A Case of *Fasciola hepatica* Mimicking Sepsis without Eosinophilia

Sepsis gibi Prezente Olan Eozinofilisiz *Fasciola hepatica* Vakası

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ABSTRACT

Fasciolosis is a rare cause of hepatobiliary system infections and caused by the trematode *Fasciola hepatica*. It primarily infects sheeps or goats, and humans are accidental hosts. On laboratory findings, marked eosinophilia is present in most of the cases. Here, we report a case of fasciolosis without eosinophilia who was presented as sepsis and responded to therapy in second dose of triclabendazole. Sepsis like clinical presentation has been reported in few cases. Forty-eight year old female patient presented with high fever, abdominal pain, hypotension and tachycardia. The patient was considered as sepsis secondary to liver abscess, which was demonstrated on the initial abdominal ultrasonography (USG) findings. Therefore, empirical antibiotic therapy was started. Due to failure of the treatment, the image was found to be compatible with fasciolosis on control magnetic resonance imaging (MRI) and USG. On detailed anamnesis, history of eating watercress was learned and the diagnosis of fasciolosis was confirmed by serological tests. *(Turkiye Parazitol Derg 2014; 38: 131-4)*

Key Words: Fasciolosis, fasciola hepatica, sepsis

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ÖZET

Fasciolosis hepatobiliyer sistem enfeksiyonlarının nadir bir nedenidir ve etkeni bir trematod olan *Fasciola hepatica*'dır. Primer olarak koyunları ve keçileri enfekte eder, insanlar rastlantısal olarak ana konak olur. Çok az vakada ise sepsis benzeri klinik prezentasyon rapor edilmiştir. Laboratuvar bulgularında belirgin eozinofili vakaların çoğunda mevcuttur.Biz, eozinofili olmaksızın sepsis benzeri tablo ile prezente olan ve ikinci doz triklabendazol tedavisine iyi cevap alınan fasciolosis olgusu sunduk. Kırksekiz yaşında kadın hastayüksek ateş, karın ağrısı, hipotansiyon ve taşikardi ile başvurdu. Hastada ilk plandaki batın ultrasonografi bulgularına göre karaciğer absesine bağlı sepsis düşünüldü. Ampirik antibiyoterapi başlandı. Ancak tedaviye yanıt alınamaması nedeniyle yapılan kontrol USG ve batın MRI incelemelerinde fasciolosis ile uyumlu görüntü saptandı. Anamnez derinleştirildiğinde su teresi yeme öyküsü olduğu öğrenilen hastada fasciolosis tanısı serolojik testler ile kesinleştirildi. *(Turkiye Parazitol Derg 2014; 38: 131-4)*

Anahtar Sözcükler: Fasciolosis, fasciola hepatica, sepsis

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INTRODUCTION

Fasciolosis is caused by the trematode *Fasciola hepatica* and is a rare cause of infections of the hepatobiliary system. It primarily infects sheep or goats, while humans are accidental hosts. Humans acquire these parasites by ingesting metacercariae encysted on contaminated water, watercress, or other aquatic plants. After ingestion of metacercariae, they encyst in the intestine, penetrate the bowel wall through the peritoneum, and then migrate through the liver parenchyma towards the biliary ducts (1, 2). Urticaria, fever, right upper quadrant abdominal pain or intermittent episodes of biliary colic, hepatomegaly, and marked eosinophilia are common signs and symptoms. Marked eosinophilia is present in most of the cases. The diagnosis requires high clinical suspicion. Radiological modalities, stool studies, and serological tests are required for confirmation of the diagnosis. Triclabendazole (TCBZ), bithionol, and praziquantel are currently the effective drugs of choice for the treatment of fasciolosis (3). Here, we report a case of fasciolosis without eosinophilia that was presented as sepsis and responded to therapy at the second dose of triclabendazole.

CASE REPORT

A 48-year-old woman was admitted to our clinic with right upper quadrant pain, nausea, and vomiting for 1 month. In the last 10 days, fever accompanied these complaints. She had no medical history of alcohol or drug ingestion, gallstone disease, or abdominal surgery. Her physical examination revealed right upper quadrant tenderness, hepatomegaly, and ascites. Her body temperature was 38.9°C, heart rate was 130 beats per minute and regular, and blood pressure was 80/60 mm Hg, while the laboratory findings were as follows: leukocyte: 15.000/mm^3, (neutrophil: 79%, eosinophil: 0.2%), alanine aminotransferase (ALT):106 IU/L, aspartate aminotransferase (AST): 82 IU/L, alkaline phosphatase: 175 IU/L (normal range: 98-279 IU/L), total bilirubin: 1.68 mg/dL, C-reactive protein (CRP): 205 mg/L (0-5), and the amylase level was within normal ranges.

