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Case Report

Amoebic enteritis: A clinicopathological analysis of 14 cases and literature review

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Abstract

Amoebic enteritis is often misdiagnosed or overlooked due to its nonspecific clinical presentation. This study presents a comprehensive clinicopathological analysis of 14 confirmed cases of amoebic enteritis, examining their clinical manifestations, histopathological features, and responses to treatment. Periodic acid-Schiff and hexamine silver stains were employed to aid in diagnosis. A review of the relevant literature is also included to improve recognition and diagnostic accuracy of this uncommon but clinically significant condition.

Keywords: Amoebic enteritis, diagnosis

Introduction

Amoebic enteritis (AE), also known as amoebic dysentery, is an infectious disease caused by *Entamoeba histolytica*, a protozoan parasite that primarily invades the intestinal tract. The infection commonly affects the cecum, colon, and rectum [1]. However, the clinical manifestations and colonoscopic findings of AE are often nonspecific and may closely resemble those of inflammatory bowel disease and other gastrointestinal disorders [2], making accurate diagnosis challenging. This study retrospectively analyzes the clinical presentations, pathological features, and special staining results of 14 patients with confirmed AE. By integrating case data with a review of the literature, the study aims to enhance the current understanding of AE and support its differentiation from other intestinal pathologies.

Case Record

Between June 2018 and May 2024, 14 cases of AE were diagnosed at Beijing Chuiyangliu Hospital. Tissue samples were fixed in 4% neutral formaldehyde, processed routinely, embedded in paraffin, sectioned at 4 μm, and stained with hematoxylin and eosin. Special stains, including periodic acid-Schiff (PAS) and hexamine silver, were applied using commercial kits (Besso Biotechnology, Zhuhai, China). The study was approved by the ethics committee of Beijing Chuiyangliu Hospital (No. 2024-009KY). Informed consent was obtained from the patients.

The clinicopathological data of the 14 patients are summarized in Table 1. All patients were male, aged 28–69 years (median age 34 years, mean age 37 years). The predominant clinical manifestations included acute abdominal pain, diarrhea, and jam-like stools, occasionally accompanied by pus, blood, and mucus. Colonoscopy revealed mucosal erosion

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Conflict of interest
The authors declare no conflict of interest related to this study.

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Table 1. Clinical data of 14 amoebic enteritis cases

Case No.	Sex	Age (year)	Location	Clinical manifestations	Endoscopic diagnosis
1	Male	37	Ileocecal region	Abdominal pain and diarrhea	Multiple ulcers in the ileocecal region
2	Male	47	Ileocecal region	Abdominal pain and hematochezia	Multiple polyps of the colon
3	Male	44	Appendiceal orifice	Abdominal pain and diarrhea	Multiple ulcers of the colon
4	Male	28	Ascending colon	Diarrhea and unclear abdominal pain	Multiple ulcerative lesions of the colon
5	Male	37	Ileocecal region	Abdominal pain and diarrhea	Ulcer of the ileocecal region
6	Male	34	Rectum	Diarrhea and mucopurulent bloody stool	Multiple rectal ulcers
7	Male	33	Ileocecal region, rectum	Abdominal pain and diarrhea, with jam-like stools	Multiple ulcers of the colon
8	Male	31	Ileocecal region, rectum	Abdominal pain hematochezia	Colonic ulcer
9	Male	30	Ileocecal region, rectum	Abdominal pain and diarrhea	Ulcerative colitis
10	Male	29	Ileocecal region, ascending colon, rectum	Abdominal pain hematochezia	Colonic ulcer
11	Male	41	Appendiceal orifice	Abdominal pain and diarrhea	Enteritis
12	Male	39	Transverse colon	Lower abdominal distension and incomplete bowel movements	Colonic ulcer
13	Male	69	Rectum	Abdominal pain and diarrhea	Enteritis
14	Male	31	Terminal ileum, ileocecal region, transverse colon, sigmoid colon, rectum	A 3-year history of ulcerative colitis with diarrhea	Colonic ulcer

and ulceration, although lesion locations varied. The ileocecal region was the most frequently affected site (8/14), followed by the rectum (6/14); other involved sites included the appendiceal orifice, ascending colon, transverse colon, and terminal ileum.

