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Bacterial Peritonitis Caused by *Kingella kingae*▽

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Kingella kingae **is a commensal of the upper respiratory tract that occasionally causes skeletal infections in children and endocarditis in children and adults. We report a case of a 55-year-old man with liver disease and tense ascites who performed a paracentesis on himself and developed *K. kingae* peritonitis and bacteremia.**

CASE REPORT

A 55-year-old man with a history of advanced hepatic cirrhosis related to hepatitis B and C and of past alcohol and intravenous drug use developed progressively worsening abdominal distension, diffuse abdominal pain, and dyspnea with exertion over a 4-week period. To relieve these symptoms, he procured a syringe with a needle and inserted it into his umbilicus, draining enough yellow fluid to soak three shirts. He felt better initially, but 3 days later, he noticed that the injection site was tender and red and his abdomen was more painful and distended. His brothers brought him to the emergency department because he was more confused than usual. At the time of presentation he reported fevers and chills but no headache, rhinorrhea, sore throat, cough, dysuria, or change in bowel habits. He was not actively using drugs or alcohol and had been compliant with his medications, which included a proton pump inhibitor, diuretics, and lactulose. When asked about the needle which he had used for paracentesis, he reported that he had taken it from a hospital and that it was not wrapped in a sterile package but did have an intact needle cap in place.

On examination, the patient was febrile (100.5°F) but hemodynamically stable. He was in mild distress due to dyspnea and abdominal pain. His sclerae were icteric, and his mucous membranes were dry, but there were no oral ulcers or pharyngeal erythema. The patient was edentulous. The heart sounds were regular, and there were decreased lung sounds at the bases. The abdomen was firm, distended, and diffusely tender. The umbilicus was erythematous, tender, and indurated, but there was no discharge. Mild edema of the lower extremities was noted, and there were a few spider angiomata on the skin of the upper chest. Asterixis was present on neurological exam.

On admission, the peripheral leukocyte count was 19,300/mm^3 with 89% neutrophils and 8% band forms. The platelet count was 103,000/mm^3. The serum creatinine level was 1.7 mg/dl, and the sodium level was 120 mmol/liter. The total bilirubin was elevated at 8.1 mg/dl, as were alanine aminotransferase and aspartate transaminase (113 and 225 U/liter, respectively) and international normalized ratio values (1.7). The clinical impression was one of acute decompensation of severe underlying chronic liver disease. Since these events are often triggered by infections, specimens of blood, urine, and peritoneal fluid were submitted for culture before he was started on antibiotics: ceftriaxone and vancomycin.

A large volume of peritoneal fluid was removed (5.5 liters), and a sample sent for analysis showed a white blood cell count of 5,725/mm^3 with a differential of 87% neutrophils and 8% lymphocytes. Gram staining showed a moderate white blood cell count, but no organisms were seen. Cultures were submitted in ESP culture bottles (Trek Diagnostic Systems, Cleveland, OH), which are normally used for blood cultures. On hospital day 2, the ESP system recorded positive growth, and Gram staining revealed short gram-negative rods. The organism showed β-hemolysis on Columbia blood agar but did not grow on MacConkey agar. The Phoenix automated microbiology system (BD Diagnostic Systems, Sparks, MD) failed to identify it. Biochemical testing revealed that it was negative for catalase and indole and positive for oxidase, and that it acidified glucose and maltose but not sucrose or lactose. On the basis of these tests, the organism was identified as *Kingella kingae*. This organism also grew in two sets of blood cultures (ESP culture bottles; Trek Diagnostic Systems, Cleveland, OH), one of which was collected on the day of admission and the other on hospital day 2. Antibiotic susceptibility tests were not performed because of the lack of CLSI standards. Based on the known susceptibility of the organism to β-lactam antibiotics (16), the patient was treated with a 4-week course of ceftriaxone, 1 g intravenously daily. Subsequent cultures of ascites and blood were negative for growth of the organism. A transthoracic echocardiogram revealed no valvular vegetations. A transesophageal echocardiogram was not performed because of the patient's known history of esophageal varices. He was discharged but subsequently returned to the hospital 4 weeks later with altered mental status, and computerized axial tomography scanning of the brain revealed a hemorrhage extending from the right thalamus to the superior pons. Magnetic resonance imaging of the brain revealed an acute right thalamic embolic infarct, although magnetic resonance angiography revealed no vascular malformations, aneurysms, or stenoses. Cultures of blood and ascites fluid remained negative, and a repeat transthoracic echocardiogram was negative for

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thrombi or valvular vegetations. The patient died several weeks later after a prolonged and complicated hospital course. No permission for autopsy was granted.

