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CASE REPORT

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Liver fluke in a young Nepalese girl: A rare diagnostic puzzle

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Key Clinical message

Fascioliasis poses diagnostic challenges to clinicians for its broad spectrum of hepatobiliary symptoms and lower detection rates. Timely and precise identification avoids long-standing hepatic complications.

Abstract

Fasciola hepatica, a trematode parasite, inhabits snails and sheep. Human hepatic fascioliasis is a neglected tropical disease with no specific tests. In this instance, the significance of clinical awareness, appropriate imaging, and serological investigations is demonstrated. A young Nepalese girl was diagnosed and managed successfully.

KEYWORDS

children, *Fasciola hepatica*, hepatic fascioliasis, liver fluke, MRI

1 | INTRODUCTION

Liver rot is an established disease in domestic and wild ruminants, mostly sheep, goats, and cattle, where the common liver fluke, or *Fasciola hepatica* (*F. hepatica*), is implicated in the chronic parasitic illness.[1,2] This along with *Fasciola gigantica* (*F. gigantica*), a larger variant of the same genus, is known to cause human fascioliasis that mainly affects the hepatobiliary system, causing necrosis, parenchymal abscesses, and subsequent hyperplasia of the bile duct epithelium, resulting in periportal fibrosis.[1] This can pose a diagnostic challenge because of its potential to mimic a wide range of hepatobiliary conditions and lower detection rates.[3]

2 | CASE REPORT

A 5-year-old girl presented to the office with complaints of intermittent episodes of dull-aching sensation over the right upper abdomen for 2 weeks. This was not associated with jaundice, vomiting, fever, or abdominal distention. She had normal bowel and bladder habits and had no prior episodes of similar events.

She was afebrile, hemodynamically stable, comfortable, and clinically anicteric. An abdominal examination revealed mild tenderness over the right hypochondrium without palpable organomegaly. The rest of the systemic examination was unremarkable. Her hemogram revealed a total leukocyte count of 10,760 cells/mm^3, a differential

count of neutrophils 33%, lymphocytes 44%, monocytes 3%, and obvious eosinophilia (20% eosinophils). Her liver function tests, serum tumor markers, and stool microscopic examination were all within normal limits.

An abdominal ultrasonogram (USG) showed a serpiginous hypoechoic structure in segment II of the liver with central hyper-echogenicity surrounding the liver parenchyma, a finding most likely suggesting fascioliasis, as shown in Figure 1. The contrast-enhanced abdominal magnetic resonance imaging (MRI) revealed a tubular non-enhancing area in segments IVa and II of the left lobe of the liver with peripheral mild to moderate enhancement in the liver parenchyma, as shown in Figure 2. Due to the possibility of inadequate yield, a lack of specific pathological findings, and associated risks, an invasive procedure like a liver biopsy was deferred.

She was planned for a course of the broad-spectrum antiparasitic agent nitazoxanide (dose: children 4–12 years, 200 mg twice daily for 7 days) This was administered with a close follow-up.

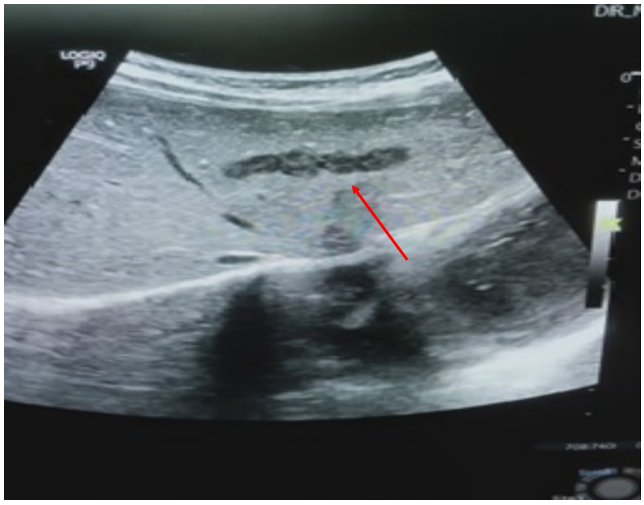

FIGURE 1 A serpiginous hypoechoic structure in segment II of the liver with central hyper-echogenicity surrounding the liver parenchyma.

