LETTERS TO THE EDITOR

Surveillance of Central Venous Catheter–Associated Bloodstream Infection in a Scottish Hematology Unit

To the Editor—In their recent article, Worth et al.[1] suggest a new case definition for central venous catheter (CVC)–associated bloodstream infection (BSI) to be applied in hematology units. Their reasons for not using the established definitions from the National Nosocomial Infection Surveillance (NNIS) system[2] are that they are complex and resource intensive. We wish to relate our experience in successfully establishing a CVC-associated BSI surveillance system in our unit using the NNIS definitions.

The hematology unit at Ninewells Hospital in Dundee, Scotland, has 13 beds and a day-patient area that cares for approximately 20 patients every weekday. During a 1-year period, data on CVC-associated BSI have been prospectively gathered. The unit's senior nurse has set up a database using Access (Microsoft) to record the number of patients with a Hickman (tunneled) catheter and the number of catheter-days per month. Information on the reason for any line being removed is also collected. The medical microbiologist (who is trained in infection control and who liaises with the unit regarding infections) performs a monthly inquiry for all positive blood culture results from the hematology unit, including the day-patient area, using the microbiology laboratory computer (LabCentre; CliniSys). Clinical information, entered into the computer by the medical microbiologists when they telephone in positive blood culture results, can be accessed to see details such as which antibiotic was used. The microbiologist then meets with the senior nurse monthly for approximately 1 hour in the hematology unit to review which patients with bacteremia meet the NNIS system diagnostic criteria for CVC-associated BSI. Any cases for which there is still doubt about the diagnosis, the medical notes can be referred to, as these are kept on the unit.

During the 1-year period (from April 2007 to March 2008), there were a total of a 29 cases of infection in 19 patients; 11 cases of infection fulfilled criterion 1 of the NNIS definitions, and 18 cases of infection fulfilled criterion 2. Coagulase-negative *Staphylococcus* was the sole organism found in the blood cultures for 17 (58.6%) of the 29 cases of infection. The hematology unit has seen a decline in the incidence of CVC-associated BSI (albeit from a high level; see Figure), which may partly be due to the implementation of surveillance. The overall rate for that year was 12 cases of infection per 1,000 CVC-days.

A rate of 11.5 cases of infection per 1,000 CVC-days among hematology patients who used tunneled lines has been reported,[3] although lower rates of infection have also been found.[4] Despite possible contributing factors, such as neutropenia and whether the patient was hospitalized or not,[5] we feel that the surveillance system we have implemented is worthwhile and has played a part in decreasing the incidence of CVC-associated BSI in our hematology unit. The key to

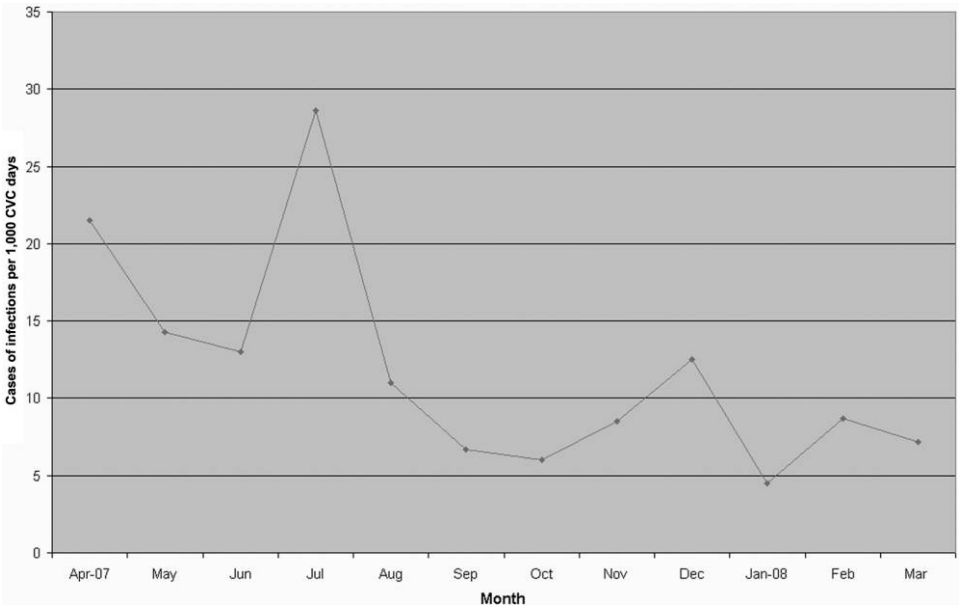

FIGURE. Monthly rates of central venous catheter (CVC)–associated bloodstream infection in the hematology unit at Ninewells Hospital during the period from April 2007 to March 2008.

establishing successful surveillance has been the collaboration between the senior nurse, who was responsible for the unit's Hickman line protocols, and the medical microbiologist, who is familiar with NNIS definitions because of interaction with other hospital areas, such as the intensive care unit, and can ensure that the bacteremia data are complete. Use of the NNIS definitions has not been onerous, and has the advantage of being well established in many countries, and therefore data can be compared between centers. Creating separate case definitions for CVC-associated BSI in patients in a hematology unit could be counterproductive, because units considering using surveillance, faced with an expanding choice of definitions, may be less likely to do it at all.

