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Case Report

A Case of Fatal Disseminated Ascariasis: Clinico-Diagnostic Dilemma

Kaushik Stuti¹, Duggal D Shalini^{2*}, Gur R³, Sharma P⁴, Bharara T⁵, Verma J⁶

¹Assistant Professor; Department of Microbiology, Hamdard Institute of Medical Sciences and Research, Delhi, India

²Specialist and Head; Department of Microbiology, Burari Hospital, Government of NCT of Delhi, Kaushik Enclave, Delhi, India

³Senior Specialist and Head of the Department, Department of Microbiology, Dr Baba Saheb Ambedkar Hospital, Rohini, Delhi, India

⁴Assistant Professor, Department of Microbiology, Veer Chandra Singh Garhwali Government Institute of medical Science and Research, Srinagar, Uttarakhand

⁵Assistant Professor, Department of Microbiology, North DMC Medical College and Hindu Rao Hospital, Delhi, India

⁶Department of Medicine, Dr Baba Saheb Ambedkar Hospital, Rohini, Delhi, India

***Corresponding Author:** Shalini Dewan Duggal, MD (Microbiology), Specialist and Head; Department of Microbiology, Burari Hospital, Govt of NCT of Delhi, Kaushik Enclave, Delhi 110084, India;
Email: shaliniduggal2005@rediffmail.com

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Abstract

Ascaris lumbricoides is the most common soil transmitted helminth in India. Hepatobiliary and Pancreatic Ascariasis (HPA) is relatively an uncommon complication. We present a case of fatal HPA with genitourinary involvement. Morbidity and mortality can be minimized by keeping in mind the importance of basic investigations which can lead to a definite diagnosis in such cases for instituting timely treatment.

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Keywords

Ascaris lumbricoides; Hepatopancreatic Ascariasis; Renal Ascariasis; Cholangiocarcinoma

Abbreviations

HPA: Hepatobiliary and Pancreatic Ascariasis

Introduction

Ascaris lumbricoides is endemic in tropical areas of the world with around 819 million people infected worldwide. It is the most common soil transmitted helminth in India despite massive deworming programs (prevalence of 0.6% in Lucknow, 91% in Andhra Pradesh) [1]. Clinical manifestations of Ascariasis arise due to effect of larval migration, adult worms, nutritional deficiencies and host immunity. They range from asymptomatic (most common) to severe forms. Hepatobiliary and Pancreatic Ascariasis (HPA) is an uncommon complication in high worm burden and poses diagnostic dilemma. HPA is relatively uncommon in Delhi. We present a case of fatal HPA with genitourinary involvement; diagnosed radiologically as cholangiocarcinoma.

Case Report

A 75-year-old diabetic and hypertensive male presented to the emergency department with vomiting, decreased appetite, weight loss, yellowish discoloration of urine and clay colored stools for one month. He was non-smoker, non-alcoholic. On examination, icterus was present, abdomen was soft and tender with moderate hepatomegaly. Liver Function Tests (LFTs) performed on two occasions prior to admission showed elevating total bilirubin (6.25 mg/dl to 19.6 mg/dl in two weeks) and alkaline phosphatase levels (2908 IU/L). Blood investigations on admission revealed anaemia, leukocytosis, markedly deranged LFTs (Total bilirubin-26.6 mg/dl, aspartate and alanine transaminases and serum lipase were 147 IU/L, 114 IU/L and 1948 U/L respectively; mildly deranged Kidney Function Tests (KFT)- serum urea 55 mg/dl; and creatinine levels 1.5 mg/dl. Tests for hepatitis B, hepatitis C and HIV were negative. Ultrasound whole abdomen revealed bilobar intrahepatic biliary radicle dilatation, minimal vascularity with multiple calculi embedded in wall of the gall bladder and compressing common bile duct. In addition, Contrast Enhanced Computed Tomography (CECT) indicated acute pancreatitis with significant extra-pancreatic inflammatory changes and multiple retroperitoneal nodes, edema of gall bladder with thickened wall (Fig. 1). Ultrasound of Kidney

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Urinary Bladder (KUB) region showed cortical cysts in both kidneys, left hydro-uretero-nephrosis, curvilinear echogenic shadows of 3 cm and 2.5 cm suggestive of vesical calculi at the vesico-uretric junction and mild prostatic enlargement.

