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Atypical Presentation of a Rare Parasitic Infection with *Fasciola hepatica*: A Multidisciplinary Case Report

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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 58-year-old
Final Diagnosis: *Fasciola hepatica* infection
Symptoms: Jaundice • fatigue • anorexia • subjective weight loss
Medication: —
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Unusual clinical course

Background: Fascioliasis is a zoonotic disease caused by *Fasciola hepatica* (*F. hepatica*). This infection is associated with a broad spectrum of clinical symptoms such as fever, eosinophilia, and gastrointestinal symptoms.

Case Report: We report a case of *F. hepatica* abdominal mass in the peri-pancreatic region in a 58-year-old man, returned from Venezuela. The patient developed abdominal pain, nausea, anorexia, and weakness. Radiological investigations showed hepatomegaly, as well as mild intra-hepatic and extrahepatic ductal dilatation. The increase in eosinophilia, elevated total IgE titer, and anamnestic data suggested the hypothesis of parasitic infection. The diagnosis was established by high serological titer against *F. hepatica*.

Conclusions: The development of abdominal mass, with jaundice and dilation of the biliary tract, does not always suggest the presence of heteroplasia. Systemic parasitosis represents a not negligible event, especially considering the personal history of life in endemic areas.

MeSH Keywords: *Fasciola hepatica* • Parasitic Diseases • Ultrasonography

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Background

Human fascioliasis is a zoonosis caused by the trematode *F. hepatica*, a liver fluke, usually infesting cattle and sheep [1]. Humans represent occasional hosts in the Fasciola life cycle. Studies have reported that fascioliasis affects about 2.6 million people in the world. This disease is common in developing countries, and it is endemic in Africa, Asia, Europe, and the Americas. High prevalence rates have been recorded in South America [2]. *F. hepatica* adults live in the biliary ducts of the host and lay eggs outside of feces in the water, where the eggs hatch, then ciliated miracidia infect snails, which are the intermediate hosts. The cercaria then leave the snail and become metacercarial cysts on aquatic plants. Humans become accidental hosts after eating contaminated uncooked aquatic plants or undercooked sheep liver or by drinking contaminated water [3]. After ingestion, the metacercariae encyst in the duodenum and cross the small intestine wall, the peritoneal cavity, and Glisson's capsule, and then reach the liver. In the hepatic bile ducts, the larvae mature and lay their eggs. About 12 weeks are required from infection to oviposition [4]. Characteristic symptoms of fascioliasis vary with the 3 stages of disease: acute, chronic, and ectopic [5]. The major clinical manifestation in the acute (or hepatic) stage are fever, abdominal pain, headache, pruritis, urticaria, weight loss, and eosinophilia. In this phase, transaminase and bilirubin levels are in the normal range or slightly elevated [6,7]. The chronic phase is generally asymptomatic because extrahepatic obstruction and cholestasis are rare [8], but the presence of the worms and eggs in bile duct can cause obstructive jaundice.

