



CASE REPORT

Classic extraintestinal amoebiasis diagnosed through unconventional methods: a case report

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ABSTRACT

Amoebiasis, caused by *Entamoeba histolytica*, is a prevalent cause of dysentery in Southeast Asia. Conventional diagnosis heavily relies on non-invasive modalities like stool microscopy and serology; however, these diagnostic modality frequently suffer from low sensitivity and are confounded by regional endemicity. We present a rare case of invasive extraintestinal amoebiasis in a 78-year-old immunocompromised male with a history of diffuse large B-cell lymphoma. The patient presented with a three-week history of severe watery diarrhea, fever, and significant weight loss, ending in hypovolemic shock. Serial stool wet mount examinations were repeatedly negative for parasites, and *Clostridioides difficile* testing was negative. Due to his immunosuppressed status, a diagnostic colonoscopy was performed, revealing diffuse mucosal inflammation and severe sigmoid ulcerations. Histopathological examination of targeted colonic biopsies provided the definitive diagnosis, revealing numerous tissue-invasive trophozoites exhibiting erythrophagocytosis, which was further enhanced via Periodic acid–Schiff staining. The patient was successfully initiated on oral metronidazole. On day 22 of admission, hepatobiliary ultrasound screening revealed a concurrent 11 cm amoebic liver abscess, which required ultrasound-guided percutaneous drainage. Systemic exposure was retrospectively validated by a strong positive amoebic IgG serology. Following a sequential course of luminal eradication with paromomycin and targeted antibiotics, the patient achieved complete clinical recovery and was discharged well. This case highlights that when conventional non-invasive screening tools fail, an unconventional, multi-modal diagnostic approach utilizing early histopathology and proactive imaging is essential to optimize survival outcomes in complex, immunocompromised hosts.

Keywords: *Entamoeba histolytica*; immunocompromised host; diagnosis; histopathological examination; amoebic liver abscess.

INTRODUCTION

Amoebiasis caused by the non-flagellated single-celled protozoan *Entamoeba histolytica*, remains a significant public health concern. It is transmitted via ingestion of infectious amoebic cyst from contaminated water or food, with the parasite alternates between a highly resilient infective cyst stage and a pathogenic trophozoite stage (Mahon & Lehman, 2023; Morán *et al.*, 2023). In Malaysia, *Entamoeba* spp. has been identified as the most prevalent intestinal protozoa in the general population (Mizan *et al.*, 2025). While most infections are asymptomatic, symptomatic cases typically manifest as diarrhea, abdominal pain, and fever (Farhana *et al.*, 2009). However, *E. histolytica* infection can also progress to invasive extraintestinal disease, presenting profound diagnostic challenges. This is particularly true in immunocompromised hosts, where

extraintestinal amoebiasis often presents with highly atypical or vague clinical symptoms that mimic other opportunistic infections or malignancies, frequently delaying crucial therapeutic intervention (Sepahvand *et al.*, 2022).

This diagnostic complexity is further intensified by the limitations of conventional laboratory modalities. Standard stool wet mount microscopy exhibits low clinical diagnostic yields in invasive scenarios, frequently failing to detect the pathogen during extraintestinal dissemination (Doganci *et al.*, 2004; Haque *et al.*, 1998; Luna-Nácar *et al.*, 2016). Furthermore, while serological and molecular assays are available, standard blood-based antibody screening faces significant baseline limitations in geographic regions where *Entamoeba histolytica* is endemic, often complicating clinical interpretation during acute presentations (Genasan *et al.*, 2026). Conversely, advanced molecular assays, although sensitive, remain

inaccessible in most clinical centers due to high costs. Consequently, a clear diagnostic gap exists when standard non-invasive protocols fail to yield a definitive diagnosis. In such scenarios, clinicians must rely on unconventional or underutilized diagnostic pathways.

Here, we report a rare case of extraintestinal amoebiasis in an immunocompromised patient where serial stool microscopy was negative. Diagnostic confirmation was achieved through an unconventional, multi-modal approach combining histopathological examination (HPE) of colonic biopsies, serial imaging, and targeted serological correlation, highlighting a critical diagnostic framework for resource-limited or challenging clinical settings.

