International Journal of Research in Medical Sciences
Mahto BK et al. Int J Res Med Sci. 2025 Feb;13(2):897-901
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20250278

Case Report

Understanding the recurrence of amoebic liver abscess in South Bihar: a clinical case report

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Received: 15 December 2024
Revised: 10 January 2025
Accepted: 15 January 2025

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ABSTRACT

Amoebic liver abscess is a common disease condition in some parts of India, including Bihar, due to the higher prevalence of amoebiasis and habitual consumption of toddy (tari). Recurrence of amoebic liver abscess is not uncommon; although it is underreported with only a few previous reports. In this study, a patient from South Bihar presented with three subsequent episodes of amoebic liver abscess over a period of six years. Possible considerations for these recurrences have been explored, which revealed the fact that recommended full-dose treatment with metronidazole followed by a luminal amebicide agent to eradicate intestinal amoebic colonization is necessary for effective treatment of an amoebic liver abscess.

Keywords: Amoebic liver abscess, Entamoeba histolytica, recurrence, luminal amebicide

INTRODUCTION

Amoebiasis is an infection of the gastrointestinal tract caused by *Entamoeba histolytica* (a parasitic amoeba) transmitted through the faeco-oral route.[1] Other non-pathogenic Entamoeba species are *Entamoeba dispar*, *Entamoeba moshkovskii*, *Entamoeba polecki*, *Entamoeba coli* and *Entamoeba hartmanni*, which are intestinal colonizers. 80% of amoebiasis cases are asymptomatic, while 20% symptomatic cases can present with diarrhoea, abdominal pain, fever and other constitutional symptoms. Amoebic liver abscess (ALA) is the most common extra-intestinal form of invasive amoebiasis.[2]

Amoebic abscesses can occur in all age groups but are 10 times more common in adults than in children, and are more frequent in males.[3] It is seen predominantly in tropical and subtropical climates and is more common in low socio-economic and unhygienic populations.[4] ALA occurs frequently in toddy or tari (a locally fermented palm wine made by a complex procedure) consumption areas, prevalent in some endemic parts of India like Bihar and Jharkhand.[5]

Presentation

20% of ALA patients had a history of amoebic dysentery. About 10% of patients presented with episodes of gastroenteritis at the time of diagnosis of amoebic liver abscess.[6] Most of the cases usually presented with acute onset upper abdominal pain and high-grade fever. The pain is intense and constant, commonly in the right upper quadrant and radiating to the scapular region and right shoulder; it increases with deep inspiration, coughing, or when the patient rests in the right decubitus position. Pain due to the left lobe abscess occurs in the epigastrium or on the left side of the abdomen and can radiate to the left shoulder. Fever is present in majority of the cases; it varies between 38°C and 40°C, frequently intermittent but sometimes constant over several days, with chills-rigors

and profuse sweating. Anorexia (a prominent symptom), weight loss (can occur early), nausea, vomiting, headache and fatigue may all be present.[7]

On physical examination

Mild icterus is quite common, but severe jaundice, however rare, can occur in complicated cases. There is restricted movement in the right hemithorax and thus, hypoventilation of the right lower lobe of the lung. The cardinal sign of amoebic liver abscess is painful hepatomegaly. Digital pressure will often produce intense pain in the liver region. On palpation, the liver is usually soft and smooth. A hard liver does not exclude the diagnosis as ALA and cirrhosis may coexist, especially in alcohol and toddy drinkers.

Hepatomegaly may not be appreciated on palpation, but increased liver span due to upward enlargement is noted in many of the cases if percussion is allowed to be performed. Intercostal tenderness most commonly at the lower right intercostal spaces, is a frequent and reliable sign even in the absence of diffuse liver pain.[8] The abruptly presenting symptoms can mimic an acute surgical abdomen.

Lesions are usually single and predominantly (~90%) found in the right lobe of the liver. The incidence of left lobe ALA ranges from 5% - 21%. The liver abscess has a thin capsular wall with a necrotic centre composed of thick material, an intermediate zone of coarse stroma and an outer zone of nearly normal tissues. Typically, abscess fluid is odourless, resembling 'chocolate syrup' or 'anchovy sauce', and sterile, although secondary bacterial infection may occur.[9] Microscopic examination of the abscess fluid shows granular eosinophilic debris with few cellular components. Amoebae are usually located at the periphery of the abscess. The term 'abscess' is a misnomer as the 'pus' is better described as liquefied necrotic liver.

Diagnosis

Amoebic liver abscess is diagnosed using a combination of clinical profile, diagnostic imaging, and serological and microbiological analyses.

Imaging

An ultrasound is the preferred initial diagnostic test for clinically suspected cases due to its quick results, cost-effectiveness, and wide availability.[10] It is particularly effective in localising abscesses; their size measurements and distinguishing them from a tumor.

