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Case Report

Abdominal CT scan findings of a child with hepatic fascioliasis: A case report on rarely reported emerging disease[☆]

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ABSTRACT

Fasciolosis is a zoonotic infection caused by trematodes *fasciola hepatica* and *fasciola gigantica*, and humans are incidental hosts. Although infrequently reported in developed nations, it is common in developing countries. Few cases have been reported in Africa, specifically in Ethiopia. This article reports a case of a 4-year-old Ethiopian child who presented with right upper quadrant abdominal pain. His complete blood count showed eosinophilia, and imaging demonstrated lesions at peripheral subcapsular parenchyma and central along the biliary tree. Serologic tests confirmed liver fluke infection with *fasciola hepatica* indirect hemagglutination test titer of 1/4000. Computed tomography imaging appearances of hepatic fasciolosis depend on the phase and course of the disease and should be considered in differential diagnosis of lesions along the biliary tree.

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Introduction

Fascioliasis is a zoonotic parasitic disease caused by liver flukes primarily targeting the liver and biliary ducts [1–3]. *Fasciola hepatica* and *Fasciola gigantica* are the 2 commonly described species causing diseases in humans [1,2]. The parasite has a complex life cycle and humans are one of the final mam-

mal hosts, sheep and cattle being the commonest hosts [3]. Animals are infected by contaminated pasture during grazing, and humans from contaminated raw water plants or cooking utensils [2,3]. Even though the significant negative impact on livestock and their products is well known, it is relatively recent, since the 1990s, that human health-related issues are well described; and expected to increase in incidences due to global warming [2–4].

Abbreviations: IgE, Immunoglobulin E; CT, Computed tomography; ESR, Erythrocyte sedimentation rate.

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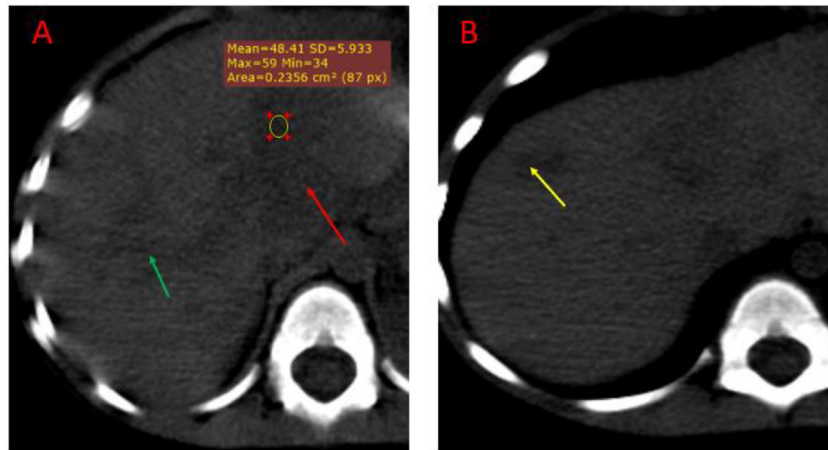


Fig. 1 – Axial noncontrast abdominal CT scan. Image A shows hypoattenuating branching type (green arrow) and hilar (red arrow) lesions. Image B shows a peripheral parenchymal lesion (yellow arrow)

These parasitic diseases are reported in large burden in Latin America, particularly in Bolivia [5]. Relatively less frequent reports have been made in Africa, Asia, and Europe. In Africa, it is noted in only 12 countries, Ethiopia being one of them, although the disease is likely underdiagnosed in most parts of the continent [2,6]. The population's dietary habits play a role in the disease burden. Ethiopians have a culture of consuming raw vegetables which could be one of the reasons for reports in Ethiopia [1,2]. Despite such predisposing habits, reports of the disease in Ethiopia are few [2].

Case presentation

A 3-year-old male child who was relatively healthy 9 weeks back started to complain of right upper quadrant pain and low-grade intermittent fever. 2 weeks before his presentation to our hospital, he developed yellowish discoloration of the eyes. Otherwise, he had no cough, bowel habit change, or significant weight loss. His past medical history was unremarkable and no similar illness in the family. The family lives in Addis Ababa, the capital of Ethiopia. His usual dietary habit commonly includes vegetable sides which consist of lettuce, cabbage, tomato, and pepper. He was vaccinated according to the national program. On physical examination, vital signs were within normal range, and anthropometric measurements were within the normal limit for his age.

