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Particular surgical aspects of endocarditis due to *Kingella kingae* with cerebral complication

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Abstract

We report 2 cases of *Kingella kingae* endocarditis leading to valvular mitral perforation in previously healthy children. *Kingella kingae* belongs to the HACEK (*Haemophilus aphrophilus*, *Actiobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens* and *K. kingae*) group of organisms known to cause endocarditis.

Keywords: *Kingella kingae* • Infective endocarditis • Stroke

INTRODUCTION

Kingella kingae is a Gram-negative, anaerobic bacterium of the HACEK (*Haemophilus aphrophilus*, *Actiobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens* and *K. kingae*) group. However, *K. kingae* belongs to the normal oropharyngeal flora found in children aged 4–10 years. It is an infrequent causal agent of endocarditis (1 of 1397) [1]. The frequency of neurological complications can be as high as 30%. Within this 30%, there is 77% probability for stroke and 46% probability for meningitis [1].

We report the surgical management of 2 cases of *K. kingae* mitral endocarditis with neurovascular complications.

CASE 1

We first report a 22-month-old girl who presented with *K. kingae* mitral endocarditis with embolic stroke and aneurysmal rupture. This case has already been described in another case report on *K. kingae* endocarditis with cerebral complication [2].

She was admitted to the hospital with fever, hypertonia and urinary retention. She was diagnosed with influenza A encephalomyelitis and meningitis by positive influenza A real-time polymerase chain reaction on a nasopharyngeal sample. We performed transthoracic echocardiogram that revealed a mild mitral regurgitation with a small perforation in the posterior lateral leaflet (P3) (Fig. 1A). No vegetation was observed on any valve, and the submitral apparatus and chordae were intact. A blood culture obtained before antibiotic treatment was positive for a sequence type 25 *K. kingae* strain. She was treated with amoxicillin and ciprofloxacin. Three weeks after admission, she was rehospitalized for mycotic aneurysm rupture leading to left parieto-occipital haemorrhagic stroke and intraventricular haemorrhage. Four months after the neurosurgical treatment, she was scheduled for surgical repair of the mitral valve (MV).

In the operating room, using a trans-septal approach, the MV was found to be normal except for a 2-mm diameter defect in the posterolateral leaflet (P3), and defect was repaired by direct suturing of the perforation. After 2 weeks, a transthoracic echocardiogram confirmed the absence of regurgitation.

CASE 2

A 9-month-old boy presented with 24-h history of fever, paleness, lethargy, right hemiparesis and sharp deviation of the mouth on the right. A magnetic resonance imaging was performed and showed an ischaemic event in the left carotid artery distribution. An echocardiogram showed a structurally normal heart but with an evidence of mitral perforation. Initial blood cultures obtained before administering antibiotics were positive, which confirmed *K. kingae* endocarditis. He was treated with cefotaxime for 6 weeks. He showed improvement in the right hemiparesis and total recovery in facial paralysis. After 1 year, the transthoracic echocardiogram revealed a mitral regurgitation three-fourths by 2 mm defect in the posterolateral leaflet of the MV (Fig. 1B). Normal function of the left ventricle was observed with a mildly dilated left atrium measuring 7, 5 cm^2, and the left ventricle was also dilated 35 mm (left ventricular end-diastolic

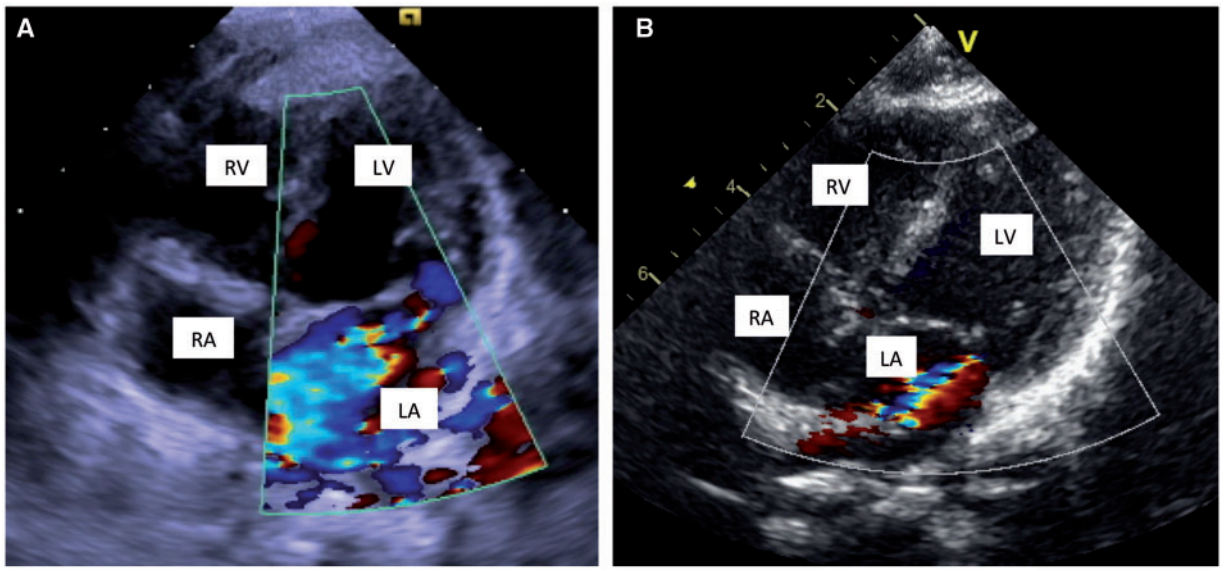

Figure 1: Transthoracic echocardiographic 4-chamber view showing mitral regurgitation. (**A**) Case 1 mitral insufficiency 4 of 4 and (**B**) Case 2 mitral insufficiency 2 of 4. RA: right atria; LA: left atria; RV: right ventricular; LV: left ventricular.

