

Case report

Hepatic fascioliasis: report of two cases

B. Andresen¹, J. Blum³, A. von Weymarn², M. Bürge², W. Steinbrich¹, St. Duewel²

¹ Department of Diagnostic Radiology, University Hospitals Basel, Kantonsspital Basel, Petersgraben 4, 4031 Basel, Switzerland

² Institute of Diagnostic Radiology and Nuclear Medicine, Thurgauisches Kantonsspital Frauenfeld, 8500 Frauenfeld, Switzerland

³ Swiss Tropical Institute, Socinstrasse 57, 4002 Basel, Switzerland

Received: 4 November 1999; Revised: 30 March 2000; Accepted: 4 April 2000

Abstract. Two cases of hepatic fascioliasis with characteristic features in US examinations and CT scans are presented. In both modalities they show tunnel-like branching and clustered areas of low echogenicity/density, which reach subcapsular regions. These cases are presented to recall the imaging features in hepatic fascioliasis especially outside endemic regions. Not only CT but also US is able to detect these characteristic lesions, which may help to make the diagnosis of hepatic fascioliasis in patients with clinical symptoms suggestive of parasitic disease.

Key words: Parasitic liver diseases – Fascioliasis – Diagnostic imaging – X-ray CT – Ultrasonography

Introduction

The liver fluke *Fasciola hepatica* is a common parasite of ruminants, especially sheep and cattle. Fascioliasis is endemic in the southern part of France, Portugal, regions of South America, North Africa, and South-East Asia [1]. Humans get infected by ingestion of contaminated aquatic plants or water [1, 2]. Only a few cases have been investigated with radiological studies [3, 4, 5]. Ultrasound and CT have been directly compared only in a few of them [3].

Fascioliasis in humans can be divided into three stages:

1. The acute stage (2–4 months), which is characterized by non-specific symptoms [2, 6] and in which the immature flukes penetrate through the wall of the small intestine into the peritoneal cavity and spread into the liver.

The parasites migrate through the liver to reach the bile ducts, where the parasites mature.

2. The latent stage (months to years), in which the mature adults are laying their eggs, whereas the host is asymptomatic [2].

3. Without treatment the infection may lead to the third, chronic phase of fascioliasis manifested as cholecystitis, cholangitis, or biliary obstruction/colic by the adult flukes [2, 7].

We demonstrate the imaging findings of one case of acute and one of chronic fascioliasis, where laboratory methods first failed to prove this parasitic disease.

Case reports

Case 1

A 47-year-old male Portuguese presented with fever, upper abdominal pain for 1 month, and recurrent nausea. Serum analyses revealed a WBC of 15.8 /nl, CRP 50 mg/l, alkaline phosphatase 192 U/l, γ GT 151 U/l, LDH 870 U/l, Ferritin 1034 μ g/l, and an eosinophilia of 40%. Serology titer for *Fasciola*, *Filaria*, and *Strongyloides* were positive (high probability for cross-reactivity [1]). Stool samples were negative. An abdominal US showed confluent tunnel-like-branching areas of low echogenicity in the liver segments 6 and 7 (Fig. 1) extending towards the capsule. Hepatosplenomegaly was present. Precontrast CT scan revealed multiple ill-defined, subcapsular clustered areas of low attenuation in the right liver lobe (segments 6 and 7). After administration of i.v. contrast, the hypodense lesions were delineated more sharply (Fig. 2).

A biopsy of the duodenum to detect liver flukes and an endoscopic retrograde cholangiopancreatography (ERCP) to exclude intraductal parasites were performed; both were normal. Two US-guided percutaneous liver biopsies revealed periportal inflammatory infiltrates with predominant eosinophils but no malignant cells.

Correspondence to: B. Andresen

Present address: B. Andresen, Institute of Diagnostic Radiology and Nuclear Medicine, Thurgauisches Kantonsspital Frauenfeld, CH-8500 Frauenfeld, Switzerland

