Is Surveillance for Multidrug-Resistant Enterobacteriaceae an Effective Infection Control Strategy in the Absence of an Outbreak?

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Multidrug-resistant enterobacteriaceae (MDRE) are an important cause of nosocomial infections. The effectiveness of screening for MDRE in the nonoutbreak setting in an attempt to prevent transmission is unknown. Patients admitted for new organ transplantation were screened for MDRE colonization. Prospective clinical data were collected, and pulsed-field gel electrophoresis and plasmid and integron analysis of isolates were performed. Colonized patients were not isolated except when required by standard precautions. Of the 287 patients, 69 (24%) were colonized, and 6 (9%) of the 69 developed clinical infections. Most colonizing isolates (66/69) were unique. No clinical infections resulted from patient-to-patient transmission. Analysis of clinical isolates from nonstudy patients demonstrated no evidence of transmission leading to clinical disease. The annual cost of a surveillance program was calculated at Canadian $1,130,184.44. Thus, the routine and costly use of MDRE surveillance and isolation precautions are not warranted in the absence of a clonal outbreak in this population.

Enterobacteriaceae may become resistant to broad-spectrum β-lactam antibiotics through several mechanisms, including the production of extended-spectrum β-lactamases (ESBL) or overproduction of native chromosomal AmpC β-lactamases [1]. Colonization or infection with multidrug-resistant Enterobacteriaceae (MDRE), which are resistant to third-generation cephalosporins and often other antibiotic classes as well, may result from acquisition of the organism from another patient or through development of resistance in the patient’s own previously susceptible strain. Clonal outbreaks of such MDRE have been well described over the past decade and have resulted in substantial morbidity and mortality [2–10]. In general, the control of multidrug-resistant organisms has relied on several strategies, including surveillance and the use of barrier precautions with or without the use of private rooms. The use of barrier precautions implies that organisms are being exchanged between patients and that, by identifying patients before clinical infection, the chain of transmission can be aborted.

Given the large number of outbreaks reported in the literature, it is tempting to assume that hospitals should develop MDRE surveillance programs, as they have for other resistant organisms, such as methicillin-resistant Staphylococcus aureus. Indeed, some authors have called for routine surveillance to detect asymptomatic MDRE colonization in an effort to control transmission [11, 12]. Although surveillance and isolation may play a role in limiting or stopping MDRE outbreaks [4–6, 11–14], it is unknown whether surveillance has a role in controlling MDRE in the nonoutbreak or endemic setting. In the absence of a particularly “fit” (i.e., virulent and easily transmissible) clone, MDRE infections may be more likely to result from the patient’s own flora rather than through transmission. Therefore, in the endemic, nonoutbreak setting, surveillance and isolation would not be expected to have a significant impact on either colonization or infection rates. Surveillance programs require substantial laboratory and personnel resources. In addition, subsequent isolation of colonized patients is expensive and may have a significant negative psychological impact [15]. Given the significant resource implications of a surveillance and isolation program, it is essential to first determine the natural history of MDRE colonization and infection in the endemic setting.

To clarify the role of routine surveillance for MDRE, we undertook a prospective 18-month cohort study of colonization and infection with MDRE exhibiting resistance to third-generation cephalosporins in a population of solid-organ transplant recip-
iente. This group was chosen because it represented a high-risk population, as determined on the basis of previous surveillance studies at our institution, and because patients were conveniently located in a single nursing unit. This study represented a true natural history study, because additional barrier precautions were not required for patients colonized with MDRE, except when required by standard precautions (e.g., the presence of draining wounds or uncontrollable diarrhea) [16].

Methods

Setting

This study was carried out on the multiorgan transplantation unit at the Toronto General Hospital site of the University Health Network (Toronto). The ward consists of 4 rooms with 4 beds, 8 rooms with 2 beds, and 4 single rooms. One sink and alcohol-based waterless hand rinses are available in each room.

Epidemiologic Methods

All new patients admitted for organ transplantation to the multiorgan transplantation unit between 1 April 1999 and 30 September 2000 were eligible for the study. No patients declined screening cultures. Rectal swabs for MDRE isolation were obtained from patients at admission, weekly for the first month, every second week thereafter, at discharge, and during subsequent admissions during the study period. When possible, rectal swabs were also obtained during outpatient follow-up visits. Patients were enrolled in the study at the time of transplantation and monitored until their last rectal swab was obtained. Patients who had a single swab obtained at admission and were discharged in <1 week were considered to be monitored for 0 days.

