MAJOR ARTICLE

Not All Patients with Vancomycin-Resistant Enterococci Need To Be Isolated

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Background. Vancomycin-resistant enterococci (VRE) have triggered multiple outbreaks. However, VRE of genotype *vanC* appear not to be associated with outbreaks. The goal of this study was to estimate the risk of bloodstream infections in patients colonized with VRE of genotype *vanC* who received care from a bone marrow transplant unit for patients with leukemia, where only standard precautions were implemented for VRE of genotype *vanC* during the last 9 years.

Methods. Since 2000, all patients in the bone marrow transplant unit underwent routine VRE rectal screening, data were prospectively entered in a database, and isolates were molecularly characterized. Infection control policy required contact isolation for patients infected with VRE of genotype *vanA* or *vanB* but only standard precautions for patients infected with VRE of genotype *vanC*.

Results. From January 2000 to July 2008, 290 isolates of VRE of genotype *vanC* obtained from 273 different patients were identified, with an incidence of 25–43 isolates/year. Of 290 isolates, 285 (98%) were identified in rectal screening swabs, 5 were from other body sites, and none required specific treatment. During the entire study period, only 1 case of bloodstream infection was detected, reflecting an incidence of 1 (0.4%) of the 273 patients, or <0.2 cases per 1000 patient-days. No outbreaks were recorded.

Conclusions. These data provide strong evidence that carriers of VRE of genotype *vanC* do not require contact isolation, thereby saving resources and potentially improving patient care. The genotype should be routinely determined in areas with a high prevalence of VRE of genotype *vanC*.

Vancomycin-resistant enterococci (VRE) were first described in Europe in 1988 [1, 2] and have rapidly spread worldwide ever since, including to the United States [3]. The percentage of nosocomial infections caused by VRE increased >20-fold (from 0.3% to 7.9%) between 1989 and 1993 in the United States [4]. This increase was mainly caused by an increase in VRE infections among intensive care unit (ICU) patients, although the same trend could also be observed in non-ICU patients [5]. The emergence of VRE has led to multiple epidemics and outbreaks in several hospital settings [6–10]. Infections with these pathogens have been associated with poor outcomes and vancomycin resistance has been shown to be an independent predictor of death in enterococcal bacteremia [11].

There are 6 recognized phenotypes of vancomycin resistance: VanA, VanB, VanC, VanD, VanE, and VanG [12]. Five of these types correspond to acquired resistance (VanA, VanB, VanD, VanE, and VanG); one type—VanC—is an intrinsic property of *Enterococcus gallinarum* and *Enterococcus casseliflavus.* The VanC phenotype is characterized by intrinsic low-level resistance to vancomycin and susceptibility to teicoplanin [13, 14].

Human enterococcal infections are mainly caused by 2 species, *Enterococcus faecalis* and *Enterococcus faecium,* which express the VanA or VanB phenotype [15], and most published outbreaks of VRE have been restricted to these 2 classes of vancomycin resistance. The resistance genes are harbored on plasmids for the VanA and VanB phenotypes, whereas the VanC phenotype is an intrinsic feature, which is not found on plasmids or transposons but is encoded chromosomally. Other spe-

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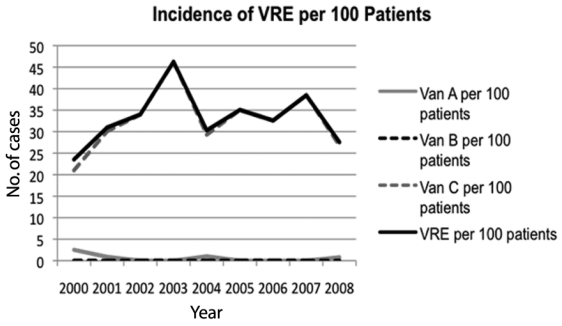

Figure 1. Incidence of vancomycin-resistant enterococci (VRE) of genotypes *vanA, vanB,* and *vanC* identified per 100 patients.

cies, like *E. gallinarum* and *E. casseliflavus,* which exhibit the VanC phenotype, are less common, and outbreaks have been reported rarely [16, 17]. In contrast to VRE of genotypes *vanA* and *vanB,* VRE of genotype *vanC* (hereafter, referred to as "VRE *vanC*") are associated with a low risk of mortality [18].

