Case Report

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Vancomycin resistant enterococcal infections in infants and neonates

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Abstract:

Vancomycin-resistant Enterococci have become one of the most challenging nosocomial pathogens with limited therapeutic options. We report three cases of Vancomycin-resistant *Enterococcus faecium* isolated from urine, perinephric collection and blood of paediatric patients over a period of one year from June 2015–2016 in a tertiary care centre in South Kerala.

Keywords:

Enterococcus faecium, nosocomial pathogens, Vancomycin-resistant Enterococci

Introduction

There are two types of Vancomycin resistance in enterococci spp. The first type is intrinsic resistance. Isolates of *Enterococcus gallinarum* and *Enterococcus casseliflavus/Enterococcusflavescens* demonstrate an inherent, low-level resistance to Vancomycin. The second type of Vancomycin resistance in enterococci spp. is acquired resistance.

Enterococci spp. can become resistant to Vancomycin by acquisition of resistance genes from another organism. Most commonly, this resistance is seen in *Enterococcus faecium* and *Enterococcus faecalis*, but also has been recognised in *Enterococcus raffinosus, Enterococcus avium, Enterococcus durans* and several other enterococcal species.

Several genes, including *vanA*,*vanB*, *vanC*, *vanD* and *vanE*, contribute to resistance to Vancomycin in enterococci spp.

E. faecium is the most frequently isolated species of Vancomycin-resistant enterococci (VRE) in hospitals and typically produces high Vancomycin (64–1000 μg/ml) and Teicoplanin (16–512 μg/ml) minimum inhibitory concentrations (MICs). These isolates typically contain *vanA* genes. *vanB* confers varied resistance to Vancomycin, ranging from moderate- to high-level resistance (MIC range, 4–1000 μg/ml) and is susceptible to Teicoplanin (MIC, 0.5–1 μg/ml).[1]

E. gallinarum and *E. casseliflavus/E. flavescens* isolates are intrinsically resistant to Vancomycin. These isolates contain Van C genes that typically produce Vancomycin MICs of 2–32 μg/ml.[1]

From the first isolation of Vancomycin-resistant *E. faecalis* and *E. faecium* in England in 1988, VRE have spread with unanticipated rapidity and are now encountered by hospitals in many parts of the world.[2]

We report three cases of Vancomycin-resistant *E. faecium* isolated from urine, perinephric collection and blood of paediatric patients over a period of one year from June 2015–2016 in a public sector, paediatric tertiary care centre in South Kerala [Table 1].

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Case 1

An eight-month-old male baby presented with a history of fever and recurrent urinary

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Table 1: Details of patients from whom Vancomycin-resistant enterococci were isolated

Serial number	Age	Sex	Clinical condition	Sample sent for culture and sensitivity
Case 1	8 months	Male	Persistent posterior urethral valve with left hydroureteronephrosis	Aspirate from perinephric abscess and urine
Case 2	2 days	Female	Meconium stained amniotic fluid with respiratory distress syndrome	Blood
Case 3	7 months	Female	Congenital acyanotic heart disease with lower respiratory tract infection	Blood

tract infection. The patient had posterior urethral valve and micturating cysto-urethrogram showed the findings suggestive of chronic cystitis. On ultrasound examination, the patient had left hydroureteronephrosis. The perinephric collection and urine of the patient was sent for culture and sensitivity to the microbiology laboratory. From both samples, *E. faecium* was identified by standard methods.

Case 2

A full-term baby was delivered by vacuum extraction, due to meconium-stained amniotic fluid. The child showed poor feeding and respiratory distress. The clinical diagnosis was early-onset neonatal sepsis. The baby was admitted in neonatal intensive care unit (ICU) and started on parenteral Piperacillin–Tazobactam and Amikacin. Two blood samples collected at one-hour interval were sent to the microbiology laboratory for culture and sensitivity. Both the samples yielded *E. faecium*.

Case 3

A seven-month-old female baby who was severely malnourished was diagnosed to have atrioventricular cushion defect, with a history of recurrent lower respiratory tract infections. The child now presented with features of sepsis. She was started on parenteral Piperacillin-Tazobactam and Vancomycin. Since there was no clinical improvement, two blood samples collected at one-hour interval were sent to the microbiology laboratory for culture and sensitivity, and injection Meropenem was added. Both the samples yielded *E. faecium* in the first subculture itself by standard methods.

Antibiotic sensitivity pattern

Isolates from all the three cases had the same antibiotic sensitivity pattern. They were sensitive only to linezolid and was resistant to Penicillin, Ampicillin, Erythromycin, high-level Gentamicin and Vancomycin by Kirby–Bauer disc diffusion method. The MIC for Vancomycin was >32 μg/ml when tested with E-test strip.

In the first case, the strain was confirmed by polymerase chain reaction for Van A gene at Christian Medical College, Vellore. The positive control for *vanA* gene was *E. faecalis* (ATCC 51,299)and negative control was *E. faecalis* (ATCC 29,212).[3]

In all the three cases, the identification of the organisms as Vancomycin-resistant *E. faecium* was confirmed by Vitek 2 (bioMerieux, France). The MICs for Vancomycin and Teicoplanin was >32 μg/ml with probably Van A phenotype as per Vitek 2 report.

Discussion

VRE is a serious threat to patients with impaired host defence mechanism. The risk factors associated with colonisation and infection with VRE include prolonged hospitalisation, especially in ICU; intrahospital ward transfers; previous antimicrobial therapy, especially Vancomycin use and third-generation Cephalosporins; exposure to contaminated medical equipment such as electronic thermometers and proximity to a previously known VRE patients or staff members who had taken care of VRE patients.[4]

E. faecalis is the predominant species accounting for 80%–90% of all clinical isolates and *E. faecium* accounts for 5%–15%. Drug resistance is more common in *E. faecium* (72%) than that in *E. faecalis* (45%). The most common phenotype seen among VRE strains is the *vanA* phenotype.

Identification of VRE to species level aids in confirming whether an isolate has intrinsic (Van C) or acquired resistance (VanA or VanB). The knowledge of the type of resistance is critical for infection control purposes. *vanA* and *vanB* genes are transferable and can spread from organism to organism. In contrast, VanC genes are not transferable, have been associated less commonly with serious infections and have not been associated with outbreaks.[5] Here, the cases had occurred at different times of the year and from different locations in the hospital, and hence, the possibility of a common source was less.

In the second case, in which there was a history of difficult labour with foetal distress, high vaginal swab of the mother also grew *E. faecium* with same antibiotic sensitivity pattern. However, in other two cases, the source could not be identified. Contact isolation was done to prevent the spread of infection. All the three cases responded well to treatment with Linezolid.

Conclusion

This case series signifies the emergence of VRE in this geographical area. The emergence and spread of this pathogen show the need for appropriate infection control policies and strict enforcement of antibiotic policies. Well-planned screening programs with barrier nursing and isolation of patient may help prevent the spread of such pathogens.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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