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Clinical and microbiological study of vancomycin-resistant enterococci isolated from colonized and infected patients with special reference to risk factors

Deepa Devhare [1]* , Sae Pol [2]

1. BVDUMC, Pune, India
2. BJ. Government Medical College, Pune, India
* Correspondence: Deepa Devhare. BVDUMC, Pune, India. Tel: +918208422612; Email: deepadevhare@gmail.com

Abstract

Background: Vancomycin-resistant enterococci (VRE) has become a growing concern in healthcare settings as a major cause of many nosocomial infections worldwide. Risk factors associated with VRE are important to study. High-risk patients need to be screened and isolated to prevent the spread of infection and colonization. The present study aims to investigate the clinical spectrum, risk factors, and source of transmission of VRE in infected and colonized patients.

Methods: A prospective observational study was carried out for 1 year. A total of 200 *Enterococcus species* isolated from clinical samples such as urine, pus, blood, sterile body fluids, and stool from 200 patients without infection were included in the study. Stool samples were screened to measure the prevalence of VRE colonization. All samples were screened for vancomycin resistance using the Kirby-Bauer disc diffusion method. Vancomycin MIC was detected using the macrobroth dilution method. Demographic and clinical history of the patients were recorded.

Results: Vancomycin resistance was detected in 7 (3.5%) of 200 enterococci isolates from clinical samples. Urinary tract infection (n = 5, 71.4%) was the most common clinical illness caused by VRE. Gut colonization was found in 12 (6%) out of 200 patients screened for VRE. A history of previous antibiotic exposure was a significant risk factor in the current study and was associated with VRE infection and colonization. Endogenous bloodstream infection caused by VRE was found in one patient with VRE colonization.

Conclusion: The findings of this study highlight the significant burden of VRE on patients, both those infected and colonized. The emergence of multidrug-resistant bacteria in healthcare settings, a consequence of inappropriate antibiotic use, is a serious concern that warrants further research and our continued attention.

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Introduction

Enterococcus is a typical commensal bacterial genus of humans' oral cavity, gut, and genitourinary tract (1,2). However, it has become a growing concern in healthcare settings as they are a significant cause of many nosocomial infections worldwide (3-5). Some species of the genus *Enterococcus,* such as *Enterococcus faecium* and *Enterococcus faecalis,* are important opportunistic pathogens that can cause severe infections in susceptible hosts (2,6). This organism can survive in the hospital environment longer due to its intrinsic resistance to several commonly used antibiotics and its ability to acquire resistance to all currently available antibiotics.

The increasing and inappropriate use of antibiotics, such as cephalosporins and especially vancomycin, has led to the emergence and increase in the prevalence of colonization and infections by vancomycin-resistant enterococci (VRE) (7). Other underlying clinical conditions and known risk factors can predispose to enterococcal colonization and infection, such as structural abnormality of the urinary tract, hematologic malignancy, neutropenia, hepatorenal insufficiency, hypoalbuminemia, diabetes mellitus, prolonged hospital stay, and use of immunosuppressants (8-12). The infections associated with VRE are urinary tract infections, endocarditis, bacteremia, wound infections, intraperitoneal infections, and meningitis (13-16).

The clinical profile and risk factors for VRE infection and colonization must be studied, as they are poorly understood (6). This helps identify the high-risk patients who have a higher chance of getting an infection with VRE. As vancomycin resistance is plasmid-encoded, precautionary and infection control measures can be taken in advance to prevent VRE infection and its spread in the hospital environment (10,13,17). Therefore, the present study was conducted to study the clinical spectrum of VRE infections, risk factors associated with VRE infection, and colonization and to determine the source of VRE transmission in a tertiary care hospital in western Maharashtra, India.

Methods

A prospective observational study was conducted at the Microbiology Department of a tertiary care hospital in western Maharashtra, India. The study period was from January 2013 to December 2013 and commenced after approval by the institutional ethics committee. (Reference No.- BMC/IEC/Pharmac/D0313005-05). Informed consent was obtained from all patients included in the study.

