

CASE REPORT

Migration route of Fasciola into the liver

Ian Lockart,*  Amitabha Das,† Neil D Merrett† and Miriam T Levy*

Departments of *Gastroenterology and †Surgery, Liverpool Hospital, Sydney, New South Wales, Australia

Key words

gastroenterology, gastrointestinal infections, hepatology, liver imaging, microbial pathogenesis.

Accepted for publication 30 August 2018.

Correspondence

Dr Ian Lockart, Department of Gastroenterology, Liverpool Hospital, Liverpool, NSW 2170, Australia. Email: ian.lockart@gmail.com

Declaration of conflict of interest: None of the authors have any conflicts of interest to declare.

Abstract

Humans usually acquire Fasciola infection by eating contaminated aquatic vegetation, such as watercress. After ingestion, Fasciola metacercariae excyst in the duodenum. In contrast to other liver flukes (Clonorchis and Opisthorchis) that migrate through the ampulla of Vater and ascend the biliary tree, Fasciola metacercariae penetrate the duodenal wall, migrate through the peritoneal cavity, and enter the liver. After a period of migrating randomly through the liver parenchyma, they eventually reach the larger biliary ducts and mature into adults. We present a case that illustrates this migration route of Fasciola.

Case Report

A 56-year-old lady presented to an outpatient clinic with 3 weeks of upper abdominal pain. She had no significant past medical history or previous abdominal surgeries. She took no regular medications, did not smoke, and did not consume alcohol. She had recently been on a 1-month holiday, traveling with her husband and two children through Vietnam, Cambodia, Malaysia, and Singapore. She returned home to Australia 3 months ago.

Initial blood tests demonstrated an elevated white cell count of $12.1 \times 10^9/L$ (4.0–10.0) and mildly elevated liver enzymes: alkaline phosphatase 304 U/L (30–110), gamma-glutamyl transferase 243 U/L (<35), alanine aminotransferase 107 U/L (5–55), and aspartate aminotransferase 58 U/L (5–55). Her bilirubin, albumin, platelet count, and international normalised ratio were normal.

A liver ultrasound demonstrated an 8 cm hypoechoic, heterogeneous mass in segment 5/6. Her serology for hepatitis B and C was negative, and a range of tumor markers (alpha fetoprotein, carcinoembryonic antigen, cancer antigen 19.9, cancer antigen 125) was normal. *Entamoeba histolytica* serology was negative, and hydatid serology was borderline. A multi-phase computed tomography scan liver demonstrated a hypodense, heterogeneous, non-enhancing subcapsular lesion (Fig. 1a). There was mild stranding of the mesentery adjacent to the inferior pole of the right lobe of the liver. A magnetic resonance imaging scan with primovist demonstrated that the lesion had cystic and hemorrhagic components and irregular rim enhancement. A positron emission tomography scan demonstrated moderately increased tracer uptake (SUV max 8.7) in the liver mass as well as increased uptake in portacaval, preaortic, and aortocaval lymph nodes. Due to initial concerns of a malignancy, she had a gastroscopy and colonoscopy, which were normal. At laparoscopy, adjacent to the mass in segment 5/6, the diaphragmatic surface of

the liver appeared nodular, and there were several adhesions (Fig. 1b). There were also areas of nodular peritoneum. Three core biopsies were taken from the liver mass, and peritoneal washings were obtained. Biopsies were also taken from an area of nodular peritoneum.

Biopsies from the liver mass demonstrated necrotizing lesions, with a mixed inflammatory infiltrate consisting of lymphocytes, plasma cells, and numerous eosinophils (Fig. 1c). Scattered Charcot-Leyden crystals were noted (Fig. 1d). The biopsy from the nodular peritoneum also demonstrated abundant eosinophils and Charcot-Leyden crystals.

Following these biopsy results, she was referred to our outpatient hepatology clinic. By this time, she had developed intermittent fevers. On review of her blood tests, a marked eosinophilia was noted, $5.9 \times 10^9/L$ (0–0.4). With a provisional diagnosis of Fasciola infection, three stool specimens were collected, and Fasciola serology was obtained. Although stool analysis did not identify any Fasciola eggs, our patient was treated empirically with two doses of 500 mg of triclabendazole, 12 h apart. The diagnosis was subsequently confirmed on serology; her Fasciola IgG ratio was markedly elevated at 14.08 (ratios <1 are negative).

Discussion

Fasciola hepatica and *Fasciola gigantica* have a similar lifecycle and are clinically indistinguishable. Humans usually acquire infection by eating contaminated aquatic vegetation, such as watercress. After ingestion, Fasciola metacercariae excyst in the duodenum. In contrast to other liver flukes (Clonorchis and Opisthorchis) that migrate through the ampulla of Vater and ascend the biliary tree, Fasciola metacercariae penetrate the duodenal wall, migrate through the peritoneal cavity, and enter the

