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Case Report

Sludge or serpent? Unmasking hepatobiliary ascariasis in a young adult female

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ABSTRACT

Hepatobiliary ascariasis (HBA) is an uncommon but significant cause of biliary obstruction, particularly in regions where Ascaris lumbricoides is endemic. We report a case involving a 21-year-old female who presented with severe epigastric pain and vomiting. Initial evaluation indicated biliary sludge and possible pancreatitis, but her symptoms worsened despite conservative management. Repeat ultrasonography revealed a coiled echogenic structure in the right hepatic and common bile ducts, raising suspicion for parasitic infestation. Magnetic resonance cholangiopancreatography (MRCP) confirmed a tubular filling defect, and endoscopic retrograde cholangiopancreatography (ERCP) enabled extraction of a live Ascaris worm. The patient was treated with albendazole and scheduled for elective cholecystectomy due to persistent gallbladder sludge. This case highlights the importance of considering parasitic infections in the differential diagnosis of biliary symptoms in endemic regions. Early imaging and prompt endoscopic intervention are crucial to prevent complications. Preventive strategies, including improved sanitation, health education, and regular deworming, are essential to reduce the burden and recurrence of ascariasis.

Keywords: Ascariasis, Biliary sludge, Cholecystitis, Parasite, Hepatobiliary

INTRODUCTION

Ascaris lumbricoides is the most prevalent helminthic infection globally, affecting over a billion people, especially in tropical and subtropical regions with poor sanitation.[1] While intestinal ascariasis is often asymptomatic, migration of adult worms into biliary tree can lead to HBA, a rare but potentially life-threatening complication.[2] HBA may present as biliary colic, cholangitis, cholecystitis, or acute pancreatitis, depending on the site and extent of infestation.[3] Differentiating HBA from more common biliary pathologies such as gallstones or sludge is crucial in endemic areas, as management and prognosis differ significantly.[4] This case report details clinical presentation, diagnostic approach, management, and prevention of HBA in young woman from an endemic region.

CASE REPORT

A 21-year-old previously healthy female presented to the emergency department with a five-day history of severe, persistent epigastric pain and recurrent vomiting. She denied fever, jaundice, or previous similar episodes. There was no history of recent travel, abdominal surgery, or known exposure to parasitic infections. Her vital signs

were stable; physical examination revealed epigastric tenderness without guarding, rigidity/rebound tenderness.

Laboratory investigations, including complete blood count, liver function tests, serum amylase, and lipase, were within normal limits. Notably, her eosinophil count was not elevated. Initial abdominal ultrasonography demonstrated gallbladder sludge without evidence of gallstones or biliary duct dilation. A provisional diagnosis of biliary sludge with possible early pancreatitis was made, and patient was admitted for conservative management. She received intravenous fluids, antispasmodics, a third-generation cephalosporin, metronidazole, ursodeoxycholic acid, and a proton pump inhibitor.

Despite initial therapy, her symptoms worsened over the next 48 hours. She developed more severe abdominal pain, persistent nausea, and vomiting. Repeat abdominal ultrasonography revealed a 12.4 cm coiled echogenic linear structure within the right hepatic and common bile ducts, with mild ductal dilation and persistent gallbladder sludge. These findings raised strong suspicion for a parasitic etiology, specifically HBA.

MRCP was performed, confirming a tubular filling defect extending from the right hepatic duct into the common bile duct (as shown in Figure 1 and 2). An urgent ERCP was performed, during which a live Ascaris lumbricoides worm was successfully extracted using a Dormia basket. The patient experienced immediate and significant relief of her symptoms following the procedure.

Post-procedurally, she was treated with a single 400 mg dose of albendazole and discharged in stable condition with outpatient follow-up. Due to persistent gallbladder sludge and to prevent potential recurrence or cholecystitis, elective laparoscopic cholecystectomy was scheduled.

At follow-up visits over the subsequent two months, the patient remained asymptomatic. Repeat ultrasonography showed no evidence of residual parasitic structures or biliary ductal dilation. She was counselled extensively on preventive strategies, including personal hygiene, safe food and water practices, and the importance of community deworming programs.

Figure 1: Filling defect in common bile duct.

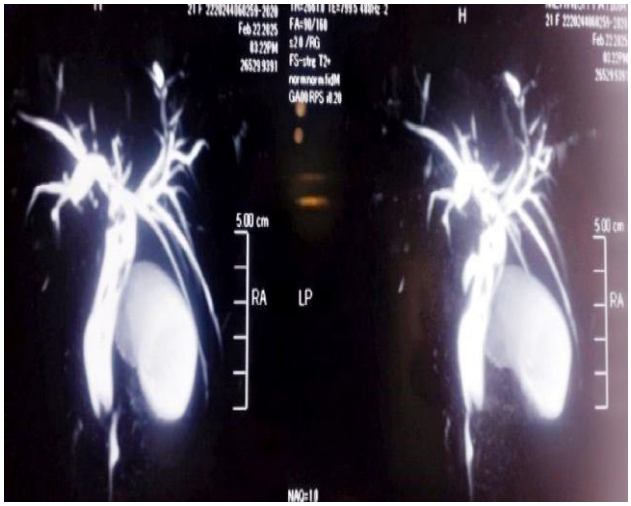

Figure 2: Tubular filling defect in the common bile duct.