Patients of all age groups and both sexes visiting the inpatient and outpatient Departments of a tertiary care hospital in western Maharashtra, India. The first group included 200 enterococcal strains isolated from clinical samples of urine, blood, cerebrospinal fluid (CSF), pleural fluid, ascetic fluid, pus, and wound swabs from patients treated at the hospital with different infections. The second group included 200 stool samples from patients without any infections who were admitted to the hospital and screened for the presence of VRE in the gastrointestinal tract.

Details of personal history along with the history of hospitalization, clinical history regarding any underlying comorbidity or predisposing factors such as diabetes mellitus, malignancy, surgical procedure, immunosuppressive drug therapy, any systemic illness, antibiotic treatment history, and duration of the hospital stay of each patient who presented infection and colonization by VRE were obtained.

Blood agar (Himedia, India) and MacConkey agar (Himedia, India) plates were used to inoculate clinical samples. Samples were inoculated on the culture media with a sterile nichrome loop and incubated under aerobic conditions at 37 °C for 24-48 h. Colony morphology, catalase test, and Gram stain were used for preliminary identification of Enterococci. For genus-level identification of *Enterococcus*, bile aesculin test, 6.5% salt tolerance test, and pyrrolidinyl arylamines (PYR) tests were used. Species-level identification was made only for isolates showing resistance to vancomycin by the Kirby Bauer disc diffusion method. Mannitol fermentation, motility, pigment production, arabinose fermentation, and arginine hydrolysis tests were used for species-level identification (14-16).

Stool samples were screened for the presence of VRE by using bile esculin azide agar plus 6 µg/mL of vancomycin (BEAV, Himedia, India). After 24 h of incubation, if at least one colony had grown along with the darkening of the medium, then Gram stain and catalase tests were performed for presumptive identification of Enterococci (17). The microbiological tests used for species level identification were the same as described above in "Isolation and identification of *Enterococcus* spp. from clinical samples" (14-16).

Vancomycin susceptibility testing:

A. Kirby-Bauer disc diffusion method was used to assess the susceptibility of enterococci isolated from clinical samples and stool samples to vancomycin. Discs containing 30 µg of vancomycin (Himedia, India) were used (18).

B. Confirmation of vancomycin resistance and determination of vancomycin minimal inhibitory concentration (MIC) was performed using the macrobroth dilution method. Serial dilutions of vancomycin (Himedia, India) were prepared in brain-heart infusion (BHI) (Himedia, India) broth. The liquid culture of each bacterial isolate was standardized to 0.5 turbidity on the standard McFarland scale. An aliquot of 10 µL of each bacterial culture was inoculated in 500 µL of BHI broth with different concentrations of vancomycin ranging from 0.5 µg/mL

to 512 µg/mL. Broths were incubated at 37 °C for 24 h. MIC was determined based on the vancomycin concentration inhibiting the bacterial isolate's visible growth evaluated in broth. Vancomycin MIC of ≥ 32 µg/mL was considered resistant, MIC of 8–16 µg/mL was considered intermediate resistance, and MIC of ≤ 4 µg/mL was considered sensitive (18,19).

Environmental swabs from VRE-infected and colonized patients' surroundings, such as beds, trolleys, saline stands, side tables, and doorknobs, were collected to identify the transmission source in these patients (20). The samples were processed aerobically on bile esculin azide agar plus 6 µg/mL vancomycin (Himedia, India) (17). Any growth obtained was followed in the same manner as described above in "Isolation and identification of VRE from stool samples" (20,21).

Microsoft Excel spreadsheets were used for data entry and analysis. Statistical significance was calculated using the Chi-square test at 5% probability ($p < 0.05$).

Results

In the present study, 200 isolates of *Enterococcus species* were isolated from different clinical samples. Out of 200 isolates of Enterococcus species, 7 (3.5%) showed resistance to vancomycin by both disc diffusion and macrobroth dilution methods. Most VRE isolates (n = 5, 71.4%) were isolated (n = 5, 71.4%) from UTI patients.

