BILIARY ASCARIASIS

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SYNOPSIS

Two cases of biliary ascariasis, one presenting as ascending cholongitis and the other as cholelithiasis are described. Its varied clinical manifestations and management are discussed.

INTRODUCTION

Invasion of biliary channels by Ascaris lumbricoides is known but its incidence varies, even in populations with high intestinal infestations rate (1). The migrating worms can produce variety of manifestations ranging from biliary colic, ascending cholangitis, oriental cholangiohepatitis, biliary duct strictures to hepatic granulomas and hepatic abscesses (2).

CASE REPORT

A 34 year old male was admitted for severe right hypochondrial pain of 2 days duration. The pain was paroxysmal, radiating to back and was accompanied by nausea and vomiting. There was no history of previous attacks of pain, jaundice or fever.

On clinical examination, he was febrile (38.5°C) but not jaundiced. The right hypochondrium was tender with mild rigidity. Murphy's sign was positive.

Investigations

White cell count was 10,400/C. mm with 70% of polymorphs 4% eosinophils and 26% Lymphocytes. Serum bilirubin: 1.6 mgms % (0.5 to 1 mgm %) Plain xrays of the abdomen and chest did not reveal any abnormality.

With the provisional diagnosis of acute cholecystitis the patient was managed conservatively with intravenous fluids, ampicillin 500 mg × 6 hourly and gentamycin 80 mg × 8 hourly. The pain subsided for a few hours but recurred with attacks of increasing severity the next day and the patient became mildly jaundiced. In view of these laparotomy was done.

On laparotomy, the gall bladder was found to be distended and thin walled with edema of the peritoneum covering the gall bladder. No stones were palpable in the gall bladder. The common bile duct (CBD) was dilated. The liver was normal. Cholecystetomy was done and the CBD was opened.

It was found to contain a live ascaris worm about 4" in length (Fig. 1) with some biliary sand. There were no stones. After removing the worm and the biliary sand from the CBD and ensuring its patency, a T-tube was placed.

Usual post operative management was instituted and tab. Mebendazole. 100 mgm bd was added from the third day for three days. Post operative T-tube cholangiogram on 8th day, showed free flow of dye into duodenum, without filling defects in the CBD.

CASE REPORT 2

A 68 years old female presented with recurrent right hypochondrial pain for one year. However, there was no history of jaundice and fever. She was afebrile and had a palpable, non tender gall bladder.

White cell count was 7,800/c. mm with polymorphs 66% and lymphocytes 34%. Total serum bilirubin was 12/mmol/l (normal range 3.4 to 20.4/mmol/L. Plain xray abdomen was normal. Ultrasound examination (done in a private clinic) showed a dilated gall bladder with a gallstone. The common bile duct was normal.

A preoperative diagnosis of mucocele of the gall bladder was made. On laparotmy, the gall bladder was thin walled, distended with bile and contained a stone. The stone was producing partial obstruction of the gall bladder outlet. The CBD was moderately dilated. Cholecystectomy was done followed by supraduodenal choledochotomy. After sucking the bile and biliary sand, dead ascaris worms (4 pieces of 1½" to 2" each) were found in the CBD and were removed. Patency of CBD was ensured and a T-tube was placed.

Few ascaris worms were palpable in the proximal small bowel. The post operative management was similar to the first case. T-tube cholongiogram on 8th day showed free flow of dye into duodenum without filling defects.

Histopathology of gall bladder revealed chronic inflammatory cells, mostly eosinophils and lymphocytes. The gall stone which was 1 cm × 1 c m in size and blackish in colour was positive for bile pigments, calcium and carbonate and negative for cholesterol.

DISCUSSION

The incidence of biliary ascariasis varies widely, even in populations with high intestinal infestation rate. In India biliary invasion by ascaris worms is very rare but is frequent in China (1). In one series from South Africa, intravenous cholongiogram revealed

