy. Hepatic resection can reduce the risk of recurrence of stones, since it removes not only intrahepatic stones but also the associated pathological bile ducts damaged by strictures.³ Indications for hepatectomy include, (i) unilobar hepatolithiasis, particularly involving the left sided; (ii) atrophy, fibrosis and multiple cholangitic abscesses; (iii) suspicion of concomitant cholangiocarcinoma; (iv) multiple intrahepatic stones with biliary strictures that cannot be treated percutaneously or endoscopically. Non-surgical therapy is most useful in bipolar hepatolithiasis without strictures or in patients who pose high surgical risk or have short life expectancies.⁴ If the liver parenchyma is diffusely affected by the disease, cirrhosis, and portal hypertension, liver transplantation may be the only alternative.

In conclusion, liver resection remains the definitive surgical option for OCH as it removes the stones as well as the strictures and the possibility of carcinoma.

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Severe acute pancreatitis caused by *Ascaris lumbricoides*

Introduction

In the Indian subcontinent, ascariasis is highly endemic in Kashmir (70%), Bangladesh (82%), and central and southwest India (20–49%).¹ We report here a case of acute pancreatitis following ascariasis within the main pancreatic duct.

Case report

A 24-year-old female presented to our Emergency Department with high grade fever and moderate, non-colicky right hypochondrial pain, associated with vomiting since 2 days. The patient was icteric, had tender hepatomegaly and liver function tests showed a serum bilirubin of 2.4 mg% and AST of 11,740 IU/ml and ALT of 7220 IU/ml. IgM anti HAV was positive and a diagnosis of acute viral hepatitis A was made. After 5 days of conservative management, the patient improved symptomatically and was discharged from the hospital. But she returned one day after her discharge with severe boring continuous abdominal pain in the epigastric region which was

radiating to the back and was associated with vomiting. Abdominal examination revealed marked epigastric tenderness and repeat liver function tests showed a serum bilirubin of 8.4 mg% ,AST: 168 IU/ml, ALT: 793 IU/ml. Serum amylase was 1346 IU/ml and serum lipase was 2249 U/L.

A clinical diagnosis of acute pancreatitis was made and the patient was kept on conservative management. Ultrasound (**Figure 1**) examination revealed hepatomegaly, an edematous gall bladder, normal common bile duct (CBD) and hypoechoic bulky pancreas more towards the tail region, with a linear echogenic structure in the main pancreatic duct extending from the region of the head of pancreas up to the tail. The ‘four-lines sign’ was distinguishable on scanning the duct in cross section. However the patient’s condition did not improve and the next day a contrast enhanced CT scan of abdomen was performed, which showed an enlarged pancreas with decreased attenuation, shaggy outline with ill-defined peripancreatic planes and peripancreatic fluid suggestive of acute edematous pancreatitis. The pancreatic duct was dilated (3 mm in diameter), in the region of the pancreatic head and body. A curvilinear tubular soft tissue attenuation filling defect was seen in contrast opacified duodenum extending up to the ampulla of Vater with an air speck within, possibly a round worm infiltrating the ampulla (USG correlation was also done). An MRCP showed a mildly dilated main pancreatic duct (3 mm in diameter) harboring a hypointense linear structure with surrounding hyperintensity in proximal part of the main pancreatic duct. The same day after the MRCP, the patient underwent an ERCP (endoscopic retrograde cholangiopancreatography, (**Figure 2**) in which an *Ascaris* protruding out of the papilla was seen. The round worm was grasped with a foreign body forceps, and retrieved

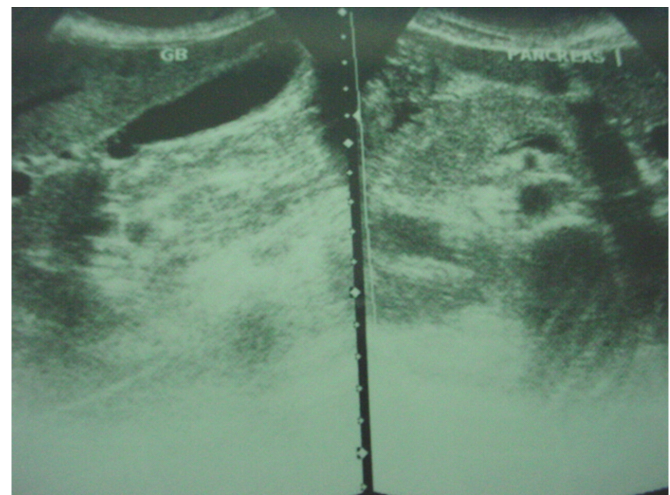


Figure 1 : Ultrasound showing a linear echogenic structure in the main pancreatic duct