Oerskovia xanthineolytica Bacteremia in an Immunocompromised Host: Case Report and Review

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Oerskovia species, once thought to be nocardiform-like bacteria, have been only rarely associated with human infection. In this report we describe a case of central venous catheter-associated infection that was successfully treated with antibiotics. With the increased use of indwelling devices, these organisms may be more commonly recognized causes of infection. Appropriate antibiotic therapy appears to successfully treat oerskovia infection and may decrease the need for removal of some indwelling access devices.

Oerskovia species have been occasionally isolated from laboratory specimens [1] but only rarely associated with clinical disease [2–8]. Most infections have occurred in patients with indwelling access devices that allow the organism to bypass nonspecific host defenses [3, 5–8]. To our knowledge, only seven cases of human infection have been previously reported [2–8]. We describe a case of Oerskovia xanthineolytica bacteremia in an immunocompromised patient with a central venous catheter and review other reported cases.

Case Report

A 49-year-old woman with a history of metastatic colonic adenocarcinoma presented for chemotherapy and evaluation of a malfunctioning central venous catheter. Fifteen months before presentation, a right subclavian Groshong catheter was placed for palliative chemotherapy because a 4-year-old left subclavian Port-a-cath (Pharmacia, North Ryde, Australia) that was placed at the time of initial chemotherapy was nonfunctional. Chemotherapy was halted 2 months before presentation when an enterovaginovesical fistula was repaired, and chronic therapy with oral trimethoprim-sulfamethoxazole was started. Two weeks before presentation, blood could no longer be withdrawn from the Groshong catheter, which had been periodically flushed at a local clinic. No more attempts were made to flush the catheter until the patient presented for reinitiation of chemotherapy. At that time a cloudy brown liquid was withdrawn from the proximal port and sent for culture. During flushing of the proximal port the following day, the patient had an immediate onset of rigors with presyncopal symptoms, tachypnea, tachycardia, and profound hypotension. Physical examination was unremarkable. Peripheral therapy with intravenous vancomycin was initiated, and her symptoms quickly abated with fluid resuscitation.

Cultures of the brown liquid, initial peripheral blood specimens, and blood specimens drawn from the Groshong catheter after vancomycin therapy that were inoculated into blood agar yielded yellow colonies with long, filamentous, branching gram-positive rods identified as *O. xanthineolytica*. The organism was β-lactamase-positive and was found to be resistant to penicillin, erythromycin, oxacillin, cephalothin, clindamycin, ciprofloxacin, norfloxacin, ampicillin/sulbactam, amoxicillin/clavulanate, amikacin, tobramycin, nitrofurantoin, gentamicin, aztreonam, tetracycline, and cefazidime. The organism was susceptible to vancomycin, trimethoprim-sulfamethoxazole, ticarcillin, and only high concentrations of piperacillin and ceftiraxone. All subsequent blood cultures during the patient’s 14-day course of vancomycin therapy (1 g twice daily) as well as 1 month after completion of antibiotic treatment revealed no organisms.

Discussion

Oerskovia species are ubiquitous, non-spore-forming, gram-positive bacilli. This group of organisms, with the exception of Corynebacterium diptheriae, possesses a low tendency for pathogenicity. Orskov first isolated this yellow-pigmented branching organism from the soil in 1938 [9]. It was first described as a *Nocardia*-like bacterium, and in 1954 Erickson designated the organism as *Nocardia turbata* [9]. On the basis of its ability to fragment into motile rods, its lack of aerial mycelia, and the presence of large amounts of galactose in the cell wall, the organism was distinguished from *N. turbata* as *Oerskovia turbata* by Prauser et al. in 1970 [10]. A second
species, *O. xanthineolytica*, was identified 2 years later by its ability to degrade xanthine and hypoxanthine [11]. Although clinical isolates have been described since 1957 [1], to our knowledge only seven cases of clinical infection have been reported in the literature [2–8]. Sottneck et al. [1] reviewed 35 diphtheroids submitted to the Centers for Disease Control over a 20-year period before 1977 [1]. Of these diphtheroids five *O. turbata* isolates were obtained from heart valves or heart tissue and eight *O. xanthineolytica* isolates were obtained from blood. The remaining isolates were recovered from urine, CSF, sputum, lung tissue, a liver biopsy specimen, tear ducts, eye discharge, a right ethmoidal sinus, and superficial wounds; however, their role in producing clinical disease was not described. In the previously reported cases of *Oerskovia* infection, infection sites included CSF [5], a kidney [3], an aortic homograft valve [2], peritoneal fluid [8], an eye [4], and blood [6, 7].

