ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

Amoebiasis (Zaheer-e-Sadique): Integrating Classical Unani Descriptions with Modern Insights on Epidemiology, Pathogenesis, Diagnosis, and Management – A Comprehensive Review

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Publication Date: 2026/05/08

Abstract: Amoebiasis, caused by the protozoan Entamoeba histolytica, remains a significant global health burden, responsible for an estimated 50 million infections and 55,000 deaths annually, with 2.2 million disability-adjusted life-years (DALYs) lost each year. The infection is transmitted via the faecal-oral route and predominantly affects populations in low-income countries with poor sanitation, though cases are increasingly identified in high-income countries due to international travel, migration, and sexual transmission among men who have sex with men (MSM). Clinically, amoebic colitis presents with a gradual onset of bloody diarrhoea, abdominal pain, and tenesmus, closely mimicking inflammatory bowel disease (IBD). Extraintestinal manifestations, of which amoebic liver abscess (ALA) is most common, represent a significant cause of morbidity and mortality. In classical Unani medicine, this condition was described under the heading of Zaheer-e-Sadique, with descriptions by Hippocrates, Ibn-e-Sina (Avicenna), Zakariyya Razi, and Ali ibn Abbas Majusi bearing remarkable concordance with the modern clinical picture. Diagnosis relies primarily on stool Entamoeba histolytica PCR, which must be specifically requested, alongside serology, antigen detection, and, where appropriate, colonoscopy with histopathology. Misdiagnosis as IBD with inadvertent immunosuppression can result in fulminant colitis and death. Treatment requires both a tissue amoebicide (metronidazole) and a luminal amoebicide (paromomycin) to eliminate intestinal carriage and prevent recurrence. This review comprehensively addresses the microbiology, epidemiology, pathogenesis, Unani classical perspective, clinical features, diagnosis, and treatment of amoebiasis to guide contemporary clinical practice.

Keywords: *Amoebiasis, Entamoeba Histolytica, Amoebic Colitis, Amoebic Liver Abscess (ALA), Zaheer-e-Sadique, Unani Medicine, Inflammatory Bowel Disease (IBD), Faecal-Oral Transmission.*

How to Cite: Dr. Syeda Ameena Tasleem; Dr. Syed Osman Pasha; Dr. Mohammed Maqbool Hussain; Dr. Juveriya Ifath; Dr. Fouzia Anjum (2026) Amoebiasis (Zaheer-e-Sadique): Integrating Classical Unani Descriptions with Modern Insights on Epidemiology, Pathogenesis, Diagnosis, and Management – A Comprehensive Review. *International Journal of Innovative Science and Research Technology*, 11(4), 3708-3716. https://doi.org/10.38124/ijisrt/26apr2106

I. INTRODUCTION

Amoebiasis is a parasitic infection of the large intestine triggered by Entamoeba histolytica, an anaerobic single-celled eukaryote that was first identified and described during the late nineteenth century. The illness manifests across a wide range of clinical presentations, from symptom-free intestinal carriage to severe necrotising colitis, and via blood-borne dissemination, to dangerous extraintestinal complications such as abscesses in the liver, lungs, pericardium, and brain. Roughly 90% of those infected stay asymptomatic, whereas the remaining 10% who progress to invasive illness account for a large share of global mortality due to infectious diseases. (1)

The term 'dysentery' was introduced by Hippocrates and derives from the Greek 'dysenteric', in which 'dys' denotes bad or difficult while 'entera' indicates the intestine.1 The clinical concept of dysentery has remained largely unchanged since ancient times: it involves the

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

passage of stools mixed with blood and mucus, along with straining and tenesmus. In Unani medicine, this condition is referred to as Zaheer-e-Sadique and has been elaborately described by renowned classical scholars such as Hippocrates, Ibn-e-Sina, Zakariyya Razi, Ali ibn Abbas Majusi, and Hakeem Ajmal Khan. This illustrates a profound heritage of clinical insight that foreshadows numerous contemporary observations. (2,3)

With the rise of globalisation, the clinical spectrum of amoebiasis has extended well beyond its traditional endemic zones in tropical and subtropical areas. Gastroenterologists and emergency physicians in developed nations need to remain vigilant and consider amoebiasis in the differential diagnosis, especially because it can closely resemble inflammatory bowel disease (IBD), and the inappropriate use of immunosuppressive treatment can lead to serious complications. This review brings together current evidence regarding the epidemiology, pathogenesis, historical descriptions, clinical manifestations, diagnosis, and management of amoebiasis.

