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CONCISE COMMUNICATION

Control of an Outbreak of Carbapenem-Resistant *Acinetobacter baumannii* in Australia after Introduction of Environmental Cleaning with a Commercial Oxidizing Disinfectant

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In the midst of an outbreak, carbapenem-resistant *Acinetobacter baumannii* was grown from samples of multiple environmental sites in an intensive care unit. A commercial oxidizing disinfectant (potassium peroxomonosulphate 50%, sodium alkyl benzene sulphonate 15%, and sulphamic acid 5%) was introduced throughout the intensive care unit, and its use coincided with cessation of the outbreak.

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In 2006, an outbreak of carbapenem-resistant *Acinetobacter baumannii* (CR-AB) occurred in an intensive care unit (ICU) at a large teaching hospital in Australia. In this report, we describe the outbreak and its successful control after attention was placed specifically on environmental cleaning with a commercially available oxidizing disinfectant (Virkon S; potassium peroxomonosulphate 50%, sodium alkyl benzene sulphonate 15%, and sulphamic acid 5%).

METHODS

The outbreak occurred in a 19-bed long-stay ICU that has an occupancy of 550–670 patient-days per month. It is within a hospital that is a major referral center for trauma, burns, and hematologic transplantation.

This assessment occurred during January 2004–December 2008. Patients in the ICU who were infected with CR-AB were identified by culture of clinical specimens. Colonization was identified by routine twice-weekly culture of endotracheal aspirates on blood and MacConkey agar, performed for surveillance purposes.^{1,2} On days 3–5 of ICU hospitalization and upon discharge from the ICU, a rectal swab sample, nasal swab sample, and wound swab sample (if a wound was present) were also collected to determine whether carriage of CR-AB and/or other multidrug-resistant organism were present.

As part of the investigation into this outbreak, 137 environmental samples were collected for culture from May to August 2006. A moistened cotton swab was swabbed over the surface and plated onto MacConkey agar. RESULTS

The outbreak. CR-AB isolates were recovered from 5 patients in 2004 but no patient in 2005. On January 30, 2006, CR-AB was isolated from the respiratory tract of a patient in the ICU. In early March 2006, CR-AB was isolated from a second patient. Subsequently, as many as 5 new patients per week were found to be colonized with CR-AB (Figure). In all, 41 patients were found to be newly colonized or infected with CR-AB during the period March 1–August 17, 2006.

In May 2006, culturing of environmental specimens commenced. Of 137 environmental sites sampled for culture in the affected ICU, CR-AB was recovered from 11: a mattress, a vital signs monitor, horizontal surfaces at the patient's bedside, a computer keyboard and mouse, and a glucometer.

The CR-AB isolates of all 4 patients identified in March 2006 were found to have identical band patterns, implying genotypic relatedness.³ We did not determine whether subsequent isolates were related to these initial isolates.

Interventions. All patients identified as being colonized or infected with CR-AB were nursed in single rooms with contact isolation. In May 2006, the area within the ICU identified as being contaminated (that is, within which environmental specimens with positive culture results were collected) was closed for a 3-day period. During this time, the area was cleaned 3 times a day for 3 days with 1% neutral detergent water solution, followed by cleaning with 70% alcohol–impregnated wipes (contact time, >1 minute). Despite this intense cleaning regimen, 2 of 12 cultures of environmental samples from horizontal surfaces at the bedside yielded positive results.

A search for a new cleaning product was undertaken after it had been clearly demonstrated that the current and increased cleaning regimens were inadequate in removing CR-AB from the environment. A commercial oxidizing disinfectant (Virkon S; potassium peroxomonosulphate 50%, sodium alkyl benzene sulphonate 15%, and sulphamic acid 5%) was identified. This was introduced throughout the ICU on August 17, 2006, and replaced all other cleaning solutions in the ICU environment.

Follow-up. After the introduction of the new cleaning product, just one new patient was found to be colonized with CR-AB during the remainder of August 2006. In September 2006, 2 patients, and in October, 3 patients, were found to be infected or colonized with CR-AB. There were then no new cases until a single case on December 31, 2007. No cases were seen in 2008, despite ongoing surveillance consisting of twice-weekly cultures of endotracheal aspirates and rectal swab samples. Use of the commercial surface disinfectant has continued from August 17, 2006, to the present time.



FIGURE. Number of patients in the intensive care unit (ICU) colonized or infected with carbapenem-resistant *Acinetobacter baumannii*, January 2004–December 2008. The new surface disinfectant was used in the ICU from August 17, 2006. From that point on, just 6 new cases were detected in more than 2 years of follow-up.

Assessment of activity of the oxidizing disinfectant against biofilm formation by A. baumannii. A strain from a patient in the March 2006 outbreak was grown as static cultures at 37°C in Luria Bertani medium. Biofilms were established by placing a polyvinyl chloride (PVC) disk (diameter, 1 cm) in the culture medium. After incubation for 20 hours, the PVC disk was removed from the culture and washed extensively in phosphate—buffered saline (PBS) to remove unbound bacteria. Each PVC disk was then subjected to 1 of 2 treatments: (1) 10-minute incubation in PBS (control) or (2) 10-minute incubation in PBS containing 0.5% this oxidizing disinfectant. Biofilm formation was then assessed by staining with crystal violet. Quantification of viable cells was performed by physically removing bacteria from the PVC disk, and enumeration was performed by direct colony counting on agar plates.

The test strain formed a biofilm in this assay. Treatment with this oxidizing disinfectant was highly efficient and resulted in the killing of all viable cells within the biofilms.

DISCUSSION

In this report, we have described a discrete outbreak of infection with CR-AB. The initial response to the outbreak in our ICU was reinforcement of basic hand hygiene and contact isolation. Despite these efforts, no reduction in the number of cases was observed. Changes in antibiotic use appear unlikely to have contributed to this outbreak (data not shown). A "common source" environmental vehicle of *A. baumannii* infection was sought, but diverse environmental sources were found to be colonized. Despite intense environmental cleaning of affected areas within the ICU, the outbreak continued.

We found a temporal association between introduction of a commercial oxidizing disinfectant and termination of the outbreak. This disinfectant had not been used previously in the institution. What are potential explanations for the effectiveness of the oxidizing disinfectant that we used and the comparative ineffectiveness of our previous environmental cleaning? With most disinfectants, viable A. baumannii remain if contact times are short or diluted agents are used.⁴ The bactericidal effects of agents against A. baumannii are considerably reduced in the presence of organic material.⁵ These principles of decreased effectiveness if contact times are short or organic material is present may have contributed to the lack of effect of our prior cleaning regimen. Alternatively, the oxidizing disinfectant that we used may have had enhanced killing activity against A. baumannii. To our knowledge, the activity of this disinfectant against A. baumannii has not been evaluated previously. Production of biofilm by devicerelated A. baumannii strains has been shown to be substantial.⁶ We have shown that our outbreak strain produced significant amounts of biofilm and that when this biofilm was treated with this oxidizing disinfectant, all of the bacterial cells were killed. There is, therefore, a theoretical basis to our observation that use of this product was associated with reduction in the presumed environmental acquisition of A. baumannii by our ICU patients.

In conclusion, we observed resolution of an outbreak of CR-AB in an ICU. What are the limitations of our observations? Temporal associations may be misleading, because epidemics may resolve of their own accord. We did not audit cleaning effectiveness, hand hygiene compliance, and other potential factors that could have influenced a reduction in CR-AB. Finally, this oxidizing disinfectant has varied in its sporicidal and antimicrobial activities.⁷ Factors that contribute to prolonged environmental contamination with CR-AB clearly need further attention.⁸

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