Bacteremia Caused by a Novel Bordetella Species, “B. hinzii”


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Bordetella spp. cause respiratory tract diseases in warm-blooded animals. Only Bordetella bronchiseptica has been reported to cause bacteremia in humans, and this rare infection usually occurs with pneumonia in immunocompromised patients. We describe “Bordetella hinzii” bacteremia in an AIDS patient without a respiratory illness. Combining biochemical phenotyping with fatty acid analysis permitted preliminary identification of this previously undescribed pathogen; identity was confirmed by DNA-DNA hybridization. This report extends the spectrum of human infections caused by the bordetellae.

Members of the genus Bordetella are well known to cause respiratory tract diseases in a variety of hosts. Bordetella pertussis and Bordetella parapertussis are etiologic agents of the human illness called whooping cough (15). Bordetella bronchiseptica is primarily a veterinary pathogen that causes snuffles in rabbits, atrophic rhinitis in swine, and kennel cough in dogs; rare human infections can result in pneumonia with or without bacteremia (20). Disease caused by Bordetella avium has been described only for poultry (rhinotraceheitis) (7, 9, 17). Here, we report bacteremia caused by “Bordetella hinzii” in an immunocompromised patient without evidence of respiratory infection. “B. hinzii,” a recent addition to the genus Bordetella (18), can cause secondary or opportunistic respiratory infections in poultry. Although it is similar to B. avium, we distinguished this isolate by combining biochemical phenotyping with whole-cell fatty acid analysis and confirmed its identity by using additional genotypic methods. This report both introduces “B. hinzii” as a potential human pathogen and extends the spectrum of infections caused by the bordetellae.

Case report. A 42-year-old homosexual man with AIDS and an indwelling left subclavian Groshong catheter was admitted to Harborview Medical Center because of fever and left-upper-extremity swelling. Ten months prior to admission, the patient was admitted with Pneumocystis carinii pneumonia and dermatomal herpes zoster viral infection. Six months prior to admission, the patient developed fever, photophobia, aphasia, and right hemiplegia. Magnetic resonance imaging showed an area of focal linear enhancement in the left frontal-parietal cortex; a thombosed intracranial artery with normal surrounding brain tissue was found at craniotomy. Pathologic examination of the thrombosed vessel revealed necrotizing arteritis and gram-negative cocci within macrophages. No organisms were seen in the surrounding cerebral cortex, and all cultures from biopsy material were sterile. A transesophageal echocardiogram showed no valvular vegetation. A left subclavian Groshong catheter was inserted. Vancomycin (1 g every 12 h) and ceftriaxone (2 g every 24 h) were administered intravenously for 4 weeks. At the completion of antimicrobial treatment, the catheter was not removed because venous access was difficult and blood tests were frequent. Azithromycin (250 mg daily) was subsequently administered for 5 months, and during this period, the aphasia and hemiplegia resolved and follow-up computerized tomography demonstrated resolution of the abnormality.

