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Blastocystis hominis and travelers

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Summary *B. hominis* is a unicellular protozoan commonly identified in stool specimens of travelers who have returned from tropical countries. It has a worldwide distribution, and infection is more common in developing countries compared to industrialized nations. Clinical features of illness which have been attributed to *Blastocystis* include nausea, anorexia, abdominal pain, flatulence and acute or chronic diarrhea. The preferred method of diagnosis is a permanently stained smear of an unconcentrated stool specimen. The presence of *B. hominis* in stool specimens of symptomatic travelers should prompt clinicians to search for other unrecognized co-pathogens. Due to controversy regarding the pathogenicity of *B. hominis* in humans, clinicians are often faced with the dilemma of whether or not they should offer treatment for *B. hominis* infection in returned travelers. The most commonly used drugs for treatment include metronidazole and trimethoprim–sulfamethoxazole (TMP–SMX), when treatment is deemed necessary. Prevention in travelers should focus on food and water precautions as the organism is transmitted by the fecal–oral route.

Introduction

Blastocystis hominis is a unicellular protozoan and one of the most common parasites found in the human intestinal tract. It was first described in the medical literature in 1911 by Alexeieff and was considered a harmless yeast at that time.[1] However, electron microscopic studies more than 50 years later in 1967 by Zierdt led to reclassification of *B. hominis* amongst protozoa.[2] Infection with *B. hominis* has a worldwide distribution and occurs in both children and adults. The proposed mode of transmission of *B. hominis* is by the fecal-oral route.[3] Travelers appear to be at increased risk of infection during travel to tropical and under-developed countries.[4-6] In this review, we explore some of the controversial issues regarding the pathogenicity of *B. hominis* in humans and suggest measures to minimize the risk of infection in travelers during trips to developing countries.

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Microbiology

B. hominis is an obligate anaerobic protozoan. In humans, it resides in the colon and cecum. Colonization has also been described in animals including pigs, monkeys, poultry and rodents. *B. hominis* does not have a cell wall and therefore there is great variation in its size and shape, ranging from 5 to 40 μm. Intracellular organelles include Golgi apparatus, mitochondria and smooth and rough endoplasmic reticulum.[7] Its mitochondria are uniquely anaerobic and have no cytochrome protein or oxidative mitochondrial enzymes.[8] *B. hominis* grows only in anaerobic conditions in culture media.

B. hominis reproduces asexually, most likely by binary fission.[8] Schizogony occurs in cultured cells and 2–30 progeny are derived from each schizont. Multiple forms of *B. hominis* have been described in culture including amoeboid, vacuolar, granular and a recently described multilayered, thick walled-cystic form (3–10 μm). The ameboid stage is often observed in old cultures and after antibiotic administration.[2] Fecal cysts are most likely responsible for external transmission as cysts can survive in water at room temperature up to 19 days.[9] However, cysts are highly susceptible to changes in the environment and disrupt at extremes of temperature and by the use of common disinfectants.[9] The amoeboid form is more likely associated with disease.[10]

Epidemiology

A higher prevalence of *Blastocystis* infection, in healthy asymptomatic adults, has been reported from developing countries (30–50%), compared with developed parts of the world (1.5–10%).[4-6,8,11,12] *B. hominis* is identified in up to15% of stool specimens submitted for routine examination in United States.[13] Reported risk factors for acquiring *Blastocystis* include travel to tropical areas,[4] consumption of untreated water, handling animals, abnormal gastrointestinal (GI) tract function, diabetes mellitus and immunosuppression secondary to malignancy, chemotherapy and HIV.

B. hominis is frequently reported in travelers returning from developing countries. In a study from Nepal, the prevalence of *B. hominis* amongst expatriates and tourists was up to 30%.[6] Similarly, journey to the Indian-subcontinent was associated with acquisition of *Blastocystis hominis* amongst German travelers in another study.[5] *B. hominis* has been reported as the most frequently identified stool parasite among foreign workers from Southeast Asia in Taiwan since 1992.[12] The increased prevalence in developing countries is likely secondary to overcrowding, poor personal and environmental hygiene, lack of safe water supply and poor sewerage and waste removal services. Working closely with animals may also be a risk factor for acquisition of *B. hominis*. Even in developing countries, infection appears to be more common in groups of lower socio-economic status. Outbreaks of gastroenteritis with *B. hominis* have been described in families.[14] An increased prevalence of *B. hominis* has also been reported amongst Peace-Corps volunteers stationed in developing countries for prolonged periods of time.[15]

An association between *B. hominis* infection and irritable bowel syndrome (IBS) is well described in the literature. Patients with IBS have a propensity to get colonized with *B. hominis* and have higher burden of organisms (five or more per high-power field) compared to a control population.[16] Patients with IBS are more likely to have elevated levels of the IgG2 sub-class of antibodies directed against *B. hominis* than do asymptomatic controls, suggesting a higher rate of prior infection or colonization in this group.[17] Patients colonized with *B. hominis* also have higher rate of co-infection with *Helicobacter pylori*,[18] likely secondary to a common route (fecal-oral) of transmission.

Pathogenesis

The pathogenic role of *B. hominis* in humans has been a subject of much controversy to date. Its pathogenic role in animals, however, has been demonstrated in some experimental studies. In a murine model,[19] infection of immunocompetent mice with fecal cysts of *B. hominis* caused weight loss and lethargy and histological examination of the cecum and colon showed intense inflammation with edematous lamina propria and mucosal sloughing. No organisms were seen invading the colonic wall and infection was self-limiting. However, in a similar study of gnotobiotic guinea pigs infected with *B. hominis*, invasion of superficial layers of colon has been demonstrated. Self-limiting myonecrosis after intra-muscular injection of *B. hominis* was observed in yet another experimental study in mice.[20]

In spite of the above-mentioned experimental animal data, the pathogenic role of *B. hominis* in humans, if any, is far from settled. Arguments that are presented to support a pathogenic role of *B. hominis* in humans are mostly based on