Of 200 stool samples screened to assess VRE colonization, 12 patients (6%) had VRE. All *Enterococcus* isolated from patients infected (7/200) and colonized (12/200) with VRE were identified as *E. faecium*.

Of 7 patients with VRE infection, 5 (71.4%) were female, and 2 (28.6%) were males; female to male ratio was 2.5:1. Out of a total of 12 patients with VRE colonization, 9 patients (75.0%) were female and 3 (25.0%) were males. The female-to-male ratio was 3:1 in VRE-colonized patients. VRE infection was found to be more in the age group 61-70 years (n = 3, 42.9%), and VRE colonization was found to be more in the age group 41-60 years (n = 8, 66.7%) (Table 1).

Table 1. Age group-wise distribution of VRE infection and colonization

Age groups(years)	Number of VRE-infected patients (n=7) (%)	Number of VRE colonized patients (n=12) (%)
1-10	0	1 (8.3)
11-20	0	0
21-30	1 (14.3)	1 (8.3)
31-40	1 (14.3)	0
41-50	1 (14.3)	4 (33.3)
51-60	0	4 (33.3)
61-70	3 (42.9)	2 (16.7)
71-80	1 (14.3)	0

Risk factors associated with VRE infection and colonization: In the present study, all patients (100%) with VRE infection had a history of antibiotic exposure in the past month before VRE isolation, which showed a statistically significant association ($p < 0.05$). The most common antibiotics exposed were cephalosporins. Only one patient with VRE infection had a history of previous exposure to vancomycin (Table 2). Other underlying medical comorbid medical conditions did not show statistical significance with VRE infection, as demonstrated in Table 3.

Table 2. Antibiotic exposure in VRE-colonized and infected patients

Antibiotic	VRE colonization (n=12) (%)	VRE infection (n =7) (%)
Cephalosporins	12 (100.0)	7 (100.0)
Aminoglycosides	2 (16.7)	2 (28.6)
Fluroquinolones	4 (33.3)	1 (14.3)
Metronidazole	2 (16.7)	2 (28.6)
Penicillin/beta lactam+ beta lactamase inhibitors	3 (25.0)	0
Vancomycin	0	1 (14.3)

Table 3. Risk factors associated with VRE infection

Risk factor	Number of patients with VRE infection (n= 7)	Percentage (%)	Statistical significance ($p < 0.05$ - statistically significant)
Antibiotic exposure in the past 1 month	7	100.0	$p < 0.05$
ICU stay for >2 weeks	3	42.9	$p > 0.05$
Urinary catheterization	3	42.9	$p > 0.05$
Chronic kidney disease	2	28.6	$p > 0.05$
Diabetes mellitus	1	14.3	$p > 0.05$
HIV infection (Immunosuppression)	1	14.3	$p > 0.05$
VRE colonization	1	14.3	$p > 0.05$

In the present study, all patients (100.0%) with VRE colonization also had a history of antibiotic exposure, specifically cephalosporins, in the past month before VRE isolation, which was found to have a statistically significant association ($p < 0.05$). Other known risk factors like hospitalization for more than two weeks, malignancy, and diabetes were also analyzed but were not found to be significant. One colonized patient had an incidence of bloodstream infection caused by VRE (Table 4). All Environmental swabs collected from VRE-infected and colonized patients' surroundings were negative for VRE.

Table 4. Risk factors associated with VRE colonization

Risk factors	Number of patients with VRE colonization (n= 12)	Percentage (%)	Statistical significance ($p < 0.05$ - statistically significant)
Antibiotic exposure in past 1 month	12	100.0%	$p < 0.05$
Prior hospitalization for > 2 weeks	6	50 %	$p > 0.05$
Malignancy	4	33.3 %	$p > 0.05$
Diabetes mellitus	3	25 %	$p > 0.05$

Discussion

In the present study, urinary tract infection was the most common infection caused by VRE. Other authors have also shown a predominance of urinary tract infections caused by VRE (1,10,22). *Enterococcus* spp. are typically found in the genital and gastrointestinal tracts as normal commensal flora. They can enter the urinary tract in susceptible individuals with predisposing factors such as indwelling catheters, instrumentation, renal failure, kidney stones, or immunosuppression, leading to endogenous infection (23).