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Reply to Graham and Olver

To the Editor—We thank Graham and Olver[1] for their interest in our study[2] of catheter-associated bloodstream infection (BSI) in hematology units, and for reporting their own experience of successfully applying the National Nosocomial Infection Surveillance (NNIS) system definition in a Scottish hematology unit. We agree that a standard definition must be employed and that this is essential if benchmarking is to be performed, and we do not support the use of ad hoc or poorly validated case definitions.

Evidently, the work flow and size of the Scottish hematology unit enabled collaboration and regular review by a medical microbiologist. It is not clear, however, how many other hematology units have devoted nursing staff and a medical microbiologist with sufficient time to perform surveillance activities for infection. It would be helpful to know the number of hours required of these nurses during the surveillance period, as a measure of resource requirements. During the first 6 weeks of our study, the number of hours required for review by an infection control practitioner for application of NNIS methodology was monitored (see Figure), and the mean number of hours required was 1.6 hours per 10 beds per week.

Furthermore, Graham and Olver[1] report experience with long-term central venous catheters (CVCs; ie, Hickman catheters), for which data on dates of insertion and removal may be more readily available to assist with the calculation of the denominator (ie, number of devices used per 1,000 CVC-days). In contrast, we studied medium-term CVCs[2] (peripherally inserted and nontunneled), of which a larger number of individual devices are used, recording of the dates of insertion and removal may not be as reliable, and closer direct monitoring is required by surveillance staff to ensure accurate data collection. A standardized strategy must be practical for a range of tunneled, nontunneled, and implanted devices, if it is to be applied to a more broad population of hematology patients.[3]

NNIS methods have been employed in intensive care unit (ICU) populations, and therefore interhospital comparisons

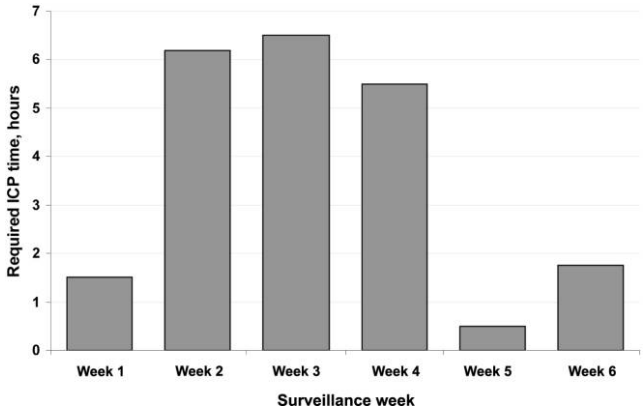

FIGURE. Number of hours required for surveillance of central venous catheter–associated bloodstream infection by an infection control practitioner (ICP) during the first 6 weeks of our study[2] in a 23-bed hematology unit.

may be performed.[4] However, the same argument does not apply for hematology patients, because their risks for infection and the nature of their care (eg, ambulatory care) may not allow for benchmarking with ICU or other populations. We question the notion that a definition that has been used predominantly in ICU populations can simply be extrapolated to non-ICU populations, without comprehensive evaluation.

Recently, the National Healthcare Safety Network (NHSN) definition for laboratory-confirmed BSI has replaced the NNIS definition,[5] and this simplified NHSN definition no longer contains the requirement for the treating physician to institute "appropriate antimicrobial therapy" (criterion 2B of the NNIS system diagnostic criteria) for classification of an infection as CVC-associated BSI. As a result of this change, longitudinal evaluation using historical data will not be possible until baseline data are accrued using the new definition. We therefore believe it is timely to consider the feasibility and applicability of surveillance definitions, in a milieu where many healthcare centers may already be implementing modified definitions for healthcare-associated BSIs.

Robust, multicenter evaluation must be performed prior to the implementation or modification of any standardized surveillance strategy, and findings at our own healthcare center's hematology unit may not reflect the findings at other hematology units. Such an evaluation must include the necessary resource requirements. We suggest that, as a key stakeholder, the hematologist, whose regular clinical contact is incorporated into his or her usual work flow, may be well positioned to inform surveillance activities or to flag potential cases for surveillance personnel. We welcome debate regarding the utility and implementation of a range of case definitions in hematology units, and we do not believe this to be counterproductive to the implementation of surveillance by individual hematology units. Such debate may contribute to future research agendas, in which the validity and ease of implementation can both be evaluated.

