



Amoebic Colitis Presenting as Subacute Intestinal Obstruction with Perforation

**Renuka Verma¹, Archana Budhwar^{1*}, Priyanka Rawat¹, Niti Dalal¹,
Anjali Bishlay¹ and Sunita Singh¹**

¹Pt. B. D. Sharma Post Graduate Institute of Medical Sciences, Rohtak-124001, Haryana, India.

Authors' contributions

This work was carried out in collaboration among all authors. Authors RV and SS designed the study, reviewed the manuscript and edited it. Author Archana Budhwar wrote the protocol and wrote the first draft of the manuscript. Author PR managed the literature searches. Authors ND and Anjali Bishlay managed the analyses of the study. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJTDH/2020/v41i1630367

Editor(s):

(1) Dr. Giuseppe Murdaca, University of Genoa, Italy.

Reviewers:

(1) Ammar M. Al-Aalim, Mosul University, Iraq.

(2) Tarunbir Singh, Guru Angad Dev Veterinary & Animal Sciences University, Ludhiana.

Complete Peer review History: <http://www.sdiarticle4.com/review-history/61831>

Case Study

Received 10 August 2020
Accepted 16 October 2020
Published 12 November 2020

ABSTRACT

Infestation with *Entamoeba histolytica* is worldwide, especially in developing areas. Presented case study included amoebic colitis in a 45 years old man complaining of abdominal distension and non-passage of stools since three days. Abdominal region was diffusely distended and tender in right iliac fossa. Plain abdominal radiography revealed prominent gut loops and minimal intergut free fluid. At laparotomy, malrotation of gut was present. Histopathological examination of intestinal samples confirmed final diagnosis of amoebic colitis post-operatively.

Keywords: Amoebic; intestinal obstruction; ileocaecal; colitis; perforation.

1. INTRODUCTION

Intestinal amoebiasis is an infestation of human intestine caused by the pathogenic enteric protozoan *Entamoeba histolytica*. *E. histolytica*

belongs to the phylum Amoebozoa in the kingdom Protista. Approximately 50 million people develop colitis or extraintestinal disease worldwide as a result of *E. histolytica* infection. Around 1,00,000 deaths were reported annually

*Corresponding author: Email: archanabudhwar@gmail.com;

from amoebic colitis. *E. histolytica* shares identical morphological characteristics with other species of intestinal amoebae (*E. dispar*, *E. hartmani*, *E. moshkovskii* and *E. bangladeshi*), which are non-pathogenic species. Prevalence is disproportionately higher in developing countries and is related to low socioeconomic conditions and poor sanitation. Infection by *E. histolytica* starts with the ingestion of mature cysts from fecally contaminated food or water. Excystation takes place within the intestine to develop the trophozoites. An inflammatory response develops after interaction of parasite with the intestinal epithelium. Extraintestinal dissemination may occur and trophozoites reach the peritoneum, liver and other areas in some cases [1-3].

Amoebic colitis has variable presentation which might range from asymptomatic infection, symptomatic noninvasive infection, acute proctocolitis to fulminant colitis with perforation [4]. Colonic amoebiasis and its manifestations are well documented in literature. However, amoebic involvement of small intestine continues to be a rare presentation.

2. CASE REPORT

A 45 year old male, vegetable seller by occupation from non-tropical region presented with history of abdominal distension, vomiting and non-passage of stools since 3 days. He had also history of loose stools 10 days back and abdominal pain since 1 month. He was a chronic alcoholic with no history of hypertension, diabetes mellitus, or tuberculosis. On physical examination, patient was afebrile and his vitals were within the normal range. Abdominal region was mildly tender and diffusely distended. Plain abdominal radiography demonstrated prominent gut loops and minimal intergut free fluid. A preliminary diagnosis of subacute intestinal obstruction was made.

Laparotomy demonstrated malrotation of gut. Duodenojejunal flexure was present over left hypochondrial region. Whole of caecum together with ileocecal junction and a part of ileum was found gangrenous and resected surgically.

Gross examination of resected gut showed necrotic and exudate covered areas.

