Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.6 No. 9 (2025): September 2025 Issue
https://doi.org/10.51168/sjhrafrica.v6i9.2147
Original Article

Phenotypic and genotypic characterization of vancomycin-resistant enterococcus isolates from clinical specimens: A cross-sectional study.

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Abstract

Background

Enterococci are Gram-positive bacteria that form part of the human gut flora but have emerged as significant nosocomial pathogens. The increasing prevalence of vancomycin-resistant enterococci (VRE) poses major therapeutic and infection-control challenges. This study aimed to determine the prevalence of vancomycin resistance among clinical Enterococcus isolates and to characterize the associated phenotypic and genotypic resistance profiles.

Methodology

A cross-sectional study was conducted in the Department of Microbiology, MKCG Medical College and Hospital, Berhampur, Odisha, from October 2017 to September 2019. One hundred non-duplicate Enterococcus isolates recovered from urine, blood, pus/wound swabs, and sterile body fluids were identified based on standard biochemical tests. Antimicrobial susceptibility was performed using the Kirby–Bauer disc diffusion method as per CLSI 2019 guidelines. Vancomycin resistance was screened on VRE agar and confirmed by MIC via E-test. Genotypic detection of vanA and vanB resistance genes was performed using multiplex real-time PCR. Basic demographic variables, including age and sex, were recorded.

Results

Of the 100 isolates, 64% were *E. faecalis*, 31% were *E. faecium*, and 5% were *E. durans*. The mean age of affected patients was 38.6 years, with a female predominance (56%). The prevalence of VRE was 23%, with *E. faecium* accounting for most resistant isolates (69.6%). VRE isolates demonstrated high resistance to ampicillin (100%), ciprofloxacin (95.7%), high-level gentamicin (82.6%), and teicoplanin (78.3%), while all isolates remained susceptible to linezolid. All VRE isolates carried the vanA gene; vanB was not detected. Heteroresistance was identified in five isolates.

Conclusion

vanA-mediated VRE is prevalent in hospital settings and associated with multidrug resistance.

Recommendation

Routine surveillance, molecular detection, and strengthened antimicrobial stewardship are essential to limit VRE dissemination.

Keywords: *Vancomycin-resistant Enterococcus, vanA gene, antimicrobial resistance, hospital-acquired infection*
Submitted: *July 25, 2025* ***Accepted:*** *August 10, 2025* ***Published:*** *September 30, 2025*

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Introduction

Enterococci are facultatively anaerobic, Gram-positive cocci that are found in the genitourinary tract and gut flora of humans and other animals [1]. Enterococci, which were once thought to be weak pathogens, have been demonstrated to cause people to have serious infections when they are acquired in a hospital. They are the second most frequent source of infections that are acquired in hospitals, specifically endocarditis, bacteremia, urinary tract infections, and wound infections [2, 3]. The most concerning aspect of enterococci is their inherent resistance to numerous antibiotics, which is made

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worse by the fact that they can develop more resistance.

In the past, vancomycin, a glycopeptide antibiotic, was thought to be the final resort for treating multidrug-resistant enterococcal infections [4]. The issue is that, since the late 1980s, VRE-caused infections have grown to be a significant clinical and epidemiological concern [5]. Longer hospital stays, higher medical expenses, higher levels of morbidity, and fewer therapeutic options are all consequences of these infections [6]. Furthermore, additional Gram-positive bacteria can acquire the resistance determinants that VRE possesses. This makes it more likely that *Staphylococcus aureus* will develop vancomycin resistance [7].

The horizontal transfer and mobilization of resistance operons vanA, vanB, vanC, vanD, vanE, and vanG are among the molecular processes by which enterococci develop resistance to vancomycin [8]. Only vanA provides resistance to teicoplanin, while vanA and vanB are the most clinically significant and provide the strongest resistance to vancomycin [9]. These resistance genes reduce the binding power of vancomycin by changing the peptidoglycan precursors' terminal D-Ala-D-Ala amino acids to D-Ala-D-Lac or D-Ala-D-Ser [10]. Finding these genotypes provides important epidemiological information on the geographic distribution of resistant bacteria at different healthcare facilities and aids in the development of targeted therapies.