Demographic, medical, epidemiologic, microbiologic (MDRE colonization status and MDRE clinical isolates), and pharmacologic (intravenous antibiotic history and immunosuppression history) data were collected prospectively for each subject and entered into an Excel 97 spreadsheet (Microsoft). MDRE clinical isolates obtained from contemporaneous study-eligible inpatients in the multiorgan transplant unit (i.e., nonstudy patients admitted to the unit during the study period) were prospectively collected and frozen at −70°C for further analysis.

Risk factors for colonization and infection were determined by χ² or Fisher’s exact test for categorical variables and by the Mann-Whitney U test for continuous variables (SPSS, version 9.0; SPSS). A multivariate model was analyzed by stepwise logistic regression. Two-sided P < .05 was considered to be significant.

Colonization status was not made available to the treating team, and no additional staff education or enforcement of infection control policy was done during the study. No attempt was made to isolate colonized patients or use extra barrier precautions (i.e., gowns and gloves) unless required under standard precautions, such as presence of an open, draining wound, uncontrollable diarrhea, or an airborne or droplet-transmitted infection.

Definitions

Duration of colonization was defined as one-half of the number of days between the last negative culture result prior to colonization and the first positive rectal culture, plus the duration of consecutive and rectal cultures, plus one-half of the number of days between the last positive culture and the first negative rectal culture after colonization. When a patient had a positive result only for his or her first or last rectal culture, it was not possible to accurately determine the duration of colonization, and these patients were censored for this calculation.

Antibiotic history collected for statistical analysis included all antibiotics used from the date of admission to the date of the last reported rectal culture or the date of the first positive clinical isolate of MDRE. If the period from the date of admission until the date of transplantation was >1 month, the antibiotic history was collected from 1 month before transplantation to the date of the last reported rectal culture or the date of the first positive clinical isolate of MDRE.

Infection with MDRE in both the study and nonstudy populations was defined as isolation of MDRE from a normally sterile site (blood or sterile body fluid) or another site (abscess or tissue) by use of a technique to minimize contamination with colonizing flora (percutaneous needle aspirate or biopsy or bronchoalveolar lavage) and a compatible clinical scenario. MDRE isolated from urine was considered to be a colonizing organism unless the organism was obtained in pure culture in amounts >10⁸ cfu/L, the sample was obtained via in-and-out catheterization, and signs and symptoms of urinary tract infection were present.

Cost Analysis

The hypothetical cost of adhering to an admission and discharge MDRE surveillance program was calculated. Costs are given in 2001 Canadian dollars. It was assumed that colonized patients were isolated in private rooms and that gowns and gloves were required for patient contact for the length of colonization from the time the organism was detected until colonization was no longer detected. With other multidrug-resistant organisms, patients are frequently isolated until 3 negative surveillance culture results are obtained; however, this additional isolation time was not included in the cost analysis. Gown and glove use, as well estimates of nursing time required to obtain surveillance cultures, were obtained from interviews with nursing staff. Supply costs were obtained from the ward supply budget. Additional nursing time necessary for the care of patients requiring contact precautions was obtained from the computerized nursing database (MISTro, version 5.3; GRASP Systems).

In addition to the above costs, the use of a private room for isolation purposes in our hospital results in a loss of potential income, because the room is then not available to patients with insurance coverage desiring private accommodation. For the purposes of the analysis, it was assumed that this additional income would be lost for each patient isolated as a result of a positive surveillance culture result. Finally, the cost of the culture swab and processing of surveillance cultures were obtained from hospital and microbiology laboratory data.
Clinical Microbiology Methods

Rectal swabs were planted on MacConkey agar (Oxoid) supplemented with 2 μg/mL cefpodoxime and incubated at 35°C overnight in 5% CO₂. Lactose-fermenting colonies were isolated and subcultured on a sheep blood agar plate (Oxoid) to ensure purity. Three colonies were sampled to undergo antibiotic susceptibility testing by means of the Vitek ESBL card (BioMérieux) [17]. Confirmation of the drug resistance phenotype was obtained by double-disk synergy testing [17] and determination of microtube broth dilution MICs [18].

