Short Communication

Two Cases of *Burkholderia cenocepacia* in Septicemic Patients

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**SUMMARY:** We herein report repeated isolation of *Burkholderia cenocepacia* from two cases of septicemia admitted to cardiothoracic and bone marrow transplant (BMT) units. The two blood cultures taken from each patient grew *B. cenocepacia*. Both patients turned afebrile after appropriate antimicrobial therapy, and the subsequent blood cultures were sterile. However, both patients had recurrence of fever after about a week, and the patient in the BMT unit died due to respiratory failure. Environmental surveillance was conducted in both units. Non-fermenting Gram-negative bacilli including *Pseudomonas aeruginosa* were isolated from environmental samples in the cardiothoracic ward.

*Burkholderia cepacia* complex has emerged as an important cause of morbidity and mortality in hospitalized patients, largely because of high intrinsic antibiotic resistance. It is one of the most antimicrobial-resistant organisms encountered in the clinical laboratory (1-3). It causes a wide variety of infections ranging from superficial to deep-seated and disseminated infections, such as pneumonia, especially in patients with cystic fibrosis, meningitis, peritonitis (in patients undergoing peritoneal dialysis), septicemia and bronchiectasis (3,4). We herein report repeated isolation of *Burkholderia cenocepacia* from two cases of septicemia admitted to the cardiothoracic ward and bone marrow transplant (BMT) unit of Nehru Hospital, Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh.

Case 1. A 40-year-old man was admitted for chemotherapy in the BMT unit of PGIMER, Chandigarh with a diagnosis of acute myeloid leukemia (AML) M4 as per French-American-British classification. His complete blood count at presentation was hemoglobin 4.4 g/dl, leukocyte count 9,700/μl and platelet count 62,000/μl. He was given standard induction therapy in the form of daunorubicin 45 mg/m2 for 3 days and cytarabine and resistance to meropenem. The patient was then switched over from meropenem to cefoperazone-sulbactam. His day 14 bone marrow was not performed. He developed febrile neutropenia and a central venous line-infection that was managed with vancomycin and co-trimoxazole with intermediate susceptibility to tetracycline and resistance to meropenem. The patient was then switched over from meropenem to cefoperazone-sulbactam (2 g twice daily intravenously) and amikacin (750 mg once daily intravenously). *B. cenocepacia* was not isolated from sputum or lung specimens from dissection or necropsy. Though the two subsequent blood cultures were sterile, the patient was febrile and died due to respiratory failure. His peripheral blood film did not show any blasts though a bone marrow examination was not performed.

Surveillance is routinely performed in the BMT unit every 3 months to point to the probable source(s) of infection, if any. The betadine (10% povidone iodine), 70% ethyl alcohol used for skin and surface disinfection, air-conditioner grill, tap mouths, suction tube, oxygen mask, curtains, settle plates

