

Original Research Article

Prevalence of fecal carriage of vancomycin resistant *Enterococci* in a tertiary care centre in south India

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ABSTRACT

Background: The number of VRE infections gradually increases worldwide and ICUs in hospitals which remains to be the major reservoir of antibiotic-resistant organisms. The current study was carried out to determine prevalence of fecal carriers of vancomycin resistant *Enterococci* in a tertiary care hospital, thereby preventing the spread of vancomycin resistant *Enterococci* to patients under high risk.

Methods: A total of 219 stool samples and rectal swabs were collected from health care workers and ICU patients and processed in vancomycin resistant *Enterococci* agar and subjected to certain phenotypic tests for its species identification. Antimicrobial susceptibility testing was done for all the vancomycin resistant enterococcal isolates.

Results: A total of 73 VRE were isolated of which 64 (36%) were from HCW and 9 (22%) were from ICU patients. The prevalence of VRE was 33% and the most common VRE isolated was *Enterococcus faecium*. All the 73 VRE isolates were susceptible to linezolid and 52% were susceptible to quinpristine/dalfopristine.

Conclusions: Screening of stool samples aids in the prevention of spread of multi-drug resistant VRE isolate from HCW to high risk patients. It's imperative to step up infection control strategies so as to curtail its spread.

Keywords: Fecal carriers, Health care workers, Vancomycin resistant *Enterococci*

INTRODUCTION

Vancomycin resistant *Enterococci* (VRE) is a major concern amongst hospital. These resistant species have the ability to survive for prolonged periods and they can get transmitted easily. The number of VRE infections gradually increases worldwide and ICUs in hospitals remains to be the major reservoir of antibiotic-resistant organisms. Colonization of VRE in healthy population is less than 1% while the prevalence rate is higher among the patients admitted in ICU's.¹

The infections caused by these organisms range from complicated urinary tract infections (UTI) to life threatening central nervous system (CNS) infections making the treatment challenging. VRE infections are

increasingly common and difficult to treat appearing usually as hospital outbreaks that present tremendous challenges for infection control. Transmission of VRE can be reduced by proper hand hygiene, use of gloves and gowns while treating infected patients, use of dedicated equipment for the patients and isolation of patients with VRE infection. Other interventions made to control the spread of VRE infection is active surveillance and screening of asymptotically colonized patients. Detection of fecal carriers of VRE is important in preventing its spread and causing infections to high risk patients.²

Thus, the present study intended to determine prevalence of fecal carriers of VRE in a tertiary care hospital, thereby preventing the spread of VRE to patients under high risk.

METHODS

This study was carried out in 178 stool samples and 41 rectal swabs received from health care workers (dietary staffs) and ICU patients respectively between January 2024 to July 2024 at KMCH Institute of Health Sciences, Coimbatore. The study was started after obtaining approval from the institutional human ethics committee. All the stool samples received in the laboratory and rectal swabs from ICU patients were included for the study.

Samples received from the same patient more than once was not included in the study. The samples were obtained after getting consent from the patients and health care workers. Samples were inoculated into vancomycin resistant *Enterococci* (VRE) agar base (HiMedia Pvt Ltd, India) with added vancomycin and meropenem supplement and incubated at 37°C for 18-24 hours. Tiny black-coloured colonies indicated the growth of vancomycin resistant *Enterococci* after subjecting to Gram stain which showed Gram-positive cocci in pairs which were spherical in shape with a characteristic spectacle arrangement. Different species of *Enterococci* were identified by performing certain phenotypic tests like

fermentation of mannitol, arabinose, raffinose, sucrose, lactose, sorbose, sorbitol and arginine dihydrolase test.

Kirby Bauer disk diffusion method was used for determining the susceptibility of the isolates to the commonly used antibiotics against *Enterococcus spp.* using the standard guidelines issued by the Clinical Laboratories Standards Institute (CLSI) 2024. The susceptibility and resistance were noted for the isolates after 24 hours of incubation at 37°C.

RESULTS

A total of 219 stool/rectal swab samples were collected. Of which 178 (81%) samples were from health care workers and 41 (19%) from the ICU patients. The prevalence of VRE in our study was found to be 33%. A total of 73 VRE were isolated of which 64 (36%) were from HCW and 9 (22%) were from ICU patients. VRE were isolated more from males both in HCW and ICU patients. The isolation rate was 33% among the HCW and ICU patients in total. About 33% (n=35) of the HCW with VRE isolates were in the age group of <35 years whereas for ICU patients 5 isolates were isolated from patients >55 years of age.

Table 1: Speciation of *Enterococcus* group.

Species	Number of strains	Group	Reaction (% positive) ^a			
			Mannitol	Sorbitol	Sorbose	Arginine
<i>E. raffinosus</i>	2	I	+(100)	+(100)	+(100)	-(0)
<i>E. faecalis</i> , <i>E. faecium</i>	60	II	+(100)	V (87)	-(0)	+(100)
<i>E. faecalis</i> , <i>E. hirae</i>	11	III	-(0)	-(0)	-(0)	+(100)

^a + Positive reaction; - negative reaction; V variable (some strains positive, some strains negative). ^b Asaccharolytic variant.