Vancomycin resistance is frequently screened for using automated systems and other phenotypic techniques such as broth microdilution, agar screening, disc diffusion, etc. [11]. These techniques, however, are likely to produce results that are unclear or even inaccurate, particularly for isolates with low levels of resistance. A specific challenge is heteroresistance, a phenomenon in which a subpopulation of bacterial cells within a single isolate exhibits higher resistance than the majority. Such subpopulations may survive vancomycin exposure even when the overall isolate appears susceptible in routine testing, potentially leading to treatment failure and underestimation of resistance prevalence. Heteroresistance has been particularly observed in *Enterococcus faecium*, complicating both clinical management and infection control strategies [11]. Molecular techniques, especially the polymerase chain reaction (PCR), which unquestionably verifies the existence of resistance genes, continue to be the gold standard for characterizing VRE [12]

In this context, the present study was undertaken to characterize vancomycin-resistant *Enterococcus* isolates from clinical specimens collected at MKCG Medical College and Hospital, Berhampur, during 2017–2019. The study focused on both phenotypic methods for antimicrobial resistance detection and genotypic analysis for vancomycin resistance genes, with the aim of generating data that would be valuable for clinical management and hospital infection control strategies.

Materials and Methods

Study Design

This was a hospital-based cross-sectional study.

Study Setting

The study was conducted in the Department of Microbiology, Maharaja Krishna Chandra Gajapati (MKCG) Medical College and Hospital, a tertiary-care teaching hospital located in Berhampur, Odisha, India.

Study Population and Sample Size

During study period, 5,672 patients underwent microbiological testing for clinically indicated reasons. From these, 100 non-duplicate *Enterococcus* isolates recovered from urine, pus/wo und swabs, blood, and sterile body fluids were included for detailed phenotypic and genotypic characterization. Repeat isolates from the same patient were excluded. The target sample size was pre-determined at 100 isolates, consistent with the study design and feasibility

Inclusion and Exclusion Criteria

Inclusion: Clinical specimens such as urine, pus/wound swabs, blood, and sterile body fluids yielding *Enterococcus* spp. Exclusion: Throat swabs, sputum, and feces, where *Enterococcus* is typically commensal.

Identification of *Enterococcus* spp.

Clinical specimens were cultured on MacConkey and blood agar and incubated at 37 °C for 24 to 48 hours. Suspected colonies were then identified using a standard battery of tests, including Gram staining, catalase, bile esculin hydrolysis, growth under

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varying conditions (6.5% NaCl broth, 10°C, and 45°C), and the pyrrolidonyl arylamidase (PYR) test.

Speciation of *Enterococcus*

Speciation was performed using sugar fermentation tests (raffinose, mannitol, sorbitol, arabinose, pyruvate) and the arginine deamination test.

Antimicrobial Susceptibility Testing

Antimicrobial resistance was evaluated following the CLSI guidelines, 2019, utilizing the Kirby–Bauer disk diffusion technique on Mueller–Hinton agar. The tested disks contained ampicillin (10µg), ciprofloxacin (5µg), high level gentamicin (120µg), high level streptomycin (300µg), vancomycin (30µg), teicoplanin (30µg), tetracycline (30µg), linezolid (30µg), and for urinary isolates, nitrofurantoin (300µg) was also included. Quality control utilized *E. faecalis* ATCC 29212 and ATCC 51299.

Screening for Vancomycin Resistance

All isolates were screened on VRE agar containing 6 µg/ml vancomycin (HiMedia, India). *E. faecalis* ATCC 51299 (VRE-positive control) and *E. faecalis* ATCC 29212 (VRE-negative control) were included.

Determination of Minimum Inhibitory Concentration (MIC)

The E-test was used to assess the vancomycin MIC (Ezy MIC™, HiMedia). Mueller-Hinton agar was inoculated with a 0.5 McFarland suspension, and E-test strips (0.016–256 µg/ml) were then applied. Guidelines from CLSI 2019 were followed in the interpretation of MIC values.

Genotypic Detection of Vancomycin Resistance Genes

A spectrophotometer was used to verify the purity of the genomic DNA after it was extracted using a HiMedia kit (MB505). Following that, a multiplex real-time PCR kit (HiMedia MBS PCR134) was used to amplify the vanA and vanB genes. There were 5µl of template DNA and controls in every 25µl reaction. With fluorescence detection set to FAM (vanA), HEX (vanB), and VIC/ROX (internal control), the thermal cycling process comprised initial denaturation (95 °C for 10 min), 40 cycles of denaturation (95 °C for 15 sec), and combined annealing/extension/detection (60 °C for 30 sec).

