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Original Research Article

PHENOTYPIC AND GENOTYPIC CHARACTERIZATION OF VANCOMYCIN RESISTANT ENTEROCOCCI (VRE) ISOLATED FROM CLINICAL SAMPLES IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Enterococci have emerged as important nosocomial pathogens due to their intrinsic resistance and ability to acquire antimicrobial resistance genes. The emergence of Vancomycin Resistant Enterococci (VRE) is a major therapeutic challenge because of limited treatment options and potential transmission of resistance determinants. The aim is to determine the prevalence of Vancomycin Resistant Enterococci among clinical isolates and characterize resistance phenotypes and genotypes by phenotypic methods and PCR

Materials and Methods: A cross-sectional study was conducted in the Department of Microbiology, Chengalpattu Medical College, from March 2016 to February 2017. A total of 200 Enterococcus isolates recovered from urine, pus, blood, and body fluids were included. Species identification was performed using conventional biochemical methods. Antimicrobial susceptibility testing was performed by Kirby–Bauer disc diffusion method according to CLSI guidelines. Vancomycin resistance was screened by vancomycin screen agar and confirmed by vancomycin minimum inhibitory concentration (MIC) using broth microdilution method. Detection of vanA and vanB genes was performed by PCR.

Results: Among 200 Enterococcus isolates, five (2.5%) were identified as VRE. Of these, three were E. faecium and two were E. faecalis. VRE isolates showed vancomycin MIC values ranging from 32–128 µg/ml. PCR analysis revealed vanA gene in three isolates and vanB gene in two isolates. All VRE isolates showed susceptibility to linezolid and tigecycline.

Conclusion: The detection of VRE with transferable resistance genes emphasizes the need for routine screening, molecular confirmation, antimicrobial stewardship, and strict infection control practices to prevent dissemination of resistant Enterococcus strains.

Keywords: Enterococcus, Vancomycin Resistant Enterococci, vanA, vanB, PCR, antimicrobial resistance.

INTRODUCTION

Enterococci are Gram-positive, facultative anaerobic cocci that are normal inhabitants of the gastrointestinal tract of humans and animals. Although traditionally considered as low-grade pathogens, Enterococcus species have emerged as important opportunistic pathogens, particularly in hospitalized and immunocompromised patients.[1] The ability of enterococci to survive under adverse environmental conditions, acquire antimicrobial resistance determinants, and persist in healthcare settings has contributed to their increasing importance as nosocomial pathogens.[2] Among the various Enterococcus species, Enterococcus faecalis and Enterococcus faecium account for the majority of human infections.[3] Enterococci are commonly associated with urinary tract infections, bloodstream

infections, wound infections, intra-abdominal infections, endocarditis, and infections related to indwelling medical devices.[4] The increasing use of broad-spectrum antibiotics and prolonged hospital exposure has contributed significantly to the emergence of multidrug-resistant enterococci.[5] Vancomycin has historically been an important therapeutic option for the treatment of serious enterococcal infections. However, the emergence of Vancomycin Resistant Enterococci (VRE) has become a major global healthcare concern. Vancomycin resistance in enterococci is mainly mediated by acquired resistance genes, particularly vanA and vanB, which encode enzymes responsible for alteration of the vancomycin binding target.[6] The vanA genotype is generally associated with high-level resistance to both vancomycin and teicoplanin, whereas vanB usually confers variable resistance to vancomycin with susceptibility to teicoplanin.[7] VRE infections are associated with increased morbidity, mortality, prolonged hospital stay, and limited therapeutic options. In addition, enterococci may act as reservoirs of transferable vancomycin resistance genes that can potentially spread to other clinically significant organisms. Therefore, early identification of VRE and characterization of resistance mechanisms are essential for effective antimicrobial therapy and infection control practices.[8] Phenotypic detection methods such as vancomycin susceptibility testing, vancomycin screen agar, and minimum inhibitory concentration (MIC) determination are routinely used for identification of VRE. However, molecular detection by polymerase chain reaction (PCR) provides rapid and accurate identification of resistance genes and helps in epidemiological surveillance.[9] The present study was undertaken to determine the antimicrobial susceptibility profile of Enterococcus isolates with special emphasis on vancomycin resistance, to detect VRE by phenotypic methods, and to identify vanA and vanB resistance genes among clinical isolates using PCR.

