

The Slipshod Stooze-Amoebic Colitis

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EDITORIAL

Amoebic colitis occurs due to infection of gastrointestinal tract with protozoan parasite *Entamoeba histolytica*. The invasive amoebic trophozoites propagate through simple binary fission. Susceptibility to invasive amoebic infection is contingent to immune status of host. Morphological similarity of *Entamoeba histolytica* to *Entamoeba dispar*, *Entamoeba moshkovskii*, *Entamoeba bangladeshi* or *Entamoeba nuttalli* is encountered.

Additionally designated as amoebiasis, amoebic trophozoites burrow through superimposed mucosa and invade submucosal tissue. Generally, implicated soft tissue is devoid of amalgamation of amoebic cysts [1,2]. Typically, amoebic infection engenders flask shaped ulcers. Exceptionally, intestinal perforation or an inflammatory mass as an amoeboma may arise [1,2]. Amoebic colitis can be specifically diagnosed with discernible parasitic trophozoites invading intestinal mucosa [1,2].

Commonly encountered in tropical or subtropical regions, no geographical zone of infection with *Entamoeba histolytica* is exempt. Immigrants to developed countries, travellers, institutionalized subjects and men with sexual contact with men (MSM) are frequently infected. Incriminated males may recurrently exhibit disseminated infection. Amoebic colitis implicates colonic segments of large intestine as cecum, right colon, rectum, sigmoid or appendix. Cutaneous lesions may ensue. Terminal ileum is devoid of infiltration with parasitic trophozoites. Haematogenous dissemination commonly occurs into brain, hepatic or pulmonary parenchyma. Recto-vesical fistula or cutaneous fistula may be configured [1,2]. Invasive infection of large intestine with *Entamoeba histolytica* is predominantly transmitted with ingestion of parasitic cysts through faecal contamination of food or water, thereby inducing faecal - oral route. Besides, sexual contact or oral-anal route may be articulated. Parasitic trophozoites may disseminate into hepatic parenchyma or associated viscera [1,2]. Parasitic cysts are resistant to digestion by gastric acid or chlorinated water. Ex-cystation may occur within small intestine with consequent trophozoite dissemination and invasion of gastrointestinal tract. Discernible genetic polymorphisms within leptin receptor may alter host susceptibility to invasion with *Entamoeba histolytica* [1,2]. Factors contributing to appearance of severe infection with *Entamoeba*

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histolytica as malnutrition, malignant neoplasms, alcoholism, young or immunocompromised individuals or pregnancy may be encountered. Cellular adhesion and demise is induced by trophozoites of *Entamoeba histolytica* [1,2]. Gal or GalNAc lectin situated upon surface of parasitic trophozoites adheres with galactose or N-acetylc-D-galactosamine residue components of O linked sugar side chains encountered within host colonic mucin. Aforesaid adherence degrades protective mucus intestinal barrier with epithelial penetration of parasitic trophozoites. Parasitic secretion of proteinases with activation of mechanisms as contact dependent cellular lysis, apoptosis and configuration of amoeba-pores initiate demise of host cell. Trophozoites of *Entamoeba histolytica* ingest remnant erythrocytes, a phenomenon designated as haemophagocytosis [1,2]. Few trophozoites appear encysted on account of signalling pathways, thus concluding propagative parasitic cycle [1,2].

Majority (90%) of individuals demonstrating *Entamoeba histolytica* infection appear asymptomatic. Clinical symptoms as mild diarrhoea, severe dysentery, pyrexia, abdominal pain or tenesmus may ensue. Alternatively, emergence of cogent clinical symptoms may be delayed [1,2]. With acute infection, symptoms pertaining to acute abdomen may ensue. Incriminated children may exhibit intussusception or necrotizing colitis with consequent perforation of gastrointestinal tract [1,2]. Subacute colonic infection may emerge within 4 weeks and manifests as worsening diarrhoea with abdominal pain [1,2]. Exceptionally, amoebic colitis is accompanied by complications such as toxic megacolon, fulminant necrotizing colitis, colonic amoeboma or perianal fistula. Fulminant necrotizing colitis is associated with significant mortality [1,2].

Grossly, clearly defined, discrete mucosal ulcers or erosions with white, yellow or haemorrhagic superficial exudate and a smooth, irregular outline are observed. Intervening mucosa is unremarkable. Mucosal ulcers may coalesce with emergence of enlarged lesions with centric necrosis. Adjacent foci of colitis or inflammatory polyps may be discerned [1,2]. Amoeboma or amoebic pseudo-tumour is generally confined to the cecum or ascending colon and represents as a firm,

well defined, annular, inflammatory thickening of colonic wall with frequent occurrence of napkin-ring deformity [1,2].