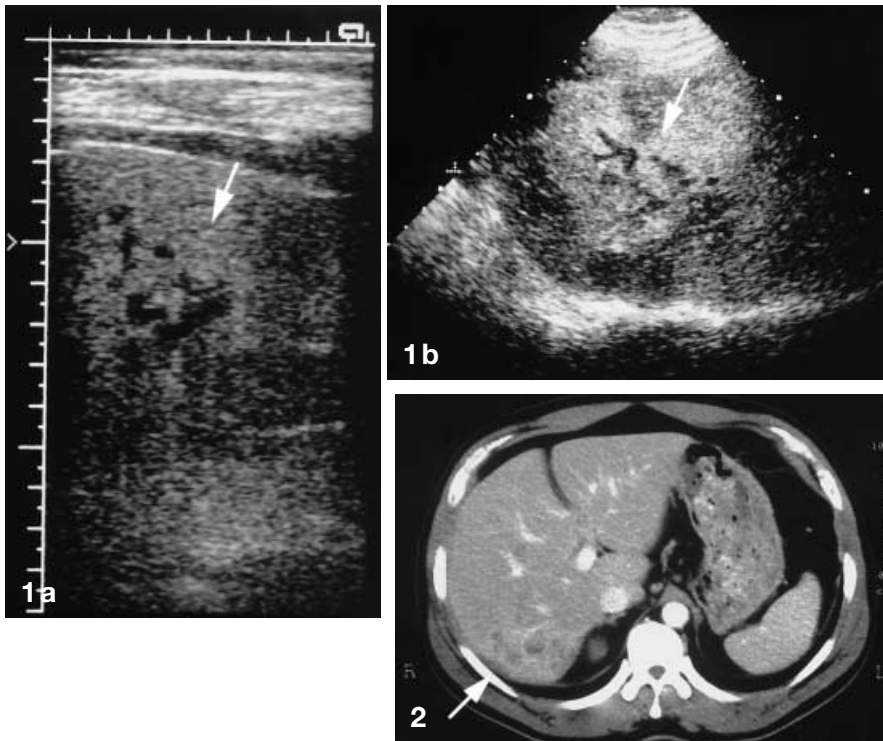


Fig. 1a, b. Case 1. Acute stage of hepatic fascioliasis. Confluent tunnel-like branching areas of low echogenicity which reach subcapsular regions besides echogenic liver parenchyma (arrows)

Fig. 2. Enhanced CT of patient 1. Subcapsular located clustered areas of low attenuation suggestive of migration tracts of the liver fluke (arrow)



Fig. 3. Case 2. Enhanced CT. Cystic and small clustered areas of decreased attenuation in the left liver lobe

Because of persistent severe eosinophilia without evidence of *Fasciola hepatica* and especially high titers for *Strongyloides*, the patient was treated with Ivermectin (in expectation of an infection by *Strongyloides*) to avoid an invasive *Strongyloidosis*. Repeated CT scans and US studies revealed no improvement. Eosinophilia persisted. Control of fecal specimen demonstrated eggs of *Fasciola hepatica*. Then the patient was successfully treated with Triclabendazol. Follow-up studies 4 months later showed decreased lesions in the liver.

Case 2

A 53-year-old female Vietnamese presented with recurrent fever, myalgia, headache, and pain in the right upper abdominal quadrant for approximately 10 weeks. Every year she visited Vietnam for 2 months. Con-

trolled serum analyses showed persistent eosinophilia (27%) and elevated levels of γ GT (186 U/l), alkaline phosphatase (271 U/l), and CRP (108 mg/l). Serum analysis revealed positive titers for *Filaria*, *Strongyloides*, and *Fasciola*. Stool samples were negative for parasitic eggs. Ultrasound showed an enlarged left liver lobe with lesions of low echogenicity predominantly, with tunnel-like and clustered lesions extending to the liver capsule. Contrast-enhanced CT showed multiple clustered hypodense areas in the liver segments 2 and 3 (Fig. 3). The intrahepatic bile ducts in the left liver lobe were dilated. Inside the gallbladder and the common bile duct inhomogeneous material was shown.

The CT and US findings led to the diagnosis of fascioliasis and the patient was successfully treated with Triclabendazol. The CT scans after 4 and 30 months revealed non-dilated bile ducts without intraluminal material and a decreased number and size of liver lesions.

Discussion

The typical but non-specific clinical symptoms and findings in acute fascioliasis, such as fever, hepatomegaly, diffuse upper abdominal pain, and marked eosinophilia [1, 2, 6], were present in both cases.

The diagnosis of fascioliasis may be confirmed by identification of eggs in fecal samples. Eggs may be detected 3–4 months after infection [1], but there are contradictory reports on the rate of positive stool samples between 0 and 72% in fascioliasis [2, 3, 4, 5, 6]. Another way to contribute to the diagnosis is the detection of anti-fluke antibodies in serum by ELISA. Antibodies appear earliest 2–4 weeks after infection [1]. Different

sensitivities for ELISA were described in literature: From 78.9% [8] up to 100% sensitivity and 97.8% specificity [9]. However, in other helminthic infections with similar clinical symptoms (e. g., schistosomiasis, clonorchiasis) cross-reactions have been reported. In chronic infections immunological tests also could fail [1].

As reported by Arjona et al. in 80% of 15 cases we found typical hypodense nodular or branching hepatic lesions on CT [6], which are better delineated after intravenous contrast.

As described by Han et al. [3] we found that the lesions extend to the liver capsule, where the immature flukes are described to penetrate into the liver. In US examinations these lesions were of low echogenicity in our cases, compared with Han et al. [3], who observed variable echogenicity. Whereas we found US and CT to be equal in their quality to make the diagnosis of fascioliasis, Arjona et al. [6] reported that US is inferior in comparison with CT in diagnosing fascioliasis.

The value of MRI in diagnosis of fascioliasis is still questionable since only a limited number of patients has been studied: whereas Van Beers et al. [5] describe no characteristic lesions, Han et al. [4] found the typical tortuous lesions as others have described on CT.

The clinical differential diagnosis includes other parasitic diseases, which cause hyper eosinophilia: echinococcosis; schistosomiasis; ascariasis; clonorchiasis; and strongyloidiasis. The radiologic differential diagnosis may be difficult, but there are some characteristic imaging features of the different parasitoses.