At every visit, a clinical examination and an ultrasonological assessment were performed. Gradual improvement was noted symptomatically and radiologically. A follow-up abdominal ultrasound after two months showed multiple hypoechoic foci within segment II of the liver that were comparatively smaller in size without posterior acoustic shadowing, indicating soft calcifications with dormant fascioliasis, as shown in Figure 3.

A follow-up hemogram showed normal blood eosinophil counts (2% eosinophils). At one year's follow-up, she remained symptom-free.

3 | DISCUSSION

Human fascioliasis is generally acquired by ingestion of food or water infested with metacercaria, or more specifically, by consumption of aquatic plants like water cress growing in a contaminated environment.[2] Whether adults or children, it is an uncommon and infrequent zoonosis wherein humans are unintentional hosts. Once in the human gut, parasitic excystation occurs soon after the penetration of the duodenal wall, followed by peritoneal invasion, penetration of the Glisson's capsule, and subsequent migration through the liver parenchyma to reach the biliary system. The mature fluke in the biliary tract lays eggs that get excreted in feces to complete the life cycle.[4]

The two clinical stages are observed: 1. The acute stage (hepatic phase) and 2. The chronic stage (biliary phase). In the acute stage, inflammation, necrosis, and subcapsular hemorrhages occur in the liver and clinically manifest with fever, right upper abdominal pain (the most frequent symptom), nausea, vomiting, urticaria, jaundice, and hepatomegaly coupled with eosinophilia.[2] While in the chronic stage, the flukes get localized in the bile ducts or other extrahepatic sites for months or even years, presenting with symptoms of cholangitis, cholestasis, pancreatitis, or biliary obstruction.[2,5]

Although rare, extrahepatic involvement of fascioliasis has also been described, affecting organs like the skin,

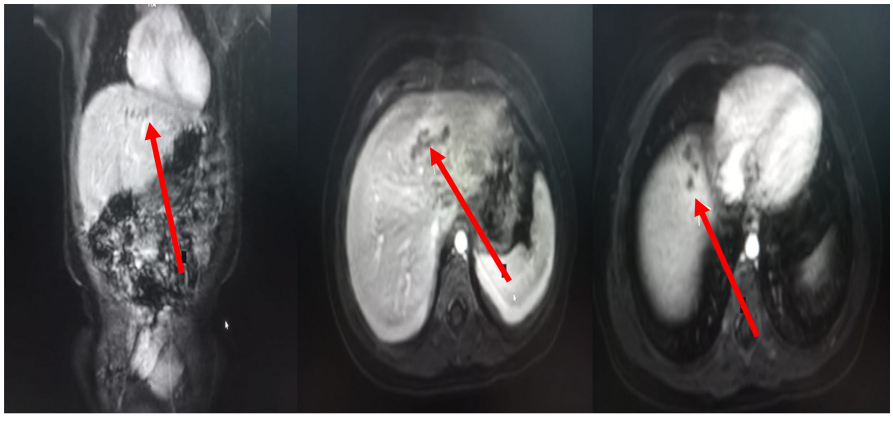

FIGURE 2 Contrast-enhanced abdominal magnetic resonance imaging shows a tubular non-enhancing area in segments IVa and II of the left lobe of the liver with peripheral mild to moderate enhancement in the liver parenchyma.

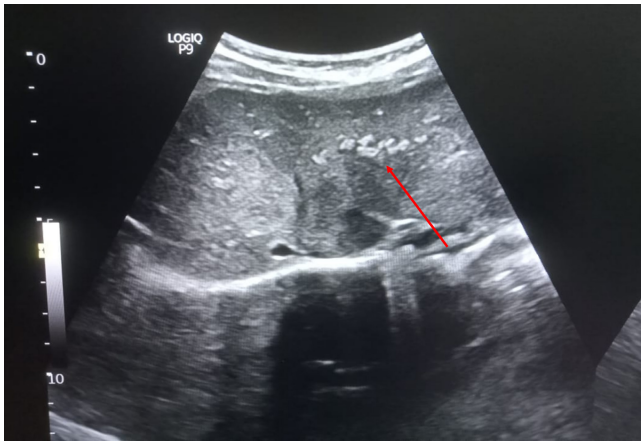

FIGURE 3 An abdominal USG at two-month follow-up shows multiple hypoechoic foci within segment II of the liver without posterior acoustic shadowing, indicating soft calcifications with dormant fascioliasis.

eyes, heart (pericarditis or cardiac conduction abnormalities), and lungs (pleural effusion).[6] In the background of these symptoms, the most striking laboratory finding in both stages is peripheral eosinophilia.[7]

Owing to a long period of latency and a biphasic mode of presentation, early identification of fascioliasis continues to pose a diagnostic challenge. In the hepatic phase, serological tests like enzyme-linked immunosorbent assay (ELISA) and indirect hemagglutination (IHA) tests are expected to have a high diagnostic yield.[3,8] But due to a lack of easy availability, both of these tests could not be used on our patient.