Definition of VRE

Isolates were classified as vancomycin-resistant if they grew on selective VRE agar and/or exhibited vancomycin MIC values above CLSI breakpoints.

Bias and Measures to Reduce Bias

Selection bias was minimized by including only first isolates per patient (non-duplicate isolates). Laboratory personnel performing susceptibility tests were blinded to PCR results to avoid measurement bias.

Ethical Consideration

This study was approved by the Institutional Ethics Committee of MKCG Medical College, Berhampur. Informed consent was waived as the study involved analysis of anonymized laboratory isolates without direct patient interaction.

Statistical Analysis

SPSS version 25 was used to evaluate the data once it was entered into Microsoft Excel. In the case of categorical variables, percentages and frequencies were used. When necessary, Fisher's exact test or the Chi-square test was used to compare proportions. Statistical significance was established at a p-value of less than 0.05.

Results

Of the total 5,672 clinical specimens collected from different inpatient and outpatient departments, 2,268 (39.98%) were positive for culture (Table 1). From these 100 non-duplicate *Enterococcus* isolates (4.41% from total specimens) were obtained for further phenotypic and genotypic characterization.

Table 1. Prevalence of *Enterococcus* isolates in clinical specimens

Total samples	Total number of culture positive samples	Total number of enterococcal isolates	Prevalence of enterococcal isolates
5672	2268(39.98%)	100	4.41%

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Age and Sex Distribution

The patient cohort had a slight female predominance (male:female ratio of 1:1.3; 44 males, 56 females). The highest incidence was observed in the 21-40 year age group (32%), followed closely by both the 0-20 and 41-60 year groups (29% each), with only 10% of isolates from the 61-80 year range. Overall, young and middle-aged females were the most affected group (Table 2).

Table 2. Age and sex distribution of enterococcal isolates(n=100)

Age(yrs)	Male	Female	Total	%age
0-20	12	17	29	29
21-40	9	23	32	32
41-60	18	11	29	29
61-80	5	5	10	10
Total	44	56	100	100

Species Distribution: Species identification revealed E. faecalis (64%) as the predominant isolate, followed by *E. faecium* (31%) and *E. durans* (5%). The predominance of *E. faecalis* is in line with previous studies highlighting its higher pathogenic potential (Figure 1).

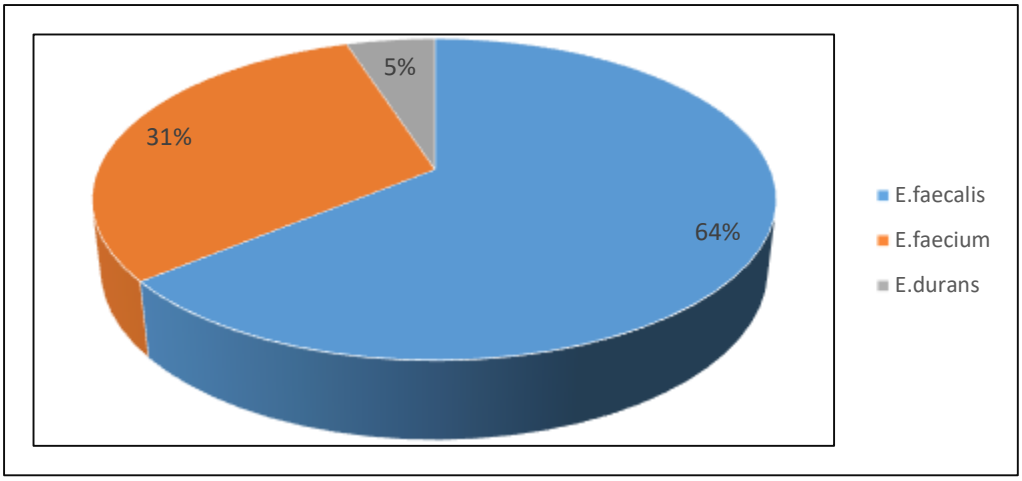

Figure1. Prevalence of enterococcal species

Clinical Units and Sample Sources

Analysis of clinical settings showed that inpatient departments contributed 86% of isolates, while only 14% were from the outpatient department. The majority were recovered from the surgical ward (58%), followed by the MICU (18%) and SNCU (14%).