CASE REPORT

A 78-year-old gentleman with a background history of hypertension, dyslipidaemia, stage 3A chronic kidney disease (CKD) and diffuse large B-cell lymphoma (DLBCL) of the neck and ocular region currently in remission for five months, presented with watery diarrhea and intermittent fever for three weeks. He had watery stools more than ten times daily without noticing any blood in his stools. These symptoms were associated with abdominal cramps and reduced appetite leading to weight loss of five kg over the same period. The fever was intermittent in nature and accompanied by chills and rigors. There was no history of recent travel, recreational water exposure, jungle trekking, and no similar symptoms were reported among family members or close contacts. He had been prescribed with prophylactic trimethoprim-sulfamethoxazole 480mg once daily (OD) in view of his malignancy and was compliant with his medications. Prior to this presentation, he had fully recovered from an episode of herpes zoster treated symptomatically by a general practitioner.

On further questioning, the patient initially presented to the emergency department of another hospital, where he was discharged with symptomatic treatment. However, his symptoms persisted despite being compliant to the prescribed medications. Upon presentation to our centre, he was found to be in hypovolemic shock with a blood pressure (BP) of 90/59 mmHg and a heart rate (HR) of 106 beats/minutes (bpm). Following intravenous fluid (IV) resuscitation, his hemodynamic parameters stabilized with his BP normalized to 120/52 mmHg, HR reduced to 87 bpm, temperature of 36.9 C and oxygen saturation (SpO₂) was 99% on room air.

On physical examination, the patient was alert but appeared dehydrated and pale, with a capillary refill time of less than two seconds. The abdomen was soft, non-tender with no organomegaly. Review of other systems was unremarkable, except for the presence of fresh blood on digital rectal examination. Initial laboratory investigations revealed a white blood cell count (WBC) of $8.9 \times 10^3/\mu\text{L}$ (Neutrophils: 80.1%, Lymphocytes: 7.1%, Monocytes: 11.2%, Eosinophils: 0.4%), hemoglobin (Hb) of 8.3 g/dL, hematocrit (HCT) of 26.8%, and a platelet count of $481 \times 10^3/\mu\text{L}$. Serum electrolytes and metabolic panels showed sodium of 138mmol/L, severe hypokalemia at 2.7mmol/L, lactate of 1.3mmol/L, bicarbonate of 23.8mmol/L, corrected calcium of 2.30 mmol/L, magnesium of 0.85 mmol/L, and phosphate of 1.21mmol/L. Liver function tests indicated hypoproteinemia (total protein: 58g/dL, albumin: 26g/dL), total bilirubin of 21.3 mg/dL, elevated alkaline phosphatase (ALP) at 322 U/L, alanine transaminase (ALT) at 124.3 U/L, and aspartate transaminase (AST) at 111 U/L. The C-reactive protein (CRP) was markedly raised at 23.22mg/dL. A peripheral blood picture confirmed moderate anemia of chronic disease with features suggesting mixed nutritional deficiencies, while the leukocytic shift and reactive thrombocytosis pointed toward an active infectious or inflammatory process. An abdominal radiograph demonstrated non-specific gas patterns without evidence of perforation or obstruction.

Based on the history and clinical findings, a preliminary diagnosis of severe acute infective gastroenteritis was made. Differential diagnoses included cytomegalovirus (CMV) colitis, relapse of lymphoma with gut involvement, tuberculosis (TB) of the gastrointestinal tract, and inflammatory bowel disease (IBD). Empirical therapy was initiated with IV ceftriaxone 2 g stat, followed by 1 g OD and IV pantoprazole 40mg OD. Serial blood and stool cultures were collected, and a colonoscopy was scheduled.

On Day 6 of admission, colonoscopy was performed, revealing the presence of ulcers and friable colonic mucosa extending from the rectum to the caecum with loss of vasculature noted particularly in the caecum. A diverticulum was observed in the ascending colon. Severe erythema with multiple deep ulcers was seen in the sigmoid colon (approximately 50cm from the anal verge) and rectum (approximately 15 cm from the anal verge). Targeted punch biopsies were obtained from the ulcerated areas and sent for HPE.

