FNG & Bilim Tıp Dergisi 2017;3(1):42-46
doi: 10.5606/fng.btd.2017.009

Case Report / *Olgu Sunumu*

A hepatic fascioliasis case diagnosed after the treatment

Tedaviden sonra tanı konan karaciğer fascioliasis olgusu

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ABSTRACT

Human fascioliasis is a rare parasitic disease caused by Fasciola hepatica, affecting mainly the liver and the biliary tract. If not suspected, it may easily be confused with other pathologies that will lead to incorrect diagnosis as well as inappropriate treatments. Late diagnosis of the hepatic fascioliasis may cause irreversible liver damage and insufficiency. In this case report, we present the computed tomography and magnetic resonance imaging findings of a hepatic fascioliasis case that was diagnosed after the treatment.

Keywords: Computed tomography; fascioliasis; human; magnetic resonance imaging.

ÖZ

İnsan fasioliyazisi, başlıca karaciğer ve safra yollarını etkileyen, Fasciola hepatica'nın neden olduğu nadir bir paraziter hastalıktır. Şüphelenilmezse yanlış tanıya ve uygunsuz tedavilere neden olacak diğer patolojilerle kolayca karıştırılabilir. Tanısı geç konulan karaciğer fascioliasis, geri dönüşü olmayan karaciğer hasarına ve yetmezliğine neden olabilir. Bu yazıda, tedaviden sonra tanı konulan bir karaciğer fascioliasis olgusunun bilgisayarlı tomografisi ve manyetik rezonans görüntüleme bulguları sunulmuştur.

Anahtar sözcükler: Bilgisayarlı tomografi; fascioliasis; insan; manyetik rezonans görüntüleme.

Fascioliasis, caused by the Fasciola species, has a very large worldwide distribution with a large prevalence rate. It may affect the hepatic tissue causing pathological sequences in both the acute and chronic stage. With presentation of wide clinical symptoms, the diagnosis of fascioliasis can be made using imaging modalities, hepatocholestatic enzyme levels, serological tests and also as noted in the recent study, nuclear and mitochondrial ribosomal ribonucleic acid (rDNA) sequence analysis.[1] Hepatic fascioliasis may be sometimes confused with other infectious or neoplastic diseases.

In this article, we present the computed tomography (CT) and magnetic resonance imaging (MRI) findings for a case of hepatic fascioliasis, in which post-treatment diagnosis was made.

CASE REPORT

A 32-year-old female patient was admitted to the hospital with complaints of right upper quadrant pain, intermittent fever, nausea, weight loss, night sweating and fatigue. Her symptoms began four months earlier. Laboratory tests revealed elevated liver enzymes (aspartate aminotransferase (AST): 120, alanine

Received: October 10, 2016 ***Accepted:*** January 23, 2017

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Presented as electronic poster in 35th National Congress of Radiology, November 11-16, 2014, Antalya, Turkey.

aminotransferase (ALT): 128 IU/L), leukocytosis (14,000/mm^3), eosinophilia (2,400/mm^3). Abdominal ultrasonography (US) was reported as normal. No definitive diagnosis was made and her symptoms improved spontaneously. Within two months, the symptoms occurred again and the laboratory results revealed mild elevation in ALP, gamma-glutamyl transferase (GGT), sedimentation rate, C-reactive protein (CRP) and ferritin levels. Other biochemical results (Hemoglobin, trombocytes, bilirubin, alpha-fetoprotein (AFP), gammaglobulin, prothrombin time (PT), activated partial thromboplastin time (aPTT) were normal; viral hepatitis serology were negative. She had no prior medical history and she was not using any medications. This time US imaging showed an echogenic fluctuating hepatic lesions in the right anterior lob of the liver. Computed tomography imaging showed a non-enhanced, unmarked, heterogeneous tubular-shaped hypodense lesion (Figure 1). Magnetic resonance imaging (MRI) showed a cystic lesion hypointense on T_1-weighted images (T_1-WI), hyperintense on T_2-WI, peripheral contrast enhanced on post-contrast weighted sequences and hyperintense on diffusion-WI (Figure 2). Magnetic resonance cholangiopancreatography (MRCP) revealed no biliary tract involvement. Eosinophilia led to the diagnosis of parasitic liver disease. Recurrent cholangitis attacks with hepatocellular damage, relocating lesions in imaging migrating from the periphery to portal hilum of the liver and eosinophilia made fascioliasis the most possible diagnosis. *Fasciolia hepatica* specific-antibodies using ELISA (after 9 weeks of the first symptoms)