Notably, in case 7, diagnosis was delayed for 8 months due to the patient's refusal to undergo follow-up colonoscopies, during which antibiotics were administered without effect. A final diagnosis of AE was confirmed via colonoscopy and histopathological examination. In case 14, the patient had been previously diagnosed with ulcerative colitis 3 years earlier but showed no improvement despite long-term medical therapy. The correct diagnosis of AE was ultimately established by colonoscopy and pathological assessment.

Endoscopic findings

Colonoscopy revealed multiple ulcerative lesions in the colon, some of which were diffusely scattered (Fig. 1A, B). These ulcers were coated with yellowish-white exudate and blood. Larger ulcer measured approximately 3.0 – 3.5 cm in diameter.

Histopathological features

Microscopic examination demonstrated moderate to severe acute and chronic inflammation of the intestinal mucosa, characterized by infiltration of lymphocytes, plasma cells, and neutrophils, along with mucosal erosion or ulceration. Gray-blue, round or oval amoe-

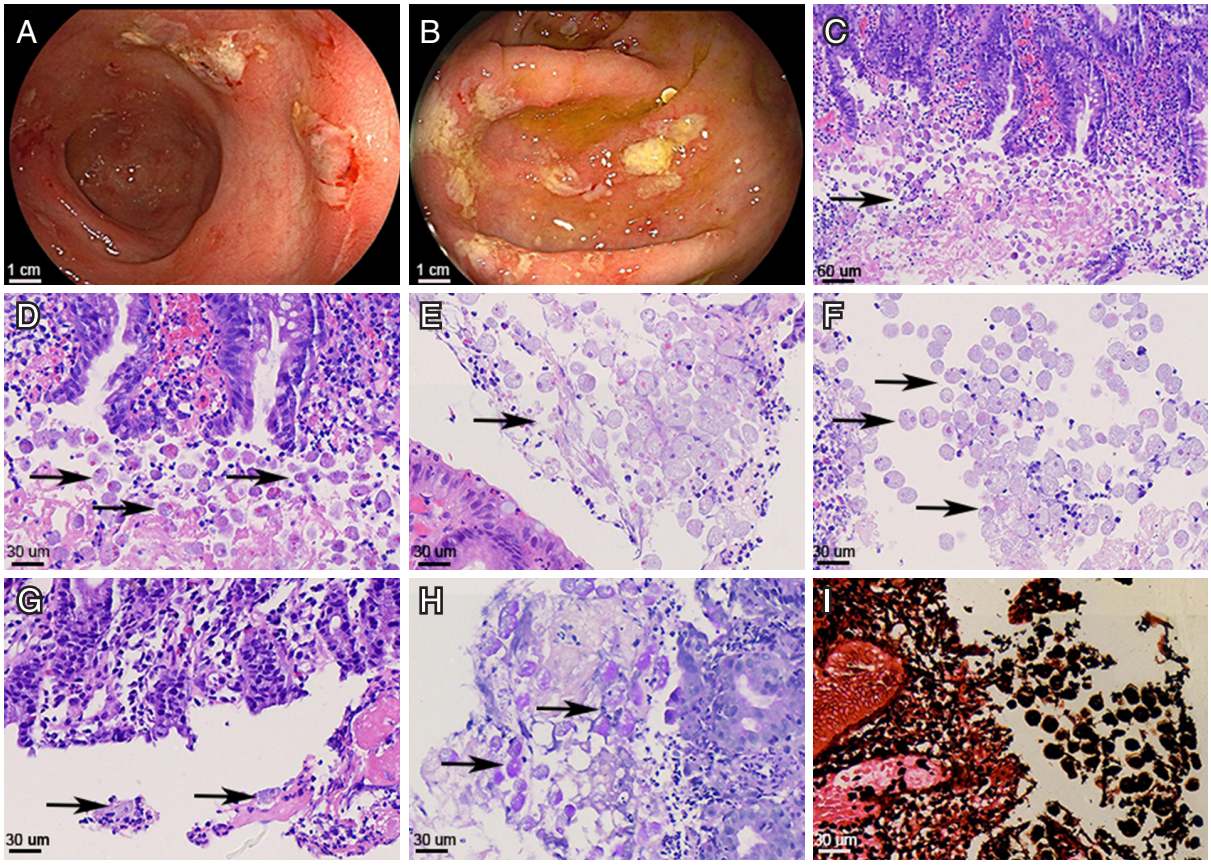

Fig. 1. (A) Colonoscopic image from case 6 showing multiple ulcers with yellow-white exudative coatings (moss) and peripheral bleeding. (B) Colonoscopy in case 7 revealed ulcerative lesions covered by yellow-white purulent exudates. (C, D) Microscopic examination of the intestinal mucosa revealed moderate acute and chronic inflammatory infiltrates. Numerous amoebic trophozoites (indicated by arrows), appearing gray-blue and round or oval in shape, were observed within necrotic debris and exudates. The trophozoites displayed eccentric or misaligned nuclei, well-distinct nuclear membranes, and prominent central nucleoli (C, H&E ×20; D, H&E ×40). (E, F) Trophozoite cytoplasm appeared foamy with light pink staining, containing glycogen vacuoles, engulfed erythrocytes, lymphocytes, and tissue fragments (H&E ×40). (G) Only 2 amoebic trophozoites (arrows) were identified under microscopic examination, highlighting the potential for misdiagnosis in cases with sparse parasitic load (H&E ×40). Special stains demonstrating positive results with (H) periodic acid-Schiff (×40) and (I) hexamine silver (×40), confirming the presence of amoebic organisms.