On routine investigations, on complete blood count his white blood cell count was elevated at $14,000 \times 10^9/L$ with eosinophilia of 55%, and other cell lines are normal. ESR was elevated, 42mm/hr. Liver function tests were normal. Hepatitis B and C viral markers were negative. Serologic studies showed a raised Ig E level of 3900ku/l and fasciola hepatica indirect hemagglutination test titer was positive at 1/4000. Stool exam was negative. Chest x-ray was unremarkable. Abdominal ultrasound showed multiple small confluent peripheral subcapsular hypoechoic hepatic lesions that extend to the hilum along the portal vein distributions. There was a 3×4 cm confluent hypoechoic lesion at the hilum. No biliary wall thickening or dilatation was seen. The gallbladder has a

free lumen and a normal wall thickness. Unfortunately, ultrasound images were not saved and we just used the report. Pre- and post-contrast abdominopelvic Computed tomography scan (CT) was done 3 weeks after the US scan. The pre-contrast study showed normal-size liver and hypoattenuating branching-type lesions that extend from the periphery to the hilum and a large confluent mass-like lesion at the hilum (Fig. 1A). There are also separate lesions at the subcapsular region which did not show apparent extension to the hilum (Fig. 1B). In the post-contrast study, the peripheral and branching type lesions showed peripheral enhancement, likely representing dilated biliary trees, and the hilar region confluent mass-like lesion shows enhancement (Fig. 2A and C). Peripheral multiple enhancing parenchymal lesions over the posterior segment are seen (Fig. 2B). There was also hypo enhancing periportal lymphadenopathy measuring 2.5×2.6 cm (Fig. 3).

The diagnosis of fasciola hepatica was made based on the patient's clinical presentation and suggestive laboratory findings and imaging studies. The patient was given 2 doses of triclabendazole, 2mg/kg/dose, one week apart. After treatment, the symptoms gradually subsided, and laboratory findings started to normalize several weeks after treatment.

Discussion

Fasciolosis is a snail-borne parasitic disease caused by trematodes of the genus *Fasciola* [4]. Worldwide, both *Fasciola hepatica* and *Fasciola gigantica* are known to cause human disease, but in eastern Africa and particularly Ethiopia, *fasciola hepatica* is the most commonly reported species in both animals and humans [2,4,7]. The infections in humans are from eating leafy water plants or raw fish meat, which is an intermediate host of the parasite [2,3]. The ingested parasite, encysted metacercariae, exodus in the duodenum and penetrates the intestine into the peritoneal cavity and then into the liver capsule to infect liver parenchyma and biliary tree [2]. The pathogenesis in the liver has 2 phases: parenchymal phase and biliary phase. The parenchymal phase starts after the parasite penetrates the liver capsule and ends when

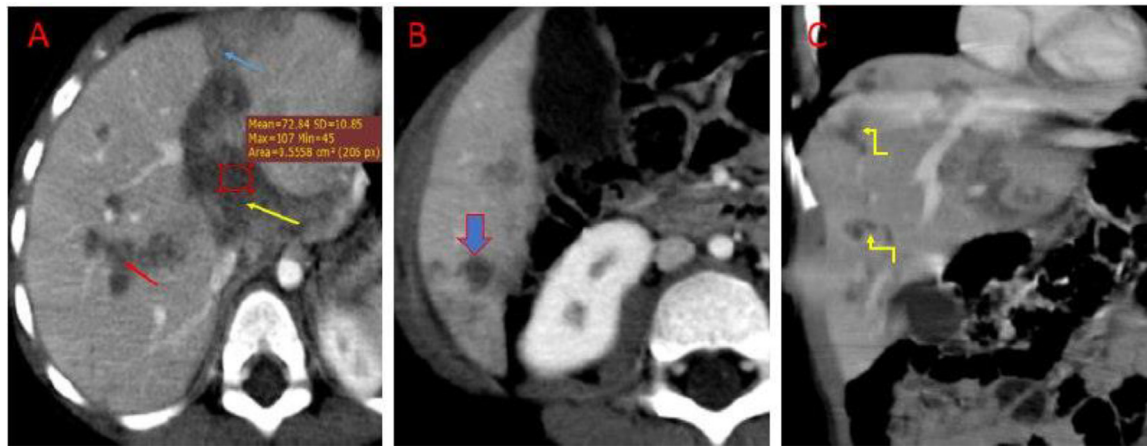


Fig. 2 – Postcontrast abdominal CT axial (A and B) and coronal (C) images: shows an enhancing hilar lesion (A: yellow arrow) which is continuous with the peripheral liver lesion (A: blue arrow). Biliary tree dilations and peripheral enhancement are demonstrated in image A (red arrow) and image C (yellow arrows). Peripheral parenchymal lesion in the posterior liver segment is shown in image B.