Molecular Microbiology Methods

**Pulsed-field gel electrophoresis (PFGE).** The genetic relatedness of MDRE isolates identified above was determined by PFGE by means of a protocol adapted from that of Gautom [19]. In brief, total DNA preparations embedded in 1% low-melt agarose plugs (Bio-Rad) were digested with SfiI (New England Biolabs) and separated in a 1% PFGE-grade agarose gel (Bio-Rad) with 0.5x Tris-borate-EDTA running buffer supplemented with 50 μM thiourea [20] by means of a CHEF DRII PFGE system (Bio-Rad). The gel was stained with 2 μg/mL ethidium bromide and photographed. Strains differing by ≤3 bands were considered to be identical [21].

**Polymerase chain reaction (PCR) detection of TEM and SHV genes.** Total template DNA was extracted from overnight cultures with Instagene (Bio-Rad), according to the manufacturer’s instructions. SHV and TEM genes were amplified by PCR [22]. PCR products were separated by agarose gel electrophoresis, excised from the gel, and purified by means of a Qiagen Gel Purification Kit (Qiagen). PCR products were either sequenced directly or cloned into pCR2.1 TOPO cloning vector (Invitrogen), according to manufacturer’s instructions, and sequenced with the M13 forward and reverse primers. Sequencing was done at the Molecular Biology Core Facility at York University (North York, Ontario) by means of the dye-deoxy-terminator method on an ABI 377 sequencer (Applied Biosystems).

**Isoelectric focusing of β-lactamases.** β-lactamase enzymes were identified by isoelectric focusing and nitrocefin overlay [22]. In brief, standardized cell-free extracts from overnight cultures were separated in precast isoelectric focusing minigels (pH 3–10; Bio-Rad), and β-lactamase activity and isoelectric point (pI) were determined by nitrocefin (1 mg/mL) overlay, comparing the bands with standards run on the same gel. The β-lactamase standards used were extracts of strains containing TEM-1 (pI, 5.4), SHV-1 (pI, 7.6), SHV-3 (pI, 7.0), SHV-4 (pI, 7.8), and SHV-5 (pI, 8.2) (provided by G. Jacoby, Lahey Clinic, Burlington, MA).

**Plasmid analysis.** Plasmids were isolated from a 5-mL overnight culture in Luria-Bertani broth by means of a modified alkaline lysis protocol [23]. Undigested plasmid DNA was separated on a 1% agarose gel stained with 2 μg/mL ethidium bromide and photographed.

**PCR amplification and DNA sequencing of integrons.** The presence of integrons was detected by PCR [22]. The intI1 gene, encoding the class I integrase, was amplified from ESBL-containing strains with use of primers intI1up (5′-ATAACGAGGACTGCGG-3′) and intI1down (5′-ATCGAATTCAGATTTGAC-3′). Strains positive for intI1 were reamplified with the intI1 downstream primer and a primer directed to the 3′ conserved sequence of the class I integrons (5′-AAACGAGCTTTGACCTGA-3′) [24]. Amplicons with unique restriction endonuclease banding patterns were sequenced to identify resistance cassettes.

**Results**

Demographics. In total, 1298 patients were admitted to the multiorgan transplantation unit over the 18-month study period. Of these, 303 were undergoing their first transplantation and underwent surveillance for MDRE colonization and infection. Contemporaneous ward patients who were not undergoing their first transplantation underwent passive laboratory-based surveillance for MDRE infection (i.e., clinical isolates). Complete data were obtained for 287 (95%) of 303 patients who received new organ transplants. Patients admitted for a short period (<1 week) who did not have at least 1 rectal swab obtained before discharge were considered to have incomplete data. The study population had the following distribution of transplant recipients: 117 (41%) renal; 99 (34%) liver; 45 (16%) lung; 17 (6%) kidney/pancreas; 13 (5%) heart; 2 (0.7%) heart/lung; and 3 (1%) other combinations.

**MDRE colonization.** Of the 287 patients, 72 were identified as being colonized with MDRE. However, 3 isolates were subsequently found to be drug-susceptible by confirmatory testing (microtube broth dilution MIC determination). Thus, 69 (24%) of 287 subjects were colonized with MDRE at some point during the study period. On average, patients were monitored for a mean of 43 days (median, 19 days; range, 0–400 days). Patients who were not colonized at any point over the duration of the study were monitored for a mean of 34 days (median, 15 days; range, 0–326 days), whereas those who were colonized were monitored for a mean of 73 days (median, 31.5 days; range, 0–400 days).