MATERIALS AND METHODS

Study Design and Setting: A cross-sectional study was conducted in the Department of Microbiology, Chengalpattu Medical College, Chengalpattu, over a period of one year from March 2016 to February 2017. The study included Enterococcus species isolated from various clinical specimens received from patients attending the tertiary care hospital.

Study Population and Sample Size: A total of 200 non-duplicate Enterococcus isolates obtained from clinical samples were included in the study. The specimens included urine, pus/wound swabs, blood, and body fluids.

Inclusion Criteria

All Enterococcus isolates recovered from clinical specimens such as urine, pus, blood, and body fluids were included.

Exclusion Criteria

Duplicate isolates from the same patient and non-enterococcal bacterial isolates were excluded from the study.

Isolation and Identification of Enterococcus Species: All clinical specimens were processed by standard microbiological techniques. Direct Gram staining was performed for specimens other than blood samples. The presence of Gram-positive cocci arranged in pairs and short chains was considered suggestive of Enterococcus species. The specimens were inoculated onto Blood agar, Nutrient agar, and MacConkey agar and incubated aerobically at 37°C for 18–24 hours. Urine samples were processed by semi-quantitative culture method using calibrated loop technique on Cysteine Lactose Electrolyte Deficient (CLED) agar. Significant bacteriuria was considered based on colony count and clinical correlation. Suspected Enterococcus colonies were identified based on colony morphology and biochemical characteristics.

Phenotypic Identification and Speciation of Enterococcus Species: Presumptive identification of Enterococcus isolates was performed using standard phenotypic methods. The isolates were initially confirmed by Gram staining, which demonstrated Gram-positive cocci arranged in pairs and short chains. Biochemical identification was carried out using catalase test, bile esculin hydrolysis, PYR test, growth in 6.5% sodium chloride, heat tolerance test at 60°C, growth at 10°C and 45°C, growth at pH 9.6, and litmus milk reduction test. The confirmed Enterococcus isolates were further characterized to species level using conventional biochemical identification methods based on Facklam and Collins criteria. Speciation was performed using carbohydrate fermentation tests, arginine hydrolysis, motility testing, pigment production, and other differentiating biochemical reactions.

Detection of Virulence Factors

Hemolysin Production: Hemolysin production was detected by inoculating isolates on 5% sheep blood agar and incubating overnight. A clear zone of complete hemolysis around colonies was considered positive.

Gelatinase Production: Gelatinase production was tested using gelatin agar. A zone of clearing or turbid halo around colonies after incubation indicated gelatinase activity.

Antimicrobial Susceptibility Testing: Antimicrobial susceptibility testing was performed by the Kirby–Bauer disk diffusion method on Mueller–Hinton agar following the Clinical and Laboratory Standards Institute (CLSI) 2016 guidelines. The inoculum was standardized to 0.5 McFarland turbidity standard before inoculation. Enterococcus faecalis ATCC 29212 was used as the quality control strain. The antimicrobial agents tested included penicillin, ampicillin, erythromycin, doxycycline, ciprofloxacin, levofloxacin, nitrofurantoin, high-level gentamicin, high-level

streptomycin, vancomycin, teicoplanin, and linezolid. The zones of inhibition were measured and interpreted according to CLSI breakpoints. Vancomycin-resistant Enterococcus (VRE) isolates were further evaluated for susceptibility to therapeutic agents including linezolid, tigecycline, and quinupristin/dalfopristin.

Detection of High-Level Aminoglycoside Resistance: High-level gentamicin resistance (HLGR) and high-level streptomycin resistance (HLSR) were detected using gentamicin (120 µg) and streptomycin (300 µg) discs on Mueller-Hinton agar. Results were interpreted according to CLSI guidelines.

