

You Traveled the World and All You Got Was Ascariasis



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ANNALS CASE

A 3-year-old girl receiving isoniazid for latent tuberculosis is referred to the emergency department (ED) for elevated liver function test results.¹ Besides recently emigrating from Honduras, the patient's history and physical examination results are unremarkable. Abdominal ultrasonography, performed to evaluate the liver, found a 10-cm×4-mm serpiginous structure in the small intestine, aka a worm! The ascariasis wasn't even in the liver. The transaminitis was from the isoniazid, and finding a huge worm in the intestines was incidental in this asymptomatic patient! Now, I know what you're thinking: do all my patients have asymptomatic worms?

Although uncommon in the United States, ascariasis remains a common helminth infection, with a prevalence of nearly 4 million people. The prevalence of ascariasis increases in lower socioeconomic and immigrant populations. Outside of the United States—wow!—ascariasis is ubiquitous in the Indian subcontinent, China, Africa, and Latin America, with a worldwide prevalence of 25%. That's an estimated 1.4 billion infected through the fecal-oral route.² Although our patient in the *Annals* case was asymptomatic, there are a few symptomatic ascariasis presentations that an emergency physician must recognize. Diagnosis first requires awareness and suspicion for the infection. Fortunately, treatment is quick and simple, with

an excellent prognosis. In this review, we will discuss high-yield and clinically relevant presentations of ascariasis.

CASE 1: LOEFFLER'S SYNDROME

A 3-year-old boy presents with 4 days of fever, cough, wheezing, and watery nonbloody diarrhea. A right-sided peripheral infiltrate on chest radiograph seals the obvious diagnosis, pneumonia. Antibiotics, discharge, and next patient...right? But what about the travel history? Did he visit rural Mexico 3 weeks before?

This presentation is classic for Loeffler's syndrome, an eosinophilic, pulmonary inflammatory reaction to migration of *Ascaris lumbricoides*. When the larva migrates to the lungs as part of its life cycle, it can cause an inflammatory reaction that mimics pneumonia. Symptoms typically occur 2 weeks after ingestion of the eggs and last 2 to 3 weeks.³ It's usually self-limited, which can lead to the false conclusion that the antibiotics cured the "pneumonia." Recognizing this presentation—transient migratory infiltrates on chest radiograph and peripheral eosinophilia—could be the only clue for a persistent underlying parasitic infection. Wheezing similar to that caused by asthma can also occur and typically responds to systemic corticosteroids.⁴ Stool studies are generally unhelpful with Loeffler's syndrome. The studies occur early in the course of illness, and the parasites typically take 40 days after the pulmonary symptoms to enter the intestines and produce eggs. Anthelmintic treatment during pulmonary symptoms may worsen the pulmonary inflammatory response. Patients should be advised to follow up in 2 months for confirmatory stool studies and treatment. This reaction, though, isn't limited to ascaris because other parasites and some medications can cause it as well. So now your asymptomatic patient, pneumonia patient, and wheezing patient all have worms.

Identification of this disease, thanks to your wide differential and thorough history taking, can prevent further complications from the ascaris infection.

CASE 2: HEPATOBILIARY ASCARIASIS

A 20-year-old woman presents with 4 to 5 days of right upper quadrant pain, worse after meals, with recent vomiting and low-grade fever. Her examination result is significant for right upper quadrant tenderness and guarding. Ultrasonography shows a thickened gallbladder wall, sludge, and a positive sonographic Murphy's sign, but no gallstones. Straightforward acalculous cholecystitis? But with further history taking, the patient reports recently returning from a Peace Corps mission in Syria...and that something may have been wiggling in her vomit.

Impressively, one third of hepatobiliary disease in endemic areas may occur as a result of ascariasis.² Although the ascarid larvae mostly reside in the jejunum, as worm load increases, so too does migration into the duodenum and even into the ampulla of Vater. Biliary involvement can also increase as a result of other factors such as pregnancy, which causes ampulla relaxation, and any previous biliary tree instrumentation. And, oh man, do these worms wreak havoc on the hepatobiliary system! Intermittent ampulla invasion can cause biliary colic. The worms can also be a nidus for gallstone formation. Ascarids can invade the bile ducts (rarely the gallbladder), leading to inflammation, obstruction, and acalculous cholecystitis. They can obstruct the ampulla of Vater or the pancreatic duct, leading to acute pancreatitis, even potentially fatal hemorrhagic or necrotizing pancreatitis. Intrahepatic duct invasion and obstruction can lead to acute cholangitis, which, like bacterial cholangitis, carries a high mortality. Dead ascarids in the intrahepatic ducts can serve as a nidus for infection and lead to hepatic abscesses. Even when they die they can cause problems! Recurrent pyogenic cholangitis can occur from changes resulting from biliary and intrahepatic tree inflammation and infection, which can cause abscesses, biliary cirrhosis, and end-stage liver failure.²

Ultrasonography commonly reveals hepatobiliary ascariasis by visualizing ascarids in the biliary tree (Figures 1 and 2). When viewed longitudinally, an ascarid appears as 4 parallel lines separated by 3 anechoic bands representing the alimentary tract of the worm ("inner-tube sign"). In cross section, it looks like a "target sign" surrounded by the intestinal lumen. In hepatobiliary ascariasis, the inner-tube sign can be found when the ascarid is in the gallbladder or common bile duct. Otherwise, the "strip sign" can be seen as a thin nonshadowing strip or the "spaghetti sign" as an overlapping longitudinal interface in the main bile duct.⁵



Figure 1. Ascaris worm in the dilated biliary system, as seen at the left. It appears as a tubular structure with echogenic walls and a hypoechoic central line. Posterior to the worm is a heterogeneous area that might be a hepatic abscess. Photo included with permission of Leandro Fernandez, MD, and SonoWorld (https://sonoworld.com/CaseDetails/Biliary_and_Hepatic_Ascariasis_with_Cholangitis_and_Hepatic_Abscess.aspx?ModuleCategoryId=639).

