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Major article

Impact of chlorhexidine-impregnated washcloths on reducing incidence of vancomycin-resistant enterococci colonization in hematology–oncology patients

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Background: Daily skin cleansing with washcloths impregnated with chlorhexidine gluconate (CHG) of patients in intensive care unit is associated with reduction in incidence of vancomycin-resistant Enterococci (VRE) acquisition. This study describes the impact on incidence of VRE colonization after the implementation of daily skin cleansing with 2% CHG-impregnated washcloths in hematology–oncology patients.

Methods: In this before-and-after study, we compared the incidence rate of VRE colonization during the baseline period (where routine soap-and-water bathing was used) with the intervention period where patients were cleansed with 2% CHG-impregnated washcloths.

Results: Acquisition of VRE decreased from 7.8% in the baseline to 3.8% in the intervention period (relative risk, 0.48, 95% confidence interval [CI], 0.21-1.09; $P = .07$). The crude relative rate of acquisition during the intervention period compared with the baseline period was 0.53 (95% CI, 0.23-1.23; $P = .13$). Patients who had been a roommate of a patient subsequently found to have VRE were at a significantly increased risk for acquiring VRE (hazard ratio, 18.8, 95% CI, 5.37-66.15; $P < .001$). However, patients admitted to the same bed number of previously known VRE-colonized patient were not at increased risk of VRE acquisition (hazard ratio, 0.37, 95% CI, 0.11-1.22; $P = .10$).

Conclusion: We did not observe a statistically significant reduction in the rate of VRE colonization in association with the use of 2% CHG-impregnated washcloths among hematology–oncology patients.

Vancomycin-resistant enterococci (VRE) was first reported in Europe in 1986[1] and in Australia in 1994.[2] Since then, VRE, predominantly *Enterococcus faecium* with the *vanB* gene, has become endemic in many hospitals in Australia.[3] Approximately 12% of patients colonized with VRE develop clinical infection.[4,5] VRE infection prolongs hospital stay, increases the cost of care,[6] and is associated with increased morbidity and mortality.[7] Rates of VRE colonization have continually increased in Melbourne hospitals, and both infection and colonization occurs predominantly in immunocompromised patient populations.[8]

Chlorhexidine is a broad spectrum antiseptic agent active against both gram-positive and gram-negative bacteria, and has been successfully assessed as an effective skin antiseptic since the early 1980s.[9,10] Chlorhexidine, as an active antiseptic can be used directly as solution, or as an ingredient in soaps, gels, or impregnated in cloths. Published studies suggest that the routine use of

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PB and SK contributed equally to this article.

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chlorhexidine-gluconate (CHG) is associated with a reduction in VRE acquisition in patients in intensive care units (ICUs).[11,12]

VRE has become endemic in many hospitals worldwide, despite the recommendations to identify, isolate, and use contact precautions. In this study, we aimed to evaluate the impact of daily use of 2% CHG-impregnated washcloths on the incidence of VRE colonization among hematology–oncology patients.

METHODS

We conducted a before-and-after study to assess the impact of 2% CHG washcloths in preventing acquisition of VRE in hematology–oncology patients at the Alfred hospital. The Alfred hospital is a 427 bed, major tertiary teaching hospital in Melbourne, Australia. The hematology–oncology ward has 34 beds and includes patients with hematologic and solid organ malignancies, including allogeneic and autologous bone marrow transplantation.

We compared incident VRE colonization in patients admitted between March and June 2010 (baseline period) and from July to October 2010 (intervention period) following the introduction of the daily use of CHG-impregnated washcloths (Clinell Washcloths, GAMA Healthcare Ltd, London, England). During the intervention period, all patients in the hematology–oncology ward were provided each day with a pack containing 4 washcloths impregnated with 2% CHG. Patients were given instructions for self-application of the washcloths as the only form of bathing or for application after showering with soap and water and drying. Washcloths could be optionally heated for 15 seconds in a dedicated microwave oven. Patients were instructed to use the washcloth according to manufacturer's instructions with the first cloth on the face, neck, and both arms (avoiding contact with the eyes); the second cloth on the axilla, chest, and back; the third cloth on the legs; and fourth cloth on the groin and perineum. Patients with extensive open wounds or who were known to be allergic to CHG were excluded from the intervention.

The primary outcome of the study was incident VRE colonization rates. Rectal swabs were taken on all new admissions to the ward and thereafter weekly during their inpatient stay. A new VRE acquisition was defined as a patient not known to have VRE before admission and not positive for VRE on rectal swab taken at admission who subsequently became positive on subsequent screening swabs after admission. Secondary outcome measures included VRE isolates from clinical sites, methicillin resistant *Staphylococcus aureus* (MRSA) isolates from clinical sites, and central line associated blood stream infections (CLABSI).