The association of infection with immunocompromised patients and foreign-body portals of entry is well established (table 1). In the case of a man with ankylosing spondylitis who required chronic glucocorticoid therapy, a preoperative culture of a scraping from the engrafted valve was negative after a 50-day incubation in nystatin, penicillin, polymyxin B, and streptomycin. However, the isolation of *Oerskovia* from seven previously discarded valves from the same medical center implies that the valve in this patient was the probable source of infection despite incubation in three antibacterial agents that probably did not adequately sterilize the valve before engraftment [2]. A 40-year-old woman with total parenteral nutrition (TPN)—dependent Crohn’s disease and documented *Oerskovia* bacteremia required readmission because of recurrent fevers that occurred after home TPN was reinitiated; TPN was later proved to be the source of her infection [7].

In the case of *O. xanthineolytica* peritonitis in a man with peritoneal dialysis—dependent renal failure, culture of the removed catheter tip was positive [8]. Although the patient with *Oerskovia* endophthalmitis did not have a chronic indwelling device, he had received a recent foreign-body injury to that eye [4]. The only case that did not appear to be associated with either a foreign body or an immunocompromised state was that of an otherwise healthy woman with *Oerskovia* pyonephrosis whose source of infection was never identified [3]. In our case the central venous catheter was clearly established as the source. We hypothesize that the patient’s chronic treatment with trimethoprim-sulfamethoxazole had prevented bacteremia until a forced injection of a high load of organisms. On the basis of the reported cases, *Oerskovia* species should be considered pathogenic in all cases of foreign-body-associated infection in immunocompromised hosts.

In general, *Oerskovia* species, like most ubiquitous organisms in nature, appear to have high resistance to antibiotics [2–8]. The organism isolated in our case proved to be suscepti-

### Table 1. Summary of data from cases of *Oerskovia* infection.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex/Immunocompromising state</th>
<th>Organism</th>
<th>Infection</th>
<th>Antibiotic therapy (route)</th>
<th>Source removal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 [2]</td>
<td>68/M Glucocorticoid therapy</td>
<td><em>Oerskovia turbata</em></td>
<td>Aortic homograft valve endocarditis</td>
<td>Pen (iv/im), Amp (iv), Em (iv), TMP-SMZ (po), Amox (po)</td>
<td>Yes</td>
</tr>
<tr>
<td>2 [3]</td>
<td>47/F None</td>
<td>Nonmotile <em>Oerskovia</em> species</td>
<td>Pyonephrosis</td>
<td>None</td>
<td>Yes</td>
</tr>
<tr>
<td>3 [4]</td>
<td>47/M Glucocorticoid therapy</td>
<td><em>Oerskovia xanthineolytica</em></td>
<td>Foreign-body-associated endophthalmitis</td>
<td>Pen (iv), Clex (po)</td>
<td>Yes</td>
</tr>
<tr>
<td>4 [5]</td>
<td>38/F None</td>
<td><em>O. xanthineolytica</em></td>
<td>Ventricularperitoneal shunt—associated peritonitis</td>
<td>Pen (iv), Rif (po)</td>
<td>Yes</td>
</tr>
<tr>
<td>5 [6]</td>
<td>3/M Leukemia and neutropenia</td>
<td><em>O. turbata</em></td>
<td>Broviac catheter—associated bacteremia</td>
<td>Amik (iv*)</td>
<td>Yes</td>
</tr>
<tr>
<td>6 [7]</td>
<td>40/F Crohn’s disease</td>
<td><em>Oerskovia species</em>¹</td>
<td>Hickman catheter—associated bacteremia</td>
<td>Vm (iv*)</td>
<td>No</td>
</tr>
<tr>
<td>7 [8]</td>
<td>70/M Renal failure</td>
<td><em>O. xanthineolytica</em></td>
<td>Peritoneal catheter—associated peritonitis</td>
<td>Vm (iv/ip), Gm (iv/ip)</td>
<td>Yes</td>
</tr>
<tr>
<td>8 [PR]</td>
<td>49/F Colon cancer</td>
<td><em>O. xanthineolytica</em></td>
<td>Groshung catheter—associated bacteremia</td>
<td>Vm (iv²)</td>
<td>No</td>
</tr>
</tbody>
</table>

