



Symposium 2.2

Therapy of VRE Infections

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Antimicrobial resistance in enterococci continues to provide difficulties for the clinician attempting to treat serious infections caused by these organisms. Enterococci are intrinsically resistant to β -lactam antimicrobials, aminoglycosides, lincosamides, and trimethoprim/sulfamethoxazole. In addition, they have acquired resistance to virtually all other antimicrobial agents including the glycopeptides and more recently the lipopeptides and oxazolidinones. Because β -lactam antimicrobials are only bacteriostatic against these agents, they are relatively ineffective when used alone to treat enterococcal endocarditis and meningitis. For such infections which require bactericidal therapy, combination therapy including an aminoglycoside has been traditionally employed. However the emergence of high-level resistance to gentamicin and other aminoglycosides has made this option impossible in a number of settings. A number of new antimicrobials have bacteriostatic and even bactericidal activity against the enterococcus *in vitro* but studies of the use of these compounds in enterococcal infections is limited. Agents with bactericidal activity against the enterococcus include quinupristin/dalfopristin (*E. faecium* only), daptomycin, telavancin, and oritavancin. Unfortunately, there are only limited data available concerning the clinical activity of these compounds for serious enterococcal infections, but several of these (especially daptomycin) are being used off-label for serious infections due to VRE. The oxazolidinone linezolid also has good activity against enterococci and has been used anecdotally to treat enterococcal endocarditis (with varying success) and enterococcal meningitis (where its marked ability to enter the CSF may be of benefit). The glycyclines have intrinsic activity against the enterococcus but unfortunately are bacteriostatic, not bactericidal. Dalbavancin, which has excellent activity against most multi-resistant gram positive organisms, does not have activity against VRE containing the *vanA* gene and thus is likely to be of little utility for serious infections due to VRE.