Upon microscopy, superficial mucosal necrosis appears to progress towards mucosal erosion and ulceration with superimposed inflammatory exudate comprised of necrotic material, fibrin and inflammatory cells admixed with parasitic trophozoites of *Entamoeba histolytica*. Amoebic trophozoites appear amalgamated within viable and necrotic tissue perimeter [1,2]. Mucosal ulcers undermine superficial mucosa and configure an ulcer crater incorporated with cellular debris, fibrin and parasitic trophozoites. Alternatively, mucosal ulcers enunciate aggregates of parasitic trophozoites abutting the submucosa [1,2]. Foci of chronic cryptitis or architectural distortion of crypts may occur. Preliminary ulcers exhibit minimal inflammation with subsequent circumscription by neutrophils, histiocytes, lymphocytes, plasma cells and occasional eosinophils. Mature mucosal ulcers appear as flask shaped lesions with a broad base and tapering apex [1,2]. Parasitic trophozoites appear as spherical to elliptical organisms imbued with abundant, vacuolated, pink-purple cytoplasm with projectile pseudopods, miniature nuclei, prominent, condensed, peripheral chromatin, miniature, centric, dot-like karyosome, circumscribing halo induced due to fixation or parasitic toxins and magnitude varying from 10µmetres to 60µmeters, simulating macrophages [1,2]. Ingestion of red blood cells by parasitic trophozoites is pathognomonic of infection with *Entamoeba histolytica* [1,2]. Spherical parasitic cysts demonstrate a magnitude of 10µmetres to 20µmeters. Mature cysts exhibit four nuclei along with chromatoid bodies and blunt, rounded edges [1,2]. Amoeboma is configured of granulation tissue admixed with chronic inflammatory cells and fibrotic tissue [1,2].

Periodic acid Schiff's stain expounds trophozoites of *Entamoeba histolytica* as deep pink or magenta. Immunohistochemistry pertaining to the parasite is absent [3,4]. Parasitic trophozoites are immune non-reactive to CD68 whereas macrophages appear immune reactive [3,4] (Figure 1 & 2).

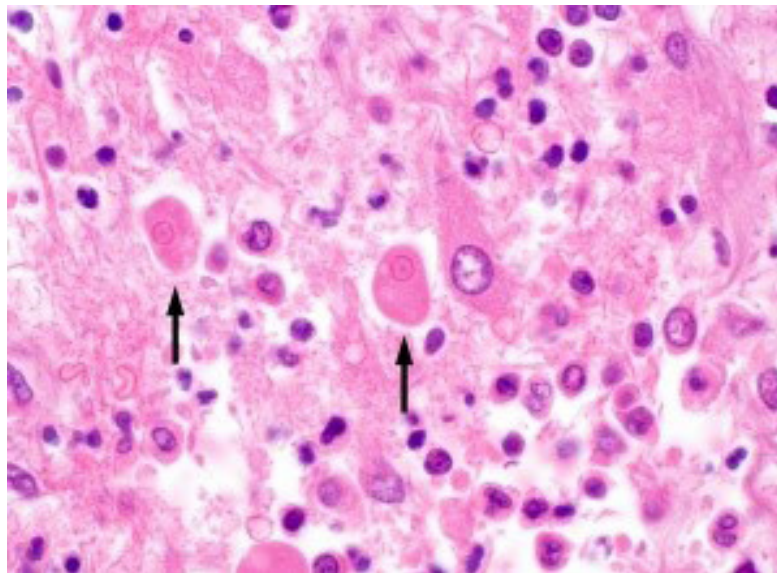


Figure 1. *Entamoeba histolytica* trophozoite depicting abundant, vacuolated, pink-purple cytoplasm, centric nucleus with prominent karyosome and a paucity of inflammatory exudate [5].

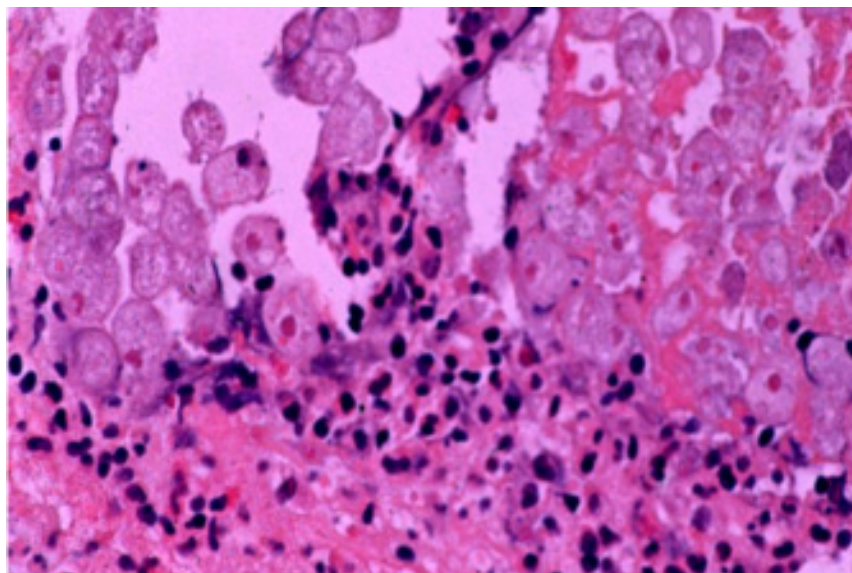


Figure 2. Proliferating parasites of *Entamoeba histolytica* with abundant, vacuolated cytoplasm, uniform nucleus with peripheral chromatin, centric karyosome and ingested red cells [6].