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Has the Time Come to Recommend the Use of Alcohol-Based Hand Rub to Hospitalized Patients?

To the Editor—Methicillin-resistant *Staphylococcus aureus* (MRSA) is a well-known and important nosocomial pathogen worldwide.[1-3] Attempts to control the spread of MRSA have relied mostly on 3 measures: (1) use of alcohol-based hand rub by healthcare workers (HCWs), (2) screening of patients with risk factors for MRSA carriage on admission, and (3) isolation of colonized or infected patients.[4-6] The role played by HCWs in the transmission of MRSA has been established,[5,7] but little is known of the role played by colonized patients in the transmission of MRSA from patient to patient.[8]

Our institution is a 230-bed tertiary care teaching hospital (with a 14-bed intensive care unit) that had 7,590 admissions in 2007. All patients with risk factors for MRSA carriage are screened within 72 hours of hospital admission. The risk factors include transfer from another hospital or nursing home, previous surgical procedure, repeated hospitalization, stay in an intensive care unit during the last 3 years, presence of open wounds, and long-term oxygen therapy. All detected MRSA carriers are placed in isolation. If private rooms are not available, then the MRSA-colonized patients are grouped with other MRSA carriers or placed in rooms occupied by patients without MRSA colonization, and a distance of at least 1 meter between patients' beds has to be assured. If a hospitalized patient is found to carry MRSA more than 72 hours after admission, surveillance cultures of nasal samples are performed for all other patients in the same room and for HCWs who have had contact with the MRSA carrier. The prevalence of MRSA has remained fairly constant during the past 4 years (ie, 4.6–5.1 cases per 1,000 admissions). The proportion of MRSA cases that were acquired by patients at our hospital was substantially reduced (from 50% to 6% of

all MRSA cases) after we implemented a rigorous infection control program based on guidelines from the Centers for Disease Control and Prevention that were adapted from the University Hospital Basel, Switzerland.[6]

We describe the transmission of MRSA by a colonized patient who had not been screened on admission because no risk factors could be identified at that time. A 56-year-old woman (case patient 1) was placed in the same room with 2 other patients. A sputum sample was collected because of her clinical presentation of lower respiratory tract infection on day 4. Two days later (day 6), detection of MRSA in the sputum was reported. Because case patient 1 had stayed with undetected MRSA colonization for more than 3 days in the same room with the 2 other patients, screening of these 2 patients was performed and consisted of nasal and throat swab samples. The nasal swab sample of 1 patient (case patient 2) was found to be positive for MRSA, whereas the screening samples of the other patient were negative. The isolates recovered from both colonized patients were susceptible to clindamycin and erithromycin, which is an extremely rare feature of MRSA isolates detected in our institution (6 of 286 MRSA isolates in past 6 years). Molecular typing of both isolates by pulsed-field gel electrophoresis showed a relatively indistinguishable banding pattern (Figure). After interviewing case patient 1 a second time, we found out that she was in contact with an MRSA carrier before she was admitted to the hospital. She also revealed that she helped case patient 2 (eg, by assisting her with drinking, arranging pillows, and turning her in bed) so as not to call nursing staff repeatedly. We also screened all HCWs who were in contact with both colonized patients and found that none had been colonized. One might argue that HCWs could have transmitted MRSA from one patient to the other and not have colonized themselves at the same time, but this is less likely because the third patient in the room remained negative for MRSA and yet had repeated contacts with HCWs and no contact with case patient 1.

Because this was the second case of probable direct patient-to-patient transmission in our hospital, we prepared an educational leaflet on hand hygiene for patients in which we recommend the use of alcohol-based hand rub during their stay in the hospital. Although no data are available on the role patients play in intrahospital transmission of MRSA, we believe that it should not be dismissed, at least in hospitals where single rooms are not readily available. We should educate our patients on how they can contribute to the global fight against MRSA, and we should give them the opportunity to actively participate in hospital-acquired infection and colonization prevention. With the growing problem of the transmission of community-associated MRSA among patients with no risk factors and no prior connections to healthcare systems, their hand hygiene could be an important factor in the successful prevention of the spread of community-associated MRSA in hospitals. We believe that the active role played by patients in some aspects of infection control could be beneficial and should be addressed in the future.

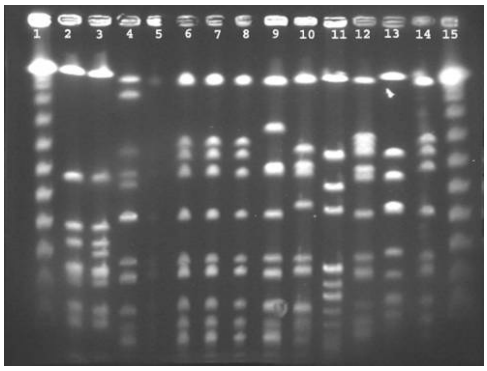

FIGURE. Findings of pulsed-field gel electrophoresis of isolates of methicillin-resistant *Staphylococcus aureus* (MRSA) recovered from patients hospitalized in our institution. *Lanes 1* and *15,* molecular weight markers; *lane 2,* MRSA isolate from case patient 1; *lane 3,* MRSA isolate from case patient 2; *lanes 4–14,* MRSA isolates from patients hospitalized in our institution before, at the same time, and after the 2 patients involved in MRSA transmission.

