

Pneumonia and Bacteremia Due to *Kytococcus schroeteri*

Ola Blennow,^a Katarina Westling,^a Inga Fröding,^b and Volkan Özenci^b

Department of Medicine, Division of Infectious Diseases,^a and Division of Clinical Microbiology,^b Karolinska Institutet, Karolinska University Hospital, Huddinge, Stockholm, Sweden

***Kytococcus schroeteri*, a saprophyte of the human skin, may cause serious infections in the immunocompromised host. Here, we describe a case of pneumonia and bacteremia due to *Kytococcus schroeteri* in an immunocompromised patient, successfully treated with linezolid and trimethoprim-sulfamethoxazole.**

CASE REPORT

A 43-year-old woman diagnosed with acute myeloid leukemia (AML) received induction therapy with daunorubicin and cytarabine with poor response and was given a new induction therapy with fludara, cytarabine, and idarubicin. She was also treated with flucloxacillin and ciprofloxacin due to a breast abscess. Ten days later, the patient was readmitted to hospital because of neutropenic fever. Chest X-ray showed a small infiltrate in the right lung, and antibiotic therapy with piperacillin-tazobactam was initiated. A chest computed tomography (CT) scan performed 4 days later showed three small, dense infiltrates in the middle and lower lobe in the right lung. Bronchoscopy with bronchoalveolar lavage (BAL) was performed, and the patient was treated with voriconazole, which later was switched to liposomal amphotericin B when fungal cultures of BAL fluid and galactomannan antigen were negative. Blood cultures, taken the day after bronchoscopy was performed, showed growth of a *Kytococcus* species in one aerobic blood culture vial out of a total of four blood culture vials, two aerobic and two anaerobic. Susceptibility testing by Etest showed that the strain had low MIC values for vancomycin, meropenem, linezolid, and trimethoprim-sulfamethoxazole. Despite concerns regarding the clinical significance of the isolate, as it was only recovered in one blood culture vial, the patient was treated with vancomycin, and piperacillin-tazobactam was switched to meropenem. The BAL fluid culture yielded a pure growth of 10^5 CFU/ml of the same *Kytococcus* species. The isolate was later identified, using sequencing, as *Kytococcus schroeteri*. The patient continued to deteriorate, and a new chest CT scan showed large, dense infiltrates in both lungs with necrosis in the middle lobe. Due to therapeutic failure and to reach better antibiotic concentrations in the lungs, vancomycin was switched to linezolid, and later, trimethoprim-sulfamethoxazole was added when the patient continued to deteriorate. Finally, 19 days after the BAL was performed, the leucopenia resolved and the patient improved. She was discharged with long-term treatment with trimethoprim-sulfamethoxazole, and 4 months later, allogeneic stem cell transplantation was successfully performed.

Blood samples were cultured using the BacT/Alert 3D (bioMérieux, Inc., Durham, NC) automated blood culture system. After 48 h of incubation, one aerobic blood culture vial signaled positive. Gram staining revealed Gram-positive cocci occurring in pairs and in tetrads. Broth from the aerobic bottle was subcultured onto blood and CLED (cystine-lactose-electrolyte-deficient) agar

plates incubated in air, chocolate agar plates incubated in 5% CO₂, and blood agar plates incubated in an anaerobic jar. Incubation at 37°C yielded growth of tiny colonies on the plates incubated in air and in CO₂ after 24 h. After 48 h, the colonies on blood and chocolate agar were convex, smooth, nonhemolytic, and muddy yellow in color. The colonies on CLED agar plates were tiny and yellow. Culture of the BAL fluid, taken 1 day before the blood cultures, yielded pure growth of Gram-positive cocci occurring in pairs and in tetrads. The colonies were similar to the ones from the positive blood culture. The bacteria were then tested for identification and antibiotic susceptibility.

