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Letters to the Editor

Palivizumab use during respiratory syncytial virus outbreak in the neonatal intensive care unit

Madam,

We read with great interest the papers by Dizdar *et al.* and by O'Connell *et al.* about the use of palivizumab during a respiratory syncytial virus (RSV) outbreak in neonatal intensive care units (NICUs), suggesting that palivizumab administration might have a role in controlling RSV outbreak and recommended early administration of palivizumab to terminate transmission as quickly as possible.^{1,2} We recently experienced a similar outbreak in our NICU, and controlled it according to the suggestions made in this paper.

There were ten preterm (median gestational age: 29.3 weeks; range: 26.2–32 weeks; birth weight: 848–1520 g), two late preterm (>35 weeks' gestational age) and four term infants in the NICU when two term newborns with bronchopneumonia and respiratory insufficiency were admitted to the NICU isolation unit between 29 February 2012 and 12 March 2012. Polymerase chain reaction (PCR) screening including RSV (A, B), coronavirus (A, B, C, D, E, OC43, HKU1), parainfluenza (1, 2, 3, 4), rhinovirus (A, B, C), influenza (A, B), bocavirus (1, 2, 3, 4), metapneumovirus and enterovirus, revealed RSV type B infection in these two patients. Although patients with RSV were cared for in separate isolation rooms, another preterm infant who had recovered from respiratory distress syndrome developed further respiratory distress after a week. Nasopharyngeal secretions obtained from this infant also revealed RSV type B infection and we decided to screen the remaining 15 infants for RSV. None of the asymptomatic patients was RSV PCR positive. In order to prevent an escalating NICU outbreak, palivizumab prophylaxis was administered to nine preterm infants, all of whom were <32 weeks of gestational age at birth, and one patient who had a congenital heart disease at a dosage of 15 mg/kg, in addition to strict contact precautions. Patients with RSV bronchiolitis recovered after about 10 days and we did not observe any additional cases with RSV.

RSV infection was brought into the NICU by two patients with RSV bronchiolitis. Following this, one preterm patient, who was recovering from respiratory distress, developed RSV bronchiolitis. As NICUs like ours embrace a family-centred model for patient care, greater difficulties complying with effective infection control measures may emerge.³ We agree

with Dizdar *et al.* and O'Connell *et al.* that palivizumab prophylaxis may have a role in the control of RSV epidemics in the NICU.^{1,2} If we had not given palivizumab prophylaxis after detection of index cases, a larger RSV outbreak might have occurred in our NICU. After a few small RSV NICU outbreaks in Turkey, the Turkish Neonatal Society now recommends RSV prophylaxis for premature infants in the NICU who are already candidates for the prophylaxis programme as outpatients when at least three RSV-positive patients are present in the NICU. This recommendation is similar to the one reported by the Spanish Neonatal Society which suggests palivizumab prophylaxis for preterm infants and newborns with haemodynamically significant congenital heart disease when such outbreaks occur.⁴

Conflict of interest statement

None declared.

Funding sources

None.

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Available online 20 June 2012

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<http://dx.doi.org/10.1016/j.jhin.2012.05.011>

Does the discovery of biofilms on dry hospital environmental surfaces change the way we think about hospital disinfection?

Madam,

We would like to highlight the importance of the principal finding of Vickery *et al.*, that multidrug-resistant organisms can be found in biofilms formed on dry hospital surfaces despite cleaning and disinfection.¹ Biofilms are problematic in healthcare settings where they are thought to be responsible for 65% of nosocomial infections and are usually reported in relation to various indwelling medical devices and prostheses, water lines and tubing on endoscopes and on wounds.^{2,3} In these settings, biofilm persistence can be prolonged, periodically 'sloughing off' and releasing planktonic bacteria, which may act as an infection source. However, to our knowledge, Vickery *et al.* have provided the first report of biofilms on hard, dry hospital surfaces, which has important implications.

Organisms in biofilms exhibit an altered phenotype compared with corresponding planktonic cells, especially regarding growth, gene transcription, protein production, and intercellular interaction.² Biofilms can form on almost any biological or inanimate surface and have been identified in various industrial and medical settings.^{2,3} They constitute a protected mode of growth, allowing bacteria to survive in hostile environments conferring reduced susceptibility to dehydration, phagocytosis, ultraviolet light, metal toxicity, acid exposure and antimicrobial agents including antibiotics, disinfectants and germicides.^{2–4}

The finding of biofilms on dry hospital surfaces strengthens the importance of several recent studies. For example, Epistal *et al.* found that biofilm-forming strains of *Acinetobacter baumannii* survived longer on dry surfaces than non-biofilm-forming strains (36 vs 15 days, $P < 0.001$), concluding that the ability to form biofilms may contribute to its persistence in the hospital environment, increasing the probability of causing nosocomial infections and outbreaks.⁵ Also, recent data indicate that bacteria living in biofilms can be up to 1500 times less susceptible to antibacterial compounds than their corresponding planktonic bacteria.^{1,3} For example, Smith *et al.* grew biofilms of clinical isolates of methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas aeruginosa* on discs of materials found in the hospital environment and treated them with three commonly used hospital biocides.³ The biocides were ineffective at killing bacteria at the concentrations recommended for use which were considerably higher than the minimum bactericidal concentration (MBC) for the planktonic populations of both organisms. The

MBCs of all three biocides were found to be 10–1000-fold higher in biofilms than concentrations recommended for use. Following biocide treatment, up to 11% of cells in MRSA biofilms and 80% of cells in *P. aeruginosa* biofilms survived. This study suggests that although biocides may be effective against planktonic populations of bacteria, some biocides currently used in hospitals are ineffective against nosocomial pathogens growing as biofilms attached to surfaces.³

Could it be that we have missed or underestimated the importance of biofilms on dry hospital surfaces? This could be the mechanism by which reservoirs of vegetative bacterial pathogens persist within the hospital environment for such extended periods. It could also be part of the reason why disinfectants that are effective for the inactivation of planktonic bacteria in laboratory tests are not effective for the eradication of a considerably lower load of the same bacterial species from hospital surfaces. In support of this, we note that the biofilms identified by Vickery *et al.* were on surfaces that had been cleaned using detergent then disinfected using 500 ppm chlorine. The presence of biofilms on dry hospital surfaces could also interfere with attempts to recover microbes through environmental sampling. This could mean that an environmental reservoir of a pathogen remains undetected or that the concentration of contamination and degree of associated risk is underestimated.

Biofilms are clearly not the only reason for failures in hospital disinfection, given the difficulty in achieving adequate distribution and contact time using manual methods, but these findings may have implications for infection control practices within hospitals and on the choice of the appropriate disinfectants for hospital surfaces. This is particularly relevant because bacterial cells in aqueous planktonic phase remain the most common model for many microbiological studies including disinfectant testing. Future testing should consider the inclusion of biofilm models to ensure that the disinfectants tested are as effective in the 'real world' as in laboratory tests.²

Conflict of interest statement

J.A.O. and S.Y. are employed by Bioquell (UK) Ltd.

Funding sources

Bioquell (UK) Ltd.

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