Tissues were also sent for *Mycobacterium tuberculosis* (MTB) culture, which showed no growth after eight weeks of incubation. Both blood and stool cultures were negative, and no parasites were identified on stool wet mount examination. Moreover, *Clostridioides difficile* antigen and toxin tests were negative. However, HPE of the colonic biopsies revealed numerous small, rounded trophozoites characterized by vacuolated cytoplasm and a single eccentric nucleus with a prominent karyosome. Erythrophagocytosis (the ingestion of red blood cells by the trophozoites) was observed within the lamina propria confirming invasive amoebiasis (Figure 1). The morphology of these parasites was further enhanced using Periodic-acid Schiff (PAS) staining (Figure 2A), while Ziehl-Neelsen staining to look for acid fast bacilli and CMV were negative (Figure 2B and 2C).

Targeted antimicrobial therapy with oral metronidazole 750mg three times daily (TDS) was initiated accordingly. Empirical IV piperacillin-tazobactam 4.5g TDS was administered alongside metronidazole for a week to address potential bacterial translocation through the deeply ulcerated mucosa. The metronidazole was maintained for a total duration of 21 days, followed by a one-week

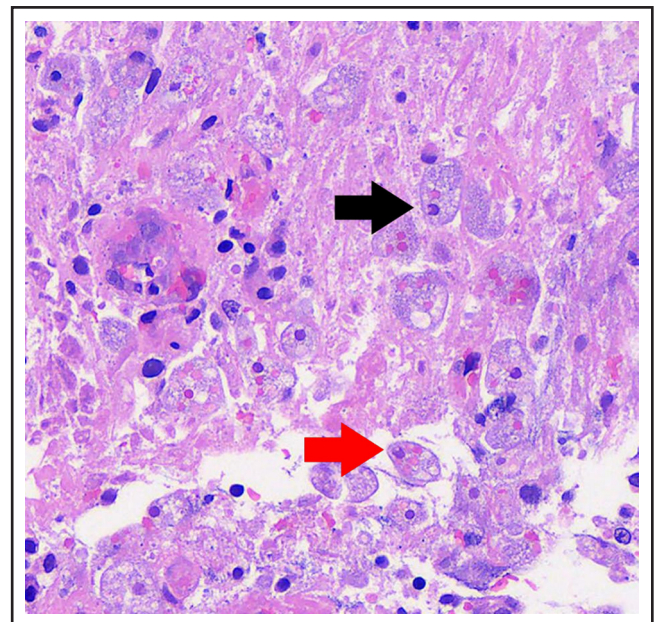


Figure 1. Hematoxylin and Eosin (H&E) staining of tissue biopsy. Note the presence of small, rounded trophozoites with vacuolated cytoplasm and a single eccentric nucleus and prominent karyosome [black arrow]. Some of the trophozoites has ingested red blood cells (erythrophagocytosis) [red arrow].

