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Letter to the Editors

Analysis of epidemics of vancomycin-resistant Enterococci in Turkey

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Abstract: The first cases with vancomycin-resistant Enterococci (VRE) isolation were reported in 1988 throughout the world, and in 1998 in Turkey. The number of the papers conducted on cases or epidemics in which VRE was isolated is in increase. In this study, it was aimed to evaluate some studies at this topic. In conclusion, it was observed that the VRE strains isolated in the same clinic within a short period had a high probability to be the same clone, that there was need for extra investigation for teicoplanin resistance in VRE strains in terms of vanB expression, that VRE colonisation was more common in patients with long term intensive care unit stay, and that eradication of VRE could be made with more strict precautions in comparison to other epidemic bacteria.

Keywords: analysis of epidemics, vancomycin-resistant, Enterococcus, Turkey

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Introduction

Enterococci, found in normal flora of gastrointestinal system, are amongst the factors of nosocomial infections. Particularly vancomycin-resistant Enterococci (VRE) can cause morbidity and mortality in patients that have long term hospitalization. The first cases with VRE isolation were reported in 1988 throughout the world, and in 1998 in Turkey. The number of the papers conducted on cases or epidemics in which VRE was isolated is in increase [1, 2]. In this study, it was aimed to evaluate some studies at this topic.

Ergani-Özcan et al. [3] isolated a total of 36 VRE strains from 10 patients in their study conducted in Antalya in 2001, and reported that six of the patients were carriers, two had urinary tract infection, two had bacteriemia, all isolates carried vanA and were all resistant to both vancomycin and teicoplanin (TEC), the isolates consisted of four clones, and the resistance genes were transferred via the transposone Tn-1546.

Yüce et al. [4] obtained eight VRE isolates from the newborns staying in the intensive care unit (ICU) in their study conducted in Izmir in 2001, and detected that low birth weight and long term antimicrobial therapy were both significant risk factors in terms of VRE colonization in the newborns.

Colak et al. [5] isolated 20 VRE strains within the year 2002 in Antalya, and reported that all their isolates were Enterococcus faecium, and they all carried vanA. They also detected that the strains consisted of five distinct clones, and the resistance genes were transferred via the transposone Tn-1546. They added that one of the patients carried two distinct VRE strains.

Aktas et al. [6] isolated VRE strains from a total of 11 patients staying in various clinics including pediatrics in their study conducted in Istanbul n 2006. They stated that all isolates were resistant to vancomycin, TEC, ampicillin, and high-level aminoglycoside. They detected vanA in all isolates. They investigated the clonal relationship using RAPD-PCR, and they detected four distinct patterns.

Comert et al. [7] detected vanA gene in all six VRE isolates obtained from the patients staiying in the ICU in their study conducted in Zonguldak in 2007. They found that all the isolates were just one single clone, and the isolates showed the same antibiotic susceptibility pattern other than the resistance to gentamicin. Using pulsed-field gel electrophrosesis (PFGE), they determined difference consisting of two bands in the isolates resistant to gentamicin.

Benzer et al. [8] isolated VRE strains from rectal swab specimens of 11 newborns staying in the ICU in Istanbul in 2009. After the first isolation, they screened all patients of the ICU, and isolated VRE strains from a total of 52 patients. They found significant association between VRE colonization and birth wights, birth weeks, staying time in the hospital, cephalosporin-use, and ventilator follow-up of the infants.

Kirdar et al. [9] isolated 20 VRE strains all of which were E. faecium from the haemotology clinics in Aydin in 2009. They detected vanA in all isolates. They reported that all isolates were resistant to both vancomycin and eritromycin, and they found that the isolates consisted of two distinct clones using PFGE and multilocus sequence typing (MLST).

Coskun et al. [2] isolated 38 VRE strains all of which were *Enterococcus faecium* within a year in 2010 in Ankara. They detected vanA in 30 isolates which were all resistant to both vancomycin and TEC, and they found vanB gene in the other eight

isolates which were resistant to vancomycin but susceptible to TEC. They determined six distinct clones using PFGE, and they found that all isolates carrying vanB belonged to one single clone sourced from the haemodialysis unit.

Atalay et al. [1] isolated a total of 19 VRE strains from either epidemic or sporadic cases in the ICU of anesthesia clinics in Izmir in 2011. They reported that 11 of these cases were male, and eight were female, and the age range was 18-96 years. They found that ten cases had VRE colonization (seven had rectal, two had urinary, and one had both rectal and urinary colonization), and the rest nine had infection. Amongst the infections, five had bacteriemia, three had catheter infection, and one had urinary tract infection, hovewer, they stated that four of these patients had rectal colonization in the past. They found vanA in the seven of eight isolates using PCR. They detected VRE in the environmental cultures within the mentioned period.

Efe-Iris et al. [10] isolated VRE strains from 21 patients in their study conducted in Istanbul in 2012. They found that those patients used nasogastric tube or venous catheter and 3rd or 4th generation cephalosporins, aminoglycoside, piperacillin-tazobactam, and levofloxacin in a significantly higher rate in comparison to the other patients, and they were all stayed in the ICU for significantly longer times.

The studies conducted in our country showed that VRE carriage was found to be significantly higher in patients with long term stay at hospital, particularly in intensive care unit, and in premature and/or low-weight newborns. It was noted that long time treatment with antibiotics such as cephalosporins and use of catheter or etc. were predisposing factors, however, these factors weren't analysed independently from long term hospitalization. In these studies, it was shown that all VRE isolates resistant to vancomycin and TEC had vanA gene, and the ones susceptible to TEC had vanB. It was detected that vanA resistance gene was transferred via transposon Tn-1546. In the molecular assays such as Pulsed-field Gel Electrophoresis, Multi Locus Sequence Typing and Random Amplification of Polymorphic DNA PCR, it was observed that the epidemics were caused by not only one single clone but multiple ones. It was noted VRE colonisation was a predisposing factor for VRE-caused infections, but that age and gender weren't determining factors for colonisation. In some of the studies, in cases of a VRE isolation from a routine culture of a patient, screening cultures were performed in the other patients or from environment, and other VRE strains were isolated, and this finding suggest that VRE may be hospital-acquired as well as it was endogeneously origined. However, there aren't any evidences proving that no detection of VREs in routine cultures of the patients means ther is no VRE colonisation in the clinic. In addition, detection of different clones in isolated VRE strains showed that not just one factor was responsible in the transmission. Not causing of the epidemics by one single clone makes us consider that glycopeptide resistance is transferred to different clones vie transposons rapidly, and this condition brings the question if the epidemics occured bu the clone itself or by the transfer of resistance. In conclusion, it was observed that the VRE strains isolated in the same clinic within a short period had a high probability to be the same clone, that there was need for extra investigation for TEC resistance in VRE strains in terms of vanB expression, that VRE colonisation was more common in patients with long term ICU stay, and that eradication of VRE could be made with more strict precautions in comparison to other epidemic bacteria.

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