Enterococcus faecium Bacteremia

Does Vancomycin Resistance Make a Difference?

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Background: Enterococcus faecium has received increased attention, primarily due to the emergence of vancomycin resistance. The purpose of this investigation was to study the epidemiological characteristics of vancomycin-resistant E faecium (VRE) bacteremia and to determine the clinical impact of vancomycin resistance on the outcome of patients with this infection.

Methods: We retrospectively analyzed the clinical features and outcome of 53 patients with E faecium bacteremia.

Results: From January 1992 until December 1995, there were 32 episodes of bacteremia caused by vancomycin-susceptible E faecium (VSE) and 21 caused by VRE. An intra-abdominal site was the most common source of bacteremia in both groups. All of the VRE and 78% of VSE bacteremia cases were nosocomially acquired. Previous administration of vancomycin was associated with VRE bacteremia (P = .001), as were indwelling bladder catheters (P = .01). Fifty-nine percent of the patients with VSE bacteremia survived vs 24% with VRE (P = .009), despite similar severity-of-illness scores. In 62% of the patients with VRE sepsis, death was related to the bacteremia (P = .01). Patients infected with VRE had longer hospitalizations than those with VSE (34.8 vs 16.7 days, respectively) (P = .004), were more likely to be on the medical service (P = .03), and on the average, had hospitalization costs of more than $27 000 per episode than did patients with VSE bloodstream infection ($83 897 vs $56 707, respectively) (P = .04).

Conclusions: Vancomycin-resistant E faecium bacteremia is a complication of prolonged hospitalization in debilitated patients. Vancomycin resistance has a negative impact on survival in patients with E faecium bacteremia and leads to higher health care costs.

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Enterococcus faecium has grown in importance as a nosocomial pathogen during the past decade.1-2 While there once was controversy regarding the pathogenic role of enterococci in causing infection, mounting evidence has now established E faecium as a cause of significant morbidity and mortality in hospitalized patients who are infected with this organism.3-6 Notorious for resistance to multiple antimicrobial agents, the acquisition of vancomycin resistance has made this organism a formidable pathogen.

Vancomycin-resistant enterococci (VRE), first described in 1988,7 are a growing problem as more hospitals throughout the United States and Europe report significant outbreaks associated with this organism.8-10 The National Nosocomial Infections Surveillance System has reported that the incidence of VRE increased from 0.3% of all enterococci in 1989 to 7.9% in 1993, with larger increases in intensive care units.11 In some medical centers, VRE have become endemic.12 Vancomycin-resistant enterococci were first identified at our medical center in 1991, and remain a significant problem despite attempts to curtail the prevalence by formulary management strategies13 and the institution of increasingly more stringent infection control practices.14 Since treatment options are limited, much attention has been focused on trying to understand the epidemiological features of VRE and the impact that vancomycin resistance has had on the ability to care for patients infected with this organism. It is only through this understanding that more effective means to control further dissemination of VRE will be identified. For these reasons, we performed a retrospective comparison of vancomycin-susceptible E faecium (VSE) and VRE bacteremia in our hospital to study the clinical and epidemiological aspects of this infection, as well as to define the clinical and economic significance of vancomycin resistance in enterococci.
PATIENTS AND METHODS

PATIENTS

Northwestern Memorial Hospital in Chicago, Ill, is a 588-bed, university-affiliated, tertiary care hospital with active surgical, trauma, solid organ transplantation, and bone marrow transplantation units. Medical records were reviewed for all patients who had a blood culture positive for *E faecium* during the 4-year period from January 1992 through December 1995, as identified by the Clinical Microbiology Laboratory of Northwestern Memorial Hospital. Seventy-two cases of *E faecium* bacteremia were identified; of these, complete medical records were available for 59 patients. Four of these bacteremias were only identified from autopsy specimens and were not included in the final analysis. Two cases were excluded because the patients were neonates and final outcomes were unknown due to patient transfer to other hospitals. Overall, 53 patients with bacteremia were included in the final analysis: 32 patients with VSE and 21 patients with VRE bacteremia. Clinically significant bacteremia was defined as the presence of 2 or more blood cultures positive for *E faecium*, or a single positive blood culture coupled with a clinically evident, or culture-positive, other site of infection.3