Phenotypic Detection of Vancomycin Resistance

Vancomycin Screen Agar: Enterococcus isolates were screened for vancomycin resistance using Brain Heart Infusion agar containing vancomycin (6 µg/ml). Growth after incubation indicated presumptive vancomycin resistance.

Vancomycin Minimum Inhibitory Concentration (MIC): Vancomycin MIC was determined by broth microdilution method according to CLSI recommendations. Vancomycin concentrations ranging from 0.5–2048 µg/ml were prepared. MIC was interpreted as the lowest concentration showing complete inhibition of visible bacterial growth.

Molecular Detection of Vancomycin Resistance Genes: DNA extraction was performed from Enterococcus isolates using a bacterial genomic DNA extraction kit. Extracted DNA was subjected to polymerase chain reaction (PCR) for detection of *vanA* and *vanB* genes. PCR amplification was carried out using specific primers targeting the resistance genes. Amplified products were analyzed by agarose gel electrophoresis and visualized under UV transillumination. The expected PCR product size for *vanA* gene was 473 bp.

Statistical Analysis

The obtained data were analyzed and presented using descriptive statistics. Results were expressed as numbers, percentages, tables, and figures. The antimicrobial susceptibility pattern and prevalence of VRE were analyzed and compared with previously published studies.

RESULTS

A total of 200 Enterococcus isolates were obtained from various clinical specimens during the study period. The isolates were recovered from patients of different age groups ranging from pediatric to elderly age groups. The highest number of Enterococcus isolates were observed among patients aged 31–45 years (61; 30.5%), followed by 46–60 years (56; 28%), 16–30 years (29; 14.5%), 61–75 years (27; 13.5%), 0–15 years (21; 10.5%), and above 75 years (6; 3%). Overall, adult patients contributed 179 (89.5%) isolates, whereas pediatric patients contributed 21 (10.5%) isolates. [Table 1]

Table 1: Age-wise distribution of Enterococcus isolates (n=200)

Age group (years)	Number of isolates	Percentage
0–15	21	10.5
16–30	29	14.5
31–45	61	30.5
46–60	56	28
61–75	27	13.5
>75	6	3
Total	200	100

Among the study population, Enterococcus isolates were more frequently recovered from males (117; 58.5%) compared with females (83; 41.5%), with a male-to-female ratio of 1.4:1. [Table 2]

Table 2: Sex-wise distribution of Enterococcus isolates

Sex	Number	Percentage
Male	117	58.5
Female	83	41.5
Total	200	100

The distribution of Enterococcus isolates according to clinical specimens showed that urine was the predominant source of isolation (129; 64.5%), followed by pus/wound swabs (35; 17.5%), blood (28; 14%), and body fluids (8; 4%) [Table 3].

Table 3: Distribution of Enterococcus isolates according to clinical specimens

Specimen	Number	Percentage
Urine	129	64.5
Pus/Wound swab	35	17.5
Blood	28	14
Body fluids	8	4
Total	200	100

Species identification revealed that Enterococcus faecalis was the predominant species isolated, accounting for 134 (67%) isolates, followed by E. faecium 60 (30%). Other species isolated included E. raffinosus (3; 1.5%), E. avium (2; 1%), and E. durans (1; 0.5%). [Table 4]

Table 4: Species distribution of Enterococcus isolates

Species	Number	Percentage
E. faecalis	134	67
E. faecium	60	30
E. raffinosus	3	1.5
E. avium	2	1
E. durans	1	0.5
Total	200	100

Among the clinical specimens, E. faecalis was the most commonly isolated species from urine (85/129), pus (24/35), blood (19/28), and body fluids (6/8). E. faecium was the second most common species isolated across all specimen types. [Table 5]

Table 5: Distribution of Enterococcus species among clinical specimens

Species	Urine	Pus	Blood	Body fluids
E. faecalis	85	24	19	6
E. faecium	39	11	8	2
E. raffinosus	2	0	1	0
E. avium	2	0	0	0
E. durans	1	0	0	0

Virulence factor analysis showed that hemolysin production was detected among 56 (28%) isolates, while gelatinase production was observed in 27 (13.5%) isolates. Hemolysin production was higher among E. faecalis isolates (45; 33.58%) compared with E. faecium (11; 18.33%). [Table 6]

