(10 g/d) was resumed orally, resulting in improvement in the skin lesions in 1 week.

Noncompliance with oral medications during October 1995 was associated with the appearance of new skin lesions. A new multidrug regimen was started in December 1995 with the combination of itraconazole (400 mg/d orally) and pentamidine (150 mg/d for 5 days and then 300 mg/d for 6 days intramuscularly). No improvement was observed. The initial therapeutic regimen (5-fluorocytosine plus itraconazole) was administered again. Following this therapy, the course of the acanthamoeba infection altered with improvement and worsening. A granular lesion was also noted on the wall of the right nasal fossa (biopsy was refused). Parasitological examination revealed free-living amebas in purulent discharge from the left maxillary sinus in February 1996. The patient died of inanition in July 1996 after 1 year of acanthamoeba infection without clinical or radiological signs of CNS involvement; autopsy was not performed.

Disseminated infections with free-living amebas in patients with HIV infection are infrequent. As of 1 October 1996, 103 cases of disseminated acanthamoeba infection had been reported worldwide [1]. Of these 103 cases, 72 were from the United States alone (including >50 cases in patients with AIDS). This infection is usually associated with a poor prognosis. Our report documents a prolonged clinical course of cutaneous and sinus infections due to Acanthamoeba in a patient with AIDS. The successive therapeutic regimens demonstrate the transient efficacy of 5-fluorocytosine. Pentamidine (in association with itraconazole) did not show efficacy against the skin lesions.

Previous reports showed variability in the activity of 5-fluorocytosine, pentamidine, and itraconazole against different Acanthamoeba strains [2–6]. Furthermore, acquired resistance of Acanthamoeba to 5-fluorocytosine was suggested by different in vitro studies and other case reports [7–8]. 5-Fluorocytosine should be included in chemotherapy for disseminated infections due to free-living amebas in combination with other antiamebic agents.

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The patient’s condition rapidly deteriorated, and he required intubation; however, extubation could be performed within 72 hours. A chest radiograph at intubation revealed a right lower lobe infiltrate; a large right pleural effusion developed over the next 5 days. Fever appeared at the time of extubation. Gram staining of sputum revealed gram-negative cocccobacillary forms although routine cultures were negative. Cultures of blood obtained simultaneously yielded CDC Group O1 bacteria. Therapy with levofloxacin and piperacillin/tazobactam was initiated.

The subsequent hospital course was difficult, marked by the development of a right hydropneumothorax with a bronchopleural fistula that resolved with chest tube placement. Gram staining and culture of the pleural fluid (obtained 5 days after initiation of antibiotic therapy) were negative. The WBC count peaked at 19,000/mm³ several days after chest tube removal, but gradual defervescence and return of the WBC count to normal then occurred. Intravenous antibiotics were given for a total of 16 days in the hospital followed by an outpatient course of oral levofloxacin and clindamycin for 42 days. Follow-up chest radiographs demonstrated stabilization of his condition, and he was doing well 1 year later.

The gram-negative bacterial strain isolated from blood culture and presumably on gram staining of sputum was analyzed at the Texas State Department of Health (Austin, TX). The organism was a motile aeroobe possessing one to two polar flagella and showed very light growth of 1-mm yellow colonies on plates of both blood agar and trypticase soy agar. It did not grow on 50% chocolate agar.
MacConkey agar. Biochemical testing revealed that the organism was oxidase-, catalase-, and nitrate-positive, and it was capable of growth at 42°C. The isolate oxidized but did not ferment glucose and did not utilize xylose, mannitol, sucrose, maltose, or lactose. It did not reduce nitrate or split urea, and the indole test was negative. The lysine, arginine, and ornithine enzymatic reactions were negative. Results of clinical laboratory testing of the strain isolated from our patient were consistent with the growth and biochemical patterns established by the CDC for CDC Group O1 organisms [1]. By microtiter dilution testing, the strain was found to be susceptible to piperacillin, ceftazidime, ceftriaxone, ciprofloxacin, chloramphenicol, amikacin, imipenem, ticarcillin/clavulanic acid, and ampicillin/sulbactam but resistant to aztreonam.

