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Intranasal octenidine and universal antiseptic bathing reduce methicillin-resistant *Staphylococcus aureus* (MRSA) prevalence in extended care facilities

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Abstract

Intranasal octenidine, an antiseptic alternative to mupirocin, can be used for methicillin-resistant *Staphylococcus aureus* (MRSA) decolonisation in the prevention of nosocomial transmission. A controlled before–after study was conducted in three extended-care hospitals in Singapore. All inpatients with >48 h stay were screened for MRSA colonisation in mid-2015(pre-intervention) and mid-2016(post-intervention). Hospital A: universal daily chlorhexidine bathing throughout 2015 and 2016, with intranasal octenidine for MRSA-colonisers in 2016. Hospital B: universal daily octenidine bathing and intranasal octenidine for MRSA-colonisers in 2016. Hospital C: no intervention. In 2015, MRSA prevalence was similar among the hospitals (Hospital A: 38.5%, Hospital B: 48.1%, Hospital C: 43.4%, $P = 0.288$). From 2015 to 2016, MRSA prevalence reduced by 58% in Hospital A (Adj OR 0.42, 95% CI 0.20–0.89) and 43% in Hospital B (Adj OR 0.57, 95% CI 0.39–0.84), but remained similar in Hospital C (Adj OR 1.19, 95% CI 0.60–2.33), after adjusting for age, gender, comorbidities, prior MRSA carriage, prior antibiotics exposure and length of hospital stay. Compared with the change in MRSA prevalence from 2015 to 2016 in Hospital C, MRSA prevalence declined substantially in Hospital A (Adj OR 0.35, 95% CI 0.13–0.97) and Hospital B (Adj OR 0.48, 95% CI 0.22–1.03). Topical intranasal octenidine, coupled with universal daily antiseptic bathing, can reduce MRSA colonisation in extended-care facilities.

Introduction

Methicillin-resistant *Staphylococcus aureus* (MRSA) is one of the most common healthcare-associated drug-resistant organisms in the world, particularly in Asia [1]. Decolonisation of MRSA carriage has been found to be effective in preventing nosocomial transmission of MRSA in various healthcare settings including intensive care units (ICUs), hospital wards and nursing homes [2]. MRSA decolonisation guidelines have included whole-body bathing with an antiseptic and topical intranasal treatment with mupirocin [3]. Intranasal topical mupirocin has been shown to be effective in eradicating nasal MRSA carriage, but the emergence of mupirocin resistance has been associated with decolonisation failure [3]. Antiseptic agents including povidone-iodine and octenidine dihydrochloride have been used as alternatives for nasal decolonisation.

Octenidine is a cationic biguanide that is structurally similar to chlorhexidine but has a broader antibacterial activity spectrum towards Gram-positive bacteria [4]. To date, there is a lack of evidence of emergence of tolerant clones to octenidine. Although octenidine has the potential to be efficacious for MRSA decolonisation, only a handful of studies have investigated the clinical effectiveness of topical intranasal octenidine [5, 6]. Universal decolonisation with a 5-day regimen of octenidine nasal gel and daily bathing with octenidine wash cloths in medical ICUs have been found to be effective in decreasing ICU-acquired MRSA clinical infections (incidence rate ratio 0.58; 95% CI 0.41–0.82) [6].

To date, the effectiveness of octenidine has yet to be investigated in extended care facilities where MRSA acquisition has been found to be four times as high as in nursing homes [7] and thrice that of affiliated acute hospitals [8]. Care delivery at extended care facilities differ from acute hospitals and present with unique infection prevention and control challenges [9]. Screening and contact precautions that are commonly implemented in acute hospitals might not be practicable in extended care facilities where treatment plans involve intensive rehabilitation and ambulation of patients.

Therefore, our study aims to assess for the effect of topical intranasal octenidine with daily universal octenidine or chlorhexidine bathing in reducing MRSA prevalence in extended care facilities.

Methods

Study design

We conducted a controlled before–after study in three extended care facilities in a healthcare network in Singapore, comparing the prevalence of MRSA colonisation in June–July 2014 (pre-intervention period), June–July 2015 (pre-intervention period) and June–July 2016 (post-intervention period). This study is part of a 3-year period prevalence study on MRSA colonisation in the three hospitals, conducted from June to July, for three consecutive years (2014–2016).

Participants

All inpatients with >48 h stay in the hospitals during June–July 2014, June–July 2015 and June–July 2016, respectively, were included in the study.

