

Original Article

Rising trend of *Enterococcus* species as pathogen, and its antimicrobial susceptibility pattern in western Gujarat

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Abstract

Introduction: Infections by enterococci pose a unique challenge as their ability to grow in extreme environments and intrinsic resistance to cephalosporin, clindamycin, and multidrug resistance. *Enterococcus faecalis* and *Enterococcus faecium* cause infections ranging from urinary tract infections (UTI) to bacteremia.

Methodology: A retrospective study on urine and blood specimens was conducted over 11 months (February–December 2024) to assess the prevalence, age-gender distribution, species isolation, and vancomycin resistant enterococci (VRE) profile to aid treatment. A total of 4,549 urine and 4,070 blood samples were processed and identified by conventional bacteriological methods. The drug susceptibility was assessed based on the Clinical and Laboratory Standards Institute (CLSI) guidelines using the disc diffusion method. *E. faecalis* (94.05%) outnumbered *E. faecium* (5.94%).

Results: Enterococci prevalence rose to 7.08% among positive samples compared to 4.1% in 2023 and 2.70% in 2022. Females had more prevalence (69.30%) than males (30.69%); and the 21–40 years age group was the most common. Both species were most resistant to ampicillin and ciprofloxacin. High level aminoglycosides (HLA) and vancomycin resistance were 54.55% and 15.15% in *E. faecalis*, and 66.67% and 33.33% in *E. faecium*, respectively. Nitrofurantoin (69.69% sensitive) and fosfomycin (78.79% sensitive) can be good options for *E. faecalis* while formulating broad spectrum therapy for UTI. VRE isolation was 7.92%.

Conclusions: The rising trend of enterococci and the alarming rates of resistance highlight the need for rational and restricted drug use, with early detection and use of the susceptibility report to prevent treatment failures and spread of resistance.

Key words: enterococci; antimicrobial resistance; vancomycin; linezolid; HLAR; UTI.

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Introduction

Urinary tract infection (UTI) affects people of all genders and ages [1]; however, it cannot be easily treated by routine conventional antimicrobials. UTI ranks second, after respiratory tract infections, among out-patient department (OPD)-based patients [2]. When a significant count of bacteria is detected in the urine, it is a medical emergency. The prevalence rate of UTI in India range between 21.8% and 31.3% [3]. *Escherichia coli* and *Klebsiella pneumoniae* are the most common uropathogens [4].

Blood stream infections (BSI) refer to infections when microorganisms invade the blood stream. When BSI is caused by bacteria, it is often termed bacteremia, and is a serious condition. Common points of entry for bacteremia causing bacteria are genito-urinary tract (25%), respiratory tract (20%), abscess (10%), surgical site infections (SSIs) (5%), and others (5%) [5]. Gram positive bacteria are commonly associated with device/catheter associated bacteremia [5].

With the evolution of different treatment methods, more invasive procedures are being performed which have definitely helped patients, but also gave rise to infection by opportunistic pathogens or the pathogens which are known as "lab-contaminants" or normal flora. *Enterococcus* is one such pathogen, and is one of the 6 pathogens causing healthcare associated infections (HAI). These 6 pathogens are referred to as ESKAPE—*Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter*, *Pseudomonas*, *Enterobacter* [6].

Enterococcus, previously classified as Group-D *Streptococcus*, is Gram-positive, catalase negative (-ve), and arranged in pairs and short chains. It is considered as normal flora of the gastrointestinal tract, biliary tract, vagina, and male urethra. It produces smooth, gray, non-hemolytic or alfa-hemolytic colonies on blood agar, and differs from *Streptococcus* by its ability to grow at 10 °C and 45 °C, in the presence of 6.5% NaCl, at pH of 9.6, and hydrolyzing esculin in the

presence of 40% bile and pyrrolidonyl arylamidase (PYR) production [7]. More than 57 species of *Enterococcus* are known [5] and *E. faecalis* and *E. faecium* are the two most promising pathogens known to humankind.

The zonal council regions of India showed the prevalence of vancomycin resistant *Enterococcus* (VRE), and it was highest in north-east (24.7%), north (16.3%), western (10.1%), central (9.2%), and eastern (9.0%) India. The lowest prevalence was in south India (2.6%) [8]. Based on these data, Gujarat, in the western part of India, was at significant risk of VRE.

Among the different virulence factors, enterococcal surface protein, gelatinase, and pillin gene clusters are implicated in biofilm formation [1]. Cytolysin and hemolysin are lethal for red blood cells (RBCs), polymorphs and macrophages [7]. Aggregation substance proteins cause clumping of organisms. Extracellular surface proteins have a role in UTI and endocarditis. Hyaluronidase and Tsp F are other factors involved [1,7].

Enterococcus causes a wide variety of infections from UTI to bacteremia. *E. faecalis* is often associated with community and *E. faecium* is with HAIs [9]. The most common type of infection caused by *Enterococcus* is UTI. Lower UTI is frequently seen in older males, whereas it is an uncommon cause of uncomplicated cystitis in young females. Upper UTI can lead to bacteremia and is often seen in older males [10]. Enterococci are responsible for 10% of all UTI cases [11].

