Colonization with vancomycin-resistant enterococci (VRE) in intensive care unit patients in Cordoba City, Argentina

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ABSTRACT

The purpose of this study was to determine the prevalence of colonization with vancomycin-resistant enterococci (VRE) among intensive care unit (ICU) patients in a hospital in Córdoba, Argentina. We collected 235 rectal swab specimens from 147 ICU patients. Resistance to vancomycin was screened with the disk diffusion method, and MICs were determined with the E-test method. Vancomycin-resistant genotypes were determined by PCR. The VRE strains were isolated from 18/147 patients (12.2%). The isolates were identified as Enterococcus faecium (94.4%), and Enterococcus gallinarum (5.6%). PCR showed that the E. faecium strains carried the vanA gene, and the E. gallinarum strain carried the vanC1 gene. Our study indicated that at least 12.2% of ICU patients were VRE carriers.

Key words: Enterococcus spp., resistance, vancomycin, colonization

Colonización con enterococos vancomicina-resistentes (EVR) en una unidad de cuidados intensivos en la Ciudad de Córdoba, Argentina. El propósito de este estudio fue determinar la prevalencia de colonización con EVR en pacientes de una unidad de cuidados intensivos (UCI) en un hospital de la Ciudad de Córdoba, Argentina. Se recolectaron 235 muestras por hisopado rectal de 147 pacientes. La resistencia a vancomicina fue estudiada por el método de difusión con discos y las CIMs fueron determinadas por E-test. Los genotipos de resistencia fueron determinados por PCR. Se aislaron cepas de EVR en 18/147 pacientes (12.2%). Los aislamientos fueron identificados como Enterococcus faecium (94.4%) y Enterococcus gallinarum (5,6%). Todas las cepas de E. faecium fueron portadoras del gen vanA y la cepa de E. gallinarum del gen vanC1 de resistencia intrínseca. Este estudio mostró que el 12,2% de los pacientes internados en la UCI fueron portadores de EVR.

Palabras clave: Enterococcus spp., resistencia, vancomicina, colonización

Bacterial resistance continues to be a problem all over the world and enterococci are not ruled out of it. During the past decade vancomycin-resistant enterococci (VRE) has become an established nosocomial pathogen in intensive care units (ICU) and, increasingly, in hospital wards (3). Thus, the ICU patients are at risk for colonization with VRE (14).

The epidemiology of VRE colonization, infection, and rapid dissemination in the United States and in Western Europe has been well described, and the prevalence rates vary among different centers (7). Once VRE colonization is detected in a hospital, a rapid increase of clinical associated infections is generally followed (1). The preponderance of both, the vanA and vanB genotypes has been shown in most countries, because of their high capacity of dissemination. The high prevalence of fecal carriage could be associated to clinical infections, and these infections may be life-threatening because choices for alternative treatments are limited (1).

In 1988, the first isolate of a VRE strain (15) was reported in France and the United Kingdom. Other protocols of survey of fecal carriage of VRE were reported some years later in different parts of the world (7). In 1996, the first clinical case of bacteremia by vancomycin-resistant Enterococcus faecium was detected in Mendoza City, Argentina (9) and in 1997, the growth of E. faecium genotype vanA was detected at the Hospital Aeronáutico, in Buenos Aires City (11). Data provided by the “Informe de vigilancia de la resistencia a los anti-microbianos Red-Whonet Argentina 2002, Documento regional OPS”, has shown an increase of vancomycin-resistance in E. faecium isolated from clinical infections rising from 0.8% in 1998 to 11% in 2002.

To determine the prevalence of colonization with VRE among ICU patients at Rawson Hospital in Cordoba City, Argentina, were collected 235 rectal swab specimens from 147 patients between November 2002 to December 2003. One to three clinical samples were obtained as follows: on admission, on day seven and on day fourteen. Rectal swabs were inoculated onto bile-esculin-azide agar plates with 6 µg/ml of vancomycin. Plates were incubated at 37 °C for 24 h (8). Colonies with a dark brown halo and
morphologically resembling enterococci were analyzed. Species identification of enterococci was made according to Facklam’s scheme (6).

Resistance to vancomycin and teicoplanin was screened by the disk diffusion method (disks from Oxoid Limited, Basingstoke, Hampshire, England), and the breakpoints used were taken from NCCLS guidelines (2000) (10). MIC for vancomycin was performed by the E-test method (AB Biodisk, Solna, Sweden). Enterococcus faecalis ATCC 29212 and E. faecalis ATCC 51299 were used as quality control strains.

Vancomycin resistance genotypes were determined by PCR. VanA, vanB, and vanC1 genes were amplified with the primers described by Dukta-Malen et al. (4).

The VRE strains were isolated from 18/147 patients (12.20%). The isolates were identified as E. faecium, 17/18 (94.4%), and E. gallinarum, 1/18 (5.6%). The VRE strains were isolated in the first sample in 12 patients, whereas the other 6, were obtained in the second sample. Vancomycin MICs of all E. faecium were ≥ 256 µg/ml. E. gallinarum MIC was 24 µg/ml. PCR was performed to 14 strains; 13 E. faecium strains carried the vanA gene, and the only E. gallinarum strain carried the vanC1 gene.

The prevalence of intestinal colonization of VRE in ICU patients at Rawson Hospital (12.20%) was similar to that reported by Coque et al. (3) in hospitals in the USA, and was higher than that reported by Endz et al. (5) in Europe (4.9%). Zanella et al. (16) reported vanA Enterococcus clinical isolates from colonized patients obtained during a nosocomial outbreak in a hospital in São Paulo, Brazil.

During the VRE colonization study there were two cases of bacteremia in immunocompromised patients admitted to ICU (data not shown). The six VRE isolated in the second sample of the study would suggest a 4.1% (6/147) ICU VRE acquisition.

Most of these strains were E. faecium as was shown by the majority of surveys in other countries, and they carried the vanA gene. A survey in Argentina showed that E. faecium carrying vanA gene is the most prevalent combination (2).

E. gallinarum that showed intrinsic resistance to vancomycin through vanC1 genotype, was the only different species found in this study. This isolate was detected because we included CIM to vancomycin by E-test for all strains. It is well known that the intrinsic resistance of E. gallinarum and E. casseliflavus cannot be detected by the diffusion method. Our purpose was to detect other phenotypes of acquired resistance, as was demonstrated by Togneri et al. (13) in an isolate of E. gallinarum with the vanA gene, isolated from a bacteremia in Argentina. It should be also pointed out that the strain of E. gallinarum isolated carried the vanC gene of intrinsically resistance to glycopeptides only; this strain was not epidemiologically relevant because this resistance is constitutive and not transferable.

Avoparcin, a glycopeptide agent that has been used extensively as a growth promoter in animals, have contributed to the development of VRE in Europe (3). We excluded the use of avoparcin in our country. Further studies are required to clarify the epidemiology of VRE at Rawson Hospital, unless the most important risk factor was the prior administration to patients of vancomycin and other antimicrobial agents.

Since the vanA and vanB resistance genes are transferable, glycopeptide resistance might be carried to other microorganisms like methicillin-resistant Staphylococcus aureus, as was recently reported (12). Therefore, these strains should be very difficult to treat with the antimicrobial drugs currently available.

The VRE isolates herein described are showing the overuse of antimicrobials. Extreme precautions should be taken to avoid the spread of these strains. Education, permanent vigilance, and appropriate use of antibiotics are the foundations to minimize this problem.

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