The Centers for Disease Control and Prevention recommend contact isolation precautions for patients colonized or infected with VRE, without mentioning specific precautions for enterococci expressing the *vanC* genotype [19]. However, many institutions in the Netherlands and Switzerland do not follow this recommendation, because this intrinsic chromosomal resistance is not found on plasmids and transposons that might spread to other bacteria. VRE *vanC* are common in Europe but are rare in the United States [11]. There are insufficient data to support the practice of nonisolation of patients colonized or infected with VRE *vanC*.

This study has 2 aims: first, to estimate the risk of invasive infections for patients colonized with VRE *vanC* and, second, to provide clinical data on the level of transmissibility.

METHODS

Setting. The University Hospital of Basel is an 855-bed tertiary care center in Basel, Switzerland. The bone marrow transplant unit performs 80–100 stem cell transplantations per year, with predominantly allogeneic transplantations (42–77 per year).

Patients and data collection. Rectal swabs to detect VRE were routinely performed during each hospitalization. The database of all consecutive VRE *vanC* isolates from patients on the bone marrow transplant unit in the University Hospital of Basel was studied from January 2000 to July 2008. Information was obtained from the computerized database of the clinical microbiology unit, and patients whose cultures from any body site yielded VRE *vanC* were identified.

An infectious diseases specialist then reviewed the medical records of these patients and collected data regarding patient demographic characteristics, underlying diseases or condition, clinical manifestations at the time of detection of VRE *vanC* from a normally sterile body fluid, prior antibiotic use, antibiotic therapy received, bacteremia with any kind of microorganism, and clinical outcome. We included 1 positive rectal swab specimen per patient per year. The study was approved by the local ethics committee as part of the quality assurance program.

Definitions. Invasive infections were defined by detection of VRE *vanC* from a normally sterile body fluid in addition to clinical assessment. Clinically significant bacteremia due to VRE *vanC* was defined by isolation of VRE *vanC* in 1 blood culture.

Species identification and genotyping. Rectal swab specimens were screened for VRE by use of a selective enrichment broth (BBL Enterococcosel Broth; Becton Dickinson) supplemented with vancomycin (6 mg/L). Subcultures were performed on BBL Vancomycin Screen Agar containing 6 mg/L vancomycin (Becton Dickinson) and blood agar. Identification

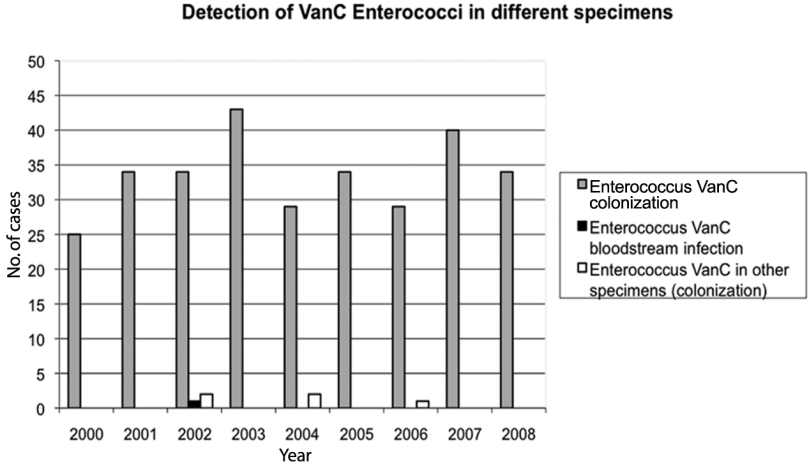

Figure 2. Detection of vancomycin-resistant enterococci of genotype *vanC* in different specimens.