Of the 69 colonized patients, 25 (36%) were colonized with MDRE at the time of entry into the study (i.e., when their first rectal swab was taken at the time of admission). Forty-six (67%) colonized patients were still positive when their last rectal swab was taken at the time of admission. Of the patients whose colonization period started after admission and ended before discharge, the mean duration of colonization was 34 days (median, 16 days; range, 6–167 days). Eleven percent of colonized subjects were intermittently colonized, and 23% were colonized with >1 MDRE during follow-up. For patients not found to be colonized at admission, colonization was detected, on average, 21 days after transplantation (median, 8 days; range, 0–245 days).

The risk factors for colonization found by univariate analysis are shown in table 1. By multivariate analysis, however, the only factors found to be significant risk factors for colonization were being the recipient of a nonrenal transplant (P = .02; odds ratio [OR], 2.10; 95% confidence interval [CI], 1.12–3.96) and prior third-generation cephalosporin use (P = .02; OR, 1.99; 95% CI, 1.10–3.61).

The colonizing strains are shown in table 2. Almost all of
One infecting isolate was the same species and had the same organism, and 1 was infected with a different, unique organism. Three of 5 patients were infected with their colonizing strain (66/69 [96%]) were unique by PFGE analysis. One *Citrobacter freundii* strain was common to 2 patients. Both clusters were epidemiologically linked, because the patients had shared the same room or adjacent rooms at some point during their hospital stay.

PCR and DNA sequence analysis showed that 12 of the 66 unique strains had the ESBL genes SHV-5 (4 strains) or SHV-12 (8 strains). The *C. freundii* strain common to 3 patients contained SHV-5, whereas the shared *E. coli* strain was an AmpC hyperproducer. All ESBL SHV-containing strains were positive for the presence of a class 1 integrase gene, but complete integrons could be amplified from only 8 of 12 strains. Restriction endonuclease molecular typing and DNA sequencing of integrons on plasmids containing the SHV-12 gene showed that 4 unique *C. freundii* strains shared the same ant(3′)-Ib–containing integron [22], which was also found in 1 strain of *E. coli* and 1 strain of *Klebsiella oxytoca*. The SHV-12 gene in these strains was not found within the integron itself, but plasmid analysis showed that these strains contained similar large plasmids (data not shown). The SHV-12, ant(3′)-Ib–bearing *E. coli* strain was isolated from a patient carrying an SHV-12, ant(3′)-Ib–containing *C. freundii* strain.

**MDRE infection.** Of the 287 new transplant recipients enrolled in the study, 66 (23%) developed infections with members of the family Enterobacteriaceae at some point during the follow-up period. Of the 69 patients colonized with MDRE during the study period, 30 (43%) developed infections with drug-susceptible Enterobacteriaceae, and only 6 (9%) developed MDRE infections. One patient developed an MDRE infection but had no prior evidence of colonization. Given that so few MDRE infections occurred, a logistic regression analysis to determine infection risk factors was not done.

The infecting MDRE strains were all unique on PFGE analysis; hence, its relatedness to the colonizing isolate could not be determined with certainty. Of importance, no clinical infection resulted from MDRE transmission from another patient.

**MDRE infections in contemporaneous transplant ward patients.** Twelve (1.2%) of 995 patients admitted to the transplant ward at some point during the study period had MDRE isolated from clinical specimens. All isolates were unique by PFGE analysis and different from all colonizing and infecting strains obtained from the core study population. Therefore, not a single symptomatic infection with MDRE could be traced to transmission from another patient over the entire 18-month period.

**Cost analysis.** The annual costs of a hypothetical admission and discharge MDRE surveillance program were calculated, assuming that 870 patients (on the basis of the same admission rate of 1298 patients/18 months) would require screening (1740 cultures/year), a colonization rate of 24%, and a median duration of colonization of 19 days. The cost of this less-aggressive surveillance program was calculated, rather than the cost of the study protocol, because it was thought that the study protocol was too aggressive (i.e., weekly surveillance swabs for the first month and biweekly thereafter) to be readily applicable to most centers. Costs included in the analysis are shown in table 3. The total annual cost of maintaining such a program is $1,130,184.44. If the cost of private rooms is excluded, the annual cost is $237,564.44.

