Kingella kingae endocarditis: A rare case of mitral valve perforation

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ABSTRACT

***Kingella kingae*, a HACEK (*Haemophilus parainfluenzae, Aggregatibacter actinomycetemcomitans, Aggregatibacter aphrophilus, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae*) organism, is a common resident of the upper airway in children; it has been associated with endocarditis in children with pre-existing heart conditions. This case report describes *K. kingae* endocarditis leading to valvular damage in a previously healthy 18-month-old child. Our patient developed a *K. kingae* bacteremia that was later complicated by meningitis, septic embolic stroke, and endocarditis of the mitral valve, leading to perforation of the posterolateral leaflet. The patient was initially treated conservatively with cefotaxime but, subsequently, required a mitral valve repair with a pericardial patch and annuloplasty. This report draws attention to the need for clinicians to be aware of the potentially serious complications of *K. kingae* infection in young children. If *K. kingae* infection is suspected then therapy should be initiated promptly with a β-lactam, followed by early echocardiographic assessment. This case also highlights the lack of specific guidelines available for *K. kingae* endocarditis.**

Keywords: Endocarditis, *Kingella kingae*, mitral regurgitation, pediatric

INTRODUCTION

Kingella kingae (*K. kingae*) is a β-hemolytic, facultative anaerobic, Gram-negative coccobacillus. It is a member of the HACEK (*Haemophilus parainfluenzae*, *Aggregatibacter actinomycetemcomitans*, *Aggregatibacter aphrophilus*, *Cardiobacterium hominis*, *Eikenella corrodens*, *Kingella kingae*) group of organisms and one study has suggested that up to 73% of children over 6 months of age carry the bacterium in their oropharynx at some point, with the carriage rate highest among those between 6 months and 4 years.[1]

Increasingly, *K. kingae* has been identified as the causative organism in a number of pediatric infections, including septic arthritis, osteomyelitis, and bacteremia.[1] While *K. kingae* infections of the skeletal system have an excellent prognosis, *K. kingae* endocarditis is associated with serious complications, including valvular abscesses and septic embolization.[2] These complications are often seen in children with pre-existing heart conditions. In this communication, we describe the complicated case of a previously healthy 18-month-old Caucasian male child who presented with *K. kingae* bacteremia and, subsequently, developed meningitis, endocarditis, a septic embolus to the frontal lobe, and perforation of the posterolateral mitral valve leaflet.

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CASE REPORT

An 18-month-old previously healthy male child presented to the emergency department (ED) with several days of flu-like symptoms. His birth and medical history were unremarkable. The patient was discharged home but was persistently febrile and had continuous vomiting, with decreased oral intake. He had no history of diarrhea. The patient returned to the ED where he was admitted to hospital with a provisional diagnosis of dehydration and possible meningitis.

On his admission, the patient was found to be febrile, pale, and tachycardic, with peripheral cyanosis and mild photophobia. His tachycardia persisted despite the administration of several boluses of normal saline; however, his perfusion was determined adequate. A new 2/6 systolic murmur was noted on auscultation. There was no hepatosplenomegaly. Laboratory tests revealed the following: Hemoglobin of 9.0 g/dL, white cell count of 25×10^9/L, platelets of 100×10^9/L, erythrocyte

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sedimentation rate of 77 mm/hr, and C-reactive protein level of 20 mg/L. Blood cultures were drawn and a lumbar puncture performed on the second day of admission revealed a white cell count of 690 cells/μL (87% neutrophils). The cultures were initially negative, so the patient was administered ceftriaxone. Over the subsequent three days, his tachycardia, pallor, and systolic murmur persisted but his neck stiffness resolved. On the third day, the blood culture grew *K. kingae*, which prompted, one week after his initial presentation, investigations into possible bacterial endocarditis. A transthoracic echocardiogram revealed mild mitral regurgitation (MR), with a small perforation in the posterolateral leaflet but no evidence of vegetations. The patient was commenced on a 6-week course of cefotaxime.

On day four of the course, he became increasingly irritable and a head computed tomography (CT) scan revealed a 17-mm-long by 9-mm-wide wedge-shaped left frontal lobe infarct, likely due to a septic embolism. There was no appreciable neurological deficit. On day seven, the patient's irritability resolved and a head CT performed 6 weeks later showed a resolving cerebral infarct.