With respect to specimen type, urine accounted for 61% of isolates, followed by blood (19%), pus (15%), and body fluids such as pleural and peritoneal fluids (5%). Among urine isolates, *E. faecalis* remained the most frequent species, whereas blood isolates displayed a more even distribution between *E. faecalis* and *E. faecium* (Table 3).

Table 3. Distribution of *Enterococcus* isolates across clinical specimens

Sample Type	Total Isolates	*E. faecalis* (%)	*E. faecium* (%)		*E. durans* (%)
Urine	61	38 (62.3%)	19 (31.1%)		4 (6.6%)
Blood	19	11 (57.9%)	8 (42.1%)		0
Pus	15	10 (66.7%)	4 (26.7%)		1 (6.7%)
Pleural fluid	3	3 (100%)	0		0

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Peritoneal fluid	2	2 (100%)	0		0
Total	100	64 (64%)	31 (31%)		5 (5%)

Antibiotic Resistance Patterns

The isolates showed high levels of resistance. Ciprofloxacin resistance was highest at 77%, followed by ampicillin at 66% and tetracycline at 61%. Comparatively, resistance to glycopeptides was lower for Teicoplanin (18%) and Vancomycin (23%). Crucially, every isolate maintained its Linezolid sensitivity (Table 4).

Table 4. Antimicrobial resistance among *Enterococcus* isolates

Antibiotic	**Total Resistant (%)**	***E. faecalis* (n=64)**	***E. faecium* (n=31)**	***E. durans* (n=5)**
Ampicillin (10 µg)	66 (66%)	36 (56.3%)	27 (87.1%)	3 (60%)
Ciprofloxacin (5 µg)	77 (77%)	46 (71.9%)	27 (87.1%)	4 (80%)
Tetracycline (30 µg)	61 (61%)	43 (67.2%)	15 (48.4%)	3 (60%)
Vancomycin (30 µg)	23 (23%)	6 (9.4%)	16 (51.6%)	1 (20%)
Teicoplanin (30 µg)	18 (18%)	5 (7.8%)	12 (38.7%)	1 (20%)
Linezolid (30 µg)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
HLG (120 µg)	46 (46%)	28 (43.8%)	17 (54.8%)	1 (20%)
HLS (300 µg)	40 (40%)	22 (34.4%)	16 (51.6%)	2 (40%)