Table 1. Characteristics of Patients with Vancomycin-Resistant Enterococci of Genotype *vanC* (VRE *vanC*) Identified in Rectal Screening Swab Specimens

Characteristic	Patients with VRE *vanC* in rectal swab specimen (n = 260)
Age, mean years	45.8
Sex	
Female	117 (45)
Male	143 (55)
Hospital stay, mean days	39.1
Autologous stem cell transplantation	57 (21.9)
Allogeneic stem cell transplantation	123 (47.3)
Chemotherapy	88 (33.8)
No antibiotic therapy	31 (11.9)
Antibiotic therapy	229 (88.1)
Amikacin	100 (38.5)
Vancomycin	51 (19.6)
Carbapenem	96 (36.9)
Cefepim	140 (53.8)
Piperacillin-tazobactam	143 (55.0)
Other	106 (40.8)
Blood stream infection	
1 Episode	61 (23.5)
2 Episodes	12 (4.6)
3 Episodes	4 (1.5)
None	183 (70.4)
Outcome	
Discharge	223 (85.8)
Transfer to another hospital	9 (3.4)
Death	28 (10.8)

of glycopeptide resistance genotypes (VanA, VanB, VanC1, and VanC2/3) and enterococcal species of VRE was molecularly confirmed by use of polymerase chain reaction (PCR) and the DNA strip GenoType *Enterococcus* assay (Hain Lifescience).

Molecular typing of 9 consecutive exemplary strains of VRE *vanC* selected at random was performed by pulsed-field gel electrophoresis (PFGE) [20]. In brief, the DNA restriction fragments were separated after *Sma*I digestion by PFGE, and dendrograms were drawn using the computer software GelCompar, version 4.5 (Applied Maths).

RESULTS

From January 2000 through July 2008, 296 isolates of VRE from all body sites were obtained from 273 different patients who received care on the bone marrow transplant unit of the University Hospital of Basel. Resistance genotype identification through PCR revealed the VanC genotype in 290 (98%) of the 296 isolates. This accounts for an incidence of 25–43 VRE *vanC* isolates per year, corresponding to 29–43 patients per year. The incidence of VRE of genotypes *vanA* and *vanB* was very low, calculated as cases per year (Figure 1).

Of the 290 VRE *vanC* isolates, 285 (98%) originated from rectal screening swab specimens, and only 5 isolates were found in other specimens, possibly suggesting invasive infection (Figure 2). The majority of the 285 rectal swabs with detection of VRE *vanC* (173 [60.7%]) were performed in the first 3 days after admission. A total of 193 (67.7%) of the 285 patients with rectal swab specimens positive for VRE *vanC* tested positive at admission. Of the remaining 92 patients, 73 had positive specimens after repeated sampling. Among the 92 patients with positive rectal swabs after the first week of admission, 35 (12.3% of 285 patients) tested positive in the second week, 18 (6.3%) in the third week, and 39 (13.7%) after 3 weeks of hospitalization.

Medical chart review could be completed for 260 of the 273 different patients. A total of 13 patients were lost to further follow-up.

The baseline characteristics of the 260 patients with detection of VRE *vanC* are summarized in Table 1. The mean age of the patients with detection of VRE *vanC* was 45.8 years, and there was a slight predominance of the male sex (55%). A total of 69.2% underwent stem cell transplantation, mainly allogeneic. The vast majority of patients (88.1%) received broad-spectrum antibiotic treatment during their hospital stay in accordance with the local guidelines of the institutions with either cefepime, piperacillin-tazobactam, or meropenem. Vancomycin was only given to 19.6%, mainly to patients with suspected catheter-related infection. Blood-stream infection with any microorganism was documented in 29.6% of all patients. The most commonly isolated pathogens were coagulase-negative staphylococci (38.4% of all 99 bloodstream isolates) and *Escherichia coli* (18.2% of all 99 bloodstream isolates). Acute myeloid leukemia and acute lymphoblastic leukemia were the underlying hematological illnesses encountered most frequently, occurring in 48% and 13% of patients, respectively.

The 5 specimens detected from body sites other than the rectum originated from 4 different patients (Table 2). After full medical chart review by 2 board-certified infectious diseases specialists, the detection of VRE *vanC* in 3 patients was interpreted as colonization. The fourth patient, who had detectable VRE *vanC* in blood cultures and in culture of a superficial wound swab specimen, showed a favorable outcome. Overall, only 1 case of bloodstream infection was detected, reflecting an incidence of 1 (0.4%) of 273 patients, or <0.2 cases per 1000 patient-days.