CLINICAL DATA COLLECTION

Clinical information obtained for each patient included the following: age, sex, length of hospital stay prior to the onset of bacteremia, and hospital unit where the patient was admitted. A determination was also made regarding community vs nosocomial acquisition of the bacteremia. Nosocomial acquisition was defined as those patients whose first positive blood culture occurred more than 72 hours after admission to the hospital, or when the patient was transferred from another hospital or chronic care facility. The source of bacteremia was identified from a culture-positive site or a clinically apparent site of infection. If another site of infection was not evident, the source was considered unknown and the bacteremia primary in origin. If *E faecium* was isolated from multiple body sites, this was documented. Information regarding previous antimicrobial agent therapy was also obtained for each patient, as well as data pertaining to the management of the infection. All medical records were reviewed for potential underlying risk factors or predisposing conditions for the acquisition of *E faecium* bacteremia. This information included liver disease, renal dysfunction, neutropenia, significant corticosteroid therapy, infection with the human immunodeficiency virus, malignant neoplasms, bone marrow transplantation, solid organ transplantation, diabetes mellitus, pulmonary disease, debilitating neurologic disease, recent trauma, recent surgical procedures, and concomitant *Clostridium difficile*–associated diarrhea or colitis.19,11-13,16,18,21 Liver disease was defined as the presence of a total bilirubin level greater than 43 µmol/L (2.5 mg/dL) and aminotransferase levels greater than twice normal. Renal dysfunction was defined as moderate if the calculated creatinine clearance was less than 1.0 mL/s (60 mL/min) or severe if it was less than 0.25 mL/s (15 mL/min). Absolute neutropenia was defined as less than 500×10⁹/L of polymorphonuclear leukocytes. A daily prednisone dosage of 20 mg (or equivalent) for at least 2 weeks was considered significant corticosteroid therapy. The presence of indwelling bladder catheters or central venous catheters and hyperalimentation therapy was also recorded.

A severity-of-illness score24 was calculated for each patient at the onset of bacteremia. This grading system assigns points on the following basis: change in mental status with stupor, 1 point; change in mental status with coma, 4 points; body temperature of 37.8°C or higher, 1 point; body temperature of 40°C or higher, 2 points; body temperature of 35.6°C or lower, 2 points; hypotension (systolic blood pressure 40 mm Hg, decrease in systolic blood pressure by 20 mm Hg, or use of intravenous pressor agents), 2 points; use of mechanical ventilation, 2 points; and cardiac arrest, 4 points. This index has previously been shown to be predictive of outcome for patients with bacteremia.

MICROBIOLOGICAL DETERMINATIONS

From January 1992 through March 1993, blood was cultured aerobically with the use of a processing system (Isolator system, Wampole Laboratories, Cranberry, NJ) and anaerobically in broth (Thiol, Difco Laboratories, Detroit, Mich). Throughout the remainder of the study period, the Isolator processing system and an automated system (ESP, Difco Laboratories) were used to culture blood aerobically and anaerobically. Identification of *E faecium* was based on standard methods, including the ability of the organism to hydrolyze esculin in the presence of bile and grow in 6.5% sodium chloride; demonstration of a lack of pigment and motility, and fermentation of arabinose.23 Susceptibility testing was performed by agar dilution on Mueller-Hinton agar according to reference methods.24

TREATMENT DATA

Therapy recorded for *E faecium* bacteremia included information on the dosage and duration of all antimicrobial agents used. Documentation was made if single, dual, or no drug therapy was attempted. Data were also collected if other interventions (such as central venous catheter removal or surgical drainage of infected tissues) were made in attempt to treat the infection and/or bacteremia.

OUTCOME DATA

Outcome determinations were based on mortality: death was considered directly caused by the *E faecium* bacteremia if the patient died following a positive blood culture with a clinical picture consistent with sepsis; death was indirectly caused by the bacteremia if the patient died of multifactorial causes, including further organ compromise by the current septic episode; and death was considered unrelated to the bacteremia if the patient died following the bacteremia of causes independent of the infectious process. Survival was defined as the patient recovering from the bacteremia and eventually being discharged from the hospital.