**Discussion.** We have shown that colonization with MDRE occurs in about one-quarter of our population of transplant recipients. Despite this substantial reservoir, transmission among patients

### Table 1. Risk factors for colonization with multidrug-resistant Enterobacteriaceae among hospitalized patients in a nonoutbreak setting, by univariate analysis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonrenal transplant</td>
<td>.002</td>
</tr>
<tr>
<td>Carbapenem use</td>
<td>.004</td>
</tr>
<tr>
<td>First-generation cephalosporin use</td>
<td>.727</td>
</tr>
<tr>
<td>Second-generation cephalosporin use</td>
<td>.048</td>
</tr>
<tr>
<td>Third-generation cephalosporin use</td>
<td>.001</td>
</tr>
<tr>
<td>Piperacillin/tazobactam use</td>
<td>.085</td>
</tr>
<tr>
<td>Quinolone use</td>
<td>.042</td>
</tr>
</tbody>
</table>

*NOTE.* Antibiotic use is counted from the time of admission until the first positive result of a culture of a swab specimen.

* a Mann-Whitney U test.

### Table 2. Resistance mechanism of colonizing organisms among hospitalized patients in a nonoutbreak setting.

<table>
<thead>
<tr>
<th>Organism, resistance mechanism</th>
<th>No. of unique isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Escherichia coli</em></td>
<td></td>
</tr>
<tr>
<td>AmpC</td>
<td>9</td>
</tr>
<tr>
<td>TEM-1/SHV-12</td>
<td>1</td>
</tr>
<tr>
<td>OXA-31/TEM-1</td>
<td>1</td>
</tr>
<tr>
<td>TEM-1/AmpC</td>
<td>1</td>
</tr>
<tr>
<td><em>Enterobacter</em> species</td>
<td></td>
</tr>
<tr>
<td>AmpC</td>
<td>34</td>
</tr>
<tr>
<td>SHV-5/AmpC</td>
<td>1</td>
</tr>
<tr>
<td>TEM-1/AmpC</td>
<td>1</td>
</tr>
<tr>
<td><em>Klebsiella</em> species</td>
<td></td>
</tr>
<tr>
<td>SHV-1/AmpC</td>
<td>3</td>
</tr>
<tr>
<td>TEM-1/SHV-12</td>
<td>1</td>
</tr>
<tr>
<td>SHV-2/SHV-5</td>
<td>1</td>
</tr>
<tr>
<td>Third-generation cephalosporin-resistant, mechanism unknown</td>
<td>1</td>
</tr>
<tr>
<td><em>Citrobacter</em> species</td>
<td></td>
</tr>
<tr>
<td>AmpC</td>
<td>26</td>
</tr>
<tr>
<td>TEM-1/SHV-12</td>
<td>4</td>
</tr>
<tr>
<td>TEM-1/AmpC</td>
<td>2</td>
</tr>
<tr>
<td>SHV-12</td>
<td>2</td>
</tr>
</tbody>
</table>
was minimal, and infection secondary to transmission was nonexistent, despite the absence of barrier precautions beyond what is recommended for standard precautions [16]. Most colonizing MDRE were of the inducible AmpC phenotype and were unique according to PFGE analysis, supporting the concept that the resistant strains arose from the patient’s own flora because of selective antibiotic pressure rather than through patient-to-patient transmission. Third-generation cephalosporin–susceptible Enterobacteriaceae species infections acquired during hospitalization have been shown elsewhere to arise from endogenous flora [25].

Surveillance for MDRE followed by the isolation of colonized patients has been thought to assist in control of the epidemic spread of ESBL- or AmpC-producing Enterobacteriaceae [4–6, 11–14]. These studies described the spread of aggressive clonal strains that colonized or infected a large percentage of patients in the at-risk population, implying that the outbreak organisms were particularly “fit” (i.e., they were readily able to colonize and/or cause invasive disease). Some authors have called for routine surveillance to detect asymptomatic MDRE colonization in an effort to control transmission [11, 12].