Study setting

Hospital A was a 100-bed rehabilitation centre, specialised in stroke, brain injury, spinal and musculoskeletal conditions. Its rooms comprised of four-bedded (20%), six-bedded (30%) and eight-bedded (49%) configurations, with only one single room which served as an isolation room. Hospital B was a 360-bed community hospital specialised in caring for patients with stroke and debilitating medical conditions. Two per cent were single rooms including an isolation room, with the remaining rooms comprising four (2%), five (7%), six (21%), eight (33%), 10 (31%) and 12 (3%) beds. Hospital C was a 116-bed community hospital focused on care for stroke and subacute medical conditions. The majority were cubicles of eight (41%) and 10 beds (52%), with six (5%) double and four (2%) single rooms which also served as isolation rooms.

Interventions

In Hospital A, universal daily whole-body chlorhexidine bathing (chlorhexidine gluconate 4%; Microshield∗4, Johnson & Johnson, Australia) has been ongoing since 2014 and continued throughout 2015 and 2016. A 5-day regimen of intranasal octenidine gel (octenidine hydrochloride, Octenisan® md nasal gel, Schülke & Mayr GmbH, Germany) applied to MRSA-colonisers from the day of admission was instituted from March to July 2016. In Hospital B, universal daily octenidine bathing (octenidine hydrochloride, Octenisan® wash lotion, Schülke & Mayr GmbH, Germany) with a 5-day regimen of intranasal octenidine (octenidine hydrochloride, Octenisan® md nasal gel, Schülke & Mayr GmbH, Germany) from the day of admission for MRSA-colonisers was implemented from March to July 2016. Prior to March 2016, Hospital B had not used any antiseptic products for MRSA decolonisation. Neither antiseptic bathing nor intranasal octenidine was administered in Hospital C throughout the study period.

There was no change to the other infection precautions undertaken by the hospitals, including infection prevention policies and practices, between January 2014 and December 2016. All three hospitals' infection prevention policies included the institution of contact precautions for MRSA-colonised patients throughout the hospital stay. Apart from the routine hand hygiene promotional activities, there was no large-scale hand hygiene campaigns conducted during that period.

Infection-related outcomes

The primary outcome of interest was the prevalence of MRSA colonisation determined by the period prevalence screening in June–July 2014, June–July 2015 and June–July 2016 at the three hospitals.

Culturing and typing

Separate nasal, axillary and groin swabs were taken by trained research nurses in a standardised manner with swabs moistened with two drops of sterile saline rolled five times in each nostril and 10 times over the skin of the axilla and groin. The samples were processed and inoculated in selective chromogenic agar media (*Brilliance* MRSA 2 agar, Oxoid, UK) by a common research laboratory. Culture plates were incubated aerobically at 35–37 °C for 18–28 h, and read by the same medical technologist who was blinded to the hospital from which the samples were collected. Positive cultures were subsequently referred to matrix-assisted laser desorption ionisation-time of flight (MALDI-TOF) mass spectrometry and cefoxitin disk diffusion test for confirmation of microbial identity and methicillin resistance.

Epidemiological and clinical data

Patients' epidemiological and clinical data were obtained from the review of a combination of electronic health records and hard copy inpatient clinical records. Data collected included demographics (age and gender), pre-existing medical conditions, history of prior MRSA carriage in the preceding 12 months, prior antibiotics exposure in the preceding 12 months and the length of stay in the extended care facility prior to screening for MRSA by the study. We defined pre-existing medical conditions as having a diagnosis of diabetes with or without complications; cardiovascular disease namely coronary artery disease or congestive heart failure; liver disease of any severity; moderate-to-severe renal disease; solid malignant tumour, leukaemia, lymphoma or any metastasis; central nervous system disease of cerebrovascular disease or dementia; and chronic obstructive pulmonary disease. The conditions were computed into the Charlson's comorbidity index (CCI) [10] and categorised into ⩽5 and >5, representing good and poor chronic health status. For prior exposure to antibiotics in the preceding 12 months, data on exposures to the various classes of antibiotics were collected.