The presence of *Enterococcus* in urine is asymptomatic and not treated, except in pregnant females, patients undergoing urosurgery, or when comorbidity is present. Many catheterized patients are colonized and just cath-removal is necessary, not the treatment [7]. Enterococcal UTI is likely to be acquired by hospitalization or intensive care unit (ICU) admission, and is often resistant to antibiotics. 15% of health-care associated UTIs are due to enterococci present in ICUs [10]. Community acquired UTIs by this species are also becoming important due to anatomical factors, prior instrumentation, and selection pressure of broad-spectrum antibiotics. VRE is now being isolated from both community and hospital-acquired UTIs.

The most common manifestations due to enterococci are bacteremia and endocarditis [10]. The source of these enterococci can be the inserted intravenous (IV)-line, genitourinary tract, abdominal infections, or biliary tract infections. The risk factors are old age, low immunity, prematurity, diabetes, and instrumentations. Intra-abdominal, soft tissue

infections are generally mixed polymicrobial and enterococci can be one among the different organisms present. Early onset of neonatal sepsis acquired by vaginal delivery and prematurity is also possible. However, meningitis is rare [7].

The mode of transmission from one patient to another is often by the hands of healthcare workers (HCW). The bacteria persist for as long as 60 minutes on hands, and for up to 4 months on surfaces which serve as reservoir for transmission of infection [10]. HCWs with the bacteria on their hands become carriers and can spread the infection while inserting catheter or IV cannula. This leads to direct inoculation of the pathogen. Acquired strains of enterococci carry antibiotic resistance genes and can be present for a long time in the gastrointestinal tract as a result of selective pressure from broad spectrum antibiotic therapy used in indoor patients [10,12,13]. The hospital environment is often associated with multi-drug resistant (MDR) enterococci. The thermometer and its handle seem to be commonly involved in VRE transmission [10,14]. Medical devices such as blood pressure (BP) cuffs, IV fluid pumps, stethoscopes, bedpans, and linen gowns play a significant role in transmission.

The hosts' risk factors for acquiring infections are prior antibiotic therapy with vancomycin and third generation cephalosporins, prior surgical exposure, and catheterization. These are also responsible for high level aminoglycoside resistant enterococci [HLAR]. Other risk factors are longer stay in hospital, exposure to VRE colonizer patient, chronic hemodialysis organ transplant, and malignancies [10].

The treatment options for this notorious organism are limited because of its intrinsic resistance to cephalosporins, clindamycin, co-trimoxazole, and aminoglycosides [except for high level]. The drugs which act by inhibiting cell wall synthesis and are bactericidal for other Gram-positive bacteria are considered static for enterococci [6,10]. Acquired resistance towards macrolides, vancomycin, tetracyclines, linezolid, and chloramphenicol does exist [6].

Hence, this study focused on enterococci, along with its prevalence, age and gender distribution, species differentiation, and VRE profile. There is limited data available on in-vitro susceptibility and other parameters for this notorious pathogen. Therefore, this study highlighted treatment plans, infection prevention, and control practices to prevent further spread and guide clinicians in the future.

Methodology

This retrospective study on urine and blood specimens was conducted over a period of 11 months from February 2024 to December 2024. A total of 4,549 urine and 4,070 blood samples were collected from patients of all ages and both genders who visited the outpatient and inpatient departments of the hospital and were diagnosed tentatively as UTI and BSI.

All specimens except blood and urine were excluded from the study. All organisms except *Enterococcus* and duplicates were also excluded. The specimens were processed according to standard bacteriological methods and identified by standard conventional methods. The samples were inoculated and incubated aerobically at 37 °C for 24 to 48 hours. Then, the isolates were primarily identified as *Enterococcus* spp. based on the colony morphology. Further identification was done by the bile esculin hydrolysis test. Potassium tellurite agar (PTA), HiCrome differential agar, and arabinose agar (HiMedia Laboratories Pvt Ltd., Mumbai, India) were used to differentiate *E. faecalis* from *E. faecium*. *E. faecalis* produces black colonies by tellurite reduction

on PTA [15] (Figure 1), blue colonies on HiCrome agar, and pink colonies on arabinose agar. *E. faecium* produces green colonies and yellow colonies on HiCrome agar and arabinose agar respectively (Figure 2).

Determination of antimicrobial susceptibility

All the isolates were analyzed with the modified Kirby-Bauer disc diffusion methods using standard techniques and measurement of zone of inhibition based on CLSI M100 34th edition guidelines [16] to determine susceptibility.

The antibiotic discs used included ampicillin (10 µg), ciprofloxacin and levofloxacin (5 µg), high level gentamicin (120 µg), high level streptomycin (300 µg), tetracycline (30 µg), nitrofurantoin (300 µg), fosfomycin (200 µg), linezolid (30 µg), and vancomycin (30 µg). The control strains used were *E. faecalis* ATCC 29212 and *S. aureus* ATCC 25923. The synergy between high level aminoglycosides (HLA) and ampicillin was assessed on all isolates. Resistance to vancomycin was noted on the basis of zone of inhibition of vancomycin < 14 mm [16].

