Unknown etiology of acute hepatitis: invasive liver flukes

Bahaa Osman Taha[1] , Essam Abdel Mohsen [1]

[1]Assiut University Internal Medicine Gastroenterology Unit

Corresponding ***author:*** *Bahaa Osman Taha, Lecturer of Internal Medicine.*

Address: Egypt, Assiut, Elgamaa Street, postal code: 71515.

Email: bahaa_osman99@aun.edu.eg.

*Doi: **10.21608/ajgh. 2025.392731.1084.***

Submit Date: 08-6-2025.

Revise Date: 25-08-2025.

Accept Date: 16-09-2025.

Background

Fasciola hepatica is a rare infectious zoonotic disease that seldom manifests as acute hepatitis. We present a case from Upper Egypt, specifically in Manfalout, Assiut, a region known to be endemic for fascioliasis. The patient, a 25-year-old male, displayed symptoms such as anorexia, vomiting, malaise, right hypochondrial pain, and fever, and was initially diagnosed with acute hepatitis. We carefully examined a patient with isolated acute hepatitis who had high levels of ALT and AST and normal levels of bilirubin and ALP to rule out viral hepatitis, autoimmune hepatitis, and

Wilson's disease. Radiological findings, including abdominal ultrasound and MRCP, revealed mild hepatomegaly and a thick gallbladder wall with no dilated biliary channels. There was eosinophilia, elevated C-reactive protein, and a high titre of fasciola antibody, suggesting fascioliasis. A single dose of albendazole at 10 mg/kg showed a significant response, resulting in an improvement in liver enzymes and a marked improvement in all reported manifestations.

Keywords: Liver flukes, acute hepatitis, eosinophilia.

Introduction

Fascioliasis is an emergent infectious disease. Egypt is considered a hotspot for fascioliasis, with a human prevalence rate of 7–11% and notably high infection rates in livestock, affecting up to 60% of cattle. [1]. Additionally, in Upper Egypt, particularly in the Assiut Governorate, periodic outbreaks of human fascioliasis have been associated with climatic and irrigation factors. These outbreaks typically peak during the summer and autumn months. [2].

Patients often overlook the early stage of liver inflammation due to the diverse range of nonspecific symptoms, such as abdominal pain, fever, and chills. Furthermore, a definitive diagnosis is typically made at a later stage, when obstructive jaundice becomes apparent.

Case report

A 25-year-old male patient presented with an acute onset of right hypochondrial abdominal pain accompanied by fever, chills, anorexia, and vomiting. An outpatient clinic initially diagnosed him with acute non-calcular cholecystitis after he sought medical advice. The diagnosis was based on

a clinical and bedside abdominal ultrasound examination, which revealed a thick gallbladder wall with positive Murphy signs. The patient received empirical medical treatment, consisting of antibiotics, antipyretics, and spasmolytics.

After one week, the patient's symptoms intensified, leading to his recurrence and subsequent admission to our hospital. The initial routine investigations revealed acute hepatitis, moderately elevated liver enzymes (ALT 659 U/L, ASL 410 U/L), and normal serum bilirubin (8.6 μmol/L).

During the physical examination, the patient exhibited a body temperature of 38.3°C, a heart rate of 110 beats/minute, a blood pressure of 120/70 mmHg, and a respiratory rate of 22 breaths/minute.

The patient looked ill with an average body build. A systemic examination revealed mildly enlarged hepatomegaly; other systemic examinations were not remarkable.

We carefully examined the patients' backgrounds to determine what caused their acute hepatitis. These tests revealed the presence of hepatitis A (HAV IgM), HBV (HBsAg, HBcIgM), HIV, CMV, infectious mononucleosis, autoimmune hepatitis (negative ANA, normal total IgG), and Wilson disease (normal serum ceruloplasmin and standard 24-hour urinary copper excretion). All investigations were negative. Abdominal ultrasound revealed mild hepatomegaly, a thick gall bladder wall, and minimal free intraperitoneal fluid collection.