Table 2: Identification of group II species^a.

Species	No. of strains	Reaction (% positive) ^b				
		Arabinose	Sorbitol	Lactose	Motility	Pigment
<i>E. faecalis</i>	23	-(0)	+(91)	+(100)	-(0)	-(0)
<i>E. faecium</i>	37	+(100)	+(92)	+(100)	-(0)	-(0)

^a Key reactions, Mannitol and arginine (+) and sorbose (-). ^b + Positive reaction; - negative reaction

Table 3: Identification of group III species^a.

Species	No. of strains	Reaction (% positive) ^b		
		Sucrose	Raffinose	Pyruvate
<i>E. hirae</i>	1	+(100)	+(100)	-(0)
<i>E. faecalis</i> (asaccharolytic variant)	10	-(0)	-(0)	+(100)

^a Key reactions, mannitol, sorbitol, sorbose (-) and arginine (+); ^b + positive reaction; - negative reaction; V- variable (some strains positive, some strains negative).

Patients were admitted in MICU for various conditions and the most common among them were pulmonary edema, heart failure and respiratory failure, post renal transplant. The mean duration of hospital stay was found to be 5 days. Out of the 219 samples, 73 VRE were isolated in VRE agar plate it grew as a minute colony with black discoloration

as depicted in Figure 1 and in Gram stain it was Gram-positive cocci in pairs with typical bi-spectacle arrangement. Several biochemical tests were performed to speciate the *Enterococcus* and their results are depicted in Table 1.

Group I species consists mostly of *E. raffinosus*, *E. avium*, *E. malodoratus* and *E. pseudoavium*. In our study we had isolated only two species of group I isolate which was arabinose and raffinose positive and was speciated as *E. raffinosus*. The members of group II species were *E. faecalis*, *E. solitarius*, *E. gallinarum*, *E. faecium*, *E. casseliflavus*, *E. mundtii*. The test for their identification is depicted in Table 2.

Enterococcus species in group III are *E. hirae*, *E. durans*, *E. faecalis* asaccharolytic variant. They were categorized

based on the sucrose and raffinose fermentation and their results are depicted below in Table 3.

The susceptibility pattern for the individual species towards different antibiotics is shown in Table 4. All the 73 VRE isolates were susceptible to linezolid and 52% were susceptible to quinpristine/dalfopristine. The sensitivity towards teicoplanin was found to be 22% and 30% to *E. faecalis* and *E. faecium* respectively.

Table 4: Susceptibility pattern of VRE species.

Antibiotics	% of sensitive isolates(N)				
	<i>E. faecalis</i> N=23	<i>E. faecium</i> N=37	<i>E. raffinosus</i> N=2	<i>E. hirae</i> N=1	<i>E. faecalis</i> N=10
Ampicillin	83 (19)	84 (31)	50 (1)	100 (1)	100 (10)
High level gentamicin	87 (20)	95 (35)	50 (1)	100 (1)	90 (9)
Tetracycline	43 (10)	57 (21)	50 (1)	100 (1)	60 (6)
Erythromycin	52 (12)	49 (18)	50 (1)	100 (1)	70 (7)
Teicoplanin	22 (5)	30 (11)	0	100 (1)	60 (6)
Linezolid	100 (23)	100 (37)	100(2)	100 (1)	100 (10)
Quinpristin/dalfopristin	-	97 (36)	50 (1)	100 (1)	-

^a Asaccharolytic variant

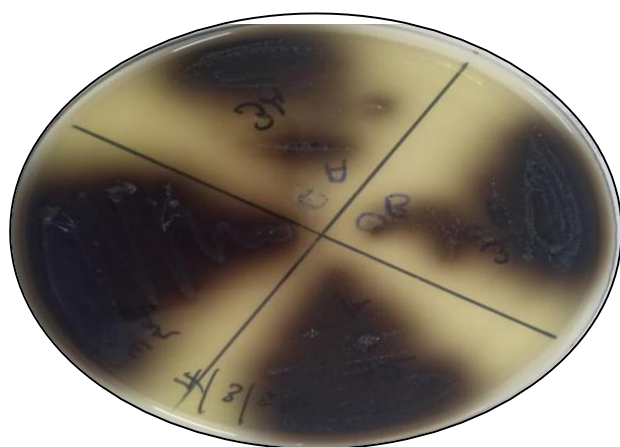


Figure 1: Colony morphology of VRE.

DISCUSSION

The emergence of VRE as a major pathogen is associated with a tendency to colonize the gastrointestinal tract (GIT), persistence in hospital settings, genomic plasticity, migrating genetic factors, and increased mortality.³ The epidemiology of VRE varies from hospital to hospital, depending on several factors, including hospital size, patient population, antibiotic use patterns, and geographic location. According to previous reports, risk factors that increase the likelihood of VRE infection or colonization may be related to host factors, hospital-specific factors, and antibiotic use.⁴ The most frequent method of nosocomial transmission of VRE is most likely when

healthcare professionals who are caring for infected patients have their hands momentarily contaminated with the bacterium. The recovery of VRE and other resistant enterococci from cultures of specimens from healthcare workers' hands lends weight to this idea.⁵ This study was done to illustrate the prevalence of VRE among health care workers from stool samples. The dietary staff of the hospital acts as a carrier for the spread of VRE among healthy individuals and patients too. The prevalence of VRE in ICU patients was also compared.