Statistical analysis

Descriptive statistics were used to calculate frequencies and percentages. The Chi square test of goodness of fit was used to assess the significance of age group distribution among enterococcal isolates across 5 age categories (0–20, 21–40, 41–60, 61–80, and 81–100 years). Gender distribution among positive urinary isolates was analyzed using the Chi square test. All statistical analyses were conducted with a significance level set at *p* < 0.05, and the results were considered statistically significant when the *p* value was less than 0.05.

Figure 1A. Growth of *Enterococcus faecalis* on bile esculin agar. **B.** Growth of *E. faecalis* on potassium tellurite agar.

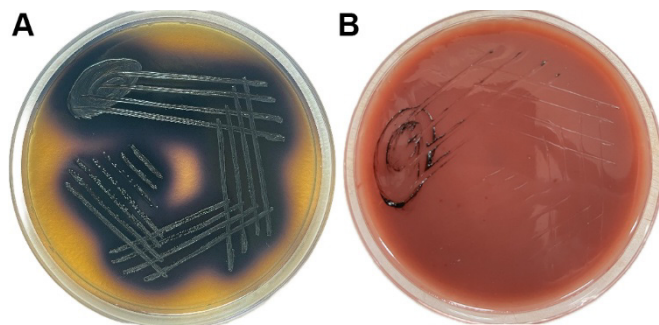


Figure 2A. Growth of *Enterococcus faecalis* (blue) and *Enterococcus faecium* (green) on HiCrome differential agar; **B.** Growth of *E. faecalis* (pink) and *E. faecium* (yellow) on arabinose agar.

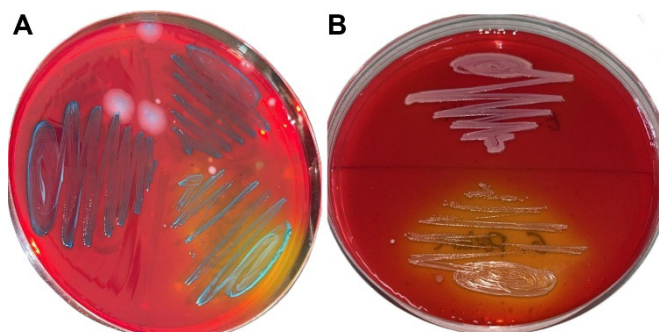


Figure 3. Year wise prevalence rate of Enterococci among culture positives at the hospital.

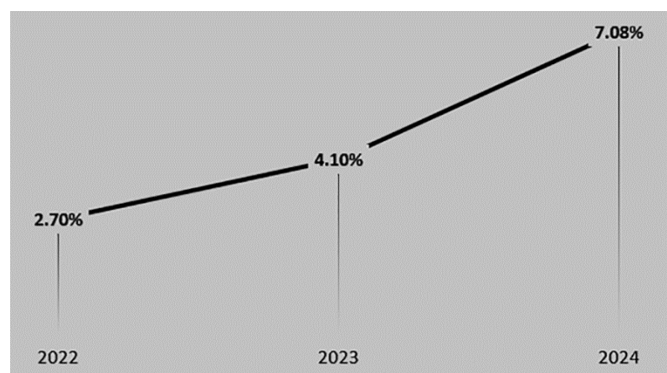


Table 1. Prevalence of *Enterococcus* species among urine and blood culture samples.

Specimen type	<i>E. faecalis</i>	<i>E. faecium</i>	Total
Urine	93 (94.89%)	5 (5.10%)	98
Blood	2 (66.67%)	1 (33.33%)	3
Total	95 (94.05%)	6 (5.94%)	101

Results

The prevalence of enterococci among culture positive samples was 2.7% in 2022, rose to 4.1% in 2023, and at the time of study in 2024 it was 7.08% (Figure 3).

A total of 101 isolates of *Enterococcus* spp. was studied over a period of 11 months. Among these, 98 isolates were found among 4,549 urine samples (2.15% isolation rate among all urine samples received at laboratory). Of the 4,549 urine samples, 1,257 were culture positive, and the rest were sterile. Thus, culture positivity rate among urine samples was 27.63%. *Enterococcus* spp. was identified in 7.79% culture positive UTIs (98 isolates among 1,257 culture positives). Among the 98 isolates from the urine samples, 93 were identified as *E. faecalis* (94.89%), whereas 5 were *E. faecium* (5.10%). The blood culture positivity rate was 4.12% (168 positive by culture/4070 total blood culture samples). *Enterococcus* spp. prevalence was 1.78% (3/168) among positive blood culture samples, of which 2 were *E. faecalis* (66.67%) and 1 was *E. faecium* (33.33%) (Table 1).

Among the 101 isolates, 70 were obtained from female patients (69.30%), and 31 from male patients

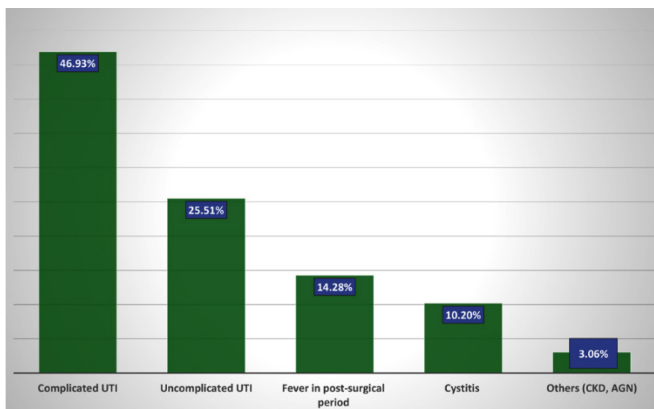
(30.69%). The *p* value for gender distribution was 0.0001, which was statistically significant.

