monitoring and feedback. Fourth, because glutamate dehydrogenase (GDH) testing does not distinguish toxigenic and nontoxigenic strains, use of this assay as the initial screening test in a 2-step algorithm may result in identification of fecal excretors of nontoxigenic *C. difficile* who would be isolated (ie, GDH positive, toxin negative). Nontoxigenic *C. difficile* strains do not cause disease and isolation is not required. A third step NAAT test would be required to confirm carriage of a toxigenic strain. Finally, if detection and isolation of fecal excretors are considered important goals of 2- or 3-step testing algorithms, it should be acknowledged that this is an imperfect detection method. Asymptomatic carriers with no diarrhea, including patients who have recently completed CDI treatment, may shed spores to their skin and the environment.[11]

In summary, we found that no patients with an alternative explanation for diarrhea and no recent antibiotic exposure had skin and/or environmental shedding of spores.[1] Based on this finding, we believe that it is reasonable to limit testing of such patients, particularly in facilities using stand-alone NAATs for CDI testing. However, our finding that antibiotic-exposed patients with <3 unformed stools within 24 hours who tested positive by NAAT frequently had skin and/or environmental contamination validates some of the concerns raised regarding restricting testing for all patients with unformed stool but not meeting criteria for clinically significant diarrhea. Testing of such patients using a 2- or 3-step algorithm may be helpful to identify fecal excretors who can be isolated to prevent transmission. Finally, because all CDI testing methods have limitations, it is essential that clinicians and infection control practitioners understand the advantages and disadvantages of the laboratory method used in their facility and appreciate the need to correlate test results with clinical assessments.

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Challenges of Long-Term MRSA Management in a Complex Continuing Care Setting

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To the Editor—Methicillin-resistant *Staphylococcus aureus* (MRSA) is a common nosocomial infectious agent with greater associated mortality and morbidity than infections caused by methicillin-susceptible *Staphylococcus aureus* isolates.[1] One considerable reservoir of MRSA is patients in long-term care facilities, who often have >1 factor predisposing them to become persistent MRSA carriers: advanced age, prior hospitalizations with greater length of stay, the presence of wounds, indwelling devices, and chronic diseases.[2] These patients have also been shown to have low rates of successful

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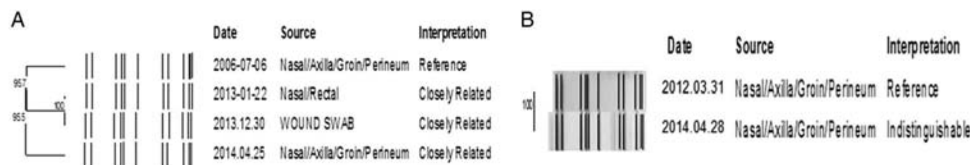

FIGURE 1. Molecular characterization by pulsed-field gel electrophoresis of methicillin-resistant *Staphylococcus aureus* (MRSA) chromosomal DNA restriction fragments of isolates from patients colonized over extended periods of time.

MRSA decolonization, and this report suggests that complex continuing care patients are similar.[2–4]

There are several methods of MRSA management, none of which are universally established: topical or systemic decolonization,[5] the implementation of isolation precautions, and follow-up screening.[3,6] Herein, we briefly describe a MRSA outbreak that occurred among long-term patients in a complex continuing care center from October 2013 through to December 2014 and the challenges concerning MRSA follow-up and management.

Patients were screened (ie, pooled swabs of axilla, nares, groin and perineum, and single swabs of all wounds and indwelling devices) upon admission or readmission to the facility. Colonized patients were on contact precautions per standard recommendations.[6] MRSA-positive patients who had no wounds or indwelling devices received topical decolonization with 2% mupirocin and 2% chlorhexidine gluconate. In addition, 1 patient also received systemic decolonization (ie, doxycycline, rifampin, chlorhexidine gluconate, and mupirocin) once. Decolonization was attempted no more than twice for any patient. Those not suitable for decolonization received daily baths with chlorhexidine gluconate. Pulsed-field gel electrophoresis was used to determine the relatedness of MRSA isolates.[7]

During this outbreak, 8 patients acquired MRSA, and 5 were previously colonized. A total of 5 unsuccessful topical decolonization attempts were made with 3 patients: 2 patients received decolonization twice, and a third patient received decolonization only once because previous failed attempts confirmed that our patients would not likely be decolonized using topical treatments.[2–4] In 1 previously positive case, a patient had acquired CMRSA-2 in July 2006 and systemic decolonization was attempted 1 month later. This patient tested positive for recolonization in July of 2009 after 463 days and 8 negative cultures. The patient subsequently tested negative 13 times over a period of 1,169 days before recolonizing again in January 2013. Thereafter, 2 negative culture times were observed, 1 involving 7 negative cultures over 127 days where the patient remained off precautions from April to December 2013, and the other involving 4 negative cultures over 56 days. Ultimately, the patient screened positive for MRSA again in April 2014. During the last negative surveillance period, the patient was not on precautions (March and April 2014). All isolates identified within this patient were >95% homologous (Figure 1a). A second previously positive patient originally tested positive for CMRSA-2 in March 2012. This patient subsequently cultured negative on 25 successive occasions over the course of 631 days and was not on precautions until screening positive for CMRSA-2 in April 2014. These isolates were indistinguishable by pulsed-field gel electrophoresis before and after a negative culture period of 631 days (Figure 1b).