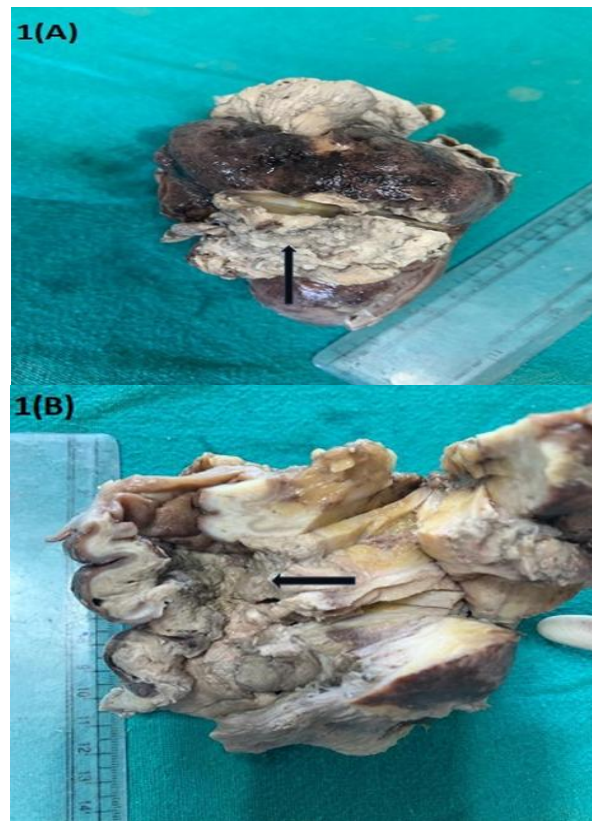


Fig. 1. (A, B). External and cut surface of ileum showing {arrow} necrotic and exudate covered areas with perforation

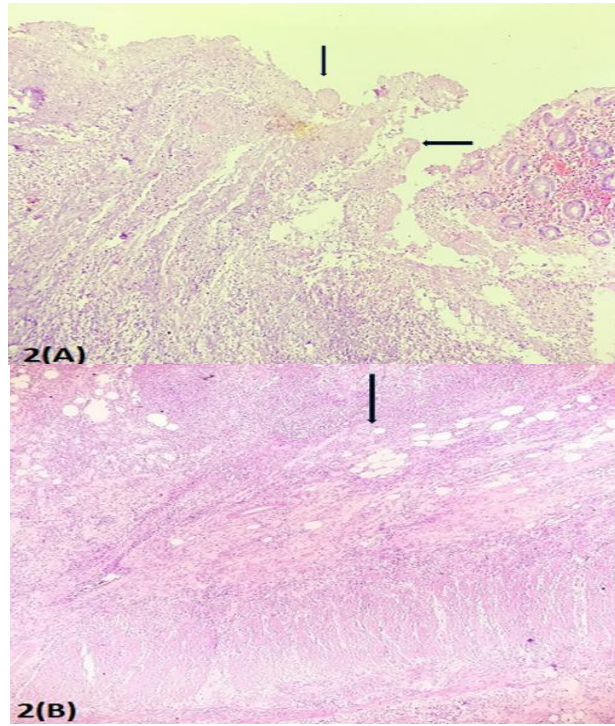


Fig. 2. (A). H & E stained slide demonstrating mucosal and submucosal ulceration of ileum {arrows}, (B). Transmural mixed inflammatory infiltrate upto serosa (100x)