Kingella is a slow-growing, fastidious, aerobic coccoid to medium-sized gram-negative rod. It belongs to the family *Neisseriaceae*, which also includes the genera *Neisseria*, *Moraxella*, *Acinetobacter*, and *Oligella*. There are four known species: *Kingella kingae*, *Kingella indologenes*, *Kingella denitrificans*, and *Kingella oralis*. Of these, *K. kingae* has been found to be the most common human pathogen. It is beta-hemolytic on blood agar and also grows on chocolate agar, but only 33% of isolates grow on MacConkey agar. Because it has a tendency to resist decolorization and is commonly found in pairs and short chains on Gram staining, it may sometimes be mistaken for a streptococcus. Biochemically, it is oxidase positive, ferments glucose and maltose, and is negative for catalase, urease, and indole (2, 3, 9, 10).

K. kingae can frequently be isolated from the mucous membranes of the upper respiratory tract. In a surveillance study done at a day-care center in Israel, *K. kingae* was isolated from 109 of 624 throat cultures (17.5%), and 34 of 48 children (70.8%) were found to carry the organism at least once during an 11-month period. The organism was detected in children ages 6 to 42 months but not in those younger than 6 months old who were attending a well-baby care clinic (13, 15). This age distribution of respiratory carriage correlates with the age distribution of invasive infections in the pediatric population. Neonates almost never get *Kingella* infections. In infants and toddlers, the most common clinical manifestations of *K. kingae* disease are septic arthritis and osteomyelitis. Similar to skeletal infections in this age group caused by other organisms, these infections are the result of hematogenous spread of the organism. Young children are particularly prone to hematogenous osteomyelitis, most likely as a result of the rich vascular supply to the growth plates of their long bones (4). Approximately half of children with *K. kingae* bacteremia have a simultaneous focal source (10). Positive blood cultures for *K. kingae* at the time of diagnosis of skeletal infection are rare, however, presumably because the primary bacteremic phase of the illness is often transient and asymptomatic (2, 14), and secondary bacteremia from seeding of the bloodstream from the infected bone does not seem to occur. Interestingly, oropharyngeal inflammation (upper respiratory tract infection; stomatitis) is a common antecedent to these infections (up to 60% of cases in one study), suggesting that the bacteria may enter the bloodstream from damaged or inflamed oropharyngeal mucosa and then subsequently seed distant sites from the bloodstream (2, 3, 13, 14).

Endocarditis is also a common manifestation of *K. kingae* infection. In contrast to arthritis and osteomyelitis, however, *K. kingae* endocarditis usually occurs in school-age children and adults (who are not as prone to hematogenous seeding of the bones); more than 60% of patients are 16 years of age or older (1, 3). Approximately 75% of cases involve structurally abnormal heart valves, and 31% have a history of antecedent disruption of the oropharyngeal mucosa (1, 3, 6).

Less-common presentations of *K. kingae* infection reported include meningitis, pneumonia, ophthalmic infections, abscesses, intervertebral diskitis, cellulitis, and epiglottitis (3, 5, 8, 9, 11). There is also one reported case of pyopneumothorax in a patient with coccidioidal pulmonary cavitary disease (9). The patient we describe here represents the first reported case of secondary peritonitis caused by *K. kingae* in a man who performed a paracentesis on himself to relieve his ascites. Also unique to this case is the likely mechanism by which this infection occurred. Most cases of secondary peritonitis are caused by bacteria which arrive in the peritoneal cavity from a gut source, most often due to rupture of an abdominal viscus. In contrast, in our edentulous patient without oral mucosal lesions to account for possible dental or mucosal seeding, the peritoneal infection was likely the result of bacteria directly injected into the peritoneal cavity. Although the patient denied licking the needle prior to using it, the most likely explanation for the clinical presentation is that he used a needle contaminated with oral flora, thus inoculating the ascites directly with *K. kingae* and then developing bacteremia subsequent to the peritoneal infection. This contrasts with the majority of the cases reported, in which bacteremia was felt to have developed directly as a result of a breach in the integrity of the oropharyngeal mucosa.

Also of interest is that our patient suffered from a stroke 4 weeks after diagnosis of *K. kingae* peritonitis and bacteremia. Because he did not have a transesophageal echocardiogram, we cannot prove that his stroke was the direct result of a cardiac vegetation, but this remains a possibility, since stroke is a commonly reported complication of *K. kingae* endocarditis (1, 3, 6, 7, 9, 12). Further supporting this notion was the radiologist's interpretation that the lesion on magnetic resonance imaging was embolic in nature.

In summary, we feel that this case illustrates how poorly performed patient-initiated medical procedures can have life-threatening infectious consequences. Our patient's self-paracentesis caused peritonitis with a commensal organism of the upper respiratory tract, an example of a common infection caused by an unusual organism brought about in an unusual way.

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