In the early stages of abscess formation, non-contrast computed tomography (CT) imaging offers superior results compared to later stages. Radiographic examination may often reveal an elevated and fixed right dome of the diaphragm. Sigmoidoscopy can demonstrate the characteristic multiple amoebic ulcers, from which biopsy samples may be obtained at the edges of the ulcers.

Blood examination

In early cases, leucocytosis may be observed, while chronic cases often present with anemia. Additionally, raised serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase may be detected.

Serological tests

Detection of Entamoeba antibodies via enzyme-linked immunosorbent assay (ELISA), which boasts a sensitivity of 90%, is a valuable tool for diagnosis.[11] Negative antibody titers effectively exclude amoebic etiology as a possibility. Recently, reverse transcription loop-mediated isothermal amplification (RT-LAMP) has emerged as a sensitive, specific, and rapid diagnostic method for amoebic liver abscess (ALA).

Stool examination

The presence of hemoglobin, neutrophils, cysts, and trophozoites may be identified in stool samples. It is recommended to collect three fresh stool samples for analysis. Standard microscopy is capable of detecting the parasite in less than 50% of cases, as trophozoites may be destroyed by factors such as drying, water, and barium. However, recent advancements in molecular testing have improved detection rates significantly.

Aspiration from liver

Diagnostic liver aspirates obtained from abscess capsules are instrumental in differentiating ALA from pyogenic liver abscess.

Complications

Potential complications include pleuropulmonary involvement (20-30%), which may manifest as sterile effusion, contiguous spread, rupture into the pleural space, or the development of hepatobronchial fistula. There is also a risk of rupture into the pericardial space (particularly with left lobe abscess) or peritonitis. Hematogenous spread can lead to abscesses in the lungs, brain, spleen, and kidneys, as well as genitourinary lesions and meningitis.[12] The estimated mortality rate is approximately 0.2–2.0% in adults, rising to as high as 26% in the pediatric population.[13]

CASE REPORT

A 34-year-old male farmer was admitted to the Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, for evaluation and management of recurrent amoebic liver abscess, which was refractory to metronidazole therapy. He has experienced three episodes of ALA over the past six years (Table 1). The patient is a Hindu male from the Nalanda district, an area with a higher incidence and prevalence of ALA, and he comes from a

low socio-economic background. Though he has a history of alcohol and toddy consumption, he denies recent intake. There is no reported history of anal intercourse, travel outside the region, or significant animal contact. Furthermore, he has no known history of immunocompromised conditions.

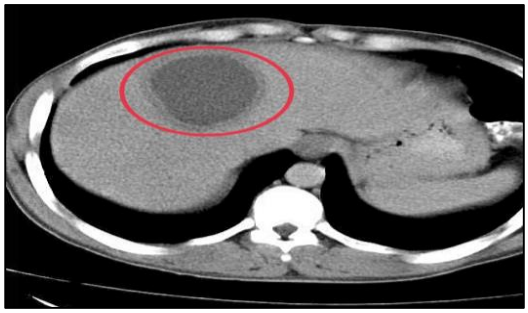

Figure 1: Solitary cystic liver lesion with low attenuation center and peripheral thick enhancing wall.

The patient's index presentation was characterized by abdominal pain, fever, loss of appetite, and malaise. After a thorough evaluation, he was diagnosed with an amoebic liver abscess (ALA) measuring 8×7 cm, as confirmed through ultrasonography, serology, and stool examinations. Treatment comprised a one-week course of injectable metronidazole and ceftriaxone, which resulted in significant symptomatic improvement without the necessity for surgical intervention. Five years later, the patient returned with a new episode marked by a one-week history of fever, chills, rigors, sweating, and vomiting. Upon admission, ultrasonography indicated the presence of a larger liver abscess (12×10 cm), confirmed to be of amoebic origin through serological examinations and stool microscopy. He was treated with injection meropenem and metronidazole for ten days, leading to substantial recovery through conservative management, again without the need for surgical intervention.

One year after this episode, the patient experienced a recurrence, presenting with fever, chills, sweating, and right upper quadrant pain, with intercostal tenderness noted upon examination. After readmission for further evaluation, a contrast-enhanced computed tomography (CECT) scan of the

abdomen revealed a liver abscess measuring 10×9 cm (Figure 1). Stool microscopy identified the presence of Entamoeba cysts, and the enzyme-linked immunosorbent assay (ELISA) for amoebiasis returned a positive result at a titer of 1:320. Additionally, stool PCR for Entamoeba was positive, while pus culture results were negative. The patient was treated with injectable metronidazole (750 mg TID) for ten days, followed by a ten-day course of diloxanide furoate (500 mg PO TID), resulting in marked clinical improvement. A repeat stool PCR for Entamoeba performed four weeks' post-treatment yielded a negative result, confirming the effectiveness of the intervention.

