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Outbreak of *Burkholderia cepacia* complex among ventilated pediatric patients linked to hospital sinks

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We investigated a cluster of *Burkholderia cepacia* complex colonization in ventilated pediatric patients. Isolates from 15 patients, 2 sink drains, and several ventilator components were found to belong to a single *B cenocepacia* clone. Hospital tap water used during oral and tracheostomy care was identified as the most likely mechanism for transmission.

Key Words: Infection control; disease outbreak; intensive care unit; pediatrics; water supply; cystic fibrosis.

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The *Burkholderia cepacia* complex (Bcc) is a multispecies complex of bacteria that commonly cause respiratory infections in persons with cystic fibrosis (CF). The species composing the ibed as member of the Bcc include *B cepacia*, *B cenocepacia*, *B multivorans*, *B stabilis*, *B vietnamiensis*, *B dolosa*, *B ambifaria*, *B anthina*, and *B pyrrocinia*. *B cenocepacia* accounts for approximately 45% of Bcc infections in persons with CF in the United States, but up to 80% of those in Canada and some European countries.[1] Pediatric outbreaks have been reported, particularly in critically ill and immunocompromised patients.[2-4] Health care associated outbreaks have been traced to contaminated respiratory therapy devices, medications, and mouthwash.[2,4-8] Here we describe the investigation of 16 pediatric patients who developed Bcc respiratory colonization.

Between July 2005 and June 2006, 9 cases of respiratory colonization with Bcc were identified at a large tertiary pediatric hospital. All cases were patients on ventilation but without CF. In each case, the treating physician deemed that the isolate represented colonization. Although no patient with CF acquired Bcc during this period, the hospital was concerned about possible spread to this vulnerable patient population. State and local health departments were notified, and a formal investigation, in conjunction with the Centers for Disease Control and Prevention (CDC), was initiated in August 2006. Because this was an outbreak investigation, institutional review board approval was not required.

A case-control study was conducted to identify risk factors for Bcc acquisition. Census logs for intensive care units (ICUs) were used to identify control subjects admitted between July 2005 and June 2006. Controls were patients aged <18 years who were admitted to an ICU within 2 weeks of the matched case. To reduce potential misclassification, controls were required to have a respiratory culture that did not grow Bcc during the 4-week window.

Investigators collected a total of 88 environmental specimens and product samples from affected units, targeting high-touch surfaces, shared equipment, tap

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water and sink surfaces, sink aerators, respiratory medications, patient care products (eg, mouthwash, saline bullets), hand hygiene products (eg, Health Stat [triclosan] antiseptic handrub, Stat-Rinse [alcohol] antiseptic hand gel; Richmond Laboratories, Crystal Lake, IL), and used ventilator components from case-patients with Bcc colonization. The investigators reviewed adherence to hand hygiene and isolation precautions, environmental cleaning, and respiratory equipment disinfection, and interviewed and observed health care workers (HCWs).

Clinical isolates were sent to the *B cepacia* Research Laboratory and Repository at the University of Michigan Medical Center. Isolates were identified to the species level using 16S rRNA and *recA* species-specific polymerase chain reaction (PCR) assays and *recA* restriction fragment length polymorphism analysis. Isolates were also genotyped by repetitive element PCR using a BOX A1R primer. Environmental specimens and product samples were sent to the CDC for bacterial culture. Clinical and environmental isolates were compared using pulsed-field gel electrophoresis and BioNumerics software (Applied Maths, Austin, TX), as described previously.[5]

Nine case-patients with Bcc respiratory colonization and 27 control patients were included in the case-control study. There were no significant differences between cases and controls with respect to sex, age, severity of underlying illness by McCabe-Jackson classification,[9] hospital unit, or outcome. No differences between cases and controls were noted in antibiotic or respiratory medication receipt or in the use of topical medications or products. There were also no between-group differences in exposure to procedures and equipment evaluated (ie, computed tomography scan, magnetic resonance imaging, ultrasonography, Doppler ultrasonography, echocardiography, and peripherally inserted central catheter line placement). The median length of hospital stay was significantly longer for cases than controls (60 days vs 16 days; $P = .02$). Using conditional logistic regression, case-patients were more likely to have been hospitalized for ≥30 days (odds ratio, 27; $P = .002$) and to have received mechanical ventilation for ≥10 days (odds ratio, 27; $P = .002$). We also identified HCWs who documented care in the medical records for cases and controls, and found that no specific HCW was associated with Bcc acquisition. Environmental specimens obtained from randomly selected HCW code pagers and stethoscopes also were negative for Bcc.

The infection control review revealed lapses in nursing and respiratory therapy procedures and suboptimal adherence to hand hygiene and isolation precautions. Practices observed included multiple insertions of suction catheters into fluid containers used for patient suctioning, and connection of suction tubing to syringes taped to the bedrail and stuffed with gauze to minimize sound.

After completion of the case-control study, infection preventionists reported identification of 7 additional patients between October 2006 and March 2007 who received ventilation, did not have CF, and had respiratory colonization with Bcc. Two colonized patients subsequently developed Bcc bacteremia, which was treated. In March 2007, infection preventionists observed HCWs using tap water when providing oral and tracheostomy care to ventilated patients and to rinse tube feeding bags. Immediate changes were implemented to halt these practices.

Respiratory isolates from all 16 patients were analyzed for species identification. One clinical isolate was identified as *B cepacia*, and the remaining 15 isolates were identified as *B cenocepacia* belonging to the same clone (Fig 1). Genetically similar *B cenocepacia*, with a high degree of relatedness, was recovered from 2 ICU sink drains as well as ventilator components (ie, disposable tubing, filters, humidification chambers, and reusable temperature probes) from colonized patients. *B cenocepacia* was not recovered from any other environmental specimens.

We report 16 cases of Bcc respiratory colonization in ventilated pediatric patients detected over a 20-month period. Respiratory isolates from 15 case-patients, 2 sink drains, and 4 used ventilator components belonging to a single *B cenocepacia* clone suggested a common source. Ongoing observations led to the identification of tap water from hospital sinks as the likely mode of transmission, and the emergence of new cases stopped only after infection preventionists recognized and ended the practice of using tap water for oral and tracheostomy care. The ICU contained both manual and automatic sinks, many of which had an aerator. Concerns regarding aerators were discussed, but we did not strongly recommend their removal. According to the CDC guidelines for preventing health care-associated pneumonia, ''no recommendation can be made about the removal of faucet aerators from areas for immunocompetent patients (unresolved issue).''[10] Although *B cenocepacia* was not cultured directly from hospital water, its recovery from drains suggests that the organism was present either in the water or in contaminated products placed in sinks. Hospital tap water has been implicated in past outbreaks, but recovering outbreak organisms is often difficult, due to inadequate sampling or variations in pathogen load within the water system. Moreover, nosocomial waterborne pathogens, such as Bcc, have a strong association with water biofilms, and when water demand is increased, organisms can be dislodged from the biofilm and released into the water supply.[11]