E. faecium was the only species isolated from all patients infected and colonized with VRE. Other studies conducted by Deshpande et al. (2013), Rahangdale et al. (2008), and Baragundi et al. (2010) also showed greater isolation of *E. faecium* among VRE isolates than any other *Enterococcus* species (24-26). *E. faecium* can acquire resistance against most of the available antibiotics by genetic transfer of resistance, which can be the reason for the higher isolation of this species from VRE isolates (27).

The female-to-male ratio of patients infected with VRE was 2.5:1. The predominance of VRE infection in females can be attributed to higher isolation of VRE in urinary tract infections, which is usually more common in females than males. Age groups commonly affected by VRE infection and VRE colonization were 41-70 years of age. The presence of comorbidities, such as diabetes mellitus, immunosuppression, and malignancies, are more common in the older age group, explaining more chances of infection and colonization with drug-resistant bacteria (10).

Various well-known risk factors and predisposing conditions associated with VRE infection are described in different studies, which include antibiotic exposure, prolonged hospitalization, invasive therapy, immunosuppression, diabetes mellitus, malignancy, and hepatorenal insufficiency (4,7-9,28). In the present study, prior antibiotic exposure was statistically associated with infection and colonization by VRE. Many other studies have shown similar findings (29-31).

Cephalosporins, fluoroquinolones, metronidazole, penicillins, and aminoglycosides were the other antibiotics to which VRE-infected and colonized patients were exposed in the present study. Previous vancomycin exposure in a patient causes selective pressure on the normal commensal flora and promotes the proliferation of VRE (6). In the present study, vancomycin exposure was present only in one patient (14.3%) of VRE infection. In previous studies, VRE infection and colonization were associated not only with vancomycin but mainly with cephalosporins, aminoglycosides, ciprofloxacin, and antibiotics used for anaerobes (10,28).

Other known risk factors, such as duration of ICU stay, invasive procedures, diabetes, immunosuppression, and malignancy, were not found to be statistically significant in the causation of infection and colonization by VRE. The reason could be the difference in sample size and prevalence of VRE (10).

One important finding in the present study was postoperative septicemia by VRE in a patient with gut colonization with VRE. This patient had undergone cardiovascular surgery and developed septicemia by VRE and died. If a patient is colonized with VRE, they have a 5-10-fold increased risk of developing serious endogenous infections with VRE (5). This highlights the need for screening of high-risk patients with predisposing factors for VRE colonization especially before any major surgical procedure, which prevents developing life-threatening infection. Also, if the infection develops in colonized patients, appropriate antibiotics should be started without delay. Necessary infection control measures can be taken while handling such colonized patients so that the spread of VRE to the environment and cross infections to other patients can be prevented (21).

Environmental swabs collected from the surroundings of VRE-infected and colonized patients were negative for VRE. This could be due to transient colonization of the surroundings by VRE (28). Other possible sources of VRE can be cross-infection from other VRE colonized patients, through the hands of healthcare workers, or through contaminated medical devices, which needs to be studied in detail (28,32,33).

Conclusion

Patients infected as well as colonized with VRE represent the actual burden of VRE. Screening high-risk patients is an important step in the prevention of the spread of VRE infection and its accurate treatment. Inappropriate use of antibiotics has led to the emergence of multidrug-resistant bacteria in healthcare settings. Antibiotic stewardship plays a major role in such a situation to reduce the inappropriate overuse of antibiotics, which can prevent the emergence of multidrug resistance in bacteria.

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Ethical statement

The study was approved by the institutional ethics committee of B.J. Govt. Medical College, Pune (With protocol reference number BMC/IEC/Pharmac/D0313005-05) and was carried out following the approved guidelines.

Conflicts of interest

None.

Author contributions

All the authors have made substantial, direct, and intellectual contributions to the work.

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