Laparoscopic appearance of fasciola hepatica infection

Guy W. Neff, MD, Rajani V. Dinavahi, MD, VeEtta Chase, MD, K. Rajender Reddy, MD

Fasciola hepatica (FH) infection is endemic in South America, the Caribbean, Europe, and Asia. The population of southern Florida, with proximity to the Caribbean and South America, may be at risk. FH infection has acute and chronic stages. In the acute stage, the patient presents with fever, hepatomegaly, and abdominal pain. In the chronic stage, the trematode can invade the bile ducts resulting in biliary obstruction, hemobilia, and liver abscesses.1,2 A high index of suspicion must be maintained to make the diagnosis of FH infection. This is a case of FH infection in a patient from southern Florida who ingested watercress. The diagnosis was based on clinical suspicion, positive serology, laparoscopic appearance, and histologic features.

CASE REPORT

The patient, a 51-year-old man, was in good health until 6 months before admission when he developed a nasal skin infection. The lesion soon progressed into an
abscess and he was treated with Keflex. Two weeks later he developed an erythematous facial skin rash and an elevated temperature. Treatment with antibiotics was resumed and the facial skin rash and fever resolved. One week after the completion of the course of antibiotics he complained of right upper quadrant abdominal pain radiating along his right flank and extending to his back, as well as a return of the low-grade fever.

Examination revealed a right hepatic lobe palpable just below the right costal margin. The spleen was not palpable. The dermatologic examination was normal with no spider angiomata. Examination of the extremities revealed no edema or asterixis.

Laboratory data were as follows: hemoglobin 13.5 g/dL, eosinophils 30%, serum albumin 3.9 g/dL (normal: 3.9-5.0 g/dL), total bilirubin 0.6 mg/dL (0.2-1.3 mg/dL), aspartate aminotransferase 46 IU/L (15-46 IU/L), alanine aminotransferase 105 IU/L (21-72 IU/L), alkaline phosphatase 162 IU/L (38-126 IU/L), amebiasis antibody of 44 U/ML (0-50), Echinococcus antibody negative, Toxocara antibody negative. Stool studies were negative for ova and parasites. Laboratory studies for acute A and B hepatitis and antibodies to hepatitis C were negative. Blood cultures were negative.

US revealed multiple hypoechoic areas in both lobes of the liver. Abdominal CT disclosed lesions in the right hepatic lobe that were heterogeneous and both solid and cystic in appearance. Over the next few months, repeated CT identified lesions in the left lobe with a change in the size and number of right lobe lesions. Abdominal laparoscopy revealed focal serpiginous, yellowish colored lesions on the surface of both lobes of the liver (Fig. 1). The lesions were palpated with a probe and were firm. Multiple liver biopsy specimens were taken and revealed “granulomatoid” abscess formation with central necrosis, cellular debris, Charcot-Leyden crystals, and eosinophils surrounded by an inflammatory infiltrate. No malignant cells were identified. Endoscopic retrograde cholangiography was obtained to rule out biliary tract disease and appeared to be normal.

Visceral larva migrans was the most plausible explanation for this clinical picture. Treatment was initiated with albendazole 400 mg twice a day for 28 days. After 2 courses of this regimen interrupted by a 2-week drug-free period, the patient’s symptoms improved but did not completely resolve.

At this point serologic studies for FH were performed by Professor Jean Dupouy-Camet of the Service de Parasitologie.

Figure 1. A-D, Laparoscopic views of yellow serpiginous lesions on the surface of the liver as identified by arrows.
tologie-Mycologie at the Centre Hospitalo-Universitaire Cochin, Port-Royal Universite Rene Descartes, Paris, France (The Centers for Disease Control in the United States does not perform serology testing for FH). The serology results were as follows: indirect immunofluorescence was 1:40 (positive greater than 1:40), indirect hemagglutinin was 1:640 (positive greater than 1:320), and the immunoelectrophoresis was positive with an “Arc 2” pattern, typically highly specific for FH.

At a follow-up examination, the patient confessed to recent ingestion of a large amount of watercress. He stated that he did so in response to a television news report that extolled the health benefits of watercress. He also admitted to having multiple salads in the ensuing months that included water chestnuts or watercress. However, he had not traveled outside of the United States in the last 20 years.

Triclabendazole, an investigational new drug from the Food and Drug Administration, was given in 2 doses, 24 hours apart. Triclabendazole is currently available in Europe; however, it is available only for compassionate use in the United States.

After treatment, the eosinophilia resolved, the hepatic biochemical profile returned to normal, and the patient became asymptomatic. A CT approximately 15 months after the initial scan, but 6 months after therapy with triclabendazole, revealed a considerable decrease in the size of the liver lesions, which were then of a subtle nature. Follow-up serology 6 months after therapy was negative by indirect immunofluorescence and immunoelectrophoresis and was 1:160 by hemaggulitnatin (threshold 1:320).

