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Opinion Article

Detection of Vancomycin-Resistant *Enterococcus* (VRE) Infections in Patients

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DESCRIPTION

The term "Vancomycin-Resistant *Enterococcus*" (VRE) refers to an illness caused by *enterococcus* bacterial strains that are resistant to the antibiotic Vancomycin. Normal habitats for *Enterococcus* bacteria include the female vaginal tract and the intestines. Fever, a rapid heartbeat, bodily redness, swelling, or soreness, and chills are the symptoms.

Treating patients with infections caused by such germs, the rise of vancomycin resistance in *enterococci* as well as the rising occurrence of high-level enterococcal resistance to penicillin and aminoglycosides provides a significant problem. Treatment options are usually restricted to antimicrobial drug combinations. Clindamycin and cephalosporin resistance almost always occurs and few isolates are sensitive to the macrolides that are currently on the market, and fluoro-quinolone resistance is now prevalent. Once VRE are established in a hospital setting, it is challenging to prevent further selective pressure in their favour due of their frequent resistance to several antibiotics.

The majority of patients from whom VRE is recovered have colonised the pathogen rather than being infected with it. Hospitals that test perirectal or rectal swab specimens from high-risk patients for VRE may find a ratio of colonised to infected individuals as high as 10:1. In one analysis, *E. gallinarum* accounted for 40% of the organisms colonising the gastrointestinal tract, yet this species didn't result in any infections. VRE infections frequently affect surgical sites, intra-abdominal sites, urinary tract sites, bloodstream sites, and vascular catheterization sites.

Hospitalized patients who are more seriously ill or disabled frequently get VRE infections. Patients with VRE bacteremia may experience mortality rates of 60 to 70 percent. It's possible that the illness caused about half of these fatalities. Enterococcal infection-related mortality was 46% in liver transplant recipients with VRE bacteremia, which was significantly greater than the 25% mortality seen during patients with vancomycin-susceptible enterococcal bacteremia. This indicates that VRE infection is a strong predictor of mortality in liver transplant patients.

Differences in mortality were found among patients with VRE and vancomycin-susceptible enterococcal bacteremias, especially after controlling for factors such as age. There is no information that VRE are more virulent than strains of the same enterococcal species that are vancomycin-susceptible. Even though 80 to 90% of VRE bacteremias have sometimes been monomicrobial, many nosocomial enterococcal bacteremias are polymicrobial. When *E. faecalis* infection was treated with dalfopristin and quinupristin, *E. faecalis* bacteremic superinfection was also observed.

Role of the microbiology laboratory in the detection of VRE

To prevent the nosocomial transmission of VRE must include early diagnosis of patients colonised or infected with VRE. Transmission prevention becomes more difficult when VRE prevalence is in significant levels. First line of defence against the spread of VRE is the microbiology laboratory.

When it pertains to determining VRE colonisation and infection and avoiding the difficult, extensive containment processes that are necessary when recognition of the disease is forced off, the laboratory's capacity to quickly and reliably identify *enterococci* and detect vancomycin resistance is crucial.

It is no longer adequate for regions where VRE have been detected to primarily perform antibiotic susceptibility testing on *enterococci* obtained from ordinarily sterile body sites such as blood or urine. In VRE outbreaks, only 45-50% of VRE isolated from clinical specimens have been recovered from blood or urine samples. As a result, *enterococci* recovered from all body sites should be examined for vancomycin susceptibility as soon as VRE have been found. Susceptibility tests on all enterococcal isolates should be highly considered in geographic regions where VRE have been identified.

Vancomycin resistance must be accurately detected by susceptibility tests, or the prevalence of VRE may be underestimated. Zones of inhibition should be evaluated under transmitted light after 24 hours of incubation in laboratories using disc diffusion method. Agar dilution, agar gradient dilution, broth macro-dilution, or manual broth micro-dilution are all used to determine MICs. 24 hours should be completed incubating these systems. Some fully automated techniques for

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checking *enterococci* for vancomycin resistance are unreliable, particularly when screening for VRE isolates with the Van B resistant determinant.

CONCLUSION

Van A VRE were detected by all susceptibility test methods, but van B VRE were frequently neglected by Vitek GPS-TA and Micro Scan rapid (sensitivities, 47 and 53% respectively), evaluating the accuracy of eight currently used susceptibility test methods (agar dilution, disc diffusion, E-test, agar screen plate, and Vitek GPS-TA and GPS-101). Van C1 and Van C2 VRE may still be detected using any method. If detection of Van A-, Van B-, Van C1-, and Van C2-mediated resistance in *enterococci* is necessary, the agar screen appears to be the most consistent and simple method for routine screening of Vancomycin-Resistant *Enterococcus* (VRE).