The isolates were identified, using the Vitek 2 system (bioMérieux), as *Dermacoccus nishinomiyaensis*/*Kytococcus sedentarius* with 95% probability. 16S rRNA PCR and sequencing were performed with the isolate as described previously (7). In the present study, sequencing of both strands was carried out using an ABI Prism BigDye terminator v3.1 cycle sequencing kit (Applied Biosystems, Foster City, CA) and 1 μM primers (5'-AGAGTTTGAT CMTGGCTCAG-3' and 5'-CCGTC AATTCATTTGAGTTT-3', respectively) on a GeneAmp 9700 thermocycler (Applied Biosystems). A BLAST search (1) showed 100% nucleotide identity to previously registered sequences of the 16S rRNA gene of *K. schroeteri* (834/834 bases).

Biochemical tests showed that the isolate was catalase positive (with 3% hydrogen peroxide). When tested, activity for alpha-D-glucosidase was negative, activity for arginine dehydrolase was positive, and hydrolysis of Tween 80 was positive, further supporting the determination that the isolate was *K. schroeteri*.

The anaerobic blood culture vials and the second aerobic vial remained negative until the end of the culture period. The reason behind the negative blood culture vials is unclear. It might be due to a small amount of bacteria in the circulation at the time of sampling.

There are no clinical breakpoints established by CLSI or EUCAST for *Kytococcus* spp. Although the authors of several previous case reports on *Kytococcus schroeteri* have used the clinical breakpoints for staphylococci for susceptibility interpretation (4–6, 8), these breakpoints are not valid for *Kytococcus* spp. Anti-

Received 22 June 2011 Returned for modification 7 August 2011

Accepted 28 November 2011

Published ahead of print 7 December 2011

Address correspondence to Ola Blennow, ola.blennow@karolinska.se.

Copyright © 2012, American Society for Microbiology. All Rights Reserved.

doi:10.1128/JCM.01245-11

TABLE 1 Antimicrobial susceptibility patterns of the present and previously published *K. schroeteri* isolates

| Antibiotic | MIC (mg/liter) for ^a : | | | | |
|-------------------------------|-----------------------------------|-------------------------------|---------------------|-----------------|--------------------|
| | Present isolate | Previous isolate reported by: | | | |
| | | Jacquier et al. (4) | Jourdain et al. (5) | Mnif et al. (8) | Le Brun et al. (6) |
| Penicillin G | 8 | | >32 | >256 | |
| Oxacillin | >256 | 1 | >32 | >256 | |
| Piperacillin-tazobactam | 128 | | | | |
| Cefotaxime | >256 | | | >256 | |
| Ceftriaxone | >256 | | >256 | | |
| Imipenem | 1 | | | 0.5 | 0.25 |
| Meropenem | 0.5 | | 0.38 | | |
| Linezolid | 1.0 | 0.19 | | | 0.25 |
| Gentamicin | 0.5 | 0.75 | 0.5 | | 1 |
| Rifampin | 0.008 | 0.06 | | | <0.002 |
| Vancomycin | 0.25 | 0.5 | 1 | 0.25 | 0.125 |
| Teicoplanin | 0.125 | 0.38 | 0.38 | 0.25 | 0.06 |
| Trimethoprim-sulfamethoxazole | 0.25 | >32 | | | |
| Trimethoprim | | | | | 0.25 |
| Levofloxacin | 0.5 | 0.5 | | | |
| Moxifloxacin | 0.125 | 0.032 | | | 0.05 |
| Ofloxacin | 1 | 1 | | | |
| Ciprofloxacin | 2 | | 0.75 | | 8 |
| Daptomycin | 1 | 1 | | | |
| Tigecycline | 0.5 | 0.5 | | | |
| Erythromycin | | | 12 | 0.5 | |

^a MICs were obtained by Etest (bioMérieux) in all cases.

biotic susceptibility testing of the present isolate was carried out by Etest (bioMérieux, Inc., Durham, NC) on Mueller-Hinton agar, and the results for 19 antibiotics are shown in Table 1. Low MICs were observed for linezolid, trimethoprim-sulfamethoxazole, vancomycin, meropenem, gentamicin, and rifampin.