II. HISTORICAL PERSPECTIVE: UNANI CLASSICAL MEDICINE

➢ *The Concept of Zaheer in Unani Medicine*

In Unani medicine, amoebic dysentery is classically referred to as Zaheer-e-Sadique. Two main types are described: Zaheer-e-Sadique (true dysentery), which corresponds closely to amoebic dysentery, and Zaheer-e-Kazib (false dysentery), which arises from intestinal obstruction. The latter resembles dysentery but typically involves milder pain and irritation. (4)

Around 400 BC, Hippocrates depicted the disease as bloody diarrhoea associated with straining and tenesmus. (1,4) In Tib-e-Akbar, Hakeem Mohammad Akbar Arzani mentioned Zaheer under the name 'Illat-ud-Dajajah' — a disorder marked by involuntary rectal movements in which only fluid, blood, and mucus are expelled despite attempts to pass faecal matter. (4)

➢ *Descriptions by Classical Unani Physicians*

In Al-Qanoon fil-Tib (The Canon of Medicine), Ibn-e-Sina (Avicenna) portrayed Zaheer as an abnormal rectal movement where the patient attempts to defecate but passes only scanty mucus accompanied by intense pain.1 He outlined several progressive stages of the illness: in the mild phase, stools are mixed with mucus; as it advances, the infection penetrates the mucosal layer and stools become admixed with that layer; with further progression, intestinal arteries are damaged, leading to bloody stools. (4)

In Kamil-al-San'a, Ali ibn Abbas Majusi described dysentery as a condition involving abnormal rectal movements that produce an urgent desire to evacuate, yet only a mucous-like fluid emerges, often mixed with saffron-coloured blood. (2)

Zakariyya Razi, in Kitab al-Hawi, characterised Zaheer as an ulceration of the rectum and regarded it as one of the most dangerous forms of ulcer.4 This account predates the modern pathological concept of flask-shaped ulcers typical of E. histolytica invasion by more than a thousand years.

Hakeem Ajmal Khan, in Kitab-al-Haziq, detailed the symptoms of Zaheer-e-Sadique as low-grade fever, frequent stools, abdominal pain, mucus in stools, shreds in stools, tenesmus, nausea, vomiting, and blood in stools, a picture remarkably similar to the contemporary clinical presentation of amoebic colitis. (4)

Abul Hasan bin Sahal Rabban-e-Tabri, in Firdaus al-Hikmat, highlighted complications of Zaheer: the appearance of watery stools resembling wax was considered a grave sign suggesting spread of inflammation throughout the intestines; if such watery stools were followed by flesh-coloured stools, it indicated weakness of the liver, possibly reflecting early recognition of amoebic hepatic involvement. (4)

These classical Unani descriptions reveal that the clinical syndrome of amoebiasis was clearly recognised and documented in pre-modern medicine. Moreover, the emphasis placed by classical Unani physicians on contaminated food and drink as a cause closely mirrors the modern understanding of faecal-oral transmission.