The abdominal ultrasonography (USG) performed for abdominal pain demonstrated a normal common bile duct with no gallstones or sludge; however, numerous hypoechoic heterogeneous lesions in the liver parenchyma compatible with liver abscess were present.With these findings, the patient was considered as having sepsis due to liver abscess; after obtaining blood cultures, we empirically initiated imipenem (500 mg four times a day). Despite treatment, the fever persisted with negative blood cultures. Since the general condition of the patient was deteriorating and the levels of ALT and AST were increasing, we performed a follow-up abdominal USG, which revealed multiple hypoechoic heterogeneous lesions, bile duct wall thickening, and a mobile vermiform structure without acoustic shadowing in the gallbladder. This appearance was supposed to be compatible with fasciolosis (Figure 1). Abdominal magnetic resonance imaging (MRI) also demonstrated hyperintense areas on T2-weighted images and hypointense areas on T1-weighted images in the liver parenchyma without dilatation of the intrahepatic or extrahepatic bile ducts (Figure 2). Based on these findings, the patient was re-questioned in detail; she then gave a history of watercress consumption 3 months before. We performed serological tests and stool examination for *Fasciola hepatica*.

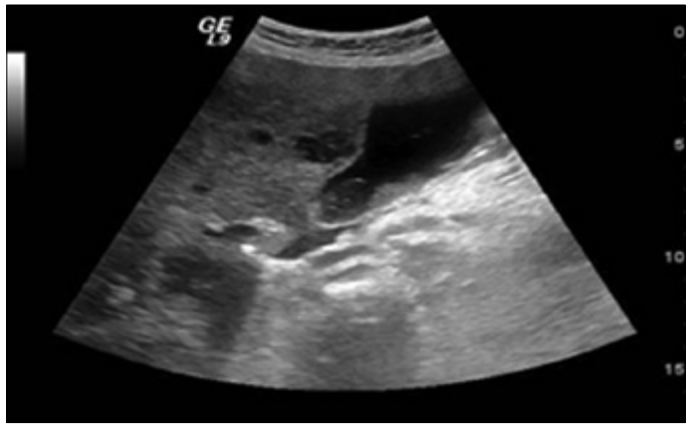

Figure 1. Bile duct wall thickening, and a mobile vermiform structure without acoustic shadowing in the gallbladder

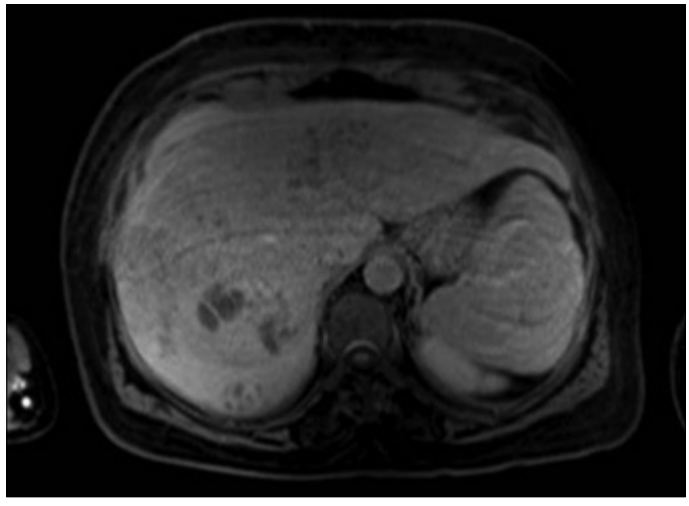

Figure 2. There were multiple lesions in the liver parenchyma without dilatation of the intrahepatic or extrahepatic bile ducts

We did not observe parasite eggs and ova in the stool. Anti-Fasciola hepatica antibody (ab) was determined 2 precipitation arcs, and *Fasciola hepatica* ab was positive at 1/1280 titer by indirect hemagglutination test. The confirmation of anti-Fasciola hepatica antibody specificity was made by the immunoblotting method in France (laboratoire Cerba; Paris, France). Test results: anti-p8-9ab: (++++), anti-p27-28 ab: (++++), anti-p42 ab: (++++), and anti-p60 ab: (++++). With the diagnosis of fasciolosis, 15 mg/kg/day TCBZ was initiated. After single-dose therapy, fever persisted. Due to clinical deterioration, one additional dose of TCBZ was required. The fever decreased, and the liver function tests declined to normal limits at the end of the first week of the second dose. She was discharged and was doing well on the first month of the follow-up visit.