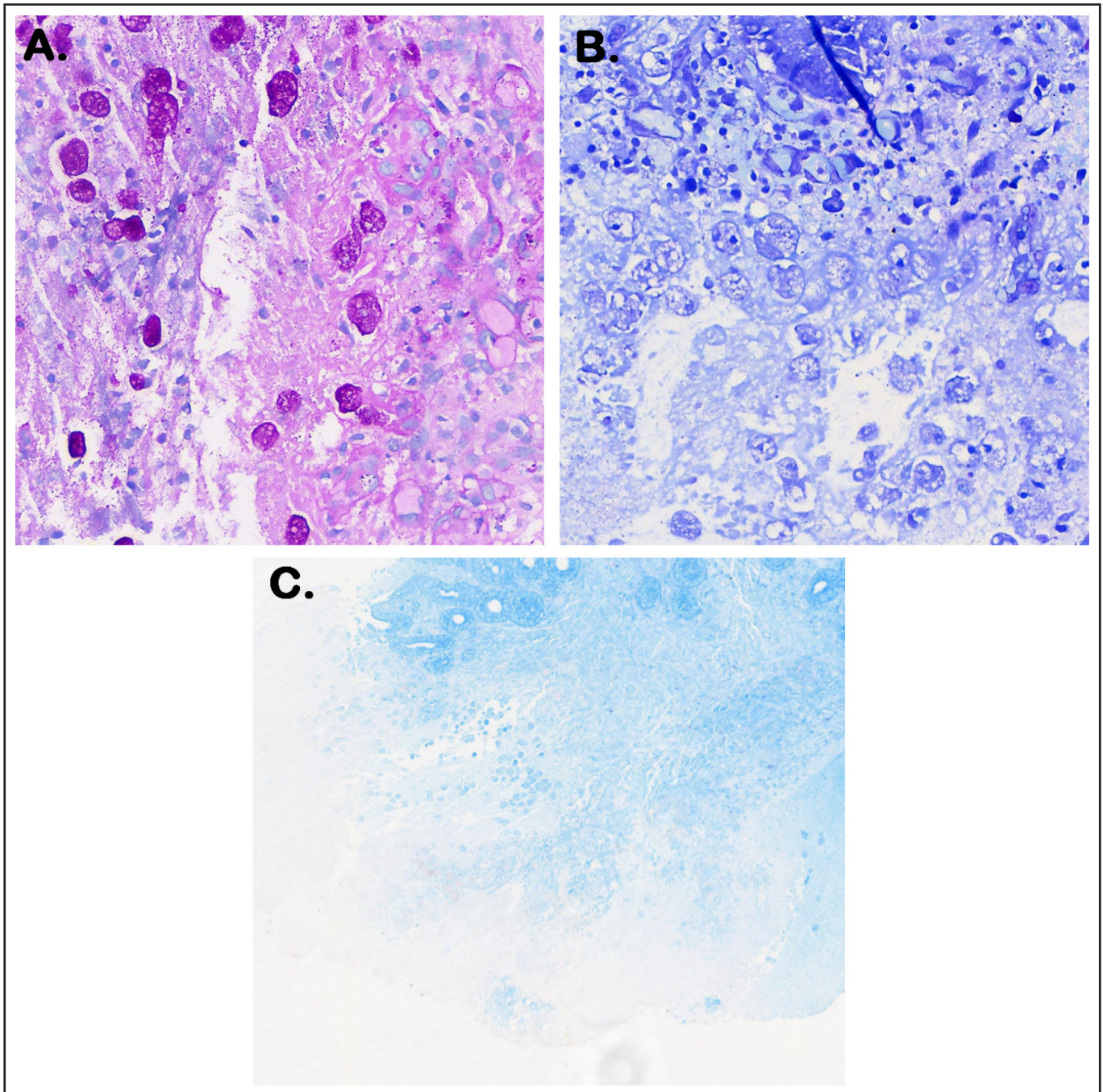


Figure 2. A) Periodic-Acid Schiff (PAS) stain highlights the parasites in magenta with pale staining intracytoplasmic vacuoles; B) Ziehl-Nelson (ZN) stain was negative for acid-fast bacilli; C) CMV staining was negative for any organisms.

course of oral paromomycin 500mg TDS to eradicate luminal cysts, as outlined in the clinical timeline (Figure 3).

On Day 22 of admission, despite significant improvement in gastrointestinal symptoms, an ultrasound of the hepatobiliary system was performed to screen for extraintestinal complications, given the invasive nature of his colonic disease and his immunocompromised status. The scan revealed a large, thick-walled echogenic collection in the liver measuring 11.2 cm x 10.9 cm x 10.7 cm without internal vascularity, confirming a concurrent amoebic liver abscess (Figure 4). Two days later, an ultrasound-guided percutaneous drainage of the liver abscess was performed, and a pigtail catheter was inserted, draining approximately 780 mL of pus over one week.

Culture of the drained pus yielded no growth, and wet mount examination detected no parasites. Concurrently, amoebic serology

demonstrated a highly elevated optical density (OD) of 3.847, which was reported as strongly positive for anti-amoebic IgG antibodies, providing essential retrospective validation of systemic exposure in tandem with histopathological findings. Intravenous ceftriaxone 1g twice daily (BD) was instituted for three weeks along with ongoing treatment for amoebiasis, to mitigate risks of secondary bacterial infection of the massive hepatic cavity.

The patient demonstrated progressive clinical and laboratory improvement (Figure 3) and was successfully discharged after 62 days of hospitalization. Upon discharge, therapy was switched to oral amoxicillin-clavulanate 625mg TDS for six weeks to achieve cavity sterilization, with a scheduled outpatient follow-up for sequential targeted hepatic ultrasonography to monitor abscess cavity regression.