Case Report

We describe the case of a 58-year-old Italian man who has been living in Venezuela for more than 20 years. He returned to southern Italy in September 2014. During this period, he developed diffuse abdominal pain, cold night sweats, nausea, anorexia, weight loss (about 8 kg in 1 month), and weakness. He underwent routine laboratory tests: WBC count of 13 300 cells/mm^3 with 70% neutrophils, 12.3% lymphocytes, 7.2% monocytes, and 8% eosinophils; hematocrit level 34.5%; aspartate aminotransferase (AST) 22 IU/L (normal range, 0–37 IU/L); alanine aminotransferase (ALT) 43 IU/L (normal range, 0–40 IU/L); total bilirubin 0.52 mg/mL (normal range, 0–1.3 mg/mL); albumin 3.3 g/dL, and G-globulins 3.07 g/dL. TORCH serology, coproculture, and stools analyses for parasites were negative. After 2 weeks, the patient showed worsening jaundice and was referred to our hospital. On the first day, we performed an abdominal ultrasound (US), which showed hepatomegaly, mild intra-hepatic and extrahepatic ductal dilatation (especially in the left lobe) in absence of the focal lesions, and splenomegaly (15 cm). The pancreas was homogeneous, without Wirsung duct dilatation. In the peri-pancreatic region, we detected a swollen hypoechoic mass of 10×7 cm (aggregate of lymph nodes), contiguous with the left hepatic lobe, showing inner anechoic components associated with enlarged lymph nodes (max 16 mm) in para-aortic and mesenteric regions (Figure 1). Laboratory tests showed an increase of serum levels of AST (142 IU/L), ALT (314 IU/L), total (5.6 mg/mL) and direct (3.7 mg/dl) bilirubin, GGT 663 U/L (11–43), ALP 2183 IU/L (80–300), and Ca19.9 131 U/ml (<33). AFP, CEA, and PSA serum levels were negative. His WBC count was 8700 cells/mm^3 with 55% neutrophils and a significant increase of eosinophils (19%). A magnetic resonance imaging (MRI) scan of the upper abdomen integrated with cholangio-pancreatography (MRCP) demonstrated a peri-pancreatic, polycyclic, and inhomogeneous mass, defined as "likely due to lymphoma", close to the head of the pancreas, enveloping the mesenteric vessels, without signs of macroscopic vascular invasion. It also confirmed severe bile duct ectasia without evidence of focal lesions (Figures 2, 3). Chest radiograph, colonoscopy, and esophagogastroduodenoscopy were negative for malignancies. Contrast-enhanced ultrasonography (CEUS) did not show any hypoechoic liver areas in the late phase and no pathologic contrast enhancement of lymph nodes was detected. The increased eosinophilia in the absence of malignancies and the subsequent detection of a high titer of total IgE (PRIST) >1000 IU/mL (normal range, 0–100) suggested the hypothesis of parasitic disease. The patient's anamnestic data revealed he had been living in South America and eating poorly cooked mutton and unsafe vegetables, so a stool analyses for parasites was repeated and a serologic test for *F. hepatica* was performed. No parasitic eggs were found in the feces, but serum ELISA revealed a high serological titer against the fluke. He was treated with 10 mg/kg triclabendazole for 2 days and then was discharged in a follow-up program. After 2 weeks, at the first check-up, the patient's jaundice seemed to be getting worst and he did not report any improvement in symptoms. Biochemical tests showed AST 66 IU/L, ALT 65 IU/L, total bilirubin 19.8 mg/mL (direct 15.5), GGT 91 U/L (11–43),

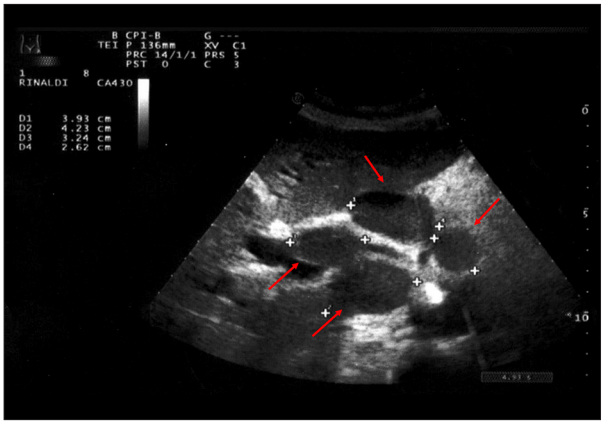

Figure 1. Nodal peri-pancreatic aggregate, adjacent to the left-lobe liver, indicated by the arrows.

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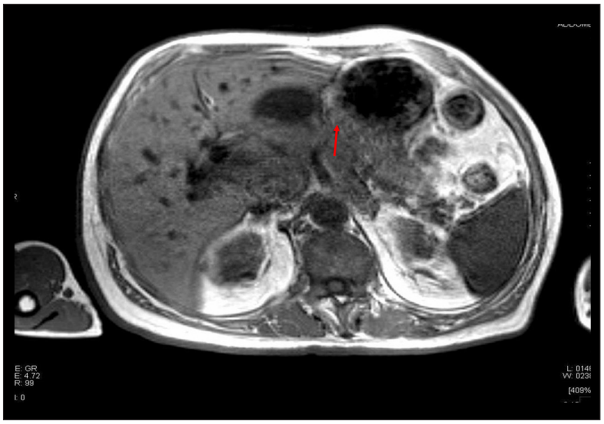

Figure 2. MRI: retroperitoneal, polycyclic, and inhomogeneous mass, defined as "likely attributable to lymphoma". The arrow indicates the main lymph node aggregates.