Although surveillance may be effective in clonal outbreaks, it is unclear whether it has a role to play in the setting of endemic MDRE colonization, in which infection may result from endogenous flora rather than transmission from another patient. Surveillance in this setting may provide little helpful information and may prompt the implementation of potentially unnecessary contact precautions. We have shown that maintaining a surveillance program for asymptomatic MDRE colonization is quite expensive in our setting and, indeed, would have provided no benefit. Even excluding the additional cost of private rooms, the annual cost was still substantial.

In the absence of an obvious clone causing clinical disease, the colonizing and infecting isolates were quite heterogeneous, in terms of both resistance mechanisms and PFGE patterns. The majority of isolates were hyperproducers of AmpC β-lactamase. Such organisms are well known to cause significant nosocomial disease [7, 9, 26, 27]. Of interest, 8 SHV-12–containing strains, from 3 separate species and isolated from 6 separate patients, shared a common large plasmid containing a class 1 integron encoding ant(3′)-Ib, which suggests that resistance genes were being transferred between patients. The fact that not all these patients were closely linked epidemiologically suggests that intermediary patients may also have been colonized with strains carrying the same integron-containing plasmid. The implication of this limited degree of resistance gene transfer is unknown; however, all these strains were colonizing rather than infecting strains.

In contrast to other studies, we grouped together ESBL- and AmpC-producing organisms, rather than limiting our study to ESBL producers. Both mechanisms of resistance confer resistance to third-generation cephalosporins, whereas the AmpC phenotype confers additional resistance to cephamycins and β-lactam–β-lactamase inhibitor combinations. Depressed AmpC mutants are more likely than ESBL producers to arise de novo in patients receiving antibiotics; however, it is reasonable to assume that, once present in the bowel flora, both would be equally transferable between patients.

The present study has several limitations. Because of morbidity associated with their requirement for transplantation, almost all study patients had been admitted previously, but data related to their previous admissions or antibiotic history were not considered. It is unlikely that patients arriving in the hospital for the first time would have such a high level of colonization with MDRE. Many patients found to be colonized at the time of admission to the study had extensive hospitalization histories and may have been colonized for a long time. The true duration of colonization of patients who were colonized at admission to the study and/or colonized at exit from the study cannot be calculated. Rather, the duration of colonization could be calculated only for those who developed and lost colonization during the study period. Thus, our reported duration of colonization represents a minimal estimate.

We lacked complete data for 16 organ transplant (mostly kidney) recipients, who, given their uncomplicated stay in the hospital, were unlikely to carry MDRE. Without these patients, the prevalence of MDRE colonization in the study population may be slightly overestimated (range, 22.7%–28.0%).

It was not possible to calculate the amount of transmission to contemporaneous but nonstudy patients, because they were screened only for MDRE infections, not asymptomatic carriage. The cost of screening such a large number of patients was considered to be prohibitive. However, it is reasonable to assume that the degree of transmission to this population would be similar to the low level found in the new transplant popu-
lation. Even should transmission have occurred to a greater degree, no infections resulted.

The sensitivity and specificity of the cefpodoxime MacConkey screening agar is unknown, because there is no recognized standard method for MDRE surveillance. Several antibiotic-supplemented differential and selective media have been used for MDRE screening of rectal swab specimens, including ceftazidime-supplemented MacConkey [5, 6] and cefotaxime-supplemented Drigalski agar [4]. We chose cefpodoxime, because it appears to be the most sensitive for detecting the presence of ESBL [28]. Given that enrichment culture methods have been shown to be more sensitive for other multidrug-resistant organisms, such as vancomycin-resistant enterococci [29] and methicillin-resistant S. aureus [30], it is likely that low-level colonization may be missed with use of this agar. Similarly, intermittent colonization may simply be due to colonization levels below the limit of detection of the screening test. This is supported by the fact that 1 patient in our study developed MDRE infection despite negative screening rectal culture results.

In conclusion, we have shown that performing MDRE surveillance in the absence of an outbreak is a costly exercise that provides little or no benefit for infection control or predicting clinical infection in this patient population. Although it is possible that the isolation and use of barrier precautions for colonized patients may play a role in managing an outbreak, we suggest that in an endemic setting in which a clonal outbreak has not been detected, a more efficient means of surveillance might be achieved by performing routine MDRE surveillance of clinical isolates. Surveillance of colonization may then play a greater role in the event that a clonal outbreak is identified.

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