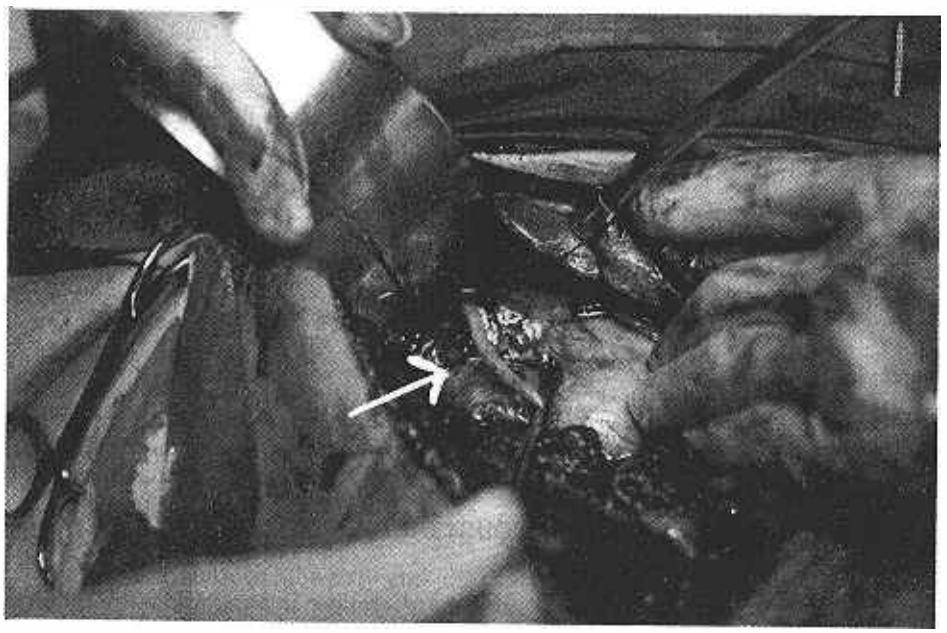

FIG. 1: Ascaris worm in the common bile duct.

biliary ascariasis in 40 of the 68 patients with intestinal infestation (3). The reason for these variations is not known.

Biliary ascariasis can produce a variety of manifestations. It can present with mild or severe recurrent right upper quadrant pain and when complicated by secondary infection as ascending cholangitis (3). Recurrent attacks of cholongitis can mimic cholelithiasis (2).

Ascaris is implicated in oriental cholangiohepatitis, characterized by cholelithiasis (often without cholecystolithiasis), biliary duct strictures and hepatic abscess. The common duct stones are usually multiple, soft and black (2).

The worm in the CBD may be alive or dead (4). The degenerated worm or ova may form a nidus for biliary sand and stones (4). Superadded bacterial infection also leads to precipitation of bilirubin by enzymatic degradation (5).

The worm may migrate into the liver where it excites granuloma formation. Degeneration of the worm in the liver with secondary infection leads to hepatic abscess. In some tropical countries, ascaris liver abscess is commoner than amoebic abscess in children (6). Ascaris in biliary tree can cause haemobilia (7) and rarely acute pancreatitis due to obstruction of ampulla of Vater (1).

Usually one or two worms are present in the biliary channels but sometimes massive invasion occurs. Complications like septicemia and hepatic abscess are higher in massive invasions and require early diagnosis and treatment (3).

Intravenous cholangiogram and ultrasonogram are highly useful in preoperative demonstration of worms. The latter may reveal typical linear, sonar opaque shadows (3). Sometimes endoscopic retrograde cholangiopancreatogram is required (2).

The following management is suggested for cases suspected or diagnosed to be biliary ascariasis by investigations (2, 3).

Most cases wil respond to conservative measures, the worms returning spontaneously to the intestine. This is enhanced if Mebendazole is given orally to deworm the intestine. Mebendazole is preferred as it is poorly absorbed from the intestine the intestinal worms are killed but the biliary worms are spared and return to the intestine. Surgery is reserved for patients who fail to respond to an adequate trial of conservative therapy or show evidence of complications like hepatic granuloma and abscess (3).

Other indications for operation are persistent right upper quadrant colic, severe (serum bilirubin 70 mmol/L) or increasing jaundice and signs of right upper quadrant peritonitis (3). Oriental cholengiohepatitis with recurrent attacks usually requires surgery as multiple stones and sediments are present in the CBD with secondary changes like biliary strictures and hepatic abscess (2).

Cases treated conservatively should be followed up with intravenous cholangiogram. If persistent filling defects are demonstrable surgery or endoscopic removal is indicated (5).

Post operatively mebendazole should be given, once gastrointestinal function is established, as reinfestation of biliary tree from intestine can occur (3). Post operative T-tube cholangiogram excludes reinfestation. If recurrent biliary invasion fails to respond to oral mebendazole flushing saline through the T-tube or endoscopic removal or re operation are indicated. Antihelminthics should not be administered through the T-tube as this will kill the worms in the bile ducts (3).

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