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Trends in *Stenotrophomonas maltophilia* Bloodstream Infection in Relation to Usage Density of Cephalosporins and Carbapenems During 7 Years

To the Editor—Stenotrophomonas maltophilia is a nonfermentative gram-negative bacillus that causes nosocomial infections, mainly in debilitated and imunocompromised patients.[1,2] In the last decade, this agent has emerged as an important nosocomial pathogen.[3-5] A study conducted from 1997 to 2001, involving 18,569 isolates of nonfermentative gram-negative bacilli, found that *S. maltophilia* was the pathogen isolated third most frequently from clinical specimens.[5] The incidence of infection due to this pathogen ranged from 3.4 to 37.7 cases per 10,000 patients discharged.[2] Prior exposure to antimicrobial agents, particularly β-lactam agents, increases the risk of infection due to *S. maltophilia.*[3] However, the relationship between usage density of β-lactams and the incidence of infection due to *S. maltophilia* remains controversial. The aim of this study was to evaluate the effect of the usage of antipseudomonal third-generation cephalosporins, fourth-generation cephalosporins, and carbapenems on the rates of bloodstream infection caused by *S. maltophilia* during a 7-year period (1999–2006).

This study was conducted at the Hospital das Clinicas, a 945-bed tertiary care university hospital, with 12 intensive care units (ICUs) that have 120 beds, and 3 transplant units, affiliated with the University of São Paulo, Brazil. The hospital has a policy of restriction of use of several antibiotics, including quinolones, third- and fourth-generation cephalosporins, piperacillin-tazobactam, vancomycin, teicoplanin, linezolid, carbapenems, and polymyxins. From 1999 through 2006, cases of *S. maltophilia* bloodstream infection were identified by reports from the hospital infection control committee. The data were prospectively collected by the infection control team, according to National Healthcare Safety Network definitions. Bloodstream infection rates were calculated using the number of patient-days and central line–days in the ICUs and the number of admissions in the non-ICU care areas as denominators. β-lactam use (in milligrams) from 1999 through 2006 was converted into the number of defined daily doses (DDDs) per 1,000 patient-days used in our hospital per year. A defined daily dose is the average daily dose in grams of a specific antimicrobial agent given to an average adult patient. We used the 2008 World Health Organization DDD values for imipenem (2 g), meropenem (2 g), a fourth-generation cephalosporin (cefepime; 2 g), and an antipseudomonal third-generation cephalosporin (ceftazidime; 4 g).[6] Data were analyzed using Epi Info 6.04 software (Centers for Disease Control and Infection). The χ^2 test for linear trend was used to evaluate the trends of incidence of bloodstream infection due to *S. maltophilia* and the use of β-lactam agents (measured in DDDs) during the study period.

From January 1999 through December 2006, data from 12 ICUs, 3 transplant units (kidney, liver, and bone marrow transplant), and 5 general wards were analyzed. The total number of patients hospitalized during the period was 316,080; there were 176,219 patient-days and 124,255 central line–days recorded in the intensive care units. We identified 100 cases of *S. maltophilia* bloodstream infection; 90% of the episodes occurred in the ICUs, and 10% in the non-ICU areas. Of the 90 cases in the ICUs, 33 (36.6%) were located in the medical ICU, 30 (33.3%) in the hematology ICU, 10 (11.1%) in the transplant ICU, 8 (8.8%) in the burn ICU, 6 (6.6%) in the surgical ICU, and 3 (3.3%) in the trauma ICU.

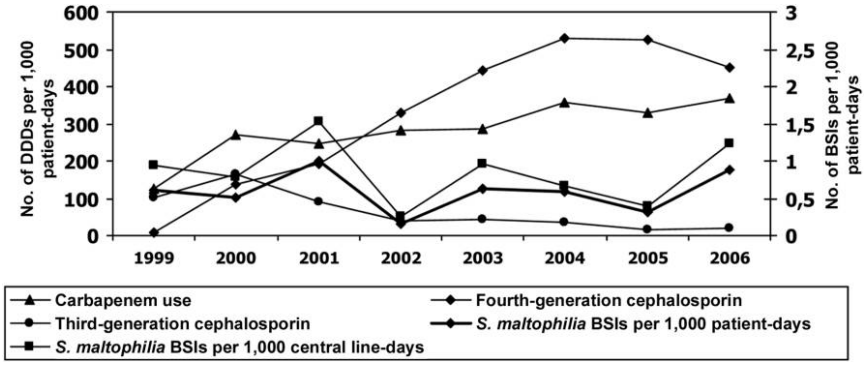

FIGURE. Comparison of median rates of bloodstream infection (BSI) due to *Stenotrophomonas maltophilia* with the use of carbapenems and third- and fourth-generation cephalosporins, in Hospital das Clinicas of the University of São Paulo, during a 7-year period. DDD, defined daily dose.