The highest number of isolates were obtained from patients in the 21–40 years age group; and 47.52% of the isolates were obtained from this reproductive age group (Table 2). The second most common age group affected was 41–60 years (26.73%), followed by 0–20 years (14.85%), 61–80 years (8.91%), and 81–100 years (1.98%). The *p* value for age distribution was statistically significant (< 0.0001).

The diagnosis of the patients with positive urinary isolates was also recorded and it was noted that the most common complaint was fever associated with burning micturition (complicated UTI, 46.93%), whereas 25 were diagnosed as uncomplicated UTI (25.51%). Cystitis was present in 10 patients (10.20%) and fever in the post-surgical period was present in 14 (14.28%). Diagnosis of chronic kidney disease (CKD), acute glomerulonephritis (AGN), and other conditions were found in 3 isolates (3.06%; Figure 4).

All 3 isolates of BSI had instrumentation history and were admitted in the ICU. Out of them, 2 were female and 1 was male. Serious complications like shock and sepsis were found in 2 isolates.

Figure 4. Diagnosis of the positive urinary isolates.



CKD: Chronic Kidney Disease; AGN: Acute Glomerulonephritis.

Figure 5. Distribution of *Enterococcus* among different wards.

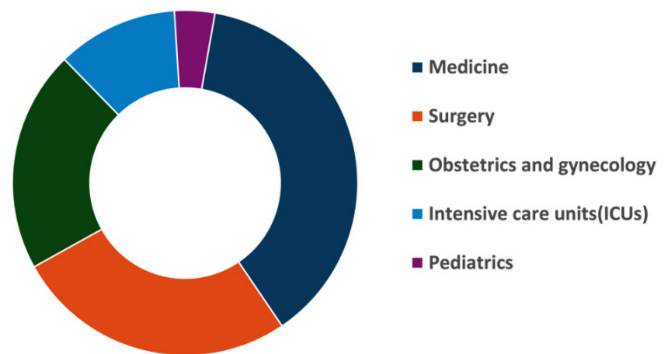
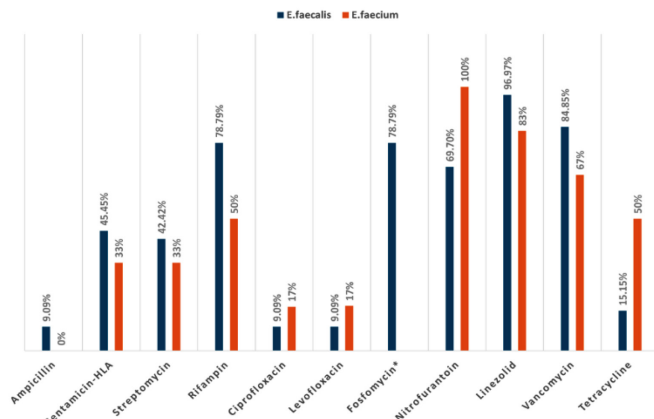


Table 2. Age group distribution among enterococcal isolates.

Age group (in years) affected	Number of enterococcal isolates belonged to respective age group (%) out of total 101 isolates	<i>p</i> -value
0–20 Years	15 (14.85 %)	<i>p</i> value: < 0.0001 (statistically significant)
21–40 Years	48 (47.52 %)	
41–60 Years	27 (26.73 %)	
61–80 Years	9 (8.91 %)	
81–100 Years	2 (1.98 %)	

Figure 6. Antimicrobial susceptibility pattern of *Enterococcus faecalis* and *Enterococcus faecium*.



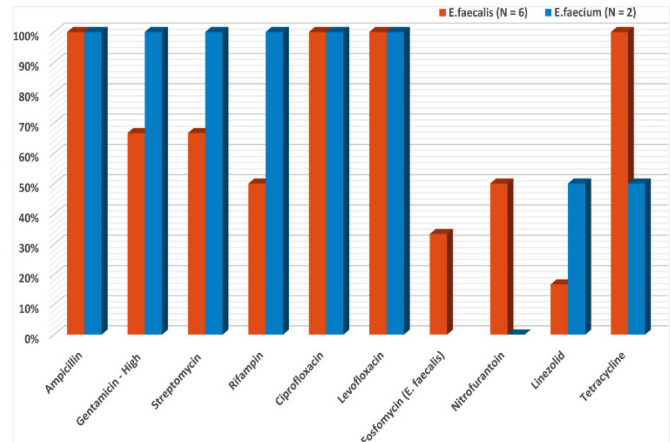
*Fosfomycin tested for *E. faecalis* only [16].

The distribution among the different wards of the tertiary care facility was also assessed and revealed that the maximum number of isolates were from medicine units (38.45%), followed by the surgical ward (26.92%), the different ICUs (11.53%), obstetrics and gynecology (21.15%), and the pediatric wards (3.84%) (Figure 5).

