Kingella kingae Intervertebral Disk Infection

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Disk inflammation in children is believed to result from infection, and Staphylococcus aureus is reported to
be the organism most commonly isolated from cases of intervertebral disk infection. A case of disk
inflammation caused by the unusual pathogen Kingella kingae is described. The antibiotic susceptibility of other
K. kingae isolates and the clinical features of 11 other previously reported cases of disk infection caused by this
microorganism are reviewed.

Disk inflammation in children is believed to result from infection, although cultures from closed and open biopsies
yield bacterial growth in only approximately 50% of cases (33). Staphylococcus aureus is reported to be the organism
most commonly isolated from cases of intervertebral disk infection (1, 18, 20, 37). The purpose of this report is to
describe a case of disk inflammation caused by the unusual pathogen Kingella kingae. We wish to call attention to the
clindamycin and vancomycin resistance of the isolate from our patient and also to the involvement of multiple disk
spaces. The antibiotic susceptibility of other K. kingae isolates, the frequency of K. kingae as a cause of disk
infection, and the clinical features of 11 other previously reported cases of disk infection caused by this microorganism
are reviewed.

Case report. An 18-month-old female was hospitalized with a 4-day history of progressive difficulty in walking. There
was no history of trauma. Two months prior to admission, she had been treated with amoxicillin for a skin
infection that was thought to be impetigo. She was seen again 1 week prior to admission with a complaint of consti-
pation. Four days prior to admission, the patient started walking with an exaggerated lumbar lordosis. This problem
progressed to walking with a “tilt” to the left and eventually a refusal to walk. The only associated problem was
irritability. There was no report of fever.

On initial examination, the axillary temperature was 37.5°C. The child was irritable and refused to walk. The patellar
reflex on the right side was slightly decreased compared with the left. The remainder of the neurologic examination
was unremarkable. There was no tenderness over the spine.

Laboratory studies included a hemoglobin value of 11.2 g/dl and a total leukocyte count of 9,800/mm³, with 1% band
cells, 38% neutrophils, 60% lymphocytes, and 1% eosinophils. The erythrocyte sedimentation rate was 45 mm/h. A
purified protein derivative skin test was negative at 48 h, with a positive Multitest-CMI control (Merieux
Institute, Inc., Miami, Fla.).

Radiographs of the spine revealed narrowed disk spaces between T₁₁ and T₁₂ and between L₁ and L₂, with irregularities of
the adjacent vertebral cortical margins. A bone scan with Tc-99m showed increased uptake only at the L₁-L₂
interspace. Computerized tomography of the involved area showed a prevertebral mass extending from T₁₁ to L₂ with
bony destruction of the vertebral bodies of T₁₁, T₁₂, L₁, and L₂. Because of the large prevertebral mass and the unusual
involvement of two noncontiguous disk spaces, a needle biopsy of the paraspinous mass was performed under direct
visualization by using biplanar fluoroscopy. Material from the aspirate was submitted for pathologic examination and
revealed an acute inflammatory response. Gram stains revealed many polymorphonuclear leukocytes, but no organ-
isms were seen. Stains for fungi and acid-fast bacilli were negative.

Blood cultures obtained at the time of admission were negative. After 3 days, cultures of material aspirated from the
paraspinal mass grew a few colonies of gram-negative coccobacilli on the chocolate agar plate only. These were
denoted as K. kingae in our laboratory, with confirmation by the Missouri State Laboratory. This bacterium presents
relatively small, usually smooth and hemolytic colonies on 24-h blood agar plates. Additional features are tabulated by
Weaver et al. (36). Disk diffusion tests for antibiotic suscepti-
bility were performed on Mueller-Hinton chocolate agar;
the organism was susceptible to penicillin, ampicillin, ticar-
cillin, cephatholin, cefuroxime, cefotaxime, gentamicin,
chloramphenicol, erythromycin, and trimethoprim-sulfa-
methoxazole; it was resistant to oxacillin and clindamycin.

However, tube dilution susceptibility testing (kindly per-
formed by J. M. Swenson at the Centers for Disease Control,
Atlanta, Ga.) revealed that the MIC of oxacillin was 1.0
μg/ml, a value that categorizes the organism as susceptible.
Results of tube dilution susceptibility tests with the other
antimicrobial agents correlated with their respective disk
diffusion results. The MIC of vancomycin was ≥32 μg/ml.

The patient was immobilized with a brace, and therapy
was initiated with intravenous oxacillin. After the culture
results became available, ampicillin was added to the therapeu-
tic regimen.