The incidence of bloodstream infection due to *S. maltophilia* did not change significantly in the intensive care units over time, ranging from 0.16 to 1.0 episode (median, 0.58 episode) per 1,000 patient-days ($P = .5$) and from 0.25 to 1.24 episodes (median, 0.84 episode) per 1,000 central line–days ($P = .1$) (Figure). In the hematology ICU and the marrow transplant units, the number of bloodstream infections due to *S. maltophilia* per 1,000 patient-days ranged from 0 to 1.6 (median, 0.7; $P = .08$).

The use of imipenem increased significantly during the study period, from 82.4 DDDs per 1,000 patient-days in 1999 to 208.4 DDDs per 1,000 patient-days in 2006 ($P < .001$). The use of meropenem also increased, from 41.2 to 160.1 DDDs per 1,000 patient-days ($P < .001$), and the use of cefepime increased from 7.8 to 449.5 DDDs per 1,000 patient-days ($P < .001$). The use of ceftazidime during the study period decreased significantly from 100.3 to 17.9 DDDs per 1,000 patient-days ($P < .001$). In the hematology unit, the use of imipenem increased from 56.8 to 152.5 DDDs per 1,000 patient-days ($P < .001$), and the use of meropenem increased from 117.4 to 428.8 DDDs per 1,000 patient-days ($P = .001$).

The effect of the use of carbapenem on rates of bloodstream infection due to *S. maltophilia* is controversial.[7,8] Metan et al.[8] showed, using multivariate analysis, that carbapenem use increased the incidence of *S. maltophilia* bloodstream infection. Sanyal et al.,[1] in a Kuwaiti hospital, found that the numbers of *S. maltophilia* isolates increased from 1993 to 1997, and that this change correlated significantly with an increase in the annual consumption of carbapenem. Del Toro et al.,[2] in a multicenter study from Spain, showed that the incidence of *S. maltophilia* infection ranged from 3.4 to 12.1 cases per 10,000 patients discharged. On the other hand, more recent studies have showed a stable incidence of *S. maltophilia* infection. Meyer et al.[4] found that the number of *S. maltophilia* isolates at German intensive care units participating in surveillance of antimicrobial use and resistance in intensive care units did not increase from 2001 to 2004, with a mean incidence of 0.13 isolates recovered per 1,000 patient-days.[4] According to Meyer et al.,[4] overall antibiotic and carbapenem use increased slightly during the 4-year period.

In our hospital, bloodstream infection due to *S. maltophilia* was more frequent in the intensive care unit (90% of cases) than in non–intensive care units. Despite the significant increase in the usage density of fourth-generation cephalosporins and of carbapenems in the hospital, the rate of bloodstream infection due to this pathogen remained stable over the 7-year study period.

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Clinical Features and Treatment Outcomes of Infections Caused by *Sphingomonas paucimobilis*

To the Editor—Sphingomonas paucimobilis isolates have been recovered from diverse sources, including hospital water systems, respiratory therapy equipment, and various clinical specimens.[1] Several case reports and case series of *S. pauci-*

TABLE. Demographic and Clinical Characteristics of 23 Patients with *Sphingomonas paucimobilis* Infection

