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Research Article

Amoebiasis Associated Colonic Infections Manifesting Clinically as Acute Appendicitis and Crohn's Disease

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I N F O

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A B S T R A C T

Amoebiasis or amoebic dysentery is a term used to describe an infection caused by the protozoan Entamoeba histolytica. Most infections are asymptomatic, but the invasive intestinal disease may occur manifesting with several weeks of cramping, abdominal pain, watery or bloody diarrhoea, and weight loss. Courses on this disease and the incidence of this parasite in industrialised nations are not yet fully investigated. We report two rare cases of a 59-year-old lady and a 30-year-old female who manifested as acute appendicitis and Crohn's disease respectively. Multi-sections from a biopsy of both cases revealed necrotic tissue along with multiple amoebic trophozoites of Entamoeba histolytica which were confirmed by doing a PAS stain.

Keywords: Amoebiasis, Crohn's Disease, Colonoscopy, Glands, RBC

Introduction

Amoebiasis or amoebic dysentery is a term used to describe an infection caused by the protozoan Entamoeba histolytica.[1] Most infections are asymptomatic, but the invasive intestinal disease may occur manifesting in several weeks of cramping, abdominal pain, watery or bloody diarrhoea, and weight loss.[2] Disseminated, extra-intestinal diseases such as liver abscess, pneumonia, purulent pericarditis, and even cerebral amoebiasis have been described. In 2010 the World Health Organisation estimated the number of infections with Entamoeba histolytica at about 50 million cases including 100000 fatal courses.[3] In most cases, this infection is a subclinical event with few or no symptoms noticeable for the patient. Courses on this disease and the incidence of this parasite in industrialised nations are not yet fully investigated. We report two rare cases of a 59-year-old lady and a 30-year-old female who manifested as acute appendicitis and Crohn's disease respectively.

Case 1

A 59-year-old female came to our hospital complaining of intense pain in the right abdomen for four days associated with nausea and vomiting. On USG appendicular lump with acute appendicitis was suggested. Open Appendicectomy was performed and sent for histopathological examination Grossly we received two containers one labelled as an appendix and the other as an appendicular lump. In container 1 we received grey-white shaggy necrotic tissue and multi sections revealed severely necrotic tissue forming

an abscess along with multiple amoebic trophozoites of Entamoeba histolytica that could be identified in the necrotic debris. (PAS stain positive). In container 2 we received fibrofatty tissue containing mostly peri appendicular tissue. Multiple sections show multiple trophozoites of Entamoeba histolytica.

Case 2

A 30-year young female came with complaints of chronic diarrhoea, abdominal pain, and anorexia for one and a half years. There were no complaints of fever. On haematological evaluation, there was severe iron deficiency anaemia. UGI endoscopy revealed gastric ulcers and Colonoscopy showed multiple colonic ulcers right> left colon. The ileocecal valve/ ileum appeared normal. A clinical diagnosis of Abdominal Koch's vs Crohn's disease was made and an ulcer biopsy from ascending colon was sent for histopathological confirmation. We received a single grey-white soft tissue measuring 0.2x0.2x0.1 cm. Multiple sections revealed superficial fragments of colonic mucosa. The colonic glands are showing mild Paneth cell hyperplasia. Occasional colonic glands are showing cryptitis. Goblet cells are maintained and there was no crypt abscess formation, Mucosa was lined by sheets of round cells with prominent nucleoli, some showing RBC engulfment.

Discussion

Entamoeba histolytica is an invasive enteric protozoan.