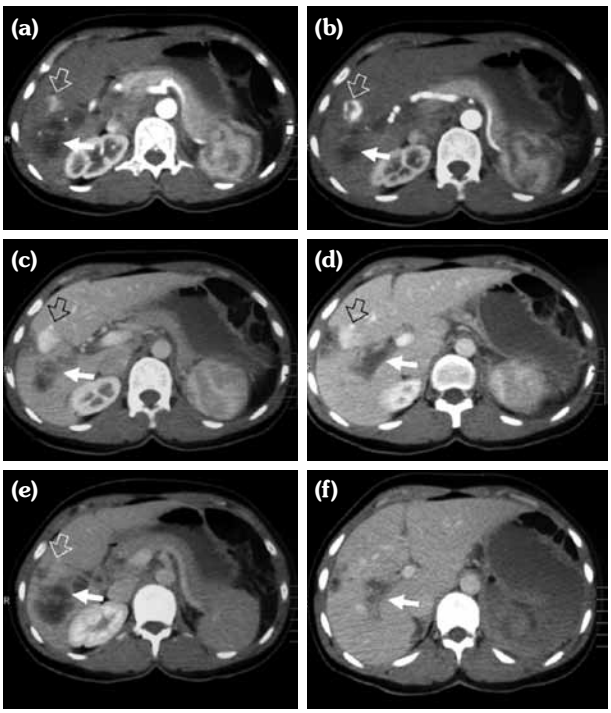

Figure 1. On contrast enhanced computed tomography images [arterial phase **(a, b)**, venous phase **(c-e)**, delayed contrasted image **(f)**] show non-filling peripheral enhanced, subcapsular and central localized hypodense tubular-tortuous lesions in the right lob of the liver (arrow). Next to the lesion peripheral nodular contrast enhancement in arterial phase and contrast fixation in venous phase suggested hemangioma (open arrow). (Computed tomography protocol: 50 mAs, 120 kV, 7 mm slice thickness; 350 mL iodine contrast material was used).

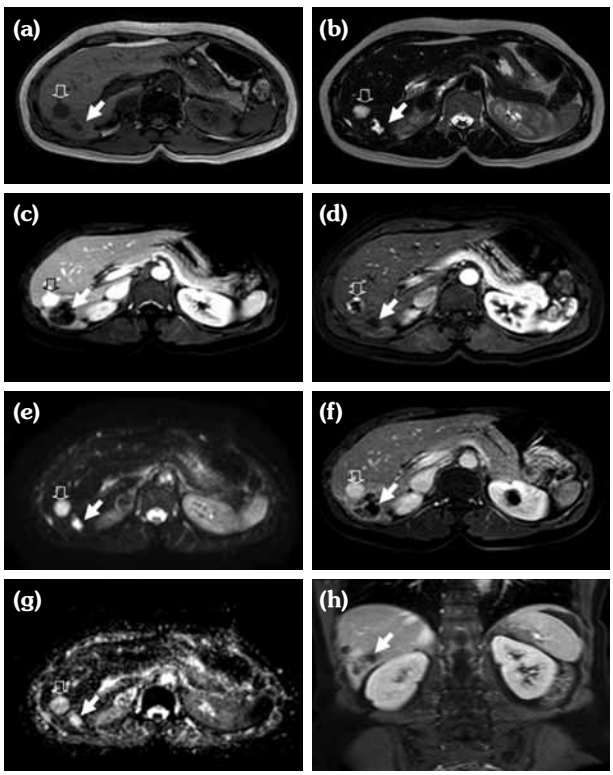

Figure 2. Magnetic resonans imaging. On T_1-WI (TE: 580, TR: 7) hypointene in signal intensity **(a)**, hyperintense on T_2-WI (TE: 100, TR: 2850) **(b, e)**, peripheral contrast enhancement non-filling tortuous appearance on contrast enhanced images (TE: 635, TR: 15) **(h)** and hyperintense on diffusion-WI (b=500) **(g)** demonstrate the tubular form cystic lesion (arrow). Next to the lesion T_1-WI hypointense **(a)**, T_2-WI hyperintense **(b, e)**, peripheral nodal enhancement on early arterial phase **(d)** contrast agent fixation on portal **(c)** and the late venous phase **(f)** suggests hemangioma (open arrow).

were negative. Fever, fluctuating mass in liver and eosinophilia levels were against *Echinococcus multilocularis* infection. Antibodies against *Echinococci* were also negative. With further query, it was discovered that the patient and her family occasionally ate herbs collected from the banks of a brook nearby, however, the rest of the family showed no symptoms or biochemical anomalies. Due to possibility of a false seronegativity (5%) and the high likelihood of fascioliasis, the patient was given two courses of triclabendazole of 10 mg/kg for three consecutive days in a month. The patient is still under follow-up and two years after the treatment shows no symptoms, biochemical studies are in normal ranges. On the post-treatment MRI, remarkable regression of the lesion was observed (Figure 3). A written informed consent was obtained from the patient.

