Case Report / Olgu Sunumu

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Long-Term Follow-Up of Computed Tomography and Magnetic Resonance Imaging Findings in Hepatic Fascioliasis

Hepatik Fasioliazis'de Bilgisayarlı Tomografi ve Manyetik Resonans Görüntüleme ile Uzun Dönem Takip Bulguları

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Abstract / Özet

Fasciola hepatica (FH) is a liver fluke that may mimic many other diseases, potentially leading to misdiagnosis. A 25-year-old patient who was admitted to our clinic with hepatic lesions and diagnosed with fascioliasis is presented in this report. Difficulties in the diagnosis of FH infestation and the role of radiological evaluation are emphasized. We suggest that FH infestation should be taken into consideration in patients being evaluated for hepatic mass and elevated liver enzymes, especially if accompanied by eosinophilia, or if the patient is known to have visited an endemic region.

Key Words: Fasciola hepatica, fascioliasis, CT, MRI

Fasiola hepatica (FH) birçok hastalığı taklit eden bir karaciğer trematodu olup, tanıda zorluğa yol açabilir. Aşağıda 25 yaşında, hepatik lezyonlar nedeni ile incelenirken fasioliasis tanısı alan bir olgu sunulmuştur. Bu olgu sunumu ile FH infestasyonunun tanısında karşılaşılan zorluklar ve doğru tanıya ulaşmada radyolojik değerlendirmenin önemi vurgulanmak istenmiştir. Karaciğer enzimlerinde yükselme ve karaciğerde kitle nedeni ile incelenen olgularda, özelikle eosinofili eşlik ediyor ve hasta endemik bir bölgede bulunmuşsa FH infestasyonu olasılığı hatırlanmalıdır.

Anahtar Kelimeler: Fasiola hepatika, fasioliazis, BT, MRG

Introduction

Hepatobiliary fascioliasis is a parasitic infestation caused by the trematode Fasciola hepatica. There has been an increase in human fascioliasis worldwide in the last decade. Increased availability of cross-sectional imaging methods and awareness of the important role of imaging in diagnosis helped to reveal that fascioliasis is endemic areas.

The disease is endemic in some Middle and Far East countries and in some parts of Central and South America. Human fascioliasis mainly involves the hepatobiliary system. It has two different phases: hepatic (acute) and biliary (chronic). The hepatic phase of the disease occurs when immature parasites pass into the liver through its capsule. The parasites migrate through the liver parenchyma to the biliary system. The biliary phase of the disease occurs in the presence of parasites in the biliary system.

We report a case of hepatic fascioliasis and discuss the role of noninvasive imaging techniques in diagnosis and follow-up with special reference to the distinction between the two stages of the disease.

Case Report

A 25-year-old male patient was admitted to our Internal Medicine Department with fever, abdominal pain, diarrhea, fatigue, and bloating. He had been treated by spasmolytic agents, omeprazole and enzyme preparates prior to hospitalization and was found to have elevated hepatic enzyme levels and eosinophilia. The patient was hospitalized due to symptom persistence. He had a personal history of allergic rhinitis and septum deviation and reported frequent work-related international traveling. On physical examination, the patient had a fever of 37.5°C, and his liver was 3 cm, soft, and mildly painful, with smooth margins on palpation. Dullness was noted on Traube's space. Other examination findings were normal. Upon admission, his leukocyte count was 13,600/mm^3, eosinophil ratio was 51%, hemoglobin (Hb) level was 13.5 g/dL, and thrombocyte count was 284,000/mm^3. Hypereosinophilic syndrome, parasitosis, eosinophilic leucosis, and lymphoma were considered as initial diagnoses. *Entamoeba histolytica* antibody was negative, total IgE was 86.9 mg/dL (normal), indirect hemagglutination test for hydatidosis, and *Toxocara canis* antibody were negative. Stool examination for parasites and eggs was uneventful. A blood sample was sent to a specialized center for *Fasciola hepatica* antibody testing for radiological examination, extensive intestinal gas was present on ultrasonography (US). Minimal irregularity in the liver parenchyma, hepatomegaly, and hardly detectable hypoechoic-isoechoic solid heterogeneous lesions were present. No lesions were observed in the gall bladder and bile ducts. The liver

