

Case report

A pitfall in purulent arthritis brought out in *Kingella kingae* infection of the knee

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SUMMARY A description of the protracted course of kingella monoarthritis is given as an illustration of the importance of accounting for the slow growth, low viable numbers, and fastidious culture requirements of microbes that may be encountered in synovial samples. The practice of carrying out synovial fluid cultures in the same way as blood cultures is recommended.

The culture of synovial fluid is a subject generally not considered to warrant particular consideration in medical microbiology texts. In our experience, however, the slow growth and/or the small number of infective organisms in synovial fluid occasionally results in a failure of routine methods of culture.

Kingella kingae, a short, slow-growing, Gram-negative rod, formerly included in the genus *Moraxella*, occurs in the normal oropharyngeal flora but may rarely cause clinical infection.¹ Altogether 10 cases of kingella arthritis (Table 1) have been reported.²⁻¹⁰ The present case illustrates both the diagnostic difficulties and the protracted course of this joint infection.

Case report

The patient was a 76-year-old retired joiner with a long history of emphysema, asthma, and chronic bronchitis. On 15 December 1982 he spent about one hour on his knees painting. As he got up he felt pain in his right knee; later the pain increased and the knee became swollen. He visited a general practitioner the following day and again four days later, by which time he was febrile. The knee was then aspirated. The fluid contained abundant leucocytes, but bacterial culture was negative. As the patient was unable to walk he was admitted to

hospital. On admission he had an erythrocyte sedimentation rate (ESR) of 92 mm/h and an elevated C-reactive protein (CRP) level. Radiologically the knee was normal.

The knee was aspirated again three times, with leucocyte counts of 20-35 × 10⁹/l, 80-90% polymorphonuclears. On each occasion the fluid was cultured for bacteria, mycobacteria, and yeasts, but with negative results. A tentative diagnosis of pseudogout was made, and the patient was treated with a glucocorticoid injection into the knee. After this he became much better and the ESR decreased to 46 mm/h. He was discharged on 18 January 1983 but readmitted on 11 February when the symptoms had recurred. Synovial fluid was examined and

Table 1 Reported cases of *Kingella kingae* arthritis

| Case | Year | Author | Patient's age | Joint |
|------|------|--------------------------------------|---------------|--------|
| 1* | 1969 | Feigin <i>et al.</i> ² | 2 years | Knee |
| 2* | 1975 | Spahr ³ | 18 months | Knee |
| 3* | 1980 | Rosenbaum <i>et al.</i> ⁴ | 42 years | Wrists |
| 4 | 1980 | Redfield <i>et al.</i> ⁵ | 4 years | Knee |
| 5 | 1981 | Vincent <i>et al.</i> ⁶ | 49 years | Knee |
| 6 | 1982 | Davis, Peel ⁷ | 13 years | Hip |
| 7 | 1983 | Gay <i>et al.</i> ⁸ | 10 months | Knee |
| 8 | 1983 | Patel <i>et al.</i> ⁹ | 18 months | Knee |
| 9 | 1983 | Powell, Bass ¹⁰ | 14 months | Knee |
| 10 | 1983 | Powell, Bass ¹⁰ | 2 years | Knee |

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*Reported as moraxella arthritis; bacterium not certainly determined as *Kingella kingae*.

cultured, with the same results as before. A further intra-articular glucocorticoid injection was given, but with a poor response.

The ESR was 64 mm/h, CRP 17 mg/l, and the patient could not walk without crutches. Nevertheless, he was discharged three days later and stayed at home until the next flare-up on 13 May, when he was readmitted with the same clinical and synovial fluid findings. All cultures were again negative. X-ray films of the knee showed pronounced osteoporosis and narrowing of the joint space medially. The patient was transferred to the Rheumatism Foundation Hospital on 19 May 1983. He had received no treatment with antibiotics.

On arrival the patient's right knee was warm and swollen; there was 10° extension limitation, and pronounced atrophy of the thigh muscles. All other joints were normal. The patient was afebrile and had a slight leucocytosis. The ESR was 90 mm/h and CRP 74 mg/l. Multiple blood cultures were negative. Arthrocentesis yielded about 1 ml of fluid containing abundant leucocytes but no crystals, and no bacteria on direct staining. The fluid was cultured according to our protocol for purulent synovial fluid on sheep blood agar, McLeod agar, and in semisolid thioglycollate at 35°C in a 5% CO₂ atmosphere; anaerobically on sheep blood agar and brucella agar with added vitamin K and kanamycin; the remaining fluid was divided between one aerobic and one anaerobic blood culture bottle (Hemobact, Orion Diagnostica, Helsinki, Finland). All primary cultures and the first blood culture bottle subcultures were negative.

At this stage an arthrotomy was performed and synovial biopsies taken, revealing a non-specific chronic synovitis. However, on the second subculture, one week after inoculation of the blood culture bottles, growth etching into the agar surface was noticed. The bacterium was later identified as *Kingella kingae* by standard methods.¹ Treatment with cefuroxime 750 mg 4 times a day for 10 days produced an excellent response. The patient was discharged with crutches on 7 July, but at follow-up five weeks later knee's condition and function were entirely normal.

Discussion

The origin of the infection may have been the patient's respiratory tract, while his prolonged kneeling may have been an aggravating factor. The importance of interrupted capillary integrity in the pathogenesis of haematogenous arthritis has recently been emphasised.¹¹ Most *Kingella kingae* infections have occurred in infants, but it is perhaps

not surprising for an opportunistic agent to appear at both ends of life.

All but one (case 6, Australia) of the previous reports of *Kingella kingae* arthritis are from the USA. Possibly this is due to some difference in microbiological practice. In tuberculous synovitis it is well known that culture of synovial tissue gives more consistent positive results than culture of synovial fluid. The same may apply to bacterial arthritis in general.¹² Evidently bacteria in the fluid represent a spill-over from infection in the tissue, and accordingly, if a biopsy is impracticable, at least the amount cultivated should be maximised in order to increase the chances of encountering viable bacteria. Particularly for a fluid that is purulent a negative culture result should not be accepted as definitive until provision has been made to exclude both an infection caused by an unusual microbe and a partially repressed conventional infection—for instance, after inadequate treatment with antibiotics. The use of blood culture bottles for this purpose has been successful in our hands in several cases of staphylococcal infection with negative cultures on solid media; moreover it may also serve to reveal slow-growing organisms.¹³

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