Three months prior to admission, the patient’s CD4 count was 31 cells per mm³. Three days prior to admission, pain and swelling developed in his left shoulder and arm. He denied cough, dyspnea, or fever. The patient was an unemployed interior decorator who lived alone without pets. In recent months, he had neither traveled nor eaten unusual foods. Medications on admission, all oral and taken once daily, included 250 mg of azithromycin, 100 mg of dapsone, 150 mg of trazodone, and 500 mg of naproxen. The physical examination at admission showed a temperature of 39.3°C and left-upper-extremity erythema and edema. Initial laboratory studies showed a hematocrit of 30%, a leukocyte count of 5.2 × 10⁹/liter, and a platelet count of 170 × 10⁹/liter. A chest radiograph was without evidence of pneumonia; the patient did not produce sputum for culture. A venous duplex examination showed an occlusive noncompressible thrombosis of the left internal jugular and subclavian veins. The Groshong catheter was removed, and the tip was sent for culture. Intravenous heparin and intravenous vancomycin (1 g twice daily) were administered. Seventeen hours after admission, the leukocyte count had risen to 11.9 × 10⁹/liter. On the second hospital day, gram-negative rods grew from four of four blood cultures drawn at the time of admission. Coagulase-negative staphylococci also grew from three of four of these blood cultures, but only from cultures drawn through the Groshong catheter; the catheter tip was culture negative. The patient received 2 g of ceftriaxone intravenously. Subsequently, his leukocyte count decreased to 4.7 × 10⁹/liter, his temperature returned to normal, and he remained afebrile for the duration of hospitalization. Peripherial intravenous access was lost on hospital day 3, and the patient refused central venous catheterization. Intramuscular ceftriaxone (1 g daily) and oral rifampin (600 mg twice daily) were started, and vancomycin was discontinued. On hospital day 6, the patient agreed to central venous catheterization, and his antimicrobial regimen was changed to intravenous ticarcillin-clavulanate (3.1 g every 6 h) and intravenous gentamicin (100 mg every 8 h). On hospital day 7, the gram-negative organism was identified as a Bordetella species resembling Alcaligenes spp. The patient’s antimicrobial agents were changed to intravenous ampicillin-sulbactam (3 g every 6 h), but this was discontinued on hospital day 15, when an erythematous maculopapular eruption devel-
oped on the upper chest. Intravenous vancomycin (1 g every 12 h) and intravenous ceftazidime (1 g every 8 h) were substituted for the ampicillin-sulbactam. The patient completed another 2 weeks (4 weeks total) of intravenous antimicrobial therapy and was discharged to home. Multiple follow-up blood cultures were negative.

Microbiological investigation. Gram-negative rods grew in BACTEC 26 medium (Becton Dickinson Diagnostic Instrument Systems, Towson, Md.) inoculated with blood obtained from the patient by peripheral venipuncture and from his central indwelling catheter. These motile organisms were oxidase positive and assimilated adipate, 3-malate, citrate, and phenylacetate (API-NFT; Biomerieux Vetek, Inc., Hazelwood, Mo.). They were nitrate and urea negative, with a biochemical phenotype (NFT profile 0000067) suggesting _B. avium_. Bacteria closely related to _B. avium_, i.e., the genus _Alcaligenes_ (9) and _Bordetella_ species other than _B. avium_, have previously been isolated from humans. In an attempt to verify the biochemical identification, we used whole-cell fatty acid analysis (Table 1). The taxonomy of the _Bordetella_ and _Alcaligenes_ genera is complex (5, 9, 11), and their fatty acid profiles are similar (8, 12). Table 1 shows they contain mostly C_{16:0}ω7c, C_{16:0}ω9c, C_{17:0}ω7c, and “SUM in feature 3” (C_{16:1}ω7 c/n = 1 and/or 3-OH C_{16:0}). _Alcaligenes xylosoxidans, B. avium_, and the patient’s isolate contain 18:0 ω9c hydroxy fatty acid derivatives (2-OH C_{18:0} and 2-OH C_{18:1}ω9c), the other bacteria do not. The fatty acid profile of our isolate most closely resembles that of _A. xylosoxidans_ (Table 1), but biochemically these organisms are very different (NFT profile 1040477 for _A. xylosoxidans_ includes the reactivities of our isolate [described above] plus nitrate reduction, glucose fermentation, and assimilation of α-gluccone and caprate). Finally, quantitative differences among the predominant fatty acid constituents distinguish our isolate from _B. avium_. Their C_{16:0}ω7c and “SUM in feature 3” contents are similar, but their C_{16:1}ω7c (isolate, 20.58% ± 0.60%; _B. avium_ 3.82% ± 0.52% [mean ± standard error]) and C_{17:0}ω7c (isolate, 19.13% ± 1.5%; _B. avium_, 36.15% ± 0.95%) contents are different. Among other possibilities, these data suggested that the isolate may represent another species of the genus _Bordetella_ that is biochemically similar to _B. avium_. We confirmed this hypothesis and identified the bacteria as “ _B. hinzii_” by a detailed comparison with the “ _B. hinzii_” type strain LMG 13501T (taxonomic and phylogenetic descriptions of “ _B. hinzii_” will be published elsewhere [18]). Their protein electrophoretic profiles are virtually identical (data not shown), the DNA-DNA homology of the two strains determined by rena-