Antimicrobial susceptibility was assessed separately for *E. faecalis*, *E. faecium*, and VRE (Figure 6). *E. faecalis* was most resistant to ampicillin (90.9% resistance rate), ciprofloxacin and levofloxacin (90.9%), and tetracycline (84.84%). Vancomycin resistance was noted as 15.15%. The least resistance was found for linezolid (3.03%). In the case of urinary isolates, there was 78.79% sensitivity to fosfomycin, and 69.70% sensitivity to nitrofurantoin. HLAR was found for gentamicin (54.55%). Rifampin sensitivity was 78.79%.

E. faecium showed 33.33% resistance to vancomycin and 16.67% resistance to linezolid. There was no sensitivity towards ampicillin, and least sensitivity towards fluoroquinolones (17%) and tetracycline (50%). HLAR was found for gentamicin (66.67%). Surprisingly, all urinary isolates were susceptible to nitrofurantoin.

Figure 7. Resistance % among vancomycin resistant enterococci (VRE) isolates (N = 8).



Vancomycin resistant enterococci (VRE)

The VRE isolates were studied in detail. Among the 101 isolates, 8 were found to be VRE (7.92% VRE isolation rate). 75% were from females and 25% from males, 50% belonged to patients in the 21–40 years age group and 50% to patients in the 61–80 years age group.

Among the 8 isolates, 6 were identified as VR-*E. faecalis* and 2 as VR-*E. faecium*. Both species were 100% resistant to ampicillin and levofloxacin. In VR-*E. faecalis*, HLAR (HLA resistance) was 66.67%, and in VR-*E. faecium* it was 100%. The VR-*E. faecalis* isolates were the least resistant to linezolid (16.67%), followed by fosfomycin (33.33%), and nitrofurantoin (50%). Nitrofurantoin susceptibility was 100% in both VR and VS-*E. faecium* whereas linezolid was susceptible only in 50% isolates of VR-*E. faecium*. The overall resistance to all antibiotics was more in VRE than vancomycin sensitive enterococci (VSE), including HLAR (Figure 7).

Discussion

The increasing presence of enterococci among culture positive samples at the study institute, with the current prevalence of 7.08%, is quite alarming. Smout et al. conducted a meta-analysis on data available from India and found that VRE prevalence was 4.8% between 2000 and 2010, and upsurged to 14.1%

Table 3. Comparative analysis of enterococcal isolates: prevalence, species distribution, and demographics across studies (2020–2024).

Study	Year of Study	Specimen from which highest number of <i>Enterococci</i> were isolated	Prevalence rate of Enterococcal spp. among culture positives	Prevalence rate of all enterococcal species		Gender affected	Common age group
				<i>E. faecalis</i>	<i>E. faecium</i>		
Present Study	2024	Urine	7.08%	94.06%	5.94%	F > M	21–40
Darji et al. [18]	2024	Urine	11.6%	72.7%	27.3%	M > Fe	31–40
Ohri et al. [19]	2023	Urine	6.62%	60%	40%	–	–
Iancu et al. [6]	2023	Urine	0.86%	47.7%	47.7%	–	–
Sikdar et al. [17]	2021	Urine	–	84.05%	15.95%	M > F	> 60
Varghese et al. [15]	2020	Pus	–	66.7%	21.4%	M > F	40–60

between 2011 and 2021 [8]. Therefore, it is necessary to understand the trend, identify the vulnerable population, and determine the effectiveness of antibiotics (Table 3).

In this study, enterococci were isolated maximally from urine samples, indicating that the most common type of infection associated with it is UTI, as was also reported in other studies. [6,17–21]. However, the study by Varghese *et al.* reported that the isolation rate of enterococci from pus samples was twice that of urine samples [15].

Enterococci were the main Gram-positive cocci isolated from UTIs. There were 101 isolates of enterococci among the 1,425 culture-positive blood and urine samples included in this study, denoting a prevalence of 7.08%. The culture positivity rate of enterococci was reported to be 0.86%, 11.6%, and 6.62% in other studies [6,18,19].

Enterococci were isolated from only 2.97% (3/101) blood specimen. Other studies have reported higher rates of isolation: 14.72% by Sikdar *et al.* [17], 11.52% by Ohri *et al.* [19], and 16.4% by Darji and Katwala [18]. However, Yadav and Agrawal [20] reported that only 1.3% *Enterococcus* were isolated from blood specimens, which is similar to the present study. The reason for this low rate is that higher numbers of blood cultures were sterile and from the neonatal and pediatric populations.

E. faecalis outnumbered *E. faecium* in all previous studies, and therefore *E. faecalis* is more common [6,11,15,17–21].

Females of reproductive age group (21–40 years) had a statistically significant higher infection rate. Possible reasons for this are close proximity of the urethra and anus, sexual activity, and hygiene; as also reported in other studies [11,20,21]. Some studies have shown that males are more affected than females; however, the difference between the two genders was quite less [15,17,18]. The middle to old age group (41–60 years) had the second highest infection rate. This group often had comorbid conditions and prior surgical history.

Enterococci were mostly isolated from urine samples of patients with diagnosis of complicated UTI,

commonly associated with fever (46.15%); followed by diagnosis of uncomplicated UTI (26.92%), denoting pyrogenic tendency and virulence of the organism. In the case of BSI patients, shock, sepsis, instrumentation history, and association with ICU was present in the case of all isolates.

