https://doi.org/10.22516/25007440.1150

Case report

Fulminant Intestinal Amebiasis in a Young Female Patient: A Case Report

Luis Adolfo Collazos-Torres,[1*] Raúl Andrés Vallejo-Serna,[2] Alejandra Collazos-Torres,[3] Jesús Arbey Hoyos-Palacios.[4]

OPEN ACCESS

Citation:
Collazos-Torres LA, Vallejo-Serna RA, Collazos-Torres A, Hoyos-Palacios JA. Fulminant Intestinal Amebiasis in a Young Female Patient: A Case Report. Revista. colomb. Gastroenterol. 2024;39(4):483-487. https://doi.org/10.22516/25007440.1150

[1] Physician, Internal Medicine Resident, Hospital Universitario del Valle Evaristo García. Cali, Colombia.
[2] Physician, Specialist in Internal Medicine. Emergency Department, Hospital Universitario del Valle Evaristo García. Cali, Colombia.
[3] Emergency General Attending Physician, Clínica Tolima. Ibagué, Colombia.
[4] Specialist in Internal Medicine, Specialist in Clinical Gastroenterology and Digestive Endoscopy. Clinical Gastroenterologist and in Digestive Endoscopist, Clínica Imbanaco, Clínica Sebastián de Belalcázar. Cali, Colombia.

***Correspondence:** Luis Adolfo Collazos-Torres.
luisadolfocollazostorres@gmail.com

Received: 13/10/2023
Accepted: 26/02/2024

Abstract

Intestinal amebiasis is an endemic disease in developing countries caused by the protozoan *Entamoeba histolytica*. It is typically asymptomatic, but in rare cases, it can present with severe manifestations. This is the case of a 30-year-old female patient who, two days after undergoing open appendectomy, developed rectal bleeding and abdominal pain accompanied by signs of hemodynamic instability. Colonoscopy revealed multiple necrotic ulcers in the mucosa with abundant clots. The patient experienced clinical deterioration, persistent bleeding, and signs of peritoneal irritation, necessitating exploratory laparotomy. Intraoperative findings included hypoperfused colon and cecal perforation, requiring total colectomy. Histopathological analysis confirmed acute severe ulcerative colitis caused by *E. histolytica*.

Keywords

Gastrointestinal bleeding, hematochezia, *Entamoeba histolytica*, amoebic dysentery, colectomy.

INTRODUCTION

Entamoeba histolytica is an extracellular enteric protozoan with a preference for the large intestine and is responsible for approximately 10% of all *Entamoeba* infections worldwide, with the remaining 90% attributed to other species. Its identification dates back to the 19th century when Lamb first described it. Later, Lösch named the organism *Amoeba coli* and demonstrated its ability to cause colonic ulceration and dysentery[1]. In 1883, Koch identified amoebas in an ulcerated human intestine, but it was not until 1903 that Schaudinn coined the term *Entamoeba histolytica* due to its capacity to cause tissue lysis[1,2].

Intestinal infection caused by *E. histolytica* is referred to as amebiasis, which ranks among the top three parasitic infections leading to death worldwide[3]. Approximately 90% of amebiasis cases are asymptomatic, while 10% result in invasive disease. However, its effects are not confined to the intestine; it can also cause extraintestinal disease, such as hepatic abscesses, pneumonia, pericarditis, and even cerebral amebiasis, although extraintestinal manifestations account for less than 1% of cases[4].

© 2024 Asociación Colombiana de Gastroenterología

CASE PRESENTATION

We report the case of a 30-year-old female patient with no known medical history who, two days after undergoing an open appendectomy, developed multiple episodes of profuse rectal bleeding, accompanied by general malaise and diffuse abdominal pain. Upon admission to the emergency department, she was in poor condition, presenting with tachycardia, dehydration, and generalized pallor. Laboratory tests revealed leukocytosis (26,750 cells/μL) predominantly due to neutrophils, hemoglobin of 9.5 g/dL, platelet count of 225,000/μL, blood urea nitrogen (BUN) of 9.0 mg/dL, serum creatinine of 0.5 mg/dL, and normal coagulation times.

