Central Venous Catheter Colonization by Linezolid-Resistant, Vancomycin-Susceptible Enterococcus faecalis

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Resistance to linezolid is rare in clinical isolates of Enterococcus faecalis. A strain resistant to this antimicrobial but susceptible to vancomycin was found to cause central venous catheter colonization in a patient who never received linezolid.

CASE REPORT

A 66-year-old man with chronic pulmonary disease and coronary artery disease underwent transhiatal esophagectomy with gastric pull-through for esophageal carcinoma. His postoperative course was complicated by respiratory failure, necessitating mechanical ventilation for nearly 3 months. He also developed Pseudomonas aeruginosa ventilator-associated pneumonia, urinary tract infection, and central venous catheter-associated bloodstream infection. His Pseudomonas infections were treated with courses of piperacillin-tazobactam, imipenem plus ciprofloxacin, and imipenem plus gentamicin.

Approximately 2 weeks after completion of the treatment for the Pseudomonas infections, a blood culture drawn through a central venous catheter grew Enterococcus faecalis susceptible to ampicillin and vancomycin but resistant to linezolid and quinupristin-dalfopristin (Table 1). Simultaneous blood culture obtained from a peripheral vein and two cultures obtained from peripheral vein 3 days later were all negative. Perirectal culture yielded an Enterococcus faecalis strain with a different antibiogram (Table 1). Pulsed-field gel electrophoresis was performed, which revealed that the perirectal and blood E. faecalis strains were not related (more than a seven-band difference). The patient did not receive antimicrobial treatment for the E. faecalis and remained afebrile and hemodynamically stable. Of note, the patient had never received linezolid or quinupristin-dalfopristin (Table 1). At 45 days prior to isolation of the E. faecalis from the central venous catheter, vancomycin-resistant E. faecium was recovered from a perirectal culture. The patient’s condition improved, and he was discharged to a long-term care facility after 91 days of hospitalization.

The widespread and often inappropriate use of broad-spectrum antibiotics in the hospital is recognized as an important contributing factor to the spread of resistance (6). Over the past 35 years, enterococci have become increasingly resistant to antimicrobials, and the first report of glycopeptide resistance was published in 1988 (8). Linezolid (an oxazolidinone) and quinupristin-dalfopristin (a streptogramin) are newer antibiotics for treating infections due to gram-positive resistant pathogens (6). Enterococcal isolates resistant to linezolid or quinupristin-dalfopristin have been described (10), and there are two reports of E. faecium being resistant to both of these agents (1, 2). However, the enterococcal isolates resistant to both linezolid and quinupristin-dalfopristin previously reported were also resistant to vancomycin (1, 2). There are also two other reports of E. faecalis isolates resistant to linezolid but vancomycin susceptible in patients who had received linezolid (4, 7). This is the first report of a linezolid-resistant, vancomycin-susceptible enterococcal isolate in a patient that had never received linezolid.

It is important to note that there are two different resistance mechanisms involved in this case. For linezolid, in at least some of the isolates, the resistance mechanism is characterized by a mutation in the central loop of domain V of the 23S rRNA of the 50S ribosomal subunit (9). For quinupristin-dalfopristin, acquired resistance to one or the other component can be caused by target modification, enzymatic degradation, and active efflux of the drug (6); however, for E. faecalis resistance is effected via an efflux pump conferring resistance to dalfopristin that seems to be intrinsic to this species (11).

Some reports indicate that most of the patients who developed linezolid-resistant colonization/infection during therapy had indwelling prosthetic devices and were receiving extended courses of linezolid therapy (3, 5). One study suggests that E. faecalis may have a greater potential for the development of linezolid resistance than E. faecium (9).

Our case has several interesting points. First, the patient never received linezolid or quinupristin-dalfopristin as antimicrobial therapy. A previous report from Turkey described four strains of E. faecium as intermediately quinupristin-dalfopristin resistant and two strains that were linezolid resistant before either antibiotic had been in clinical use in that country (1). However, antibiotics of the same classes (e.g., virginiamycin, a streptogramin) have been used in animal feed in Europe. It is also possible that the linezolid resistance emerged in an E. faecalis isolate in another patient and was nosocomially transmitted to the patient described above. Second, in our case the isolate was ampicillin and vancomycin susceptible; thus, either drug could be used in conjunction with an aminoglycoside to provide synergistic bactericidal therapy. Third, although he had several risk factors for acquiring a vancomycin-resistant strain (long

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duration of hospitalization, long duration of exposure to anti-
microbials [including vancomycin], severe underlying disease,
hospitalization in an intensive care unit near other patients
with vancomycin-resistant enterococci colonization, age, and
surgical procedure), this strain was vancomycin susceptible.
However, this patient was also found to be colonized with
vancomycin-resistant enterococci (two different species) in ad-
dition to this organism. In our institution, there was only 1 of
822 enterococcal strains that was resistant to linezolid in 2005.

In conclusion, we believe that it is necessary to know the
susceptibility patterns of enterococcal strains for the “newer
antibiotics” in order to better understand the microbiological
and more importantly, the epidemiologic characteristics of
gram-positive pathogens with different resistance mechanisms.
Individual patients may be colonized with multiple enterococ-
cal species with various susceptibility patterns. In addition, it is
also important to implement infection control practices against
these resistant strains, with an emphasis on contact precautions

and more careful use of antibiotics to prevent selection of
resistance.

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<thead>
<tr>
<th>Antibiotic</th>
<th>Blood isolates</th>
<th>Rectal isolate</th>
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<tbody>
<tr>
<td></td>
<td>MIC (mg/liter)</td>
<td>MIC interpretation</td>
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<tr>
<td>Ampicillin</td>
<td>8</td>
<td>S</td>
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<tr>
<td>Vancomycin</td>
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<td>S</td>
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<tr>
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<td>24</td>
<td>R</td>
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<tr>
<td>Quinupristin-dalfopristin</td>
<td>32</td>
<td>R</td>
</tr>
<tr>
<td>Rifampin</td>
<td>2</td>
<td>I</td>
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</tbody>
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*S, susceptible; R, resistant.*