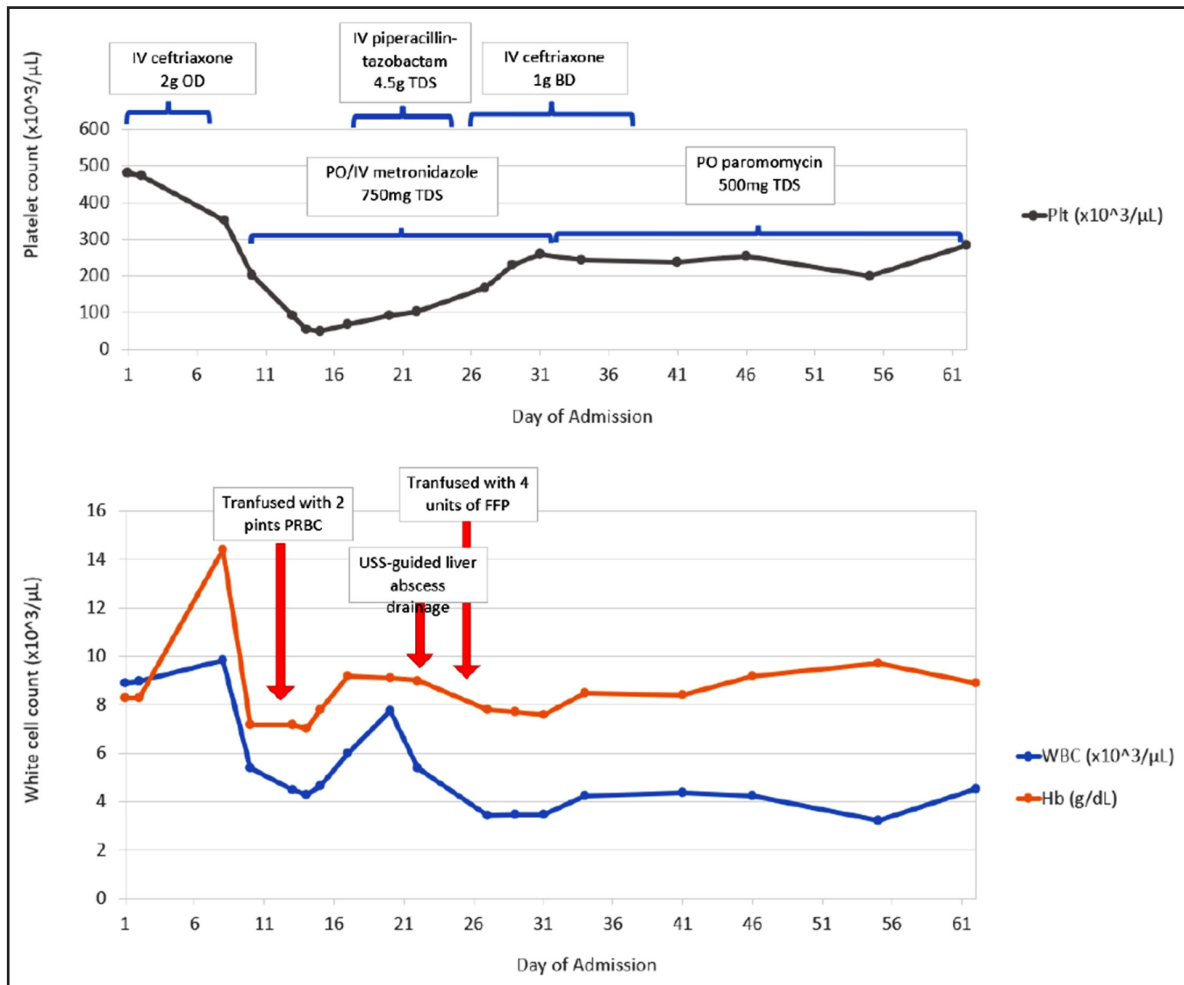


Figure 3. Clinical timeline in relation to antimicrobial therapy and complete blood count trend throughout the patient's admission. NB: Blue line – White blood cell; WBC (x10³/μL), Orange line – Hemoglobin; Hb (g/dL), Grey line – Platelets; Plt (x10³/μL).