DISCUSSION

Epidemiology and pathogenesis

HBA is a significant but often underdiagnosed cause of biliary tract disease in endemic regions such as India, Bangladesh, and parts of Africa and Latin America.[1] In India, HBA may account for up to 10-15% of all cases of biliary disease.[3] The life cycle of Ascaris lumbricoides involves ingestion of embryonated eggs from contaminated food or water, followed by larval migration through the lungs and maturation in the intestines. Adult worms can migrate through the ampulla of Vater into the biliary or pancreatic ducts, often triggered by fever, anesthesia, or bowel manipulation.[4]

Clinical presentation

Clinical manifestations of HBA depend on the site and extent of infestation. Patients may present with biliary colic, obstructive jaundice, acute cholangitis, cholecystitis, or acute pancreatitis.[3] In children and young adults, HBA is more common due to the smaller diameter of the biliary ducts and higher worm burden. Our patient presented with severe epigastric pain and vomiting, mimicking biliary sludge or early pancreatitis, which led to an initial misdiagnosis.

Diagnostic challenges

Diagnosis of HBA can be challenging, particularly in the absence of classic laboratory findings such as eosinophilia or elevated liver enzymes. In up to 30% of cases, laboratory parameters may be normal, as seen in our patient.[5] Imaging plays a crucial role in diagnosis:

Ultrasonography: First-line modality due to availability and cost-effectiveness. Typical findings include a coiled echogenic structure without posterior acoustic shadowing ("strip sign" or "spaghetti sign").[6] However, worms may

be mistaken for biliary sludge or missed altogether in early scans.

MRCP: Provides detailed anatomical information and is useful in equivocal cases, confirming the presence and extent of tubular filling defects.[7]

ERCP: Gold standard for both diagnosis and treatment, allowing direct visualization and removal of the worm.[8] Stool examination for ova and parasites may support the diagnosis but is less useful in acute biliary presentations.

Management

The management of HBA depends on clinical severity and the presence of complications:

Conservative therapy: Includes intravenous fluids, analgesics, antibiotics, and anti-helminthic agents such as albendazole or mebendazole. Most uncomplicated cases respond well to this approach.[5]

Endoscopic intervention: ERCP is indicated for patients with persistent obstruction, cholangitis, or pancreatitis, enabling both diagnosis and extraction of the worm.[8] In our patient, ERCP was crucial for prompt relief and prevention of further complications.

Surgical intervention: Reserved for cases with complications such as perforation, abscess, or failure of endoscopic therapy. Cholecystectomy may be considered in patients with persistent gallbladder pathology or recurrent symptoms.[9]

Post-procedural anti-helminthic therapy is essential to eradicate residual intestinal worms and prevent recurrence. Our patient was treated with albendazole and scheduled for elective cholecystectomy due to persistent gallbladder sludge.

Prognosis

With timely diagnosis and appropriate management, the prognosis of HBA is excellent, with most patients recovering without long-term sequelae. However, delayed or missed diagnosis can lead to serious complications such as cholangitis, pancreatitis, biliary cirrhosis, or hepatolithiasis.[3]

Prevention

Prevention of ascariasis requires a comprehensive public health approach: Improved sanitation: Ensuring access to clean water and proper sewage disposal to reduce soil contamination with Ascaris eggs.[2]

Health education: Promoting hand hygiene, safe food handling, and thorough washing of the fruits and vegetables.

Regular deworming: Mass deworming programs in endemic areas, especially targeting children and high-risk populations, have been shown to significantly reduce the prevalence of ascariasis.[2]

Community awareness: Educating communities about the risks of parasitic infections and the importance of seeking medical attention for persistent abdominal symptoms.

Personal hygiene: Encouraging regular handwashing with soap, especially before meals and after using the toilet.

The patient and her family were counselled on these preventive measures, and she was advised to participate in local deworming initiatives**.**

CONCLUSION

This case highlights the importance of considering hepatobiliary ascariasis in the differential diagnosis of biliary symptoms, especially in patients from endemic regions. Early imaging and timely endoscopic intervention are critical to prevent complications and ensure favorable outcomes. Public health measures focused on sanitation, hygiene, and regular deworming are essential to reduce the burden of ascariasis and prevent recurrence.

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