DISCUSSION

Fascioliasis is highly pathogenic disease. It is caused by Fasciola hepatica, a trematoda and

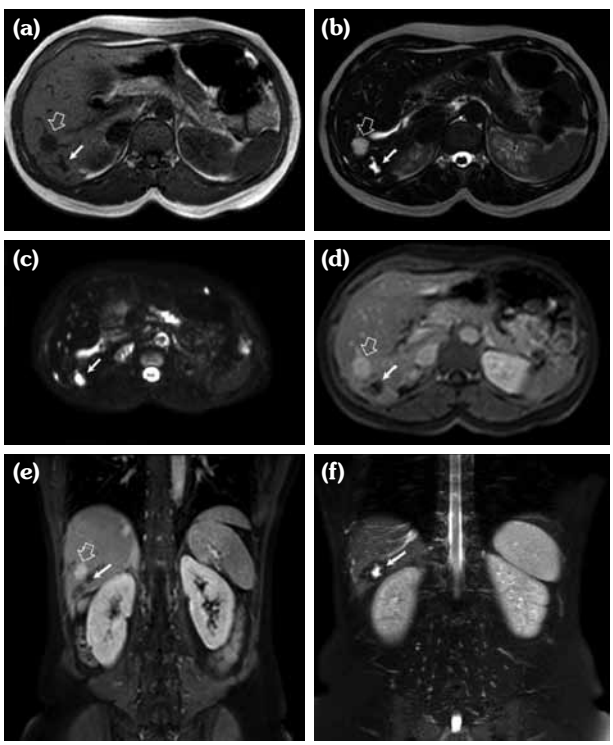

Figure 3. Post-treatment magnetic resonance imaging shows regression in the size of the lesion in all sequences (arrow) [T_1-WI (TE: 580, TR: 7) **(a)**, STIR (TE: 80, TR: 3000) **(b, f)**, post-contrast images (TE: 635, TR: 15) **(d, e)**, DWI (b=500) **(c)**]. Hemangioma (open arrow).

acquired by eating freshwater plants such as watercress (by eating metacercariae). In humans, the pathogenesis of the disease depends on the number of infecting flukes and appears to be similar to that reported in animals. Human fascioliasis can no longer be considered only as a secondary zoonotic disease, but must be considered an important human parasitic disease.[2]

There has been a marked increase in the number of reports of humans with fasciola infection since 1980 and several endemic geographical distribution studies have been mentioned.[3]

After the incubation period (two weeks to three months), clinical and laboratory findings emerge (Table 1). Depending on the clinical symptoms, two different forms of fascioliasis has been defined. In the acute (or hepatic phase), the parasite passes through the Glisson's capsule and enters the liver continuing its migration towards the biliary system.[4-6] Thus, the clinical and laboratory findings during this stage (fever, abdominal pain, weight loss and eosinophilia) are due to the inflammation in response to migration of the larva within the liver.[5] Once the parasite enters the biliary system, the complaints of the patient decrease or even entirely disappear. The chronic (or biliary stage) is characterized by the presence of adult flukes in intrahepatic and common bile ducts. Eosinophilia, fever and abdominal pain resolves during this stage.[4]

Also regarding the duration of symptoms and ultrasonographic findings, the disease can be categorized into acute and chronic stages (Table 1). The larvae mature in the liver and migrate slowly for two to four months until they reach the larger bile ducts.[2,5] In the case of patients who have experienced symptoms for less than four months and no echogenic motility in the gallbladder on US examination, the patient is considered in acute stage, while more than four months symptoms continuity in association with positive US findings, the patient is considered to be in chronic stage.[7]

In our case, there were signs of cholangitis with slightly elevated liver enzymes (AST, ALT, ALP, GGT) and leukocytosis. However, the presence of a history of recurrent symptoms in the current patient, might be indicative of a temporary biliary obstruction caused by the parasite. Also, the