III. MICROBIOLOGY AND LIFE CYCLE

➢ *The Genus Entamoeba*

Entamoeba histolytica is the sole species within the protozoal genus Entamoeba that is known to cause amoebiasis. (5) At least seven other Entamoeba species have been identified in humans: E. gingivalis, which inhabits the buccal cavity, and in the intestine — E. coli, E. hartmanni, E. polecki, E. dispar, E. moshkovskii, and E. bangladeshi. The latter three are morphologically identical to E. histolytica under light microscopy, making molecular diagnostic methods essential for precise species identification. (6)

E. dispar, initially described by Brumpt, represents the most common non-pathogenic species. The advent of molecular diagnostics has allowed accurate distinction between E. histolytica and non-pathogenic amoebae, thereby significantly refining epidemiological assessments of the actual burden of invasive disease. (7,8)

➢ *Life Cycle*

Entamoeba histolytica is a single-celled anaerobic eukaryote that possesses a two-stage life cycle consisting of the infective cyst and the invasive trophozoite. The infection is acquired through ingestion of cysts present in food or water contaminated with human faeces. (9) The life cycle includes three distinct forms:

- *Trophozoite:*

This is the invasive stage, measuring 10–60 micrometres in size. Its cytoplasm is divided into a clear ectoplasm and a granular endoplasm containing red blood

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

cells and food vacuoles. It exhibits active motility through pseudopodia (finger-like projections). Trophozoites are excreted only in the faeces of patients with active dysentery. (10)

- *Precystic form:*

An intermediate stage between the trophozoite and cyst, oval-shaped with blunt pseudopodia and measuring 10–20 microns. (10)

- *Cyst:*

The infective stage present in the stool of infected persons. It is spherical, 10–20 microns in diameter, enclosed by a smooth chitinous wall that provides resistance to gastric acid. It starts as uninucleate and undergoes binary fission to become quadrinucleate. (10)

Upon reaching the terminal ileum, the cysts undergo excystation and release four trophozoites. (9) The galactose/N-acetylgalactosamine (Gal/GalNAc) lectin enables the trophozoites to attach to the colonic epithelium. Tissue damage occurs through amoebapores that lyse host cells, along with cysteine proteases and an inflammatory cascade involving IL-1 and IL-8, resulting in invasion by trophozoites and the characteristic flask-shaped ulcers in the submucosa. (9)

Trophozoites reproduce by binary fission. Those remaining in the intestinal lumen re-encyst before being excreted and can persist in the environment for up to 90 days. (11) Entry into the portal circulation allows haematogenous spread to extraintestinal sites, most frequently the liver. Zoonotic transmission from animals to humans is considered uncommon. (12)

IV. EPIDEMIOLOGY

➢ *Global Burden*

Amoebiasis represents a major global health challenge, causing approximately 55,000 deaths annually and resulting in the loss of 2.2 million disability-adjusted life-years (DALYs). (13,14) Worldwide, an estimated 50 million individuals are infected with E. histolytica each year, although about 90% of them remain asymptomatic. (15) The infection primarily impacts low-income countries (LICs) where water supplies are contaminated and sanitation facilities are inadequate.

Tropical and subtropical areas of Central and South America, Asia, and Africa bear the highest burden. Amoebiasis occurs most frequently in countries such as Bangladesh, India, Brazil, Colombia, Mexico, and China, where it accounts for 20–40% of reported infectious diarrhoea cases in certain regions. (16) Over the past three decades, global morbidity as measured by DALYs has declined owing to better sanitation and availability of effective treatment; nevertheless, an upward trend has been observed in high-income countries, largely driven by migration and international travel. (17)

➢ *Epidemiology in High-Income Countries*

In the United Kingdom, E. histolytica infection is notifiable by laboratories under the Health Protection Regulations (2010), and approximately 100 infections are reported every year, likely underrepresenting the true prevalence. (18) GeoSentinel Surveillance Network data indicate E. histolytica is the third most commonly isolated infection globally among returning tourists with gastrointestinal disorders, responsible for 0.3–10% of travel-related diarrhoea. (19)

Travel to an endemic area most often precedes infection. In the USA, 50% of infections occur in migrants from Mexico, Central and South America, India, and Pakistan. (20) In Spain, 46% of infections occurred in migrants and 56% had travelled to an endemic area, with symptoms commonly developing after, not during, the travel period, sometimes months or years after exposure. (21)