bic trophozoites were frequently identified within necrotic debris and exudate. These trophozoites exhibited eccentrically placed nuclei with clear nuclear membranes and prominent central nucleoli. Their foamy cytoplasm stained light pink and contained glycogen vacuoles and phagocytosed red blood cells, lymphocytes, or tissue fragments (Fig. 1C-G). PAS and hexamine silver stains were positive in cases 4 and 5 (Fig. 1H, I).

Treatment and outcomes

Nine patients received oral metronidazole (750 mg ter in die (tid)) and diloxanide (500 mg, tid) for 10 days. One patient with AE complicated by liver abscess was treated with intravenous metronidazole (500 mg every 8 h for 14 days), followed by oral metronidazole (750 mg tid for 10 days). Clinical symptoms such as abdominal pain, diarrhea, and purulent stools were significantly alleviated in these patients following treatment.

Discussion

E. histolytica infection can result in AE [3], a disease with variable clinical severity. Presentations range from mild abdominal discomfort to severe ulcerative colitis with mucus and blood (amoebic dysentery), appendicitis, or granulomatous lesions (amebomas) [4,5]. Consistent with prior studies [6,7], our data revealed that the ileocecal junction is the most frequently affected site, followed by the rectum.

E. histolytica is transmitted via the fecal-oral route, typically through ingestion of contaminated food or water, and does not require an intermediate vector. Upon entering the host, cysts undergo excystation in the terminal ileum, releasing motile trophozoites capable of mucosal invasion. These trophozoites adhere to and penetrate the intestinal epithelium, leading to mucosal damage, diarrhea, and colitis. While many infections are self-limiting, some progress to more severe disease, with trophozoites reverting to cysts and being excreted in feces, thereby perpetuating the transmission cycle [8,9]. The frequent involvement of the ileocecal region can thus be attributed to the early proliferation and invasion of trophozoites at this site. Clinical symptoms may include abdominal pain, diarrhea, jam-like stools, and mucinous bloody discharge. Accordingly, it is recommended that endoscopic evaluation in suspected AE cases pay close attention to the ileocecal and rectal regions for targeted biopsy.