Table 6: Virulence factor production among Enterococcus isolates

Species	Hemolysin positive (%)	Gelatinase positive (%)
E. faecalis	45 (33.58)	16 (11.94)
E. faecium	11 (18.33)	11 (18.33)
Total	56 (28)	27 (13.5)

Antimicrobial susceptibility testing showed variable resistance patterns among Enterococcus isolates. Among E. faecalis, maximum susceptibility was observed with linezolid (100%), followed by teicoplanin (99.25%) and vancomycin (98.5%). Among E. faecium, linezolid showed 100% susceptibility followed by teicoplanin (96.67%) and vancomycin (95%). Penicillin showed the highest resistance among both species. [Table 7]

Table 7: Susceptibility pattern of major Enterococcus species

Antibiotic	E. faecalis Sensitive (%)	E. faecium Sensitive (%)
Penicillin	22.38	13.33
Ampicillin	40.29	35
Ciprofloxacin	43.28	31.67
Nitrofurantoin	89.41	71.79
Vancomycin	98.5	95
Teicoplanin	99.25	96.67
Linezolid	100	100

High-level aminoglycoside resistance was observed among Enterococcus isolates. Combined high-level gentamicin and streptomycin resistance was detected in 51 (38.05%) E. faecalis isolates and 30 (50%) E. faecium isolates. [Table 8]

Table 8: High-level aminoglycoside resistance pattern

Resistance pattern	E. faecalis	E. faecium
HLGR alone	20 (14.92%)	6 (10%)
HLSR alone	44 (32.83%)	14 (23.33%)
HLGR + HLSR	51 (38.05%)	30 (50%)

Vancomycin resistance was detected among five isolates, giving an overall VRE prevalence of 2.5%. Of these, two isolates were E. faecalis and three were E. faecium. [Table 9]

Table 9: Distribution of VRE isolates

Specimen	E. faecalis	E. faecium	Total
Urine	1	1	2
Pus	1	1	2
Blood	0	1	1
Total	2	3	5

PCR analysis of VRE isolates demonstrated that three isolates carried the *vanA* gene and two isolates carried the *vanB* gene.

Table 10: Genotypic characterization of VRE isolates

Resistance gene	Number of isolates	Percentage
vanA	3	60
vanB	2	40
Total	5	100

Overall, the study showed predominance of E. faecalis, higher isolation from urinary samples, considerable multidrug resistance, and emergence of VRE with both vanA and vanB genotypes among clinical Enterococcus isolates.

DISCUSSION

Enterococci have emerged as important opportunistic and nosocomial pathogens due to their intrinsic resistance mechanisms, ability to acquire antimicrobial resistance determinants, and capacity to survive in hospital environments. In the present study, 200 Enterococcus isolates obtained from various clinical specimens were analyzed for species distribution and antimicrobial susceptibility pattern. The predominance of Enterococcus faecalis (67%) followed by Enterococcus faecium (30%) was observed. Similar predominance of E. faecalis has been reported in studies by Srivastava et al, and Barman et al., where E. faecalis was the major clinical isolate, accounting for more than 50% of Enterococcus infections.[10,11]

However, some studies have reported increased isolation of E. faecium, particularly among hospital-associated infections, reflecting geographical variation and differences in antimicrobial exposure.[12]

In the present study, urine was the most common specimen yielding Enterococcus isolates (64.5%), followed by pus/wound samples (17.5%) and blood (14%). Similar findings were reported by Peters et al., where urinary isolates constituted the majority of Enterococcus infections.[13]

Enterococci are well-recognized causes of urinary tract infections, especially among hospitalized patients, elderly individuals, and patients with urinary instrumentation. Studies by Gupta et al. also demonstrated urine as the predominant source of Enterococcus isolation, supporting the findings of the present study.[14]

The antimicrobial susceptibility pattern revealed considerable multidrug resistance among Enterococcus isolates. In this study, E. faecalis and E. faecium showed high susceptibility to linezolid (100%), teicoplanin (99.25% and 96.67%), and vancomycin (98.5% and 95%, respectively). Similar excellent activity of linezolid against Enterococcus isolates has been reported by Billström et al. and Sreeja et al.[15,16]