A total colonoscopy was performed (**Figure 1**), which revealed multiple mucosal ulcerations with necrosis, clots, and patchy ulcerations in the rectum, descending colon, and transverse colon, with diffuse bleeding that could not be controlled endoscopically. Following the procedure, the patient experienced significant rectal bleeding, prompting an abdominal CT angiography, which showed irregular, concentric mural thickening of the cecum and ascending colon without evidence of contrast extravasation. Due to the persistence of bleeding and a hemoglobin drop to 6.9 g/dL, an abdominal aortogram was performed. At the level of the right hypogastric artery, contrast extravasation was observed in the distal branch, leading to endovascular embolization of the affected vessel. Initial differential diagnoses included severe ulcerative colitis and ischemic colitis, which were rapidly ruled out based on the clinical presentation and the absence of relevant medical history or risk factors.

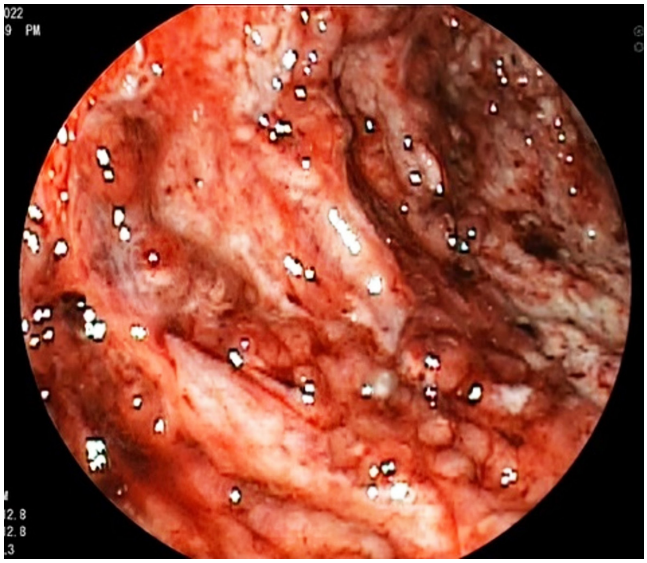


Figure 1. Ischemic colon with multiple ulcerations, mucosal denudation, and abundant clots observed during colonoscopy. Author's File.

The patient had a torpid clinical course despite the treatment provided. Due to the development of signs of peritoneal irritation, an exploratory laparotomy was performed, revealing peritonitis in the right two quadrants, along with a hypoperfused and pale colon and a perforation in the cecum. A total colectomy was performed (**Figure 2**), and the patient completed her recovery in the intensive care unit (ICU), where vasopressors and invasive mechanical ventilation were successfully discontinued. *Pluralibacter gergoviae* was isolated from the peritoneal fluid culture, while blood cultures were negative. She was treated with meropenem, and due to her favorable clinical progression, she was discharged from the hospital with an ileostomy. However, one week later, the patient was readmitted due to worsening abdominal pain, accompanied by nausea and fever. A review of the colon pathology report revealed severe, perforating acute colitis caused by *Entamoeba histolytica*, along with acute suppurative peritonitis (**Figures 3** and **4**). The Infectious Disease team recommended treatment with intravenous (IV) metronidazole 750 mg every eight hours for ten days, followed by oral (PO) paromomycin 500 mg every eight hours for seven days, resulting in the patient's full recovery.



Figure 2. Total colectomy. The ischemic colon is shown with fibrinopurulent membranes, multiple necrotic areas, and perforations. Author's File.

DISCUSSION

Acute lower gastrointestinal (GI) bleeding, historically defined as bleeding originating below the ligament of Treitz, has been reclassified to distinguish it from bleeding originating in the midgut. It is now limited to bleeding that arises from the colon, rectum, or anus[5,6]. Its defini-

