

EPIDEMIOLOGICAL AND CLINICAL PROFILE OF AMOEBIASIS IN HOSPITALIZED CHILDREN IN MOSUL CITY

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ABSTRACT

Background: Amoebiasis, caused by the protozoan parasite *Entamoeba histolytica*, presents a major public health challenge in developing countries characterized by substandard water sanitation infrastructure. **Objectives:** This prospective case series aimed to investigate the clinical, macroscopic, biological, and socio-demographic parameters of pediatric amoebiasis cases admitted to Al-Khansaa Maternity and Pediatric Teaching Hospital in Mosul. **Methods:** From March 1 to June 30, 2014, 96 children under 10 years of age exhibiting diarrheal symptoms and microscopically confirmed *E. histolytica* trophozoites in fresh stool samples were monitored sequentially. **Results:** The study cohort comprised 59 males (61.14%) and 37 females (38.54%), establishing a male-to-female ratio of 1.6:1. Infants aged 1–12 months were highly vulnerable, making up 46.87% (n=45) of cases, with a peak frequency among those aged 6–12 months (32.29%, n=31). Urban residents constituted 63.54% (n=61). Paradoxically, 86.45% (n=83) of infected families utilized municipal tap water. Clinical evaluation showed that 67.7% (n=65) presented with fever ($\geq 38^{\circ}\text{C}$), 75% (n=72) with abdominal pain, and 55.2% (n=53) with macroscopically distinct blood and mucus (clinical dysentery). Mild-to-moderate dehydration was identified in 61.4% (n=59), while 3.1% (n=3) suffered severe dehydration. Concomitant stool cultures for dysenteric samples revealed secondary bacterial infestations with *Escherichia coli* in 22.6% (n=12) and *Salmonella* in 3.7% (n=2); no *Shigella* growth was observed. **Conclusions:** Infant cohorts aged 6–12 months and formula/mixed-fed children exhibit heightened vulnerability. Traditional municipal chlorination proves insufficient against amebic cysts, necessitating mandatory water boiling and targeted dual tissue/luminal therapeutic regimens.

KEYWORDS: Amoebiasis, *Entamoeba histolytica*, Pediatric Gastroenteritis, Stool Culture, Mosul Sanitation.

1. INTRODUCTION

Amoebiasis is clinically defined as an intestinal or extraintestinal infection caused by the protozoan parasite *Entamoeba histolytica*, occurring with or without overt clinical symptoms.^[1] The organism is found worldwide, but exhibits its highest endemic transmission rates in developing countries where the infrastructural barriers between human excreta and municipal water or food systems are severely compromised.^[1,5] Globally, diarrheal diseases represent a premier driver of childhood mortality and structural morbidity, causing an estimated 2.5 million deaths annually alongside long-term

deleterious effects on physical linear growth and cognitive performance.^[2, 19] *E. histolytica* represents a prominent etiological agent within this diarrheal burden.

E. histolytica exists in two distinct morphological stages: the motile, invasive trophozoite and the dormant, infectious cyst.^[1, 2] Trophozoites typically reside in the lumen or mucosal layers of the large intestine, measuring 7–30 μm in diameter, and are characterized by a single spherical nucleus with fine peripheral chromatin and a central nucleolus.^[1,3] In clinical dysentery, these trophozoites exhibit hematophagous characteristics,

ingesting host erythrocytes, and can expand up to 60 μm in diameter.^[2] Trophozoite pseudopodia mediate both directional motility and active phagocytosis, acting as critical determinants of pathogenicity.^[2, 6] Conversely, the non-motile, spherical cyst stage measures 5–20 μm and contains up to four nuclei when mature, allowing it to survive environmental stressors and gastric acidity.^[1, 3] Colonization is initiated solely via the ingestion of these mature cysts.^[1]

Five other non-pathogenic species of the genus *Entamoeba*—namely *E. coli*, *E. hartmanni*, *E. gingivalis*, *E. moshkovskii*, and *E. polecki*—frequently colonize the human gastrointestinal lumen without inducing tissue breakdown, presenting substantial confounding challenges during diagnostic microscopic evaluations.^[2, 6]