**DISCUSSION**

The infection caused by *Fasciola hepatica* (liver fluke), a leaf-shaped trematode, is primarily a zoonotic disease acquired by ingesting contaminated watercress or water.1,2 A snail is an intermediate host, whereas humans are accidental hosts and other herbivores, such as sheep, goats, and cattle, serve as primary hosts. Most human cases occur in Latin America, Mediterranean countries, England, Germany, Poland, Russia, East and South Africa, Southeast Asia, and the Caribbean.1,2 FH infection is rare in the United States but should be considered in patients who have lived in or traveled to endemic areas.

The life cycle of *Fasciola hepatica* begins with the ingestion of the encysted metacercariae, which are on aquatic plants such as wild watercress.1,2 Once the metacercariae are in the intestines they penetrate and traverse the intestinal wall and enter the peritoneal cavity. The metacercariae then migrate to the liver and penetrate the capsule. After entering the hepatic parenchyma and eating their way into the biliary radicles, an inflammatory response ensues resulting in bile duct destruction and symptoms. They also mature in the biliary system and begin to pass eggs. The prolonged tissue migration through host tissues may be associated with a long period of symptoms.

The clinical manifestations of FH infection can be classified into 3 groups: acute, chronic, and asymptomatic. In acute cases of FH infection there is a classic triad of prolonged fever, hepatomegaly, and/or abdominal pain.1,2 During the early period of migration within the liver, hemobilia may result in anemia.3 Other organ systems can be involved, including the heart and lungs with resultant pleural effusions, electrocardiography abnormalities, and even seizures.1 The chronic disease state may include any of the following: extrahepatic biliary duct obstruction with colic, jaundice, cholangitis, pancreatitis, hemobilia, hemoperitoneum, and possibly hepatic masses. The time period for advancement to the chronic stage has been reported to be between 3 and 9 years.2 Although the majority of the patients with FH infection are asymptomatic, 18% of patients diagnosed with FH infection have been reported to be asymptomatic.4

The diagnosis of FH should be considered in patients with one or more of the following features: history of watercress ingestion, eosinophilia, fever of unknown origin, granulomatous hepatitis, biliary colic, or cholangitis with a normal US and a family history of fascioliasis. Identifying parasitic eggs in the feces is important in the early course of the disease although they often are scanty or absent. Repeated stool collections are needed, and pretreatment with anti parasitic drugs may obscure results. Eosinophilia is an early indication of FH infection.

Serologic assays are the mainstay of diagnosis and allow diagnosis in the acute stage, even before the parasite eggs can be identified in the feces. The various serologic tests include complement fixation, immunofluorescence, counterelectrophoresis, double diffusion, and indirect hemaggulitnatin. Although extremely sensitive, they lack optimal specificity and may cross-react with other parasitic infections, such as echinococcosis, and therefore the clinical picture and radiologic studies serve as supporting evidence for a specific diagnosis of FH infection. Enzyme-linked immunoabsorbent assay (ELISA) testing for FH has a sensitivity of 98%,5 and an indirect hemaggulitnatin test, when greater than 1:160, has a sensitivity of greater than 90%.6

CT is the imaging study of choice and may demonstrate 2 identifiable types of lesions. The first is a hypodense nodular area that can be singular or multiple resulting from the deposition of the parasite. The second is a tunnel-like branching hypodensity resulting from the migration of the parasite through the liver.1
The characteristic laparoscopic appearance of the FH is yellowish-white nodules in a serpiginous or cord-like configuration, as seen in our patient.\textsuperscript{7,8} Other reports have suggested that the hepatic nodules may have a ribbed or vermiform configuration.\textsuperscript{9}

Histology in FH infection demonstrates granulomas and must be differentiated from other well-recognized causes including tuberculosis, sarcoidosis, Q fever, Whipple's disease, Crohn’s disease, non-Hodgkin’s lymphoma, scleroderma, and toxoplasmosis.\textsuperscript{10} The most typical histologic finding in FH infection is central necrosis, cellular debris, and Charcot-Leyden crystals encompassed by eosinophils and inflammatory infiltrate.\textsuperscript{10} In some cases ova and parasites can be identified.\textsuperscript{10} The addition of a forceps capsular biopsy may improve the diagnostic accuracy and decrease the number of samples required. This specimen can be obtained easily with the laparoscopic approach.

Most cases of FH are diagnosed as a result of a high degree of suspicion, the presence of eggs within the feces, eosinophilia, and appropriate serologic tests. In addition, patients may require imaging procedures such as CT and possibly histologic evaluation. If the diagnosis remains in question, laparoscopic visualization should assist with the diagnosis and facilitate procurement of an accurately guided biopsy.

ACKNOWLEDGEMENT

We wish to thank Edward T. Ryan, MD, from Massachusetts General Hospital for facilitating serological testing.

REFERENCES