Letters to the Editor

Association of Blastocystis hominis with Human Disease

Sheehan et al. (3) and Pikula (Z. P. Pikula, Letter, J. Clin. Microbiol. 25:1581, 1987) bear witness to the fact that Blastocystis hominis is a common inhabitant of the human intestinal tract. These authors, like those they cite, have found B. hominis in some persons in whom they identified none of the commonly recognized pathogens, and in others in association with recognized pathogenic protozoa or bacteria. Surveys cite Blastocystis prevalence rates in the 10 to 15% range.

To support "the emerging concept of B. hominis... causative of human disease" (3), one must first rule out the presence of other known pathogens or disease states. This has not been done. The authors of both of the reports referred to above apparently based their conclusions on the results of examinations of single stool specimens. It has long been known that the examination of a single stool sample reveals only a fraction of the Entamoeba histolytica or other protozoan infections. Sawitz and Faust (2) found this figure to be approximately 40% for E. histolytica and that a series of six stool examinations was needed to detect some 95% of infections. Modern techniques may have altered these figures (there are no comparable recent studies), but it is impossible to draw conclusions based on the examination of one or two specimens.

We monitored a series of 32 patients found to have B. hominis infections over a period of at least 30 months (1). All had six or more stool examinations performed, and 27 patients were found to have one or more recognized pathogens—E. histolytica, Giardia lambia, or Dientamoeba fragilis—often undetected in the initial stool examinations. All became and remained asymptomatic after appropriate therapy, while retaining B. hominis. The other five patients fulfilled the criteria for irritable bowel syndrome, and B. hominis was considered an incidental finding.

It is interesting that of the 23 patients with five or more B. hominis cells per 40× microscopic field tabulated by Sheehan et al. (3) the 4 who had stool examinations performed as part of a routine employee screen were all asymptomatic, whereas most of the others had histories suggestive of possible parasitic infections, inadequately investigated with a single stool examination. Furthermore, the figure of "five or more (B. hominis) per 40× field" used by these authors and others as an index of pathogenicity suggests an unparalleled situation in which an organism becomes pathogenic only when its numbers as measured in the stool specimen reach a critical threshold. Is it not at least equally possible that increased numbers of B. hominis may at times result from disturbance of gastrointestinal function caused by some other infection?

Repeated stool examinations are time-consuming and costly, but without them any conclusions as to the pathogenicity of B. hominis cannot be considered valid.

LITERATURE CITED


Authors' Replies

Markell and Udell's criticism of our report, "Association of Blastocystis hominis with Signs and Symptoms of Human Disease" (9), focused on methodology and conclusions. Purged stool specimens are a widely used and superior modality for the diagnosis of intestinal protozoa (1, 5). In a study conducted by Andrews (2), it was reported that "six examinations of normal stools detected 75% percent of all the protozoan species found in the normal and purged stools; the single purged specimen showed 88.9%, adding nine species which had not been previously found."

It is interesting that in their exhaustive study of 32 patients with B. hominis infection, Markell and Udell (7) failed to detect any "recognized" pathogen in 16% of their study group. All except one in this group presented with diarrhea of long duration. Markell and Udell classified these subjects as having irritable bowel syndrome and considered the presence of B. hominis incidental. However, Vannatta et al. (10), Ricci et al. (8), LeBar et al. (6), and Cohen (4) all reported the successful treatment of B. hominis, with drugs other than iodoquinol, which resulted not only in the eradication of B. hominis infection but concurrent loss of symptoms as well. Curiously, Markell and Udell have deduced that the successful treatment of B. hominis in the above-mentioned published case reports "represents the elimination of B. hominis but of some undetected pathogen..." (7).

Our experience, as well as that of other investigators (3), suggests that B. hominis may be responsible for a clinical syndrome consisting of diarrhea, flatus, abdominal discomfort, and anorexia. Whether the syndrome is due to B. hominis itself or to the interaction of B. hominis with other organisms has yet to be resolved.