2. Pathogenesis and Clinical Characters

Following ingestion, cysts pass uninjured through the stomach and undergo excystation within the alkaline milieu of the small intestine, releasing eight distinct motile trophozoites that migrate to the cecum and colon.^[1, 6] Trophozoites adhere to colonic mucin glycoproteins via a specific galactose and N-acetyl-D-galactosamine (Gal/GalNAc) lectin.^[10] This binding allows the parasite to resist host complement-mediated lysis by inhibiting membrane attack complex formation.^[1, 10]

Host susceptibility is modified by nutritional, physiological, and immunological factors. Corticosteroid treatments, active systemic malignancies, severe malnutrition, and pregnancy significantly heighten the risk of fulminant, life-threatening invasive infections.^[2, 10] Host clearing mechanisms are primary cell-mediated, utilizing interferon-gamma and tumor necrosis factor to activate macrophages and polymorphonuclear neutrophils for effective trophozoite elimination.^[10] Pathological hallmark lesions comprise shallow, flask-shaped mucosal ulcers covered with yellowish-gray exudates that contain dense aggregates of hematophagous trophozoites.^[11, 12] If these destructive processes traverse the muscularis mucosae and serosa, colonic perforation and peritonitis occur.^[3, 11]

3. PATIENTS AND METHODS

3.1 Study Design and Setting

A prospective case series design was implemented at Al-Khansaa Maternity and Pediatric Teaching Hospital in Mosul city, over a fixed 4-month duration from March 1 to June 30, 2014. The institutional setting is a premier pediatric training facility operating continuously in the region since 1986. Ethical approval was maintained via structured verbal consent protocols obtained directly from the mothers, fathers, or legal guardians of all pediatric candidates prior to enrollment.^[24]

3.2 Patient Selection and Case Definition

The potential sample pool comprised all pediatric patients of both sexes under 10 years of age admitted to the acute care wards or outpatient clinics for diarrheal complaints during 5 working days per week. The specific case definition required the concurrent presence of clinical diarrheal symptoms and positive microscopic identification of active, motile *E. histolytica* trophozoites during general stool examinations (GSE).^[1, 2] Children presenting only with non-motile cysts or those confirmed to have alternative parasitic infestations (e.g., *Giardia lamblia*) were excluded.

3.3 Data Collection and Laboratory Analysis

Socio-demographic and clinical parameters were collected through structured, direct face-to-face interviews using a standardized questionnaire tool. Captured parameters included age, biological sex, geographical residence, feeding history (categorized into exclusive breastfeeding, exclusive bottle formula feeding, mixed feeding, or ordinary family food), household water supply source (tap water vs. alternative raw tankers, rivers, or wells), daily bowel motion frequencies, and associated systemic features like vomiting or abdominal pain.

Clinical examinations assessed the degree of dehydration using the Clinical Dehydration Scale (categorized as none, some/moderate, or severe).^[24] Core axillary temperature was measured using electronic thermometry, with an added correction factor of +0.5°C; values $\geq 38.0^\circ\text{C}$ were defined as febrile.^[25] Detailed abdominal palpation focused on identifying hepatomegaly.

Stool samples were examined macroscopically and microscopically within 30 minutes of defecation to ensure trophozoite viability. Direct smears were prepared with physiological saline to identify motile trophozoites, red blood cells (RBCs), and pus cells. For all patients presenting with positive trophozoite findings and concomitant bloody diarrhea ($n=53$), fresh fecal specimens were cultured on MacConkey agar, Salmonella-Shigella (SS) agar, and tetrathionate broth enrichment media. Cultured media were incubated aerobically for 18–24 hours at 37°C . Subcultures from tetrathionate broth onto fresh SS agar were sustained for an additional 24 hours to optimize the recovery of fastidious bacterial pathogens like *Salmonella* and *Shigella* species.