The isolation rate was the highest from the medicine unit (19.80%), followed by surgical wards (13.86%), gynecology (10.89%), and ICUs (5.94%). Pediatrics had the lowest infection rate (1.98%). A similar ward-wise distribution was also reported by Nisarta *et al.* [21]. This difference in ward distribution can be explained by the fact that a greater number of females with complaints of UTI are presented to clinicians of the medicine department and diagnosed as UTI. In addition, invasive procedures and usage of instruments are more frequent in surgical and gynecology wards.

Since this coccus is intrinsically resistant to commonly used broad spectrum drugs, only few drugs are available for treatment. Therefore, the role of the clinical microbiology lab is to identify *Enterococci* at species level, determine susceptible and resistant cocci, establish pathogenicity, and guide the selection of proper therapy and infection control practices. Routine testing of HLAR is needed in the case of blood and body fluids isolates because of increase in resistance [10].

A comparison of the resistance to different antimicrobial agents reported in different studies is provided in Table 4.

E. faecalis

E. faecalis was most sensitive to linezolid (96.96%), followed by vancomycin. The two most promising drugs—nitrofurantoin (69.69% sensitive) and fosfomycin (tested for *E. faecalis* only in accordance to CLSI) [16] (78.78% sensitive)—can be considered as therapeutic options while formulating broad spectrum antibiotics for UTI. Although nitrofurantoin is more sensitive compared to other drugs, it has effect only against uncomplicated UTI [22] and its resistance was less compared with other drugs in previous studies ranging from 5.84–35.13% [11,15,17,19]. The advantage of fosfomycin over nitrofurantoin is that being cidal it can act by

Table 4. Comparison of resistance to antimicrobial agents reported in different studies.

Studies	<i>E. faecalis</i> resistance (%)					<i>E. faecium</i> resistance (%)				
	HLAR	NIT	CIP	VAN	LZ	HLAR	NIT	CIP	VAN	LZ
Present Study-2024	54.55	30.3	90.90	15.15	3.03	66.67	0	83.33	33.33	16.67
Ohri <i>et al.</i> [19]-2023	51.42	0	71.07	2.94	0	71.90	23.21	85.54	29.89	0
Sikdar <i>et al.</i> [17]-2021	58.39	5.84	76.64	3.28	0.36	34.62	7.69	61.54	15.38	5.77
Varghese <i>et al.</i> [15]-2020	22	10	28	4	0	43.8	25	56.2	6.2	0
Singh <i>et al.</i> [11]-2019	54.05	35.13	72.52	8.55	1.8	85.18	77.77	90.74	27.77	5.55

HLAR: high level aminoglycoside-Gentamicin; NIT: nitrofurantoin; CIP: ciprofloxacin; VAN: vancomycin; LZ: linezolid.

inactivating the Mur A enzyme and inhibiting cell wall synthesis. However, the spectrum of activity of nitrofurantoin is against both *E. faecalis* and *E. faecium*, whereas fosfomycin has been tested by the CLSI [16] only for *E. faecalis* and observed to have sensitivity of 78.79% (more than nitrofurantoin) in the present study. Fosfomycin can also be used for complicated UTI, prostatitis, and pyelonephritis. The present study is the only study that describes the in-vitro susceptibility of fosfomycin against *E. faecalis* and rifampin.

Aminoglycosides combined with a cell wall active agent can show synergy and such a combination can be given to the patient. The resistance to gentamicin was 54.55% in HLAR testing and other studies were similar to the present study (Table 4) [11,15,17,19].

The inference of testing HLAR is: [a] if isolate is susceptible to HLA only and not to ampicillin, combination of vancomycin + gentamicin can be given. [b] If only ampicillin is susceptible and not HLA, synergy is absent and necessitates combination of ampicillin + ceftriaxone (max. dose). [c] If all the agents like ampicillin and vancomycin are resistant but HLA is sensitive, daptomycin should be combined with HLA [22].

The most resistant antibiotics are ampicillin (36 to 64.6% resistance) and ciprofloxacin (61.54 to 85.54% resistance) [11,17–19]. Vancomycin resistance has increased over time, ranging from 4% to 32% in different studies [11,17–20] and 15.15% observed in this study. Linezolid showed the least resistance and should be reserved (Table 4).

E. faecium

E. faecium isolates were more resistant than *E. faecalis* in previous studies and in the present study because of the biofilm formed with carriage of antibiotic-resistant genes [1]. The trend of *E. faecium* isolation requires throughout the investigation of each isolate, history of patient, and hospital environment surveillance.

The highest resistance was noted towards ampicillin (100% in this study; 53–76% in other studies

[11,17,19]), followed by ciprofloxacin (range of 56–90%, Table 4). HLAR to gentamicin was more than *E. faecalis*; a range of 35 to 85% was noted in different studies. Fortunately, in the present study, all isolates were susceptible to nitrofurantoin; however, resistance was found towards it in different studies (Table 4). Vancomycin resistance increased to 33.33% in this study; consistent with a higher tendency of becoming VRE. Only 1 (out of 6) isolate was found to be linezolid resistant. Rifampin resistance was 50% but it should not be used as monotherapy.

