Methicillin-Resistant *Staphylococcus aureus* Nosocomial Acquisition and Carrier State in a Wound Care Center

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**Objective:** To assess methicillin-resistant *Staphylococcus aureus* (MRSA) nosocomial acquisition and carrier state in a wound care center.

**Design and Setting:** The results of an intervention to control MRSA were compared with those of historical controls at the wound care center of university-based Hôpital Broussais, Paris, France.

**Patients:** Patients admitted for specific care of chronic ulcers and surgical wounds.

**Main Outcome Measures:** Incidence rates of MRSA carriers and acquisition in wounds.

**Results:** Of 88 patients admitted during a 3-month preintervention period in 1993, 18 (21%) were MRSA carriers. Of 334 patients admitted in 1994 and 395 in 1996, 65 (19.5%) and 81 (20.5%) were MRSA carriers, respectively (P = .80). In 1993, 6 (9%) of 70 patients without MRSA acquired MRSA wound infections; the corresponding numbers were 6 (2.2%) of 269 in 1994 and 3 (0.9%) of 314 in 1996. Despite that the number of MRSA carriers remained stable at admission to the wound care center, the rate of MRSA infections in wounds per 100 non-carriers decreased significantly between the preintervention period and subsequent years: 1994 (P = .02) and 1996 (P = .002).

**Conclusions:** Although our results are limited by the use of historical controls, they showed that simple infection control measures, such as the use of soap and water and barrier precautions associated with staff education, seemed to significantly reduce MRSA infection rates in patients with chronic skin breaks.

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**ETHICILLIN-resistant *Staphylococcus aureus* (MRSA) is increasingly common in hospitals worldwide and has recently spread to community populations.** In 1993-1996 in French hospitals, the proportion of *S aureus* resistant to methicillin was 40%, and the infection rate was 0.45% in a recent multicenter survey. The high endemicity of MRSA poses a problem for drug therapy because of the possible development of resistance to glycopeptides, which could lead to untreatable infections. In addition, MRSA is a financial burden on the health care system. As this organism can spread easily from person to person, MRSA is introduced into hospitals by the admission of a patient with infection or colonization who serves as a reservoir. The control of MRSA requires the implementation of infection control measures, particularly inside high-risk wards, including rapid detection of MRSA by screening every patient at risk at admission, identifying MRSA carriers using specific labeling, rigorous hand washing by staff, isolation of patients with colonization and infection, and MRSA eradication, if possible, by the use of antiseptic agents.

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Open wounds, particularly when they are chronic, provide a portal of entry for MRSA to the underlying tissues, which can readily lead to local or generalized infection. In addition, the presence of wounds in geriatric patients is a risk factor for MRSA colonization at any other site. Recently, pressure ulcers and surgical wounds were found to be independently associated with an increased rate of subsequent MRSA infection in general hospital populations. The epidemiology of MRSA colonization and infection in wounds has been studied in the elderly, but no data are available for large cohorts of patients with miscellaneous wounds. The aim of this study was to assess the MRSA nosocomial acquisition and carrier state in a large cohort of patients admitted to a ward for...
PATIENTS AND METHODS

SETTING AND PATIENTS

The wound care center (WCC) of university-based Hôpital Broussais, Paris, France, is a vascular rehabilitation ward with 51 beds and 350 to 400 admissions per year. It is dedicated to the care of chronic ulcers (venous ulcers, arterial ulcers, and neurotrophic ulcers) and wounds due to vascular surgery (distal amputation and bypass graft). The study population was composed of all patients admitted in 1994 and 1996, with a mean length of stay of 35 days.

STUDY DESIGN

We conducted a 3-month incidence survey from September 1, 1993, to November 30, 1993, consisting of systematic recording of wound infections acquired in the ward. The cohort included 88 patients, of whom 14 patients (16%) acquired wound infections due to various microorganisms. Of these 88 patients, 70 did not have MRSA at admission. Of these 70 patients, 6 (9%) acquired MRSA wound infections during their stay.

From January 1, 1994, to December 31, 1994, a program to control MRSA was implemented, including specific control measures and a continuous 12-month survey to assess the incidence of both MRSA carrier state in wounds and nosocomial acquisition. A second 12-month survey was conducted from January 1, 1996, to December 31, 1996, using the same methods to assess the medium-term efficacy of the control program.