Comparison of the results obtained by PFGE of 9 representative strains of VRE *vanC* detected during the entire study period revealed no evidence of identity. No evidence of an outbreak was recorded during the entire study period.

Table 2. Patients with Detection of Vancomycin-Resistant Enterococci of Genotype *vanC* (VRE *vanC*) in Specimens from Various Body Sites

Patient	Age, years	Sex	VRE *vanC* in rectal swab specimen	VRE *vanC* in clinical specimen	Clinical diagnosis	Outcome
1	51	M	Yes	Deep wound swab	Colonization	Cured
2	53	M	Yes	Blood and superficial wound swab	Bloodstream infection	Cured
3	63	F	No	Respiratory tract specimen	Colonization	Deceased
4	69	F	Yes	Superficial wound swab	Colonization	Cured

DISCUSSION

Throughout the study period, 296 isolates of VRE were detected from all body sites tested. The *vanC* genotype was detected in 290 isolates and thus represented the vast majority of all VRE isolates. This finding represents an incidence of 25–43 VRE *vanC* isolates per year. The predominance of the *vanC* genotype has also been reported in other European countries [21]. In contrast, the *vanA* and *vanB* genotypes account for ~67% and 25% of all VRE isolates in the United States, respectively [22]. This difference may be because avoparcin—a glycopeptide—was used in large amounts in the European food industry until it was banned in April 1997 by the European Commission. This was not the case in the United States. In countries using avoparcin, vancomycin-resistant enterococci were commonly found in the commensal flora of food animals, in meat from these animals, and in the commensal flora of healthy humans, despite very limited use of vancomycin in hospitals [23].

Of the 290 VRE *vanC* isolates, 285 (98%) originated from rectal swab specimens, accounting for an incidence that ranged from 21 to 46 VRE *vanC* isolates per 100 patients throughout the entire study period. Gordts et al [24] described a prevalence of 3.5% for VRE colonization of the intestinal tract in hospitalized patients, with the vast majority of VRE of genotype *vanA*. The study included all patients admitted to the hospital and did not focus on a bone marrow transplant unit. The duration of stay in a hematology department was, however, identified as a risk factor for VRE colonization.

During the entire study period from January 2000 to July 2008, only 1 case of bloodstream infection due to VRE *vanC* was detected among colonized patients. This reflects an incidence of 1 (0.4%) of 273 patients, or <0.2 cases per 1000 patient-days. Our data therefore demonstrate an extremely low risk for invasive infection caused by VRE *vanC* in colonized patients on a hematology transplant unit. To our knowledge, this is the first study to assess the risk of invasive infection in patients on a hematology transplant unit who are colonized with VRE *vanC*.

The patient with bloodstream infection due to VRE *vanC* had a favorable clinical outcome. However, VRE *vanC* have been reported to cause serious infections, including endocarditis [25, 26], meningitis [27], liver abscess [28], and primary bacteremia [25].

Reid et al [25] reported 20 cases of VRE *vanC* bloodstream infection with serious outcomes: 4 patients died within 6 days after admission to the hospital, and another 4 patients died 1–2 months after the episode of bacteremia. The authors therefore concluded that *E. gallinarum* and *E. casseliflavus* may cause serious invasive disease. However, all the patients described had serious underlying diseases, so it was difficult to attribute their mortality directly to infection with VRE *vanC*. Furthermore, a large proportion (45%) of the described patients had polymicrobial bacteremia in which aerobic gram-negative bacilli were the predominant blood coisolates recovered, which further complicates the analysis of the relationship between mortality and the detection of VRE *vanC* in blood cultures.

A low risk of mortality associated with bacteremia due to *E. casseliflavus* and *E. gallinarum* was postulated by Choi et al [18] in their analysis of 56 cases of bacteremia due to VRE *vanC*. However, the majority of the described patients (75%) had biliary disease as an underlying condition, and only 1 patient had an underlying hematological illness. This study supports our finding of a favorable clinical outcome of VRE *vanC* bacteremia in a limited manner, because of the different patient characteristics.