Statistical methods

First, we used appropriate descriptive statistics to summarise the patients' demographics, their health status, prior MRSA carriage and antibiotics exposure, and length of stay, by hospital. Second, we compared the prevalence of MRSA colonisation in the pre-intervention and post-intervention periods, by hospital. Univariate analysis was carried out using χ^2 test for categorical variables, and Kruskal–Wallis test for continuous variables. Next, we explored relationships between the hospital, various patient characteristics and exposures, and MRSA colonisation, using logistic regression models. We then constructed multivariable logistic regression models, accounting for potential confounding. In the initial multivariable logistic regression model, we included variables decided *a priori* as factors associated with MRSA colonisation based on prior knowledge from literature review. Then, we ran a forward selection algorithm, adding factors

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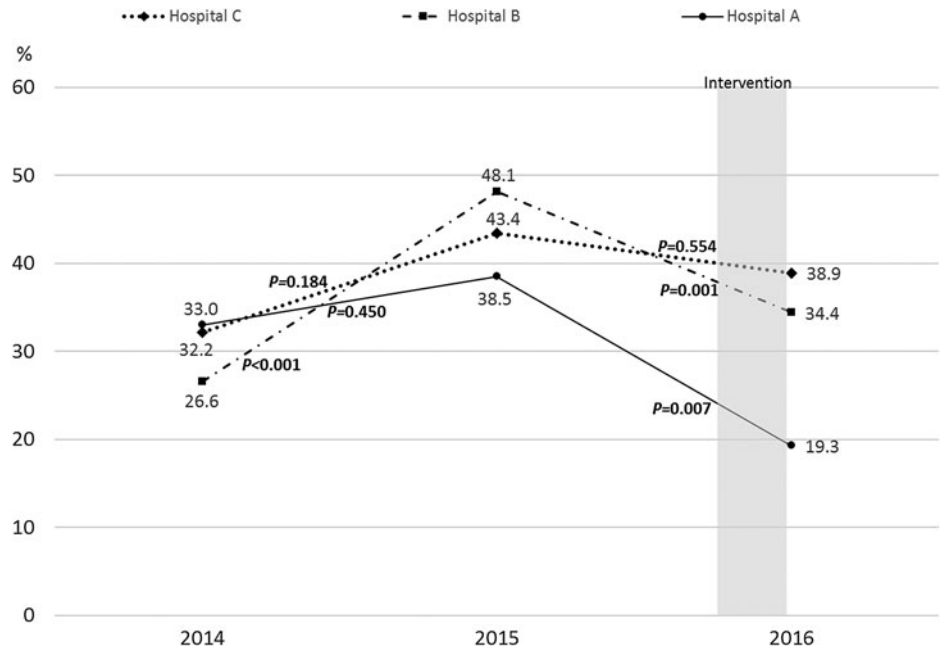

Fig. 1. MRSA prevalence in Hospitals A, B and C, in 2014, 2015 and 2016.

Table 1. Characteristics of study participants in Hospitals A, B and C, in 2015 and 2016

Factor	Hospital A (*N* = 161)	Hospital B (*N* = 570)	Hospital C (*N* = 166)	*P*-value
Age, mean years (s.d.)	60.9 (13.5)	72.6 (11.0)	76.9 (10.2)	<0.001
Male gender, *N* (%)	105 (65.2%)	271 (47.5%)	78 (47.0%)	<0.001
Charlson's comorbidity index >5, *N* (%)	12 (7.5%)	73 (12.8%)	14 (8.4%)	0.079
Prior MRSA carriage in preceding 12 months, *N* (%)	23 (14.3%)	85 (14.9%)	34 (20.5%)	0.188
Prior antibiotics exposure in preceding 12 months, *N* (%)	103 (64.0)	370 (64.9)	102 (61.4)	0.714
Length of stay in hospital, median days (IQR)	16 (8–30)	26 (14–43)	18 (12–30)	<0.001

IQR, interquartile range; *N*, number; s.d., standard deviation.

that met a *P* value of <0.05 for statistical significance without introducing collinear variables. Statistical interactions between hospitals and year of MRSA screening were explored and the product term was included in the model. Finally, to ensure that all potential confounders were captured, variables not selected were manually added back one-by-one and kept in the model if there was more than 20% change in the coefficients of existing predictors. Effect measure modification due to hospital was further assessed.

All analyses were performed using Stata version 13 (StataCorp 2013, College Station, Texas, USA).

Results

A total of 1255 patients were screened for MRSA (358 in 2014, 462 in 2015 and 435 in 2016). The participation rate was 90%. The remaining had refused swabbing. MRSA prevalence was similar among the hospitals in 2014 (Hospital A: 33.0%, Hospital B: 26.6%, Hospital C: 32.2%, $P = 0.453$) and 2015 (Hospital A: 38.5%, Hospital B: 48.1%, Hospital C: 43.4%, $P = 0.288$) (Fig. 1). From 2015 to 2016, MRSA prevalence declined significantly in Hospital A (2015: 38.5%, 2016: 19.3%, $P = 0.007$) and Hospital B (2015: 48.1%, 2016: 34.4%, $P = 0.001$), but remained similar in Hospital C (2015: 43.4%, 2016: 38.9%, $P = 0.554$) (Fig. 1).