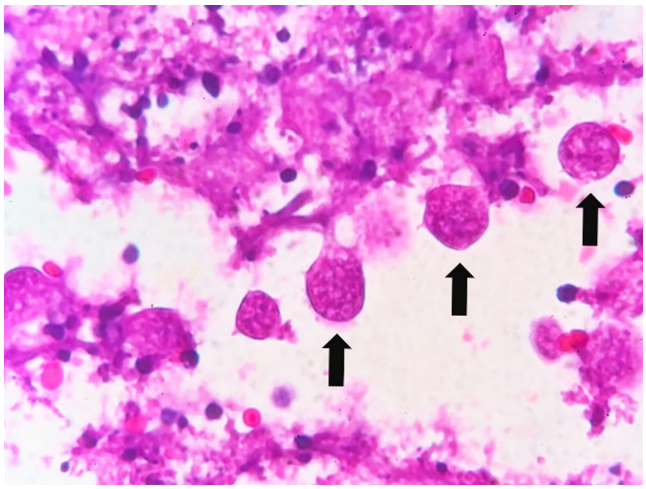


Figure 3. Abundant acute inflammatory infiltrate and multiple *Entamoeba histolytica* trophozoites (black arrows). Hematoxylin and eosin stain of a colon biopsy section at 10x magnification. Author's File.

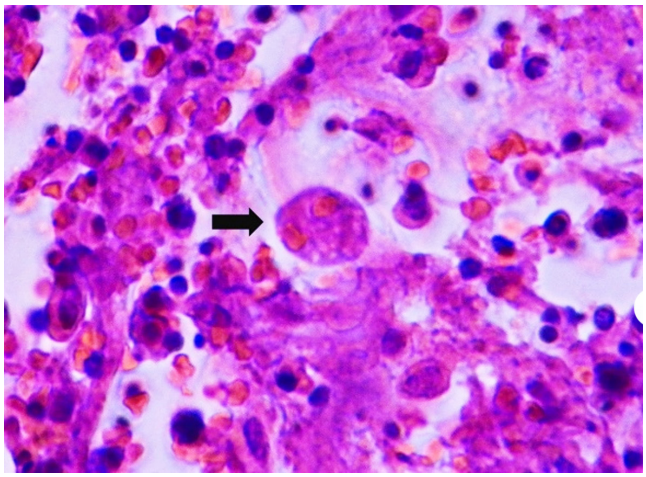


Figure 4. Abundant acute inflammatory infiltrate and a trophozoite of *Entamoeba histolytica* with erythrophagocytosis (black arrow). Hematoxylin and eosin stain of a colon biopsy section at 10x magnification. Author's File.

tion, which is somewhat arbitrary, refers to recent onset within a period of fewer than three days[5]. Acute lower GI bleeding has an incidence of 20 to 30 cases per 100,000 person-years and accounts for 20% of all GI bleeds[7]. It is most commonly associated with colonic sources, such as diverticulosis or angiodysplasia[8], and over 80% of cases resolve spontaneously[5,8]. Clinically, it often presents as hematochezia, though melena may occur less frequently. Advanced age and comorbidities are linked to higher morbidity and mortality[6].

Diverticular disease is the leading cause of acute lower GI bleeding, accounting for 30% to 65% of cases[9]. This is followed by ischemic colitis (5% to 20%), which generally affects elderly patients with cardiovascular risk factors[6,7]; hemorrhoids (5% to 20%), more common in younger individuals[6,7]; colorectal polyps and neoplasms (2% to 15%); angiodysplasia (5% to 10%), which is more prevalent in those over 60 years[6,7]; and inflammatory bowel disease (3% to 5%)[6]. Infectious colitis is one of the least common causes, accounting for only 2% to 5% of cases[6].

The first-line diagnostic tool is colonoscopy, which can provide a definitive diagnosis in 74% to 100% of cases. It also allows for a more precise description of the lesion, even in the absence of active bleeding, and in many cases facilitates therapeutic intervention[10]. The timing of colonoscopy remains a subject of debate. Although urgent or early colonoscopy (within 6 to 12 hours) has been shown to improve diagnostic yield and shorten hospital stays, it does not significantly affect major outcomes, such as mortality[10], compared to elective colonoscopy (performed within 36 to 60 hours)[10,11].

Additional studies, such as CT angiography, are reserved for hemodynamically unstable patients in whom active bleeding is suspected, as it provides a rapid and minimally invasive method to localize the bleeding site and guide subsequent endoscopic or radiological therapy. If a positive finding is observed, catheter angiography can enable embolization of the responsible vessel, thus avoiding emergency laparotomy whenever possible[12], as was the case in our patient.