If not found on ultrasonography, they may surprise you on magnetic resonance imaging, endoscopic retrograde cholangiopancreatography, or direct surgical visualization. Invasive procedures are indicated for obstructive findings, abscess or cholangitis (each of which presents increased septicemia risks), or removal of dead ascarids.² Nonetheless, invasive procedures may be circumvented by prompt ascariasis diagnosis because medical anthelmintic therapy alone can often be definitively curative.

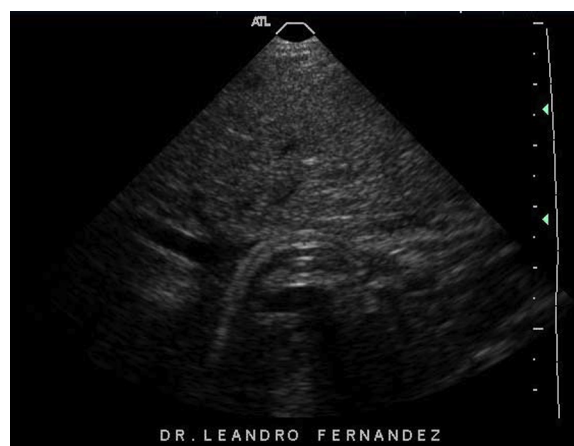


Figure 2. The ascaris worm, extending from the right lobe to the porta hepatis, is visualized by a double hyperechoic line with a hypoechoic center. Photo included with permission of Dr. Leandro Fernandez and SonoWorld (https://sonoworld.com/CaseDetails/Biliary_and_Hepatic_Ascariasis_with_Cholangitis_and_Hepatic_Abscess.aspx?ModuleCategoryId=639).

CASE 3: INTESTINAL ASCARIASIS

A 32-year-old man with months of intermittent gastrointestinal symptoms presents with 3 days of abdominal pain and vomiting. He has a distended abdomen with diffuse tenderness. Laboratory results are unremarkable, but an abdominal radiograph shows air-fluid levels and a round opacity occluding the distal small intestine. Ready to close the differential on small bowel obstruction, you begin fluid resuscitation, prepare for a computed tomography (CT) scan, and consult your surgical colleagues...and then you discover the patient recently returned from 6 months in Bangladesh.

Intestinal obstruction is the most common complication of ascariasis. Besides the obvious mechanical obstruction by an entangled ball of worms, ascariasis can also cause obstruction and inflammation through a spasm-inducing neurotoxin. It can even cause volvulus or intussusception.⁴ Children tend to be more susceptible because of their smaller intestinal diameter.

CT scan, as well as ultrasonography, can help diagnose intestinal obstruction. Although the evidence is limited, diatrizoate per os can also be a therapeutic agent. Its hyperosmolarity can result in an influx of luminal fluid, which can separate and lubricate the worms, as well as dehydrate and shrink them. It alone may help alleviate the obstruction.⁴ Surgical intervention should be considered if there are signs of strangulation, perforation, peritonitis, complete obstruction, or presence of intraperitoneal free air. If immediate surgical management is not indicated, an observation period for resolution is advisable. Anthelmintics should not be administered while obstructive symptoms are present because death or paralysis of the worms may worsen the obstruction.⁵ Once the obstruction is resolved, anthelmintic therapy should be initiated, with follow-up treatment.

Yep, so the patient with bowel obstruction has worms too.

ANTHELMINTIC TREATMENT

The benzimidazoles albendazole and mebendazole are the mainstays of treatment for ascariasis and have minimal adverse effects for the regimens recommended. Albendazole has ovicidal and larvicidal effects, with systemic activity. Absorption is improved with a fatty meal, but the drug should be ingested on an empty stomach for intestinal infections such as ascariasis. In most cases, only a single dose is indicated. Mebendazole has poor absorption, is used specifically for intestinal parasites, and is unaffected by last oral intake. However, it is currently not available in the

United States. A 3-day course is recommended. Mebendazole is not recommended in pregnancy, and seizures have been reported in children younger than 2 years.⁶ You may want to warn patients that they will pass dead worms during the next week. If a sample of the worms is available in the ED, it should be sent for pathologic examination. As always, hand washing and hygiene are important because they may prevent reinfection. Redosing of anthelmintics is recommended for continued symptoms. Outpatient screening for reinfection should occur in 2 to 3 months because, as you now know, untreated ascariasis is often asymptomatic.

CONCLUSION

Ascariasis is an extremely common infection worldwide and may present to an ED near you even in the good ol' US of A. At high risk are immunocompromised patients, immigrants from ascariasis-endemic areas (almost everywhere else), and low socioeconomic status. Take a good travel history because identification and treatment of this parasite can prevent unnecessary testing and invasive procedures.

To follow up on our initial case, the child had a great outcome after treatment with albendazole and worm passage (which we hope is less uncomfortable than it sounds).

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