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Case report

Kingella kingae infective endocarditis in a healthy adult

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Introduction

Infective endocarditis generally results from turbulence or trauma to the endothelial surface of the heart followed by a transient bacteremia that seeds the damaged endothelial area. A 2009 study by Bor et al. reported that infective endocarditis (IE) affects approximately 12.7 per 100,000 people yearly and has been steadily increasing [9]. Of the cases of IE with known pathogens, *Staphylococcus aureus* and streptococci make up the vast majority, with the HACEK organisms (*Haemophilus, Aggregatibacter, Cardiobacterium, Eikenella,* & *Kingella*) only comprising approximately 1.4–3% of all cases annually [10]. The HACEK organisms are fastidious gram-negative bacteria that are typically oropharyngeal commensals and while of low-virulence, are known to cause a spectrum of infections in children. Interestingly, the rates of prevalence of endocarditis in any member of this group of bacterium changes depending on the age of the patient. In this case report we present a healthy male who developed IE secondary to *Kingella kingae*, which is exceedingly rare in adults.

Case report

A 27 year old male of Ashkenazi Jewish descent was admitted to our hospital with a one day history of abdominal pain. For the previous six months the patient was living in Israel with his wife and young son where he was undergoing rabbinical training. Two weeks prior to admission while still in Jerusalem, he notes that he developed a fever with chills as well as upper extremity weakness but with no other symptoms. He stated that his wife had a similar disease course slightly earlier. His body aches continued until he returned to the U.S where he developed worsening fevers along with vomiting and abdominal pain. He denied any chest pain, rash, or intravenous drug use. On physical exam he was afebrile and without any evidence of pharyngitis, oral ulcers, irregular heart sounds or murmurs. His fingernails and palms of his hands and soles of his feet were without any obvious lesions. He had only mild abdominal tenderness. His white blood cell count was 11.3 $\times$ 10^9 cells/L. A urine analysis was positive for protein, ketones, bilirubin, urobilinogen, and mucous. No urine cultures where done. A CT of his abdomen and pelvis showed multiple splenic infarcts, bilateral renal infarcts, a moderate amount of pericardial effusion and hepatosplenomegaly. Blood cultures were drawn and he was started empirically on intravenous vancomycin and pipercillin-tazobactam.

A transthoracic echocardiogram showed a patent foramen ovale (PFO) and normal valves but no vegetation. A follow up transesophageal echocardiogram confirmed the patent foramen ovale, the normal valves, and again no vegetation was seen. Both a complete hypercoagulable work up and a lower extremity duplex were negative. Blood cultures grew *Kingella kingae* in 3 sets with a 4th growing coagulase negative staphylococcus thought to be a contaminant. Cardiology was consulted and determined that the PFO was likely not a factor in his septic embolic infarcts and therefore did not require further intervention. *Kingella* susceptibilities were performed and his treatment was switched to intravenous ceftriaxone for a total of four weeks. Repeat blood cultures were sterile. A CTA chest/abdomen/pelvis was conducted to evaluate for an occult source of the emboli, but revealed no evidence of intramural hematoma, dissection or aneurysm. His splenic infarctions demonstrated no significant change and some bilateral renal infarctions resolved whereas others appeared to be new. He continued to improve during his hospital course and was eventually discharged to home to complete his four-week course of antibacterials. He was followed by both hematology and infectious disease services six weeks after discharge. At this time, he had a repeat CT of the chest, abdomen and pelvis which showed improvement in his splenic and renal infarcts.

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Discussion

Kingella kingae is a facultative anaerobic β-hemolytic gram-negative bacterium commonly arranged in pairs or short chains, first identified in the 1960s by Dr. Elizabeth King. *K. kingae* has been established as a common agent of occult bacteremia and predominant etiology of joint and bone infections in children 6–36 months old [6], however reports of infection caused by *K. kingae* among adults are rare.

