

Case Report

Common bile duct worm induced pancreatitis: a rare case report from an urban area

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ABSTRACT

Ascaris is the most common intestinal helminth to infest humans in the world. In India, it is endemic to Kashmir and rarely occurs in an urban setting. It has a varied presentation including pancreatitis. Here, we present a case report of a female patient suffering from pancreatitis induced by common bile duct ascariasis in a non-endemic urban area. A 56 year old lady presented with pain upper abdomen and nausea with tenderness in her epigastrium. Contrast enhanced computed tomography was suggestive of pancreatitis. Patient then vomited a worm and further similar history of worm emesis and passage in faeces was elicited. Magnetic resonance cholangiopancreatography showed linear filling defect in distal CBD. 5 live worms were removed via endoscopic retrograde cholangiopancreatography. Vermifuge was started and her follow-up was uneventful. Therefore, there should be a strong suspicion in patients presenting with pancreatitis having a past history or family history of passage of worms in vomits or faeces, even in non-endemic areas.

Keywords: *Ascaris*, Pancreatitis, MRCP, ERCP, Vermifuge

INTRODUCTION

Ascariasis, as a cause of pancreatitis, is the most common after gall stones in the state of Kashmir in India that is endemic to *Ascaris*.¹ In other parts of India, especially urban areas, *Ascaris* infestation is rare. It is possible for ascariasis to occur in the non-endemic areas due to easier transportation between rural and urban areas.² Therefore, the suspicion of *Ascaris* induced pancreatitis should be more.

CASE REPORT

A 56 year old lady presented to the surgery emergency with complaints of pain in her upper abdomen radiating to the back for 5 days with nausea for 1 day. There was no history of vomiting, fever, jaundice, constipation or

dyspepsia. She had no history of any previous comorbidities or surgery.

The patient's general condition was poor. She was tachypneic at 24 breaths/min and was tachycardic at 104 beats/min with a blood pressure 86/68 mmHg. She was icteric as well. Abdominal Examination showed that the abdomen was soft, tender in epigastrium and left hypochondrium with guarding. There was no rigidity, no lump or organomegaly, no free fluid and bowel sounds were present. Basing on the history and examination, a presumptive diagnosis of acute pancreatitis was kept. Patient was kept nil per oral. Intravenous fluids and analgesics were given and bowel rest was advised.

Preliminary blood investigations showed leukocytosis with TLC 19800/mm³. Serum amylase and lipase at

presentation were 399 IU/L and 719 IU/L respectively. Liver enzymes were raised. Ranson's scoring at presentation and 48hrs after presentation were both three.

Table 1: Blood investigations of patient at 0 hour and after 48 hours.

Blood investigations	0 hour	48 hours
Hct (%)	34.3	27.8
TLC (/mm ³)	19800	18100
T.bil/d.bil (mg/dl)	2/0.4	3.0/1.7
S. amylase/S. lipase (IU/L)	399/719	494/650
AST/ALT (IU/L)	53/28	-
LDH (IU/L)	450	-
BUN	21	16
Calcium (mg/dl)	-	7.3
PaO ₂ (mmHg)	-	89
Base deficit (mEq/L)	-	4.5
Fluid deficit (L)	-	7
CRP (mg/L)	110	
Ranson's scoring	3	3

Lipid profile (TG/Chol/LDL/HDL): 129/156/67/68.



Figure 1: Worm in vomitus of patient.

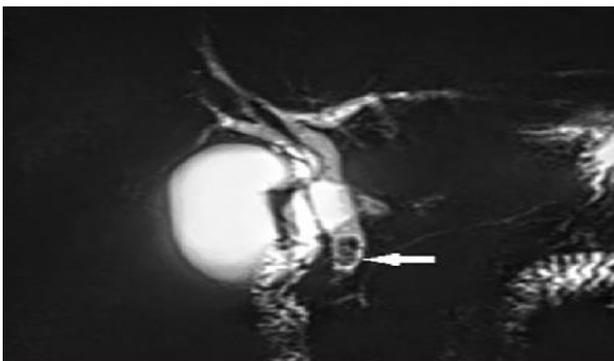


Figure 2: MRCP of patient showing dilated CBD and filling defect in CBD.

Ultrasound (USG) showed an obscured pancreas. There were no gall stones and the common bile duct was normal. In view of non-improvement of Ranson's scoring and abdomen still being tender with guarding, contrast enhanced computed tomography (CECT) abdomen was done which revealed a mildly bulky pancreas, peripancreatic fat stranding and heterogeneity, suggestive

of acute pancreatitis. The common bile duct (CBD) was dilated with a thick enhancing wall and distal tapering, suggestive of cholangitis. There was also presence of bilobar intrahepatic biliary radical dilatation (IHBRD) and multiple cholangiolar abscesses.