Amoebic colitis requires segregation from conditions such as appendicitis, Crohn's disease, ulcerative colitis, aggregated tissue macrophages, non-pathogenic amoeba, infection with bacterial pathogens as *Shigella*, *Escherichia coli*, *Salmonella*, *Campylobacter* or *Clostridioides difficile*, pseudomembranous colitis, pyogenic hepatic abscess, tuberculosis of gastrointestinal tract or ischaemic bowel disease [3,4]. Pertinent categorization of infection with *Entamoeba histolytica* is contingent to diverse epidemiological factors, cogent clinical symptoms and features discerned upon plain radiography or colonoscopy.

Amoebic liver abscess generates specific anti-amoebic antibodies in incriminated individuals [3,4]. Amoebic hepatic cyst is devoid of bacterial microorganisms. Upon aspiration, isolation of parasites is uncommon [3,4]. *Entamoeba histolytica* infection can be appropriately detected with microscopic examination of faecal matter wherein ova and parasitic trophozoites may occur. Nevertheless, majority of subjects are asymptomatic and segregation amidst diverse *Entamoeba* species may be challenging. Besides, stool antigen or stool nucleic acid can be evaluated [3,4]. Antigen detection or sensitive nucleic acid amplification

tests (NAATs) may demarcate *Entamoeba histolytica* from morphologically identical amoebic species. Serological evaluation of antibodies to *Entamoeba histolytica* is optimal and beneficially adopted although segregation of current from preceding infection is challenging [3,4]. Upon computerized tomography (CT), amoebic colitis is denominated as •acute colitis demonstrating a nonspecific, diffuse thickening of intestinal wall with submucosal oedema. Contrast administered CT exhibits a stratified or target pattern [3,4]. •chronic colitis delineates prominent, thickened intestinal wall. Segregation from malignant metamorphosis of the colon may be challenging. Incrimination of cecum and rectum with sparing of terminal ileum is frequently discerned [3,4]. Plain radiographs or barium enema may demonstrate foci of mucosal ulceration. •amoeboma enunciates localized thickening of intestinal wall with minimal contrast enhancement upon CT [3,4]. •fulminant necrotizing colitis exemplifies a prominent, thickened intestinal wall with an attenuated, enhancing rim upon contrast enhanced CT. Focal mucosal ulceration may occur as contiguous or skip lesions. Ultrasonography and computerized tomography is optimally adopted for discerning amoebic liver abscess [3,4]. Amoebic colitis is appropriately managed with amoebicidal or luminal anti-parasitic agents. Amoebicides as metronidazole or tinidazole can be employed to eradicate invading parasitic trophozoites[3,4]. Paromomycin is beneficial in exterminating luminal cysts and circumventing disease relapse or parasitic transmission to companions. Parasitic infection associated with complications may necessitate surgical excision of perforated or necrotic segment of gastrointestinal tract. Additionally, resuscitation with intravenous fluids and ingestion of broad spectrum antimicrobials is recommended in order to manage peritonitis [3,4].

CONCLUSION

Amoebic colitis occurs due to infection of gastrointestinal tract with protozoan parasite *Entamoeba histolytica*. Factors contributing to severe infection with *Entamoeba histolytica* emerge as malnutrition, malignant neoplasms, alcoholism, young or immunocompromised individuals or pregnancy. Amoebic colitis implicates colonic segments of large intestine as cecum, right colon, rectum, sigmoid or appendix.

Typically, amoebic infection engenders flask shaped ulcers whereas intestinal perforation or an inflammatory mass as an amoeboma may ensue occasionally. Parasitic trophozoites appear admixed within inflammatory exudate confined to focal mucosal ulceration as spherical to elliptical organisms imbued with abundant, vacuolated, pink-purple cytoplasm with projectile pseudopods, miniature nuclei, prominent, condensed, peripheral chromatin, miniature, centric, dot-like karyosome and a circumscribing halo, staining deep pink or magenta upon periodic acid Schiff's stain. Amoebic colitis requires segregation from conditions such as appendicitis, Crohn's disease, ulcerative colitis, aggregated tissue macrophages, non-pathogenic amoeba, infection with bacterial pathogens as *Shigella*, *Escherichia coli*, *Salmonella*, *Campylobacter* or *Clostridioides difficile*, pseudomembranous colitis, pyogenic hepatic abscess, tuberculosis of gastrointestinal tract or ischaemic bowel disease. Antigen detection or sensitive nucleic acid amplification tests (NAATs) are optimal in demarcating *Entamoeba histolytica* from morphologically identical amoebic species. Amoebic colitis is appropriately managed with amoebicidal or luminal anti-parasitic agents.

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5. Image 1 Courtesy: Pathology outlines.
6. Image 2 Courtesy: PLOS.