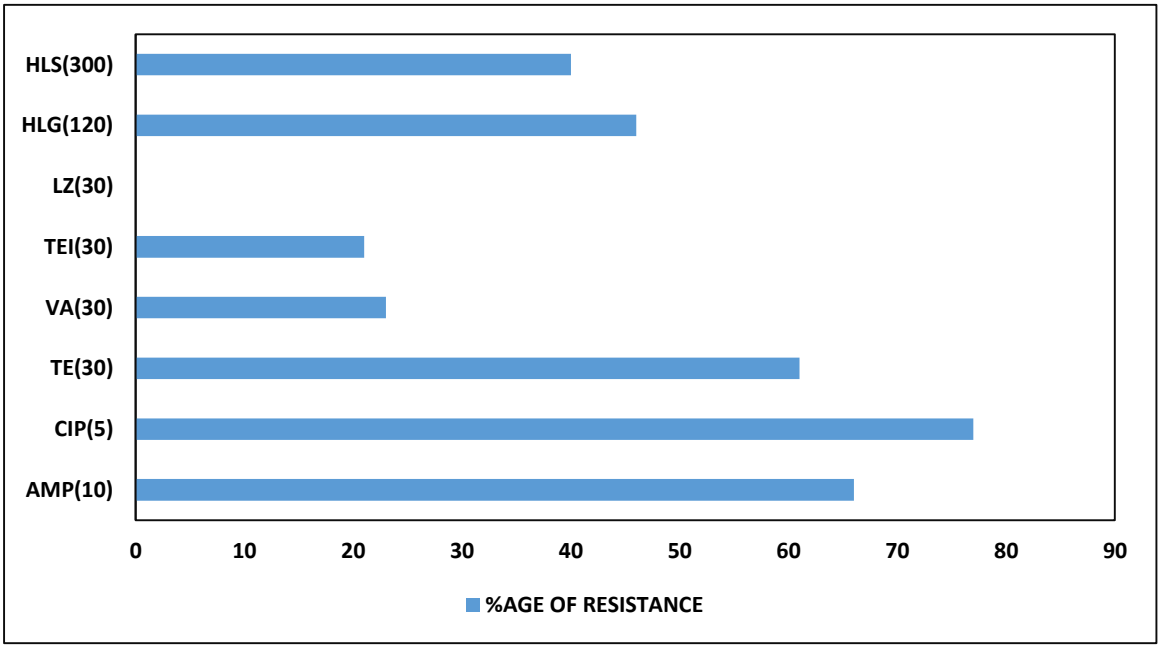

Figure 2. Antibiotic resistance among enterococcal isolates

Species-wise, *E. faecium* exhibited the highest multidrug resistance rates, significantly surpassing *E. faecalis* and *E. durans*. In urinary isolates, Nitrofurantoin resistance was recorded in 19.6%, more frequent among *E. faecium* (31.6%) compared to *E. faecalis* (15.8%).

Phenotypic Results

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Out of 100 isolates, 23% were identified as vancomycin-resistant enterococci (VRE). The majority were *E. faecium* (69.6%), followed by *E. faecalis* (26.1%) and *E. durans* (4.3%). Notably, all VRE originated from inpatient settings, particularly the MICU and surgical wards (Table 5).

Table 5. Distribution of VRE species (n=23)

Species	Number (%)
E. faecium	16 (69.6%)
E. faecalis	6 (26.1%)
E. durans	1 (4.3%)

Comparison of phenotypic detection methods (disc diffusion, vancomycin screen agar, and E-test) demonstrated complete concordance, with all three methods detecting the same 23 isolates, confirming 100% agreement (Table 6).

Table 6. Comparison of phenotypic methods for VRE detection

Method	No. of VRE detected (n=23)
Disc diffusion	23
Vancomycin screen agar	23
E-test (MIC)	23

VRE antibiotic resistance was concerning. In addition to the high resistance rates to Ciprofloxacin (95.7%), Teicoplanin (78.3%), and High-Level Gentamicin (82.6%), all isolates exhibited 100% resistance to Ampicillin. Tetracycline and high-level streptomycin resistance were observed in 30.4% and 60.9% of cases, respectively. Linezolid's function as an essential therapeutic drug was maintained because neither of the isolates displayed resistance to it (Table 7).

Table 7. Antibiotic resistance pattern of vre

Antibiotic(µg)	No.of isolates resistant	%Age (n=23)
AMP(10)	23	100
CIP(5)	22	95.7
TE(30)	7	30.4
TEI(30)	18	78.3
HLG(120)	19	82.6
HLS(300)	14	60.9
LZ(30)	0	0

The VanA phenotype, which demonstrates simultaneous resistance towards both vancomycin and teicoplanin, was expressed by 18 of the 23 VRE isolates (78.3%), according to further classification (Table 8).

Table 8. Distribution of resistance to both vancomycin and teicoplanin(vana phenotype

Species	No. resistant to VanA phenotype (%)
E. faecalis	5 (7.8%)
E. faecium	12 (38.7%)
E. durans	1 (20%)
Total	18 (18%)

Additionally, five isolates (21.7%) exhibited heteroresistance, of which the majority (80%) were *E. faecium.*

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Genotypic Results

Genotypic analysis was performed to confirm phenotypic resistance mechanisms. PCR amplification revealed that all 23 VRE carried the vanA gene, confirming the vanA genotype as the molecular determinant of glycopeptide resistance in our setting.

Amplification curves and gel electrophoresis images demonstrated clear banding for vanA in resistant isolates, with appropriate positive (*E. faecalis* ATCC 29212) and negative controls. No other vancomycin resistance genes were detected (Figure 2).

