

Original

Community-acquired urinary tract infection caused by vancomycin-resistant *Enterococcus faecalis* clinical isolate

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SUMMARY

We present a case of urinary tract infection caused by vancomycin-resistant *Enterococcus faecalis*. The patient is a 62-year-old woman showing no recent admittances. The isolated microorganism was identified by MicroScan® (DADE) and API® (BioMerieux) and susceptibility was assessed by disk diffusion, E-test® and broth microdilution. The isolate was identified as *Enterococcus faecalis* and showed high MIC for vancomycin (>128 mg/l) and teicoplanin (8 mg/l) but was susceptible to ampicillin. The transmission routes of vancomycin-resistant enterococci in the community and their clinical implications remain uncertain. Healthy carriers have already been described in several countries but this case report represents an unusual finding.

Key words: *Enterococcus faecalis* - Vancomycin-resistance - Urinary tract infection - Community-acquired infection

Infección urinaria causada por un aislamiento clínico de *Enterococcus faecalis* resistente a la vancomicina

RESUMEN

Presentamos un caso de infección urinaria causada por *Enterococcus faecalis* resistente a vancomicina. La paciente es una mujer de 62 años que no había tenido ingresos recientes. El microorganismo aislado fue identificado por MicroScan® (Dade) y API® (BioMerieux) y la sensibilidad fue confirmada mediante difusión con disco, E-test® y microdilución en caldo. El aislamiento fue identificado como *Enterococcus faecalis* y mostraba altas CMI para vancomicina (>128 mg/l) y teicoplanina (8 mg/l), pero era sensible a ampicilina. Las vías de transmisión de los enterococos resistentes a la vancomicina en la comunidad y sus implicaciones clínicas continúan siendo poco conocidas. Ya se han descrito casos de portadores en distintos países, pero éste de una paciente sin ingresos previos resulta poco frecuente.

Palabras clave: *Enterococcus faecalis* - Resistencia a vancomicina - Infección del tracto urinario - Infección comunitaria

INTRODUCTION

Enterococcus spp. were first recognized as an important cause of hospital-acquired infections in the mid 1970s. Their rising pathogenicity was probably due to the use of third-generation cephalosporins, to which these bacteria are intrinsically resistant. They are one of the most common microorganisms recovered from nosocomial urinary tract and wound infections (1).

Two of the main reasons why enterococci are usually isolated from the hospital environment are their intrinsic resistance to several antibiotics and their ability to acquire resistance to nearly all known antibiotics.

In 1988 the isolation of a vancomycin-resistant *Enterococcus faecalis* strain was reported for the first time (2). Later on, vancomycin-resistant enterococci (VRE) spread to many countries causing significant nosocomial outbreaks.

Currently, there are five recognized phenotypes of vancomycin resistance: VanA, VanB, VanC, VanD and VanE.

Community-acquired infection and VRE in healthy carriers have rarely been described (3). There seem to be epidemiological differences between the US and Europe (4). In the US, few cases have been reported and the most likely hypothesis is that patients colonized in hospitals would be the source of transmission. On the other hand, in Europe glycopeptide-containing animal feeds are believed to be responsible for VRE intestinal colonization in farm animals and pets. Consequently, VRE might reach humans through the food chain or through contact with domesticated animals (5).

We report a community-acquired urinary tract infection (UTI) caused by vancomycin-resistant *E. faecalis*.

PATIENT AND METHODS

The patient was a 62-year-old female, who was hysterectomized 15 years ago. She reported four surgical interventions due to a cystocele. The last operation took place 11 years ago, and she reported no further admittances at any hospital.

Over the past several years, the patient also suffered from repeated urinary tract infections, with the most recently isolated microorganisms being *E. coli* (four times) and *S. aureus* (once). Those acute episodes were treated with several antibiotics (amoxicillin/clavulanic acid, fosfomicin, etc.). She had been under preventive treatment with trimethoprim/sulfamethoxazole for 6 months as well.

