CASE REPORT

HEPATOBILIARY ASCARIASIS COMPLICATED BY PANCREATITIS

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Ascaris lumbricoides is the commonest organism causing soil-transmitted helminth infection. It is particularly common in poor sanitary conditions. Nevertheless, involvement of the gallbladder by *Ascaris* is a rare entity. A lady presented to us with long-standing history of vague abdominal symptoms suggesting dyspepsia. Ultrasound showed a tube like structure invading the biliary channels. Serum amylase was elevated and the patient was managed conservatively, as for acute pancreatitis. She improved clinically, but subsequent imaging with magnetic resonance cholangiopancreatography revealed worm in the gallbladder. Laparoscopic cholecystectomy was done and *Ascaris lumbricoides* was removed. *Ascaris* infestation is an important differential diagnosis of patients with upper abdominal symptoms and screening with stool examination and ultrasound is warranted in high-risk population.

Keywords: *Ascaris lumbricoides*, soil-transmitted helminthes, gallbladder, cholecystitis, pancreatitis, dyspepsia

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INTRODUCTION

Ascaris lumbricoides and other soil-transmitted helminthes are highly prevalent throughout the world, particularly in Asia. In 2010, an estimated of 819 million people globally have been infested with *A. lumbricoides*, constituting the most common infection among the other soil-transmitted helminthes. Above all, significant disability has been reported with *A. lumbricoides*, amounting towards 1.10 million years lived with disability (YLDs).[1]

Poor hygiene and sanitary conditions promote the transmission of *A. lumbricoides*, which is spread through faeco-oral route.[2] Gallbladder is a rare site of invasion by this worm, accounting for only 2.1% of patients with biliary system involvement.[3] In this report, we describe a case of hepatobiliary ascariasis involving the gallbladder and complicated by pancreatitis.

CASE REPORT

A 52 year old female presented in April 2014, to the emergency department Mayo hospital Lahore, Pakistan, with the complaint of epigastric pain for three days. The pain was gradual in onset, severe in intensity, sharp and squeezing in nature, radiating to back and right hypochondrium, relieved by lying supine and aggravated by leaning forward. It was associated with nausea and vomiting. The patient experienced five to six episodes of yellow coloured vomitus per day, containing undigested food particles. Four hours before reaching the hospital, she vomited out a long, round, tubular "snake-like creature", which prompted her to visit the emergency department. There was no history of blood in vomitus, constipation, diarrhoea, or fever. Our patient experienced dyspepsia for four years, which was managed by primary care physicians by over-the-counter preparations. Multiple gastroscopies, done during these four years were non-contributory in determining a cause of recurrent dyspepsia. There was no personal history of diabetes mellitus, hypertension, cardiac disease, dyslipidaemia, smoking or alcohol consumption. No relevant family history was found.

On physical examination, her vitals were: pulse 102/minute, blood pressure 130/70 mmHg, temperature 99.0^0F and respiratory rate 18/minute. The patient was jaundiced. Her abdomen was soft, but tender in epigastrium and right hypochondrium. Abdominal viscera were not enlarged. Clinically there was no ascites and bowel sounds were audible. Other systemic examinations were unremarkable.

Laboratory tests revealed haemoglobin 116g/L, white cell count $10.4X10^3$/µL, platelet count 189×10^3/µL, total bilirubin 6.0 mg/dl, ALT 620 IU/L, AST 541 IU/L, alkaline phosphatase 439.6 IU/L, serum total proteins 6.3 g/dl, serum albumin 3.5 g/dl, urea 18.2 mg/dl, creatinine 0.5 mg/dl, sodium 137 mmol/L, potassium 4.2 mmol/L, chloride 104 mmol/L, LDH 318.4 IU/l and serum amylase 2900 IU/L. Her fasting lipid profile was normal. Ultrasound abdomen showed a mobile tube like structure in the gallbladder, with normal calibre of biliary channels.

