CASE REPORT

BILIARY ASCARIASIS: A WANDERING WORM

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Abstract

Ascaris lumbricoides infection usually results in mild gastrointestinal symptoms. However, heavy worm load infections can cause major complications. In one-third of the latter cases, the worms enter the bile duct causing cholangitis, biliary colic and acute pancreatitis. We describe an eleven-year-old Indian boy who presented with high grade fever, vomiting of worms, jaundice and right hypochondrial pain. Examination revealed an ill, malnourished and jaundiced child. His abdomen was tender with positive Murphy's sign and hepatomegaly. Urgent ultrasound showed hepatomegaly with dilated intrahepatic and common bile ducts (CBD). There were tubular echogenic filling defects with central sonolucency along the long axis of the CBD. He was fasted, started on intravenous fluids and broad spectrum antibiotics. As there was no improvement after 24 hours, endoscopic retrograde cholangio-pancreaticography (ERCP) was performed: it showed worms in the CBD. A stent was inserted to decompress the bile ducts and to ensure biliary drainage. The patient improved, the fever settled, jaundice resolved, serum amylase and transaminase levels normalised. It was planned to remove the stent at a later date.

Key words: *Ascaris; biliary; paediatric*

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Introduction

Ascariasis is a human helminthic infection caused by the nematode *Ascaris lumbricoides*.[1] It is a soil transmitted disease. The life span of the adult worm is 16 to 18 months. The female worm can release millions of eggs which require moist soil for embryonation. The disease is transmitted by swallowing the eggs e.g. via soil to fingers by toys, raw vegetables or water contaminated with human faeces. The eggs hatch in the small intestine liberating larvae which migrate via the circulation to the lungs. There they penetrate the alveolar walls to enter the epiglottis and are swallowed to reach their final habitat. The time from swallowing to maturation is approximately 60 to 75 days.

Ascariasis is globally distributed, affecting more than 1.4 billion people.[1] The majority of infections occur in the developing countries and the incidence is increasing due to world travel and population migration. Transmission occurs predominantly in the tropics and rural areas with dense population, poor sanitation, poor personal hygiene and low levels of health education. Persons infected are usually asymptomatic or may only have mild gastrointestinal symptoms, e.g. mild colic or occasional worms passed out in the stool or vomitus. Major complications are restricted to heavy worm load infections with an annual estimate of 1.2 million cases world-

wide and 20,000 deaths in epidemic areas.[1] In one-third of cases, the worm enters the bile duct and causes cholangitis, biliary colic and acute pancreatitis.[1-5] We describe a patient with acute cholangitis and pancreatitis due to biliary ascariasis.

Case report

An eleven-year-old Indian boy presented with a three-day history of fever, chills and rigors associated with nausea, vomiting, anorexia, jaundice, dark urine and right hypochondrial pain. He had two episodes of having vomited worms. He swam frequently in a river discharging sewage behind his house. He is the fourth of eleven siblings who are from a poor socio-economic background. He has stopped schooling and helps at their vegetable farm which has no toilet, water or electricity. Both parents and siblings also have a history of passing worms in their stools.

Physical examination revealed an ill, malnourished boy (weight, height and head circumference below the third centile) with high grade fever, pallor and jaundice. He did not have clubbing, spider naevi or palmar erythema. Blood pressure was 120/80 mmHg, heart rate 100/min and respiratory rate 40/min. His abdomen was distended and tender with positive Murphy's sign. There was a firm enlarged tender liver of 10cm below the right subcostal margin. There was no palpable spleen or ascites. Urgent ultrasound showed an enlarged liver with dilated intrahepatic and common bile ducts (CBD). The gallbladder wall was thickened. There were tubular echogenic filling defects with central sonolucency along the long axis of the CBD. A diagnosis of acute cholangitis and pancreatitis due to ascariasis was made.

Blood investigations showed a hypochromic, microcytic anaemia with neutrophilia (haemoglobin 7g%, haematocrit 28%, mean corpuscular volume 70fl, total white cell count $24x10^3$). Serum amylase was markedly raised (1262 U/l) (normal 8–85 U/l). Liver function tests showed a mixed hyperbilirubinaemia (total bilirubin 71 μmol/l, unconjugated 40 μmol/l, conjugated 31μmol/l), hypoalbuminaemia (27g/l) and raised liver enzymes (alanine transaminase 213 U/l, aspartate transaminase 154 U/l). Prothrombin time was prolonged at 16.2s (control 12s). Stool examination showed presence of ascaris worms and eggs. He was fasted, started on intravenous fluids and a naso-gastric tube was inserted for aspiration of gastric contents. He was given intravenous broad spectrum antibiotics (cefuroxime, netromycin and metronidazole), vitamin K, prophylactic cimetidine and oral paracetamol. Nasogastric losses were replaced with intravenous Hartmann's solution. As he did not improve clinically after 24 hours, endoscopic retrograde cholangio-pancreaticography (ERCP) was performed under general anaesthesia. Multiple worms were seen in the stomach and duodenum (ascaris and trichuris). The ampulla was patulous and the CBD was dilated with a worm inside (Fig. 1). A plastic stent was inserted to decompress the bile ducts and ensure biliary drainage. Post-ERCP, the patient developed ileus due to hypokalaemia which was corrected with potassium in the intravenous drip. There was also pleural effusion and ascites which were due to hypoalbuminaemia; the latter was correct-