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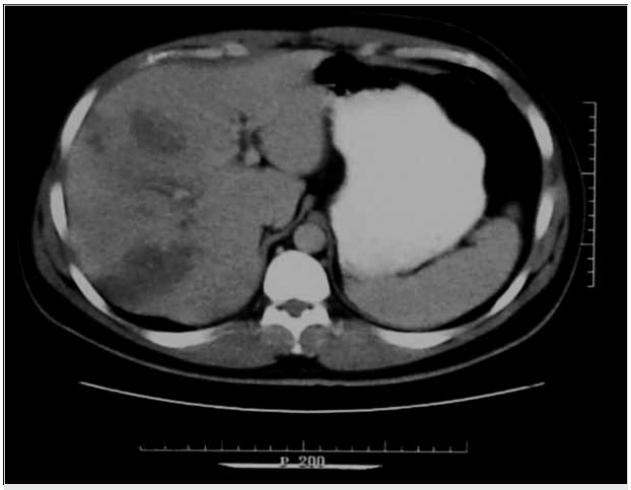

Figure 1. A contrast-enhanced CT scan demonstrates multiple, clustered, hypodense nodular lesions with peripheral contrast enhancement in the liver

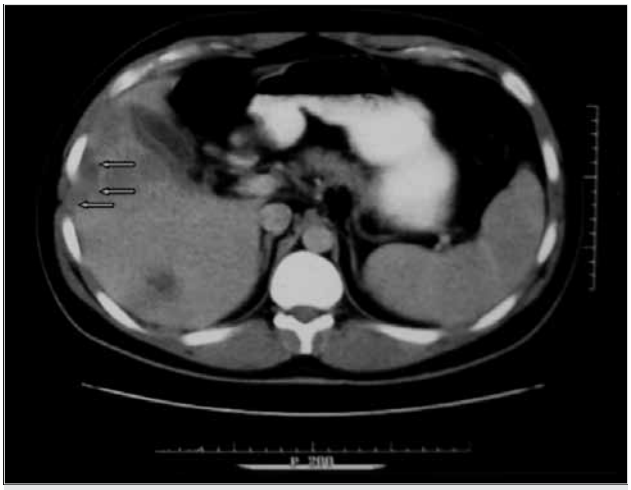

Figure 2. Infiltration of the liver in the parenchymal phase. Effusion is evident on subcapsular hypodense curvilinear lesions (arrows) in the anterior segment of the right lobe on axial contrast-enhanced CT

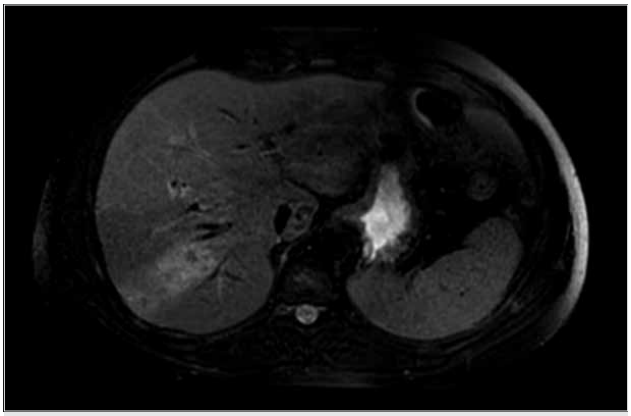

Figure 3. Highly hyperintense lesions are observed in the posterior segment of the right lobe on axial T2*WI MRI

US was not repeated in the follow-up due to the indistinct nature of the liver lesions and low diagnostic value. Three-phasic upper abdomen computerized tomography (CT) and magnetic resonance imaging (MRI) scans were performed after US examination. The patient was followed by CT imaging and MRI before, immediately, and 6 months after treatment.