Linezolid remains an important therapeutic option for infections caused by resistant Enterococci, particularly Vancomycin Resistant Enterococci (VRE). In contrast, penicillin showed poor activity in the present study, which is comparable with observations from previous studies where beta-lactam resistance was frequently encountered among clinical Enterococcus isolates.[16]

High-level aminoglycoside resistance (HLAR) is clinically significant because it eliminates the synergistic effect of aminoglycosides combined with cell wall-active agents. In the present study, HLAR was observed in 38.05% of E. faecalis and 50% of E. faecium isolates. Similar findings were reported by Padmasini et al., who demonstrated increasing aminoglycoside resistance among Enterococcus isolates.[17] The higher resistance among E. faecium emphasizes its role as a more resistant nosocomial pathogen. Vancomycin resistance was detected among 5 isolates (2.5%) in the present study. Among these, three isolates carried the vanA gene and two carried the vanB gene. Similar low prevalence of VRE has been reported by Gupta et al., who documented VRE rates of approximately 1–2% among clinical Enterococcus isolates.[14] In contrast, higher rates have been reported from tertiary care hospitals with prolonged antibiotic exposure and intensive care settings. The predominance of the vanA genotype in the present study is consistent with several reports where vanA-mediated resistance remains the most common mechanism worldwide.[9]

Phenotypic detection by vancomycin MIC showed resistant isolates with MIC values ranging from 32–128 µg/ml, and genotypic detection showed complete correlation with vanA/vanB gene detection. Similar concordance between phenotypic and molecular methods has been reported by Padmasini et al., demonstrating the reliability of PCR-based detection of vancomycin resistance genes.[17]

Molecular confirmation of VRE is essential because resistant Enterococci may serve as reservoirs of

transferable resistance genes capable of dissemination to other clinically important organisms. Virulence factor analysis showed hemolysin production in 28% and gelatinase production in 13.5% of isolates. Similar observations were reported by Farman et al., who documented hemolysin and gelatinase production among clinical Enterococcus isolates.[18]

These virulence factors contribute to tissue invasion, persistence, and severity of infection, highlighting the pathogenic potential of Enterococcus beyond antimicrobial resistance. The findings of this study demonstrate the increasing importance of Enterococcus as a multidrug-resistant nosocomial pathogen. Continuous surveillance of antimicrobial susceptibility, early detection of VRE, molecular characterization of resistance genes, and strict infection control practices are essential to prevent transmission and limit therapeutic challenges.

Limitations

The present study had certain limitations. The study was conducted in a single tertiary care hospital with a limited study duration; therefore, the findings may not represent the overall prevalence and resistance pattern of Enterococcus in the community or other healthcare settings. The sample size was moderate, and only selected clinical specimens were included. Molecular characterization was restricted to detection of vanA and vanB genes; other vancomycin resistance determinants and additional antimicrobial resistance genes were not investigated. Furthermore, follow-up of patients and clinical outcomes related to Enterococcus infections were not assessed. Despite these limitations, the study provides valuable information regarding species distribution, antimicrobial resistance pattern, and molecular detection of VRE, which can support infection control strategies and antimicrobial stewardship programs.

CONCLUSION

Enterococci have emerged as important multidrug-resistant nosocomial pathogens with significant clinical implications. In this study, E. faecalis was the predominant species isolated from clinical samples, followed by E. faecium. Urine was the most common source of isolation, indicating the major role of Enterococcus in urinary tract infections. High-level aminoglycoside resistance and resistance to commonly used antibiotics were observed among isolates. Vancomycin-resistant Enterococci were detected with a prevalence of 2.5%, and molecular analysis revealed the presence of both vanA and vanB genes. The high susceptibility of VRE isolates to linezolid and tigecycline highlights their importance as therapeutic options. Continuous surveillance, early detection of VRE, rational antibiotic use, and strict infection prevention measures are essential to control the emergence and spread of resistant Enterococcus strains.

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