Presumptive diagnosis of acute pancreatitis with obstructive jaundice due to cholangiocarcinoma and obstructive uropathy was made. On day 12 of his stay in hospital, the patient vomited out four dead worms. Two of these were sent to the Microbiology department for identification. They were unsegmented round worms, 25-30 cm in size with pointed ends, tail end was straight with characteristic vulvar waist, suggestive of female adult of *Ascaris lumbricoides* (Fig. 2). A cola colored urine specimen was received for routine microscopy which showed presence of about five pus cells and six granular casts per high power field, but no ova or parasites. Laboratory investigations done during his stay did not show any significant improvement in the LFTs and KFTs, warranting Magnetic Resonance Cholangiopancreatography from a higher center, so the patient was transferred. However, the patient succumbed before any surgical intervention could be undertaken.

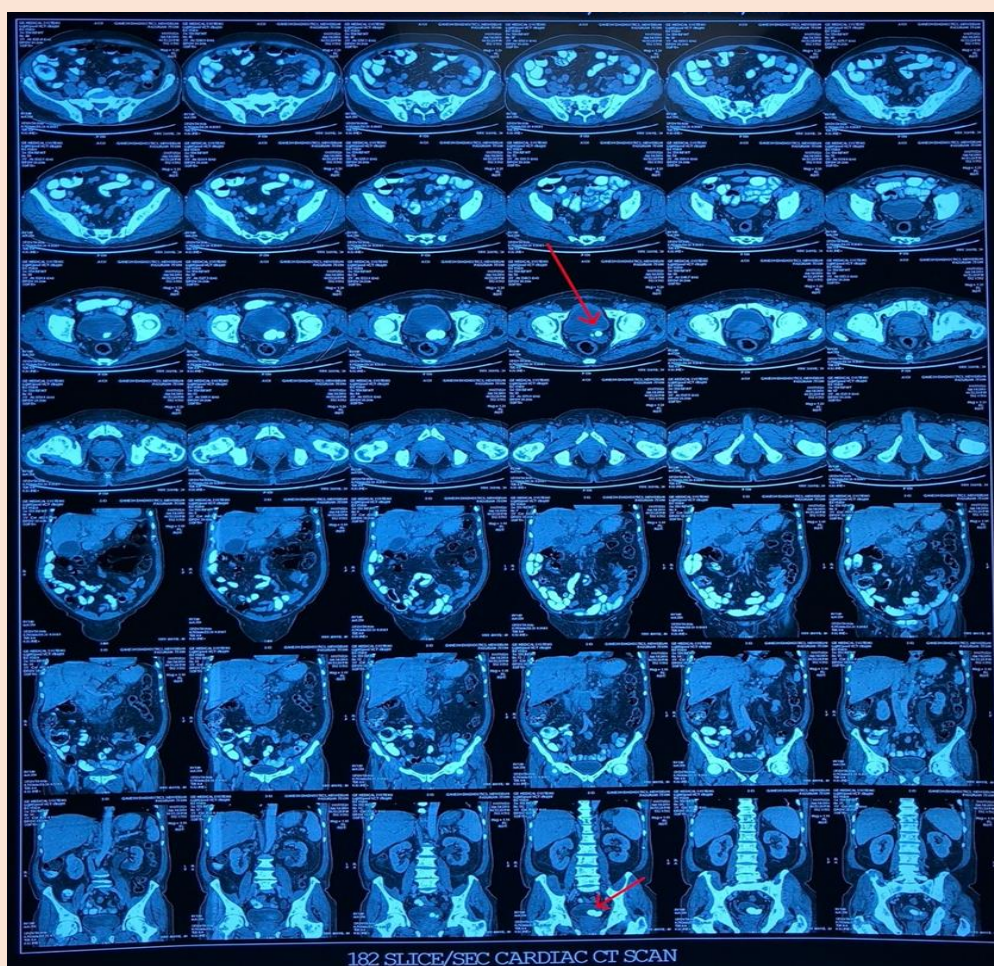


Figure 1: CECT whole abdomen showing curvilinear echogenic shadows of 3 cm and 2.5 cm in lumen of the urinary bladder embedded at the vesico-ureteric junction.



Figure 2: Two adult females of *Ascaris Lumbricoides* vomited by the patient, 25-30 cm in size with the characteristic vulvar waist.