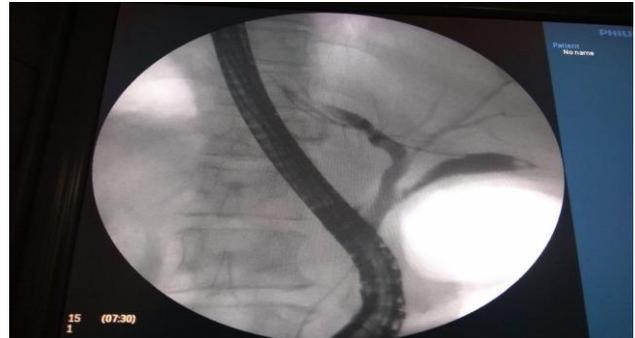


Figure 3: Cholangiogram showing multiple linear filling defects in upper CBD and left hepatic duct.



Figure 4: ERCP of patient showing worm in CBD.



Figure 5: Ascaris worms extracted via ERCP.

The patient then vomited a single live worm. Similar history of worm emesis and passage of worms in flatus and faeces was then elicited in the patient 6 months ago and in the family 8 months ago. A suspicion of hepatopancreatic ascariasis was kept. Magnetic resonance cholangiopancreatography (MRCP) was done showing a dilated common bile duct, linear filling defect in the distal common bile duct with focal dilatation of biliary radicles and cholangiolar abscess formation.

The patient then underwent endoscopic retrograde cholangiopancreatography (ERCP). The cholangiogram showed a linear filling defect in the common bile duct and further ERCP helped in extraction of 5 live round worms. Patient got symptomatically relieved. antihelmintics in the form of a stat dose of tablet albendazole 400 mg OD was prescribed for vemifuge.

Patient was followed up, 4 weeks later with complete resolution of her symptoms.

DISCUSSION

Ascariasis is the most common intestinal helminth infesting humans in the world. 25% of the world’s population is infected with ascariasis. The endemic parts of the world include China, South America, Africa and India.¹ In India, the endemic areas include Kashmir, Bihar and Tamil Nadu. In Kashmir, 70% of the population are infected with ascaris worm some time in their lives.² Poor sanitation is the most important risk factor.³

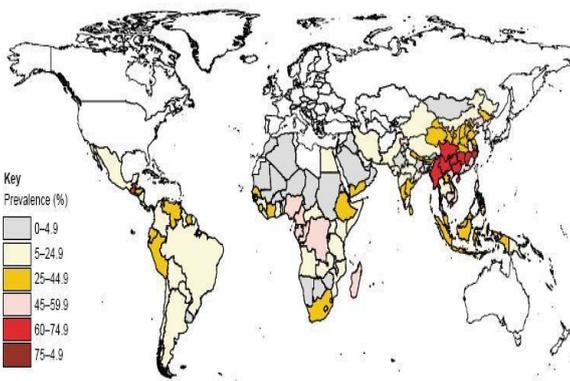


Figure 6: World prevalence of ascariasis.⁴

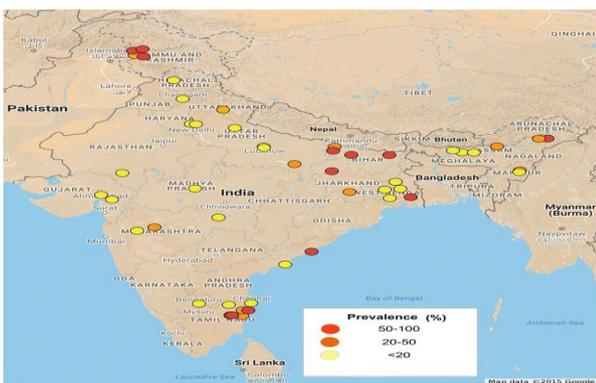


Figure 7: Prevalence of ascariasis in India.⁵

Ascariasis can cause a broad spectrum of diseases and can infect intestine, peritoneum, appendix, pancreas, lungs, hepatobiliary system or pancreas. Hepatopancreatic ascariasis may cause biliary colic, acalculous cholecystitis, cholangitis, pancreatitis or hepatic abscesses. Pancreatitis constitutes 5% of all cases

of hepatopancreatic ascariasis. 23% of pancreatitis patients in Kashmir are due to hepatopancreatic ascariasis, 78% of which is mild and 22% cases are severe.⁶ Pancreatitis due to hepatopancreatic ascariasis is more common in the age group of 35 to 42 years and is rare in children due to a small calibre biliary tree. It has a male preponderance with male to female ratio of 3:1.² Most cases of *Ascaris* related pancreatitis are mild, although a small group of patients with severe *Ascaris* infection and pancreatitis die.