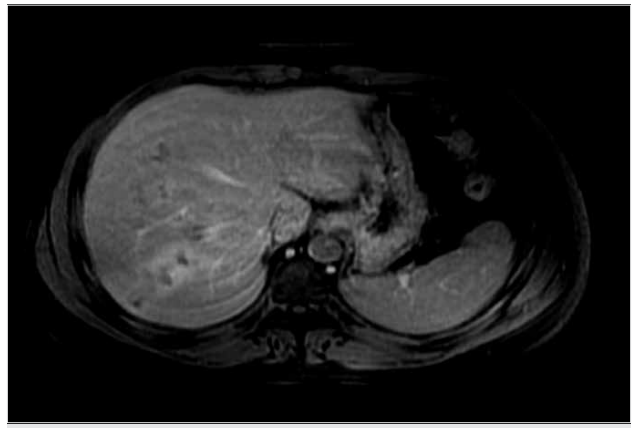

Figure 4. On axial T1 C+ MR images, multiple clustered nodular lesions are observed in the posterior segment of the right lobe, and hypointense lesions with peripheral contrast enhancement are observed in the liver

Abdominal computerized tomography (CT) was initially performed without contrast, and then, 120 mL intravenous contrast medium (Ultravist 300, Bayer, Berlin, Germany) was administered by an automatic injector at 3 mL/second. After contrast injection, imaging was obtained in the hepatic arterial phase (20 seconds), portal venous phase (60 seconds), and late phase (10 minutes).

Multiple, diffuse, hypodense nodular lesions and accompanying curvilinear or linear hypodense striations, as well as subcapsular effusion, were observed on dynamic triphasic CT evaluation (Figures 1, 2). Nodules had irregular margin, with the biggest diameter measuring 2-3 cm. After IV contrast injection, CT scan demonstrated multiple, clustered, hypodense lesions, with peripheral contrast enhancement in the liver. It was noted that lesions were better delineated in the portal venous phase. MRI revealed similar findings with CT. Lesions were iso-hypointense on T1-weighted images of abdominal MRI. They were visualized as hyperintense and/or isointense with a hyperintense halo on T2-weighted images. Additionally, the lesions were also highly hyperintense on T2-weighted axial plane fat suppression MRI in the posterior segment of the right lobe (Figure 3). On MRI, following IV contrast (Magnevist, Bayer, Berlin, Germany), multiple nodular lesions were observed in the posterior segment of the right lobe, while hypointense lesions with intensive peripheral contrast enhancement were observed at the center (Figure 4).

Fasciola hepatica antibody indirect fluorescent antibody test (IFA), which was performed in a foreign laboratory, was reported to be positive at 1/160 titration (normal value: <1:10). After the patient was diagnosed with fascioliasis by eosinophilia and serological evidence of antiparasitic antibodies, a single dose of triclabendazole was administered for treatment. Control CT and MRI were performed at months 6 and 12.

Eosinophil ratio was 40.2% at 3 months after treatment. Eosinophilia was decreased to 5% at 6 months. Significant regression of the lesions was noted at the 6-month radiological follow-up by CT (Figures 5 and 6).

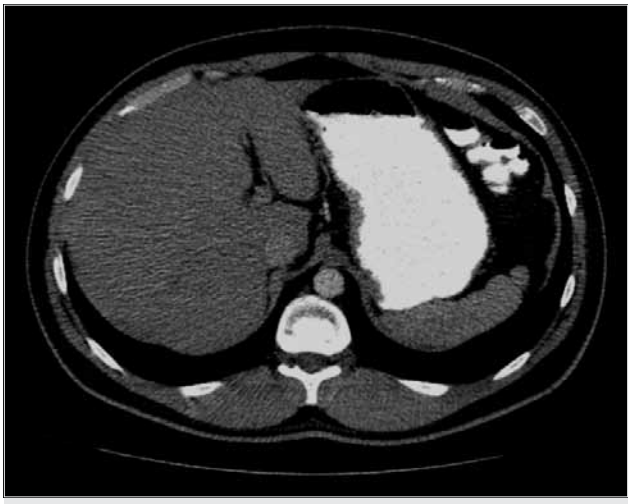

Figure 5. Significant regression is visible on axial contrast-enhanced CT compared to the hypodense nodular lesions observed in the anterior and posterior right lobes of Figure 1