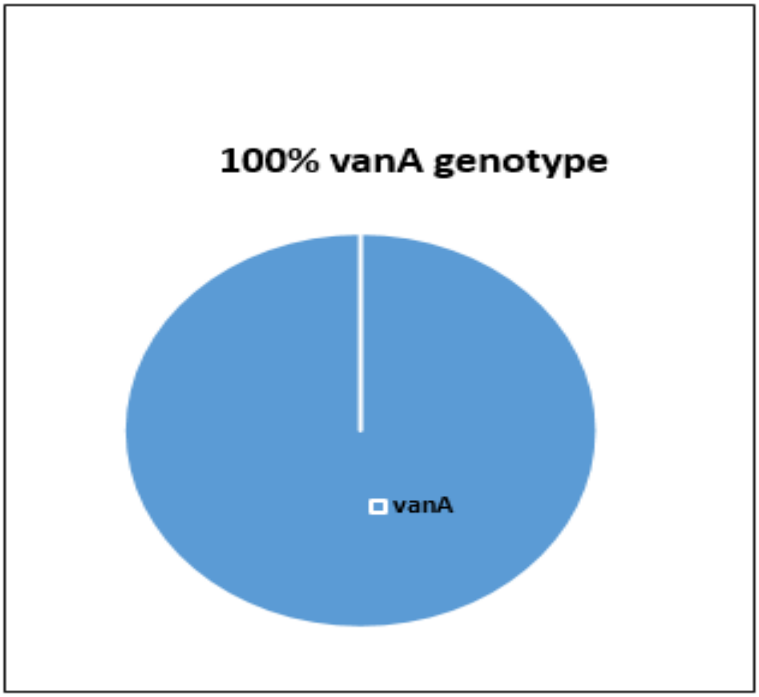

Figure 3. All the VRE were of vanA genotype.

Thus, all phenotypically resistant isolates were confirmed to be genotypically vanA-positive VRE, establishing a complete correlation between phenotypic and molecular findings.

Discussion

Vanover et al. [13] noted the growing clinical relevance of VRE with a focus on the nosocomial infections' burden of multidrug-resistant *E. faecium* and *E. faecalis*. In the study, the prevalence rate of *Enterococcus* spp. in clinical specimens was 4.41%, consistent with prior hospital-based studies, which reported rates of isolation between 3% and 7% [14,4]. Our cohort had a male to female ratio of 1:1.3, and the most common age group was 21-40, suggesting a slight predominance in younger and middle-aged women. This has been documented in the literature and explained by increased susceptibility of women, possibly due to UTI colonization having hormonal and anatomical correlates [3,15]. With a 64% predominance of *E. faecalis*, followed by *E. faecium* (31%) and *E. durans* (5%); the species distribution results show *E. faecalis* to be the most predominant. This is not surprising as the *E. faecalis* predominance is well understood due to its increased virulence and colonization capacity [2]. Interestingly, *E. faecium*, along with being less prevalent compared to *E. faecalis*, also had the greatest antimicrobial resistance, which is consistent with findings in the larger multicenter studies [16,17]. In our study, this suggests *E. faecalis* will continue to be the most common. *E. faecium*, however, is likely to be the

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most challenging to treat due to its multidrug resistance.

Most clinical sources are from inpatients, especially from the surgical wards (58%) and MICU (18%). This indicates hospital environments as an important site for VRE colonization and infection. The most frequent sample type was urine (61%), followed by blood (19%) and pus (15%). This is consistent with the literature, where enterococcal infections most frequently present as urinary tract infections [18]. Although *E. faecalis* was the most commonly isolated pathogen in urine samples, blood cultures showed higher rates of *E. faecalis* and, more significantly, *E. faecium*. This implies that *E. fecium* is a more invasive pathogen that might be disproportionately associated with serious infections of the bloodstream [19]. Susceptibility profiles showed high resistance to ciprofloxacin (77%), ampicillin (66%), and tetracycline (61%), while resistance to the glycopeptides was considerably lower with vancomycin (23%) and teicoplanin (18%). This is consistent with the literature, where enterococci remain susceptible to linezolid [20, 21]. VRE isolates are of high clinical relevance, with 100% ampicillin, 95.7% ciprofloxacin, and 78.3% teicoplanin showing resistance. This indicates limited treatment options and the importance of linezolid as an alternative.

Using disc diffusion, vancomycin screen agar, and the E-test MIC, the complete concordance demonstrated the reliability of the combined phenotypic methods in detecting VRE. Vancomycin and teicoplanin resistance were high in the VanA phenotype, which predominated (78.3%). In contrast, a subgroup (21.7%), primarily *E. faecium*, showed heteroresistance. Such findings support older literature that VanA resistance, particularly teicoplanin, is the most clinically significant and widely distributed in hospital isolates [22,23]. Testing of resistant phenotypic isolates confirmed the genotypic presence of the vanA gene, while vanB and the other van operons were absent. This completes the correlation of phenotypic and genotypic results, suggesting vanA predominated in our hospital and aligns to other reports that vanA is the principal determinant of high-level glycopeptide resistance in hospital settings [24]. The absence of vanB and vanC genes indicates limited heterogeneity of local vancomycin resistance mechanisms; however, surveillance is necessary to spot new resistance vancomycin resistance. Strict infection control procedures are essential due to the high occurrence of VRE in healthcare institutions, especially in high-risk areas like operating rooms and intensive care units [25]. Early identification of vanA-positive VRE can inform targeted therapy and prevent horizontal transmission of resistance genes to other Gram-positive pathogens, particularly *Staphylococcus aureus*, which remains a significant clinical concern [26].