4. RESULTS

4.1 Socio-Demographic Patterns

A total of 96 pediatric patients met the diagnostic inclusion criteria and were monitored. Biological sex distribution showed 59 males (61.14%) and 37 females (38.54%), resulting in a male-to-female ratio of 1.6:1. Age cohort segmentation demonstrated that the vast majority of cases occurred in early childhood, with 95.83% ($n=92$) of patients under 5 years of age. Infants aged 1–12 months represented the single largest

vulnerable class, making up 46.87% (n=45) of the study sample. Within this infant group, peak prevalence occurred in the 6–12 month age range, accounting for

32.29% (n=31) of the total cohort. No cases were observed in neonates under 1 month of age.

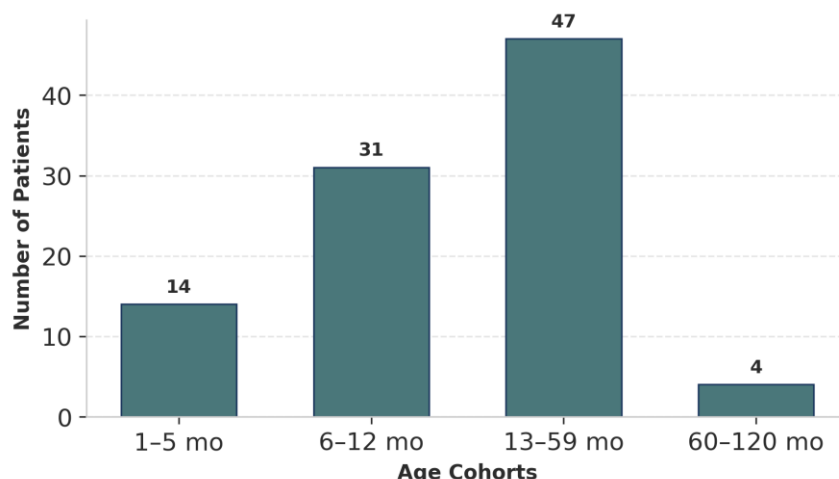


Figure 4.1: Age distribution of the pediatric study sample (N=96), highlighting high vulnerability in infants.

Socioeconomic evaluation showed that 53.12% (n=51) of families lived in moderate conditions, 40.63% (n=39) in low conditions, and 6.25% (n=6) in high socioeconomic conditions. Geographical analysis showed a higher proportion of urban residents (63.54%, n=61) compared

to rural residents (36.46%, n=35). Regarding domestic water infrastructure, 86.45% (n=83) of families used chlorinated municipal tap water supplies, whereas only 13.54% (n=13) relied on unpurified sources like tankers, rivers, or private wells.

Table 4.1: Distribution of the study sample according to household socioeconomic state.

Socioeconomic Stratification	Case Frequency (n)	Percentage (%)
Low Socioeconomic State	39	40.63%
Moderate Socioeconomic State	51	53.12%
High Socioeconomic State	6	6.25%

4.2 Nutritional Profile

Analysis of feeding histories showed that the lowest incidence of amoebiasis occurred among exclusively breastfed infants, who accounted for only 8.33% (n=8) of cases. In contrast, children on mixed feeding regimens

(combination of breast milk and formula bottles) represented the largest proportion at 42.71% (n=41). Solid family foods accounted for 31.25% (n=30) of cases, while exclusive bottle feeding accounted for 17.71% (n=17).