In the biliary phase; however, the diagnosis relies on the detection of eggs in the bile or stool, albeit with lower sensitivity. Furthermore, a variety of invasive techniques, such as fine-needle bile aspiration from the gallbladder or endoscopic or percutaneous transhepatic bile sampling from the duodenum or biliary ducts, have been suggested depending on individual suitability and expertise.[9]

Occasionally, moving parasites in the gallbladder or the dilated bile ducts can be demonstrated on a USG. In the absence of mobile parasites, only non-specific changes like focal hypo/hyperechoic lesions, ductal ectasia, wall thickening, or diffuse involvement of the liver can be seen on USG.[6] Computed tomography (CT) scan and MRI features might vary depending on the stage of the disease process at presentation. On CT, focal capsular thickening and enhancement secondary to the penetration of parasites through the liver capsule in the hepatic phase are noted, while residual parenchymal calcification might be seen in the biliary phase.[9] Hepatic parenchymal lesions are best diagnosed on CT, as it can appropriately identify the classical "tunnel and caves sign" corresponding to the path of migration of *F. hepatica* through the liver parenchyma.[10]

An MRI, on the other hand, demonstrates the characteristic evolutionary pattern of fascioliasis, reflecting the life cycle of the parasite in the early parenchymal phase even without contrast as capsular hyperintensity on T2-weighted images. It may also provide additional details about complications such as hemorrhagic lesions and abscess formation.[9] Endoscopic retrograde cholangiopancreatography (ERCP) appears to have a dual role in the diagnosis and treatment of fascioliasis, as it allows the detection and extraction of motile parasite(s). Common findings are linear, elliptical, or rounded filling defects inside a dilated biliary or pancreatic duct.[10]

Many other disease processes presenting with similar image findings can be taken into consideration; nonetheless, fascioliasis should always be suspected when there are numerous confluent non-enhancing hypodense lesions aligned within a tract next to the liver capsule.[2] Biopsy of these lesions may reveal eosinophilic infiltration, coagulation necrosis, or microabscesses, none of which are of specific diagnostic value.

Therapeutically, triclabendazole is an available and well-tolerated option for pediatric fascioliasis, both in the acute and chronic phases. A single dose of 10 mg/kg or two 5 mg/kg doses are usually effective with a high cure rate.[11] Most adverse effects seem to be related to the expulsion of dead or dying flukes from the hepatobiliary system, with commonly observed ones being abdominal pain, sweating, obstructive jaundice, elevated hepatic enzymes, urticaria, pruritus, rashes, headache, dizziness, and vertigo.[11] However, with the re-emergence and increasing incidence of the disease, triclabendazole resistance has also been observed globally owing to alteration in the mechanisms of drug uptake, efflux, and detoxification as suggested mechanisms of resistance. An alternate drug, nitazoxanide, a broad-spectrum antiparasitic agent with minimal adverse effects, is seen to be equally effective and well tolerated, as was noted with our patient.[7]

4 | CONCLUSION

Hepatic fascioliasis is a zoonosis with the potential for delay or misdiagnosis, resulting in complications secondary to long-standing hepatic inflammation. In endemic regions, when children present with nonspecific abdominal findings in the background of eosinophilia and suggestive radiological findings, fascioliasis should always be considered a possibility.

AUTHOR CONTRIBUTIONS

Anwesha Pandey: Conceptualization; data curation; formal analysis; investigation; methodology; writing – original draft; writing – review and editing. **Aakash Mishra:**

Conceptualization; data curation; formal analysis; investigation; methodology; writing – original draft; writing – review and editing. **Sagar Khadka:** Data curation; investigation; supervision; writing – review and editing. **Sunil Raja Manandhar:** Data curation; formal analysis; investigation; methodology; supervision; writing – review and editing. **Ashish Lal Shrestha:** Conceptualization; data curation; formal analysis; investigation; methodology; supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT
The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT
The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT
Not needed.

CONSENT
Written informed consent was obtained from the patient's guardian for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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