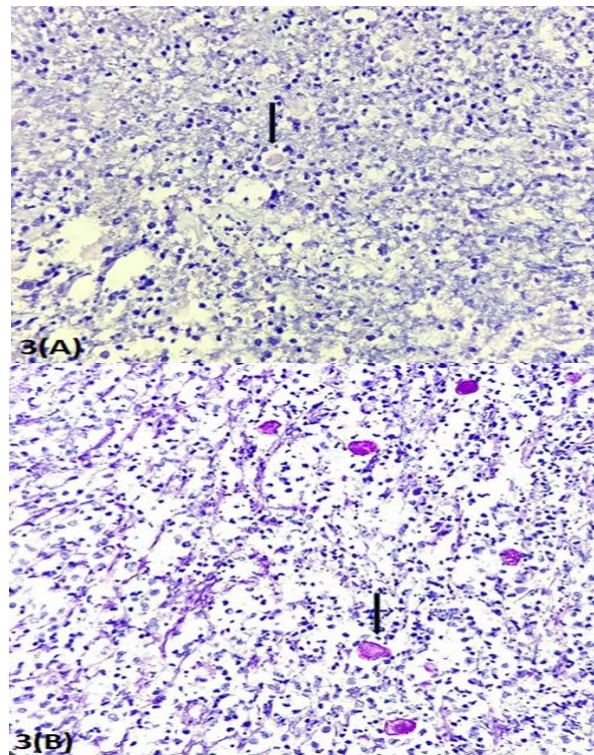


Fig. 3. (A). H & E stained slide showing round to oval bodies, (B). Arrow showing amoeba highlighted in PAS staining (400x)

Two perforations with surrounding areas of ulceration were identified (Fig. 1). Histopathological examination from perforation and exudate covered areas show mucosal and submucosal ulceration with extensive areas of necrosis, karyorrhectic debris and infiltration by mixed inflammatory infiltrate comprising of eosinophils, neutrophils, lymphocytes, histiocytes and plasma cells (Fig. 2). Few embedded amoebic trophozoites of size 15-20 μm , round to oval with abundant cytoplasm, central round small nuclei with prominent nuclear membrane were seen. These organisms were better appreciated on PAS stain (Fig. 3). No granuloma or evidence of malignancy were identified. The lymph nodes showed reactive hyperplasia, increased number of eosinophils and were negative for tuberculosis.

3. DISCUSSION

The amoebiasis is globally widespread, the developing countries of tropics and subtropics bear the highest burden of amoebiasis, particularly in regions with inadequate hygiene and access to sanitation [5].

The causative organism of amoebiasis is *Entamoeba histolytica* which is a protozoan parasite and affects mainly gastrointestinal tract and the liver in human body. Ingestion of the cysts of the parasite from food or water contaminated with faeces causes gastrointestinal manifestation. Once ingested, trophozoites are formed by excystation. These trophozoites migrate to the large intestine and more cysts are produced by binary fission there by invading the intestinal epithelium. These trophozoites can pass to extra-intestinal sites via hepatic portal circulation to liver or further hematogenously disseminate to distant organs like brain and lungs. Symptoms are seen in weeks or may develop years after infection [5,6].

The pathological features of amoebic colitis include mucosal thickening, multiple discrete ulcers with regions of normal colonic mucosa in between, diffuse inflammation, mucosal oedema, necrosis and perforation of the intestinal wall. The hallmark of amoebic colitis is amoebic invasion through the mucosa and into the submucosal tissues. The classic flask-shaped ulcer of amoebiasis is formed by lateral extension through submucosal tissues [7].

Entamoeba infections are generally asymptomatic in more than 90% cases. Increased disease severity and mortality are

noted in young age, pregnancy, malignancy, malnutrition, alcoholism and corticosteroid use. Amoebic colitis usually encompasses a subacute onset, with symptoms starting from mild diarrhea to severe dysentery with abdominal pain. Symptoms tend to be frequently missed and the differential diagnosis is broad. Infectious causes that need to be excluded include shigella, salmonella, campylobacter, and enteroinvasive and enterohemorrhagic *Escherichia coli*. Noninfectious causes include inflammatory bowel disease (IBD), intestinal tuberculosis, diverticulitis, and ischemic colitis. It is important to differentiate amoebiasis from IBD because corticosteroid, the core treatment for IBD, is contraindicated in amoebic colitis. On endoscopy, Discrete mucosal ulcers with intervening normal mucosa compared to the presence of mucosal changes around ulcers should raise a suspicion of colonic amoebiasis whereas later is suggestive of IBD. On histology, necrotic material admixed with proteinaceous material and mucin, significant surface epithelial changes like shortening and tufting adjacent to ulcers, mild chronic inflammation extending into the deep mucosa, and mild architectural alteration are characteristic features of amoebiasis [1,8-10].