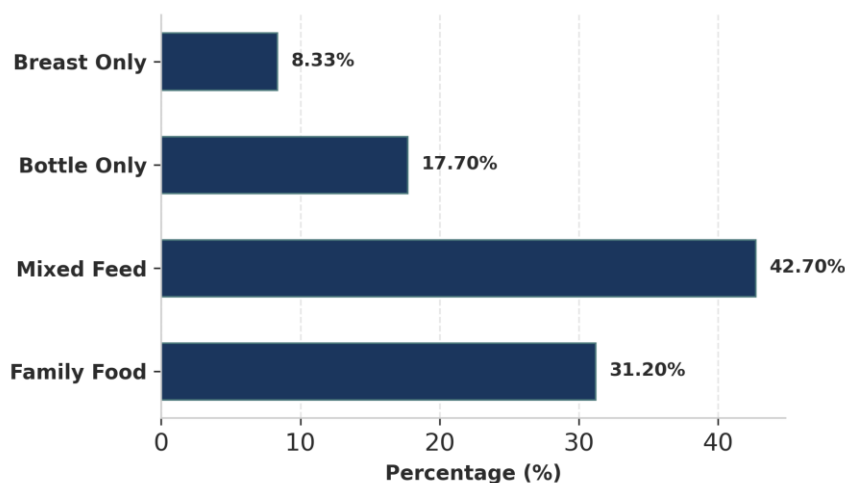


Figure 4.2: Horizontal distribution of feeding modalities, showing lower infection rates in exclusively breastfed children.

4.3 Clinical and Macroscopic Presentations

Regarding bowel motion frequencies, 67.71% (n=65) of the children presented with fewer than 10 watery or loose motions per day, while 32.29% (n=31) had severe diarrhea exceeding 10 motions daily. Abdominal pain was a common symptom, reported in 75.00% (n=72) of patients, while vomiting occurred in 20.83% (n=20) of

cases. Systemic evaluation identified pyrexia (temperatures $\geq 38^{\circ}\text{C}$) in 67.71% (n=65) of the children. Dehydration mapping showed that 61.46% (n=59) had mild-to-moderate clinical dehydration, while 3.12% (n=3) presented with severe dehydration. No clinical cases of hepatomegaly or amebic liver abscesses were detected.

Table 4.2: Comprehensive clinical sign and symptom frequencies among the study sample.

Clinical Sign / Symptom Category	Frequency (n=96)	Percentage (%)
Bowel Motion Frequency < 10 times/day	65	67.71%
Bowel Motion Frequency ≥ 10 times/day	31	32.29%
Pyrexia (Axillary Temp $\geq 38.0^{\circ}\text{C}$)	65	67.71%
Abdominal Pain / Colic	72	75.00%
Concomitant Vomiting	20	20.83%
Mild-to-Moderate Dehydration	59	61.46%

4.4 Microbiological Co-Infections

Stool culture evaluations performed on the 53 patients with amebic dysentery showed that 73.58% (n=39) carried normal gastrointestinal flora, with no additional aerobic bacterial pathogens isolated. However, secondary

bacterial co-infections were confirmed in 26.42% of these dysenteric cases. Pathogenic *Escherichia coli* was isolated in 22.64% (n=12) of specimens, and *Salmonella* species were identified in 3.77% (n=2). No *Shigella* growth was observed in any culture.

Table 4.3: Distribution of secondary aerobic bacterial isolations from dysenteric stool specimens.

Microbiological Culture Isolation	Frequency (n=53)	Percentage (%)
Normal Colonic Flora Only	39	73.58%
Pathogenic <i>Escherichia coli</i> Isolation	12	22.64%
<i>Salmonella</i> Species Isolation	2	3.77%
<i>Shigella</i> Species Isolation	0	0.00%

5. DISCUSSION

This prospective case series provides an analysis of the demographic and clinical parameters of pediatric amoebiasis within the Mosul region. The observed male-to-female ratio of 1.6:1 aligns with regional epidemiological studies conducted in northern Iraq by Salim (2004)^[26] and Alla (2002)^[27], which reported a similar male predominance in symptomatic presentations. This gender distribution may be linked to distinct behavioral exposure patterns or variations in healthcare-seeking practices for male children in certain communities.^[26]

