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## **ENTEROCOCCAL INFECTIONS, VANCOMYCIN RESISTANT**

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Enterococci are gram-positive cocci that occur in singles, pairs and short chains. They were previously classified as Streptococci. *Enterococcus faecalis* and *Enterococcus faecium* are the two major species. Other species are *E. avium*, *E. casseliflavus*, *E. durans*, *E. gallinarum*, *E. hirae*, *E. malodoratus*, *E. mundtii*, *E. raffinosus*, *E. solitarius* and *E. pseudoavium*.

### **Epidemiology**

A major habitat of Enterococci is the gastrointestinal tract of humans and of other animals, where they make up a significant portion of the normal gut flora. Concentrations of enterococci can reach  $10^8$  colony forming units (CFU) per gram in human stool. Most enterococci isolated from human stools are *E. faecalis*, although *E. faecium* is also commonly found in the gastrointestinal tract of humans. Most clinical isolates of enterococci are *E. faecalis*, which account for 80 to 90% of the organisms encountered in the clinical microbiology laboratory. *E. faecium* accounts for 5 to 10% of isolates.

Small numbers of enterococci are occasionally found in oropharyngeal secretions, vaginal secretions and on the skin, especially in the perineal area.

Enterococci are able to grow and survive under harsh conditions and consequently occur in a wide variety of environments. They are present in soil, vegetation, food, water, surface water contaminated by animal feces, sewage and a wide variety of living animals. They survive well on environmental surfaces. They have been cultured on various hospital environments including fingertip cultures, gloved hands, stethoscopes, bedrails, telephones, thermometers, laboratory bench tops and upholstery.

Most enterococci do not have classic virulence factors and are mostly causing infection in severely ill patients. The resistance of enterococci to multiple antimicrobial agents allows them to survive and proliferate in patients receiving antimicrobial chemotherapy. This explains their ability to cause infections in patients receiving a number of different broad-spectrum antimicrobial agents.

The source of these infections remains uncertain. Enterococci are part of the normal gut flora of almost all humans which may be the main source of infections (endogenously acquired). However, in many patients the organisms causing such infections often appear to be exogenously acquired. Enterococci may be spread between patients and even from one institution to another. Enterococci causing nosocomial infections are occasionally found on the hands of medical personnel and have frequently been isolated from environmental sources in hospitals and nursing homes. The environment may simply have been passively contaminated by stool or urine from infected patients. In fact, studies suggest direct cross-infection is rare. Resistant organisms from patients or hospital personnel first colonize the gastrointestinal tract (or occasionally the skin and groin or other contiguous areas) before causing infections in patients. Some health care workers colonized with enterococci in their own gastrointestinal tracts may be responsible for colonization of the patients under their care. Once colonized with resistant enterococci, patients may become carriers for months or even years. In addition, devices such as electronic rectal thermometers may also aid in the spread of resistant organisms.

Risk factors for acquiring nosocomial enterococcal infections include:

- gastrointestinal colonization
- serious underlying disease
- long hospital stay
- prior surgery
- renal insufficiency
- neutropenia
- transplantation (especially liver and bone marrow)
- urinary or vascular catheters
- residency in an intensive care unit
- prior antibiotic therapy (especially with vancomycin, cephalosporins, or aminoglycosides) is also a major risk factor for the acquisition of resistant enterococci. Other antimicrobial agents, including aztreonam, imipenem and ciprofloxacin, have been associated with nosocomial enterococcal infections.

Enterococci are naturally resistant to a variety of antibiotics commonly used to treat infections due to being gram-positive organisms. The majority of strains express chromosomal resistance genes.

- Aminoglycosides (low level)
- $\beta$ -lactams (high Minimum Inhibitory Concentrations – MIC)
- Lincosamides (low level)
- Trimethoprim-sulfamethoxazole (*in vivo* only)

Besides being intrinsically resistant to a large number of antimicrobial agents, Enterococci can also easily acquire new mechanisms of resistance. These antimicrobials are penicillins (organisms with and without beta-lactamase), macrolides, tetracyclines, lincosamides, chloramphenicol, vancomycin, quinolones, quinupristin/dalfopristin (Synercid), and linezolid. Vancomycin resistance is of particular concern.

Enterococci account for 12% of all nosocomial infections and are the third most common cause of nosocomial infections in the USA.