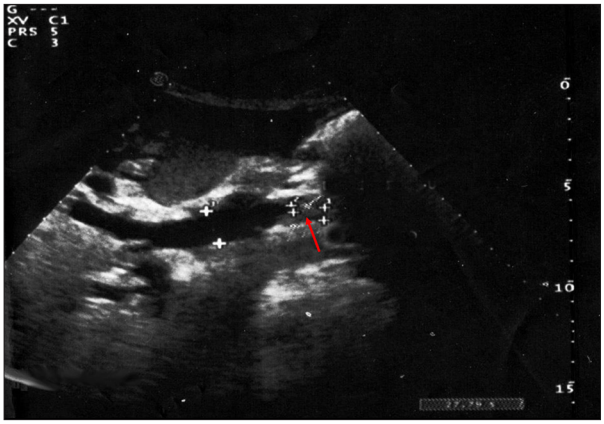

Figure 4. The concentric wall thickening, with a visibility of the residual lumen of the common bile duct indicated by the arrow.

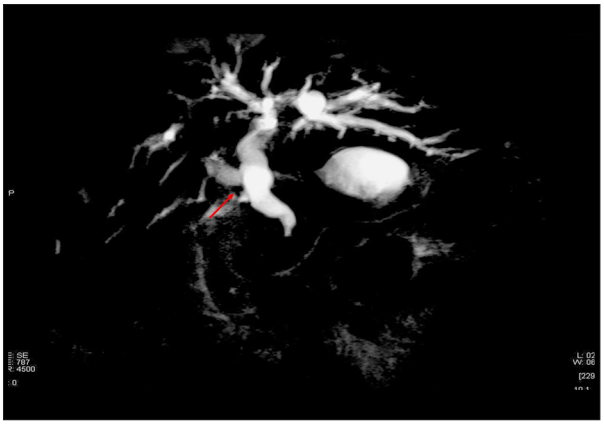

Figure 3. Cholangiographic sequences: dilatation of the bile ducts in the absence of solid lesions. The arrow indicates the point of greatest dilatation of the main bile duct.

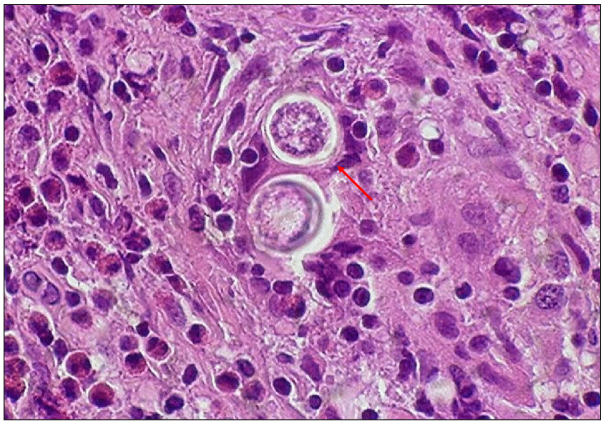

Figure 5. Sites of granulomatous inflammation with presence of several eggs stained in light purple, indicated by the arrow.

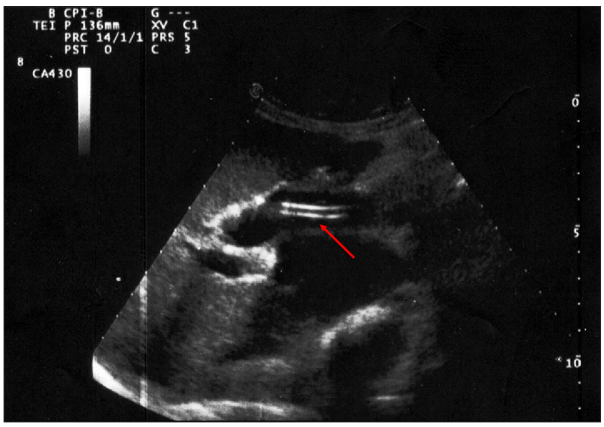

Figure 6. The endoprosthesis in the common bile duct indicated by the arrow.