Stool examination can aid in detecting amoebic cysts or trophozoites, but its diagnostic sensitivity remains suboptimal due to procedural and technical limitations [6]. In our study, 5 of the 14 patients had negative stool tests for trophozoites or cysts. Colonoscopy is a valuable diagnostic tool, particularly in the early stages when lesions appear as scattered mucosal erosions [7]. As the disease progresses, classic AE may present as flask-shaped ulcers with narrow necks and broad bases, accompanied by mucosal congestion, edema, and inflammatory exudates. Lesions may also appear as ulcers with a yellowish-white purulent coating and surrounding necrotic tissue [10]. However, with increased antibiotic use and improved host immunity, these characteristic lesions have become less common. In our cohort, all 14 patients exhibited ulcerative changes in the ileocecal region, colon, and rectum. Clinically, these lesions were initially misdiagnosed as nonspecific ulcerative colitis. Notably, 1 patient (case 14) had a 3-year history of presumed ulcerative colitis that was unresponsive to treatment; pathological reevaluation ultimately confirmed AE. This is a diagnostic challenge that shows how difficult it is to differentiate amoebic colitis from other ulcerative colitis based on colonoscopic findings alone.

Histopathological examination of intestinal biopsies remains the gold standard for AE diagnosis. The identification of trophozoites in biopsy specimens confirms the presence of amoebic colitis. Morphologically, trophozoites are round with a vesicular nucleus and cytoplasm containing glycogen vacuoles, ingested erythrocytes, lymphocytes, and tissue debris. Despite their diagnostic significance, trophozoites may be sparse in some biopsy samples due to sampling error, potentially resulting in false negatives.

The histopathological changes in AE, including cryptitis and crypt abscesses, may resemble those of other chronic inflammatory conditions, necessitating careful differential diagnosis. The differential diagnosis includes chronic ulcerative colitis, characterized by continuous and diffuse mucosal erosions with multiple superficial ulcers observed on colo-

noscopy. Histological findings in these cases typically demonstrate persistent chronic inflammation, crypt architectural distortion, and crypt abscesses [11,12]. Crohn's disease primarily affects the terminal ileum and exhibits segmental or skip-pattern involvement. Early lesions manifest as aphthous ulcers that progressively enlarge and coalesce into longitudinal or fissuring ulcers, ultimately creating a cobblestone mucosal appearance. Histopathological examination reveals mucosal ulceration accompanied by noncaseating epithelioid granulomas [13,14]. Intestinal tuberculosis is often associated with concurrent pulmonary or systemic tuberculosis. Colonoscopic findings in these patients commonly include mucosal congestion, edema, and horizontally oriented ulcers with irregular "mouse-bite" margins. Histologically, transmural granulomatous inflammation may be observed, sometimes accompanied by central caseous necrosis, surrounded by thick lymphocytic sheaths that may coalesce into nodular patches. Acid-fast staining confirms the presence of *Mycobacterium tuberculosis* [15-17]. Additionally, intestinal amebiasis must be differentiated from gastrointestinal malignancies, such as adenocarcinomas and lymphomas, through detailed histopathological evaluation and adjunctive diagnostic tools.

Following treatment with metronidazole and adjunctive anti-inflammatory therapy (diclofenac), 9 patients experienced substantial clinical improvement, such as resolution of abdominal pain, diarrhea, hematochezia, and purulent stools. These outcomes highlight the effectiveness of accurate diagnosis and appropriate therapy for AE. In contrast, delayed or incorrect diagnoses can prolong morbidity. For example, case 7 initially declined colonoscopy and received empirical antibiotic therapy for 8 months without improvement. A subsequent colonoscopy and biopsy confirmed AE, and the patient responded promptly to targeted treatment. Similarly, case 14 had been misdiagnosed with ulcerative colitis for 3 years. Following colonoscopic and pathological reassessment, AE was correctly diagnosed, and the patient achieved full clinical recovery with anti-amoebic therapy.

A key limitation of this study is the lack of comparative evaluation of endoscopic and histological features before and after treatment, which could have provided additional insights into the disease course and therapeutic response.

In classic cases, AE can be readily diagnosed based on characteristic clinical features such as chronic abdominal pain, diarrhea, and jam-like stools. However, in atypical or nonclassic presentations, diagnosis requires high clinical suspicion and targeted endoscopic biopsy from the affected intestinal segments. Accurate histopathological identification of trophozoites—using conventional microscopy and special stains such as PAS and hexamine silver—is essential for definitive diagnosis. Early recognition and appropriate treatment of AE are crucial for favorable patient outcomes and for preventing unnecessary treatment delays or misdiagnosis with other chronic inflammatory or neoplastic intestinal diseases.

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