*NOTE. Amik = amikacin; Amox = amoxicillin; Amp = ampicillin; Clex = cephalaxin; Em = erythromycin; Gm = gentamicin; Pen = penicillin; PR = present report; Rif = rifampin; TMP-SMZ = trimethoprim-sulfamethoxazole; Vm = vancomycin.*

* Unclear whether peripheral or central venous catheter.

¹ Not identified to the species level.

² Central venous catheter.
ble to only vancomycin, trimethoprim-sulfamethoxazole, and ticarcillin. Although our patient was receiving chronic therapy with trimethoprim-sulfamethoxazole, sterilization of the catheter lumen would not be expected with oral administration, and treatment with intravenous vancomycin was required to clear the infection. Vancomycin and trimethoprim-sulfamethoxazole appear to be the only antibiotics to which the organism has been consistently susceptible [2-8] and are, therefore, recommended as the antibiotic treatments of choice.

Although all patients described to date have survived oerskovia infection, response to antibiotic therapy has been variable. Removal of the access device was not required in only one previous case [7]. Though intrathecal antibiotics were not administered in the case of ventriculoperitoneal shunt-associated meningitis, the authors speculated that a lack of response to antibiotics to which the organism was highly susceptible in vitro was likely due to poor penetration of the CSF [5]. In the case of Broviac catheter-associated infection due to _O. turbata_, the organism was susceptible to amikacin; however, persistently positive blood cultures necessitated removal of the catheter [6].

Even though the patient with oerskovia peritonitis was treated with both intravenous and intraperitoneal vancomycin and gentamicin, persistently positive cultures eventually required removal of the peritoneal catheter [8]. The efficacy of two intraperitoneal treatments over a 7-day period in a patient undergoing 2-L peritoneal exchange dialysis four times daily was likely to be low. Our patient responded to a 14-day course of intravenous vancomycin administered via alternating ports of a Groshong catheter. Although concern for seeding of the indwelling left subclavian Port-a-cath was high, blood cultures remained negative 1 month after treatment. A course of antibiotics to which the organism is susceptible that are administered through the foreign-body device to allow for prolonged antibiotic contact with the device would therefore seem a reasonable approach to treatment of oerskovia infection before removal of the device is considered.

Our case illustrates the isolation of _Oerskovia_ species and the successful treatment of oerskovia infection involving sterile sites in immunocompromised patients with indwelling foreign-body access devices. In some cases the presence of these organisms may be incorrectly attributed to contamination by diphtheroids if microbiological identification is incomplete. Suspicion for oerskovia infection in this patient population must therefore remain high when gram-positive rods are isolated. Until results of antibiotic susceptibility testing become available, a trial of treatment with either vancomycin or trimethoprim-sulfamethoxazole through the foreign-body device is recommended before removal of the device, as the patients are frequently dependent on their access devices.

References