ALP 405 IU/L (80–300), WBC count 10 200 cells/mm^3, with 54% neutrophils and 10% eosinophils. A new US was performed: the liver and the spleen did not appear enlarged anymore, the mesenteric and celiac lymph nodes showed a dimensional reduction of about 4 cm; therefore, mild-grade dilatation of intra-hepatic bile ducts was confirmed. A severe common bile duct (CBD) dilatation of 16 mm was found to be associated with a concentric wall thickening of the distal tract (about 6.5 mm) and a residual lumen of about 3 mm (Figure 4). The endoscopic retrograde cholangio-pancreatography (ERCP) revealed an enlarged and nodular appearance of the ampulla of Vater, a dilatation of the middle and proximal tract of the CBD, and an irregular stenosis with a jagged appearance in the distal portion. Sphincterotomy was performed and a self-expanding, flexible, metal biliary stent 7 cm in length was placed endoscopically. During the procedure, a bile sample was collected for parasitological testing and a biopsy specimen from the wall thickening was taken, but no living parasites were found in the common bile duct. The biopsy specimen revealed fragments of biliary mucosa and sites of granulomatous inflammation with presence of several eggs (Figure 5). After 2 months,

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the patient returned to our institution for the second check-up, and the symptoms and biochemical values are normalized. Moreover, a new US was performed; the abdominal exploration showed the correctly placed stent in the CBD and a further volume reduction of celiac and mesenteric lymph nodes (with a maximum diameter of 3 cm) (Figure 6). One year later, the patient returned for a check-up, and the clinical condition appeared optimal. Laboratory tests were consistently normal and the last US exam did not show any evidence of residual disease. The follow-up US showed residual lymph nodes (diameter less than 1 cm) and the stent placed in the CBD.

Discussion

Infection with the liver fluke *F. hepatica* is a cosmopolitan zoonosis throughout the sheep-raising areas of the world [9]. Human infections have been reported, particularly from South America, Europe, Africa, China, and Australia [10]. Although fascioliasis is endemic in developing countries, cases have been reported in other countries due to increased traveling and immigration [11]. The total estimated number of infected people is 2.4 million and the number at risk is more than 180 million throughout the world [3]. In our patient, the history of living in a geographical area at risk induced the suspicion of the fluke, together with some laboratory findings at the admission (eosinophilia and high IgE titer). Collecting a precise and relevant history was particularly important in the diagnostic process. The consumption of undercooked liver of infected animals and contaminated plants and water are the main risk factors for infection. With the wide spread of fascioliasis, doctors need to consider the history of travel and immigration of patients for diagnosis [10,11]. Typical symptoms that may be associated with fascioliasis can be divided by phases of the disease: the acute or liver phase, the chronic or biliary phase, and ectopic or pharyngeal fascioliasis [12]. In the acute (or hepatic) stage, our patient experienced diffuse abdominal pain, cold night sweats, nausea, anorexia, weight loss, weakness, and eosinophilia, but no fever or pruritus. Transaminase levels were only minimally elevated (ALT), and bilirubin was in the normal range. Our patient was in the biliary phase just before admission to our hospital. This chronic phase is generally asymptomatic and only rarely causes extrahepatic obstruction and cholestasis, as seen in our case [13,14]. Traditionally, diagnosis of *F. hepatica* infection involves detecting eggs in stool, although this method is unreliable and depends on the skill of the person performing the examination, and stools analyses in our case were persistently negative [15]. Use of immunological tests such as ELISA are important in diagnosis of fascioliasis, they have high sensitivity (90%) [16,17]. Some authors reported 2 key advantages of ELISA over fecal examination: 1) the ability to store samples frozen for a long time without apparent loss in diagnostic power and 2) reduced labor and the ability to process large batches [18]. Flukes and eggs can also be detected in other sites, including duodenal fluid, duodenal and biliary aspirates, and in surgical (laparotomy, cholecystectomy, sphincterotomy) or biopsy specimens [15]. In our case, we found the presence of several eggs in the biopsy specimen taken during the ERCP performed at first check-up 2 weeks after the end of therapy. US findings are non-specific after 8 weeks of infection, when the parenchymal phase, with focal hypoechoic/hyperechoic lesions and increased echogenicity of the liver, develops [15]. In our patient, at the first ultrasound exam we observed hepatomegaly associated with mild intra- and extrahepatic ductal dilatation in the absence of intra-hepatic focal lesions and a retro-pancreatic, swollen hypoechoic lesions, which were contiguous to the left hepatic lobe and were also associated with enlarged (max 16 mm) para-aortic and mesenteric lymph nodes. Many drugs have been used to treat fascioliasis. Unlike other trematodes, *F. Hepatica* does not respond to praziquantel. Bithionol and triclabendazole are the most valid therapeutic choices [19]. A single dose of triclabendazole is required. It is very effective against mature and immature flukes. A single dose of 10 mg/kg Triclabendazole can be administered. In more severe infections, as in our case, two 10-mg/kg doses, 12 h apart, have been prescribed [20]. In our patient, medical therapy was followed by a partial biochemical response, with a reduction in hyper-transaminase, eosinophilia, and lymph node volume, but an increase in bilirubin and jaundice, and the persistence of symptoms. After drug therapy, biliary obstruction occurs in the chronic phase; therefore, this phase is controlled by an endoscopic mechanical clearance of the bile ducts [21–24]. In addition, *F. hepatica* can generate hyperplasia and hypertrophy in the duct epithelium, favoring an increase of proline concentration [25]. As a result, periductal fibrosis with thickening of the duct walls leading to obstruction may occur. Furthermore, the ultrasound findings can reveal these changes: it has been reported that the ultrasound parenchymal phase can lead to the ductal phase (after 8 weeks), characterized by ductal ectasia and thickening of the wall duct [15]. Two weeks after infection, ductal dilatation is shown through thin hypoechoic lines parallel to the portal areas at the beginning of the ductal phase, followed by an increase in biliary dilatation and tortuousness of the bile ducts. The role of US is functional in the ductal phase, while it appears limited in the parenchymal phase. That was the finding of our second ultrasound, which reported a severe CBD dilatation (16 cm) and a concentric wall thickening affecting the distal tract (wall thickness of 6.5mm), with a visibility of the residual lumen of about 3 mm. It has been reported that magnetic resonance imaging (MRI) can also be employed to determine liver parenchymal involvement, but mild ductal dilatation is poorly appreciated and appears on T2W images as hyperintense areas parallel to hypointense lines, corresponding to the portal vessels [15]. In our case, the MRI of the superior abdominal region, integrated with Cholangio-MR sequences, confirmed the