The patient was managed conservatively in high dependency unit. She was kept *nil per oral* (NPO) and a nasogastric tube was passed. Her

intake/output charts were maintained and vitals monitored three hourly for the first forty-eight hours of admission. The Ranson's score on admission was one. Acute Physiology and Chronic Health Evaluation II (APACHE II) score was two. The Bedside Index for Severity in Acute Pancreatitis (BISAP) score was zero. Empirical antibiotic and mebendazole 100 mg 12 hourly was given.

Our patient improved clinically. Her Ranson's score was zero at 48 hours. She became pain free on third day of admission. Serum amylase became normal after seven days of therapy. Serum lipase was also reported as normal (24IU/l). Her liver function tests (LFTs) improved gradually and became normal on tenth day of admission. A follow-up hepatobiliary ultrasound revealed an immobile echogenic linear shadow with central lucency in the gall bladder cavity. Subsequently magnetic resonance cholangiopancreaticography was done, which showed a thin tortuous hypo-intense band in the lumen of gall bladder suggesting the possibility of worm (Figures 1-3). Intrahepatic and extra hepatic biliary channels were of normal calibre.

Surgery was planned and the patient was shifted to surgical floor. Laparoscopic cholecystectomy was done and *Ascaris lumbricoides* was extracted (Figure-4). Gross examination of specimen showed a 10×0.3 cm worm and multiple gall stones. Histological examination of the sample revealed mucosal hyperplasia, Rokitansky-Aschoff sinuses, and fibro-collagenous wall with focal infiltrations by lymphocytes (chronic cholecystitis).

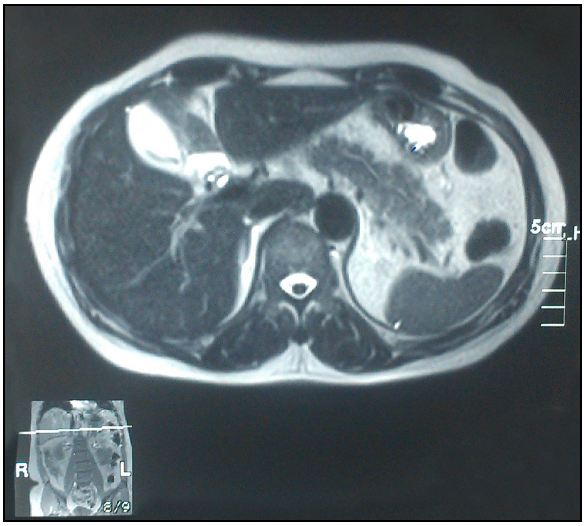

Figure-1: T2 weighted transverse image showing a hypo-intense band against the hyper-intense signals of gallbladder

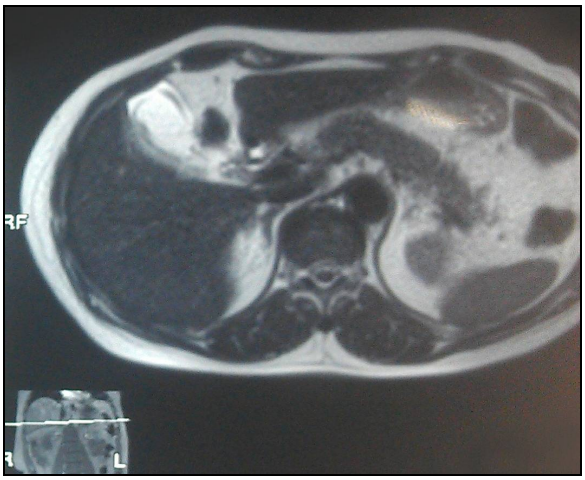

Figure 2: T2 weighted transverse image showing a hypo-intense band looping along the wall of gallbladder, involving the cystic duct

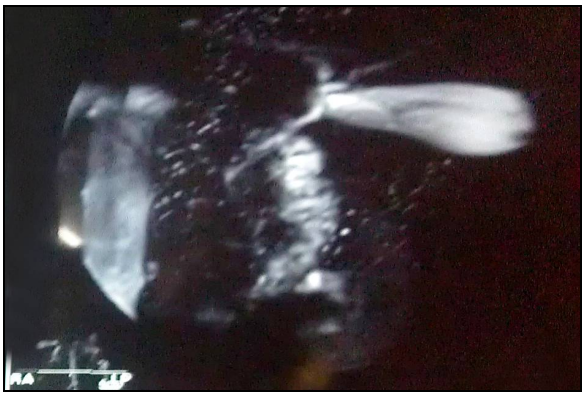

Figure-3: T2 weighted coronal image showing a linear smooth edged hypo-intense band in the gallbladder