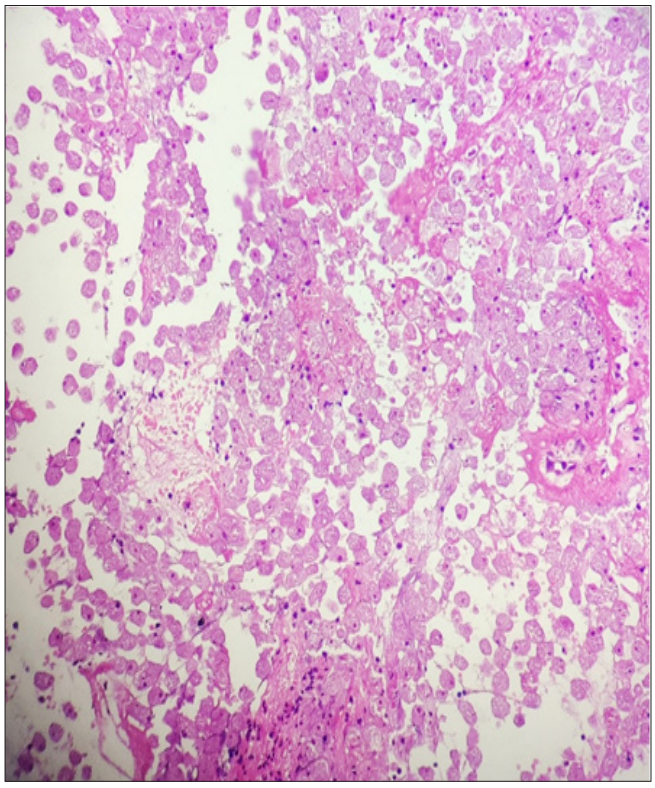

Figure 1.PAS Stain was Positive for Amoeba

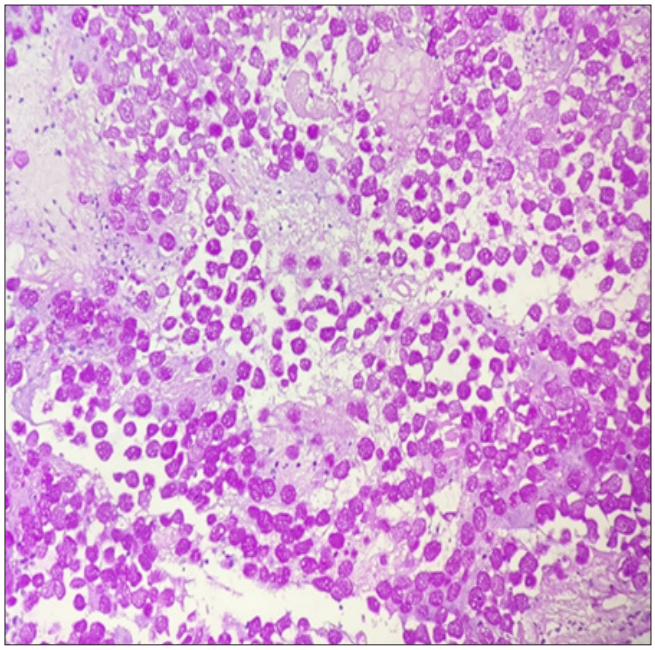

Figure 2.A Histopathological Diagnosis of Amoebic Colitis with Non-Specific Chronic Inflammation was Made

Infection begins typically with the ingestion of mature, quadrinucleated cysts found in fecally contaminated food or water. Excystation occurs in the small intestine with the release of motile trophozoites, which migrate to the large intestine. Through binary fission, trophozoites form new cysts, and both stages are shed in faeces, but only cysts have the potential to transmit disease due to the protection conferred by their wall. Cysts can survive days to weeks in the external environment, while trophozoites are rapidly destroyed once outside the body or by gastric secretions if ingested. Trophozoites have the capacity to adhere and lyse the colonic epithelium and subsequently spread haematologically through the portal vein system to distant sites such as the peritoneum, liver, lung, or brain. Adherence to the colonic mucus layer and colonization is through the Gal/GalNAc lectin, which targets galactose and N-acetyl-D-galactosamine residues found on O-linked sugar side chains of mucins.[4] Virulence among E. histolytica species is currently under investigation, and the presence of certain enzymes has been linked to an increased risk for invasive disease. For instance, glycosidases such as sialidase, N-acetylgalactosamidase, and N-acetylglucosaminidase are needed to remove the branched polysaccharides from mucin cells. This allows the trophozoites to degrade the protective mucous barrier and subsequently penetrate the colonic epithelium increasing the risk of metastasis to distant sites.[5] Approximately ninety per cent of Entamoeba infections are asymptomatic. Risk factors that are associated with increased disease severity and mortality include young age, pregnancy, malignancy, malnutrition, alcoholism, and corticosteroid use.[6] Amoebic