➢ *Risk Factors*

Acquisition of infection in non-endemic countries is well documented. In one Australian study, approximately 8% of infected individuals reported no history of travel to endemic areas. (22) Other documented modes of transmission include post-surgical acquisition and use of contaminated colonic irrigation equipment. (23,24) Sexual transmission is also recognised; epidemiological studies conducted in high-income countries have shown a higher prevalence of Entamoeba carriage among men who have sex with men (MSM). (25)

E. histolytica infection affects children and adults equally and can spread within households. Studies among adults demonstrate a clear male predominance, particularly in cases of amoebic liver abscess (ALA), where men account for 72–76% of adult cases. (16) The use of immunosuppressive medications, especially corticosteroids, is associated with increased severity of infection. This is particularly relevant clinically due to the significant overlap in symptoms between amoebiasis and inflammatory bowel disease (IBD). (26)

V. PATHOGENESIS

➢ *Modern Pathogenesis*

Following ingestion of viable cysts through the faecal-oral route, the cysts pass through the stomach unharmed due to the protective chitinous wall that resists gastric acid. Upon reaching the small intestine, the enzyme trypsin dissolves this wall, allowing metacystic trophozoites to emerge. These trophozoites then migrate to the colon, where they colonise the mucosal surface and crypts of the large intestine. (9)

The Gal/GalNAc lectin enables trophozoite adherence to the colonic epithelium. Cysteine proteases and amoebapores cause rupture of host cells, while an IL-1 and IL-8-mediated inflammatory response promotes further invasion by the trophozoites. (9) The resulting lesions most commonly develop in the caecum but can extend throughout the colon down to the anal canal, forming the characteristic

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

flask-shaped ulcers. Some trophozoites may penetrate the bloodstream via the portal circulation, leading to extraintestinal involvement in organs such as the liver, lungs, and brain.

Zakariyya Razi's description in the 10th century of dysentery as an ulcer of the rectum remarkably anticipated the modern pathological concept of flask-shaped ulceration in the colon.

➢ *Humoral Pathology: Unani Perspective*

In Unani medicine, Zaheer-e-Sadique is categorised as Marz-e-Murakkab Maddi (compound material disease). According to Unani principles, the pathology of any disease is determined by three key factors: Mizaj (temperament), Tarkeeb (composition), and Ittesal (continuity). Any disturbance in these elements results in Sue-e-Mizaj, Sue-e-Tarkeeb, and Taffaruq-e-Ittesal, respectively. (4)

In amoebiasis, the cysteine proteinase secreted by E. histolytica disrupts the protective role of the intestinal mucosal barrier; in Unani terminology, this corresponds to Sue-e-Mizaj (altered temperament). Invasion of the mucosal layer by E. histolytica leads to discontinuity and breach of the mucosal surface, which is described in Unani terms as Taffaruq-e-Ittesal. (4) Although this conceptual framework predates the germ theory of disease, it describes the core pathological processes of intestinal amoebiasis with striking precision.

VI. CLINICAL FEATURES

➢ *Intestinal Amoebiasis*

Approximately 90% of individuals infected with E. histolytica remain asymptomatic, while 10% of those carrying pathogenic cysts progress to invasive amoebiasis within one year. (27) Patients with amoebic colitis usually present with a gradual onset of worsening cramping abdominal pain, weight loss, and watery, mucoid or bloody diarrhoea spanning several weeks. This presentation often makes differentiation from inflammatory bowel disease (IBD) especially difficult. (15)

On physical examination, findings may be minimal or include abdominal tenderness (67%), distension (33%), and, rarely, a palpable abdominal mass known as an amoeboma. This fibroinflammatory mass most commonly occurs in the caecum or ascending colon and can mimic a Crohn's phlegmon. (20)

Progression to necrotising or fulminant colitis is seen in about 0.5% of cases. (28) Affected patients typically develop fever, bloody diarrhoea, vomiting, and abdominal pain with rebound tenderness. Around three-quarters of those with fulminant amoebic colitis develop toxic megacolon or perforation, carrying a mortality rate of 40%. (29)