In alveolar hydatid disease cystic lesions have amorphous coalescent calcifications and are found typically in a central location [11] in contrast to hepatic fascioliasis.

Hepatic manifestations of chronic schistosomiasis include periportal thickening on US [12] and hypodense lesions in non-enhanced CT; the latter are markedly enhanced in contrast-guided CT scans [13], which is not seen in fascioliasis. In schistosomiasis japonica CT demonstrates typical peripheral septal and capsular calcifications [11], which are also distinguishable from fascioliasis.

Ascaris may enter the common bile duct and lead to obstruction causing similar symptoms as seen in chronic fascioliasis [14]. The dilatation of bile ducts [15, 16] is similar to chronic fascioliasis, whereas the typical finding in acute fascioliasis with subcapsular tunnel-like branching areas in ascariasis is missed.

Fascioliasis cause dilatation of the central bile ducts, in contrast to clonorchiasis, which shows diffuse peripheral ductal dilatation [10].

Hepatic manifestations of strongyloidiasis are rare. In the case with a *Strongyloides stercoralis* hyperinfestation US showed lesions with an inner hyperechogenic area and an outer hypoechogenic rim. The CT scan showed hypodense lesions with a central ring-like area of increased attenuation [17].

In the chronic stage of fascioliasis, US may reveal echogenic material (which represents liver flukes) in

the gallbladder, but the ERCP is the radiological method of choice since it can be used for therapeutic intervention simultaneously [7, 18].

Two cases with typical symptoms and imaging findings in hepatic fascioliasis in the acute and chronic stage are presented. In fascioliasis diagnosis by identification of eggs in fecal samples and detection of anti-fluke antibodies often failed. These cases demonstrated that not only CT, but also US, is helpful in the diagnosis of fascioliasis.

References

- Chen MG, Mott KE (1990) Progress in assessment of morbidity due to fasciola hepatica infection: a review of recent literature. *Trop Dis Bull* 4:R2–R37
- Bjorland J, Bryan RT, Strauss W et al. (1995) An outbreak of acute fascioliasis among Aymara Indians in the Bolivian Altiplano. *Clin Infect Dis* 21: 1228–1233
- Han JK, Choi BI, Cho JM et al. (1993) Radiological findings of human fascioliasis. *Abdom Imaging* 18: 261–264
- Han JK, Han D, Choi BI, Han MC (1996) MR findings in human fascioliasis. *Trop Med Int Health* 1: 367–372
- Van Beers B, Pringot J, Geubel A et al. (1990) Hepatobiliary fascioliasis: noninvasive imaging findings. *Radiology* 174: 809–810
- Arjona R, Riancho JA, Aguado JM, Salesa R, Gonzalez-Macias J (1995) Fascioliasis in developed countries: a review of classic and aberrant forms of the disease. *Medicine* 74: 13–23
- Danilewitz M, Kotfila R, Jensen P (1996) Endoscopic diagnosis and management of *Fasciola hepatica* causing biliary obstruction. *Am J Gastroenterol* 91: 2620–2621
- Apt W, Aguilera X, Vega F, Miranda C et al. (1995) Treatment of human chronic fascioliasis with Triclabendazol: drug efficacy and serologic response. *Am J Trop Med Hyg* 52: 532–535
- Shaheen HI, Kamal KA, Farid Z et al. (1989) Dot-enzyme-linked immunosorbent assay (DOT-ELISA) for the rapid diagnosis of human fascioliasis. *J Parasitol* 75: 549–552
- Han JK, Jang HJ, Choi BI et al. (1999) Experimental hepatobiliary fascioliasis in rabbits: a radiology–pathology correlation. *Invest Radiol* 34: 99–108
- Stoupis C, Taylor HM, Paley MR et al. (1998) The rocky liver: radiologic–pathologic correlation of calcified hepatic masses. *Radiographics* 18: 675–685
- Hatz C, Jenkins JM, Ali QM et al. (1992) A review of the literature on the use of ultrasonography in schistosomiasis with special reference to its use in field studies. 2. *Schistosoma mansoni*. *Acta Trop* 51: 15–28
- Fataar S, Bassiony H, Satyanath S, Rudwan MA et al. (1985) CT of hepatic Schistosomiasis Mansoni. *AJR* 145: 63–66
- Akata D, Ozmen MN, Kaya A, Akhan O (1999) Radiological findings of intraparenchymal liver *Ascaris* (hepatobiliary ascariasis). *Eur Radiol* 9: 93–95
- Severen van M, Lengele B, Dureuil J (1987) Hepatic ascariasis. *Endoscopy* 19: 140–142
- Rocha M de S, Costa NS, Costa JC et al. (1995) CT identification of ascaris in the biliary tract. *Abdom Imaging* 20: 317–319
- Rawat B, Simons ME (1993) Strongyloides stercoralis hyperinfestation. Another cause of focal hepatic lesions. *Clin Imaging* 17: 274–275
- Riedtmann H-J, Obeid T, Aeberhard P, Sakman P (1995) Fasciola hepatica: a rare cause of acute cholecystitis with cholestatic icterus. *Schweiz Med Wochenschr* 125: 1642–1648