DEFINITIONS

Methicillin-resistant S. aureus carrier was defined as a patient with MRSA in wound cultures obtained less than 48 hours after admission. Methicillin-resistant S. aureus acquisition was defined as the isolation of MRSA in samples taken 48 hours after admission from former noncarriers. According to the Centers for Disease Control criteria,13 wound infection was defined as the presence of 2 of the following clinical signs (localized pain or tenderness, swelling, and redness or heat) and isolation of MRSA in pure culture. If both clinical and microbiological criteria were not met, the presence of MRSA in wound cultures was considered colonization.

SURVEILLANCE METHODS

A standard form was used to collect data for the inpatients. The information gathered included date of admission, age, admission category, underlying diseases (eg, diabetes mellitus), type of wound, site of acquisition, and clinical and microbiological features of infection. Each month, all cases involving MRSA (carrier at admission or acquisition) were evaluated by the WCC staff and the infection control team. The classification of patients (MRSA carriers or patients with MRSA acquisition) depended on the date that cultures positive for MRSA were obtained (at admission or 48 hours after admission). The time to acquisition was noted. All nosocomial wound infections (including bacteria other than MRSA) were also recorded during the study period. All results were reported monthly by the infection control team to the WCC staff.

SPECIFIC CONTROL MEASURES

Before the intervention, the systematic management of wounds consisted of regular attention to changes in the appearance of wounds and regular care with Vaseline gauze dressings. Since January 1, 1994, infection control measures have been implemented in the ward.

For management of all wounds, we implemented the following measures: (1) the revision of nursing procedures, including proper and rigorous hand washing using soap and water, (2) the use of gloves and gowns by personnel who have direct contact with wounds, (3) the systematic use of disposable devices, and (4) the washing of the extremities with soap and water during dressing changes.

In addition, for specific management of wounds with MRSA, the following measures were implemented: (1) the staff of the microbiology laboratory labeled patients’ laboratory results and charts when a culture was positive for MRSA, (2) the staff were educated about the risk of cross-transmission, (3) regular feedback was given to the staff about new cases of MRSA, and (4) when changing dressings, patients with MRSA were treated after patients without MRSA.

Strict patient isolation was not possible because there are only 11 single rooms in the ward.

MICROBIOLOGICAL METHODS

Samples were obtained using cotton-tipped swabs from all skin wounds at admission and all wounds during the stay, if clinical worsening occurred. The samples were delivered to the microbiology laboratory, placed on blood agar plates, and incubated at 37°C for 72 hours. Using standard laboratory procedures, MRSA was isolated and identified. The detection of methicillin resistance was performed according to the methods recommended by the Comité de l’Antibiogramme de la Société Française de Microbiologie.14

CASE-CONTROL STUDY

A case-control study was conducted to identify specific risk factors for MRSA wound infections. Cases were patients infected with MRSA and controls, patients with MRSA colonization during the same period.

ANALYSIS OF DATA

The carrier incidence rates were calculated by dividing the number of MRSA carriers by the number of admissions to the ward; it was expressed per 100 admissions.

The acquisition incidence rates were expressed in 2 different ways. The rate was first calculated by dividing the number of patients who acquired MRSA at the hospital by the number of patients without MRSA at admission and expressed per 100 noncarriers. It was also calculated by dividing the number of patients who acquired MRSA at the hospital by the total length of stay of all patients admitted to the ward during the period; it was expressed per 1000 resident-care days.

Incidence data were compared using the χ² test or Fisher exact test. In the risk factor analysis, qualitative data were compared using Fisher exact test, and quantitative data using the t test. All tests were 2-tailed; P≤.05 was considered significant.
treatment of chronic ulcers and postoperative wounds. The effect of an intervention to control MRSA was assessed by comparing the results before (historical controls) and after the intervention.

### RESULTS

#### CARRIERS

Of 334 admitted patients in 1994, 65 (19.5%) were MRSA carriers; of 395 admitted patients in 1996, 81 (20.5%) were MRSA carriers ($P = .80$). The characteristics of MRSA carriers in 1994 and 1996 were similar with respect to age, prevalence of diabetes mellitus, admission category, and type of skin breaks (Table 1). The characteristics were also comparable to those of all patients admitted to the ward during the same period (data not shown).