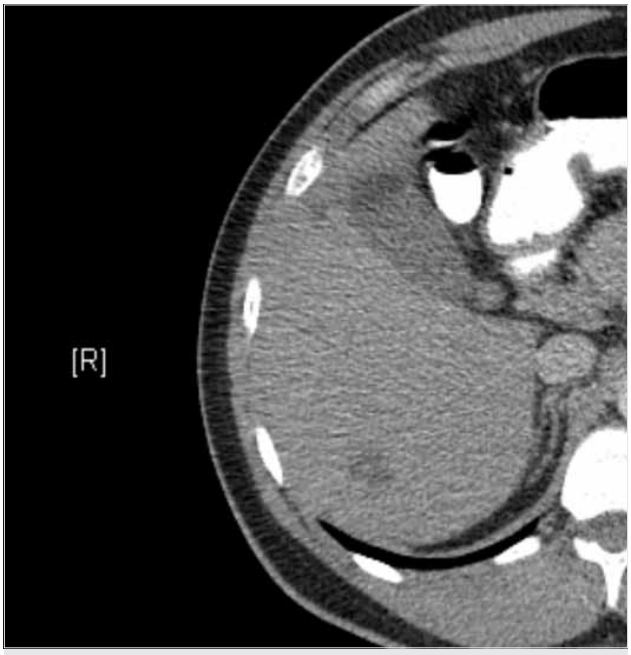

Figure 6. A single hypodense nodule is present in the posterior right lobe on axial contrast-enhanced CT in comparison with Figure 2

Discussion

Fascioliasis is not an uncommon parasitosis. It is caused by trematodes belonging to the *Fasciola* genus and is seen all over the world. This infestation, with a high seroprevalence, especially in rural areas of our country, may be related to ingestion of uncooked or unwashed green herbs, such as watercress (1, 2).

Typical, though nonspecific, clinical symptoms of fascioliasis are fever, hepatomegaly, diffuse upper abdominal pain, and prominent eosinophilia. However, if it is not considered in the differential diagnosis, it is difficult to clinically, microbiologically, and radiologically identify fascioliasis. While right upper quadrant pain, fever, allergic reactions, and significant eosinophilia are observed in the acute period, recurrent biliary colic may be seen in the chronic period. The diagnosis can be based on serological tests or detection of parasite or eggs on direct examination of stool, aspirate bile, or liver tissue (1, 2). While the parasite may not be present in stool during the acute infection period, serological tests may be positive. Antibodies against all parasites, as well as coproantigens and excretory secretory proteins, can be detected by enzyme immunoassay or immunoblot methods. Despite sensitivity values reaching 90%, the specificities of these tests are low (2).

In order to be able to detect *Fasciola hepatica* eggs or parasite, *F. hepatica* metacercaria, after entering the human body, must evolve and reach adulthood. During this evolution phase, lasting 3 to 4 months, symptoms, such as abdominal pain, fever, weight loss, cough, and chest pain, may be present, associated with parasites attacking the intestinal wall, liver tissue, bile ducts, and even lung or brain tissue (1, 2). During this period, in which the eggs can not be detected in stool or bile, the microbiological diagnosis is only possible by serological tests. Successful diagnosis in the early period can be determined in 91% of the cases by using the 78 sandwich ELISA method to detect excretory-secretory (ES) antigen in the stool or blood. Detection of antibody in the serum by DOT-ELISA and ES- ELISA technique has been found to be 97% sensitive and 93% specific (3).