Enterococcus organisms with resistance to vancomycin were first reported in 1988. Since that time the rate of resistance has continued to increase. In Louisiana, the rate was 3% in 1996, 4% in 1998 and had very slowly increased to 6% in 2003.

### **Clinical Description**

Enterococci can colonize or infect patients who receive antibiotic therapy for unrelated non-enterococcal infection. Colonization is usually completely asymptomatic. The majority of patients with severe enterococcal infections are colonized prior to infection. Distinguishing between enterococcal contamination, colonization and infection can be difficult but it is very important in attempting to target pathogens and to reinforce the importance of the judicious use of antimicrobials.

Urinary tract infections are the most common type of clinical disease produced by enterococci and urine cultures are the most frequent sources of enterococci in the clinical microbiology laboratory. Among healthy ambulatory patients, less than 5% of UTIs are caused by enterococci, compared to higher rates among hospitalized patients. Risk factors for enterococcal UTI include urinary tract instrumentation, in-dwelling catheterization, genitourinary tract disease and prior antimicrobial exposures.

Nosocomial bacteremias are increasing in numbers. Portals of entry for enterococcal bacteremia include the urinary tract, intra-abdominal (or pelvic) sepsis, wounds (especially thermal burns, decubitus ulcers, or diabetic foot infections), intravenous or intra-arterial catheters, or cholangitis. Enterococcal endocarditis remains a common cause of bacteremia, accounting for up to 20% of native valve infections.

Enterococci are frequently found as part of a mixed aerobic and anaerobic flora in intra-abdominal and pelvic infections.

They are frequently isolated from mixed cultures with gram-negative bacilli and anaerobes in surgical wound infections, decubitus ulcers and diabetic foot infections and in these cases, their contribution to the disease is uncertain.

Patients with anatomic defects of the central nervous system, prior neurosurgery, or head trauma are at risk for enterococcal meningitis.

### **Laboratory Tests**

Antibiotic sensitivity panel that includes testing for vancomycin resistance by one of the following methods:

1. Disk diffusion-Kirby-Bauer method  
Not available at the State Laboratory
2. MIC-e-test method  
Not available at the State Laboratory for diagnostic purposes

Interpretation:

1. Disk diffusion-Kirby-Bauer method, results indicating that the organism is resistant to vancomycin.
2. MIC-e-test method of 16 or greater for vancomycin is resistant (and a MIC of 4 to 8 for vancomycin is intermediately resistant).

### **Surveillance**

Enterococcal invasive disease is a condition with reporting required within 5 business days.

### **Case Definition**

A case of vancomycin resistant Enterococcus (VRE) is defined as an Enterococcus organism that is resistant to vancomycin (MIC of 16 or greater or Kirby Bauer results of resistant), and was cultured from a sterile body site or is isolated from a newly infected site (i.e. acute decubitus).

An invasive disease is defined as isolation of the microorganism from the following sterile body sites: CSF, pleural fluid, peritoneal fluid, joint fluid, placenta, amniotic fluid, surgical specimen.

### **Intervention**

The main purpose of reporting is to monitor trends in invasive enterococcal disease. No investigation is necessary for individual cases.

Upon the receipt of a report of a case of vancomycin resistant Enterococcus, confirm the diagnosis.

This can be accomplished by reviewing the attached lab report sensitivities that record resistance to vancomycin (MIC of 16 or greater for vancomycin) or contact the physician and/or hospital.

Patients infected or colonized with VRE may be cared for in any patient care setting with minimal risk of transmission to other patients provided appropriate infection control measures are taken.

### **Prevention of transmission**

Persons infected or colonized with VRE are more likely to transmit the organism. Transmission depends primarily on which body site(s) harbor the bacteria, whether the body fluids are excreted and how frequently health care providers touch these body sites. Body fluids which are excreted create a risk for transmission of the organism and possibly, contamination of the environment.

Reinforce employee education about basic infection control measures -especially handwashing between patient contacts.

Patient care equipment should be dedicated for patients identified with VRE (i.e., blood pressure cuffs, thermometer, stethoscopes), otherwise, shared equipment should be appropriately cleaned and disinfected between patient use.

**Long term care facilities may NOT refuse to accept a resident with VRE colonization or infection, as long as the facility is able to address the medical needs of the patient.**

Long term care facilities should have some system in place for alerting HCWs and visitors that a resident is on contact precautions, such as labeling the chart or the door of the room, without compromising that resident's privacy.

**Hospital precaution and isolation:** Contact precautions