Amoebic colitis can also present as ameboma which usually manifest as gut obstruction or lower gastrointestinal bleeding. It may mimic colon carcinoma, Crohn's disease, Non-Hodgkin's lymphoma, tuberculosis, fungal infection, AIDS associated lymphoma and Kaposi's sarcoma in colonoscopy findings. Amebomas are due to repeated episodes of untreated or partially treated amoebic colitis which emphasizes the importance of early diagnosis of amoebic colitis. Serious complications like fulminant necrotizing colitis, toxic megacolon, and fistulizing perianal ulcerations are presented when diagnosis and treatment is not done timely [8,11].

The diagnosis of *Entamoeba histolytica* may be done by various diagnostic tools including microscopy, serology, antigen detection, molecular techniques, and colonoscopy with histological examination. Recently stool molecular RT-PCR has evolved as the "gold standard" accurate diagnostic assay to differentiate *Entamoeba* species [1,8].

All *Entamoeba histolytica* infections should be treated for their potential risk of invasion and further spread. Systemic drugs such as

metronidazole or tinidazole are highly effective for elimination of *E. histolytica* trophozoites [3].

4. CONCLUSION

Amoebiasis is not an uncommon intestinal infection but can rarely present as an intestinal obstruction and perforation. It is one of the differential diagnoses of intestinal obstruction and inflammatory bowel disease. Early recognition followed by antiamoebic treatment, combined with urgent aggressive resectional surgery are necessary to cut back morbidity and mortality in complicated cases of amoebic colitis.

CONSENT AND ETHICAL APPROVAL

As per university standard guideline, participant consent and ethical approval have been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Wang H, Kanthan R. Multiple colonic and ileal perforations due to unsuspected intestinal amoebiasis-Case report and review. *Pathol Res Pract* [Internet]. 2020;216(1):152608.
2. Mi-ichi F, Yoshida H. Unique features of entamoeba sulfur metabolism; compartmentalization, physiological roles of terminal products, evolution and pharmaceutical exploitation. *Int J Mol Sci*. 2019;20(19):1-13.
3. Roure S, Valerio L, Soldevila L, Salvador F, Fernández-Rivas G, Sulleiro E, et al. Approach to amoebic colitis: Epidemiological, clinical and diagnostic considerations in a non-endemic context (Barcelona, 2007-2017). *PLoS One*. 2019;14(2):1-10.
4. Moorchung N, Singh V, Srinivas V, Jaiswal S, Singh G. Caecal amebic colitis mimicking obstructing right sided colonic carcinoma with liver metastases: A rare case. *J Cancer Res Ther*. 2014;10(2):440-2.
5. Shirley DAT, Farr L, Watanabe K, Moonah S. A review of the global burden, new diagnostics and current therapeutics for amoebiasis. *Open Forum Infect Dis*. 2018;5(7):1-9.
6. Gupta SS, Singh O, Shukla S, Raj MK. Acute fulminant necrotizing amoebic colitis: A rare and fatal complication of amoebiasis: A case report. *Cases J*. 2009;2(9):1-4.
7. Stanley SL. Amoebiasis. *Lancet*. 2003;361(9362):1025-34.
8. 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;4601420:1-6.
9. Singh R, Balekuduru A, Simon EG, Alexander M, Pulimood A. The differentiation of amebic colitis from inflammatory bowel disease on endoscopic mucosal biopsies. *Indian J Pathol Microbiol*. 2015;58(4):427-32.
10. Pai SA. Amebic colitis can mimic tuberculosis and inflammatory bowel disease on endoscopy and biopsy. *Int J Surg Pathol*. 2009;17(2):116-21.
11. Sugi Subramaniam RV, Ravichandran P, Senthil Kumar P, Sukumar R, et al. An interesting presentation of ameboma – A case report and review of literature. *Gastroenterol Pancreatol Liver Disord*. 2018;6(4):1-4.

© 2020 Verma et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

The peer review history for this paper can be accessed here:

<http://www.sdiarticle4.com/review-history/61831>