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Short communication

Vancomycin resistant *enterococcus* causing surgical site infections in a tertiary care hospital

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Presence of Vancomycin Resistant Enterococcus (VRE) is increasingly being reported. In the present study we reported the prevalence of Vancomycin Resistant Enterococcus causing Surgical Site Infections (SSI) in a tertiary care hospital. Pus from surgical site wound was cultured and Enterococcus species was identified as standard microbiological methods. The antimicrobial susceptibility for Vancomycin was determined by Minimum Inhibitory Concentration (MIC) by broth dilution method. Patients (n = 2566) who had undergone surgery were included in the study. Enterococcus species had grown in cultures in 112 (4.3%) patients. Vancomycin resistance was observed in 9 isolates (8.03%). The rampant use of higher antibiotics in hospital has already paved way for multi drug resistance. The presence of Vancomycin Resistance must caution both physicians as well as hospital administrators on the urgent need for stringent hospital infection control programmes.

Key words: Vancomycin resistant enterococcus, VRE, surgical site infection.

INTRODUCTION

During the 1960s and 1970s, it was seen that gram-negative organisms were leading causes of hospital acquired infections. Gram-positive organisms were typically sensitive to most antibiotics (Swartz et al., 1994). However, with the emergence of gram-positive organisms as leading causes of hospital acquired infection in the 1990s, called for a re-evaluation of public research priorities (Swartz et al., 1994). One of the organisms that caused concern was Vancomycin Resistant *Enterococcus* (VRE) that was first reported in France in 1988 by Uttley et al. (Uttley et al., 1988). Since then, the incidences of Vancomycin Resistant *Enterococcus* have been reported in many parts of the world (Linden et al., 1996; Tenover and McDonald, 2005; Fica et al., 2007). The number of cases infected with VRE, as reported by the US Centers for Disease Control and Prevention, has risen from 99 in 1992 to 278 in 1994 (Tenover and McDonald, 2005). The more recent reports are both distressing as well as alarming. Studies by Khudaier et al. (2007) propose that prolonged hospital stay and the irrational use of vancomycin were important risk factors for both vancomycin-resistances in *Enterococci* (Khudaier et al., 2007). Other significant factors that were associated with the emergence of vancomycin-resistant Enterococci were renal dialysis and or renal failure, prior aminoglycoside and the use of third generation cephalosporins. They also emphasized on the importance of screening for VRE in clinical samples (Khudaier et al., 2007). Vancomycin resistant *Enterococcus* is known to cause significant mortality and morbidity (Khudaier et al., 2007; Roghmann et al., 1997; Schaberg et al., 1991). The present study aims at determining the prevalence of vancomycin resistance in Enterococcus causing surgical site wound infections.

MATERIALS AND METHODS

Patients who underwent surgery in a tertiary hospital in Mysore were observed for surgical site wound infections from January, 2005 - January, 2006. A wound swab was used to collect the pus from the incision site using a sterile swab and cultured for growth. *Enterococcus* that grew on culture was identified by biochemical tests. An antimicrobial susceptibility testing by Kirby Bauer Disc diffusion method was carried out on all the isolates. The antimicrobial susceptibility testing included antibiotics such as Amikacin, Gentamicin, Ciprofloxacin, Ofloxacin, Penicillin, Ampicillin and Imipenem. Vancomycin resistance was determined by Minimum Inhibi-

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Table 1. Organisms causing surgical site wound infections.

Organisms	Surgical site infection (total N = 2566)	
	Number (No)	Percentage (%)
Escherichia coli	10	0.89
*Enterococcus faecalis (*Vancomycin sensitive *Enterococci)*	43	3.82
Klebsiella spp.,	2	0.18
Methicillin Resistant Staphylococcus aureus	19	1.69
Methicillin Sensitive Staphylococcus aureus	26	2.31
Pseudomonas aeruginosa	33	2.93
Vancomycin Resistant Enterococcus	9	0.18
Total	135	

Inhibitory Concentration (MIC) by broth dilution method. ATCC strain *Enterococcus faecalis* 29212 was used as aquality-control organism.

RESULTS

A total of 2566 patients underwent surgical procedures that included minor surgeries such as Appendisectomy, Tubectomy and Prostatectomy and major surgeries such as Hysterectomy, Gasterectomy, Gastero-jejunovagotomy. Minor surgeries were carried out in 69.1% and major surgeries in 30.8% of the patients who were included in the study. The age groups of the patients ranged from 25 - 65 years, mean age 43.04 ± 10.87 years. The patient population comprised of 43.8% males and 56.2% females. *Enterococcus* species was one of the pathogens that caused surgical site wound infections 112 (4.3%) samples. The other etiological agents were *Klebsiella* species, *Pseudomonas aeruginosa*, *Escherichia coli* and *Staphylococcus aureus*. The percentages of the organisms isolated from surgical site wound infection are shown in Table 1. Minimum Inhibitory concentration yielded 9 of the 112 isolates of the *Enterococcus* species as resistant to Vancomycin. This translates into 8.03% Vancomycin resistance in *Enterococcus*. A single isolate was moderately sensitive (0.89%) and the remaining 102 (91. 07%) were sensitive to Vancomycin. The VRE isolates were resistant to most of the other antibiotics tested. With regard to *S. aureus*, all the isolates were sensitive to Vancomycin. Amikacin sensitivity for Vancomycin resistant organisms was 33%, Gentamicin was 44%, and Ciprofloxacin was 22%. All the VRE strains were resistant to Penicillin and Ampicillin. All the strains were sensitive to Imipenem.