After metacercaria of *Fasciola hepatica* penetrate the intestinal wall or peritoneal cavity from the gastrointestinal tract, they surpass the Glisson capsule and reach the liver. The metacercaria lead to inflammatory events in the liver for 1 to 3 months, later passing to the bile ducts. Nonspecific, microabscess-like lesions with heterogeneous echogenicities are observed during the hepatic invasion period. After transmission to the bile ducts in the second phase, irritation and inflammation of the ducts occur (1, 2).

Although it is non-invasive and inexpensive, US may not be diagnostic in the hepatic phase, secondary to poorly defined nodules. It is more useful in the biliary stage of the disease.

Parasites may be seen as echogenic particles floating in the gall bladder on US examination (1, 4). Multiple or solitary liver lesions, dilatation and edema of the main bile duct, periportal lymphadenopathy, splenomegaly, ectopic inflammations, subcapsular effusion, and liver calcifications are significant CT imaging and MRI findings in hepatobiliary fascioliasis at baseline and long-term follow-up (1).

Associated CT findings can be summarized as multiple, hypodense, often peripherally or centrally localized nodular lesions with blurred margins and a tendency to join, linear or curvilinear hypodense striations, single hypodense nodular lesions or periportal lymphadenopathy, linear hypodensity compatible with isolated subcapsular effusion, main bile duct wall thickening and dilatation of bile ducts, and opacification of the liver capsule (1, 4, 5). Lack of contrast enhancement in hypodense lesions in the hepatic arterial, portal venous, and equilibrium phases is an important feature for differentiation from other focal liver lesions (1). These lesions can be better visualized after contrast injection and either remain hypodense or become isodense with the liver (1, 5). Nodules are nonspecific and may be misinterpreted as necrotic neoplasia and abscess. Acute cholangitis and hepatic abscess formation may develop. Dilated bile ducts and thickening of the liver capsule may be observed. The lesions may decrease in size and disappear due to parasite migration to the bile ducts. Parenchymal

calcifications persist (1, 5). In the biliary phase, the parasite may be visualized in the bile ducts by US, which are dilated and have irregular wall thickening. These findings are also nonspecific and particularly resemble sclerosing cholangitis and cholangitis seen in HIV-positive patients. CT does not provide additional benefit in the biliary phase. In the case of cholangitis, it is virtually impossible to diagnose fascioliasis by CT in the absence of other CT findings, and other etiological causes should be considered. Aspiration under US guidance and serological and microbiological examinations of the material can also be of diagnostic value (1, 4).

Multiple scattered hypodense nodular lesions and accompanying curvilinear hypodense subcapsular striations, as well as effusion, were identified in the liver of our patient. Thickening of the liver capsule in the late phase (equilibrium phase), biliary phase findings, and periportal lymphadenopathy were not observed.

Unlike other trematodes, *F. hepatica* does not sufficiently respond to praziquantel. A single dose of triclabendazole is recommended as first choice for treatment in this infestation, with a successful outcome in over 80% of cases. A second dose may be necessary in cases that are not cured by the first treatment dose. However, a second dose was not required in our patient due to the improvement of symptoms and cholestasis enzymes 1 month after treatment. The clinical efficacy of triclabendazole was investigated in 82 patients and found to be effective and safe at the 20-mg/kg dose (2, 6).

The spectrum of biliary fascioliasis ranges from recurrent colic to acute cholangitis. The long-term complications are gall stones, sclerosing cholangitis, and biliary cirrhosis. In addition to confirmation of the infestation diagnosis in the biliary phase, endoscopic retrograde cholangiopancreatography may provide a treatment opportunity for endoscopic parasite removal (7, 8).

Conclusion

The role of US is limited in the parenchymal phase; US is helpful for diagnosis and follow-up in the biliary phase. MRCP is particularly effective in the biliary stage. CT and MRI are beneficial in the hepatic phase and may also help in reflecting the phase and activity of the disease. Considering that the lesions are best visualized in the portal hepatic venous phase sections, it can be suggested that contrast-enhanced CT and MRI (portal venous phase) are useful in the diagnosis and follow up of hepatic fascioliasis.

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