Patient	Age in years, sex	Underlying condition(s)	Type of infection	Source of isolate	Nosocomial infection	Indwelling device
1	48, M	Hepatocellular carcinoma	Cholangitis	Blood	Yes	None
2	66, M	Asthma, AOSD	Wound infection	Wound	Yes	None
3	28, M	Herpes occipitoradialis	Ear pyoderma	Pus	Yes	None
4	69, M	Lung cancer	Neutropenic fever (pneumonia)	Blood	Yes	None
5	64, F	Breast cancer	Neutropenic fever (unknown focus)	Blood	Yes	Tunneled CVC
6	8, M	ALL	Neutropenic fever (unknown focus)	Blood	Yes	Tunneled CVC
7	50, M	Hepatocellular carcinoma	Catheter-related infection	Catheter tip	Yes	Nontunneled CVC
8	52, M	Lymphoma	Catheter-related infection	Blood	Yes	Tunneled CVC
9	59, F	AML	Catheter-related infection	Blood	Yes	Tunneled CVC
10	1, M	Anaplastic ependymoma	Catheter-related infection	Catheter tip	Yes	Nontunneled CVC
11	56, F	Multiple myeloma	Catheter-related infection	Blood	Yes	Tunneled CVC
12	17, M	Ewing sarcoma	Catheter-related infection	Blood	Yes	Tunneled CVC
13	47, M	Lymphoma	Catheter-related infection	Blood	Yes	Tunneled CVC
14	48, F	Breast cancer	Catheter-related infection	Blood	Yes	Tunneled CVC
15	55, M	ESRD	CAPD peritonitis	Dialysate	Yes	CAPD catheter
16	62, M	ESRD	CAPD peritonitis	Dialysate	Yes	CAPD catheter
17	14, F	ESRD	CAPD peritonitis	Dialysate	Yes	CAPD catheter
18	<1, F	Chylothorax	Primary bacteremia	Blood	Yes	Nontunneled CVC
19	71, F	Head and neck cancer	Primary bacteremia	Blood	Yes	Nontunneled CVC
20	2, F	Aplastic anemia	Primary bacteremia	Blood	Yes	Tunneled CVC
21	<1, M	Neonatal sepsis	Primary bacteremia	Blood	Yes	Nontunneled CVC
22	45, F	None	GI infection	Blood	No	None
23	27, F	None	GI infection	Blood	No	None

NOTE. ALL, acute lymphocytic leukemia; AML, acute myelocytic leukemia; AOSD, adult-onset Still disease; CAPD, continuous ambulatory peritoneal dialysis; CVC, central venous catheter; ESRD, end-stage renal disease (and receiving peritoneal dialysis); GI, gastrointestinal.

mobilis infection have been published.[2-8] However, little is known about the clinical features of *S. paucimobilis* infections. Thus, we retrospectively analyzed patients with infections caused by *S. paucimobilis* to evaluate the clinical features and treatment outcomes associated with this pathogen.

The database at the clinical microbiology laboratory was reviewed to identify patients who had *S. paucimobilis* infection from January 2000 through September 2007 at Samsung Medical Center, Seoul, Republic of Korea. Patients were included in the study if a culture was positive for *S. paucimobilis*, and their medical records were reviewed. Only true infection for each patient was included in the analysis.

We defined clinically significant *S. paucimobilis* infection as recovery of *S. paucimobilis* from culture of specimens from patients with clinical features compatible with systemic inflammatory response syndrome.[9] We defined antibiotic therapy as inappropriate if an antibiotic agent active against *S. paucimobilis* (as determined by in vitro susceptibility testing) at the usual recommended dosage was not administered during the first 48 hours after diagnosis of infection. The definition of catheter-related infection required the presence of no apparent source for the bacteremia except the central venous catheter and required the isolation of the organism in semiquantitative culture (more than 15 colony-forming units of *S. paucimobilis* recovered from a culture of the central venous catheter tip). Possible catheter-related infection was indicated by the finding of a positive blood culture result with no apparent source of the bacteremia except the catheter.

The recovery of *S. paucimobilis* from specimens was accomplished by the processing of blood cultures, body fluids, or catheters in a Bactec Model 9240 (Becton-Dickinson) or BacT/ALERT 3D (bioMérieux). Identification of *S. paucimobilis* and antibiotic susceptibility testing were performed on the Vitek II automated system (bioMérieux).

During the study period, a total of 79 isolates of *S. paucimobilis* were identified. The patients corresponding to 23 of these isolates were enrolled; 56 patients were excluded, because their isolates were considered to represent colonization or contamination. The mean age of patients ($\pm$SD) was 38.7 $\pm$ 24.8 years, and 15 patients (65.2%) were male. The most common types of infection were catheter-related infection (in 8 patients [34.8%]), followed by primary bacteremia (in 6 [26.1%]), continuous ambulatory peritoneal dialysis peritonitis (in 3 [13.0%]), and gastrointestinal infection (in 2 [8.7%]) (Table). Of the 8 catheter-related infections, 2 were definitely related to catheters and 6 were possibly related to catheters. Six of these infections (75.0%) were cured without catheter removal. Central venous catheters were removed from 2 patients for cure.

Of the *S. paucimobilis* isolates, 13.6% (3 of 22) were resistant to amikacin; 20.0% (4 of 20) were resistant to cefo-

taxime; 4.5% (1 of 22) were resistant to imipenem; 21.7% (5 of 23) were resistant to ciprofloxacin; and 18.1% (4 of 22) were resistant to the combination of trimethoprim and sulfamethoxazole. (Not all antimicrobials were tested in all isolates.) Twenty-one patients (91.3%) were classified as having nosocomial infection. Only 2 patients (9.7%) were considered to have community-acquired infection; both of the patients had infectious colitis and did not have underlying disease.

