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The Thistle QA CEU No is: **MT00025**.

Each attendee should claim **THREE** CEU points for completing this Quality Control Journal Club exercise, and retain a copy of the relevant Thistle QA Participation Certificate as proof of registration on a Thistle QA EQA.

Cycle 24 - Organism 8:

Enterococcus faecalis

Enterococci are part of the normal intestinal flora of humans and animals but are also important pathogens responsible for serious infections. The genus *Enterococcus* includes more than 17 species, but only a few, *Enterococcus faecalis* and *Enterococcus faecium*, account for most clinical infections in humans. With increasing antibiotic resistance, enterococci are recognized as feared nosocomial pathogens that can be challenging to treat.

Enterococcus species are hardy, facultative anaerobic organisms that can survive and grow in many environments. In the laboratory, enterococci are distinguished by their morphologic appearance on Gram stain and culture (gram-positive cocci that grow in chains) and their ability to (1) hydrolyze esculin in the presence of bile, (2) grow in 6.5% sodium chloride, (3) demonstrate pyrrolidonyl arylamidase and leucine aminopeptidase, and (4) react with group D antiserum. They were formerly known as group D streptococci until assigned their own genus.

E faecalis and *E faecium* are the most prevalent species cultured from humans, accounting for more than 90% of clinical isolates. *E faecium* represents most vancomycin-resistant enterococci (VRE). Isolation of enterococci resistant to multiple antibiotics has become increasingly common in the hospital setting.

Enterococci have both an intrinsic and acquired resistance to antibiotics, which make them important nosocomial pathogens. Intrinsically, these species tolerate or resist beta lactam antibiotics because they contain penicillin-binding proteins (PBPs); therefore, they are still able to synthesize some cell wall components. They are intrinsically resistant to penicillinase-susceptible penicillin (low level), penicillinase-resistant penicillins, cephalosporins, nalidixic acid, aztreonam, macrolides, and low levels of clindamycin and aminoglycosides. They use already-formed folic acid, which allows them to bypass the inhibition of folate synthesis, resulting in resistance to trimethoprim-sulfamethoxazole.

Enterococci also have acquired resistance, which includes resistance to penicillin by beta-lactamases, chloramphenicol, tetracyclines, rifampin, fluoroquinolones, aminoglycosides (high levels), and vancomycin. The genes that encode intrinsic or acquired vancomycin resistance result in a peptide to which vancomycin cannot bind; therefore, cell wall synthesis is still possible.

Unlike streptococcal species, enterococci are relatively resistant to penicillin, with minimum inhibitory concentrations (MICs) that generally range from 1-8 mcg/mL for *E faecalis* and 16-64 mcg/mL for *E faecium*. Therefore, exposure to these antibiotic agents inhibits but does not kill these species. Combining a cell wall-active agent with an aminoglycoside may result in synergistic bactericidal activity against enterococci.

The acquisition of vancomycin resistance by enterococci has seriously affected the treatment and infection control of these organisms. VRE, particularly *E faecium* strains, are frequently resistant to all antibiotics that are effective treatment for vancomycin-susceptible enterococci, which leaves clinicians treating VRE infections with limited therapeutic options.

Infections commonly caused by enterococci include urinary tract infections, endocarditis, bacteremia, wound infection, and intra-abdominal and pelvic infections. Many infecting strains originate from the patient's intestinal flora. From here, they can spread and cause urinary tract, intra-abdominal, and surgical wound infections. Bacteremia may result with subsequent seeding of more distant sites. Individuals at risk include critically ill patients who have received lengthy courses of antibiotics (particularly those in long-term care facilities), solid organ transplant recipients and patients with hematologic malignancies, and health care workers.

According to recent NNIS surveys, enterococci remain in the top 3 most common pathogens that cause nosocomial infections.

CPD Questions:

1. What were enterococci known as previously?
 2. What is the role of the "peptide" mentioned above in vancomycin resistance?
 3. Why are enterococci so prevalent in nosocomial infections?
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