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colitis generally has a subacute onset, with symptoms that can range from mild diarrhoea to severe dysentery, with abdominal pain and watery or bloody diarrhoea. Symptoms tend to be nonspecific and the differential diagnosis is broad. Unusual but serious complications such as fulminant necrotizing colitis, toxic megacolon, and fistulizing perianal ulcerations can occur, especially when diagnosis and treatment are not timely. Patients that develop necrotizing colitis have a mortality rate of 40% and for those with concomitant liver abscess mortality increase to 89%.[7] These patients appear toxic, with fever, bloody diarrhoea, and signs of peritoneal irritation. The development of toxic megacolon has been linked to corticosteroid use and is unresponsive to ant-amoebic therapy, requiring immediate surgical intervention. Exclusion of inflammatory bowel disease is exceptionally important, given that misdiagnosis and treatment with corticosteroids can lead to these serious complications. Infectious causes that need to be excluded include shigella, salmonella, campylobacter, and enteroinvasive and enterohemorrhagic Escherichia coli. Non-infectious causes include inflammatory bowel disease, intestinal tuberculosis, diverticulitis, and ischemic colitis.[8]

The formation of an ameboma is another uncommon manifestation that may occur in amebic colitis. It tends to present with pain and swelling in the right iliac fossa, or with symptoms of bowel obstruction. Macroscopically, amebomas resemble a mass (or multiple masses) typically localized in the cecum or ascending colon and consist of localized hyperplastic granulation tissue. Ameboma formation has been generally associated with untreated or partially treated amoebic colitis. Given that its appearance can resemble lymphoma, neoplasm, tuberculosis, abscess, or inflammatory bowel disease, colonoscopy and histopathological examination of the biopsied material are warranted to exclude other sinister lesions.[9] Intestinal tuberculosis and inflammatory bowel disease can present similarly to amoebic colitis. Endoscopic features such as cryptitis and crypt abscesses, as well as erosions and ulceration of the rectum, which are commonly seen in ulcerative colitis, may also be present in amebic colitis. The mucosa between the amebic ulcers may appear normal, mimicking Crohn's disease and intestinal tuberculosis. Because the cecum is the most commonly affected area in amoebiasis, normal terminal ileum, transverse and descending colon can help distinguish it from IBD and infectious colitis.[10] Combining serological testing with PCR or antigen detection is currently the best diagnostic approach. Using combined techniques will increase the specificity and sensitivity in the diagnosis of E. histolytica infection. Further, this method allows clinicians to distinguish acute infection from chronic or previously treated infection.

Conclusion

To conclude, Amoebiasis continues to be a large health issue in developing countries, particularly in children. With increased travel and emigration to developed countries from endemic areas, the incidence and prevalence of amoebiasis continue to increase. Since most patients are asymptomatic, diagnosis and treatment can be challenging for clinicians, potentially leading to the continuous spread of the disease. E. histolytica should be considered as a differential diagnosis of colitis and with certain extra-intestinal manifestations, particularly when certain demographics (i.e., gender, race, travel history) are present.

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Conflict of Interest: None

References

1. R. Haque, C.D. Huston, M. Hughes, E. Houpt, and W. A. Petri, "Amebiasis," The New England Journal of Medicine. 2003;348;(16):1565-1573.
2. A. Cheepsattayakorn and R. Cheepsattayakorn, "Parasitic pneumonia and lung involvement," Bio Med Research International. 2014.
3. "United States Centers for Disease Control and Prevention. Amebiasis-biology," http://www.cdc.gov/parasites/amebiasis/biology.html.
4. T. E. Bercu, W. Petri, and J. W Behm, "Amebic colitis new insights into pathogenesis and treatment," Current Gastroenterology Reports. 2007;429-433.
5. C. Ximénez, R. Cerritos, L. Rojas, "Human amebiasis breaking the paradigm?" International Journal of Environmental Research and Public Health. 2010;7(3):1105-1120.
6. I. Salit, K. Khairnar, K. Gough, and D. Pillai, "A Possible Cluster of Sexually Transmitted," Clinical Infectious Diseases. 2009;49(3):346-353.
7. V. Prakash and SS. Bhimji, "Amebic Liver. StatPearls Publishing," 2017. https://www.ncbi.nlm.nih.gov/books/NBK430832/.
8. G. Maltz and C. M. Knauer, "Amebic Liver Abscess A 15-Year Experience," American Journal of Gastroenterology. 1991;86(6):704-710.
9. T. Wuerz, JB. Kane AK. Boggild, and "A Review of Amoebic Liver Abscess for Clinicians in a Nonendemic Setting," Canadian Journal of Gastroenterology & Hepatology. 2012;26(10):852835:729-733.
10. J. Gunther, S Shafir, B Bristow, and F Sorvillo, "Short report. Amebiasis-related mortality among United States residents. 1990-2007," The American Journal of Tropical Medicine and Hygiene. 2011;85(6):1038-1040.