Table 1 Symptoms of Amoebic Colitis

Symptom	Prevalence
Diarrhoea (watery or bloody)	Very common >90%
Abdominal pain	Common >60%
Fever	Less common <40%
Weight loss	Uncommon <25%
Rectal bleeding without diarrhoea	Rare
Abdominal mass (amoeboma)	Rare
Fulminant / necrotising colitis	~0.5% of cases

➢ *Amoebic Liver Abscess (ALA)*

Amoebic liver abscess (ALA) is the most frequent extraintestinal manifestation of amoebiasis, occurring in approximately 1% of infected individuals. (15) Once E. histolytica reaches the liver, it triggers an inflammatory response that leads to hepatocyte necrosis. This characteristically results in a single, well-defined abscess surrounded by a rim of connective tissue and filled with brown "anchovy sauce" pus consisting of necrotic hepatocytes, a few trophozoites, and inflammatory cells. (30)

Symptoms generally develop 8–20 weeks (median 12 weeks) after travel to an endemic area, although cases of ALA have been documented more than 20 years after the last exposure to an endemic region. (30) Typical symptoms include fever and dull right upper quadrant pain that may radiate to the epigastrium, chest, or shoulder. Concomitant diarrhoea is reported in only 38% of patients. (30) On physical examination, hepatic tenderness and hepatomegaly are found in about 50% of cases.

➢ *Rare Extraintestinal Manifestations*

Table 2 Rare Extraintestinal Manifestations of Amoebiasis

Site	Risk	Key Clinical Features	Prognosis
Liver (ALA)	Most common (1% of infected); male predominance 72–76%	Fever, right upper quadrant pain, hepatomegaly; 'anchovy sauce' pus	Mortality 1–3% (uncomplicated); worse with encephalopathy, hypoalbuminaemia
Pulmonary	2–3% of extraintestinal; more common with ALA	Cough, haemoptysis, dyspnoea, pleuritic pain	Mortality >16%; most respond to medical therapy

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

Pericardial	Very rare; left lobe ALA rupture	Pericardial effusion, tamponade, constrictive pericarditis	Left ALA drainage + pericardiocentesis recommended
Neurological	Very rare; always concomitant ALA	Brain abscess, meningoencephalitis, focal neurology	Fatal if undiagnosed early; up to 8 weeks treatment
Cutaneous	Very rare; perianal risk from poor hygiene	Painful perianal ulceration, erythematous ulcers	Trophozoites isolable from ulcer exudate
Genital	Very rare; risk factor anal sex	Penile, vaginal, cervical ulcers; foul smelling discharge	Treat as other forms; trace and treat partner

VII. DIAGNOSIS

➢ *Laboratory Diagnosis*

Differentiating E. histolytica from the non-pathogenic E. dispar is essential for accurate diagnosis and treatment. The UK Health Security Agency recommends PCR as the method of choice for diagnosis of E. histolytica in both symptomatic and asymptomatic patients. (18)

Light microscopy of a fresh stool is widely available, inexpensive, and quick, but cannot morphologically distinguish E. histolytica cysts from those of non-pathogenic species such as E. dispar or E. moshkovskii. (31) Trophozoites will usually only be seen in a loose 'hot stool' examined within 20 minutes of production.

Antigen detection using ELISA tests for E. histolytica-specific antigens from stool or liver abscess aspirate, with reported sensitivities and specificities of over 80%. (32) PCR is the gold standard for intestinal amoebiasis, achieving 92–100% sensitivity and 89–100% specificity, and can accurately differentiate E. histolytica from non-pathogenic species.