Two weeks after completion of his antibiotic regimen, the patient returned with increasing fatigue and diaphoresis during exertion. An echocardiogram revealed a 3-mm perforation of his posterolateral mitral valve leaflet with severe MR. No vegetations were seen on any valve and his submitral apparatus and chordae were intact. Due to progressive dyspnea, the patient was scheduled for surgical repair of his mitral valve.

In the operating room, a pre-operative transesophageal echocardiogram (TEE) revealed a discrete 4-mm hole in the posterolateral leaflet of his mitral valve [Figure 1]. Normal left ventricular function was observed with a mildly dilated left atrium measuring 2.5 cm × 2.5 cm and a mitral valve annulus of 21 mm. Under cardiopulmonary bypass, the left atrium was entered using a trans-septal approach and the mitral valve was examined. A well-circumscribed 4-mm diameter defect in the medial component of the posterolateral leaflet was identified. The defect was repaired with a small patch of autologous pericardium. As the patient's annulus was slightly dilated, a suture annuloplasty was performed from the anteromedial to the posterolateral commissure. After weaning from cardiopulmonary bypass, the TEE confirmed trivial mitral regurgitation [Figure 2]. The postoperative recovery was unremarkable.

DISCUSSION

K. kingae infection most commonly results in bacteremia and septic arthritis in children; meningitis and endocarditis are rarely observed.[1] From a review in 2004 and one subsequent report, 16 cases of *K. kingae* endocarditis have been reported.[3,4] Of these, four were previously healthy, developmentally normal children. Two of the four children experienced mitral leaflet perforation.

In one report, 40-50% of *K. kingae* infections had concomitant mucosal lesions acting as the possible route of inoculation.[5] We observed no concomitant oral pathology in the patient in our study; however, he was likely still teething, thus, making the gingiva a potential entry point for the organism. This route of infection could explain the incidence rate pattern of *K. kingae* infection, which peaks in the 6- to 11-month-old population,

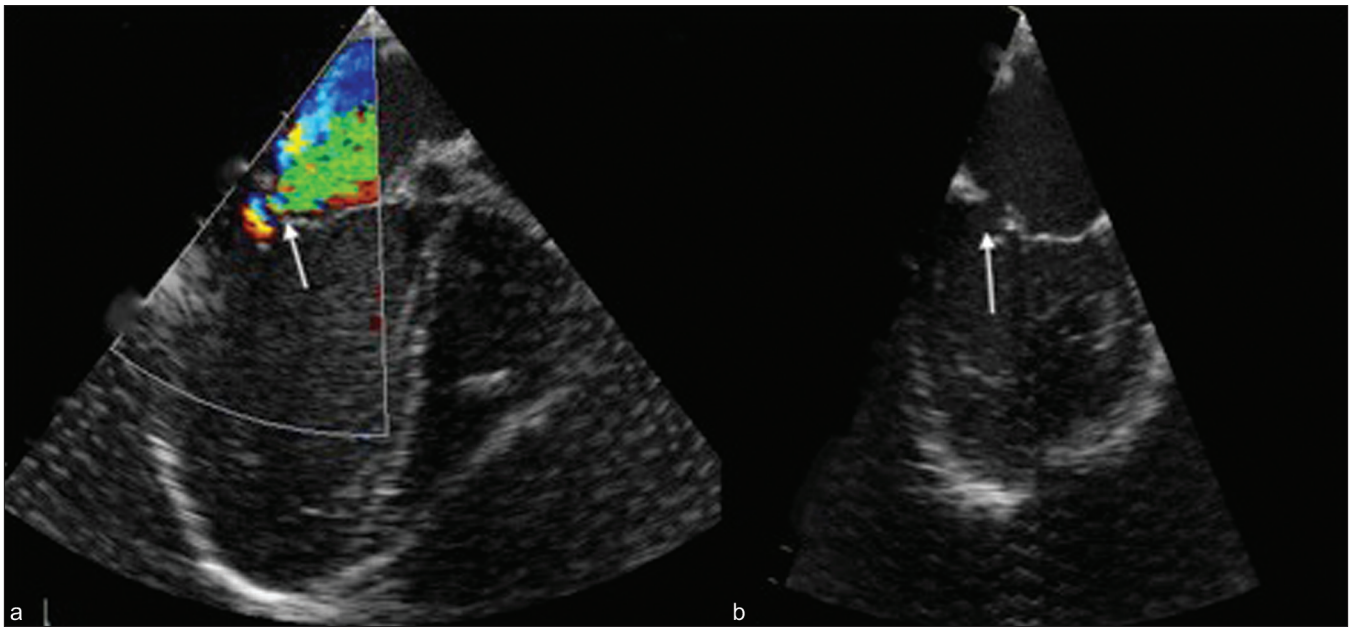

Figure 1: (a) Preoperative trans-esophageal echocardiogram (TEE) showing mitral regurgitation through a 4-mm hole in the posterolateral leaflet. (b) Preoperative TEE revealing a perforation in the posterior leaflet of the mitral valve