During the present episode, she consulted her physician because of typical UTI symptoms (dysuria and bladder tenesmus) and a urine sample was collected.

The urine sample was cultured in CLED agar using a calibrated loop. The identification of the infective agent

and its drug susceptibility were first achieved by a commercially available method following manufacturer's recommendations (*MicroScan*[®], DADE).

Identification was later confirmed by API rapid strep system (BioMerieux).

To rule out *Enterococcus* spp. intrinsically resistant to vancomycin, two different methods were used:

- 1) The absence of motility was observed with direct microscopic detection (6).
- 2) The absence of pigmentation was determined by culture on TSA (trypticase-soy agar).

Vancomycin resistance was also estimated by several methods:

- Vancomycin and teicoplanin disk diffusion in Mueller-Hinton agar according to NCCLS recommendations (7).
- Vancomycin and teicoplanin *E-test*[®] (AB biodisk).
- Broth microdilution following NCCLS methods (8).

RESULTS

After 24 hours of incubation, more than 100,000 CFU/ml of a Gram-positive coccus was isolated and later identified as *E. faecalis*. This isolate was immobile and nonpigmented in TSA.

The microorganism showed vancomycin resistance (MIC >128 mg/l) and a high MIC for teicoplanin (8 mg/l by broth microdilution), but it was ampicillin susceptible (MIC = 0.5 mg/l).

Table 1 shows susceptibility results obtained with the different procedures performed.

Symptoms ceased with antibiotic treatment and further urine cultures were negative.

Table 1. Susceptibility results for the antibiotics tested.

	Disk diffusion (mm)	<i>E-test</i> [®] (mg/l)	Broth micro-dilution (mg/l)	<i>MicroScan</i> [®] (mg/l)
Ampicillin	NT	NT	0.5	0.5
Vancomycin	6	>256	>128	>16
Teicoplanin	10	16	8	>16

(NT: not tested).

DISCUSSION

The identification of a VRE strain as the cause of a community-acquired urinary tract infection is an unusual finding.

In the US, there is little evidence that VRE transmission occurs in the community. The opposite is true in Europe, where these microorganisms have been isolated from different animal sources and from healthy individuals. In Spain, some studies have demonstrated the presence of VRE in animals and food (9).

Several conditions are considered risk factors for colonization or infection by VRE, and these are mainly related to hospitalization. In this case, the source of infection remains uncertain because none of the main risk factors described were found (4).

Some identification methods may confuse *E. faecalis* resistant to vancomycin with intrinsically vancomycin-resistant enterococci; motility and pigmentation tests are therefore recommended to discriminate between the two types of resistance. In this case, both tests were negative.

Several mechanisms of resistance have been described in *Enterococcus* spp. (VanA, VanB, VanC, VanD and VanE), but only VanA, VanB and VanE have been observed in *E. faecalis* (10–12). VanA phenotype shows high-level resistance to vancomycin and teicoplanin, VanB phenotype exhibits high-level resistance to vancomycin but low-level resistance to teicoplanin and VanE isolates are susceptible to teicoplanin and show low-level resistance to vancomycin.

Van B, whose MIC values for vancomycin are between 4 and more than 1000 mg/l and whose MIC values for teicoplanin are between 0.5 and 32 mg/l, is probably the phenotype involved in our case.

However, there is not always an exact correlation between genotype and phenotype in glycopeptide-resistant enterococci (13). Recent reports suggest that mutations in the *vanS* regulatory gene could result in impaired resistance to teicoplanin among vancomycin-resistant enterococcal isolates possessing the *vanA* gene cluster (14). Furthermore there have been reports of isolation of vancomycin-resistant enterococci with van B phenotype-van A genotype incongruence in Taiwan (15) and Korea (16). Unfortunately we were not able to confirm our findings with genetic methods.

Even though community-acquired VRE infections are not as alarming as hospital-acquired ones, we should not overlook epidemiological surveillance because these microorganisms may cause treatment failures.

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