No differences in severity of illness or mortality between different enterococcal species were found by De Perio et al [29], who compared the outcomes of 33 patients with non–*E. faecalis* and non–*E. faecium* enterococcal bacteremia with those of patients with *E. faecalis* bacteremia. In Japan, of 9 patients with VRE *vanC* bacteremia, 4 died; however, the authors of the study did not specify whether death was directly attributed to infection with VRE *vanC* [30].

All VRE *vanC* strains detected during the entire study period revealed no evidence of identity by PFGE analysis. The majority of rectal swabs (60.7%) that had detection of VRE *vanC* were performed within the first 3 days after admission, whereas 92 rectal swab specimens tested positive after the first week of hospitalization. For 73 of the 92 patients, the initial rectal swab

specimen obtained at admission tested negative for VRE, suggesting nosocomial transmission. The vast majority of patients (88.1%), however, received broad-spectrum β-lactam treatment during their hospital stay, selecting for enterococci in the gastrointestinal flora and, thus, facilitating detection of VRE. [31]. In addition, D'Agata et al [32] found antibiotic exposure to be significantly associated with higher VRE density in stool; the sensitivity of rectal swab specimen cultures was only 58%, ranging from 100% at VRE densities of $\geq$7.5 $\log_{10}$ colony forming units (cfu) per gram of stool to 0% at densities of $\leq$4.5 $\log_{10}$ cfu per gram of stool. Therefore, it is conceivable that the delayed detection of VRE relates to the sensitivity of the culture method, rather than being evidence of nosocomial transmission. Stable incidence rates could be documented, and no additional outbreaks were recorded by our continuous surveillance system during the observed study period. We therefore concluded that no transmission of any clinical relevance had taken place, although contact isolation precautions for patients colonized or infected with VRE, which are recommended by the Centers for Disease Control and Prevention, were not implemented. However, our data were collected in a highly specialized bone marrow transplant unit with a very high standard of routine infection control strategies. This may limit the application of our results to normal hospital wards.

Only 2 outbreaks due to VRE *vanC* have been reported so far. The first outbreak was reported to involve 9 otherwise healthy patients who underwent total knee arthroplasty in a single orthopedic unit. They consecutively developed prosthesis-associated infection caused by a single strain of *E. gallinarum* nearly 4 months later. To find a source of exposure, environmental cultures and select personnel cultures were performed; results of these cultures, however, were negative. In summary, no source was implicated, and no major breaches in infection control could be identified [16].

The second outbreak was identified because of an unusual increase in the number of infections caused by vancomycin-resistant *E. gallinarum* in a Colombian tertiary care teaching hospital. Eleven cases were identified, and *E. gallinarum* could be isolated in cultures of blood from 4 patients, surgical secretions from 4 patients, paranasal sinus secretion from 1 patient, a lung abscess from 1 patient, and urine from 1 patient. The mortality of the patients infected with *E. gallinarum* was higher than that of control subjects (18% vs 9.7%), although this difference was not statistically significant. The authors therefore concluded that VRE *vanC* are capable of spreading in a hospital environment and can cause a substantial number of nosocomial infections. Furthermore, they recommended the implementation of infection control measures if an outbreak occurs [17].

Several limitations of this study should be discussed. Patients are mainly kept in single or 2-bed rooms. Less than 10% of all hospital beds are in 3- or 4-bed rooms. In addition, we cannot rule out special precautions for patients with hematological malignancies. However, these precautions were never applied to these patients, although they can be ordered by the treating physicians, and such precautions were not documented in the charts.

Despite these 2 reports of outbreaks, we believe that our findings clearly demonstrate no need for contact isolation of patients colonized or infected with VRE *vanC*. We therefore propose that the Centers for Disease Control and Prevention guidelines [19] should be adapted for VRE *vanC*, to avoid unnecessary isolation days. Furthermore, we could demonstrate an extremely low risk of invasive infection in colonized patients with underlying hematological disorders and immunosuppression.

These data provide strong evidence that carriers of VRE *vanC* do not require contact isolation, thereby saving resources and potentially improving patient care. The genotype of VRE should be routinely determined in areas with a high prevalence of VRE *vanC*.

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