The complete absence of infants under 1 month of age is consistent with the established incubation period for *E. histolytica*, which typically ranges from 2 to 4 weeks before clinical manifestations appear.^[2, 5] The high prevalence observed in infants aged 6–12 months (32.29%) marks a critical transitional period characterized by the decline of maternally acquired passive immunity, the introduction of potentially contaminated weaning foods, and increased exploratory hand-to-mouth behaviors.^[2, 11] This vulnerability is further illustrated by the feeding modality data; exclusively breastfed infants accounted for only 8.33% of infections, whereas children on mixed formula bottle regimens accounted for 42.71%. Breast milk provides protective secretory IgA antibodies and avoids the sanitation risks associated with contaminated water used to prepare formula bottles.^[28, 29] In bottle-fed infants,

transmission often occurs via the fecal-oral route through unwashed hands or improperly sterilized equipment.^[11, 30]

The higher percentage of urban cases (63.54%) likely reflects a selection bias inherent to the study setting, as Al-Khansaa Hospital is located within Mosul city. Rural families often face geographic and financial barriers to tertiary care, leading them to seek treatment at local primary centers.^[26] A key finding is that 86.45% of infected children came from households using chlorinated municipal tap water. This underscores a significant public health issue: standard chlorine concentrations used in domestic water treatment grids are insufficient to kill *E. histolytica* cysts.^[2, 23] Consequently, relying solely on standard municipal chlorination without boiling water fails to clear the parasite from the supply.^[23, 31]

The clinical presentation data, showing that 67.71% of patients had fewer than 10 bowel motions per day and 75.00% experienced abdominal pain, align with literature describing the typical gradual onset of colicky pain and moderate diarrheal frequency (6–8 motions daily).^[2, 23] Similar frequencies were reported by Haque (2003) in Bangladesh^[31] and Alla (2001) in Mosul^[27], where moderate daily motions constituted 71.42% of cases.

However, the observed pyrexia rate of 67.71% is notably higher than standard textbook descriptions, which report

fever in only about one-third of intestinal amoebiasis cases.^[2, 5] This discrepancy is likely because this study was restricted to hospitalized inpatients, thereby selecting for more severe clinical presentations. Additionally, secondary bacterial co-infections may have contributed to the elevated temperatures.^[31] Stool culture analysis revealed that 22.64% of the dysenteric cohort had concomitant *E. coli* infections, and 3.77% had *Salmonella*. The three cases that developed severe dehydration (3.12%) all presented with concurrent pathogenic *E. coli*. This suggests a synergistic pathogenic effect, where bacterial co-infection may increase mucosal permeability and enhance the tissue-invasive capabilities of amebic trophozoites.^[33, 34]

6. CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSIONS

1. Infants aged 6–12 months represent the primary risk group for symptomatic pediatric amoebiasis, with an observed male-to-female ratio of 1.6:1.
2. Exclusive breastfeeding provides a protective effect, as evidenced by the significantly lower infection rate (8.33%) compared to mixed or formula bottle feeding modalities.
3. Standard municipal water chlorination is insufficient for preventing transmission, as 86.45% of infected cohorts utilized tap water networks.
4. The typical clinical profile for hospitalized pediatric amoebiasis includes moderate diarrheal frequency, colicky abdominal pain, acute pyrexia, and macroscopic blood and mucus in stool specimens.
5. Bacterial co-infections, particularly with pathogenic *E. coli*, are common in amebic dysentery and are associated with an increased risk of severe dehydration.

6.2 Recommendations

1. Promote exclusive breastfeeding during the first 4–6 months of life and encourage its continuation up to 2 years, while discouraging the use of infant feeding bottles.
2. Implement strict hygiene protocols for the storage and preparation of weaning foods.
3. Advise families to boil tap water before drinking to ensure the destruction of chlorine-resistant amebic cysts.
4. Emphasize thorough handwashing with soap after defecation, before handling food, and prior to feeding children.
5. Establish regular health education programs for mothers regarding child nutritional and sanitary needs.
6. Ensure accurate identification of *E. histolytica* infections, utilizing a combination of tissue amebicides (e.g., metronidazole) followed by a luminal agent to clear residual intestinal cysts and prevent relapse.
7. Implement mandatory, periodic screening of food handlers in commercial and institutional settings to detect and treat asymptomatic cyst carriers.

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