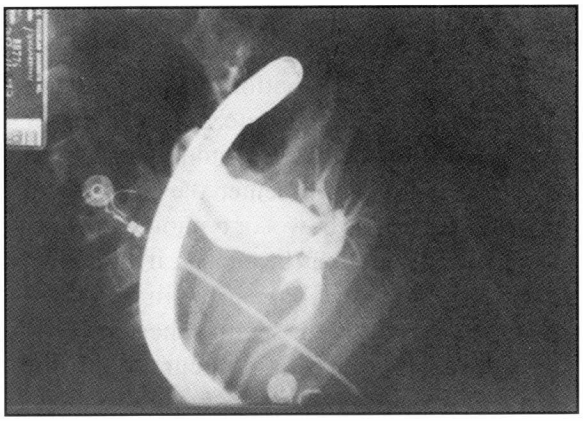

Fig. 1. Dilated common bile duct with long tubular filling defect (ascaris worm) seen during ERCP.

was corrected with intravenous albumin 20% infusions. The patient improved with the fever settling, nasogastric losses reduced to minimal, jaundice resolved and serum amylase and transaminases normalised. Blood culture was negative. Oral feeding was started on day 10. The patient was discharged after treatment with repeated courses of oral albendazole. Albendazole was also prescribed to the other family members. Advice on personal hygiene and sanitation was given and the family was referred to the local social welfare department for further follow-up and provision of proper water and toilet facilities. We planned to remove the stent at a later date. However, the patient defaulted follow up.

Discussion

Ascariasis is known to cause linear growth stunting, reduced cognition and protein energy malnutrition.[1] A heavy worm load of 13–40 worms can cause 4g protein loss per day in a daily diet consisting of 35–50g protein. Catch-up growth is seen after its treatment.[1] Although usually a mild disease, on occasions it may be life-threatening.

Pulmonary ascariasis (Loeffler's syndrome) is caused by waves of larval migration and maturation.[1] This can lead to fever, cough, wheeze, haemoptysis and urticaria. Chest X-rays may show alveolar shadows and consolidation. There may be eosinophilia.

Heavy worm load infection is the cause of intestinal obstruction, acute appendicitis or appendicular perforation. Peritoneal ascariasis can occur if the worms enter the peritoneum through gangrenous bowel or typhoid/amoebic/tubercular ulcers. It may be fatal or, if the patient survives, this will result in chronic granulomatous peritonitis with adhesions.[1]

Hepatobiliary ascariasis[1-5] occurs when the worm in the duodenum enters and causes blockage to the Ampulla of Vater. The worm can enter the bile duct, gallbladder and pancreatic duct causing biliary colic, acalculous cholangitis, acute cholecystitis, acute pancreatitis and hepatic abscesses. Intrahepatic lithiasis is frequently associated with nucleation of ascaris eggs. Periampullary carcinoma and extrahepatic cholangiocarcinoma are also associated with ascariasis.

The finding on abdominal ultrasonography of tubular echogenic filling defects with central sonolucency is typical and diagnostic of biliary ascariasis.[1,2,5]

Our patient developed life-threatening acute cholangitis and pancreatitis due to the heavy ascaris infection. Like most worm-infected children, he also had a mixed worm infection with *trichuris trichura*.[1] The cholangitis is treated with broad spectrum intravenous antibiotics, intravenous fluid replacement, fasting and gut rest/decompression, analgesics/antipyretics and support for a decompensated liver with correction of the coagulopathy and electrolyte imbalance, intravenous albumin 20%, maintaining normoglycaemia, avoidance of hepatotoxic drugs and maintaining a normal urine output. Antihelminth agents (e.g. albendazole) help to paralyse the worms which are then expelled by peristalsis. This will depopulate the number of worms in the duodenum and will encourage the worm in the biliary system to move out into the duodenum, hence unblocking the biliary system.

Failure of conservative medical therapy indicates the necessity of an early, emergency ERCP to decompress the biliary system and ensure biliary drainage.[2,3,6,7] Hence, it is important that these children are transferred to centres where these facilities are available. In this case, a stent was placed for biliary decompression. Sometimes, the worm needs to be removed with an endoscopic Dormia basket.[1-5]

Several drugs are available and effective for treatment of ascariasis. Drugs of choice include albendazole (Zentel®), mebendazole (Vermox®), pyrantel pamoate (Combantrin®), levamisole and piperazine salts.[1] Albendazole is suitable for

of children with albendazole improves nutritional status. The same patient may need repeated courses of treatment in order to eradicate heavy worm infections.

The importance of prevention and control[1] of this common but potentially fatal disease needs to be emphasised. On-going basic health education on personal hygiene (handwashing with soap and clean water, foot wear, trimming of finger/toe nails), provision of safe water supply, proper disposal of stool, environmental sanitation and properly cooked food need to be reinforced.

Conclusion

Ascariasis can cause mild but chronic diseases affecting the general health of the population as well as life-threatening complications which need urgent treatment. Effective prevention control programmes will curb its prevalence.

Acknowledgement

We wish to thank the Director General, Ministry of Health for permission to publish this paper.

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