Discussion

Humans are infected by ingestion of embryonated eggs of *Ascaris lumbricoides* in contaminated food and water. Infections are mostly asymptomatic but, with heavy worm burden, complicated syndromes like *Ascaris* pneumonia, intestinal obstruction, Appendicitis, peritoneal and gastric ascariasis, HPA, stunted growth may manifest. Only a handful cases of Renal and genitourinary ascariasis have been described [2,3].

Cases of HPA have been reported from all across the globe (Syria, Saudi Arabia, Philippines, South Africa) and frequently from high prevalence states of Kashmir, Tamil Nadu, Assam and West Bengal in India [4]. Extensive reviews by Khuroo, et al., mention 23% cases of acute pancreatitis being attributed to HPA with a mortality rate of 3% [5]. Delhi has a low prevalence of Ascariasis (1.4%). A study conducted in Delhi included 15 cases (all recovered) over a span of 18 months [6]. Barring this study, only three cases of HPA have been reported from Delhi in the last five years (one incidental on autopsy) [7-9].

HPA is more common in adult females (female: male= 3:1). A widened ampullary orifice due to hormonal changes or surgical interventions involving the common bile duct is a predisposing factor. Adult worm resides in jejunum but in heavy infestation, it moves toward duodenum and ampulla of Vater to invade the common bile duct, hepatic duct, gall bladder, pancreatic duct or the cystic duct. The condition may be asymptomatic if the worms exit but symptomatic if they are trapped and die. The dead worms act as a nidus for stone formation or cause stasis due to dysmotility leading to varied presentations [10]. Biliary sludge containing cholesterol crystals, mucin and calcium bilirubinate granules provide an appropriate environment. This patient presented with calculi in the gall bladder and urinary bladder and vomited dead worms. Bile concentration upto 10% may stimulate *Ascaris* eggs to hatch and promote larval migration, whereas concentrations beyond 20% may impair the same [11]. Mortality may result due to necrotizing pancreatitis, the likely cause of death in this patient.

HPA poses a diagnostic dilemma in areas of low prevalence due to its deceptive presentation. The worms may move out of the ducts within 24 hours of inducing pancreatic or biliary symptoms. Diagnosis is aided by ultrasound showing echogenic curvilinear shadows with writhing movements along with dilatation of involved duct; ERCP shows filling defects with smooth, long linear characteristics; or laboratory tests including CBC, LFTs, serum amylase and lipase levels. Stool routine microscopy may show embryonated and unembryonated eggs, adult worms may be visualized in vomitus/other body orifices or during postmortem examination. Symptomatic treatment is warranted in cases of HPA along with an antihelminthic (mebendazole, albendazole, ivermectin and pyrantel pamoate) to paralyse the worms causing their expulsion. Endotherapy is indicated if there is no relief after six weeks, whereby dead worms are endoscopically removed.

Cases of biliary ascariasis associated with cholangiocarcinoma have been reported in literature [12]. Cholangiocarcinoma was considered a differential diagnosis in this case. It peaks in the seventh decade of life with pruritus, right upper quadrant pain, weight loss, fever, clay colored stools and dark urine. The patient collapsed before MRCP could be performed, hence commenting on the presence/absence of carcinoma will be difficult.

An unresolved aspect was the important finding of deranged KFTs along with curvilinear echogenic shadows at the vesico-uretric junction. This could indicate obstructive uropathy due to vesicle calculi formation, a foreign body, or parasites like *Diectophyma renale*, *Schistosoma hematobium* and *Ascaris lumbricoides*. Another explanation can be the arrested development of their larvae in aberrant sites. Involvement of the renal system is very rare in ascariasis; two reports of *Ascaris* induced urinary retention and four cases of acute interstitial nephritis have been reported emphasizing the role of genetic susceptibility in aberrant presentations [2]. *Ascaris* burden has also been attributed to susceptibility of specific genes on Chromosome 1 and 13.

This case exemplifies an unusual presentation of Ascariasis and suggests that it should be considered as a differential diagnosis in such clinical manifestations, especially in an endemic country like India.

Conclusion

Ascariasis in humans may have varied manifestations depending on the social, demographic and genetic factors. Morbidity and mortality can be minimized by keeping in mind the importance of basic laboratory investigations (stool microscopy) which can lead to a definite diagnosis by supplementing radiological modalities in such cases for instituting timely treatment.

Conflict of Interest

The authors declare that they have no conflict of interest.

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