COST DATA

Through the hospital management engineering database, information was collected to determine the total cost of

Continued on next page
inpatient care for 33 patients with *E. faecium* bacteremia. One liver transplant recipient with VRE was excluded from analysis because of excessive hospitalization costs related to complications from transplantation, and 1 patient with VSE was excluded because of excessive costs related to surgery. The final calculations were based on 31 patients with VSE bacteremia and 20 patients with VRE bacteremia. From these data, the average cost of hospitalization was determined for each group of patients.

**STATISTICAL ANALYSIS**

All clinical, microbiological, and outcome data were assessed for statistical significance by the use of the Fisher exact test (2-tailed) and computation of *P* values. The Student *t* test was used to compare continuous variables. *P* <.05 was considered statistically significant.

**RESULTS**

**DEVELOPMENT OF BACTEREMIA**

During the 4-year study period, 32 episodes of VSE bacteremia and 21 episodes of VRE bacteremia were reviewed. Of these, 26 VSE bacteremias (81%) compared with 18 VRE bacteremias (86%) were determined to be clinically significant. The annual number of VRE bacteremias slowly increased throughout the study period; from January 1992 through December 1993, 8 VRE bacteremias were identified. However, 13 VRE bacteremias occurred during the last 2 years of the study period. In contrast, the number of VSE bacteremias declined from 13 cases in 1992 to 6 cases in 1993, 6 cases in 1994, and 7 cases in 1995.

The mean age of patients with VSE was 56.7 years, and that of patients with VRE was 58.3 years. Nosocomial acquisition of bacteremia occurred in 25 patients with VSE (78%) compared with all of the patients with VRE (*P* =.02). Patients with VRE had longer hospital stays prior to the development of bacteremia than did patients with VSE, with a mean of 34.8 days vs 16.7 days, respectively (*P* =.004). The intensive care units were the most frequent hospital location for development of enterococcal bacteremia: 38% (12/32) of patients with VSE compared with 57% (12/21) of those with VRE (*P* =.09). Table 1 summarizes the clinical features of the patients with *E. faecium* bacteremia.

In Table 2, the various sources of infections identified in these patients and their microbiological features are listed. There were no statistically significant differences in the sources of the bacteremia. An intra-abdominal site was the most common source of bacteremia for both the VSE and VRE groups: 15 (47%) and 8 (38%), respectively. A trend toward multiple sources for the bacteremia was seen in the patients with VRE, but this did not reach statistical significance (*P* =.07).

Microbiological data revealed that polymicrobial bacteremia was relatively common, and occurred with equal frequency in both the VSE and VRE bacteremic episodes: 14 patients with VSE (44%) vs 9 patients with VRE (43%) had more than 1 organism recovered from blood cultures. The mean number of blood cultures positive for *E. faecium* was higher in the VRE group (Table 2). The mean (±SD) severity-of-illness scores at the onset of the bacteremic episodes were equivalent for both groups (4.2±3.8 for the VSE group vs 4.5±3.4 for the VRE group). In addition, the VSE and VRE groups had comparable numbers of patients with a severity-of-illness score greater than or equal to 5: 13 (41%) and 8 (38%), respectively.

Data pertaining to antimicrobial agent therapy prior to the development of bacteremia were retrievable for 29 VSE and 20 VRE cases (Table 3). Patients in both groups frequently received antimicrobial chemo-

Table 1. Clinical Features of *Enterococcus faecium* Bacteremia*

<table>
<thead>
<tr>
<th>Feature</th>
<th>VSE (n=32)</th>
<th>VRE (n=21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>26 (81)</td>
<td>9 (43)</td>
<td>.004</td>
</tr>
<tr>
<td>F</td>
<td>6 (19)</td>
<td>12 (57)</td>
<td></td>
</tr>
<tr>
<td>Mean (+SD) SIS</td>
<td>4.2±3.8</td>
<td>4.5±3.4</td>
<td>.76</td>
</tr>
<tr>
<td>SIS ≥5</td>
<td>13 (41)</td>
<td>8 (38)</td>
<td>.22</td>
</tr>
<tr>
<td>Clinically significant</td>
<td>26 (81)</td>
<td>18 (86)</td>
<td>.27</td>
</tr>
<tr>
<td>Unit of acquisition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICU</td>
<td>12 (38)</td>
<td>12 (57)</td>
<td>.09</td>
</tr>
<tr>
<td>Surgical</td>
<td>3 (9)</td>
<td>2 (10)</td>
<td>.36</td>
</tr>
<tr>
<td>Medical</td>
<td>9 (28)</td>
<td>1 (5)</td>
<td>.03</td>
</tr>
<tr>
<td>Oncology</td>
<td>8 (25)</td>
<td>6 (29)</td>
<td>.24</td>
</tr>
<tr>
<td>Nosocomial</td>
<td>25 (78)</td>
<td>21 (100)</td>
<td>.02</td>
</tr>
<tr>
<td>Mean (+SD) length of stay, d</td>
<td>16.7±16.1</td>
<td>34.8±23.3</td>
<td>.004</td>
</tr>
</tbody>
</table>

