An Overview of Intestinal Amoebiasis

C.E Omuemu *MBBS, FWACP, MPH*
Consultant Gastroenterologist, Senior Lecturer

R. Ugiagbe *MBBS*
Senior Registrar
Department of Medicine
University of Benin Teaching Hospital, Benin City, Edo State

ABSTRACT

Intestinal amoebiasis describes infestation by Entamoeba spp restricted to the large intestine. It is a common disease thriving in areas of poor hygiene. Most cases present as self limiting diarrhea or dysentery but a small proportion present with severe disease and complications. Treatment is relatively simple in uncomplicated cases but the emphasis must be on preventive measures (improved hygiene) rather than treatment.

INTRODUCTION

Several enteric amoebae dwell in the lower gastrointestinal tract (GIT) but fortunately, most are non-pathogenic. Amoebiasis refers to infection with the protozoan *Entamoeba histolytica.*[1] About 90% of infections are asymptomatic while 10% produce a spectrum of clinical syndromes affecting the GIT and other organ systems..[1] Infection is restricted to the gastrointestinal tract is termed intestinal amoebiasis.

EPIDEMIOLOGY

About 10% of the world's population is infected with Entamoeba species, the majority with the non invasive Entamoeba dispar. The prevalence of Entamoeba infection ranges from 1-40% of the population in Central and South America, Africa and Asia, and from 0.2-10.8% in endemic areas of developed countries such as the USA. [2,3,4,5]

Infection with E. histolytica is a common cause of acute diarrhoea in developing countries, accounting for up to 38% of cases. [6]

LIFE CYCLE

The average incubation period is two to six weeks but patients may present months to years after the initial infection. Humans are often asymptomatic carriers. Infectious cysts are shed in the stool and can survive for several weeks in a moist environment. Ingested cysts release motile trophozoites in the small intestine and these usually remain as harmless commensals in the large bowel but may invade the bowel mucosa causing colitis. From there they may also enter the portal venous system leading to abscesses of the liver and rarely the lungs and other organs. The trophozoite may not encyst in patients with active dysentery.[1]

Amoebiasis is usually transmitted by the faeco – oral route aided by poor personal hygiene and contaminated food or water. Spread through contaminated instruments introduced into the GIT has been suspected particularly colonic irrigation devices. [11] Other risk factors include communal living, oral and anal sex, compromised immune system and migration or travel from endemic areas.

PATHOGENESIS

The infectious form is *E. histolytica* cyst which has four nuclei. Ingested cysts colonize the surface of colonic mucin epithelial cells. Cysts release trophozoites which reproduce under anaerobic conditions without harming the host.

Dysentery occurs when amoeba attach to colonic epithelium as they cause epithelial cell apoptosis, invade the crypts of colonic glands and burrow into the lamina propria. The organisms subsequently burrow laterally to create a flask shaped ulcer with a narrow neck and broad base. [10]

Amoebic proteins involved in tissue invasion include cysteine proteinases which break down proteins of extracellular matrix, lectin on parasite surface that binds to carbohydrate on surface of colonic epithelial cells and red blood cells, as well as channel-forming protein called amoebapore which makes holes in plasma membrane of host cells and lyses them.[10]

PATHOGENESIS

Amoebiasis most frequently involves the caecum and ascending colon, followed by sigmoid, rectum and appendix. The entire colon is involved in severe full-blown cases. Amoebae usually mimic the appearance of macrophages due to their comparable size and large number of vacuoles but the parasites have a smaller nucleus which contains a large karyosome. [10]

CLINICAL FEATURES

Most infections are asymptomatic.[1] Symptomatic amoebic colitis presents with diffuse lower abdominal pain, mild diarrhoea, malaise and occasionally weight loss. Caecal involvement may mimic acute appendicitis. In severe cases, stools may be passed 10-12 times daily with little faecal material consisting mainly of blood and mucus. Less than 40% of patients with amoebic dysentery are febrile. Chronic forms of amoebic colitis may mimic inflammatory bowel disease. [1,7,9]

LABORATORY DIAGNOSIS

Diagnosis is confirmed by microscopic examination of fresh stool for haematophagous trophozoites of *E.histolytica*. Since trophozoites are killed rapidly by water, drying and barium it is important to examine three fresh stool specimens before excluding the diagnosis.[1,9] Cysts may be found in suitably preserved faecal specimens. Culture of *E. histolytica* is not routinely available. Sigmoidoscopy with biopsy of the edge of ulcers or biopsy of colonic masses may also yield trophozoites. [1,7]

Serology using ELISA techniques or agar gel diffusion assays is important in differentiating *E.histolytica* from non-pathogenic *E.dispar*.

Imaging techniques (barium enema, abdominal ultrasound and computerized tomography) are of limited value. Amoebomas are usually identified as a mass by barium enema, but biopsy is necessary for differentiation from carcinomas and other mass lesions.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of intestinal amoebiasis include bacterial diarrhoeas caused by Campylobacter, enteroinvasive *E. coli* and *Shigella*, *Salmonella* and *Vibrio* species.

The typical patient with amoebic colitis has less fever, haem positive stools with few neutrophils, correct diagnosis however requires bacterial cultures, microscopic examination of stools, and amoebic serologic testing.

TREATMENT

Drugs are classified according to their primary site of action into luminal and tissue amoebicides. Tissue amoebicides are used in the treatment of amoebic colitis and invasive amoebiasis. Examples include metronidazole, tinidazole and ornidazole.[1,9] Luminal agents are useful for the eradication of cysts in patients with colitis or a liver abscess as well as treatment of asymptomatic carriers. Examples are iodoquinol, paramomycin and diloxonide furoate. Emetine and chloroquine are rarely used due to side effects. [1, 7]

COMPLICATIONS

Intestinal amoebiasis and its complications have a wide variety of presentations including noninvasive intestinal colonization, acute amoebic proctocolitis, chronic nondysenteric intestinal amoebiasis, amoeboma, toxic megacolon, amoebic peritonitis and amoebic strictures. Intestinal amoebiasis may be complicated by granuloma of the large intestine,colonic perforation and haemorrhage, perianal ulceration, and dissemination via the blood stream leading to extra intestinal amoebiasis most commonly abscess formation in the liver and sometimes in the brains or lungs. Toxic megacolon usually occurs in children. [9]

PROGNOSIS

Fulminant amoebic colitis is reported to have 55-88% mortality. Between 40,000-100,000 people with amoebiasis will die each year, placing this infection next to malaria and schistosomiasis in mortality caused by protozoan parasites. [9,12]

PREVENTION

Health education with emphasis on personal hygiene, proper cleaning of uncooked fruits and vegetables, discouraging use of human faeces as manure, protection of water supplies from faecal contamination and screening of food handlers is needed. There is need for prompt treatment of patients and screening of household members/institutional contacts and epidemiologic investigation aimed at identification of clusters of cases. [7 ,9]

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