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Isolation of Two Strains of *Kingella kingae* Associated with Septic Arthritis

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Two new cases of infection, a presternal abscess and a spondylodiscitis caused by the recently classified bacterium *Kingella kingae*, are reported. The main bacteriological characteristics and the susceptibility of the two isolates to antimicrobial agents are described. The pathology of *K. kingae*, particularly among children, is reviewed.

Kingella kingae is an uncommon, short gram-negative rod known as an occasional resident of the rhinopharyngeal flora. It is an infrequent cause of septic arthritis, particularly in childhood (6). Its real frequency is probably unknown, perhaps because of its fastidious nutrition. The purpose of this paper is to report two new cases of septic infection (including arthritis) caused by *K. kingae*.

Case 1. A 9-month-old girl was hospitalized with a presternal, erythematous, and painless swelling measuring about 2.5 cm in diameter without any signs of trauma at the involved site. The only associated clinical sign was a discrete rhinitis. X rays of the sternum were normal. The abscess was incised, and the purulent material obtained was examined. No microorganisms were seen on the Gram- or Ziehl-Nielsen-stained smears of the aspirate. After 3 days of incubation, gram-negative rods were isolated, in addition to an anaerobic bacterium which was not identified. The gram-negative bacterium was identified as *K. kingae*, which was confirmed at the Institut Pasteur (H. H. Mollaret). This strain was susceptible by disk diffusion susceptibility testing to all the 17 antimicrobial agents tested, which included penicillin, ampicillin, carbenicillin, amoxycillin plus clavulanic acid (Augmentin), cephalothin, cefoxitin, cefotaxime, moxalactam, gentamicin, dibekacin, amikacin, erythromycin, rifampin, chloramphenicol, colistin, tetracycline, and co-trimoxazole. The patient was treated with only local surgical drainage without antimicrobial therapy. The abscess recurred 1 month later. It was treated with complete success by local application of a diamidine antiseptic.

Case 2. A 12-month-old boy refusing to walk and stand was admitted with rhinopharyngitis which had been developing for a month. A spondylodiscitis of the L5-S1 intervertebral disk was diagnosed 9 days later (postadmission) and radiologically confirmed. Laboratory investigations showed a total white blood cell count of 8,200/mm^3 (neutrophils, 13%; eosinophils, 2%; lymphocytes, 73%; and monocytes, 12%). The erythrocyte sedimentation rate was 30 mm/h. Needle aspiration of the L5-S1 space was performed. A sample of slightly sanguineous fluid was withdrawn from the disk space and cultured. No organisms were seen on the Gram-stained smears. Later (2 days), a gram-negative diplobacillus was isolated in a pure culture and identified as *K. kingae*, which was confirmed by the Institut Pasteur. The strain was also susceptible to all 17 antimicrobial agents tested. The patient was treated with intravenous moxalactam (150 mg/kg per day) and intravenous netilmicin (60 mg/day). This therapy was maintained for 7 days, followed by orally administered cefaclor (100 mg/kg per day) for 2 months. The patient was immobilized. Reexamination 5 months later revealed the patient to be asymptomatic. The sedimentation rate was 8 mm/h. New X rays demonstrated persistent disk space narrowing.

Our strains were isolated on 5% (vol/vol) horse blood agar (Bio-Mérieux, Lyon, France). Like those previously isolated by other researchers (8), the strains were catalase negative and oxidase positive and required serum for growth (horse serum, 5% [vol/vol]; Institut Pasteur Production, Paris, France). One of the two strains was nitrite reductase positive and microaerophilic. Both colonies were surrounded by distinct zones of β-hemolysis. Slight acid was produced on cystine trypticase agar (Bio-Mérieux) with 5% horse serum from glucose, galactose, and maltose (76% of *K. kingae* isolates were maltose positive). Casein was hydrolyzed. All the strains tested by standard methods on Mueller-Hinton agar (Institut Pasteur Production) with 5% horse blood were highly susceptible to β-lactam antibiotics, aminoglycosides, chloramphenicol, tetracyclines, erythromycin, and co-trimoxazole.

K. kingae, individualized by Henriksen and Bøvre in 1976 (5) and originally named *Moraxella kingii*, is a gram-negative diplobacillus that usually produces β-hemolysis, hydrolyzes casein, ferments maltose, and is nitrite reductase negative. These four characteristics distinguish *K. kingae* from *Kingella denitrificans*. However, one of our isolates was nitrite reductase positive (usually only 3% positive).

K. kingae colonizes human mucous membranes. Strains have been isolated from the throat, nose, blood (endocarditis) (2), bone lesions, and joints. As often as not, only an infection of the upper respiratory tract is found in anamnesis (4). In one case, infection resulted from immunodepressive therapy (7). This bacterium can cause mild or severe infections: in 1983, Powell and Bass reported nine cases of septic arthritis (6). We have described two additional cases here to point out the importance of this species for arthritis (1) or para-osseous tumors (3, 9), particularly among children (there was only one adult among the nine cases reported by Powell and Bass). The disease evolves in a subacute manner, with neither great deterioration of the general state nor with direct evidence of visible organisms (Gram or Ziehl-Nielsen stain). The treatment is simple, as this bacterium is still susceptible to most antibiotics.

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LITERATURE CITED

1. **Bosworth, D. E.** 1983. *Kingella* (*Moraxella*) *kingae* infections in children. Am. J. Dis. Child. **137**:650–653.
2. **Christensen, C. E., and G. C. Emmanouilides.** 1967. Bacterial endocarditis due to "*Moraxella* new species I." N. Eng. J. Med. **277**:803—804.
3. **Davis, J. M., and M. M. Pell.** 1982. Osteomyelitis and septic arthritis caused by *Kingella kingae*. J. Clin. Pathol. **35**:219–222.
4. **Gay, R. M., T. W. Lane, and D. C. Keller.** 1983. Septic arthritis caused by *Kingella kingae*. J. Clin. Microbiol. **17**:168–169.
5. **Henriksen, S. D., and K. Bøvre.** 1976. Transfer of *Moraxella kingae* Henriksen and Bøvre to the genus *Kingella*. gen. nov. in the family *Neisseriaceae*. Int. J. Syst. Bact. **26**:447–450.
6. **Powell, J. M., and J. W. Bass.** 1983. Septic arthritis caused by *Kingella kingae*. Am. J. Dis. Child. **137**:974–976.
7. **Redfield, D. C., G. D. Overturf, N. Ewing, and D. Powars.** 1980. Bacteremia, arthritis and skin lesions due to *Kingella kingae*. Arch. Dis. Child. **55**:411.
8. **Snell, J. J. S., and S. P. Lapage.** 1976. Transfer of some saccharolytic *Moraxella* species to *Kingella* Henriksen and Bøvre 1976, with descriptions of *Kingella indologenes* sp. nov. and *Kingella denitrificans* sp. nov. Int. J. Syst. Bact. **26**:451–458.
9. **Wong, A. S., J. Dyke, D. Perry, and D. C. Anderson.** 1978. Paraspinal mass associated with intervertebral disk infection secondary to *Moraxella kingii*. J. Pediatr. **92**:86–88.