Compared to the western world, India has a significantly lower prevalence of VRE infections, albeit it has been rising over the past ten years.⁶ The overall prevalence of VRE in this study was found to be 33%. In a study conducted by Rajesh et al, the intestinal carriage rate of VRE in ICU patients was found to be 29%.¹ According to published data, the rate of VRE colonization differed widely by region, with the USA reporting greater rates (12.3%) than Europe (2.7%), South America (7%), Asia (5.3%), and Oceania (4.4%). According to Zakias et al, the average rate of VRE colonization in US hospitals is 12.5%, however certain institutions reported substantially higher rates of 42% at admission, which are much higher than the rates observed in the current study.⁵ In a study conducted by Joseph et al, the prevalence of stool colonization of VRE was found to be 5.3% among hospital employees.⁶ In our study the prevalence of VRE colonization among HCW was found to be 36% which is higher when compared to the other studies and among ICU patients it was 22% which is lesser when compared to the study

conducted by Rajesh et al.¹ On comparing the prevalence of intestinal carriage of VRE among HCW and ICU patients, it was found that the risk is higher in HCW and this is statistically significant. It has been reported that the widespread use of third-generation cephalosporins and medications with strong anti-anaerobe activity exerts antibiotic selection pressure, which increases the risk of VRE colonization and infection.⁹

In a study by Joseph et al, out of 110 VRE isolates *E. faecium* accounts for 64% of HCW.⁶ In another study by Rajesh et al, for ICU patients, 77.2% of VRE isolates were *E. faecium* and 23.6% were *E. faecalis*.¹ Similarly in our study, 51% of the VRE isolates were *E. faecium* followed by *E. faecalis* which is about 45%. Vancomycin resistance is widely seen in *Enterococcus faecium* when compared to other *Enterococcus* species.

VRE colonization has been shown to raise the risk of subsequent VRE infections, but this finding is not universal; rates ranging from 0-45% have been recorded in different studies, while the risk in patients who are not colonized is less than 2%.⁵ In a study by Rajesh et al, only one of the 214 non-colonized patients was infected, compared to 4 of the 88 patients who had VRE colonization (4.5%).¹ In our study, none of the ICU patients who were colonized by VRE developed infections on VRE subsequently. In another study, done by Papadimitriou-Olivgeries et al, it was discovered that 4 out of 107 patients who had been colonized became infected with the same strain.¹⁰ They made the crucial insight that patients with VRE colonization could also contract VRE isolates in the future.

The options for treating serious infections caused by *Enterococcus* have been significantly constrained by the propensity of these species to quickly acquire resistance genes and the availability of some specific mechanisms giving resistance to antibiotics like aminoglycosides and glycopeptides.¹¹ Strains of VRE that contain the *vanA* gene show a high level of resistance to vancomycin and teicoplanin, whereas the *vanB* gene is resistant only to vancomycin but sensitive to teicoplanin.¹² In this study, about 88% and 70% of *E. faecalis* and *E. faecium* were resistant to teicoplanin respectively. Both the isolates of *E. raffinosus* were resistant to teicoplanin. It appears that *vanA* may be the phenotype for vancomycin-resistant *E. faecalis* and *E. faecium*, based on the Kirby Bauer disc diffusion results. Despite being uncommon, linezolid-resistant enterococci outbreaks have been observed recently. Even without any prior exposure to linezolid, cases of linezolid-resistant vancomycin-resistant *E. faecium* infection have been documented.⁸ In a study by Pradnya et al, all the VRE isolates were sensitive to linezolid which is similar to our study.¹² Quinpristin/dalfopristin is a combination of two antibiotics that is used to treat infections by *Staphylococci* and Vancomycin-resistant *Enterococcus faecium*. 97% of *E. faecium* were found sensitive to quinpristin/dalfopristin in our study and they are intrinsically resistant to *E. faecalis*.

There are some limitations of the study. Vancomycin administration or the use of broad-spectrum cephalosporin antibiotics is widely cited as a risk factor for VRE infection or colonization. However, our investigation was unable to conclusively link the usage of antibiotics to the occurrence of VRE strains in fecal flora.

CONCLUSION

The likelihood that an HCW will be exposed to a particular pathogen is presumably correlated with how common the pathogen is in patient populations or the environment. The presence of VRE in the stools of the HCW suggests that VRE form part of the normal human fecal flora or can be acquired in the community. The dietary staffs who are involved in food handling and their supply for the patients may pose a high risk for the spread of VRE which is evident in our study. They remain as a silent carrier and are responsible for transmission of VRE infection. The prevalence of VRE in the community is a further element that has to be researched for an accurate assessment of occupational risk because these organisms are becoming progressively isolated from non-hospitalized people.

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