Serology detects IgG antibody to E. histolytica-specific antigen with sensitivity of 65–92% and specificity over 90%, useful for both intestinal and extraintestinal disease. However, sensitivity is only approximately 60% for invasive intestinal amoebiasis, and serology can remain positive for years after infection, limiting its utility in endemic regions. (31)

Table 3 Laboratory Diagnostic Tests for Entamoeba Histolytica

Diagnostic Method	Specimen	Sensitivity	Specificity	Advantages	Limitations
Light Microscopy	Stool	<60%	~95%	Cheap; widely available; screens other parasites	Cannot differentiate E. histolytica from non-pathogenic species
Antigen ELISA	Stool / Liver aspirate	Up to 88%	>80%	Differentiates Entamoeba species	Needs fresh stool; reduced sensitivity after therapy
PCR (Gold Standard)	Stool / Liver aspirate	92–100%	89–100%	Definitive species differentiation	Expensive; requires laboratory expertise
Serology (IgG)	Serum	65–92%	>90%	Useful for intestinal and extraintestinal disease	Low sensitivity (~60%) for intestinal disease; false positives in endemic regions
Point-of-care Antigen	Stool	28–100%	80–100%	Cheap; rapid; no specialist lab needed	Cannot differentiate Entamoeba species reliably
Histopathology	Colonic biopsy / tissue	Variable	Operator-dependent	Routine practice; no pre-test suspicion required	Trophozoites may be missed; mimics Crohn's disease

➢ *Colonoscopy and Histopathology*

Although colonoscopy is not routinely required for the diagnosis of amoebic colitis, patients presenting with gastrointestinal symptoms — especially severe or persistent cases — are frequently assessed by endoscopy. Amoebic colitis typically produces patchy inflammation, with or without pale exudate and ulceration. The lesions predominantly affect the caecum and ascending colon, followed by the rectum. (33)

Ulcers vary in size from small erosions to larger lesions exceeding 2 cm and are usually multiple and discrete. A mucosal “bump” sign — a small inflammatory nodule less than 1 cm infiltrated by trophozoites, has been suggested as a pathognomonic endoscopic finding. (33)

Histopathology often reveals flask-shaped ulcers at low magnification. At higher magnification, E. histolytica trophozoites can be identified in the ulcer debris, frequently showing erythrophagocytosis. Inexperienced observers may confuse these trophozoites with macrophages or detached enterocytes. The primary diagnostic challenge in biopsies is distinguishing amoebic colitis from inflammatory bowel disease (IBD), particularly Crohn’s disease, since the ulceration can be patchy and features of chronicity such as crypt distortion and fibrosis may coexist.

➢ *Imaging*

CT, MRI, and ultrasonography are effective modalities for diagnosing ALA. ALA typically appears on ultrasound as a cystic intrahepatic hypoechoic lesion with thick walls, most commonly in the right hepatic lobe near the capsule. By CT, there is a non-enhancing centre with an

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

inflammatory ring following contrast administration. (34) The right hemidiaphragm may be elevated. ALA is most commonly unilocular, as opposed to pyogenic abscess which is most commonly multilocular.

Plain abdominal radiography may show non-specific features of colitis. CT may help identify deep ulceration, patchy distribution of colitis, and omental wrapping, but CT alone should not be used to diagnose amoebic colitis since features may be indistinguishable from IBD.

VIII. DIFFERENTIAL DIAGNOSIS

The most important clinical differential diagnosis for amoebic colitis is inflammatory bowel disease (IBD), especially Crohn's disease. Both disorders can present with similar features, including cramping abdominal pain, bloody diarrhoea, weight loss, and endoscopic findings such as patchy ulceration and mucosal inflammation. Misdiagnosis is particularly common in non-endemic areas, where clinicians may not suspect amoebiasis.

A systematic review of 24 cases of amoebic colitis found that the use of corticosteroids led to rapid disease progression, with higher rates of colonic perforation, amoeboma formation, rectovaginal fistula, and extraintestinal spread. More than half of the patients required emergency surgery, and a quarter died. (35)

Other important differential diagnoses include bacillary dysentery (shigellosis), Salmonella and Campylobacter infections, diarrhoeagenic E. coli, intestinal tuberculosis, Clostridium difficile colitis, and colorectal carcinoma (which may be mimicked by an amoeboma). (36)

In classical Unani medicine, conditions producing similar symptoms were grouped under various forms of Ishal (diarrhoea) and Qulanj (colic). These were differentiated according to accompanying symptoms, the character of the stool, seasonal factors, and dietary history. (4)

