scape of conditions (ie, efficacy and compliance) under which preoperative bathing may be economically favorable. Describing the parameters that our model was most sensitive to can help aid the direction of future clinical research. Future studies that evaluate both the local compliance to and efficacy of chlorhexidine bathing with a nonwoven fiber cloth are needed and will allow decision makers to determine the potential value of preoperative bathing in their facilities.

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Effect of Intranasal Mupirocin and Chlorhexidine Body Wash on Decolonization of Community-Associated Methicillin-Resistant *Staphylococcus aureus*

To the Editor—Community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infection first emerged in early 1990s in Australia.[1] Most patients with CA-MRSA infection present with mild skin and soft-tissue infection, but some patients may present severe clinical manifestations, such as necrotizing pneumonia, severe sepsis, or necrotizing fasciitis, resulting in fatalities.[2-5]

In Hong Kong, CA-MRSA infection has been a notifiable infectious disease since January 2007. A case of CA-MRSA infection is defined as an occurrence of clinically compatible illness with isolation of MRSA from any clinical specimen with the following genetic characteristics: presence of staphylococcal cassette chromosome *mec* (SCC*mec*) gene type IV or V and Panton-Valentine leukocidin gene.

We telephone-interviewed all CA-MRSA infection case patients, using a standardized questionnaire to obtain clinical and relevant exposure history. We offered empirical decolonization therapy (5-day course of twice-daily mupirocin nasal ointment 2% and daily body wash using 4% chlorhexidine gluconate) to all patients and their home contacts. Before the therapy, nasal and axilla swabs were taken from the patients and their home contacts to screen for CA-MRSA colonization. For CA-MRSA carriers identified during initial screening, nasal and/or axilla swabs were collected 2 weeks after the therapy was finished.

Studies on the effectiveness of decolonization therapy for MRSA carriage in healthcare settings have been widely reported.[6] However, studies specific to CA-MRSA decolonization have seldom been reported. We carried out a retrospective review of CA-MRSA infections in Hong Kong to measure the percentage of CA-MRSA carriers who could be successfully decolonized and the associated factors.

We reviewed case records of all patients with CA-MRSA infection reported from 2007 to 2009. The information we retrieved included demographic characteristics, past health, relevant exposure history, SCC*mec*-typing results of the MRSA isolates of the patients, and screening-swab results before and after decolonization therapy.

We defined a CA-MRSA carrier as a person with a screening swab positive for CA-MRSA before decolonization therapy. Successful decolonization was defined as microbiological clearance of CA-MRSA at the carriage site after decolonization therapy. Percentage of successful decolonization was the percentage of carriers that had screening swabs negative for

TABLE 1. Results of Bivariate Analysis for Factors Affecting the Effect of Decolonization Therapy among 101 CA-MRSA Carriers, Hong Kong, 2007–2009

Predictor variables	Percentage of decolonization failure		
	Patients with predictor variable	Other patients	Risk ratio (95% CI)
South Asian ethnicity	50	14	3.5 (1.6–7.8)
Age ≥ 33 years	12	24	0.5 (0.2–1.2)
Male	20	15	1.3 (0.5–3.2)
Presence of SCC*mec* type V	23	15	1.5 (0.7–3.5)
History of diabetes mellitus	40	17	2.4 (0.8–7.7)
History of hospitalization[a]	0	19	0.0 (NC)
History of antibiotic use[a]	23	18	1.3 (0.4–3.8)

NOTE. CA-MRSA, community-associated methicillin-resistant *Staphylococcus aureus*; CI, confidence interval; NC, not calculable; SCC*mec*, staphylococcal cassette chromosome *mec*.
[a] Within 1 year of illness onset.

CA-MRSA after completion of decolonization therapy. To assess the factors affecting the effect of decolonization therapy, we compared the percentages of successful decolonization according to patients' demographic characteristics and exposure history.

For bivariate analysis, we computed the risk ratio and 95% confidence interval (CI) for each variable. A statistically significant result was defined as one with a P value less than .05. For multivariate analysis, variables with $P \leq .2$ in the bivariate analysis were included in a binomial regression model. Data analyses were performed with Stata, version 10.

From 2007 to 2009, 823 cases of CA-MRSA infection were recorded. CA-MRSA colonization screening was performed in 500 patients (61%), and 449 of them (90%) received decolonization therapy. We identified 148 CA-MRSA carriers. Among these, 101 received decolonization therapy and had post-decolonization screening swabs taken. They included 61 males and 40 females, with a median age of 33 years (range, 29 days–73 years). Eighteen patients remained positive for CA-MRSA (nasal swab, 9; axilla swab, 7; both, 2) after decolonization therapy. The percentage of successful CA-MRSA decolonization was 82% (83/101; 95% CI, 75%–90%). The median intervals from therapy completion to collection of post-decolonization screening swabs for those who could and could not be decolonized were 16 and 20 days, respectively.

The percentage of decolonization failure was higher among patients of South Asian origin (50%) than among those of other ethnicities (14%; risk ratio, 3.5; 95% CI, 1.6–7.8; Table 1). In addition, patients aged 33 years and over had a lower percentage of decolonization failure than those aged less than 33 years, but the association was not statistically significant ($P = .11$). These two variables were included in the binomial regression model. Adjusting for age, we found that patients of South Asian origin had a risk of decolonization failure 2.1 times higher than that of patients of other ethnicities (adjusted risk ratio, 3.1; 95% CI, 1.4–6.9).