VRE

The presence of VRE is a cause of concern in any healthcare facility because only few antibiotics are effective against it. This study identified 7.92% VRE isolation which falls in the range of 4%–13.72% (Table 5) [6,15,17–20]. The antimicrobial profile for VRE was different from that of VSE. VRE was 100% resistant to ampicillin, ciprofloxacin and tetracycline. The least resistance was towards fosfomycin, nitrofurantoin (judicious use in case of VRE in UTIs is recommended), rifampin, and linezolid. Linezolid and daptomycin were the most effective drugs in other studies also, but they should be used with caution.

Prevention and control measures for VRE and VSE are important because of their rapid spread and rising trends of infection. Establishment of a hospital infection control policy and committee with implementation of an antibiotic stewardship program is recommended. The interventions that can be taken by the antibiotic stewardship program are:

1. Hand hygiene, including decontaminating the hands of HCWs before and after contact with patients. Hospital environmental contamination can be prevented by using standard disinfecting agents for cleaning of high touch/frequently touched surfaces such as tables, toilets, handles, lifts etc.
2. Use of barrier precautions (gloves and mask) and sterile gowns when attending VRE-infected patients, and hand washing practices.
3. Periodic surveillance of culture of patients at high risk of carriage
4. Cleaning of rooms used by VRE patients to control further spread.
5. Medical equipment should not be shared among patients.
6. Daily bathing of patient (pre- and post-operative) by chlorohexidine soap has shown to decrease the chance of acquiring VRE by 50% [10,23].
7. Active surveillance of asymptomatic patients for VRE colonization should be implemented. The goal

Table 5. Prevalence of vancomycin resistant enterococci (VRE) in various studies.

Study	Year Of Study	Prevalence (%)
Present Study	2024	7.92%
Darji et al. [18]	2024	3.63%
Ohri et al. [19]	2023	13.72%
Iancu et al.[6]	2023	9.56%
Sikdar et al. [17]	2021	5.21%
Varghese et al. [15]	2020	4%
Yadav et al. [20]	2020	9.6%

is to identify every single colonized patient and keep them in contact isolation. Periodic culture should be taken for surveillance, and 3 consecutive negative rectal swabs at least 1 week apart indicates clearance of VRE [10,24,25].

8. The use of vancomycin and linezolid should be restricted. These antibiotics should not be used with intrinsic resistance like cephalosporin for treating enterococcal infection. The correct dose and correct duration should be administered to decrease antibiotic pressure.
9. Use of fluoroquinolones as broad -spectrum antibiotic should be avoided as there are high chances of clinical failure. Instead, fosfomycin and nitrofurans can be used, depending on sensitivity.

Conclusions

Given the rising trend in VREs, especially in the western part of India, it is crucial that microbiology laboratories are aware and vigilant regarding isolation of these bacteria from cultures. Every case of *Enterococcus* isolation should be followed up by a study of its antibiotic susceptibility pattern. The patients and their contacts should be screened for VRE. In addition, the surveillance of the hospital environment and ICUs should be implemented. HCWs hygiene practices should be improved, along with strict implementation of an antibiotic stewardship program. Nitrofurans and fosfomycin should be used as treatment options for UTIs, instead of using only fluoroquinolones, which is currently used widely as a broad-spectrum antibiotic. *Enterococcus* is a current and future threat and should not be ignored as normal flora.

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Conflict of interest

No conflict of interest is declared.