*VSE indicates vancomycin-susceptible enterococci; VRE, vancomycin-resistant enterococci.†Unless otherwise indicated.
‡Unit where patient was hospitalized at the onset of bacteremia.
§Median length of stay prior to onset of bacteremia.

Table 2. Features of *Enterococcus faecium* Bacteremia*

<table>
<thead>
<tr>
<th>Feature</th>
<th>VSE (n=32)</th>
<th>VRE (n=21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (±SD) positive blood cultures</td>
<td>1.6±0.7</td>
<td>2.3±1.1</td>
<td>.02</td>
</tr>
<tr>
<td>Polymicrobial bacteremia</td>
<td>14 (44)</td>
<td>9 (43)</td>
<td>.22</td>
</tr>
<tr>
<td>Source of bacteremia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intra-abdominal</td>
<td>15 (47)</td>
<td>8 (38)</td>
<td>.18</td>
</tr>
<tr>
<td>Urinary tract</td>
<td>1 (3)</td>
<td>1 (5)</td>
<td>.49</td>
</tr>
<tr>
<td>Wound</td>
<td>1 (3)</td>
<td>0 (0)</td>
<td>.60</td>
</tr>
<tr>
<td>Soft tissue</td>
<td>1 (3)</td>
<td>0 (0)</td>
<td>.60</td>
</tr>
<tr>
<td>Vascular catheter</td>
<td>4 (13)</td>
<td>3 (14)</td>
<td>.31</td>
</tr>
<tr>
<td>Multiple sources</td>
<td>1 (3)</td>
<td>4 (19)</td>
<td>.07</td>
</tr>
<tr>
<td>Unknown source</td>
<td>9 (28)</td>
<td>5 (24)</td>
<td>.24</td>
</tr>
</tbody>
</table>

*VSE indicates vancomycin-susceptible enterococci; VRE, vancomycin-resistant enterococci.
†Unless otherwise indicated.
therapy prior to the development of enterococcal bacteremia. Previous administration of vancomycin was more commonly associated with VRE bacteremia compared with VSE bacteremia (16 [80%] vs 10 [34%] patients, respectively; *P=.002). Aminoglycoside use was also associated with the development of VRE bacteremia (*P=.005). There were no significant differences between the enterococcal bacteremia groups observed with the use of third-generation cephalosporins, aztreonam, or fluoroquinolones.

**Table 4** summarizes the serious underlying conditions and risk factors for the acquisition of *E faecium* bacteremia. Although both groups had many underlying illnesses, there were few statistically significant differences between those with VRE and VSE bacteremia. Malignant neoplasms and surgical procedures were common to both groups. A known malignant neoplasm was present in more than half of the patients with *E faecium* bacteremia. Recent surgery was documented in 10 (31%) vs 8 (38%) of the patients in the VSE and VRE groups, respectively. Interestingly, infection with the human immunodeficiency virus was seen significantly more often in the VRE group (7 [22%] compared with 0 [0%] in the VSE group (*P=.02). Although neutropenia (6 [19%] in VSE vs 8 [38%] in VRE; *P=.08) and solid organ transplantation (0 [0%] in VSE vs 3 [14%] in VRE; *P=.06) were identified more frequently in patients with VRE, these did not reach statistical significance. There was no difference in the use of central venous catheters; however, indwelling urinary catheter use was significantly higher in patients with VRE compared with those with VSE (13 [62%] vs 9 [28%], respectively; *P=.01).