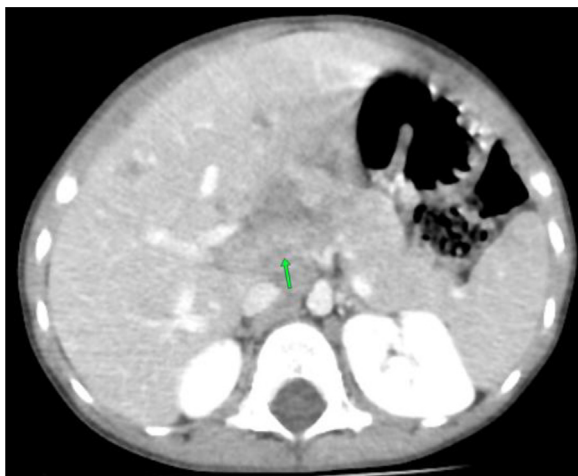


Fig. 3 – Axial contrast-enhanced abdominal CT image showing hypo enhancing periportal confluent lymphadenopathies (green arrow).

it reaches the biliary tree, then biliary phase follows. Both mechanical and chemical damages as well as the clinical manifestations will depend on the degree of infestation. The leaf-shaped adult worms can be seen with the naked eye, *F. hepatica* measuring 3*1.5cm and *Fasciola gigantica* measuring 7.5*1cm [2]. The adult worms will release eggs which will pass through faeces and later hatch to miracidia. The miracidia will be consumed by snails, intermediate host, and cercariae will leave the snail to encyst on aquatic plants and ready to initiate another infectious cycle [2].

Clinical presentations of fasciola infections are widely variable and range from nonspecific systemic manifestations like fever to relatively specific right upper quadrant pain and jaundice [3]. Our patient had an intermittent low-grade fever, right upper quadrant pain and jaundice; which are among the commonly mentioned presentation of this disease entity [8]. More-

over, the clinical presentations are affected by the phase of the disease. The parenchyma phase is the first phase and it starts after the parasite penetrates the liver [8]. Infected individuals at this phase will have manifestations including fever, hepatomegaly, right hypochondriac pain, nausea vomiting, and myalgia; and these features make clinicians consider liver abscess which is by far more common than fascioliasis. The biliary phase is more chronic and patients will be chronically sick looking with weight loss and wasting. They will also have jaundice, malaise, and epigastric and right upper quadrant pain [2,8].

Ultrasound and computed tomography are commonly used imaging modalities. The imaging appearance, extent of findings, and type of findings depend on the duration of illness and phase of infection [9]. In the parenchymal phase, ultrasound will show predominantly peripheral or diffuse hyperechoic or hypoechoic lesions [9]. In our patient a peripheral subcapsular hypoechoic lesions were seen, which is one of the described imaging findings in this phase. These early phase findings can be variable but more consistent after 8 weeks post-infection [9]. In the biliary phase ultrasound will show ductal dilatations with wall thickening [10]. In our patient, hypoechoic confluent type lesions were seen around the hepatic hilum extending from the subcapsular lesions. On the ultrasound report, there was no biliary tree dilatation and the reason for this could be the time gap between the US and CT. The parenchymal findings can be seen together with ductal dilatation [9,10]. These findings were also seen in our case. Computed tomography findings in the parenchymal phase include focal or multiple oval to round peripheral hypodense lesions with peripheral enhancement [9], just like in our case. In the biliary phase, ductal dilatations with periportal thickening are the dominant features [8,10]. In our patient, the CT showed branching-type hypoattenuating lesions with peripheral enhancement consistent with biliary tree dilation and cholangitis. The other frequently described imaging finding is periportal lymphadenopathy [10], which was also seen in our case.

Diagnosis of fasciola is commonly settled by laboratory tests [8]. There are specific confirmatory tests like postmortem examinations and egg detection in stool. Other indirect evidence of infection like ELISA and immunoassay techniques can also be used. Stool egg detection is commonly done but sensitivity is low [4]. In our case, the diagnosis was confirmed by a serological test, fasciola hepatica indirect hemagglutination test titer.

The preferred treatment of choice is oral triclabendazole 10mg/kg which is repeated after 12 hours. This agent is active against both the adult and infective larvae. Less effective alternative agents include oral artesunate, metronidazole, and albendazole [2]. Our patient was treated with 2 doses of triclabendazole given 12 hours apart. He showed clinical and imaging improvement after treatment.

Conclusion

Hepatic fasciolosis usually presents in parenchymal phase but less commonly can present with mixed biliary and parenchymal phases at the same time depending on the time of imaging in the disease course. Here we report a confirmed case of hepatic fasciolosis with imaging appearance of mixed biliary and parenchymal phases.

Availability of data and materials

The data supporting the findings of the case are available upon request to the corresponding author.

Author contributions

All authors contributed to this research and read and approved the final version of the manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent

Written informed consent was obtained from the patient's parents for anonymized patient information to be published in this article.

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