K. schroeteri is a recently defined member of the genus *Kytococcus* (1) that is a normal saprophyte of the human skin. However, *K. schroeteri* has been reported as a cause of infective endocarditis, mainly in patients with prosthetic valves (2, 10), and of spondylodiscitis (4). In immunocompromised patients, three cases of *Kytococcus schroeteri* pneumonia with growth in BAL fluid have been reported. One patient had been receiving maintenance therapy with 20 mg/day prednisone due to asthma bronchiale (9), and two patients had AML (3). The outcome in all three cases was fatal. In the first reported case (9), the patient was given ceftriaxone and ofloxacin as antibiotic therapy, and the etiological diagnosis was found postmortem. The two patients with AML (3) were both initially given empirical antibiotic therapy with ceftazidime and vancomycin for neutropenic fever. After blood cultures showed growth of Gram-positive cocci, rifampin was added. In both cases, the patient died despite intensive care.

In the present case, the patient was initially treated with vancomycin, but when the lung function deteriorated, vancomycin was switched to linezolid 6 days after the blood culture became positive. As the neutropenia resolved 7 days later, the patient deteriorated, and trimethoprim-sulfamethoxazole was added. In retrospect, the last deterioration may have been due to the immunoreconstitution, and it is questionable if trimethoprim-sulfamethoxazole really was needed. There was some concern about combining two drugs with known myelotoxicity, linezolid

and trimethoprim-sulfamethoxazole, but the patient's bone marrow function recovered as expected.

There are published antibiotic susceptibility patterns in four cases of *K. schroeteri* infections: two with endocarditis, one with spondylodiscitis, and one with a shunt infection (4–6, 8). The present isolate had antibiotic susceptibility patterns similar to those in previously published data (Table 1). All *K. schroeteri* isolates for which data were published had low MICs against linezolid, vancomycin, rifampin, imipenem, meropenem, and trimethoprim-sulfamethoxazole. In contrast, high MIC levels were observed for oxacillin, piperacillin-tazobactam, cefotaxime, and ceftriaxone (3).

In conclusion, we report a case of pneumonia and bacteremia due to *Kytococcus schroeteri* in a patient with AML that was successfully treated with linezolid and trimethoprim-sulfamethoxazole. The hitherto-published reports in the same patient group were all related to fatal outcomes. The reasons for the high mortality due to invasive *K. schroeteri* infections are still unknown. Further studies investigating the immune response in affected patients and the virulence factors associated with *K. schroeteri* are warranted.

ACKNOWLEDGMENT

We are particularly grateful to Stina Boräng for molecular methods.

REFERENCES

1. Becker K, et al. 2002. *Kytococcus schroeteri* sp. nov., a novel Gram-positive actinobacterium isolated from a human clinical source. *Int. J. Syst. Evol. Microbiol.* 52:1609–1614.
2. Becker K, et al. 2003. Prosthetic valve endocarditis due to *Kytococcus schroeteri*. *Emerg. Infect. Dis.* 9:1493–1495.
3. Hodiament CJ, Huisman C, Spanjaard L, van Ketel RJ. 2010. *Kytococcus schroeteri* pneumonia in two patients with a hematological malignancy. *Infection* 38:138–140.
4. Jacquier H, et al. 2010. Postoperative spondylodiscitis due to *Kytococcus schroeteri* in a diabetic woman. *J. Med. Microbiol.* 59:127–129.

5. Jourdain S, et al. 2009. *Kytococcus schroeteri* infection of a ventriculo-peritoneal shunt in a child. *Int. J. Infect. Dis.* 13:e153–e155.
6. Le Brun C, Bouet J, Gautier P, Avril JL, Gaillot O. 2005. *Kytococcus schroeteri* endocarditis. *Emerg. Infect. Dis.* 11:179–180.
7. Liderot K, Larsson M, Borang S, Ozenci V. 2010. Polymicrobial bloodstream infection with *Eggerthella lenta* and *Desulfovibrio desulfuricans*. *J. Clin. Microbiol.* 48:3810–3812.
8. Mnif B, et al. 2006. Endocarditis due to *Kytococcus schroeteri*: case report and review of the literature. *J. Clin. Microbiol.* 44:1187–1189.
9. Mohammedi I, et al. 2005. Fatal *Kytococcus schroeteri* bacteremic pneumonia. *J. Infect.* 51:E11–E13.
10. Renvoise A, Roux V, Casalta JP, Thuny F, Riberi A. 2008. *Kytococcus schroeteri*, a rare agent of endocarditis. *Int. J. Infect. Dis.* 12: 223–227.