#### ACQUISITION

Of 269 patients without MRSA in 1994, 12 patients (4.5%) acquired MRSA, including 6 with colonization (2.2%) and 6 with infection (2.2%); the colonization and infection rates were 0.39 per 1000 resident-care days. Of 314 patients without MRSA in 1996, 11 patients (3.5%) acquired MRSA, including 8 with colonization (2.5%) and 3 with infection (0.9%); the colonization and infection rates were 0.61 and 0.23 per 1000 resident-care days, respectively. The ages, prevalence of diabetes mellitus, and type of skin breaks in patients who acquired MRSA were similar in 1994 and 1996 (Table 2).

There was a significant decrease in the rate of MRSA wound infections per 100 noncarriers between the preintervention period in 1993 (6 [19%] of 70) and the postintervention period in 1994 (6 [2.2%] of 269; $P = .02$) and 1996 (3 [0.9%] of 314; $P = .002$).

### RISK FACTORS FOR INFECTION VS COLONIZATION

The 9 patients with infection were compared with the 14 patients with colonization (Table 3), and the patients with infection were similar to those with colonization. There was no statistically significant difference between the 2 groups in terms of age, prevalence of diabetes mellitus, nature, number and duration of wounds, wound grafts, frequency of antibiotic therapy before hospitalization, presence of simultaneous infectious sites, presence of invasive devices, and prior hospital admissions. Many of the members of both the group with infection and the group with colonization had previously

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### Table 1. Characteristics of MRSA Carriers*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>1994 (n = 65)</th>
<th>1996 (n = 81)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean ± SD</td>
<td>76 ± 22</td>
<td>72 ± 14</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>22 (33)</td>
<td>27 (33)</td>
</tr>
<tr>
<td>Admission from other ward of hospital</td>
<td>22 (34)</td>
<td>33 (41)</td>
</tr>
<tr>
<td>Other hospital</td>
<td>36 (55)</td>
<td>33 (41)</td>
</tr>
<tr>
<td>Community</td>
<td>7 (11)</td>
<td>13 (16)</td>
</tr>
<tr>
<td>Day-care center</td>
<td>0 (0)</td>
<td>2 (2)</td>
</tr>
<tr>
<td>Medical admission</td>
<td>46 (71)</td>
<td>69 (85)</td>
</tr>
<tr>
<td>Surgical admission</td>
<td>19 (29)</td>
<td>12 (15)</td>
</tr>
<tr>
<td>Skin breaks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arterial ulcer</td>
<td>14 (17)</td>
<td>24 (22)</td>
</tr>
<tr>
<td>Venous ulcer</td>
<td>14 (17)</td>
<td>26 (24)</td>
</tr>
<tr>
<td>Mixed ulcer</td>
<td>22 (27)</td>
<td>11 (10)</td>
</tr>
<tr>
<td>Neurotrophic ulcer</td>
<td>2 (3)</td>
<td>3 (3)</td>
</tr>
<tr>
<td>Distal amputation</td>
<td>17 (21)</td>
<td>18 (17)</td>
</tr>
<tr>
<td>Bypass graft</td>
<td>3 (4)</td>
<td>10 (9)</td>
</tr>
<tr>
<td>Other</td>
<td>9 (11)</td>
<td>16 (15)</td>
</tr>
</tbody>
</table>

*Data are presented as the number (percentage) of patients unless otherwise indicated. MRSA indicates methicillin-resistant Staphylococcus aureus.