MRSA colonisation in the nares, as well as on the axilla and groin, of patients declined significantly in Hospital A (nares 2015: 20.8%, 2016: 6.0%, $P = 0.006$; axilla and groin 2015: 28.2%, 2016: 14.5%, $P = 0.033$) and Hospital B (nares 2015: 28.6%, 2016: 17.2%, $P = 0.001$; axilla and groin 2015: 38.6%, 2016: 22.9%, $P < 0.001$), but not in Hospital C (nares 2015: 23.7%, 2016: 20.5%, $P = 0.618$; axilla and groin 2015: 32.9%, 2016: 28.9%, $P = 0.577$).

In years 2015 and 2016, Hospital A had younger (mean age (standard deviation) in years, A: 60.9 (13.5), B: 72.6 (11.0), C: 76.9 (10.2), $P < 0.001$) and more male (A: 65.2%, B: 47.5%, C: 47.0%, $P < 0.001$) patients than the other hospitals (Table 1). The length of stay was the longest in Hospital B (median days (interquartile range), A: 16 (8–30), B: 26 (14–43), C: 18 (12–30), $P < 0.001$). However, there was no difference between the hospitals in the proportion of patients with CCI > 5 (A: 7.5%, B: 12.8%, C: 8.4%, $P = 0.079$), prior MRSA carriage in the preceding 12 months (A: 14.3%, B: 14.9%, C: 20.5%, $P = 0.188$) and prior antibiotics exposure in the preceding 12 months (A: 64.0%, B: 64.9%, C: 61.4%, $P = 0.714$). There were no significant differences in the colonisation pressures in the respective hospitals between

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Table 2. Multivariable analysis of factors associated with MRSA colonisation in Hospitals A, B and C, in 2015 and 2016

Factor	OR	(95% CI)	*P*-value
Age (years)	1.02	(1.00–1.03)	0.015
Male gender	2.14	(1.57–2.90)	<0.001
Charlson's comorbidity index >5	1.79	(1.13–2.84)	0.013
Prior MRSA carriage in preceding 12 months	3.62	(2.41–5.44)	<0.001
Prior antibiotics exposure in preceding 12 months	1.35	(0.98–1.87)	0.070
Length of stay in hospital (days)	1.01	(1.00–1.01)	0.001
Year 2016 (*vs.* 2015)	1.19	(0.60–2.33)	0.621
Hospital			
Hospital B (*vs.* Hospital C)	1.47	(0.84–2.57)	0.176
Hospital A (*vs.* Hospital C)	1.07	(0.52–2.21)	0.846
Interaction between year and Hospital B	0.48	(0.22–1.03)	0.060
Interaction between year and Hospital A	0.35	(0.13–0.97)	0.044

CI, confidence interval; OR, odds ratio.

the pre-intervention and post-intervention periods. Known MRSA colonisers among inpatients prior to the period prevalence screening by the study in Hospital A was 18.0% in 2015 and 10.8% in 2016 ($P = 0.198$), in Hospital B was 15.3% in 2015 and 18.7% in 2016 ($P = 0.274$), and in Hospital C was 26.3% in 2015 and 25.6% in 2016 ($P = 0.911$).

In years 2015 and 2016, after adjusting for age, gender, CCI > 5, prior MRSA carriage in the preceding 12 months, prior antibiotics exposure in the preceding 12 months and length of stay in the hospital prior to MRSA screening, there were significant interactions between the hospitals and the year of screening (Table 2).

From 2015 to 2016, declines in MRSA prevalence were observed in all three hospitals in the unadjusted analysis. After adjusting for age, gender, CCI > 5, prior MRSA carriage in the preceding 12 months, prior antibiotics exposure in the preceding 12 months and length of stay in the hospital prior to MRSA screening, MRSA prevalence reduced by 58% in Hospital A (Adj OR 0.42, 95% CI 0.20–0.89) and 43% in Hospital B (Adj OR 0.57, 95% CI 0.39–0.84), but remained similar in Hospital C (Adj OR 1.19, 95% CI 0.60–2.33) (Table 3).

