

# Letters to the Editor

## Association of *Blastocystis hominis* with Human Disease?

The paper by Qadri et al. (2) on the clinical significance of *Blastocystis hominis* is another classic example of guilt by association prevalent in the literature which has grown up around this organism in the past few years. In their retrospective study, these authors discuss findings in the examination of 19,252 stool specimens from 12,136 patients seen in their hospital in Saudi Arabia during a 2-year period. Of this number, 647 patients were found to have *B. hominis* in their stools. Specimens were examined by saline and iodine-stained wet mounts and by concentrate, with the more accurate trichrome stain reserved for "all liquid specimens and those in which identification of parasite(s) was difficult or equivocal." It is generally recognized that stained fecal films are the single most productive means of stool examination for protozoa (4). Our own experience showed (1; E. K. Markell and M. P. Udkow, Letter, J. Clin. Microbiol. 26:609-610, 1988) that even with examination of trichrome-stained smears by two technologists and one of us (E.K.M.), an occasional pathogen was missed on six consecutive examinations.

When the authors eliminated the 132 patients in whom potential pathogens (bacterial, protozoal, or helminthic) had been found, 515 patients remained, and only 44.4% of these were symptomatic (even including an array of nonspecific symptoms such as fatigue, headache, and depression!). Of these 239 symptomatic patients, 26 had a diagnosis of duodenal ulcer, 3 had peptic ulcer, 17 had ulcerative colitis, 2 had bleeding hemorrhoids, and 18 had leukemia. Another 22 patients had a clinically significant eosinophilia, not characteristic of *Blastocystis* infection despite the report of Sheehan et al. (3). No mention was made of any special types of examination for helminth parasites (schistosomes, *Strongyloides stercoralis*) which might not be found on the usual stool examinations but would be a diagnostic consideration in patients with gastrointestinal symptoms and an eosinophilia of 9 to 11%, especially in the area in which these authors were working. Treatment for *B. hominis* infection was undertaken in 43 of these patients after three to five stool examinations had revealed only *B. hominis*, symptoms persisted or recurred, or eosinophilia was present. We are told that resolution of symptoms occurred in all cases after 7 to 10 days of therapy and that none had *B. hominis* in their stools at follow-up examination after 3 to 6 months. Again, *B. hominis* can best be identified on stained smears, without which it would be most difficult to be certain of its absence. Seven to ten days seems a reasonable time in which to expect resolution of symptoms from many nonspecific causes, and it would be interesting to know what percentage of patients who were not so treated had an equally good outcome.

In our series of 32 patients, 27 were eventually found to have a recognized pathogen (*Entamoeba histolytica*, *Dientamoeba fragilis*, or *Giardia lamblia*) in addition to *B. hominis* and were treated appropriately with iodoquinol, metronidazole, or quinacrine. (At the time we commenced our study, iodoquinol was the drug being recommended for treatment of *B. hominis* infection and quinacrine or metronidazole was used only to treat those patients who had giardiasis.) On reexamination approximately 4 weeks after completion of treatment, all 27 patients were found still to be

infected with *B. hominis*, as was the case with the 5 patients in whom we were able to find only *B. hominis*. None of those with recognized pathogens remained symptomatic after treatment. Again, posttreatment specimens were stained with trichrome and examined by three different persons.

In the series reported by Qadri et al. (2), 5.3% were found to have *B. hominis* in their stools, a smaller number than has been found in most studies. More patients were found to have *Entamoeba coli* (9.1%) and *Endolimax nana* (5.8%). We might speculate what association could have been made between either of these organisms and the symptoms presented by the affected patients if we did not have foreknowledge that they are "nonpathogens."

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### Author's Reply

Markell and Udkow have raised the same points again regarding our paper (5) which were amply clarified by D. J. Sheehan in response to their earlier letter (E. K. Markell and M. P. Udkow, Letter, J. Clin. Microbiol. 26:609-610, 1988). The methodology used in our laboratory is consistent with the standard procedures (8). Stained fecal films are necessary for the identification of trophozoites in diarrheal stools, and they augment identification when it is difficult or equivocal; but anyone with reasonable laboratory experience knows that they are not the "single most productive" method. If potentially pathogenic parasites are not seen in the presence of symptoms, we do examine four to six specimens collected on alternate days, but the Kaiser Foundation Medical Center is probably the only institution where the same specimen is examined by two technologists and a physician. Laboratories handling 50 to 200 specimens a day cannot afford such luxury. During my 20 years of experience in the United States and Saudi Arabia, we did not have any difficulty in recovering *Strongyloides stercoralis* using the very same methods. This parasite does not seem to be endemic here, not because of inadequate methodology but because of temperatures reaching 120 to 125°F (48.9 to 51.7°C), coupled with low humidity of 15 to 20%. We do not have any difficulty in finding schistosomes, which account for 1.5 to 2.0% of all parasites, including *Blastocystis hominis* (6). Markell and Udkow state that eosinophilia is not

characteristic of *B. hominis* infection without any supporting reference or data.