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ultrasound findings, such as hypertrophic stenosis of the choledochus visible until the retro-pancreatic portion. Reports in the literature show ultrasounds can provide information useful in the diagnosis of the disease, and magnetic resonance cholangio-pancreatography (MRCP) can be superior [26]. The role of the CEUS was to exclude the presence of the secondary hepatic lesions, which was useful in considering the initial neoplastic hypothesis. Moreover, the evidence of the singular vascular pedicle at the hilum confirmed the reactive nature of the lymphadenopathy, according to the EFSUMB guidelines and recommendations on the clinical practice of CEUS [27,28]. In previous case reports, the US feature of hepatic fascioliasis are often multiple, ill-defined, hypoechoic lesions throughout both lobes of the liver, or various grades of ductal dilatation [29,30]. CBD wall concentric thickness was confirmed by MRCP and led to the decision to perform an ERCP. Even if the diagnosis is achieved through ultrasound or CT, ERCP should be the first choice in the chronic phase. ERCP and sphincterotomy are used successfully and safely to remove the parasites in the bile duct in case of obstruction [31]. In our case, therapy was probably effective to eradicate the infection and that is why no living mobile parasites were removed from the common bile duct. Despite this, it was not enough to resolve the obstruction, hyperplasia, and hypertrophy in the duct epithelium. Moreover, the presence of eggs in the bile duct probably determined an inflammation reaction, which could have been the main cause of bile duct stricture. Biliary stenting via ERCP was placed to resolve the obstruction and jaundice. Clinical manifestations of the disease can be quite varied; the appearance of a liver abscess, cholecystitis or cholangitis, have been described in literature [32–34].

Conclusions

Presence of an abdominal mass, with jaundice and dilatation of biliary tract, suggests, in most of cases, the presence of heteroplasia. However, we must not overlook the existence of non-oncological diseases, which, although rare, can mimic the procession of symptoms and seriously delay diagnosis. In particular, systemic parasitosis is important, especially when there is a history of living in endemic areas. A diagnostic hypothesis based on teamwork by clinicians, radiologists and pathologists allows exclusion of the principal differential diagnoses (including hydatic cyst disease, amoebic and pyogenic liver abscess) and to establish a correct diagnosis and rapid interventional therapeutic approach that can be life-saving. The pivotal role of US was remarkable in all stages of the disease. The interpretation of the sonograms enabled us to identify lymph node lesions in the early phase, to exclude secondary liver lesions by CEUS, and to evaluate the effectiveness of the therapy during follow-up.

Conflict of interest

None.

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