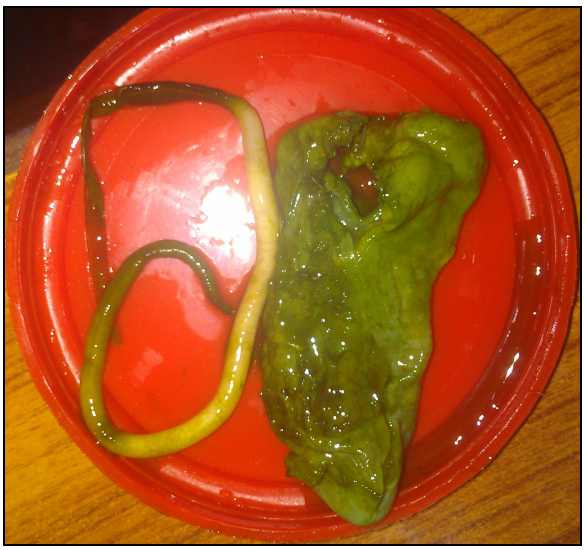

Figure-4: Gross specimen of gallbladder and Ascaris lumbricoides after laparoscopic cholecystectomy

DISCUSSION

A. lumbricoides is known to cause an indolent course of infection in the gastrointestinal tract. Fertilized eggs of the worm, found in the excreta of infected humans, when ingested due to unclean eatables and drinking water result in transmission of infection to other hosts.[4] The eggs hatch into larvae that penetrate the duodenum wall to enter bloodstream. Thereafter, it invades different organ systems including the pulmonary vasculature, from where it breaks free through the alveolar wall. Eventually it is coughed up to re-enter the gastrointestinal tract lumen, where it matures to adult form.[5]

Ascaris residing in the jejunum may invade the biliary or pancreatic ducts, resulting in bile duct obstruction, cholecystitis, cholangitis, pancreatitis, or liver abscesses.[4] High parasite load in the intestine increases the chance of *A. lumbricoides* moving through the narrow tortuous cystic duct to invade the gallbladder, promoting an attack of acute cholecystitis. Gallbladder Ascariasis often requires surgical intervention, since antiparasitic drugs have a very low excretion rate through the bile.[3] Medical treatment is less effective, and cholecystectomy is particularly required when *A. lumbricoides* fails to move out of the gallbladder with antiprotozoal drugs, or when the worm is resting dead in the gallbladder, or when there is associated gallstones.[3] Remains of worm in the bile duct or gallbladder may result in a severe inflammatory response leading to ductal necrosis, calcification, stone formation, stenosis or fibrosis.[4]

Early diagnosis of *A. lumbricoides* infection can be difficult, particularly as a result of vague symptom presentations and constant mobility of the worm within the gastrointestinal tract lumen. Our patient had a history of vague abdominal symptoms suggesting acid peptic disease since 4 years, which had been treated by primary care physicians as non-ulcer dyspepsia. Imaging with ultrasound has an important role in viewing the parasite. Sonography reveals *A. lumbricoides* as an echogenic linear, tubular structure in longitudinal view or having a pseudotumour appearance in the transverse view.[6] Magnetic resonance imaging (MRI) has recently been much helpful in identifying *Ascaris* and signs of any associated complications like pancreatitis. On magnetic resonance cholangiopancreatography, intraductal worms are seen as linear hypo-intense filling defects.[7] In high prevalence areas, stool examination for parasite ova[8] and ultrasound imaging[9] could prove good screening tools.

A. lumbricoides infestation must be kept on the differential diagnosis in patients with vague abdominal symptoms suggesting refractory acid peptic disease. Screening of suspected patients in endemic areas could be very useful in helping cure millions and preventing further spread from the "Ascaris Mary".

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