The patient received symptomatic nonspecific treatment in the form of antipyretics, antiemetics, proton pump inhibitors, and hepatic support in the form of silymarin. Unfortunately, follow-up patients continue to complain of hallmark symptoms such as abdominal pain, fever, and a 'toxic' appearance. The complete blood count reveals an elevated white blood cell count, with

eosinophilia being particularly pronounced. The patient denied any previous drug history or exposure to toxic substances that could explain acute hepatitis and eosinophilia.

The presence of fever and elevated levels of C-reactive protein (56.8 mg/l) strongly suggests an infectious cause. The stool analysis conducted over three consecutive days using repeated samples showed no abnormalities.

The patient was also investigated for typhoid fever and brucellosis using the Widal and Malta tests, respectively.

The reported patient resides in Manfalout, Assiut, a region known for its endemic fascioliasis, primarily due to issues with the water source. Along with the presence of eosinophilia and elevated CRP levels, the indication of an infectious source heightens the concern about fascioliasis.

The Fasciola antibody titre was significantly elevated by 1/1280, with a normal reference range of 1/160, which is the reason for the presence of eosinophilia. Abdominal MRCP revealed only slight enlargement of the liver with a normal diameter of the common bile duct and no apparent signs of dilatation of the biliary channels.

We had planned to perform a liver biopsy, but the patient declined to proceed. Consequently, we opted to initiate a therapeutic trial for fascioliasis.

Unfortunately, triclabendazole was not available at this time, so we administered a single dose of albendazole to the patient at a dosage of 10 mg/kg. Two weeks after the follow-up, the patient showed clinical improvement, with her liver enzymes returning to within the normal range (ALT 36 U/l, AST 22 U/l) and her fasciola antibody level decreasing to less than 1/160.

The patient was monitored monthly for liver function and clinical findings over three consecutive months, during which he exhibited no abnormalities. Throughout the follow-up period, we focused on preventive measures, given that the reported case occurred in an endemic region where there is a risk of reinfection.

Discussion

Fasciola hepatica is a zoonotic infection that is commonly undiagnosed during the early hepatic stage, resulting in a late diagnosis and the appearance of persistent symptoms during the biliary stage, such as jaundice and abdominal pain.[3]. Contaminated food serves as a means of transmitting the illness to humans, who act as intermediate hosts in the life cycle of a trematode fluke. [4].

Due to its nonspecific symptoms, such as fever, eosinophilia, and abdominal pain, the acute invasive stage of fascioliasis often remains undiagnosed. This stage can persist for up to 4 months, during which adult flukes migrate to the biliary ducts and trigger an inflammatory response that leads to obstructive jaundice. Both acute hepatitis and cholangitis typically have an uncommon presentation, with a slight elevation in liver enzymes. [5].

The patient's clinical appearance, along with significantly elevated ALT and AST liver enzymes by more than 10 times, indicated a diagnosis of acute hepatitis, which was extensively investigated for relatively common etiologies.

In this case, eosinophilia is considered a key indicator of fasciola suspicion. While eosinophilia is a common presentation of fasciola infection in approximately 40–80% of cases [6]There are other conflicting differential diagnoses of eosinophilia, such as drug-induced hepatitis. [7].

However, the invasive stage of the flukes, which causes acute hepatitis in our patients, may explain the presence of disproportionately severe abdominal pain, fever, a toxic look, and sonographic findings of minimal free intraperitoneal fluid. [8].

Stool analysis is often inconclusive during the early invasive stage and may yield negative results. In contrast, serological antibody titers play a more significant role during the acute stage of our case, as these assays can detect infection weeks before egg shedding occurs. This serological assay offers a critical diagnostic advantage during the acute phase and may serve as a reliable tool for the early (prepatent) diagnosis of fascioliasis. [9].

Single-dose albendazole may be effective in treating acute hepatitis caused by the invasion stage of fasciola infection; however, triclabendazole remains the preferred treatment for fascioliasis. Albendazole can serve as an alternative, particularly in cases where resistance is present or non-availability.[10].

