Enterococci have emerged as important nosocomial pathogens. They are the second most common organisms recovered from nosocomial urinary tract and wound infections and the third most common cause of nosocomial bacteremia. These organisms have been able to survive in the hospital environment because of their intrinsic resistance to several commonly used antibiotics and their ability to acquire resistance to currently available antibiotics, either by mutation or by receipt of foreign genetic material through the transfer of plasmids and transposons. Bactericidal synergy between a β-lactam antibiotic and an aminoglycoside is needed to treat most serious enterococcal infections such as endocarditis. The synergistic bactericidal effect between aminoglycosides and β-lactam is lost if there is high-level resistance to either class of drug. Until recently vancomycin was virtually the only drug that could be consistently relied on for the treatment of infections caused by multi-drug resistant enterococci. The emergence of enterococci with resistance to vancomycin, seen predominantly in the species *E. faecium*, has been followed by an increase in the frequency with which this species is recovered.

Since the first report of vancomycin-resistant enterococci (VRE) in 1988 in England, VRE has spread with unanticipated rapidity and have been reported from many countries. From 1989 through 2002 the percentage of nosocomial enterococcal infections reported to the center for disease control and surveillance system (CDC) due to VRE increased from 0.3% to 27.5%.

Six types of glycopeptide resistance have been described in enterococci that can be distinguished on the basis of the sequence of the structural gene for the resistance ligase (*vanA, vanB, vanC, vanD, vanE, and vanG*), transferability, levels of resistance, and the spectrum of glycopeptides to which the strains are resistant. The VanA type is characterized by high-level inducible resistance to both vancomycin and teicoplanin and is mediated by transposon *Tn1546* or closely related elements. VanB type strains have variable levels of inducible resistance to vancomycin but susceptible to teicoplanin. VanC type resistance is an intrinsic property of *E. gallinarum, E. casseliflavus* and *E. flavescens* and displays a low-level resistance to vancomycin only. VanD type strains are resistant to moderate
levels of vancomycin and teicoplanin. VanE and VanG, recently described in 
*E. faecalis*, are characterized by a low-level of resistance to vancomycin and 
susceptibility to teicoplanin.

Despite the increasing reports of VRE in different countries, the 
prevalence of VRE in Indian hospitals is unknown and very little data is 
available on occurrence of VRE and infections caused by them. The 
genotype involved has not been studied in detail either. At our center, 
vancomycin is used for treatment of serious infections due to methicillin 
resistant *Staphylococcus aureus* (MRSA), which are endemic in many units. 
Oral vancomycin is used to treat *Clostridium difficile* associated colitis. A 
need was felt to screen for VRE to know the magnitude of the problem and 
affected units so that a control policy for our hospital could be formulated. 
Since enterococci are normal inhabitants of gastrointestinal and genitourinary 
tract, urine and stool samples were screened for VRE. The isolates were 
confirmed and speciated, their genotype confirmed by polymerase chain 
reaction (PCR) and sequencing of PCR products was carried out. The risk 
factors for acquisition of VRE were also studied.

The present study has been carried out with the following objectives:

(1) Isolation and identification of *Enterococcus* spp. from clinical 
samples (urine / stool).

(2) Screening of *Enterococcus* strains for vancomycin resistance.

(3) Determination of MICs of *Enterococcus* strains for vancomycin.

(4) Characterization of vancomycin resistance at gene level using 
PCR.

(5) Sequencing of PCR products.

(6) Multiple sequence alignment of *van* genes sequences.