Table 1. Stage categorization of Fasciolias depending on clinical, laboratory and imaging findings[4-7]

Clinical and laboratory findings		Duration of symptoms and ultrasonographic findings	
Acute (Hepatic phase)	Chronic (Biliary stage)	Acute	Chronic
• The parasite passing through the Glisson's capsule, entering liver and migrating towards the biliary system, • Thus, the clinical (fever, abdominal pain, weight loss) and laboratory (eosinophilia) findings are due to the inflammation.	• Presence of adult flukes in intrahepatic and common bile ducts, • Resolving of eosinophilia, fever and abdominal pain.	• Symptoms less than 4 months, • US: No motile echogenic changes in the gallbladder.	• Symptom more than 4 months, • Positive US findings.

symptoms persisting for 4-6 months, suggested the patient was in the chronic stage.

Some symptomatic clues may help in understanding the stage of the disease. For example, in the acute phase, fever and arthralgia are more common symptoms, while, epigastric pain and right-upper quadrant pain are more frequent symptoms in chronic stage patients.[7] In the study by Mailles et al.,[8] asthenia (89%), fever (67%), myalgia (61%), right upper quadrant abdominal pain (72%) were reported to be the most common symptoms. Similarly in our case, the patient presented with right upper quadrant pain, fever, nausea, weight loss and night sweats.

Mailles et al.[8] also reported that leukocytosis was present in 14 of their 18 patients (median: 10,200 cells/mm^3). In our case, leukocyte count was 14,000 cells/mm^3. And also elevation of ALT and AST (suggesting hepatic inflammation) may occur during acute phase, it gradually decreases in the chronic stage. The ALP and GGT counts indicated cholestasis, are commonly highest in the chronic stage.[9] Particularly in the acute stage, eosinophilia often exceed the 5% limit; whereas, it may be present in about 50% in chronic stage patients. Hence, eosinophilia may be a valuable predictive finding for fascioliasis in patients with permanent clinical condition.[8]

The US, CT and MRI are non-invasive modalities for detection of the disease.

In the acute stage (parenchymal invasion by the larva), CT may demonstrate linear or branching subcapsular-peripheral low attenuation clustered nodular non-enhancing lesions, with different sizes and maybe vermiform structures in the gallbladder.[10-12]

The MRI appearance is similar to CT in shape, can better characterize the hemorrhagic nature and extension of the lesion. The US has large variety of finding and is non-specific in shape.

In chronic stage (maturation of flukes), ductal changes are observed predominantly in the central bile ducts, of which can be shown through CT, MRI and US. Also, US is capable of demonstrating the movement of the worm with-in the bile duct and flukes in the gallbladder as mobile vermiform structures.[11,13,14]

Due to an absence of eggs in the stool, in the first 3-4 months, serologic tests are very important. In the study by Karahocagil et al.,[3] all cases in their study were seropositive for fascioliasis in ELISA testing compared to none seropositive cases in the control group. However, in three patient with consistent clinical without radiological findings, the final diagnosis was made through an ELISA test. In the current case, despite the negative ELISA test, typical CT and MRI findings raised the suspicion of the presence of fasciolosis.

Hepatic fascioliasis may sometimes be confused with other clinical problems such as acute hepatitis, other parasitic infestation, biliary tract disease, hepatic amoebiasis and malignancy (i.e. hepatocellular carcinoma). Inflamed or hemorrhagic hepatic nodular lesions may be misinterpreted as metastases, leading to incorrect treatment or interventional procedure.[9]

Triclabendazole (a benzimidazole derivative), is effectively used in the treatment of fascioliasis. The Center of Disease Control (CDC) has recommended triclabendazole as the first line drug for fascioliasis since 1998.[15,16]

Our patient had both acute and chronic stage symptoms within the first 4-6 months, and also both of biochemical and radiological findings suggested the high possibility and presence of hepatic fascioliasis and also improvement in findings were observed after triclabendazole administration. In light of the literature, as was in the current case, it is noteworthy to mention the importance of non-invasive methods that provided diagnosis from treatment.

In conclusion, although it has varying clinical and laboratory findings, fascioliasis should be considered in differential diagnosis of patients with abdominal pain, fever, weight loss, eosinophilia and elevated Immunoglobulin E (IgE). In fascioliasis, radiological examinations may reveal diagnosis even prior to the emergence of the clinical findings.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

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