Letter to the Editor

Uncommon *Kingella kingae* lytic bone lesions in children

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Kingella kingae has emerged as an important cause of osteoarticular infections (OAI) in young children, ranging from 48 to 82%, as a result of improved culture methods and nucleic acid amplification techniques.¹ Usually these infections are mild and have a favorable prognosis,^{2–5} but severe infections have been reported.¹ Mallet *et al.* conducted a retrospective study and identified 10 unusual *K. kingae* OAI with bony lytic lesions and growth cartilage damage.¹ We also report three patients with these atypical lesions.

From 2012 to 2014, three children (Table 1) with *K. kingae* lytic OAI were identified. All had low-grade fever, mild ankle or feet tumefaction and refusal to bear weight. The gap between symptom onset and admission was 12 ± 6 days. Two reported previous upper respiratory illness. All had slightly elevated inflammatory markers (average: leukocytes, 8900 cells/mm³; sedimentation rate, 32.3 mm/h; C-reactive protein, 1.93 mg/dL). Each had a single lytic lesion: in the posterior talus (Fig. 1a); in the postero-inferior tubercle of calcaneus, with liquid collection and thickening of adjacent plantar fat pad (Fig. 1b); and in the distal lateral metaphyseal peroneus, with peri-maleolar soft-tissue thickening and ankle effusion (Fig. 1c).

by an oral course (26 ± 5 days). At 6 months follow up, there were no clinical or radiologic lesions.

In the present cases, several characteristics of *K. kingae* infections can be identified: predominance in early childhood, previous upper respiratory symptoms, clinical paucity, low inflammatory markers and favorable response to antibiotics.³

The low virulence of *K. kingae* is reflected in the shorter length of hospital stay, fewer adverse events and better outcome.³ Recently, however, Mallet *et al.* reported unusual severe *K. kingae* OAI with osteomyelitis and metaphyseal or epiphyseal abscess.¹ Nevertheless, in their cases, there was a rapid clinical and laboratory response and neither acute nor chronic complications, nor recurrence, were reported. We verified the same pattern in the present patients. Also, other authors have reported *K. kingae* predilection for small bones of the feet, with articular damage, without penetrating trauma.³ We therefore question whether we might have been too interventional, because the management of *K. kingae* OAI is usually less invasive, mostly without the requirement of surgical intervention and shorter treatment duration. The need for differential diagnosis in these atypical cases, however, meant that decisions needed to be made quickly.

Table 1 Clinical and demographic data

Patient	Sex	Age at infection (months)	Baseline health status	Leukocytes (cells/mm ³)	CRP (mg/dL)	ESR (mm/h)	Site of infection	I.v. antibiotic (days)	Treatment duration (days)
1	M	38	Healthy	14 600	1.7	52	Right posterior talus	5	33
2	M	33	Healthy	9700	1.19	50	Left calcaneus	9	30
3	F	25	Healthy	7300	0.97	19	Right distal peroneus	9	39

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.

Arthrotomy with bone abscess curettage was performed in two and arthrocentesis with bone puncture in one patient. Empirical i.v. antibiotherapy with flucloxacillin and gentamycin was started. Histology showed inflammatory cells and bone sequestrum or abscess. Bone aspirate/synovial fluid culture identified *K. kingae*, while blood cultures were sterile. Antibiotherapy was changed for i.v. amoxicillin-clavulanate or cefuroxime (8 ± 2 days) followed

Mallet *et al.* speculate that the less pronounced inflammatory response observed might be attributed to *K. kingae* molecular detection, with possible lower bacterial loads.¹ In the present cases, however, *K. kingae* was isolated on culture and the recovery was also excellent. Most likely, the indolent and low-virulence nature of the bacteria explains this favorable outcome.

Nevertheless, we should not underestimate the potential risk of *K. kingae* OAI, even in cases of lytic bone lesion when the prognosis seems to be favorable.

Disclosure

The authors declare no conflicts of interest.

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Fig. 1 Bony lytic lesions in the (a) right posterior talus; (b) postero-inferior tubercle of the left calcaneus, with thickening of adjacent plantar fat pad; and (c) right distal lateral metaphyseal peroneus, with peri-maleolar soft-tissue thickening and ankle effusion.

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