Table 1: Clinical spectrum of different episodes of amoebic liver abscess (ALA).

	First episode (2018)	Second episode (2023)	Third episode (2024)
Age at presentation	28 years	33 years	34 years
Symptoms	Fever, upper abdominal pain, anorexia and malaise for 1 month	Fever with chills, sweating and vomiting for 1 week	Fever with chills, sweating and upper abdominal pain for 5 days
Clinical examinations	Temp- 99°F, pallor, tender hepatomegaly, intercostal tenderness	Temp- 99.5°F	Temp- 100°F, intercostal tenderness
Lab investigations	Hb- 6.8 g/dl, TLC-14000/cubmm, CRP- 38 mg/dl, ALT- 45 U/l, AST- 88 U/l, ALP- 266 U/l	Hb- 11g/dl, TLC-24000/cubmm, CRP- 77 mg/dl, ALT-89 U/l, AST-145 U/l, ALP- 389 U/l	Hb- 10g/dl, TLC-34000/cubmm, CRP- 88 mg/dl, ALT-181 U/l, AST-266 U/l, ALP- 465 U/l
Imaging	Ultrasound revealed right lobe ALA of size 8×7 cm	Ultrasound revealed right lobe ALA of size 12×10cm	Right lobe liver abscess of size 10×9cm (CECT Abdomen)
Stool microscopy	Entamoeba cysts and trophozoites+	Negative	Entamoeba cysts+
ELISA for amoebiasis	1:640	1:80	1:320
Abscess fluid culture	Not done	Not done	Negative
Treatment given	Metronidazole 500 mg iv TID for 7 days Ceftriaxone 1g iv BD for 7 days	Metronidazole 500 mg iv TID for 10 days Meropenem 1g iv TID for 10 days	Metronidazole 750 mg iv TID for 10 days Diloxanide furoate 500 mg PO TID for 10 days

DISCUSSION

The recurrent nature of the amoebic liver abscess in this case can be attributed to several factors. During the first episode, the patient was treated with injection metronidazole at a subtherapeutic dose of 500 mg TID for a shorter duration of 7 days. Current guidelines recommend 750 mg TID for 10 days, followed by 10 days of treatment with a luminal amoebicide.[12] The suggested dosages for luminal agents include Diloxanide furoate (Amicline) 500 mg PO TID, Nitazoxanide (Nizonide) 500 mg PO BD, Paromomycin 500 mg PO TID, Iodoquinol 650 mg PO TID, diiodohydroxyquinoline (Iodometrol) 300 mg PO TID and Quinidochlor (ENERO QUINOL) 500 mg PO TID. In managing the second episode, a similar subtherapeutic dose of metronidazole was administered, and no luminal agent was provided.

Singh et al, found a recurrence rate of 9% within 2 years among patients who were not treated with a luminal agent.[14] Additionally, a study by Irusen EM et al, reported a 72% prevalence of asymptomatic luminal colonization by pathogenic *E. histolytica* at the initial presentation of amoebic liver abscess, suggesting the importance of using a luminal amoebicide.[15] Treatment with metronidazole alone for the eradication of intestinal colonization proved insufficient in more than 50% of cases, even when the liver abscesses were resolved.[15] Numerous studies suggest that without the inclusion of a luminal amoebicide, metronidazole therapy is ineffective in managing asymptomatic colonization.[16-19]

While *E. histolytica* may develop metronidazole resistance in vitro, clinical data on resistance remain poorly understood.[20] Although the patient denied recent consumption of toddy and alcohol, the possibility of amoebic liver abscess due to recent toddy consumption cannot be ruled out, given his history of alcoholism and the high prevalence of toddy use in his region. Furthermore, the patient's low socio-economic status and poor hygiene could have also contributed to the recurrence of the amoebic liver abscess.

CONCLUSION

In this case report, we emphasize the critical role of luminal amoebicides in the effective management of amoebic liver abscess. Relying solely on metronidazole therapy is insufficient for completely eradicating intestinal amoebic colonization, often leading to repeated episodes of relapse. Therefore, it is essential to follow metronidazole treatment with a 10-day course of a luminal agent, such as diloxanide furoate or paromomycin. This approach is vital for ensuring comprehensive treatment and preventing recurrence.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Mahto BK, Kumar S, Kumar S, Kumari R, Shreya, Kushwah HS, Kumar A, et al. Understanding the recurrence of amoebic liver abscess in South Bihar: a clinical case report. Int J Res Med Sci 2025;13:897-901.