Conclusion:

Fasciola infection should be considered in patients with unexplained acute hepatitis-associated eosinophilia, especially in endemic regions.

Footnotes.

Peer-Reviewers: Ahmed Fathy (Professor of Internal Medicine), Ahmed Gad (Professor of Internal Medicine), Nevin Fouad (Professor of Internal Medicine).

E- Editor: Salem Y Mohamed, Emad Fawzi Hamed.

This work was conducted in accordance with the CARE guidelines.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interests

There are no conflicts of interest related to this study.

Acknowledgments: The authors express their heartfelt gratitude to the resident doctors in the Internal Medicine Department, Gastroenterology Unit, at Al-Raghi Hospital—Dr. Dina Sameh, Dr. Flobater Talaat, Dr. Nardine Nady, and Dr. Demina Adel—for their committed contributions to patient management.

We obtained written consent from the patient.

Author contributions:

Bahaa Osman Taha contributed to the conception, design, writing, and data analysis of the study.

Essam Abdelmohsen contributed to the investigation, material preparation, and data collection.

All the authors have read and approved the final manuscript.

The patient gave written consent to participate in the study.

The data are available as requested.

References.

1. Zanaty N, Ibrahim N, Ramadan HK-A, Ahmad AA, Saad-Hussein A. Significance of climate change in the emergence of human fascioliasis in Upper Egypt. Trop Dis Travel Med Vaccines. 2024; 10:24. doi:10.1186/s40794-024-00234-z.

2. Dar Y, Amer S, Mercier A, Courtioux B, Dreyfuss G. Prevalence of *Fasciola hepatica* infection in cattle, buffaloes, and humans in the Nile Delta, Egypt. Parasites Vectors. 2017; 10:470. doi:10.1186/s13071-017-2412-z.

3. Lee RY, White RV, Shin BJ, Collins BG. Hepatic parasitic abscess resistant to traditional anthelmintics: difficulty of diagnosis and treatment of fascioliasis in the USA. BMJ Case Rep. 2021;14(7): e242613. doi:10.1136/bcr-2021-242613.

4. Keiser J, Utzinger J. Food-borne trematodiases. Clin Microbiol Rev. 2009;22(3):466-83. doi:10.1128/CMR.00012-09.

5. Tanabe M, Caravedo M, White A, Cabada M. An update on the pathogenesis of fascioliasis: what do we know? Res Rep Trop Med. 2024; 15:13-24. doi:10.2147/RRTM.S397138.

6. Kaymak M, Babür CC, Olcay L. *Fasciola hepatica* infection as a cause of severe hypereosinophilia. Turk J Hematol. 2013;30(1):88-90. doi:10.4274/tjh.2012.0016.

7. Martínez-Cabriales SA, Shear NH, González-Moreno EI. Liver involvement in the drug reaction, eosinophilia, and systemic symptoms. World J Clin Cases. 2019;7(6):705-16. doi:10.12998/wjcc. v7.i6.705.

8. Lalor R, Cwiklinski K, Calvani NE, et al. Pathogenicity and virulence of the liver flukes *Fasciola hepatica* and *Fasciola gigantica*, which cause the zoonotic disease fasciolosis. Virulence. 2021;12(1):2839-67. doi:10.1080/21505594.2021.1996520.

9. Espinoza JR, Maco V, Marcos L, Sáez S, Neyra V, Terashima A, et al. Evaluation of Fas2-ELISA for the serological diagnosis of human fascioliasis. Am J Trop Med Hyg. 2007;76(5):977-82. doi:10.4269/ajtmh.2007.76.977.

10. Babják M, Königová A, Burcáková Ľ, Komáromyová M, Dolinská M, Várady M. Assessing the efficacy of albendazole against *Fasciola hepatica* in naturally infected cattle by in vivo and in vitro methods. Vet Sci. 2021;8(11):249. doi:10.3390/vetsci8110249.