Bleeding caused by infectious colitis typically presents as acute diarrhea (lasting fewer than 14 days) with blood in the stool, often in the context of recent travel to endemic areas or consumption of contaminated food[7,13]. This diagnosis is made when an invasive or non-invasive pathogen triggers diffuse mucosal inflammation of the colon. Infectious colitis should be suspected in patients with acute diarrhea, particularly when they present with heavily blood-stained stools, tenesmus, urgency, or evidence of fecal inflammatory markers. The diagnosis is confirmed when diffuse colonic inflammation is observed during colonoscopy[13]. Given that Latin America is considered an endemic region for *E. histolytica*, the presence of bloody diarrhea in this context should prompt clinicians to actively investigate for this pathogen[14].

Amebiasis is a common parasitic infection in many tropical and subtropical regions of the world, particularly in areas with poor sanitation. It can present in various forms: asymptomatic infection (90%), non-invasive symptomatic infection (6%–8%), and acute amebic colitis (dysentery)[15]. Nearly all infections are acquired through the consumption of food or water contaminated with feces containing cysts, a scenario that is relatively frequent in developing

countries[4,16]. Less common transmission routes include oral-anal sexual contact and contaminated endoscopes or enema devices[16].

Amebic cysts enter the human intestinal lumen and produce trophozoites capable of invading intestinal epithelial cells[17]. This process involves the parasite's Gal/GalNAc lectin binding to the galactose (Gal) or N-acetyl-D-galactosamine (GalNAc) carbohydrates of the host cell membrane[18]. After adhering to mesothelial cells, the amoeba employs cytotoxic mechanisms to induce cell death and tissue invasion, including apoptosis, phagocytosis, and trogocytosis[19,20]. In rare cases (<0.5% of cases), fulminant necrotizing amebic colitis occurs, leading to gangrenous necrosis of the large intestine. The mortality rate for this condition is extremely high, ranging from 55% to 100%, with secondary sepsis due to perforation-related peritonitis being the leading cause of death[15].

In our patient's case, the preceding episode of appendicitis raises the suspicion that the accumulation of parasites may have triggered acute inflammation of the appendix, even though the histopathological report of the appendix did not mention trophozoites. Amebic appendicitis is relatively uncommon, with an incidence of 0.5% to 2.3% in endemic areas[21,22]. However, its association with fulminant necrotizing colitis, as observed in our patient, is even rarer, requiring a high index of suspicion for timely diagnosis.

CONCLUSION

Intestinal amebiasis, caused by *Entamoeba histolytica*, is an endemic parasitic infection in our region. Although it is often asymptomatic, it has the potential to become severe and invasive, posing a life-threatening risk. One of its most frequent manifestations is lower GI bleeding, which, when presenting as an initial symptom, poses a diagnostic challenge due to its wide range of possible etiologies. However, a detailed and thorough medical history, an evaluation of risk factors, and a high index of suspicion guide the selection of appropriate diagnostic studies and facilitate a definitive diagnosis.