A repeat computerized tomography scan performed 10
days after initiation of therapy showed a slight decrease in
the size of the paraspinal mass. Patellar reflexes were
symmetrical. The erythrocyte sedimentation rate decreased
to 19 mm/h after 2 weeks of treatment. The child was treated
for 8 weeks with parenteral antibiotics. Follow-up evalua-
tion, 17 months later, revealed the patient to be asympto-
matic. Follow-up radiographs at that time demonstrated
reconstitution of the T₁₁-T₁₂ disk space with a prominent
residual bony defect along the anterosuperior margin of the
T₁₂ vertebra. The L₁-L₂ disk space was completely recon-

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TABLE 1. Disk infection caused by *K. kingae*—clinical data

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (mo)</th>
<th>Sex</th>
<th>Level of disk inflammation</th>
<th>ESRb (mm/hr)</th>
<th>Temp maximum (°C)</th>
<th>Leukocyte count</th>
<th>Duration of symptoms before diagnosis (days)</th>
<th>Antibiotic treatment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22</td>
<td>F</td>
<td>L4-L5</td>
<td>46</td>
<td>39.0</td>
<td>10,000</td>
<td>18</td>
<td>Ampicillin, dicloxacillin</td>
<td>32</td>
</tr>
<tr>
<td>2</td>
<td>22</td>
<td>F</td>
<td>T11-T12</td>
<td>65</td>
<td>39.0</td>
<td>14,200</td>
<td>2</td>
<td>Methicillin</td>
<td>10</td>
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<tr>
<td>3</td>
<td>48</td>
<td>F</td>
<td>L2-L3</td>
<td>65</td>
<td>39.0</td>
<td>10,900</td>
<td>60</td>
<td>Cefuroxime, penicillin</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>60</td>
<td>F</td>
<td>L4-L5</td>
<td>40</td>
<td>38.0</td>
<td>9,600</td>
<td>15</td>
<td>Gentamicin, TMP-SMXc</td>
<td>9</td>
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<tr>
<td>5</td>
<td>15</td>
<td>M</td>
<td>T11-T12</td>
<td>32</td>
<td>38.0</td>
<td>14,400</td>
<td>15</td>
<td>Naftilcin, ampicillin</td>
<td>30</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
<td>M</td>
<td>L5-S1</td>
<td>30</td>
<td>Normal</td>
<td>8,200</td>
<td>10</td>
<td>Moxalactam, netilmicin</td>
<td>25</td>
</tr>
<tr>
<td>7</td>
<td>36</td>
<td>F</td>
<td>L1-L2</td>
<td>33</td>
<td>Normal</td>
<td>12,800</td>
<td>21</td>
<td>Cefotaxime, netilmicin</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>18</td>
<td>M</td>
<td>L5-S1</td>
<td>42</td>
<td>Normal</td>
<td>11,500</td>
<td>15</td>
<td>Oxacillin, aminglycoside</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>24</td>
<td>F</td>
<td>L7-L4</td>
<td>57</td>
<td>Normal</td>
<td>10,700</td>
<td>10</td>
<td>Ampicillin, gentamicin</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>60</td>
<td>F</td>
<td>L7-L4</td>
<td>50</td>
<td>Normal</td>
<td>8,800</td>
<td>10</td>
<td>Oxacillin, aminglycoside</td>
<td>5</td>
</tr>
<tr>
<td>11</td>
<td>18</td>
<td>F</td>
<td>L7-L2</td>
<td>45</td>
<td>37.5</td>
<td>9,800</td>
<td>4, 7d</td>
<td>Oxacillin, ampicillin</td>
<td>This report</td>
</tr>
<tr>
<td>Present case</td>
<td>18</td>
<td>F</td>
<td>L7-T11-L12</td>
<td>45</td>
<td>37.5</td>
<td>9,800</td>
<td>4, 7d</td>
<td>Oxacillin, ampicillin</td>
<td>This report</td>
</tr>
</tbody>
</table>

a F: Female; M: male.
b ESR: Erythrocyte sedimentation rate.
c TMP-SMX: Trimethoprim-sulfamethoxazole.
d Four days of difficulty in walking; seven days of constipation.

Literature review. *K. kingae* has been reported with increasing frequency as a cause of bone and joint infections, bacteremia, and endocarditis. When it was first identified as a unique species in 1966 by E. O. King, it was classified as *Moraxella* new species 1 (M-1), in the family Neisseriaceae. It was later renamed *Moraxella kingii* by Henriksen and Bøvre (15). In 1976, because of its distinctive genetic and biochemical characteristics, it was allocated to a new genus *Kingella* as *K. kingae* (16). The organism is a fastidious, slowly growing, short gram-negative rod.

At least 11 cases of *K. kingae* disk space infection have been reported previously (Table 1). Probably the earliest reported case was included in the report of Spiegel et al. (32) that described 48 children with intervertebral disk space inflammation. Fifteen biopsies were performed, and of the four that were positive, one identified a *Moraxella* species. Ten additional cases of *K. kingae* disk infection have been reported in the last 12 years (3–5, 9, 10, 25, 39, 40). The ages of the affected children ranged from 12 to 60 months. None of the children had underlying illnesses. Eight cases were in females, and three cases were in males. Fever was observed in five of seven children. When measured, the erythrocyte sedimentation rate was elevated, and the peripheral leukocyte count was also usually increased. The lower thoracic and lumbar disk spaces were most frequently affected. All of the children were treated with antibiotics, and the outcome was uniformly favorable. In all eight of the cases for which follow-up information was available (7 weeks to 36 months), the children were asymptomatic; the only radiologic evidence of any residual abnormality was disk space narrowing (seven of seven examined).