### Table 2. Characteristics of Patients Who Acquired MRSA*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>1994 (n = 12)</th>
<th>1996 (n = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean ± SD</td>
<td>67 ± 16</td>
<td>75 ± 10</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>5 (42)</td>
<td>4 (36)</td>
</tr>
<tr>
<td>Skin breaks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arterial ulcer</td>
<td>3 (23)</td>
<td>2 (20)</td>
</tr>
<tr>
<td>Venous ulcer</td>
<td>5 (39)</td>
<td>3 (30)</td>
</tr>
<tr>
<td>Mixed ulcer</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Neurotrophic ulcer</td>
<td>2 (15)</td>
<td>2 (20)</td>
</tr>
<tr>
<td>Distal amputation</td>
<td>3 (23)</td>
<td>3 (30)</td>
</tr>
<tr>
<td>Bypass graft</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Other</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Time to acquisition, y, mean ± SD</td>
<td>34.9 ± 18.7</td>
<td>33.5 ± 23.7</td>
</tr>
</tbody>
</table>

*Data are presented as the number (percentage) of patients unless otherwise indicated. MRSA indicates methicillin-resistant Staphylococcus aureus.

### Table 3. Comparison of Patients With MRSA Infection and Colonization*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Colonization (n = 14)</th>
<th>Infection (n = 9)</th>
<th>$P$†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 75 y</td>
<td>6 (43)</td>
<td>4 (44)</td>
<td>&gt;.99</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>5 (36)</td>
<td>2 (22)</td>
<td>.70</td>
</tr>
<tr>
<td>Major arteritis</td>
<td>4 (29)</td>
<td>6 (67)</td>
<td>.10</td>
</tr>
<tr>
<td>Ulcers</td>
<td>12 (86)</td>
<td>5 (56)</td>
<td>.16</td>
</tr>
<tr>
<td>Surgical wounds</td>
<td>2 (14)</td>
<td>4 (44)</td>
<td>.16</td>
</tr>
<tr>
<td>No. of wounds &gt; 3</td>
<td>5 (36)</td>
<td>2 (22)</td>
<td>.65</td>
</tr>
<tr>
<td>Delay of wound healing &gt; 12 mo</td>
<td>11 (79)</td>
<td>4 (44)</td>
<td>.18</td>
</tr>
<tr>
<td>Wound grafts &lt; 1 mo</td>
<td>7 (50)</td>
<td>5 (56)</td>
<td>&gt;.99</td>
</tr>
<tr>
<td>Prior antibiotic therapy &lt; 1 mo</td>
<td>12 (86)</td>
<td>7 (78)</td>
<td>&gt;.99</td>
</tr>
<tr>
<td>Other infectious sites</td>
<td>1 (7)</td>
<td>3 (33)</td>
<td>.26</td>
</tr>
<tr>
<td>Presence of invasive devices</td>
<td>3 (21)</td>
<td>6 (67)</td>
<td>.08</td>
</tr>
<tr>
<td>Prior hospital admissions &lt; 6 mo</td>
<td>11 (79)</td>
<td>9 (100)</td>
<td>.25</td>
</tr>
<tr>
<td>Time to acquisition, d, mean ± SD</td>
<td>37.6 ± 19.9</td>
<td>29.0 ± 22.2</td>
<td>.37</td>
</tr>
</tbody>
</table>

*Data are presented as the number (percentage) of patients unless otherwise indicated. MRSA indicates methicillin-resistant Staphylococcus aureus.
†Two-tailed Fisher exact test for qualitative data and the t test for quantitative data.
received antibiotics (78% vs 86%) and had been hospitalized before their present admission (100% vs 79%).

**NOSOCOMIAL WOUND INFECTIONS**

A total of 34 wound infections (23 in 1994 and 11 in 1996) were acquired by the patients in the WCC, with the isola-
tion of 37 microorganisms. *Staphylococcus aureus* was the main causative bacterium, with the isolation of 21 microorganisms (57%), and MRSA was responsible for 9 episodes (27% of all nosocomial wound infections). The other organisms were *Streptococcus agalactiae* (n=7), *Pseudomonas aeruginosa* (n=5), *Streptococcus pyogenes* (n=2), *Proteus mirabilis* (n=1), and *Coryne-
bacterium* species (n=1). *Pseudomonas aeruginosa* and methicillin-sensitive *S aureus* were responsible for 2 mixed infections.