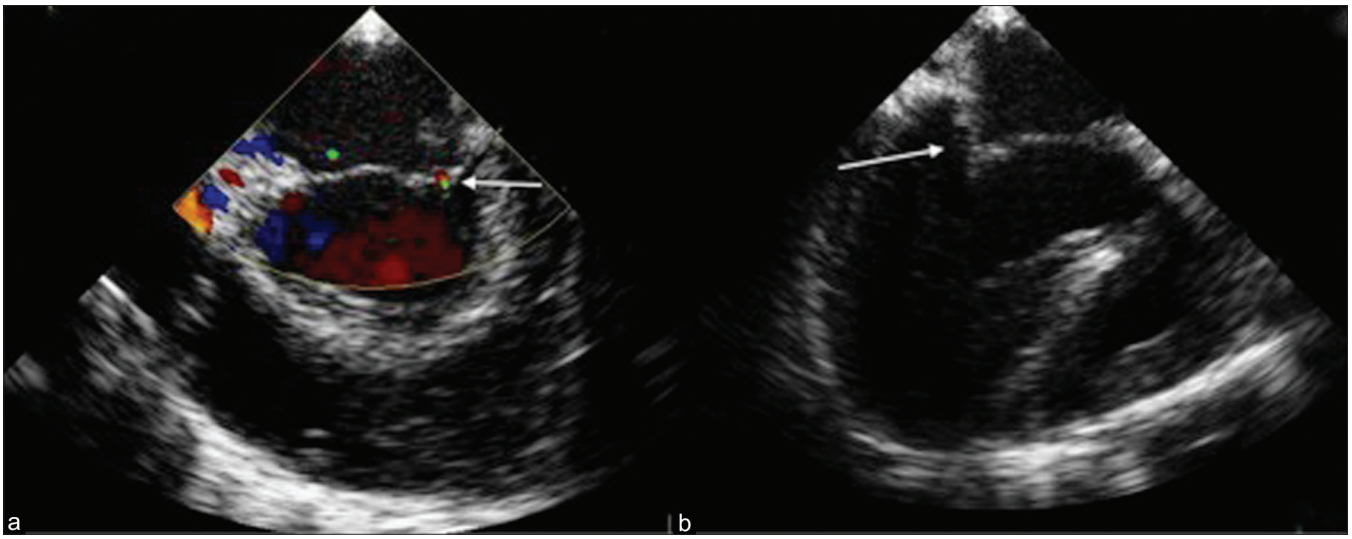

Figure 2: (a) Post-repair, Intra-operative Trans-Esophageal Echocardiogram (TEE) showing minimal Mitral Regurgitation. (b) Post-repair, Intra-operative Trans-Esophageal Echocardiogram (TEE) showing the newly intact posterior leaflet of the Mitral valve

with 60% of infections occurring before 24 months of age.[1] Furthermore, a recent study of 322 cases of *K. kingae* infection confirmed a seasonal pattern to invasive *K. kingae* disease with viral infections frequently detected concomitantly.[6] With respect to our patient, his initial presentation with flu-like symptoms may have represented a predisposing viral infection.

K. kingae has fastidious growth requirements. It may take 5 days or longer to be isolated and does not grow in MacConkey Agar.[7] It is also resistant to alcohol decolorization, making it falsely appear Gram-positive.[7] This lengthens the pre-diagnosis phase and, despite empirical treatment, *K.kingae* endocarditis is associated with a higher mortality rate than viridans streptococcal endocarditis.[7]

This case reflects how the current investigation and management strategy for invasive *K. kingae* disease can fail to prevent serious complications. Interestingly, a recent report has shown that early surgical removal of a leaflet vegetation can be effective in preventing complications of embolism and infarction.[4] In summation, if *K. kingae* infection is suspected, the patient should immediately be administered appropriate β-lactam as well as subjected to cardiac assessment.

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