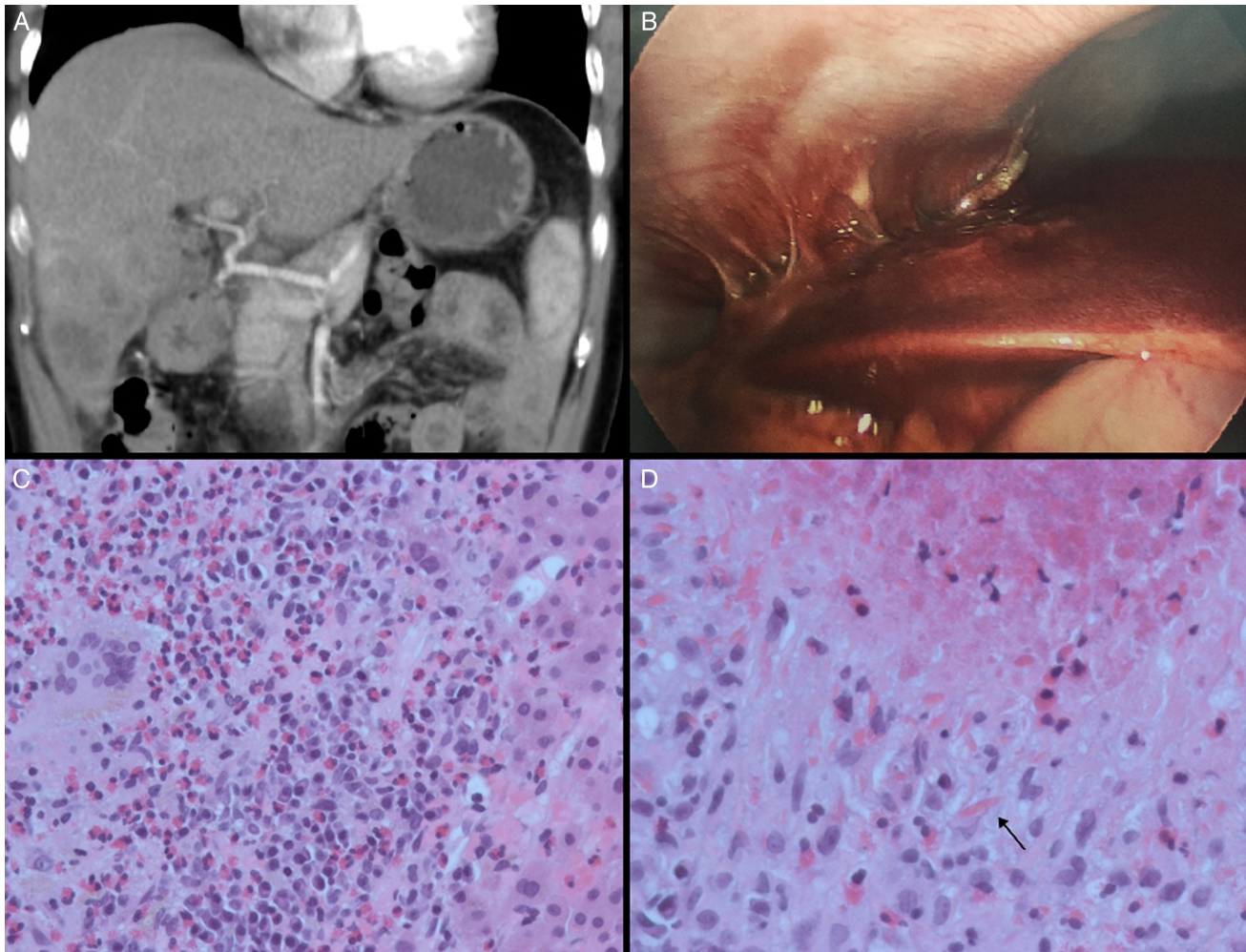


Figure 1 (a) Arterial-phase computed tomography liver. (b) Laparoscopy. (c) Liver biopsy demonstrated abundant eosinophils and (d) Charcot-Leyden crystals.

liver. After a period of migrating randomly through the liver parenchyma, *Fasciola metacercariae* eventually reach the larger biliary ducts and mature into adults. This route of migration explains the laparoscopic findings on the surface of our patient's liver and the results of the peritoneal biopsy.

Our patient presented during the acute (hepatic) phase of *Fasciola* infection. Common symptoms during this phase are right upper quadrant pain and fevers. Symptoms can last 2–4 months and are caused by the tissue destruction and inflammation as the young flukes migrate through the liver parenchyma. A peripheral eosinophilia is almost always present. As demonstrated by our case, stool analysis is usually negative during the acute (hepatic) phase, and *Fasciola* serology is required to make a diagnosis. Serology becomes positive 2–4 weeks after infection. Eggs do not appear in the stool until 3–4 months, when the liver flukes have finished their migration into the larger biliary ducts and matured into adults.

Mature liver flukes can live in the bile ducts for up to 13.5 years. *F. hepatica* flukes can grow up to 2.9 cm in length, and

F. gigantica flukes can reach 5.2 cm. The chronic (biliary) phase may be asymptomatic or may be complicated by biliary obstruction and infection. During this phase, a diagnosis can be established by identifying eggs in the stool, duodenal aspirates, or bile specimens. Adult flukes may also be identified at endoscopy or in surgical specimens. A peripheral eosinophilia may or may not be present during the chronic (biliary) phase.

Triclabendazole can be used in both the acute and chronic phases of infection as it is active against both immature and adult liver flukes. Our patient was treated with two doses of triclabendazole and improved over the following weeks. Her abdominal pain resolved, and her liver function tests and eosinophil count returned to normal.

This case illustrates the migration route of *Fasciola* through the peritoneal cavity into the liver. It also demonstrates the need for *Fasciola* serology during the acute (hepatic) phase of infection as well as the importance of reviewing the eosinophil count when investigating a patient with a liver mass.