All patients received initial empirical antibiotic therapy: broad spectrum cephalosporins with or without aminoglycosides (15 patients); fluoroquinolones (4); first- or second-generation cephalosporins (2); carbapenem (1); and a glycopeptide (1). Of 23 patients, 10 (43.5%) received inappropriate initial empirical antibiotic therapy. However, all patients survived despite inappropriate initial therapy. The presence of atypical lipopolysaccharide constitute bound to the outer membrane of *S. paucimobilis,* with the accompanying deficiency of endotoxin activity, has been proposed to explain the low virulence of *S. paucimobilis.*[1,2] The favorable outcome in our study (all cases survived despite initial inappropriate antibiotic treatment) may support the conclusion that *S. paucimobilis* has a low virulence.

Infections caused by *S. paucimobilis* are usually associated with the use of various indwelling devices, according to the case reports.[2,5,8] This study revealed that two-thirds of patients (17 [73.9%] of 23) had an indwelling device, including central venous catheters and continuous ambulatory peritoneal dialysis catheters. The catheter-related infections caused by *S. paucimobilis* had a good clinical outcome, mostly without catheter removal, in this study.

Most *S. paucimobilis* infections reported in the literature have been nosocomial infections or have been related to nosocomial outbreaks.[2,4,5] This trend was true in the present study as well. There were 2 patients with community-acquired infection; both were admitted to the emergency department with fever and diarrhea, and neither had any healthcare-associated risk factors or underlying diseases. To our knowledge, this is the first report about *S. paucimobilis* as a cause of diarrheal disease in immunocompetent hosts.

The *S. paucimobilis* isolates in this study exhibited antibiotic susceptibility trends that differed from those in other studies. Previous reports suggested that third-generation cephalosporins or aminoglycosides are the antibiotics of choice for the treatment of infection caused by this organism.[1,10] However, 20.0% of the isolates in our study were resistant to cefotaxime, and 13.6% were resistant to amikacin. Carbapenems were the most effective therapy in our study. These differing results reinforce the need to treat these infections with individualized antibiotic therapy, guided by the in vitro susceptibility of each clinical isolate.

Even though we examined only 23 patients with *S. paucimobilis* infection, this is the first study, to our knowledge, to evaluate the clinical features and treatment outcomes of *S. paucimobilis* infections in more than 10 patients.

In summary, our results showed that most *S. paucimobilis* infections are nosocomial and that they are commonly associated with indwelling medical devices. Clinicians should consider *S. paucimobilis* a notable hospital-acquired pathogen, especially in cases involving a device-related infection.

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Surveillance for Mupirocin Resistance Among Methicillin-Resistant *Staphylococcus aureus* Clinical Isolates

To the Editor—Mupirocin is an antimicrobial agent that has a unique mechanism for inhibiting the synthesis of proteins: it selectively binds to bacterial isoleucyl-tRNA synthetase.[1] It has been widely used in an effort to decolonize methicillin-resistant *Staphylococcus aureus* (MRSA) carriers, as a means of controlling the spread of this pathogen. The prevalence of mupirocin resistance among MRSA isolates varies considerably. A significant increase in mupirocin resistance following the widespread use of mupirocin has been reported,[2] and some centers have observed high rates of mupirocin resistance despite low rates of mupirocin use.[3]

In 2007, OSF St. Francis Medical Center, a tertiary care hospital in central Illinois, implemented a comprehensive infection control program that included identifying MRSA colonization among high-risk patients and using topical mupirocin ointment to eradicate MRSA carriage. This program was used in conjunction with other practices, which included patient isolation and cohorting, education, and hand hygiene, to control the spread of multidrug-resistant pathogens. Surveillance for mupirocin resistance was begun in anticipation of the possibility that the emergence of mupirocin-resistant isolates could limit the therapeutic options available for the control and prevention of MRSA infections.

From July through October 2007, a total of 156 nonduplicate MRSA clinical isolates were consecutively collected from the OSF System Laboratory at St. Francis Medical Center. The isolates were screened for mupirocin resistance using a 5-μg and a 20-μg mupirocin disk. The minimum inhibitory concentration (MIC) of mupirocin was measured using Etest strips (AB Biodisk). Isolates were classified as being susceptible (ie, having an MIC of 4 μg/mL or less), having low-level resistance (an MIC of 8–256 μg/mL), or having high-level resistance (an MIC of 512 μg/mL or greater). Multiplex polymerase chain reaction amplification was performed for the simultaneous detection of genes to identify *S. aureus* (*nuc* gene), methicillin resistance (*mecA* gene), and high-level mupirocin resistance (*mupA* gene), as described elsewhere.[4]