Generalizability

The findings apply primarily to tertiary-care hospital settings with similar antimicrobial usage patterns and infection-control practices. However, the predominance of vanA-mediated VRE and multidrug-resistant *E. faecium* aligns with trends reported across Indian and international healthcare facilities.

Limitations

The study was limited by the single-center design and sample size. Molecular analysis included only vanA and vanB genes; other resistance determinants (vanC, vanD, vanE) were not evaluated. Clinical outcomes and treatment response were not assessed.

Data Availability

Data supporting the findings of this study can be made available by the corresponding author upon reasonable request.

Conclusion

According to the current investigation, there is a substantial burden of VRE (23% of 100 *Enterococcus* isolates, primarily *E. fecium*), which is sensitive to linezolid despite displaying widespread multidrug resistance (e.g., to ampicillin, ciprofloxacin, and teicoplanin). The vanA gene discovered in all VRE isolates confirmed high levels of vancomycin/teicoplanin resistance, and the heteroresistance presented therapeutic problems. The study highlights the use of both genotypic and phenotypic techniques for accurate VRE detection and calls for strict infection control, enhanced surveillance, and antibiotic stewardship to curb the spread of VRE.

Recommendation

In order to track vancomycin resistance and new multidrug resistance, hospitals should use ongoing

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surveillance of *Enterococcus* isolates. Strict infection control procedures are crucial, and these include isolation in high-risk locations, environmental cleaning, and hand hygiene. Vancomycin and broad-spectrum antibiotics must be used sparingly, according to antimicrobial stewardship initiatives. Rapid identification of resistant strains should be achieved using molecular detection of the vanA and vanB genes. To stop nosocomial transmission and enhance patient outcomes, it is advised that staff members receive regular training, that patients who have been colonized with VRE be identified early, and that patients receive tailored treatment based on their susceptibility profiles.

Acknowledgement

We thank the Department of Microbiology, MKCG Medical College and Hospital, Berhampur, for providing laboratory support throughout the study.

List of Abbreviations

VRE: The acronym for Vancomycin-Resistant *Enterococcus*.
MIC: Used to denote Minimum Inhibitory Concentration.
CLSI: Stands for the Clinical and Laboratory Standards Institute.
PCR: The standard abbreviation for Polymerase Chain Reaction.
HLG: Indicates High-Level Gentamicin resistance.
HLS: Signifies High-Level Streptomycin resistance.
ATCC: Refers to the American Type Culture Collection.
UTI: Commonly refers to a Urinary Tract Infection.
MICU: Identifies the Medical Intensive Care Unit.
SNCU: Represents the Special Newborn Care Unit.
AMP: The abbreviation for the antibiotic Ampicillin.
CIP: The abbreviation for the antibiotic Ciprofloxacin.
TE: The abbreviation for the antibiotic Tetracycline.
TEI: The abbreviation for the antibiotic Teicoplanin.
LZ: The abbreviation for the antibiotic Linezolid.
VRE Agar: Refers to Vancomycin-Resistant *Enterococcus* Selective Agar.

Source of funding

This research received no external funding.

Conflict of interest

The authors declare that they have no competing interests

Author contributions

Concept and study design: Author 1 & Author 2
Laboratory work and data collection: Author 1
Data analysis and interpretation: Author 1 & Author 3
Manuscript drafting: Author 1
Manuscript review and editing: All authors
All authors approved the final version of the manuscript.

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Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.6 No. 9 (2025): September 2025 Issue
https://doi.org/10.51168/sjhrafrica.v6i9.2147
Original Article

PUBLICATION DETAILS

Student's Journal of Health Research (SJHR)
(ISSN 2709-9997) Online
(ISSN 3006-1059) Print
Category: Non-Governmental & Non-profit Organization
Email: studentsjournal2020@gmail.com
WhatsApp: +256 775 434 261
Location: Scholar's Summit Nakigalala, P. O. Box 701432, Entebbe Uganda, East Africa