The joint effect (simultaneous influence) of Hospital A and Year 2016 on the reduction of MRSA prevalence (Adj OR 0.45, 95% CI 0.20–0.99, $P = 0.048$) deviated substantially from the sum of the individual effects of Hospital A (Adj OR 1.07, 95% CI 0.52–2.21) and Year 2016 (Adj OR 1.19, 95% CI 0.60–2.33) (Fig. 2).

Except for one patient who discontinued the application of intranasal octenidine after developing mild periorbital swelling that was eventually assessed not to be an allergy attributable to the antiseptic product, all patients had complied with the interventions implemented at the respective hospitals. The decline in MRSA prevalence in Hospital A from 2015 to 2016 was significantly greater than the change in MRSA prevalence from 2015 to 2016 in Hospital C (Adj OR 0.35, 95% CI 0.13–0.97) (Table 2). The decline in MRSA prevalence in Hospital B was also substantial (Adj OR 0.48, 95% CI 0.22–1.03), when compared with the change in MRSA prevalence in Hospital C. However, the

Table 3. Unadjusted and adjusted analyses of change in MRSA colonisation between 2015 and 2016 in Hospitals A, B and C

	Hospital C		Hospital B			Hospital A		
MRSA prevalence	OR	(95% CI)	OR	(95% CI)	*P*-interaction[a]	OR	(95% CI)	*P*-interaction[a]
Unadjusted analysis								
2015	1.00	Reference	1.00	Reference	0.289	1.00	Reference	0.108
2016	0.83	(0.45–1.54)	0.57	(0.40–0.79)		0.38	(0.19–0.78)	
Adjusted analysis[b]								
2015	1.00	Reference	1.00	Reference	0.060	1.00	Reference	0.044
2016	1.19	(0.60–2.33)	0.57	(0.39–0.84)		0.42	(0.20–0.89)	

CI, confidence interval; OR, odds ratio.
[a]Multiplicative scale.
[b]Adjusted for age, gender, Charlson's comorbidity index >5, prior MRSA carriage in preceding 12 months, prior antibiotics exposure in preceding 12 months, length of hospital stay prior to MRSA screening.

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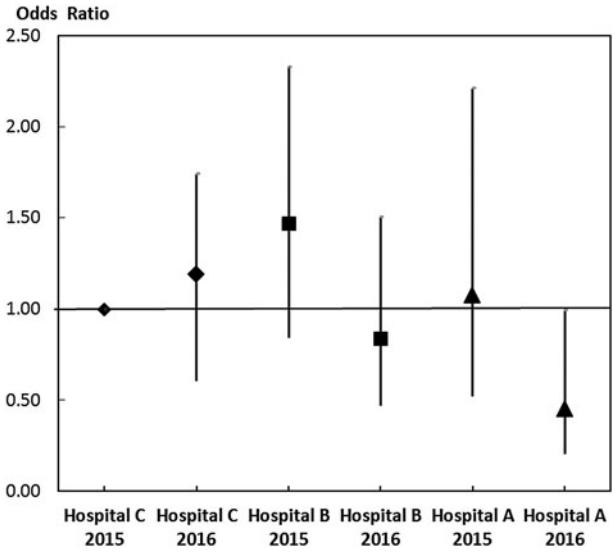

Fig. 2. Joint effects* (simultaneous influences) of Hospitals A, B and C, and years 2015 and 2016, respectively, on the prevalence of MRSA colonisation
*adjusted for age, gender, Charlson's comorbidity index >5, prior MRSA carriage in preceding 12 months, prior antibiotics exposure in preceding 12 months, length of hospital stay prior to MRSA screening. **Prevalence of MRSA colonization in Hospital C in 2015 served as the reference.

decline in MRSA prevalence from 2015 to 2016 in Hospital A was not statistically different from the decline observed in Hospital B (Adj OR 0.73, 95% CI 0.31–1.71), suggesting similar effectiveness in the interventions implemented in the two hospitals.

Discussion

This is the first clinical study assessing the effectiveness of topical intranasal octenidine and universal antiseptic bathing with chlorhexidine or octenidine on the reduction of MRSA prevalence in extended care facilities. The reduction in the prevalence of MRSA colonisation by 43–58% suggest the effectiveness of intranasal octenidine on decolonisation of MRSA carriage and nosocomial transmission in extended care facilities. The decline in MRSA colonisation of 58% in Hospital A from 2015 (chlorhexidine bathing) to 2016 (chlorhexidine bathing and intranasal octenidine) was similar to the 60% reduction in multidrug-resistant organisms reported in another study involving universal chlorhexidine bathing and intranasal povidone-iodine [11].