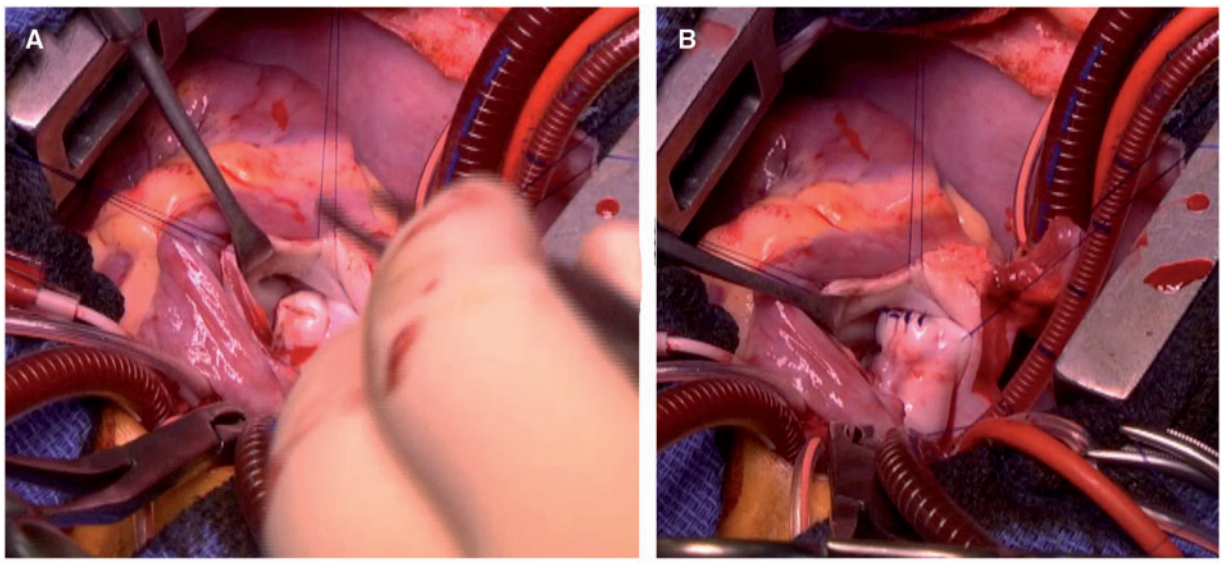

Figure 2: Intraoperative images showing (**A**) perforation of the mitral valve and (**B**) after direct suturing of the perforation.

diameter = 115% of theory). Because of progressive left ventricular dilatation and risk of recurrences, he was scheduled for surgical repair of the MV.

In the operating room, using the same trans-septal approach, the MV was tested. A 2-mm diameter defect in the posterolateral leaflet (P3) was identified. The edge of the perforation was fibrous. There was no abscess or vegetation. We decided to perform direct suturing of the perforation (Fig. 2).

For the 2 cases, no complications occurred during the perioperative period in the first year.

DISCUSSION

K. kingae endocarditis is rare but shows increasing frequency [1]. It is associated with a very high rate of neurological complications (30%) [1–3].

In the literary reviews [3], it seems that *K. kingae* endocarditis has a preferential location in the MV (90%) and particularly in the posterior leaflet (3 of 4 cases). The MV is most often (95%) without abnormality [3]. The pathogenic mechanism is mixed, including formation of vegetation and perforation of the valve.

There is no consensus on the optimal timing of surgery for endocarditis due to *K. kingae*. We recommend late operation for patients with neurological complications. First, the repair is easy (direct suture of perforation) when the endocarditis is not active and the edge of the MV is fibrous. Second, there is a high risk of cerebral complication with risks of postoperative neurological deterioration caused by secondary cerebral haemorrhage related to cardiac surgery. However, in patients without neurological complications, treatment should be performed immediately, because early cardiac surgery may prevent vegetation embolization.

In the literary reviews, it seems we are the first to describe the direct repair of the MV without patch for *K. kingae* endocarditis.

When the perforation of the valve is simple after cooling down the infective process, the minimal use of prosthesis (PTFE ring and pericardium patch) is encouraged. The MV repair without prosthetic annuloplasty ring or pericardial patch is probably better for the recurrence rate of endocarditis and for the durability of MV repair [4]. Glutaraldehyde-treated autologous pericardium may develop a calcification of the autologous pericardium after long-term MV stenosis or regurgitation [5].

CONCLUSION

Kingella kingae endocarditis is rare and associated with a very high rate of neurological complications. In *K. kingae* endocarditis, with neurological complications, the MV repair must be executed after the treatment of the mentioned neurological complications and endocarditis. The MV repair should be done, if possible, without using prosthetic material (rings) or a pericardium patch.

Conflict of interest: none declared.

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