Our study suggests that a decolonization therapy regimen consisting of a 5-day course of intranasal mupirocin and chlorhexidine body wash could eliminate CA-MRSA colonization in 82% of carriers. This is similar to the results of a study conducted by Ellis et al[7] in a group of healthy soldiers in the United States, where the percentage of successful CA-MRSA decolonization under a regimen of twice-daily intranasal mupirocin was 88% (95% CI, 78%–94%).

Factors affecting the effect of decolonization therapy have rarely been reported in the literature. In our study, we found that the percentage of decolonization failure was higher among South Asian individuals than in other ethnic groups. The underlying reason for this observation is unclear. We postulated that compliance and proper technique for the application of the decolonization therapy, as well as the socioeconomic status of the patients, might be the possible intervening factors. Further study should be conducted to explore the reasons behind the association.

We did not follow up with the patients after collection of post-decolonization screening swabs, and hence the long-term effect of decolonization therapy was unknown. Moreover, some important factors that may alter the effect of the decolonization therapy, such as patient's compliance with the therapy and their socioeconomic status, were not assessed in this study.

In conclusion, our study found that a 5-day course of intranasal mupirocin and chlorhexidine body wash could eliminate CA-MRSA colonization in 82% of carriers. Ethnicity of the patients may be associated with the effect of the therapy, and further study should be done to explore the reasons behind the association.

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Novel KPC Variant from a Carbapenem-Resistant Strain of *Klebsiella pneumoniae* in a Chinese Hospital

To the Editor—Klebsiella pneumoniae carbapenemase (KPC) was first reported to be the carbapenem-hydrolyzing β-lactamase from a carbapenem-resistant strain of *K. pneumoniae* in 1996[1] and was termed KPC-1. Two years later, KPC-2 was discovered by the same group.[2] However, a bla_{KPC-1} sequence error was found, and the bla_{KPC-1} and bla_{KPC-2} sequences were confirmed as identical soon afterward.[1] To date, 9 other variants (KPC-3 to KPC-11) have been reported (http://www.lahey.org/studies). Among all of these carbapenemases, KPC-2, which had been discovered in many areas, was the predominant one and played a crucial role in carbapenem resistance of *K. pneumoniae*. We isolated a strain of *K. pneumoniae* carrying a novel KPC variant from an inpatient in the First Affiliated Hospital, College of Medicine, Zhejiang University, in the Hangzhou city of China.

An 81-year-old man with acute exacerbation of chronic obstructive pulmonary disease accompanied by gastrointestinal hemorrhage was admitted to our hospital on February 27, 2010. An exploratory laparotomy was performed on March 20, and colorectal polyps were found and removed. Three days later, abdominal drainage appeared, and a strain of *K. pneumoniae*, labeled zjm002, was isolated from the drainage fluid.

The isolate was identified by Vitek gram-negative identification cards (bioMérieux). Antimicrobial susceptibility tests of 9 antibiotics were performed by the microdilution method with cation-adjusted Mueller–Hinton broth (Oxoid) according to the recommendations of the Clinical and Laboratory Standards Institute (CLSI). The minimum inhibitory concentrations of 26 other antibiotics were determined by the Etest technique (bioMérieux) according to the manufacturer's instructions. The susceptibility breakpoints were interpreted as recommended by the CLSI and previous reports. The strain zjm002 was sensitive to amikacin, tigecycline, chloramphenicol, and trimethoprim-sulfamethoxazole and intermediate sensitive to tetracycline, but it was resistant to all other 27 antibiotics (Table 1).

A modified Hodge test (MHT), which might assist in confirming the presence of carbapenemase, was carried out according to Endimiani et al.[3] As a result, the positivity of MHT for this isolate indicated that it carried carbapenemase. Since MHT could not exclusively detect the KPC-type carbapenemase, we detected bla_{KPC} and an additional 39 β-lactamases genes, including 13 class A carbapenemases genes (bla_{TEM}, bla_{SHV}, bla_{CTX-M1}, bla_{CTX-M2}, bla_{CTX-M8}, bla_{CTX-M9}, $bla_{CTX-M25}$, bla_{PER}, bla_{VEB}, bla_{GES}, bla_{CARB}, bla_{RTG}, bla_{LAP}), 10 class B carbapenemases genes (bla_{IMP}, bla_{VIM}, bla_{SIM}, bla_{SPM}, bla_{GIM}, bla_{AIM}, bla_{NDM}, bla_{KHM}, bla_{TMB}, bla_{DIM}), 8 class C carbapenemases genes (bla_{LEN}, bla_{OKP}, bla_{DHA}, $bla_{ACT/MIR}$, $bla_{LAT/CMY}$, $bla_{MOX/CMY}$, bla_{FOX}, bla_{ACC}), and 8 class D carbapenemases genes (bla_{OXA-1}, bla_{OXA-2}, bla_{OXA-10}, bla_{OXA-23}, bla_{OXA-24}, bla_{OXA-48}, bla_{OXA-51}, bla_{OXA-58}). Primer pairs for bla_{KPC} and bla_{TEM} polymerase chain reaction detection were 5′-ATGTCACTGTATCGCCGTCTA-3′ and 5′-TTACTGCCCGTTGACGCCCAA-3′ for bla_{KPC} and 5′-AGGAAGAGTATGATTCAACA-3′ and 5′-CTCGTCGTTTGGTATGGC-3′ for bla_{TEM}. The amplicons were sequenced on an ABI PRISM3730 sequencer analyzer (Applied Biosystems). As a result, bla_{KPC} and bla_{TEM} were detected, whereas the remaining 38 genes were not. The presence of bla_{TEM}, confirmed as bla_{TEM-1}, was determinated by sequencing and Blast analysis. The amino acid sequence of KPC showed an amino acid change