**TREATMENT OF BACTEREMIA**

With respect to treatment, patients with VRE bacteremia were just as likely to receive some form of specific antimicrobial agent therapy as their VSE counterparts: 15 (71%) vs 24 (75%) patients, respectively (Table 5). Combination therapy, usually with a β-lactam antibiotic or vancomycin plus an aminoglycoside, was given equally to the patients of both groups. Not surprisingly, use of vancomycin therapy was significantly higher in the VRE group, with 18 (56%) patients with VRE receiving this agent compared with 4 (19%) patients with VRE (*P=.006). Ampicillin, given at high doses as a continuous infusion, was used more frequently in the treatment of bloodstream infections caused by VRE. Unconventional and investigational therapies, such as a combination product of quinupristin and dalfopristin,25 an experimental streptogramin, were used to treat patients in the VRE group. Central venous catheter removal as an additional therapy was significantly more common in patients with VRE bacteremia (10 [48%] vs 6 [19%] for VSE; *P=.02). There was no difference in surgical debridement of infected tissue between the 2 groups.

**OUTCOME AND HOSPITAL COST ANALYSIS**

Overall mortality for the VSE group was 41% (n=13), but only one fourth of these deaths (9% mortality rate [n=3]) could be directly attributed to the bacteremia. Mortality for the VRE group was significantly higher, with death occurring in 16 patients (76%) (*P=.009). One half (38% mortality rate) of these deaths were directly caused by VRE bacteremia, and in an additional 5 cases (24%), VRE contributed indirectly to the mortality of the patient. Interestingly, while there was no significant difference in mortality between patients in whom enterococci were isolated in multiple as opposed to a single blood culture, there was a trend toward more directly attrib-
able mortality in the group with VRE (7 cases vs 0 VSE cases; P=.09). However, no death was found to be directly attributable to VRE bacteremia when the organism was isolated from only a single blood culture. Table 6 summarizes outcome and costs associated with enterococcal bacteremia. Average cost of hospitalization for a patient with VRE bacteremia was $83 897 compared with $56 707 for a patient with VSE bacteremia (P=.04). A 32.4% cost difference was identified between these 2 cohorts.

### Table 6. Outcomes of Enterococcus faecium Bacteremia *

<table>
<thead>
<tr>
<th>Outcome Measure</th>
<th>VSE  (n=32)</th>
<th>VRE  (n=21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>13 (41)</td>
<td>16 (76)</td>
<td>.009</td>
</tr>
<tr>
<td>Directly related</td>
<td>3 (9)</td>
<td>8 (38)</td>
<td>.01</td>
</tr>
<tr>
<td>Indirectly related</td>
<td>6 (19)</td>
<td>5 (24)</td>
<td>.24</td>
</tr>
<tr>
<td>Unrelated</td>
<td>4 (13)</td>
<td>3 (14)</td>
<td>.31</td>
</tr>
<tr>
<td>Survival</td>
<td>19 (59)</td>
<td>5 (24)</td>
<td>.009</td>
</tr>
<tr>
<td>Total cost of hospitalization, $‡</td>
<td>83 897</td>
<td>56 707</td>
<td>.04</td>
</tr>
</tbody>
</table>

* VSE indicates vancomycin-susceptible enterococci; VRE, vancomycin-resistant enterococci. ‡Average total cost of hospitalization for 31 patients with VSE bacteremia and 20 patients with VRE bacteremia. See “Results” section of the text for details.

**COMMENT**

Our study confirms and extends what has been observed in patients with VRE bacteremia: that this is currently a nosocomial infection acquired by patients who have severe underlying medical conditions, who have prolonged hospital stays, and who have been previously treated with broad-spectrum antimicrobial agents, especially vancomycin.3,13,25,26 Additionally, vancomycin resistance directly adds to the cost of care of these patients, accompanied by an increase in infection-related mortality. Most patients in our series were hospitalized for more than 2 weeks prior to the onset of the bacteremia. Interestingly, men acquired VRE more frequently than women; however, our data contained no explanation for this finding. Patients often acquired the organism in an intensive care unit, and no one was admitted with community-acquired VRE.

