Invasive *Kingella kingae* Resulting in a Brodie Abscess

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*Kingella kingae* is an increasingly recognized cause of pediatric infections [1–6]. Once largely relegated as the last part of the “HACEK” acronym for what was originally termed “culture negative endocarditis,” the pathogen is identified as the causative agent for osteoarticular infections (OAI)s such as septic arthritis and osteomyelitis in addition to the more traditionally recognized endocarditis [7]. The diagnosis is challenging because the symptoms and laboratory studies are frequently unremarkable or nonspecific. Two cases were recently reported from France involving a soft tissue abscess as well as a femoral Brodie abscess following upper respiratory infections in children [8]. To our knowledge, we report the first North American case of a Brodie abscess caused by invasive *K kingae* infection.

CASE

The patient was a 14-month-old full-term male without any significant medical history who initially presented to the pediatric emergency department (PED) with a 5-day history of a limp. Preceding his gait abnormality, he had a brief upper respiratory infection with subjective fevers. As the respiratory symptoms resolved, he developed a left-sided limp that fluctuated in severity from barely noticeable to hardly being able to bear weight. At home, he had responded well to ibuprofen prior to his parents bringing him in for evaluation. Physical examination was unremarkable other than an antalgic gait. He had no tenderness in his knee or his hip, and he had a full range of motion on exam. In the emergency department, plain films of the lower extremity were unremarkable. The peripheral white blood cell count was 11 700/mm³, the erythrocyte sedimentation rate was 26 mm/hour, and the C-reactive protein was 1.4 mg/dL. Given these unremarkable laboratory results and imaging studies along with his improvement in the PED with ibuprofen, he was discharged home with the provisional diagnosis of toxic synovitis.

The patient had a fluctuating course over the subsequent days with the parents noting that he seemed to improve with limited activity and continued ibuprofen. At times, his parents thought the patient had symptom-free days, but on the 15th day of gait symptoms the patient was believed to have acute worsening of his limp and discomfort and the family returned to the PED for further testing. Repeat radiographs revealed a lucent area of the proximal tibia. The hips and femur were unremarkable (data not shown). Repeat peripheral white blood cell count was 11 300/mm², the erythrocyte sedimentation rate was 35 mm/hour, and the C-reactive protein was 0.8 mg/dL. The patient had a magnetic resonance imaging (MRI) of the left lower extremity that revealed an ill-defined area of proximal left tibial marrow edema with a central 9-mm rim-enhancing fluid collection, which was believed to have some minimal extension through the proximal physis and into the epiphysis with modest surrounding soft tissue edema and enhancement (Figure 1). The diagnosis of osteomyelitis with an organized abscess (Brodie abscess) was favored, but malignancy could not be excluded on the basis of radiologic imaging.

The patient was admitted to the hospital with orthopedic as well as oncology consultations. Because the patient was not acutely ill, empiric antibiotic therapy was
with held and the patient was taken to the operating room by the orthopedic service for incision and drainage of the lesion. The lesion was a well demarcated area of abscess in the lateral proximal tibia adjacent to and entering the periosteum of the growth plate. The abscess was easily removed surgically without complication. Involvement of the infectious disease service led to routine culture as well as inoculation into an aerobic blood culture bottle. Empiric coverage for presumed osteomyelitis was initiated postoperatively with clindamycin and ceftriaxone. Gram-negative rods in the aerobic bottle grew after approximately 24 hours, so antibiotic coverage was reduced to single coverage with ceftriaxone. The patient was discharged home on postoperative day 2 with a peripherally inserted central catheter for daily ceftriaxone administration. Identification of the pathogen proved to be difficult due to insufficient growth for speciation in the hospital laboratory, and colonies of the organism were sent to both the Texas Department of Health Services for enhanced growth and identification techniques at an outside laboratory for 16s ribosomal RNA sequencing. Both laboratories confirmed K. kingae. Of note, peripheral blood cultures were never sent with initial laboratory studies during the patient’s hospitalization.

The patient had an unremarkable recovery, and he completed 4 weeks of intravenous antibiotic therapy with ceftriaxone. Follow-up imaging almost 1 month postoperatively showed normal bone growth and no recurrence of the abscess (Figure 2). Cardiac echocardiography was negative for vegetative lesions, and follow-up laboratory studies showed normalization of the erythrocyte sedimentation rate and the C-reactive protein.

**DISCUSSION**

*K. kingae* has increasingly become recognized as an important cause of pediatric acute OAs, especially in younger children [1–6,9]. Some authors note that with the advent of improved diagnostic techniques, including polymerase chain reaction (PCR), *K. kingae* is likely the primary pathogen for OAs in young children. *K. kingae* inhabits the oropharyngeal flora of young children and can be transmitted between children, and studies have suggested that PCR testing for the bacteria in the oropharynx may increase the diagnostic yields for OAI infections [10]. One report of a daycare outbreak of *K. kingae* OAs related to a single strain of bacteria was successfully managed by oropharyngeal prophylaxis with oral rifampin [11].

A recent case report from France by Basmaci et al [8] was remarkably similar to our patient. In their 2 cases, rhinovirus infection was specifically identified. In the case of our patient, no specific viral etiology was identified, but he did have a definite preceding viral upper respiratory tract infection. The association of upper respiratory tract infection with invasive *K. kingae* infection has been noted previously, and it is hypothesized that viral damage to respiratory mucosa enables *K. kingae* invasion from the oropharynx to the blood and subsequent hematogenous spread to cause OAI [1]. Brodie abscesses have traditionally been associated with *Staphylococcus aureus* infections, which are typically considered more virulent infections.

**Figure 1.** Magnetic resonance imaging of the left lower extremity on August 26, 2012.

**Figure 2.** Follow-up film on September 24, 2012.
than *K. kingae* infections [12]. Although *K. kingae* infections are increasingly recognized as a source of OAI with improved imaging as well as bacteriological diagnostic techniques, it is not clear whether the natural history of the organism is one of an infection that typically clears itself before clinical recognition in most cases, whether the virulence is changing in a way that is leading to more clinically significant OAI infections, or whether enhanced awareness of the organism is leading to an increase in diagnoses. The widespread use and availability of higher-resolution MRI may allow detection of abscesses that would previously have gone unrecognized.

Much of the literature on the increasing prominence of *K. kingae* in OAI comes from Europe and Israel, and to our knowledge our case is the first North American report of a Brodie abscess caused by *K. kingae*. Detection of this fastidious organism is challenging, and lack of appreciation for the potential etiology may hamper diagnosis. Inoculation of sample material into an aerobic blood culture bottle is recommended to increase isolation of this fastidious organism; in the absence of this method, our patient may have been another “culture negative” OAI. Use of other techniques such as PCR of the purulent material or PCR of the oropharynx before surgery may have also indicated the likely pathogen. The role of oropharyngeal carriage in relation to the pathogenesis of *K. kingae* infections needs to be further elucidated to help discriminate asymptomatic background carriage from pathogenic organisms, but the potential use of PCR techniques and recognition of the pathogen earlier in the course of this patient’s course may have prevented the morbidity associated with surgical intervention and a prolonged course of antibiotics. Further investigation is also necessary to determine the prevalence of *K. kingae* infections in the United States and to develop guidelines to aid early diagnosis and treatment.

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### References