IX. TREATMENT

➢ *Principles of Treatment*

All patients infected with E. histolytica should receive treatment, regardless of whether they are symptomatic or not. The primary aim of therapy is to eradicate both the invading trophozoites and the intestinal carriage of cysts. Effective treatment necessitates the use of two different agents: a systemically absorbed tissue amoebicide to target invasive forms and a luminal amoebicide to clear cysts from the intestinal lumen. This combination approach has been demonstrated to prevent recurrence of the disease. (37)

➢ *Tissue Amoebicides*

Tissue amoebicidal agents belong to the nitroimidazole class of antibiotics, mainly metronidazole and tinidazole. A Cochrane review demonstrated that tinidazole is more effective than metronidazole, with a failure rate of 1% for tinidazole compared to 5% for metronidazole, and it is associated with fewer adverse effects. (37) However, tinidazole has not been available in the United Kingdom since 2021, making metronidazole the currently recommended first-line tissue amoebicide. Patients should avoid alcohol consumption during treatment and for at least four days after completing nitroimidazole therapy because of the risk of a disulfiram-like reaction. (38)

➢ *Luminal Amoebicides*

Luminal amoebicides include paromomycin, diloxanide furoate (no longer available in the UK), iodoquinol, and nitazoxanide. Paromomycin, an aminoglycoside that acts by disrupting RNA translation, is the preferred luminal agent in the UK and achieves cure rates of up to 85% in asymptomatic carriers. (38) Although more than 90% of patients with amoebiasis respond to nitroimidazoles, parasite persistence is observed in 40–60% of cases if a luminal amoebicide is not used subsequently.

Table 4 Treatment Regimens for Entamoeba histolytica Infection

Condition	First-line Treatment	Luminal Amoebicide	Notes
Amoebic Colitis	Metronidazole 800 mg TDS PO for 5 days OR Tinidazole 2 g OD for 3 days (unavailable in UK)	Paromomycin 25–35 mg/kg/day in 3 divided doses for 7 days	Avoid alcohol during and 4 days after nitroimidazole therapy
Amoebic Liver Abscess (ALA)	Metronidazole 800 mg TDS PO for 5–10 days OR Tinidazole 2 g OD for 5 days (unavailable in UK)	Paromomycin 25–35 mg/kg/day for 7 days	Consider drainage if abscess >10 cm, left lobe, no clinical response after 3–4 days, or imminent rupture
Asymptomatic Carriage	Luminal amoebicide only	Paromomycin 25–35 mg/kg/day for 7 days	No tissue amoebicide required for asymptomatic carriers
Extraintestinal / Extrahepatic	Consult infectious diseases team	As per ALA protocol	Longer treatment durations may be needed (up to 8 weeks for cerebral amoebiasis)

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

➢ *Management of Amoebic Liver Abscess*

ALA does not routinely require percutaneous drainage. Drainage may be considered if: clinical response is not seen after three or four days of medical treatment; the abscess is greater than 10 cm (increased risk of rupture); or the abscess is in the left lobe, where rupture may involve the pericardium. (39) The abscess may take many months to resolve fully on imaging, but this does not require extended drug therapy.

➢ *Unani Treatment Principles*

Classical Unani management of Zaheer-e-Sadique was based on well-established principles of Usool-e-Ilaj. It included dietary regulation (Tadbeer-e-Ghiza), administration of laxative drugs, Habis-ud-Dam (haemostatic agents), Muqawi-e-Amah (intestinal tonics), Muhalil-e-Auram (anti-inflammatory drugs), and Qabizat (astringents). (4)

Among the single drugs commonly used were Marorphali (Helicteres isora), which contains 4-quinolone alkaloids and malatyamine responsible for its antidiarrhoeal effects; Beekh-e-Khatmi (Althaea officinalis), valued for its soothing demulcent action on intestinal irritation; Teewaj (Holarrhena antidysenterica), whose bark and seeds contain conessine with notable amoebicidal activity; and Beal (Aegle marmelos), appreciated for its demulcent properties. (4)

Compound Unani formulations frequently employed included Safoof-e-Muqliyasa, Safoof-e-Teen, Habb-e-Pechish, Sharbat-e-Belgiri, and Tiryaq-e-Pechish. These preparations demonstrate a refined pharmacopoeial approach that specifically addresses the core pathological features of intestinal inflammation, mucosal damage, and haemorrhage.