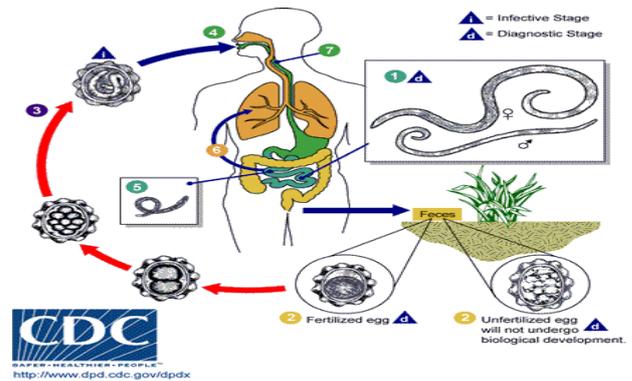


Figure 8: Life cycle of *Ascaris lumbricoides*.⁷

Ascaris lifecycle starts with ingestion of an egg. It develops into a larval stage in the alimentary canal where it can invade into the portal venous system and then into lungs. It migrates upwards to the trachea and then into the oesophagus where it is swallowed and then comes back down into the alimentary canal where it develops into an adult worm.



Figure 9: USG showing 4 line sign.¹¹



Figure 10: USG showing spaghetti appearance.¹¹

When there is a high worm load with duodenal ascariasis, worms have a tendency to enter any orifice they sense, termed as wanderlust. Here they breach the ampulla of Vater and enter the common bile duct, pancreatic duct, common hepatic duct, cystic duct or gall bladder causing the spectrum of hepatopancreatic ascariasis diseases. In the biliary tree the worm wriggles persistently causing sphincteric spasm and dysmotility thereby causing pancreatitis.⁸

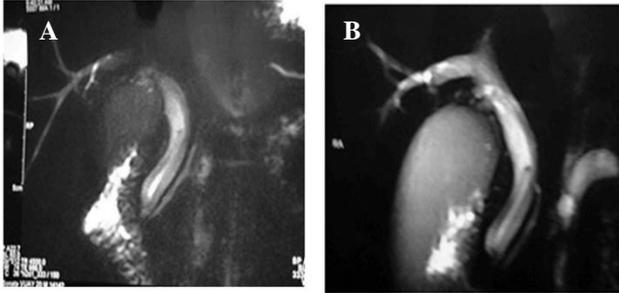


Figure 11: A) and B) MRCP showing filling defect in CBD.¹²

Risk factors for developing pancreatitis due to ascariasis include previous cholecystectomy, sphincterotomy, pregnancy or fasting.⁹



Figure 12: ERCP showing worm in CBD.¹⁴

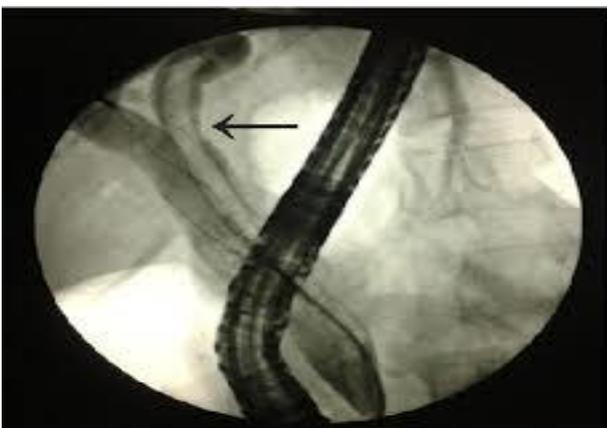


Figure 13: Filling defect in cholangiogram.¹⁴

CBD worm induced pancreatitis may present like a typical pancreatitis case. The patient will have complaints of pain upper abdomen with or without radiation to the back, nausea or vomiting. Patient may have worm emesis. The patient may also present with icterus if the worm is obstructing the biliary tree. Patient would have a tender epigastrium and left hypochondrium with guarding. Blood investigations would show leukocytosis with or without eosinophilia. Eosinophilia is seen only in 5-12% of extra-pulmonary ascariasis. Stool would be positive for ova.¹⁰