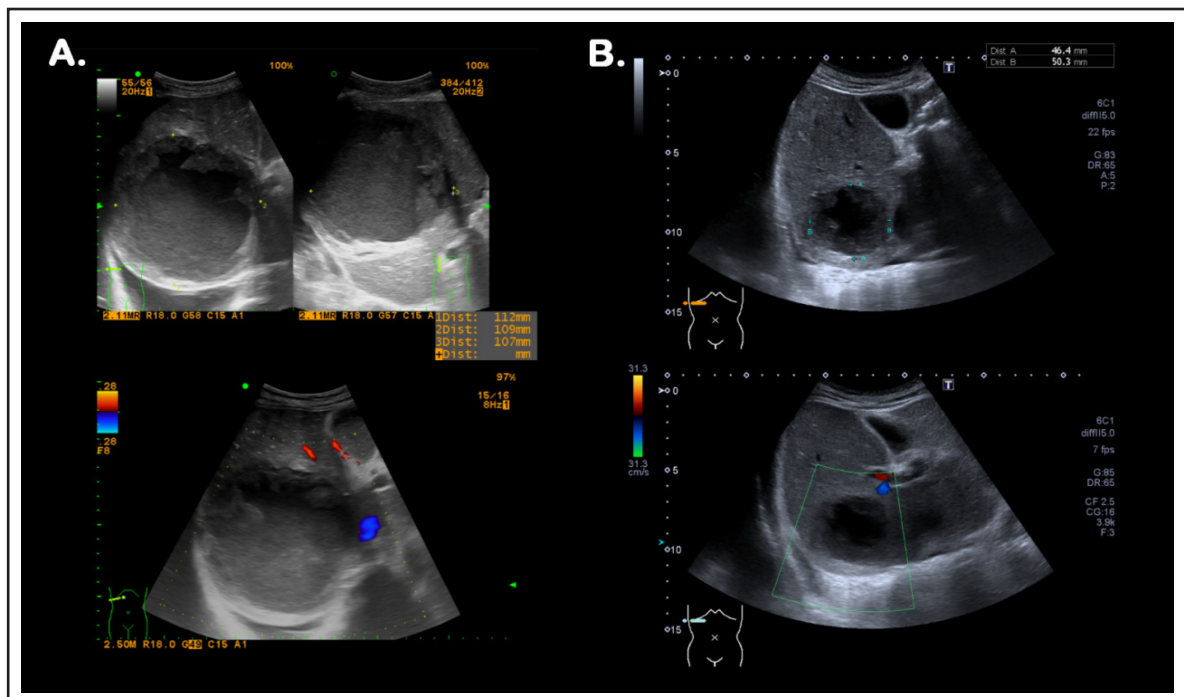


Figure 4. Ultrasound of the hepatobiliary system at Day 22 of admission. (A) Top: Presence of a large, thick-walled echogenic collection at the right liver lobe measuring 11.2cm x 10.9cm x 10.7cm. Bottom: Doppler ultrasonography demonstrates that the lesion is non-vascular; (B) Ultrasound of the abdomen at Day 44 of admission (the abscess has reduced to 4.6 cm x 5.0 cm in size).

Table 1. Summary of blood investigation throughout the patient's admission

DOA	D1	D2	D8	D10	D13	D14	D15	D17	D20	D22	D23	D27	D29	D31	D34	D41	D46	D55	D62
Date	1/1	2/1	8/1	10/1	13/1	14/1	15/1	17/1	20/1	22/1	23/1	27/1	29/1	31/1	3/2	10/2	15/2	24/2	3/3
WBC ($\times 10^3/\mu\text{L}$)	8.9	8.98	9.84	5.38	4.47	4.3	4.64	6.01	7.74	5.40	3.38	3.41	3.45	3.46	4.25	4.38	4.24	3.21	4.51
ANC	6.68	6.47	6.62	4.83	3.55	3.0	3.29	4.42	6.0	3.82	2.11	1.97	1.77	1.45	2.06	2.23	1.94	1.93	2.62
Hb (g/dL)	8.3	8.3	14.4	7.2	7.2	7.0	7.8	9.2	9.1	9.0	8.0	7.8	7.7	7.6	8.5	8.4	9.2	9.7	8.9
Plt ($\times 10^9/\mu\text{L}$)	481	473	351	201	92	53	47	67	91	102	113	168	230	259	243	238	252	199	282
CRP (mg/mL)	23.22	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
PT (sec)	–	18.0	–	23.7	–	–	22.1	–	–	33.2	–	23.6	–	29.1	–	15.5	–	–	14.0
PTT (sec)	–	42.7	–	46.4	–	–	59.9	–	–	82.2	–	N/A	–	>180	–	41.4	–	–	51.3
INR	–	1.36	–	1.82	–	–	1.69	–	–	2.62	–	1.81	–	2.28	–	1.15	–	–	1.03