The risk factors that was associated with surgical site infections was age > 45 years (adj. OR = 3.74, p value = 0.012). The other group that were significantly at risk were diabetics (adj OR = 0.26, p value < 0.001). Surgeries such as Gasterectomy, Cholescystectomy, Prostatectomy and Hysterectomy were risk factors (adj OR = 39.43, 12.71, 10.51 and 9.51; p value = <0.001, 0.037, 0.013 and 0.015 respectively).

DISCUSSION

Enterococcus species, by large is the second most common pathogen causing nosocomial urinary tract infection and the third most common pathogen causing bacteremia in the United States (Clevel, 1990). Closer home, the prevalence of VRE cannot be discounted (Khudaier et al., 2007). A major reason for the survival of *Enterococcus* in hospital environment is their intrinsic resistance to several commonly used antibiotics and, perhaps more important, their ability to acquire resistance to all currently available antibiotics, either by mutation or through the transfer of plasmids and transposons (Cetinkaya et al., 2000). We isolated 4.3% E. species from surgical site infections alone which may be only the tip of the iceberg. E. species causing the other nosocomial infections will definitely add over these numbers. While *Enterococcus* is inherently resistant to most of the drugs, vancomycin resistance complicates treatment and management of the disease. The prognosis is also affected leading to increased mortality and morbidity (Roghmann et al., 1997). The other factors that are also affected are the added burden on costs incurred by the patient in the form of prolonged hospital stay and medication. While the prudent use of higher antibiotics such as vancomycin are advocated in hospital settings; education programs for the different categories of health care workers, a well equipped laboratory to identify vancomycin resistance in Enterococci, implementation of infection control measures, screening of health care workers in order to identify carrier rates, surveillance cultures in high prevalence areas such as intensive care units and operation theatres are immediate requirements in order to keep the spread of vancomycin resistance under control.

REFERENCES

Cetinkaya Y, Falk P, Mayhall GC (2000). Vancomycin Resistant *Enterococci.* Clin. Microbiol. Rev. 3: 686-707.

Clevel DB (1990). Movable genetic elements and antibiotic resistance in *Enterococci*. Eur. J. Clin. Microbiol. Infect. Dis. 9: 90–102.

Fica A, Jemenao MI, Bilbao P, Ruiz G, Sakurada A, Perez de Arce E,

Zuniga I, Gompertz M (2007). Emergence of vancomycin-resistant *Enterococcus* infections in a teaching hospital in Chile. Rev. Chilena. Infect. 24: 462-671.

Khudaier BY, Tewari R, Shafiani S, Sharma M, Emmanuel R, Sharma M, Taneja N (2007). Epidemiology and molecular characterization of vancomycin resistant Enterococci isolates in India. Scand J. Infect. Dis. 39: 662-670.

Linden PK, Pasculle AW, Manez R, Kramer DJ, Fung JJ, Pinna AD (1996). Differences in outcomes for patients with bacteremia due to vancomycin-resistant *Enterococcus faecium* or vancomycin-susceptible *E. faecium*. Clin. Infect. Dis. 22: 663-670.

Roghmann M-C, Qaiyumi S, Johnson JA, Schwalbe R, Morris JG Jr. (1997). Recurrent vancomycin-resistant *Enterococcus faecium* bacteremia in a leukemia patient who was persistently colonized with vancomycin-resistant *Enterococci* for two years. Clin. Infect. Dis. 24: 514-515.

Schaberg DR, Culver DH, Gaynes RP (1991). Major trends in the microbial etiology of nosocomial infection. Am. J. Med. 91(Suppl. 3B): 72S-75S.

Swartz MN. Hospital-acquired infections: diseases with increasingly limited therapies.1994. Proc. Natl. Acad. Sci. U S A. 91: 2420-2427.

Tenover FC, McDonald LC (2005) . Vancomycin-resistant *Staphylococ*ci and *Enterococci*: epidemiology and control. Curr. Opin. Infect. Dis. 18(4): 300-305.

Uttley AHC, Collins CH, Naidoo J, George RC (1988). Vancomycin-Resistant *Enterococci*. Lancet 1: 57–58.