Their doubts about the clinical significance and effective treatment of this parasite appears to be based on their experience with 32 patients, 27 of whom had pathogenic parasites, that was published 4 years ago (4). Sheehan (Markell and Udkow, Letter) has dealt with this aspect succinctly, and Markell and Udkow's opinion is contrary to increasing evidence of the pathogenic potential of *B. hominis* (2, 3, 5, 7, 9).

They correctly state that 55% of our patients were asymptomatic. It is possible that strains of *B. hominis* differ in virulence, which has been shown for *Giardia lamblia* (1). Since most infections with *G. lamblia*, *Entamoeba histolytica*, and other microorganisms are subclinical, one might speculate whether our esteemed colleagues would consider them nonpathogenic because the hosts are "asymptomatic."

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## Typing and Nosocomial Candidiasis

Fungi, especially *Candida* spp., are emerging as major nosocomial pathogens in both immunocompetent and immunocompromised patients. The rational development of effective measures to prevent and control nosocomial candidiasis needs a thoughtful understanding of the epidemiology of *Candida* spp., with particular reference to the reservoir and mode of transmission of the organisms.

Isenberg et al. recently provided some interesting insights into these issues, documenting the role of one individual as the source of a *Candida tropicalis* outbreak of cardiac bypass surgery wound infections involving eight patients (2). In fact, they examined fingertip and nasopharyngeal cultures of all personnel in contact with these patients, isolating *C. tropicalis* from one nurse only. Apart from this microbiological evidence, other important confirmations resulted from additional considerations: (i) the suspect nurse was the only individual involved in the surgical operations of all infected patients, (ii) the transmission of *C. tropicalis* occurred only while she was working as a scrub nurse and not while she was working as a circulating nurse, and (iii) her removal from the cardiac team terminated the cluster outbreak. The authors concluded by ascribing the cluster epidemic to the hands and oropharynx of the scrub nurse, acting as a common source.

We recently had the opportunity to investigate a nosocomial outbreak of systemic *C. albicans* infection involving eight patients who had received parenteral nutrition (3a). This epidemic had some similarities to the one reported by Isenberg et al., especially as to the possible role of one staff member as the common source of infection. In fact, the microbiological surveillance of personnel showed that two physicians and three nurses harbored *C. albicans* in the

nasopharynx. We typed the isolates using DNA fingerprinting, a molecular method proposed as a helpful epidemiological tool for the study of *Candida* species (5) and successfully applied to the investigation of outbreaks of systemic *C. albicans* infections (3, 6), as well as to subspecies delineation of nosocomial isolates of *C. tropicalis* (4). DNA fingerprinting revealed that the same DNA pattern was shared by the *C. albicans* isolates from all patients and one nurse acting as a specialized nutrition nurse, whereas different DNA fingerprints were observed in the isolates recovered from the other staff members.

In the outbreak investigated by Isenberg et al., the attribution of the outbreak to the positive scrub nurse had immediate practical consequences (she was removed from the cardiac team) and has important scientific implications for both the understanding of the epidemiology of nosocomial candidiasis and the planning of relevant control interventions. In our opinion, however, the typing of *C. tropicalis* isolates would have reinforced and definitively proven the conclusions about the role of the scrub nurse as the single source of the outbreak, in particular ruling out the possibility that not all the cultured strains had the same origin. In reality, such a possibility appears to be quite unlikely in the case in question, even considering that *C. tropicalis* can be found in routine cultures from the nose, throat, skin, vagina, and gastrointestinal tract of healthy individuals (1). However, this possibility was well documented in an outbreak of systemic *C. albicans* infection which occurred in a neonatal intensive care unit (6). In that apparently single outbreak, DNA analysis revealed that two distinct strains were actually involved.