This case represents the first report of the CDC Group O1 bacterium as the etiologic agent of necrotizing aspiration pneumonia. The development of this pneumonia appears to have followed the witnessed aspiration event; therefore, we speculate that the source of this microorganism was either oropharyngeal or gastrointestinal flora. Although the pneumonia may have been the product of a polymicrobial infection, no other organisms other than CDC Group O1 bacteria were isolated from cultures of specimens collected at the time of the initial febrile episode.

The CDC has received 62 different clinical isolates of this organism from various sources. The sources are quite diverse and include blood (55% of isolates), CSF (6%), pleural fluid (6%), wound (5%), cervix (5%), and other sites (23%), including vagina, heart valves, lymph nodes, eye, intravenous fluid, platelets, serum, scapula, finger, bone marrow, peritoneal fluid, allergenic extract, a water bath, and “unknown” [1]. Although the invasive potential for this organism is obvious, no clinical data have appeared in the literature, and the clinical significance of these isolates is unknown. This report suggests that this bacillus is invasive enough to cause necrotizing pneumonia with complicating bronchopleural fistula and bacteremia.

Therapy consisted of simple local drainage as well as dual antimicrobial treatment directed by the reported susceptibility patterns of the isolate. A broad pattern of susceptibility to antibiotics was apparent, but we would recommend dual therapy for infections with the CDC Group O1 bacterium on the basis of occasional recalcitrance of syndromes caused by similar nosocomial gram-negative rods [2].

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Chronic Diarrhea Associated with Vibrio alginolyticus in an Immunocompromised Patient

Vibrio alginolyticus is a halophilic gram-negative organism that is considered to be part of the normal marine flora. In the warmer months, it can reach concentrations sufficient enough to cause disease in humans. It mainly affects persons who have had direct contact with seawater or those who have handled shellfish. V. alginolyticus is most commonly associated with wound infections, otitis media, and otitis externa [1]. This organism has only rarely been associated with acute diarrheal illness, and we were unable to find any reports of chronic diarrhea caused by V. alginolyticus. We describe a unique case of chronic diarrhea associated with V. alginolyticus infection in an immunocompromised patient.

A 38-year-old homosexual man was admitted to our medical service with multiple complaints including a 3-month history of diarrhea as well as more recent development of odynophagia and dysphagia. He reported about five episodes of watery nonbloody stool per day as well as a 25-lb weight loss over that same period. He denied taking any recent trips to the sea or eating any raw seafood. The patient had not been tested for HIV infection before admission.

At admission, laboratory analyses revealed the following values: WBCs, 3,200/mm^3; hemoglobin, 12.9 mg/dL; and hematocrit, 38.2%. After fluid therapy, his hematocrit fell to 33.8%. Electrolyte analysis showed the following values: sodium, 134 mEq/L; potassium, 4.4 mEq/L; chloride, 98 mEq/L; bicarbonate, 27 mEq/L; blood urea nitrogen, 7 mg/dL; creatinine, 0.6 mg/dL; and glucose, 83 mg/dL. The patient’s total protein level was 8.6 g/dL, and his albumin level was 3.4 g/dL. Three stool samples sent for determination of fecal leukocyte counts were all negative, as were two samples sent for detection of Clostridium difficile toxin. Two stool samples were also sent for culture. ELISA and western blotting were reactive for antibody to HIV, and his absolute CD4 cell count was subsequently found to be 28/mm^3.

During hospitalization, the patient underwent esophagogastroduodenoscopy to evaluate the symptoms of odynophagia and dysphagia, which revealed mild duodenitis, gastritis with small gastric ulcers, and severe esophagitis suggestive of candidal infection. A biopsy specimen of the esophagus was positive for acute herpetic esophagitis, and pathology of esophageal brushings was consistent with herpes simplex virus infection as well as infection with Candida species. The patient was treated with acyclovir and fluconazole with subsequent resolution of his dysphagia and odynophagia.

The stool samples were plated directly onto MacConkey agar, trypticase soy agar with 5% sheep blood, MacConkey agar with sorbitol, Hektoen enteric agar, and campylobacter sheep blood agar (10% sheep blood). After 48 hours of incubation, a moderate quantity of gram-negative, small, curved, oxidase-positive rods was observed growing only on the plate with trypticase soy agar with 5% sheep blood. The isolate was identified as V. alginolyticus by means of the Negative Breakpoint Combo panel (Dade International, MicroScan Division, West Sacramento, CA). Subculturing the isolate to thiosulfate citrate bile salts sucrose agar yielded

References

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