Antibiotic susceptibility. *K. kingae* is usually susceptible to the antibiotics commonly used in the treatment of bone and joint infections in children (2, 3, 5, 6, 14, 19, 21–27, 29, 39, 34). In previous studies, the MIC of penicillin was usually low (<0.24 μg/ml) (19, 23, 24). An important exception is oxacillin or clindamycin, to which only 31% of *K. kingae* isolates from clinical samples reported in the literature were susceptible (Table 2). Importantly, a few strains were also resistant to other antibiotics such as oxacillin (2 of 34), erythromycin (5 of 51), and trimethoprim-sulfamethoxazole (7 of 77). Emergence of resistance to ciprofloxacin was reported in one case (13). Most of these data are based on the results of disk diffusion tests, which are not standardized for fastidious microorganisms and may be unreliable, as was the case for the oxacillin test on our isolate.

**Discussion.** *K. kingae* infections occur most frequently in children, in whom they cause osteomyelitis, septic arthritis, and, less often, endocarditis and bacteremia (7, 12). Acute disk inflammation with *K. kingae* has been reported only for children (3–5, 9, 10, 25, 32, 39, 40). Since the organism is occasionally found in the human respiratory tract, infection is thought to result from hematogenous spread from the nasopharynx (2).

Although Waldvogel and Vasey recommend biopsy in every case of disk inflammation (35), in practice only a small percentage of children with disk inflammation have had direct cultures of the disk space performed. Because *S. aureus* is the organism most frequently isolated from these cultures, initiation of empiric therapy with an antistaphylococcal agent has been suggested. Most authors recommend

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>No. of isolates</th>
<th>% of isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin</td>
<td>58</td>
<td>100</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>58</td>
<td>100</td>
</tr>
<tr>
<td>Oxacillin</td>
<td>34</td>
<td>94</td>
</tr>
<tr>
<td>Aminoglycoside</td>
<td>58</td>
<td>100</td>
</tr>
<tr>
<td>Cephalosporin</td>
<td>38</td>
<td>100</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>45</td>
<td>100</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>41</td>
<td>100</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>51</td>
<td>90</td>
</tr>
<tr>
<td>Clindamycin or lincomycin</td>
<td>32</td>
<td>31</td>
</tr>
<tr>
<td>TMP-SMXc</td>
<td>77</td>
<td>91</td>
</tr>
<tr>
<td>Trimethoprim</td>
<td>13</td>
<td>0</td>
</tr>
</tbody>
</table>

* Data from references 5 and 14.

b TMP-SMX: Trimethoprim-sulfamethoxazole.
biopsy only in cases with unusual clinical findings or a poor response to therapy (1, 17, 28, 37).

In 10 reviews of disk inflammation in children (1, 8, 11, 17, 18, 20, 28, 31, 32, 37), cultures of material obtained by biopsy or aspiration of the disk space grew S. aureus in 27 of 63 cases; there was no growth from 28 of the cases. In three cases, diphtheroids were isolated, and Klebsiella species, Streptococcus pneumoniae, Staphylococcus species, beta-hemolytic streptococci, and Moraxella species (presumably K. kingae) were each recovered in one case. Ten additional cases of disk infection caused by K. kingae have been reported, either as single cases or in reviews of K. kingae infections. It is unlikely that these numbers reflect the true proportion of K. kingae in all cases of disk infection in children, since it is probable that there has been a bias toward the reporting of a more unusual organism. However, it is clear that K. kingae must now be recognized as an important cause of disk space infection in children. The apparent increase in frequency of reported cases of K. kingae infection may reflect improvement in diagnostic techniques to isolate and identify this organism. Many workers have noted the fastidious nature of the organism, its low growth rate, and its variable appearance on a Gram stain.

Although K. kingae is susceptible to most of the antibiotics commonly used for treatment of disk infection, the organism is usually resistant to clindamycin. In addition, our strain was resistant to vancomycin, and two other strains (5, 14) were resistant to oxacillin. Both clindamycin and oxacillin are commonly used for empiric therapy of bone or joint infection presumed to be caused by S. aureus. Vancomycin is an alternative in treatment failure or when a methicillin-resistant staphylococcal infection is suspected. The previously reported patient with disk inflammation caused by an oxacillin-resistant isolate of K. kingae (5) had her therapy changed from oxacillin to josamycine as soon as the results of susceptibility tests were known, and the patient enjoyed a favorable clinical outcome. Treatment failures have been reported for several patients with severe K. kingae infection caused by isolates that were resistant to the antibiotics used in their initial therapy (9, 14, 38). In at least one case, resistance was confirmed by a tube dilution test (14).

In conclusion, we report this case of disk inflammation caused by K. kingae to emphasize that empiric antistaphylococcal therapy initiated for treatment of disk inflammation in children may require adjustment if the patient does not respond satisfactorily. If it is not possible to obtain material for culture, a drug effective against K. kingae, such as penicillin or ampicillin, should be added.

REFERENCES


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