Introduction

Enteroccocci are bacteria that are normally present in the human intestines, female genital tract and are often found in the environment. These bacteria can sometimes cause infections. Vancomycin is an antibiotic that is often used to treat infections caused by enterococci. In some instances, enterococci have become resistant to this drug and thus are called vancomycin-resistant enterococci (VRE). Most VRE infections occur in hospitals. VRE can live in the human intestines and female genital tract without causing disease (often called colonization). However, sometimes, it can cause infections of the urinary tract, heart valves (endocarditis), blood stream, meninges and wounds ***(CDC, April 2008).***

The epidemiology of VRE has not been clarified; however, certain patient populations are at increased risk for VRE infection or colonization. These populations include critically ill patients or those with severe underlying disease or immunosuppression (e.g., patients in intensive care units (ICUs) or in oncology or transplant wards), persons who have had an intraabdominal or cardio-thoracic surgical procedure or an indwelling urinary or central venous catheter, and persons who have had a prolonged hospital stay or received multiantimicrobial and/or vancomycin therapy ***(Montecalvo et al., 1994).***

The isolation of vancomycin-resistant enterococci (VRE) was first reported in 1988 in the United Kingdom and France and then in hospitals in

the United States. VRE are also encountered in many countries, especially in Europe and the United States ***(Bo et al., 2007).***

There are five recognized phenotypes of vancomycin resistance, *van*A, *van*B, *van*C, *van*D, and *van*E. Two of these (*van*A and *van*B) are mediated by newly acquired gene clusters not previously found in enterococci. *van*A and *van*B resistance phenotypes were described primarily in enterococcus faecalis (*E. faecalis)* and enterococcus faecium (*E. faecium)* ***(Cetinkaya et al., 2000).***

*Van*A phenotype is characterized by acquired inducible and high-level resistance to vancomycin and teicoplanin, whereas *van*B phenotype is characterized by variable levels of resistance to vancomycin with in vitro susceptibility to teicoplanin ***(Thierry et al., 2005).***

A third phenotype, *van*C, shows low-level resistance to vancomycin without teicoplanin resistance ***(Reina et al., 2000).***

Studies suggest that once vancomycin-resistant enterococci are introduced in a facility, and particularly after they have spread to multiple patients or wards, control is very difficult ***(Belinda et al., 2001).***

Detection of VRE colonization relies on culture techniques using selective media. The most effective medium identified for screening for VRE has been bile esculine azide agar supplemented with 6 µg of vancomycin per ml ***(Nathan et al., 2007).***

Direct multiplex PCR assay on specimens, using *vanA* and *vanB* primers, provides rapid results and is more sensitive than culture on selective media ***(Suzanne et al., 2003).***