K. kingae can be a normal inhabitant of the oropharynx with rates of colonization that peak at rates of 3–12% between the ages of 6–48 months old [1]. It is believed that disrupted respiratory or buccal mucosa (through upper respiratory tract infections, aphthous stomatitis, or acute gastroenteritis) may facilitate bacterial invasion and hematogenic disseminations [2,4,5]. Outbreaks of invasive *K. kingae* infections, including almost the entire spectrum of the disease (osteomyelitis, bacteremia, and endocarditis), have been reported from French, American, and Israeli day care facilities [6]. Poor dental hygiene or dental extractions have been reported frequently in adolescents and adult patients prior to initial infection. In children osteoarticular infections (OAIs) are the most common form of *K. kingae* invasive disease [1]. Likely because of the prevalence of vaccination for *H. influenzae* type b, *K. kingae* has now become the most common gram-negative bacterium causing osteoarticular infections in children less than 3 years of age [3]. Of the endocardial infections in children those that were secondary to *K. Kingae* have been reported by some case series to be approximately 7% [1,6]. It usually affects previously healthy children aged less than 4 years, whereas older children and adults frequently have predisposing conditions [6]. Interestingly, a report by Amit et al showed that although a variety of *K. kingae* strains circulate within the pediatric population of Israel, a small subset is responsible for most clinical infections, and a few exhibit significant associations with particular clinical disease [7].

Most isolates of *K. kingae* strains are broadly susceptible to β-lactams, cephalosporins, aminoglycosides, Trimethoprim-sulfamethoxazole, and fluoroquinolones [3]. Established treatment for *K. Kingae* endocarditis consists of a high dose intravenous β-lactam antimicrobial as monotherapy or combined with an aminoglycoside drug for 4–7 weeks duration [6]. Despite *K. kingae*'s remarkable susceptibility to antimicrobial drugs, serious complications such as mycotic aneurisms, pericarditis, valvular insufficiency, congestive heart failure, CVAs, embolic arthritis and cellulitis are common complications [1,6].

In stark contrast to case reports involving children, there have been only approximately 40 cases reported of *K. kingae* endocarditis in adults since 1967. Of those cases only 6 have been in patients with no pre-existing health conditions [4,6]. Also interestingly native valves are primarily affected in children with *K. kingae* endocarditis (up to 95% of cases) whereas native and prosthetic valve involvement are reported at similar frequencies in adults. Given the rarity of adult cases of *K. kingae* infection, our case represents a unique situation. The patient likely had vegetative endocarditis that led to shower emboli causing impressive splenic, renal, and likely hepatic infarcts. He did not have any recent dental work, had no obvious cardiac malformation and no systemic immunosuppression. However he had spent a considerable amount of time in Jerusalem and had a young child (less than 3 years of age) likely in daycare or associated with other children in day care. In a limited single center study, Brandle et al. reported that parents of young children, less than 4 years of age, had a colonization rate (8.8%) similar to that of children younger than 4 years [8]. While pharyngeal carriage has been thought to be universal across countries, a report from Amit et al which compared Jewish children and Bedouin children in Southern Israel showed that Jewish children had a significantly higher incidence of invasive disease [1]. Given these two reports it is likely that our patient had transmission of a virulent strain from his young child.

Conclusion

In this paper we report the case of a healthy man without any cardiac history who developed *Kingella kingae* endocarditis. While not uncommon in children, this infection is exceptionally rare in healthy adults and has not been reported in these hosts in recent literature. We hypothesize that he likely had horizontal transmission from his young son ultimately leading to his endocarditis. While it is unlikely that there is a cultural or racial link between the reported incidences of *K. kingae* it is interesting to note that our patient returned from a region where colonization has been proven and increased virulence has been suspected.

Conflict of interest

There are no conflicts of interest to disclose.

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Authors' contribution

MD, TM, and JC cared for the patient searched the scientific literature, wrote the report and revised the report.

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