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, cefotaxime, 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, intravascular fluid, platelets, sternum, scapula, finger, bone marrow, peritoneal fluid, allergic 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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References
characteristic yellow colonies, which were the only type of organism identified. Cultures were negative for Salmonella, Shigella, Campylobacter, and Escherichia coli O157:H7.

Antimicrobial susceptibility testing for the isolate revealed susceptibility to aminoglycosides, fluoroquinolones, trimethoprim-sulfamethoxazole, and tetracycline and resistance only to ampicillin. The patient was treated with a single 1-g oral dose of ciprofloxacin and subsequently reported complete resolution of his diarrhea.

*V. alginolyticus* has only rarely been associated with acute diarrheal illness [2–8]. In the seven studies, a total of 15 cases of diarrhea due to *V. alginolyticus* were reported from 1980 to 1995. In four of these 15 cases, another enteric pathogen along with *V. alginolyticus*, including Campylobacter, Shigella, E. coli, and Vibrio parahaemolyticus, was isolated, suggesting that *V. alginolyticus* may not have even been the causal agent in these cases [5–7]. In an additional eight cases, it was not specified whether *V. alginolyticus* was the sole organism recovered [3, 4]. Only three prior cases clearly demonstrate pure isolation of *V. alginolyticus* from a patient with acute diarrhea [2, 6, 8]. Chronic diarrhea was not described in any of the 15 cases. A MEDLINE search of the English-language literature from 1966 to 1999 revealed no reports of chronic diarrhea associated with *V. alginolyticus*.

Our case is also of interest because it involved a homosexual man who was immunocompromised secondary to AIDS. This situation raises the question of whether patients with AIDS or homosexual males are at increased risk of developing *V. alginolyticus* infections. An AIDSLINE search of the English-language literature from 1980 to 1999 revealed no other case reports of such infections. There have, however, been several case reports of gastroenteritis caused by other *Vibrio* species in patients with AIDS. One report described acute diarrhea caused by *Vibrio fluvialis* in a man with AIDS [9], and another discussed a case of sepsis, peritonitis, and gastroenteritis in a homosexual man with AIDS that was found to be caused by *Vibrio vulnificus* [10]. In both of these cases, the patients had eaten seafood (scallops and raw oysters, respectively) prior to symptom onset, suggesting that the infections were food-borne.

**Acalculous Cholecystitis Associated with Plasmodium falciparum Infection**

Acute acalculous cholecystitis (AAC) has been described in association with various infectious agents [1–3]. Although gastrointestinal manifestations are not uncommon in malaria [4], AAC as a complication of this infection has not been previously reported. We report the first case of AAC attributed to an acute infection with *Plasmodium falciparum*.

A 26-year-old female was admitted to the hospital with a 5-day history of abdominal pain, nausea, vomiting, fever, and chills. She had been seen elsewhere 2 days previously and was diagnosed with gastroenteritis. She was told to take acetaminophen, but when her symptoms persisted, she came to the hospital. At admission, she was lethargic but capable of being aroused with a temperature of 39.5°C, pulse rate of 120, and blood pressure of 80/35 mm/Hg. The neck was supple, and no rashes were present. Results of chest and cardiovascular examinations were normal. The abdomen was diffusely tender with rebound in the right upper quadrant.

Laboratory investigations showed a WBC count of 4.5 × 10⁹/L, hemoglobin level of 104 g/L, and platelet count of 56 × 10⁹/L. The total bilirubin concentration was elevated to 60 mmol/L (indirect bilirubin level, 51 mmol/L). Transaminase and alkaline phosphatase values were normal. Abdominal ultrasonography was performed and revealed a thickened gallbladder wall (5 mm) surrounded by a thin rim of fluid. No stones were visible, and a diagnosis of acalculous cholecystitis was made. The patient was treated with intravenous fluids and broad-spectrum antibiotics, and an emergency cholecystectomy was scheduled. However, on further questioning, it was determined that the patient had returned from Togo, Africa, 2 weeks before hospitalization.

It was her first trip home in 10 years, and she had stayed there for 2.5 weeks but did not take any prophylaxis. Examination of a...