DISCUSSION

The World Health Organization has classified fasciolosis as an important human parasitic disease. Humans acquire these parasites by ingesting metacercariae encysted on contaminated water, watercress, or other aquatic plants (1, 2). Clinical suspicion is very important for diagnosis. History of travel and food consumption should be taken very carefully. Diagnosis of fasciolosis

requires a high index of clinical suspicion in patients admitted with clinical signs, such as right upper quadrant abdominal pain, fever, and jaundice. Eosinophilia is also a significant laboratory finding (3, 4). In our patient, the common clinical signs and symptoms were fever, right upper quadrant abdominal pain and tenderness, nausea, hypotension, and tachycardia. Neutrophilic leukocytosis was present, but the eosinophil count was within normal ranges, and USG revealed numerous hypoechoic heterogeneous lesions compatible with liver abscess. Based on these findings, the patient was primary misdiagnosed for sepsis due to liver abscess. Similar clinical findings may be present in liver abscess, sclerosing cholangitis, cholecystitis, ruptured hydatid cyst, acute viral hepatitis, malignancy, and other parasitoses, such as ascariasis or clonorchiasis (5, 6). Eosinophilia is common in the acute phase of the disease. Eosinophil count is reported to be variable in patients and particularly higher in acute cases. Eosinophil count is not a surrogate marker in the biliary phase of fasciolosis, as in our case (7, 8).

Fasciolosis has two phases: hepatic and biliary. The clinical manifestations of fasciolosis differ according to the stage of the disease. The hepatic phase of the disease begins 1 to 3 months after ingestion of the encysted larva, with penetration and migration through the liver parenchyma toward the biliary duct. This phase is characterized by urticaria, fever, right upper quadrant abdominal pain, hepatomegaly, mild hepatitis, and eosinophilia. USG may not be diagnostic in the hepatic phase. Heterogeneity of the liver parenchyma, hypoechoic nodules, and cystic lesions produced by migration of the trematodes are nonspecific USG findings of the hepatic phase. During the second stage-the biliary phase-the parasite passes in the biliary tract and usually presents with intermittent episodes of biliary colic with or without fever, cholangitis, and jaundice. Abdominal USG provides information, especially in the biliary stage of the disease. Biliary dilation, bile duct wall thickening, and periductal fibrosis are common findings (9, 10). In our case, the first abdominal USG, probably performed at the end of the first stage of disease, was not specific for diagnosis. However, 2 weeks later, follow-up USG additionally revealed vermiform structures in the gallbladder and was compatible with fasciolosis.

Computed tomography and MRI have similar values on the diagnosis; MRI is especially helpful for determining the stage and activity of the disease. Hypointense lesions on T1-weighted and hyperintense lesions on T2-weighted images, dilatation, or wall thickening of the biliary ducts are important findings (11). In our case, MRI also demonstrated similar lesions on liver parenchyma, except for biliary duct enlargement.

Microscopic identification of the characteristic egg and ova in the stool and serological tests may be helpful for the exact diagnosis. In the acute phase of fasciolosis, eggs may not be detectable in stool examination; we also could not detect eggs in stool, which may be explained with the history and clinical findings of our case being compatible with the acute phase (12). Diagnosis should be confirmed by clinical and radiologic findings, supported by serology (13). Antigen detection with ELISA in the serum is very useful for diagnosis and has a sensitivity rate of 92.4% and specificity (14). The specificity of the indirect hemagglutination test is 96.9% for the diagnosis of fasciolosis, while the specificity is 100% by using immunoblotting (15, 16). We could not detect any eggs in the stool examination of the patient. However, the result of serum indirect hemagglutination test for *F. hepatica* was positive at a titer of 1/1280 and confirmed by immunoblotting.

The recommended treatment for fasciolosis is TCBZ (10-20 mg/kg/day). The response to TCBZ is reliable, and it can be used as a criterion of the diagnosis. Generally,

single-dose therapy is enough for response. Nevertheless, multiple doses of TCBZ may be required in severe infections and persistent disease after one dose therapy (17). Due to clinical deterioration, one additional dose TCBZ was required in our case.

CONCLUSION

In conclusion, a high clinical suspicion and history-taking are very important for the diagnosis of fasciolosis. It should be kept in mind that fasciolosis may present without eosinophilia and jaundice and may also mimic sepsis.

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