References

1. Codelia-Anjum A, Lerner LB, Elterman D, Zorn KC, Bhojani N, Chughtai B (2023) Enterococcal urinary tract infections: a review of the pathogenicity, epidemiology, and treatment. *Antibiotics* 12: 778. doi: 10.3390/antibiotics12040778.
2. Hareendranath G (2021) Fosfomycin susceptibility in multidrug resistant urinary *Escherichia coli* isolates. *J Evol Med Dent Sci* 10: 414–418. doi: 10.14260/jemds/2021/92.
3. Prakash D, Saxena RS (2013) Distribution and antimicrobial susceptibility pattern of bacterial pathogens causing urinary tract infection in urban community of Meerut City, India. *ISRN Microbiol* 2013: 1–13. doi: 10.1155/2013/749629.
4. Tulara N (2018) Nitrofurantoin and fosfomycin for extended spectrum beta-lactamases producing *Escherichia coli* and *Klebsiella pneumoniae*. *J Glob Infect Dis* 10: 19. doi: 10.4103/jgid.jgid_72_17.
5. Tille PM (2022) Bailey & Scott's diagnostic microbiology. 15th edition. Elsevier, St. Louis, Missouri.
6. Iancu A-V, Arbune M, Zaharia E-A, Tutunaru D, Maftai N-M, Peptine L-D, Ţocu G, Gurău G (2023) Prevalence and antibiotic resistance of *Enterococcus* spp.: a retrospective study in hospitals of southeast Romania. *Appl Sci* 13: 3866. doi: 10.3390/app13063866.
7. Procop GW, Church DL, Hall GS, Janda WM (2020) Koneman's color atlas and textbook of diagnostic microbiology, 7th edition. Jones & Bartlett Learning.
8. Smout E, Palanisamy N, Valappil SP (2023) Prevalence of vancomycin-resistant enterococci in India between 2000 and 2022: a systematic review and meta-analysis. *Antimicrob Resist Infect Control* 12: 79. doi: 10.1186/s13756-023-01287-z.
9. Hoge CW, Adams J, Buchanan B, Sears SD (1991) Enterococcal bacteremia: to treat or not to treat, a reappraisal. *Clin Infect Dis* 13: 600–605. doi: 10.1093/clinids/13.4.600.
10. Gilmore MS, Clewell DB, Ike Y, Shankar N (2014) Enterococci: from commensals to leading causes of drug resistant infection. *Massachusetts Eye and Ear Infirmary, Boston*.
11. Singh Naruka H, Chand AE, Meena H (2019) Prevalence of various *Enterococcus* species and their antibiotic resistance pattern among urinary isolates in tertiary care center in South Eastern Rajasthan. *IP Int J Med Microbiol Trop Dis* 5: 18–22. doi: 10.18231/2581-4761.2019.0005.
12. Donskey CJ, Chowdhry TK, Hecker MT, Høyen CK, Hanrahan JA, Hujer AM, Hutton-Thomas RA, Whalen CC, Bonomo RA, Rice LB (2000) Effect of antibiotic therapy on the density of vancomycin-resistant enterococci in the stool of colonized patients. *N Engl J Med* 343: 1925–1932. doi: 10.1056/NEJM200012283432604.
13. Ubeda C, Taur Y, Jenq RR, Equinda MJ, Son T, Samstein M, Viale A, Succi ND, Van Den Brink MRM, Kamboj M, Pamer EG (2010) Vancomycin-resistant *Enterococcus* domination of intestinal microbiota is enabled by antibiotic treatment in mice and precedes bloodstream invasion in humans. *J Clin Invest* 120: 4332–4341. doi: 10.1172/JCI43918.
14. Livornese LL, Dias S, Samel C, Romanowski B, Taylor S, May P, Pitsakis P, Woods G, Kaye D, Levison ME, Johnson CC (1992) Hospital-acquired infection with vancomycin-resistant *Enterococcus faecium* transmitted by electronic thermometers. *Ann Intern Med* 117: 112–116. doi: 10.7326/0003-4819-117-2-112.
15. Varghese V, Menon AR, Nair KP (2020) Speciation and susceptibility pattern of enterococcal species with special

- reference to high level gentamicin and vancomycin. *J Clin Diagn Res* 14: DC01–DC06. doi: 10.7860/JCDR/2020/44014.13708.
16. Lewis JS (2024) Performance standards for antimicrobial susceptibility testing, Thirty-fourth edition. Clinical and Laboratory Standards Institute.
 17. Sikdar S, Sadhukhan S, Majumdar AK, Bhunia S, Sarkar S, Bhattacharjee SG (2021) Phenotypic characterisation, virulence determination and antimicrobial resistance pattern of *Enterococcus* species isolated from clinical specimen in a tertiary care hospital in Kolkata. *J Clin Diagn Res* 15: DC06–DC09. doi: 10.7860/JCDR/2021/48616.15077.
 18. Darji SM, Katwala JR (2024) Prevalence and antimicrobial susceptibility pattern of *Enterococcus* species isolated in clinical samples from a tertiary care centre. *J Popul Ther Clin Pharmacol* 31: 3445–3453. doi: 10.53555/jptcp.v31i6.6903.
 19. Ohri S, Singh K, Sidhu SK, Oberoi L (2023) Prevalence and antimicrobial resistance in *Enterococcus* species. *Asian J Pharm Clin Res*. 16: 30–33. doi: 10.22159/ajpcr.2023.v16i6.47283.
 20. Yadav RK, Agarwal L (2022) Enterococcal infections in a tertiary care hospital, north India. *Ann Afr Med* 21: 193–197. doi: 10.4103/aam.aam_110_20.
 21. Nisarta A (2016) Study of antimicrobial resistance in enterococci at Government Medical College, Bhavnagar, Gujarat, India. *Int J Curr Microbiol Appl Sci* 5: 826–836. doi: 10.20546/ijcmas.2016.509.093.
 22. Sastry AS, Priyadarshi K, Sarumathi D (2023) Essentials of antimicrobial stewardship. New Delhi: Jaypee Brothers Medical Publishers.
 23. Climo MW, Sepkowitz KA, Zuccotti G, Fraser VJ, Warren DK, Perl TM, Speck K, Jernigan JA, Robles JR, Wong ES (2009) The effect of daily bathing with chlorhexidine on the acquisition of methicillin-resistant *Staphylococcus aureus*, vancomycin-resistant *Enterococcus*, and healthcare-associated bloodstream infections: results of a quasi-experimental multicenter trial. *Crit Care Med* 37: 1858–1865. doi: 10.1097/CCM.0b013e31819ffe6d.
 24. Boyce JM, Potter-Bynoe G, Chenevert C, King T (1997) Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 18: 622–627. doi: 10.1086/502213.
 25. Hospital Infection Control Practices Advisory Committee (HICPAC) (1995) Recommendations for preventing the spread of vancomycin resistance. *Infect Control Hosp Epidemiol* 16: 105–113. doi: 10.1086/647066.