Mupirocin resistance was detected in 37 (23.7%) of the 156 MRSA clinical isolates. Of these, 29 isolates (18.6%) exhibited low-level resistance, and 8 isolates (5.1%) exhibited high-level resistance. Mupirocin-resistant isolates were recovered from various clinical specimens, including blood (7), sputum (3), urine (3), skin and soft tissues (18), and specimens from other body sites (6). There were no significant differences between mupirocin-susceptible and mupirocin-resistant isolates with regard to specific sites of isolation. Reduced susceptibility to non–β-lactam antimicrobial agents (erythromycin, clindamycin, levofloxacin, and gatifloxacin) was common among mupirocin-resistant isolates; however, there were no significant differences, compared with mupirocin-susceptible isolates, except with regard to susceptibility to clindamycin (detected in 70.3% of mupirocin-resistant isolates vs 48.7% of mupirocin-susceptible isolates; $P = .02$). Most of the MRSA isolates (more than 95%) were susceptible to gentamicin, trimethoprim-sulfamethoxazole, and tetracycline, regardless of mupirocin susceptibility. A multiplex polymerase chain reaction was performed for 32 MRSA isolates exhibiting mupirocin resistance. The *mupA* gene was detected in all 8 isolates with high-level mupirocin resistance but was not detected in isolates with low-level mupirocin resistance.

Because there are no Clinical and Laboratory Standard Institute guidelines for interpretive criteria for mupirocin, several investigators have proposed interpretive criteria using disks with various concentrations of mupirocin (Table 1). Using a 5-μg mupirocin disk, Finlay et al.[5] found that isolates with a zone-of-inhibition diameter of 14 mm or more were mupirocin susceptible. We found 6 MRSA isolates that were more accurately classified by the Etest as having low-level resistance (16–24 μg/mL). The reliability of using a 5-μg mupirocin disk to detect mupirocin-resistant isolates improved when a larger zone-of-inhibition diameter was applied. Using the breakpoint of 19 mm or more advocated by Creagh and Lucey,[6] we did not observe any very major errors (ie, false-susceptible test results). A 20-μg mupirocin disk was evaluated as a tool to discriminate between low-level and high-level mupirocin resistance. As suggested by the British Society for Antimicrobial Chemotherapy,[7] the breakpoints should be 6 mm or less for high-level resistance and 7–26 mm for low-level resistance. Using these breakpoints, we observed no very major errors. However, we did observe major errors (ie, false-resistant test results) in several isolates that were classified as having low-level mupirocin resistance, on the basis of the

TABLE 1. Disk Diffusion Testing Interpretive Criteria for Mupirocin (Mpc) Susceptibility Proposed by Various Investigators

Study, concentration of Mpc in disk	Zone-of-inhibition diameter, mm, by susceptibility level		
	Susceptible	Low resistance	High resistance
Finlay et al. [5]			
5 μg	$\geqslant$14	$\leqslant$13	$\leqslant$13
Creagh and Lucey [6]			
5 μg	$\geqslant$19	$\leqslant$18	$\leqslant$18
Andrews et al. [7]			
5 μg	$\geqslant$22	$\leqslant$21	$\leqslant$21
20 μg	$\geqslant$27	7–26	$\leqslant$6
Palepou et al. [8]			
25 μg	>26		<10
de Oliveira et al. [9]			
5 μg	$\geqslant$14	$\leqslant$13	$\leqslant$13
200 μg	$\geqslant$14	$\geqslant$14	<14

zone-of-inhibition diameter, but were also classified as being mupirocin susceptible, on the basis of MICs using the Etest.

Our study demonstrated that there was a high prevalence of mupirocin resistance among MRSA clinical isolates, compared with other surveillance studies that found a geographic variation in the prevalence of mupirocin resistance among MRSA isolates that ranged from 4% to 17%.[10] Although the prevalence of mupirocin resistance prior to this finding was not known, it is plausible that the higher rate could be the consequence of frequent exposure to mupirocin.

The determination of MICs by the Etest is simple, reproducible, and shows a good correlation with agar dilution testing but is relatively expensive. Disk diffusion testing is cheap and easy to perform, but defining breakpoints for mupirocin susceptibility remains problematic. The finding of inconsistent results, which were mainly the result of the different methods used, makes it difficult to judge which interpretive criteria should be applied. We observed the limited usefulness of the disk diffusion method, finding that the susceptibility of the pathogens could not be predicted accurately. Nevertheless, diffusion testing using the 5-μg mupirocin disk has potential as a screening method for prediction of mupirocin resistance. The breakpoint, however, should be 18 mm or less, to increase sensitivity, as proposed by Creagh and Lucey.[6] If the diameter of the inhibition zone is 18 mm or less, an Etest can then be performed to distinguish between high-level and low-level mupirocin resistance.

Although this study was limited by the relatively small number of clinical isolates, the finding of a high rate of mupirocin resistance supports the need to establish effective infection control measures. Further work is required to determine the impact of widely used topical mupirocin and the burden of mupirocin-resistant isolates.

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