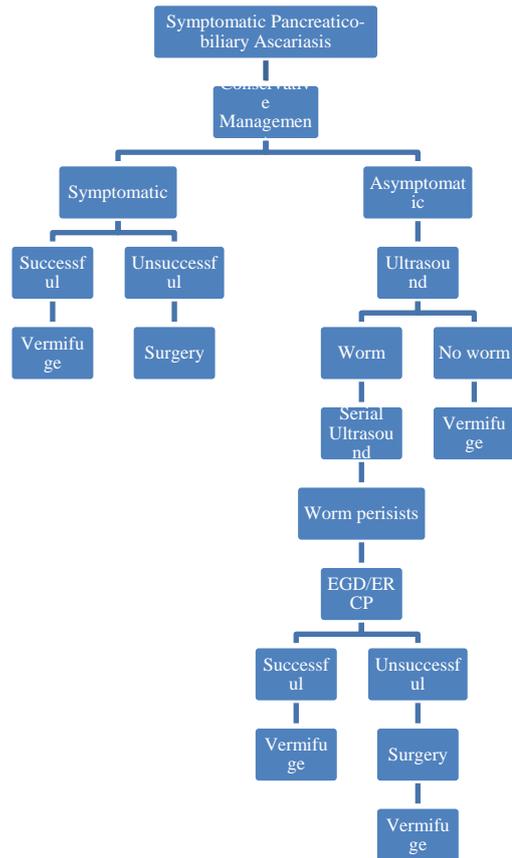


Figure 14: Algorithm for management of CBD worm induced pancreatitis; conservative management includes analgesics, anti-spasmodics and antibiotics.¹⁷

EGD - Esophagoduodenoscopy; ERCP: Endoscopic Retrograde Cholangiopancreatography.

Ultrasound has a 50-80% sensitivity of detecting biliary ascariasis. The sensitivity is less for pancreatic ascariasis and single worms. USG showing signs like strip sign, 4 line sign, spaghetti like appearance are suggestive of biliary ascariasis. These signs may be associated with biliary or pancreatic dilatation, hepatic abscesses, air in the biliary or pancreatic ducts or gall bladder wall thickening. Air may be present in the biliary or pancreatic ducts and there may be presence of intrahepatic abscesses.¹¹

Plain radiograph of abdomen may show air fluid levels suggestive of obstruction or whirlpool pattern of intraluminal worms. Biliary worms may calcify which can be seen on X-ray abdomen. Chest radiograph may show fleeting opacities during pulmonary migration. Barium meal follow-up shows linear or coiled filling defects, sometimes with thin central track of barium outlining the worm's intestinal tract. CECT abdomen demonstrated dilated IHBRD (at periphery), bulky pancreas (in cases of pancreatitis) and dilated bowel loops with filling defects after administration of oral contrast medium (in cases of obstruction).¹²

Magnetic resonance imaging (MRI) and MRCP are good imaging modalities to detect the above condition. Axial images in T2 weighted sequence show a dot hypointense signal in the CBD around which the bile signals are hyperintense. MRCP shows intraductal worms as linear hypointense filling defects. Its use for diagnosing CBD worms has not been studied.¹¹

A patient presenting with a strong suspicion of *Ascaris* induced pancreatitis i.e. presenting with features suggestive of pancreatitis and a history of passage of worms in vomits and faeces should first be resuscitated and kept nil per oral. If the patient remains symptomatic, ERCP should be done to remove the worms followed by vermifuge. The patient should first undergo USG if asymptomatic. If there is an absence of worms, conservative management should continue with vermifuge. ERCP is the treatment of choice if serial ultrasounds show presence of CBD worms. This is followed by vermifuge. In the event that ERCP fails, patient should be planned for surgery at the earliest. Laparoscopic CBD exploration and worm extraction should be done. It is converted to open if complicated by bowel perforation or peritonitis. Therefore, the first-line treatment is ERCP combined with antihelminthic drugs. First choice drugs used for eradication are albendazole and mebendazole and alternatively, levamisole.^{13,14} This is successful in 90% patients.¹⁵ Persistence of worm in the biliary tree due to failure of removal or non-detection can cause a myriad of complications. Persistence of a dead worm is a nidus for stone formation and furthermore, obstructive jaundice can be a possibility. Dead worms may calcify and cause recurrent pancreatitis. Even successful removal of worm may be followed by reinvasion of the biliary tree. This occurs in 15-28% cases at one year. Antihelminthic therapies act against the adult worm but not against the larvae. Therefore, patients should be re-evaluated in 2-3 months following therapy with repeat stool microscopy and ultrasonography.¹⁶

CONCLUSION

Ascariasis is an uncommon cause of biliary pancreatitis. There should be a strong suspicion in patients presenting with pancreatitis having a past history or family history of passage of worms in vomits or faeces even in non-

endemic areas. Endoscopic removal is the treatment of choice in addition to antihelminthic medications.

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