Our study found that a 5-day regimen of intranasal octenidine for MRSA colonisers and daily antiseptic bathing with chlorhexidine or octenidine universally for all inpatients in the general wards of extended care facilities can reduce MRSA prevalence in such healthcare settings. Using a modified microbroth dilution method, adhering to the Clinical and Laboratory Standards Institute's guidelines, fresh colonies of MRSA were used to determine the minimum inhibitory concentration (MIC) levels to chlorhexidine and octenidine for the range of susceptibility testing from 0.125 to 8.0 mg/l. Each isolate was tested in triplicates and incubated at 37 °C for 16–20 h. MRSA remained susceptible to chlorhexidine and octenidine throughout 2015 (pre-intervention) and 2016 (post-intervention), with none of the MRSA isolates having MIC levels of >4 mg/l to chlorhexidine or >2 mg/l to octenidine. Susceptibilities to chlorhexidine were similar in 2015 (MIC = 4, 82.9%; MIC = 2, 16.5%; MIC = 1, 0.6%) and 2016 (MIC = 4, 86.4%; MIC = 2, 13.1%; MIC = 1, 0.6%), whilst susceptibilities to octenidine differed slightly between 2015 (MIC = 2, 7.7%; MIC = 1, 92.3%) and 2016 (MIC = 2, 13.1%; MIC = 1, 86.4%; MIC = 0.5, 0.6%). Our observations that gender, prior antibiotics exposure and duration of hospitalisation stay increased the risk of MRSA colonisation were consistent with the findings reported in other studies [12–16].

Strengths and limitations

Our study has several strengths. It had a high participation rate of 90%, rendering any selection or non-participation bias very unlikely. There was also no difference in the age (mean age in participants 71.2 years and non-participants 71.8 years, $P = 0.584$) and gender (proportion of males in participants 50.6% and non-participants 45.9%, $P = 0.219$) distributions between those who participated and did not participate in the study in 2015 and 2016. Furthermore, standardised protocols were used for sample collection, sample processing and testing by a single research laboratory, with further confirmation of MRSA colonies with MALDI-TOF. Hence, any potential measurement error was likely to be minimal. The blinded microbiological evaluation of the samples negated the possibility of detection bias. However, our study could be limited by the inability to adjust for unknown confounders that were not measured. Nonetheless, we attempted to adjust for some of the major risk factors for MRSA colonisation in the multivariable analyses. The study is also limited by the period prevalence assessments of MRSA colonisation and the lack of serial measurements of the same patient over time. Follow-up longitudinal studies are necessary to assess for the incidence and acquisition of MRSA infections.

As there was no change in infection prevention and control policies and strategies between the pre-intervention and post-intervention periods, changes in MRSA prevalence were likely to be attributable to the new interventions implemented by the study. Any change due to time would have been observed in the control hospital, Hospital C, in which no new intervention was implemented. The observation that the declines in MRSA prevalence from 2015 to 2016 in both Hospitals A and B were significantly greater than the change in MRSA prevalence over the same period in Hospital C further supports the conclusion on the effectiveness of the interventions on MRSA reduction.

Generalisability

The observation that the prevalence of MRSA colonisation in the extended care facilities (38.5–48.1%) in this study being similar to the prevalence rates of MRSA colonisation reported in long-term care settings (20–50%) and higher than in acute hospitals (5–10%) in the USA [17] suggests similarities among extended and long-term care facilities worldwide. As such, our findings can be generalised to similar high prevalence healthcare settings internationally. Topical octenidine nasal gel could serve as an alternative to intranasal mupirocin ointment for MRSA decolonisation in areas with a high prevalence of high-level resistance to mupirocin in MRSA, such as Singapore [18].

Conclusion

Topical intranasal octenidine coupled with universal daily chlorhexidine or octenidine bathing can reduce the prevalence of MRSA colonisation in extended care facilities. Intranasal octenidine can be used in settings with high prevalence of mupirocin

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resistance for MRSA decolonisation for the prevention of nosocomial transmission. Longitudinal studies should be conducted to assess for its effectiveness in the reduction of acquisition of MRSA colonisation and infection.

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Conflict of interest. None.

Ethical standards. Ethics approval was obtained from the National Healthcare Group's Domain Specific Review Board (NHG DSRB Ref 2013/00965).

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