Abbreviation: DOA, Day of Admission; WBC, White blood cell ($\times 10^3/\mu\text{L}$); Hb, Haemoglobin (g/dL); Plt, Platelets ($\times 10^3/\mu\text{L}$); CRP, C-reactive protein (mg/dL).

DISCUSSION

Amoebiasis caused by protozoan *Entamoeba histolytica*, remains a critical public health challenge in tropical and subtropical regions especially in areas with poor sanitation. In Malaysia, the documented prevalence of gastrointestinal amoebiasis varies significantly. A-10-year retrospective data from a Malaysian university hospital estimated prevalence rates between 1.0% and 14.4% (Jamaiah & Shekhar, 1999). More recently, a comprehensive systematic review demonstrated a pooled local prevalence of 7.0%, although with profound heterogeneity ranging from 1.0% to 47.0% across different individual studies (Genasan *et al.*, 2026). This wide statistical disparity is closely linked to geographical variations, study designs, and population demographics, with past local surveillance focused on symptomatic individuals or specific indigenous communities. While global mortality rates for extraintestinal manifestations like amoebic liver abscess (ALA) remain low, ranging from 0.8% in the United States to 1.2% in regional settings like Thailand, underreporting and misdiagnosis remain major obstacles to understanding the true disease burden (Congly *et al.*, 2011).

The conventional diagnostic workflow for intestinal protozoal infections relies almost exclusively on non-invasive screening tools, yet these diagnostic modalities present significant technical limitations. Stool wet mount microscopy is inherently compromised by its extreme reliance on immediate sample processing and the technical expertise of the laboratory personnel (Fotadar *et al.*, 2007). Trophozoites degrade rapidly and lose their characteristic motility unless samples are evaluated within one hour of shedding (Tanyuksel & Petri, 2003).

Furthermore, standard light microscopy cannot reliably differentiate *E. histolytica* from morphologically identical, non-pathogenic commensals such as *E. dispar*, *E. moshkovskii*, or *E. bangladeshi* (Sardar *et al.*, 2023). Even the microscopic hallmark of erythrophagocytosis (ingestion of erythrocytes by trophozoites) is no longer considered specific to *E. histolytica*, as it has been occasionally documented in *E. dispar* infections (Kantor *et al.*, 2018). In our patient, serial stool wet mounts were repeatedly negative, providing a false sense of diagnostic reassurance that initially delayed the consideration of a parasitic etiology.

While IgG serology offers an alternative non-invasive pathway, its diagnostic utility is heavily confounded in endemic settings like Malaysia. A significant portion of the local population harbors persistent baseline antibody titers from previous exposure; consequently, a positive IgG result cannot reliably distinguish an active, acute extraintestinal infection from a past infection (Genasan *et al.*, 2026). This diagnostic ambiguity indicates that serology serves better as a retrospective validation rather than a primary decision-making tool, thereby necessitating a reliance on tissue-based diagnostics. Definitive discrimination requires advanced molecular tools like quantitative PCR (qPCR), which remain inaccessible in many

secondary and tertiary clinical centers due to cost constraints and infrastructure limitations (Anuar *et al.*, 2012).

In the presented case, resolving the diagnostic dilemma required a shift toward an unconventional diagnostic pathway consisting of early colonic biopsy and HPE with complementary PAS staining. Furthermore, diagnosis of a parasitic diarrheal illness via tissue biopsy is highly unconventional and typically reserved for ruling out malignancies or inflammatory bowel disease (IBD) in non-endemic regions (Cooney *et al.*, 2024). However, given the patient's history of hematological malignancy, diagnostic colonoscopy was expedited to exclude a lymphoma relapse.