Despite following recommended best practices,[6] this outbreak was triggered by previously positive patients who intermittently screened negative for MRSA and, therefore, were not always on contact precautions. Long durations of intermittent colonization with a single MRSA isolate were observed in these patients (Figure 1), which has been noted in other studies, the longest being 51 months.[3] These findings indicate either low levels of colonization where MRSA is present yet undetectable by regular culture methods, or intermittent shedding of MRSA.

A key challenge is the duration of MRSA colonization; 3 complex continuing care patients remained colonized for years, and 1 patient was colonized as long as 7 years and 9 months. The longest length of colonization previously observed in any group of patients was 5.56 years, with a median follow up of 940 days.[8] These patients are potential MRSA reservoirs and can remain on contact precautions receiving chlorhexidine gluconate baths for years. Patients on precautions utilize limited resources, such as single rooms.[6] Although the colonized patients in this report were in single rooms, there may be benefits in cohorting carriers. Cohorting may free single rooms for other needs and may alleviate certain psychological effects (ie, increased anxiety, depression, loneliness and adverse events) as well as limited healthcare worker contact, which can be issues for patients on precautions.[6,9] Systemic decolonization may also be considered in place of topical decolonization, which may reduce contact precautions whereby patients can screen negative by typical culture methods for months. However, no standard protocols exist, and there may be adverse effects with systemic decolonization that would not occur with topical methods.[5,6,10] Systemic decolonization would necessitate increased surveillance frequency to avoid undetected periods of intermittent shedding. There remains a paucity of data in support of systemic decolonization, as many studies involve varied patient groups, small sample sizes, and a lack of data to support the significant effectiveness of systemic decolonization over topical decolonization after a year.[5,8,10] Consideration of systemic methods of decolonization should be considered, as this patient population will likely remain in complex continuing care as chronic carriers.

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Resistant Superbugs: Race against Time

To the Editor—*Pseudomonas aeruginosa* is one of the most notorious bacteria isolated from nosocomial infections. The growing threat of antimicrobial resistance in *P. aeruginosa* relies on its intrinsic resistance as well as on the transferable resistance determinants that further reduce their spectrum of susceptibility. Surveillance by hospitals to track the emergence of newer strains of *P. aeruginosa* is important to prevent its outbreak. In the present study, a total of 207 nonduplicate *Pseudomonas* isolates were collected over a period of 2 years (2013–2015) from various clinical samples of admitted patients (eg, pus, urine, wounds, and burns). The susceptibility of these isolates was tested against antimicrobial agents according to the Clinical and Laboratory Standards Institute (CLSI) broth microdilution procedure and interpretation criteria.[1] Among these isolates, 26 showed resistance to the following antibiotics: cefepime (89%), ceftriaxone (54%) gentamicin (79%), netillin (39%), ciprofloxacin (59%), and olfloxacin (34%). Based on the restriction pattern of 16S rRNA gene (Msp1 and Hha1), these 26 isolates were divided into 9 strains of *P. aeruginosa*. Among these 9 strains, 67% showed elevated minimum inhibitory concentrations (MICs) for imipenem (MIC, ≥10 μg/ml) and meropenem (MIC, ≥30 μg/ml). In a few studies from India, the rate of carbapenem resistance in *P. aeruginosa* isolates has been reported to vary from 12% to 43%.[2,3] PCR amplification with NDM-1 primers (forward: CTCGCACC GAATGTCTGGC and reverse: GCGGCGTAGTGCTC AGTGTC) showed amplification in all the carbapenemase producers. The high prevalence rate of carbapenemase producers could be linked to poor control of antibiotic usage in India.[4] Tigecycline, which was approved by the Food and Drug Administration in 2005, and the "old" antibiotic colistin are among the remaining treatment options for these difficult-to-treat infections.[5] Among the carbapenem-resistant *P. aeruginosa* strains, 42% and 35% showed resistance to tigecycline (16–50 mg/L) and colistin (16–500 mg/L), respectively (Figure 1). Among these isolates, 2 (M-30 and R-32) showed resistance to all the last-resort antibiotics tested (ie, imipenem, meropenem, colistin, and tigecycline). This is the first study from India that has reported the emergence of a 'superbug' *P. aeruginosa* that is resistant to last-resort antibiotics.

Due to lack of stringent measures, almost all antimicrobial agents are available to both public and private-sector outpatients in India. Decades of overuse and misuse of antibiotics by both the public and clinicians has led to the evolution of these superbugs. A decline in the development of new antimicrobial agents and the simultaneous increase in resistance to available treatment options pose a threat to the successful treatment of infections caused by these

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