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for air, and skin swabs from the patient were subjected to culture but grew no non-fermenting gram-negative bacilli (NFGNB). Case 2. A 27-year-old man was admitted to the cardiology department with high-grade fever and chills lasting 1 month. He was a known case of mitral stenosis. On examination, he was found to be pale, but no cyanosis/etem or lymphadenopathy or clubbing was noted. His blood pressure was 110/70 mmHg, pulse rate was 100/min, respiration rate was 26/min. Echocardiography indicated large vegetations (12 x 12 mm) on the anterior mitral leaflet and another (2 x 3 mm) on the posterior mitral leaflet with borderline mitral stenosis and severe mitral regurgitation. Laboratory investigations revealed hemoglobin = 8.6 g/dl, erythrocyte sedimentation rate = 52 mm, total leukocyte count 16,100/μl, differential leukocyte count = N 79, L 15, E 4 while serum electrolytes were within normal range. C reactive protein was negative, Widal titers were within normal limits, urine culture was sterile and Cryp
tococcus latex agglutination was negative. The patient was diagnosed to have infective endocarditis and was being treated for the same. Two blood cultures were received on the same day during the initial week of the admission. Approximately 5 ml of blood was aseptically collected and added to each of two bottles containing 50 ml of Tryptone Soy broth and Bile broth (Hi Media). Both the bottles grew B. cenocepacia when incubated aerobically at 37°C and subcultured on sheep blood agar and MacConkey agar after 48 h incubation (both were sterile after 24 h incubation). The rods did not oxidize glucose and therefore were labeled as B. cenocepacia. B. cenocepacia could not be confirmed by RFLP, as the strain could not be revived. B. cepacia complex grows well on most laboratory media but may lose viability on blood agar in 3 to 4 days (5). The isolates were found to be susceptible to cefazidime, and the patient responded to the same (2 g twice daily intravenously) for 1 week. Following antimicrobial therapy, he became afebrile, and the subsequent blood cultures were sterile. However, he turned febrile again and did not respond to antibacterial and antifungal therapy until the time when he was discharged on request. The size of the vegetations (anterior mitral valve = 12 x 12 mm and posterior mitral valve = 2 x 3 mm on echocardiography) remained unchanged during the additional week of hospital stay. The causative organism for infective endocarditis was likely B. cenocepacia, as no other organism grew in culture. Since the patient was lost for follow up we could not confirm the same. The betadine (10% povidone iodine), 70% ethyl alcohol used for skin and surface disinfection, needle, cotton pack, instrument tray, swabs from bed linen and other materials in the patient’s cubicle, and skin swabs from the patient were cultured, and only aerobic spore-bearing bacilli were isolated from some samples and were not considered significant. A second intensive surveillance was conducted to identify the environmental source, e.g., samples from washbasins, tap mouths and handles and soaps used for hand washing were collected. Cultures from three taps and corresponding soaps grew NFGNBs (including P. aeruginosa), but B. cepacia complex could not be isolated. For over 3 months in 2004-2005, out of 5,437 blood cultures we isolated 70 (41.2%) lysine-positive NFGNBs (B. cepacia complex and Stenotrophomonas maltophilia) among 170 NFGNB. In 2005-2006 (September to December) out of 7,779 blood cultures, 74 (29.1%) lysine-positive NFGNB were obtained out of 254 NFGNB. In 2006-2007 (September to December) out of 8,601 blood cultures we isolated 25 (11.36%) lysine-positive NFGNB among 220 NFGNB. In these two nosocomially acquired cases, B. cenocepacia was isolated twice from both cases. The two patients had never been in the same ward. The strains of the two cases were isolated at a gap of more than 6 months from different wards. Therefore, in the present scenario, these two cases do not appear to be epidemiologically linked, and unlikely to be from the same point source. A limitation of this study is that pulsed-field gel electrophoresis could not be performed on these four isolates to confirm their clonality. Lysine-positive non-fermenters (B. cepacia complex and S. maltophilia) are emerging as important pathogens in India with an increasing incidence particularly during the winter months. Hospitalized patients can acquire organisms from many sources including the environment, but it is almost impossible to screen all environmental surfaces and there is no guarantee that the implicated organism will be isolated (6-8). Potential pathogens can be established in favorable niches in the hospital environment, which may act as a significant reservoir for transmission to patients (6,9). Recently, bacteriological standards for assessing surface hygiene have been proposed, and the presence of a recognized pathogen (in any amount) and/or heavy burden of organisms (regardless of identity) is considered as constituting a potential risk to patients. A heavy microbial burden in the environment may mask the finding of a particular pathogen and also implies the presence of an epidemiologically related pathogen. Environmental surveillance in the BMT and cardiothoracic wards was unsuccessful in the isolation of B. cenocepacia, but oxidase-positive NFGNB were isolated in both instances. Since B. cenocepacia and NFGNB are epidemiologically related, it is possible that there is some environmental niche harboring the pathogen (6).

REFERENCES