X. PREVENTION AND PUBLIC HEALTH CONSIDERATIONS

➢ *Prevention Strategies*

Prevention is of utmost importance and focuses on personal hygiene practices, avoidance of contaminated food and water, and public education about sexual transmission. For individuals travelling to endemic regions, key recommendations include consuming only commercially bottled or properly boiled water; avoiding ice unless it is prepared from purified water; refraining from eating raw vegetables; consuming only thoroughly cooked foods; and using bottled or boiled water for handwashing, cooking, and brushing teeth. (40)

Clinical management of confirmed cases requires strict universal enteric precautions, which may be stopped 48 hours after diarrhoea has completely resolved. Both symptomatic and asymptomatic household members, co-travellers, and sexual contacts should be screened with stool PCR testing and treated if found positive. (18)

➢ *IBD Misdiagnosis Prevention*

It is essential to exclude E. histolytica infection in every patient with a new or suspected diagnosis of inflammatory bowel disease (IBD) before initiating immunosuppressive therapy. The most dependable diagnostic method to rule out active infection is stool PCR for E. histolytica, which can be performed as a single test or as part of a broader pan-enteric panel. (26)

When immunosuppressive treatment for IBD needs to be started urgently, stool amoebic PCR testing should be prioritised and the treatment plan adjusted according to the results. If clinical suspicion is high, empirical therapy for amoebiasis may be commenced once stool samples have been collected.

➢ *Future Directions*

There is currently no licensed vaccine available for amoebiasis. However, the Gal/GalNAc lectin has been extensively studied as a promising antigenic target for vaccine development. (41)

Several novel drug targets linked to various amoebic cellular processes have also been proposed. Emerging research indicates that changes in the gut microbiome may prompt E. histolytica to shift from a harmless commensal state to an invasive pathogen. This observation has led to speculation about a possible role for probiotics in prevention or as adjunctive therapy, although more studies are needed to establish their efficacy. (41)

XI. CONCLUSION

Amoebiasis continues to pose a substantial global health burden, with clinical relevance extending well beyond traditional endemic zones. The condition was clearly recognised and documented by classical physicians, including Hippocrates, Ibn-e-Sina (Avicenna), Zakariyya Razi, Ali ibn Abbas Majusi, and Hakeem Ajmal Khan. Their detailed accounts of its clinical features, pathological changes, and treatment approaches show striking consistency with current medical understanding. In classical Unani medicine, the entity known as Zaheer-e-Sadique — marked by tenesmus, bloody mucoid stools, and intestinal ulceration linked to contaminated food and drink — closely anticipated the key characteristics of amoebic colitis, now explained by the invasion of colonic mucosa by Entamoeba histolytica.

The foremost modern clinical challenge associated with amoebiasis lies in its ability to closely resemble inflammatory bowel disease (IBD). Incorrect diagnosis followed by inadvertent immunosuppressive therapy can rapidly lead to fulminant and potentially fatal colitis. Gastroenterologists, infectious disease specialists, and surgeons must remain vigilant and consider amoebiasis in the differential diagnosis of any patient presenting with colitis, particularly those with a history of travel to or migration from endemic areas, men who have sex with men (MSM), or individuals on immunosuppressive medications.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26apr2106

Accurate diagnosis is best accomplished through stool PCR specifically targeting E. histolytica, which should be explicitly requested along with a complete travel and exposure history. Routine stool PCR screening is strongly advised for all patients with newly suspected IBD before starting immunosuppressive treatment. Effective management requires combination therapy with both tissue and luminal amoebicides to ensure complete eradication and prevent relapse. The integration of ancient clinical insights with contemporary molecular diagnostics provides the most effective approach to the modern management of this persistent parasitic disease.

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