Histopathological examination provided the definitive diagnosis in this case, enabled by the visualization of numerous rounded trophozoites invading the lamina propria established a diagnosis of tissue-invasive amoebiasis. Although the biopsy fragments were too small to preserve the classic morphology of a 'flask-shaped' mucosal ulcer (Pritt & Clark, 2008), the direct visualization of tissue-invasive trophozoites bypassed the diagnostic limitations of routine stool examinations.

The severe clinical course observed in this case emphasizes the complex interplay between parasite virulence and host immune defenses. The transition from asymptomatic luminal colonization to invasive, extraintestinal disease is mediated primarily by host factors, including advanced age, nutritional status, and explicit immunosuppression (Marie & Petri, 2014).

Similar to our case, Sepahvand *et al.* (2022) reported that immunocompromised patients with invasive amoebiasis frequently present with atypical, vague, or silent clinical symptoms that mimic severe bacterial sepsis or gastrointestinal malignancies, which often results in critical delays in targeted therapy. However, our case features a highly unique presentation; despite our patient's advanced age and profound immunosuppression secondary to recent chemotherapy for DLBCL, he did not experience rapid, fulminant bowel perforation or toxic megacolon. Instead, his extraintestinal disease manifested as a massive, 11 cm amoebic liver abscess that remained entirely asymptomatic from a hepatic perspective, which was discovered only on Day 22 via ultrasound screening. This highlights a crucial clinical lesson where, in the context of altered cell-mediated immunity, extensive extraintestinal dissemination can develop silently without the classic focal signs of right upper quadrant pain or localized abdominal tenderness. This demonstrates that deep tissue invasion and hematogenous spread via the portal circulation do not always trigger the expected inflammatory alerts in an immunocompromised host.

The primary therapeutic strategy for invasive amoebiasis displays a coordinated, multi-phased antimicrobial approach aimed at eliminating both tissue-invasive trophozoites and persistent luminal cysts (Cooney *et al.*, 2024). In this case, targeted therapy was initiated with oral metronidazole, a potent nitroimidazole tissue amebicide. Metronidazole undergoes reductive activation within

the anaerobic environment of the parasite, generating reactive cytotoxic intermediates that disrupt helical deoxyribonucleic acid (DNA) structures and inhibit nucleic acid synthesis (Hernández Ceruelos et al., 2019). While generally well-tolerated, its prolonged use necessitates careful monitoring for rare but serious neurotoxic adverse reactions, such as peripheral neuropathy or encephalopathy.

Tissue amebicides such as metronidazole undergo extensive absorption in the upper gastrointestinal system, which often prevents them from reaching sufficient therapeutic concentrations within the colonic lumen. This pharmacokinetic limitation leaves up to 10% of patients at risk of passing infectious cysts or experiencing a clinical relapse. Achieving absolute parasitological clearance therefore requires a multi-phased approach; in this case, the 21-day metronidazole course was followed sequentially by a seven-day course of paromomycin. As an aminoglycoside with negligible oral bioavailability, paromomycin remains localized within the bowel lumen, binding directly to the 16S ribosomal RNA subunit to disrupt protein synthesis in luminal trophozoites and cysts (Bissuel et al., 1994). Both metronidazole and paromomycin share overlapping gastrointestinal side-effect profiles including nausea and abdominal cramps, leading to both were administered sequentially rather than concurrently. This deliberate, phased approach successfully achieved total clinical and parasitological clearance without compounding gastrointestinal toxicity in an already fragile, immunocompromised patient.

CONCLUSION

This case demonstrates the importance of deploying a flexible, multi-modal diagnostic approach when managing severe diarrheal illness in vulnerable, immunocompromised patient populations. Reliance on conventional stool microscopy alone carries an unacceptably high risk of false-negative results, which delay crucial antiparasitic intervention. When standard non-invasive testing fails to match the clinical picture, clinicians should not hesitate to utilize alternative diagnostic pathways including early endoscopic tissue biopsy, special histopathological staining, and serial cross-sectional imaging. Maintaining a high index of suspicion and utilizing these unconventional diagnostic tools is essential to ensure rapid pathogen identification, prevent fulminant complications, and optimize survival outcomes in complex clinical settings.

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Conflict of Interests

All authors have no conflicts of interest to declare. The case study was exempted from the local Medical Research and Ethics Committee review and obtained the approval with NMRR ID-26-02319-FYP. Written informed consent was obtained from the patient for publication of this case report as well as any accompanying images.

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