**COMMENT**

By comparing the 2 survey periods, our study revealed that the rate of MRSA carriers (20%) at admission re-
maind stable. This percentage confirms that patients ad-
mitted to the WCC were at particularly high risk for MRSA acquisition, probably because they had a combination of several known risk factors, such as old age, previous and prolonged hospital stays, previous antibiotic treatment, and diabetes mellitus (some patients), and had under-
gone invasive procedures.13 Girou et al16 studied samples from the nose and/or wounds and/or perineum in a sub-
group of patients with generalized dermatoses in a der-
matological intensive care unit in Paris. They reported a similar rate of MRSA carriers at admission (18%). Our results are consistent with data obtained from 9 studies in nursing homes from 1982 to 1996 cited by Bradley,12 who reported rates of MRSA carriers at admission of 10% to 54%. Because the MRSA carrier state often lasts sev-
eral months, patients with MRSA frequently have colo-
nization at readmission.17 In our ward, 40% to 50% of the patients are readmitted for care of chronic wounds and are often transferred during their hospital stay be-
tween various wards. Because of cost and personnel con-
siderations, selective screening of MRSA carriers in French university hospitals is required. However, the way of select-
ing the population to be screened has not been de-
termined.18 Our data suggest that patients with chronic ulcers and postoperative wounds should be identified, screened, and isolated in all wards. Other subpopula-
tions need to be defined as well. Such targeted strategies could prevent the spread of MRSA within hospitals and also possibly in the community where MRSA emerged recently.19

We found a 3.5% to 4.5% acquisition rate in pa-
tients with chronic skin breaks. These results are con-
sistent with Girou et al,16 who found a 3.6% MRSA ac-
quisition rate in patients with various skin diseases and an 8.5% rate among patients with generalized dermato-
es. Our methods differed from those of Girou et al, who considered both true infections and colonizations with-
out clinical signs. We performed bacterial cultures only if clinical worsening occurred, which did not allow us to calculate precisely the total MRSA colonization rates (with and without clinical signs) and thus underesti-
imated the true rate of MRSA acquisition. Only system-
atric screening, for instance weekly, could provide information about true MRSA colonization rates. A cost-
benefit analysis would be required to assess whether this is warranted.

In many patients with skin breaks, it is doubtful whether there is a clinical wound infection because loc-
alsigns are misleading, especially if they are not found in combination with other symptoms20 that justify bacterial sample analysis. Among 23 patients with clinical signs, only 9 (39%) with the isolation of MRSA were re-
tained to a true local infection according to the Centers for Disease Control criteria,13 which led to a possible sys-
tematic treatment. Despite the limitations of the risk factor analysis due to the small numbers of patients in the 2 groups, the failure to identify a specific factor for in-
flection (Table 3), particularly diabetes mellitus, which is a known risk factor for wound infection,21 was an un-
expected finding in our study. In wounds, colonization and infection may be 2 stages of the same pathological process, the infection state resulting from an imbalance between the pathogen's virulence and the host's defense factors.22

As *S aureus* is part of the commensal flora and no normal microbial flora in leg ulcers can be established,20 optimal therapy for such wounds with MRSA coloniza-
ion has not been defined. In our ward, careful local care, mainly based on washing and wound cleaning, seems to be beneficial enough to avoid the need for systemic antibi-
totics when wounds have MRSA colonization. A regu-
lar and particular vigilance for the development of new local signs in patients with skin breaks is essential for the early detection of such pathogenic colonizations.

This study showed a significant decrease in the inci-
cidence of infection between the period before the imple-
mentation of the control program and subsequent years, 1994 (P = .02) and 1996 (P = .002). Many measures have been recommended for controlling the spread of MRSA in hospitals. However, there is controversy regarding which measures should be preferentially implemented. Our intervention to control MRSA consisted of system-
atric wound management associated with the strict ap-
plication of simple barrier precautions, such as using gloves and gowns and regularly washing the hands and skin with soap and water. These simple measures could be applied in most long-term care or rehabilitation fac-
cilities with high MRSA incidence. Other proposed mea-
ures,23 such as cohort nursing or placing all MRSA pa-
tients in single rooms or in separate wards, are not easily applicable in most facilities.

Although the results of our study are limited by the use of historical controls, they showed that simple in-
flection control measures, such as the use of soap and wa-
ter and barrier precautions associated with staff educa-
tion, seemed to significantly reduce MRSA infection rates in patients with chronic skin breaks.

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