To examine the potential confounding effect of other VRE-colonized patients on the ward, we considered 2 covariates; the VRE status of patients who had previously occupied the same bed number (same bed number may represent the same bed or same bed space) earlier occupied by a VRE-positive patient, and the VRE status of patients who occupied the same room as a patient who was later diagnosed as VRE positive. In our hospital, patients are placed in contact precaution in single rooms following the result of positive screening swab or clinical isolate of VRE. However, patients colonized with VRE may have shared a room with patients not colonized with VRE until diagnosis of VRE via screening swabs or in clinical specimens. Patients could only occupy the same bed or same number bed after the terminal cleaning of the room and bed with sodium hypochlorite after the discharge of a VRE-positive patient.

Rectal swabs were taken either by the patients themselves following instruction or by nursing staff. The swabs were then plated onto bile aesculin media (BBL Enterococcosel agar, Cockeysville, MD) with vancomycin 6 μg/mL and incubated at 37°C for up to 72 hours. Enterococci species were identified using the VITEK-2 system (bioMérieux, Marcy l'Etoile, France). Polymerase chain reaction was used to detect the *vanA* or *vanB* genes as described previously.[13] VRE colonization was defined if an isolate of *E. faecalis* or *E. faecium* with *vanA* or *vanB* gene was detected.

Policies to prevent transmission of VRE at our hospital include isolation of patients with known VRE infection or colonization in single rooms with dedicated bathrooms. Gloves, but not gowns, are stipulated for entry into patients' rooms. Infection control signs are placed on doors notifying staff of the isolation requirements. Rooms of patients with known VRE colonization, infection, or colonization with other multiresistant organisms are cleaned daily using 1,000 ppm sodium hypochlorite solution. Terminal cleaning of the rooms was also performed with sodium hypochlorite (1,000 ppm) following discharge of patients known to be colonized or infected with VRE. Routine cleaning practices for the rooms of nonisolated patients included daily neutral detergent cleaning.

Before the study period, other interventions to reduce bloodstream infection had been introduced. This included the use of chlorhexidine-impregnated foam dressings around central venous line exit sites (BioPatch, Ethicon, Johnson & Johnson, North Ryde NSW, Australia) in 2007, and ongoing audits of central venous line nursing practice (since May 2007). Hand hygiene compliance had been monitored in the ward and was known to be moderately high during the study period (78% in August 2010).

When designing this study, we had preliminary data suggesting 12% of patients became colonized with VRE during their hospital stay, and that with approximately 100 admissions each month we would have 80% power to detect a 60% reduction in the risk of acquisition with a 3-month baseline and 3-month postintervention period. The intervention effect was measured in terms of relative risk, hazard ratio (HR), and incidence rate ratio. The relative hazard of colonization was calculated using a Cox proportional hazards model, adjusted for VRE status of patients that had previously occupied the same bed as the index patient, and the colonization status of other patients in the same room. Statistical significance was tested using χ^2 test for categorical variable and Wilcoxon rank sum test or t test for continuous variables. A P value of $<.05$ was considered statistically significant. Statistical analyses were performed using STATA (version 10, 2007, Stata Corp, College Station, TX).

Ethical approval for the study was granted from the Alfred Health Human Research Ethics Committee and Monash University Human Research Ethics Committee.

RESULTS

During the study period, a total of 479 patients were found to be negative on initial VRE screening of 753 total admissions. Of these patients, 40 were excluded in the data analysis because they had a previous history of VRE carriage, leaving 439 patients (baseline period n = 229; intervention period n = 210) available for further analysis. Patient characteristics are shown in Table 1.

During the study period, a total of 26 (5.4%) of all uncolonized patients acquired VRE. During the baseline period, 18 (7.8%) previously uncolonized patients acquired VRE, whereas during the intervention period, 8 (3.8%) patients were found to be newly colonized (relative risk, 0.48, 95% confidence interval [CI], 0.21-1.09; $P = .07$). The incidence rate in baseline and intervention period is shown in Table 2.

The relative hazard of VRE acquisition during the intervention period compared with the baseline period was 0.53 (95% CI, 0.23-1.23, $P = .13$). Patients sharing the same room with another patient later found to be VRE colonized were at increased risk of colonization (33.3% vs 5.15%; $P = .003$); however, the patients who were cared for in the same bed number previously occupied by VRE