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Case report: Non-ulcerative amebiasis of rectum¹

Short communication

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Studies of man and animals indicate that lysis of intestinal epithelium precedes movement of *Entamoeba histolytica* into the lamina propria (Prathap and Gilman, 1970; Pittman et al., 1973; Takeuchi and Phillips, 1975; Takeuchi et al., 1977; Chadee and Meerovitch, 1985). Here we describe trophozoites of *E. histolytica* in rectal tissue without detectable lytic or other injury to any of the tissues, including intestinal epithelium, lamina propria, and muscularis mucosae.

A 69-year-old black Zairian woman living in the Ubangi territory was imprisoned for sorcery and died suddenly after 2 days of punitive starvation. Autopsy at Karawa Mission Hospital revealed no significant gross lesions. Microscopically, there was a myocarditis of undetermined cause, but this was mild and not thought to be the cause of death. Sections of rectum showed minimal autolysis characterized by separation of epithelial cells from the basement membrane. Trophozoites of *E. histolytica* were in most sections of the rectum studied. They measured 20 μ m in maximum diameter and contained a nucleus with a prominent central karyosome. Their cytoplasm was amphophilic, granular, and sometimes vacuolated (Fig. 1). One trophozoite contained a red blood cell. The trophozoites occurred singly and in clusters of up to 4 or

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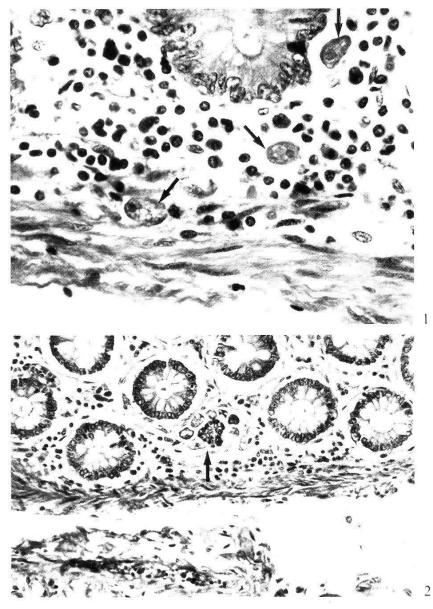


Fig. 1. Three trophozoites (arrows) of *E. histolytica* in lamina propria and muscularis mucosa of rectum. One lies between epithelium and basement membrane and appears to be constricting the adjacent epithelial cells. Hematoxylin and eosin, \times 415, AFIP MIS # 85-7403.

Fig. 2. A cluster of 7 *E. histolytica* trophozoites in the space between the basement membrane and epithelium. Adjacent tissue is normal or possibly slightly inflamed, but the inflammation cannot be related to the invading trophozoites. Hematoxylin and cosin, $\times 165$, AFIP MIS # 85-7398.

more (Fig. 2). They were in the lamina propria and muscularis mucosae (Fig. 1). Many were in the space between the epithelium and basement membrane (Figs. 1 and 2). The characteristic lytic changes of colonic amebiasis were not present. Elsewhere in the rectum there was a slight diffuse infiltrate of the lamina propria by plasma cells, lymphocytes, and eosinophils, but these were no more than usually is seen in colons of Africans, and none of the inflammatory cells was concentrated around or near the invading trophozoites. This mild inflammation therefore was not a response to the invading trophozoites. Additional sections, taken routinely, of colon, ileum, liver, lung, and pericardium revealed no trophozoites or necrotizing lesions that suggested amebiasis.

The source of the trophozoites remains unclear. It seems likely, however, that they migrated to and invaded the rectum – either from the lumen of the large intestine where they had not caused lesions or from inapparent lesions in the colon that we did not detect. But in the rectum there is invasion of intestinal mucosa and lamina propria without lytic activity and without inflammation and ulceration. We think this is because the trophozoites penetrated the rectal mucosa post mortem. If so, the absence of lysis and necrosis could be explained by the lack of host response. Takeuchi and Phillips (1975) suggested that lysis of host tissue may be a consequence of infiltrating polymorphonuclear leukocytes. They showed experimentally that invading trophozoites penetrate epithelial tissue between cell junctions and that necrosis followed contact between amebae and infiltrating neutrophils. Direct contact between trophozoites and neutrophils resulted in immediate lysis of the neutrophils with spillage of hydrolytic enzymes. The invasion of trophozoites in our present case appears to support the view that lysis requires a reaction by the host. This case also reveals that trophozoites of E. histolytica are capable of non-lytic penetration of intestinal epithelium. In summary, we believe the absence of necrosis in the rectum of this corpse is a consequence of the absence of a neutrophilic response.

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