REFERENCES

1. Carrero JC, Reyes-López M, Serrano-Luna J, Shibayama M, Unzueta J, León-Sicairos N, et al. Intestinal amoebiasis: 160 years of its first detection and still remains as a health problem in developing countries. Int J Med Microbiol. 2020;310(1):151358. https://doi.org/10.1016/j.ijmm.2019.151358
2. Pinilla AE, López MC, Viasus DF. Historia del protozoo Entamoeba histolytica. Rev Méd Chile. 2008;136(1):118-24. https://doi.org/10.4067/S0034-98872008000100015
3. Kantor M, Abrantes A, Estevez A, Schiller A, Torrent J, Gascon J, et al. Entamoeba Histolytica: Updates in Clinical Manifestation, Pathogenesis, and Vaccine Development. Can J Gastroenterol Hepatol. 2018;2018:4601420. https://doi.org/10.1155/2018/4601420
4. Haque R, Huston CD, Hughes M, Houpt E, Petri WA. Amebiasis. N Engl J Med. 2003;348(16):1565-73. https://doi.org/10.1056/NEJMra022710
5. Qayed E, Dagar G, Nanchal RS. Lower Gastrointestinal Hemorrhage. Critical Care Clinics. 2016;32(2):241-54. https://doi.org/10.1016/j.ccc.2015.12.004
6. Gralnek IM, Neeman Z, Strate LL. Acute Lower Gastrointestinal Bleeding. N Engl J Med. 2017;376(11):1054-63. https://doi.org/10.1056/NEJMcp1603455
7. Hawks MK, Svarverud JE. Acute Lower Gastrointestinal Bleeding: Evaluation and Management. Am Fam Physician. 2020;101(4):206-212.
8. Whitehurst BD. Lower Gastrointestinal Bleeding. Surg Clin North Am. 2018;98(5):1059-72. https://doi.org/10.1016/j.suc.2018.06.007
9. Strate LL, Naumann CR. The Role of Colonoscopy and Radiological Procedures in the Management of Acute Lower Intestinal Bleeding. Clin Gastroenterol Hepatol. 2010;8(4):333-43. https://doi.org/10.1016/j.cgh.2009.12.017
10. Beck KR, Shergill AK. Colonoscopy in Acute Lower Gastrointestinal Bleeding. Gastrointestinal Endosc Clin North Am. 2018;28(3):379-90. https://doi.org/10.1016/j.giec.2018.02.009
11. Strate LL, Gralnek IM. ACG Clinical Guideline: Management of Patients With Acute Lower Gastrointestinal Bleeding. Am J Gastroenterol. 2016;111(4):459-74. https://doi.org/10.1038/ajg.2016.41
12. Oakland K, Chadwick G, East JE, Guy R, Humphries A, Jairath V, et al. Diagnosis and management of acute lower gastrointestinal bleeding: guidelines from the British Society of Gastroenterology. Gut. 2019;68(5):776-89. https://doi.org/10.1136/gutjnl-2018-317807
13. Iqbal T, DuPont HL. Approach to the patient with infectious colitis: clinical features, work-up and treatment. Curr Opin Gastroenterol. 2021;37(1):66-75. https://doi.org/10.1097/MOG.0000000000000693
14. Thielman NM, Guerrant RL. Clinical practice. Acute infectious diarrhea. N Engl J Med. 2004;350(1):38-47. https://doi.org/10.1056/NEJMcp031534
15. Beg MY, Bains L, Mahajan R, Lal P, Choudhury S, Kumar NP, et al. Fulminant Necrotising Amoebic Colitis of Whole of Large Bowel: A Rare Complication of a Common Infectious Disease. Case Rep Infect Dis.

2020;2020:8845263.
https://doi.org/10.1155/2020/8845263
16. Jessurun J. The Differential Diagnosis of Acute Colitis: Clues to a Specific Diagnosis. Surg Pathol Clin. 2017;10(4):863-85.
https://doi.org/10.1016/j.path.2017.07.008
17. Uddin MJ, Leslie JL, Petri WA. Host Protective Mechanisms to Intestinal Amebiasis. Trends Parasitol. 2021;37(2):165-75.
https://doi.org/10.1016/j.pt.2020.09.015
18. Petri WA, Smith RD, Schlesinger PH, Murphy CF, Ravdin JI. Isolation of the galactose-binding lectin that mediates the in vitro adherence of Entamoeba histolytica. J Clin Invest. 1987;80(5):1238-44.
https://doi.org/10.1172/JCI113198
19. Begum S, Quach J, Chadee K. Immune Evasion Mechanisms of Entamoeba histolytica: Progression to Disease. Front Microbiol. 2015;6:1394.
https://doi.org/10.3389/fmicb.2015.01394
20. Ralston KS, Solga MD, Mackey-Lawrence NM, Somlata, Bhattacharya A, Petri WA Jr. Trogocytosis by Entamoeba histolytica contributes to cell killing and tissue invasion. Nature. 2014;508(7497):526-30.
https://doi.org/10.1038/nature13242
21. Ito D, Hata S, Seiichiro S, Kobayashi K, Teruya M, Kaminishi M. Amebiasis presenting as acute appendicitis: Report of a case and review of Japanese literature. Int J Surg Case Rep. 2014;5(12):1054-7.
https://doi.org/10.1016/j.ijscr.2014.10.035
22. Otan E, Akbulut S, Kayaalp C. Amebic Acute Appendicitis: Systematic Review of 174 Cases. World J Surg. 2013;37(9):2061-73.
https://doi.org/10.1007/s00268-013-2079-5