As for the presence of underlying illness, both groups of patients had numerous but similar comorbidities and equivalent severity-of-illness scores. Malignant neoplasms, neutropenia, corticosteroid therapy, recent surgery, liver dysfunction, and renal impairment were commonly present. Many of these same underlying conditions have been associated with bacteremia and other infections caused by VRE.3,26 Although not statistically significant, neutropenia and solid organ transplantation were present more frequently in patients who acquired VRE. We did not find VRE bacteremia in our patients infected with the human immunodeficiency virus, likely reflecting shorter lengths of hospitalization (average, 8 days; range, 4-14 days) for these patients.

The use of indwelling bladder catheters was also associated with VRE bacteremia, although the urinary tract was not found to be a common source for VRE bloodstream infections. This may be explained in part by the serious underlying diseases in the patients with VRE. However, Morris and colleagues18 found that urinary tract catheterization was a risk factor for the development of urinary tract infections caused by this organism.

Broad-spectrum antimicrobial agent therapy appears to be an important predisposing factor to E faecium bacteremia. We found a strong correlation between parenteral vancomycin use and the subsequent development of VRE. This supports previous findings by Morris and coworkers,18 who found that patients with urinary tract infection caused by VRE were more likely to have prior exposure to parenteral vancomycin than those with VSE urinary tract infections. Our findings differ from those of Linden et al,23 who report similar patient exposure to vancomycin prior to the development of VRE and VRE bloodstream infection. Aminoglycoside use was also found to correlate with VRE bacteremia. However, our data may have been influenced by antibiotic formulary changes that occurred during the study period.19 Early in 1993, a policy restricting the use of third-generation cephalosporins at our medical center resulted in substantial decreases in cephalosporin use and increases in aminoglycoside use. Our finding may be explained by this and the fact that VRE bacteremia has been increasingly common at our center since the beginning of 1994. For similar reasons, we did not find a strong correlation between third-generation cephalosporin use and the acquisition of VRE. However, previous investigations at our medical center did find use of third-generation cephalosporins as a risk factor for enterococcal bacteremia.3,6

While we were not able to identify many differences in the patient populations who developed E faecium bacteremia, we were able to highlight dramatic differences in the outcomes of these groups. Despite similar comorbidities and comparable severity-of-illness scores, mortality was much higher in patients with VRE bloodstream infection. Although our patients
with VRE were just as likely to receive aggressive antibiotic therapy combined with removal of foreign bodies and surgical drainage, the high mortality in this group likely reflects the lack of effective therapy for VRE bloodstream infection. Similarly, data collected by Linden and colleagues,23 the National Nosocomial Infections Surveillance System,17 and Montecalvo and colleagues26 also show higher mortality rates with Enterococcus faecium bacteremia when vancomycin resistance is present. In contrast, Wells et al27 found comparable and overall lower mortality rates in patients with both VRE and VSE bacteremia (17% vs 27%); however, these investigators did not attempt to separate patients based on single and multiple blood cultures positive for VRE. Aside from this one report, accumulating evidence confirms that vancomycin resistance does indeed have a significant adverse impact on the outcome of patients who develop Enterococcus faecium bacteremia.

In our study, trends toward higher attributable mortality with multiple blood cultures positive for VRE were seen. Failure to achieve statistical significance is likely a reflection of the small sample size. In many instances, a single blood culture positive for VRE in the absence of an obvious source of infection likely represents skin colonization and blood culture contamination, similar to that seen with coagulase-negative staphylococci in other patients. A reasonable alternative to immediate institution of unconventional antimicrobial therapy is to observe these patients, repeat cultures, and perform other conservative measures, such as the removal or replacement of intravenous and urinary catheters. More aggressive therapeutic measures can then be undertaken if additional blood cultures are positive.

In addition to the high mortality, we demonstrated substantial additional hospitalization costs associated with VRE bacteremia. In today's health care environment, understanding the financial impact of this multidrug-resistant organism is crucial since controlling infection with VRE becomes both medically necessary and economically important.

In conclusion, VRE bacteremia is a serious and costly complication of prolonged hospitalization in severely debilitated patients. Previous treatment with parenteral vancomycin appears to be a major factor in the acquisition of VRE bacteremia. As a medical community we must be more judicious in the use of this antibiotic as well as other extended-spectrum antimicrobial agents. This study reinforces the serious nature of the problem of reemerging pathogens with multiple drug resistance and suggests that future resources and efforts must be directed at finding more efficacious therapy plus preventing further nosocomial spread of this pathogen.

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REFERENCES