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A detailed description of procedures used in our laboratory was not given in my letter (Z. P. Pikula, Letter, J. Clin. Microbiol. 25:1581, 1987) because I wanted to emphasize two other points: the frequency of Blastocystis hominis in our material and a significant association of Entamoeba histolytica and B. hominis. A similar observation was given by Sheehan et al. (5).

My results were based on at least three fecal examinations. It is recommended that three fecal examinations be performed before a patient is declared negative for E. histolytica and other amoebas (1). Of course, if indicated, more than three examinations must be done.

If it is not contraindicated, I examine purged stools. The first portion of the stool is examined for the presence of eggs of intestinal helminths and cysts of intestinal protozoa by saline smears, iodine-stained smears, and a flotation technique (2). The liquid portion of the stool with tissue detritus and mucus is examined for the presence of intestinal parasites, especially E. histolytica and other amoebas, by using standard unstained wet mounts and smears stained by Quin- sel stain. Cultivation, now routine, was not done for all patients in 1986. If indicated, other procedures for intestinal parasites are used (examination of duodenal juice, the Baerman technique for larvae of Strongyloides stercoralis, etc.).

Six of our patients had B. hominis in large numbers and without other pathogens. All clinical and other examinations were performed. Three of the patients had noninfective diseases of the bowel (carcinoma coli, irritable bowel syndrome, or diverticulosis). Three others had no signs of any other diseases. Based on data given by Ziertd (C. H. Ziertd, Clin. Microbiol. Newsl. 5:57-59, 1983), LeBar et al. (3), Ricci et al. (4), and Vannatta et al. (6) about successful treatment of patients with metronidazole, this drug was introduced (500 mg three times a day for 10 days). After treatment, the symptoms resolved and B. hominis disappeared from the stools (S. Telabaslic, Z. P. Pikula, and A. Drnda, unpublished data).

The figure of five or more B. hominis cells per 40× field is just an aid in routine work; like many other criteria in the laboratory, it is not absolute. If B. hominis is found without other pathogens and without signs of some noninfective disease of the intestine, it may be considered a potential pathogen. This problem will be solved after further clinical, parasitological, and immunological investigations.

LITERATURE CITED


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Disk Susceptibility Testing of Penicillin-Resistant Pneumococci

In their article "Screening Pneumococci for Penicillin Resistance," Swenson et al. (2) recommend "that the oxacillin screen test be used to detect penicillin resistance in pneumococci" and "that the methicillin screen test not be used." They based this recommendation on the observation that 3 of 34 relatively resistant strains were falsely characterized as susceptible by using methicillin disks and the breakpoint for resistance of >25 mm recommended by Jacobs et al. (1). Oxacillin tested under the same ambient air conditions with Mueller-Hinton sheep blood agar showed no false susceptibility (0 of 34 strains). The difference between these observations is not significant (χ² = 1.39, chi-square test, Yates modification). The 5-µg methicillin disk tested on Mueller-Hinton sheep blood agar by Jacobs et al. (1) showed 2 of 29 strains to be falsely susceptible, compared with 1 of 29 strains with 1-µg oxacillin disks, leading them to recom-

mend the use of either disk. They used Mueller-Hinton agar plus 5% lysed horse blood.

We have analyzed a further 78 pneumococcal strains, including 19 relatively resistant strains (MIC, ≥0.12 µg/ml by a serum broth microdilution method), with Columbia agar base plus 5% horse blood and ambient air incubation. We found 2 of 19 strains to be falsely susceptible with both methicillin and oxacillin disks.

The results of these three studies with different media thus fail to show a difference in false susceptibility between the oxacillin and methicillin disks using the criteria of >25 mm for susceptibility to methicillin and ≥20 mm for susceptibility to oxacillin. We do agree that the National Committee for Clinical Laboratory Standards recommendation for oxacillin (≥20 mm; 3) should not be used for methicillin (2).

False resistance also occurs with both methicillin disks (2