Cureus
Part of SPRINGER NATURE

Open Access Case Report

Presumed Kingella kingae Septic Arthritis in an 18-Month-Old Child: The First Suspected Case Reported From Georgia

Review began 09/30/2026
Review ended 10/18/2026
Published 10/21/2026

© **Copyright** 2026
Khurtsilava et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118338

Ia Khurtsilava [1, 2], Ekaterine Gozalishvili [1], Darejan Kanjaradze [3], Sophiko Osiashvili [1], Teimuraz Mikeladze [4]

1. Pediatrics, Petre Shotadze Tbilisi Medical Academy, Tbilisi, GEO 2. Pediatrics, Tbilisi Pediatric Private Clinic, Tbilisi, GEO 3. Pediatric Intensive Care Unit, Tbilisi Pediatric Private Clinic, Tbilisi, GEO 4. Pediatrics, University of Georgia, European University, Tbilisi, GEO

Corresponding author: Ia Khurtsilava, ia.khurtsilava@gmail.com

Abstract

Kingella (K.) kingae is an important cause of osteoarticular infections (OAIs) in young children, particularly between 6 and 36 months of age. However, these infections can be difficult to recognize because affected children often have mild systemic symptoms, only moderate elevations in inflammatory markers, and negative conventional cultures. Diagnosis is especially challenging in settings where *K. kingae*-specific molecular testing is not available.

We describe an 18-month-old girl who developed refusal to bear weight and pain on movement of the left hip following a recent febrile respiratory illness. Despite remaining relatively well clinically, she had persistent limitation of hip movement, elevated inflammatory markers, progressive thrombocytosis, and a hip joint effusion on ultrasonography. These findings raised concern for septic arthritis. Intravenous ceftriaxone was started, followed by rapid resolution of fever and gradual improvement in mobility. Serial ultrasonography showed reduction of the joint effusion, and the child subsequently regained independent walking. Blood cultures remained negative. Joint aspiration was not performed because the effusion was small and the child was already showing clear clinical and laboratory improvement. As *K. kingae*-specific polymerase chain reaction (PCR) testing was not available locally, microbiological confirmation could not be obtained, and the diagnosis remained presumptive.

To the best of our knowledge, this is the first suspected case of *K. kingae* septic arthritis reported from Georgia. The case highlights an important diagnostic challenge: septic arthritis caused by *K. kingae* may present with relatively mild systemic findings and can easily be mistaken for transient synovitis or remain classified as a culture-negative infection. Greater awareness of this pathogen and wider availability of molecular testing could improve diagnosis, allow more targeted antimicrobial treatment, and help clarify the currently unknown burden of *K. kingae* OAIs in Georgia.

Categories: Pediatrics, Orthopedics, Infectious Disease
Keywords: kingella kingae, k. kingae, kocher criteria, osteoarticular infections, septic arthritis

Introduction

Septic arthritis in children is a serious osteoarticular infection (OAI) that requires prompt recognition and treatment to reduce the risk of joint damage and long-term complications. It most often develops through hematogenous spread of bacteria. Although relatively uncommon, with an estimated annual incidence of 4-5 cases per 100,000 children in developed countries, delayed diagnosis and treatment may lead to cartilage damage, growth disturbance, and permanent joint dysfunction. The hip, knee, and ankle are most frequently affected, with typical manifestations including fever, joint pain, restricted movement, limping, or refusal to bear weight [1].

Staphylococcus (S.) aureus has traditionally been considered the predominant cause of pediatric septic arthritis. However, the introduction of molecular diagnostic techniques has substantially changed our understanding of the etiology of OAIs in young children. *Kingella (K.) kingae*, a fastidious Gram-negative coccobacillus, is now recognized as a leading cause of OAIs in children younger than 4 years, particularly between 6 and 36 months of age. Septic arthritis is its most common osteoarticular manifestation, accounting for approximately 53%-82.8% of reported *K. kingae* OAIs [1,2].

Unlike infections caused by more virulent organisms such as *S. aureus*, *K. kingae* septic arthritis often has a subtle clinical presentation. Children may appear relatively well, fever may be absent or low-grade, and inflammatory markers may be only moderately elevated despite significant functional impairment. This presentation may resemble transient synovitis or other nonbacterial musculoskeletal conditions, making early recognition challenging [1,2].

Diagnosis is further complicated by the fastidious nature of *K. kingae*. The organism grows slowly and poorly when synovial fluid or bone exudate is plated directly onto routine solid media. Colonies are small and may take 48-72 hours to appear, even in a CO_2-enriched atmosphere. Synovial fluid is thought to contain factors that inhibit its growth, and the bacterial inoculum in infected joints is often low. Recovery improves considerably when specimens are inoculated into aerobic blood culture bottles, which dilute these inhibitory factors. Even so, many cases remain culture-negative. On Gram stain, the organism appears as Gram-negative coccobacilli in pairs or short chains, which may resist decolorization and be overlooked. Bacteremia is usually transient and low-grade, so blood cultures are also frequently negative [2,3]. Molecular techniques, particularly *K. kingae*-specific polymerase chain reaction (PCR), have considerably improved detection and suggest that the burden of *K. kingae* infection was historically underestimated [2]. In settings where molecular testing is unavailable, cases may therefore remain undiagnosed and be classified as culture-negative OAIs.

We report a case of septic arthritis in an 18-month-old child with clinical and laboratory features suggestive of *K. kingae* infection and a rapid, favorable response to ceftriaxone. Although microbiological confirmation was not possible, this represents, to the best of our knowledge, the first suspected case of *K. kingae* septic arthritis reported from Georgia. This case highlights an important diagnostic consideration in settings where pathogen-specific molecular testing is unavailable. It underscores the need for increased clinical awareness and improved laboratory diagnostic capacity for *K. kingae*, particularly in young children presenting with Gram stain-negative or culture-negative OAIs.

Case Presentation

A previously healthy, 18-month-old girl presented to the emergency department with recurrent high-grade fever. Her recent medical history was notable for an upper respiratory tract infection associated with fever up to 39-40 °C for 3 days, followed by a maculopapular rash. Based on the clinical course, roseola infantum had been suspected, and the child had been managed conservatively with antipyretics and adequate hydration. After a two-day afebrile period, however, high-grade fever recurred, prompting presentation to the emergency department.

On admission, the child was irritable and had bilateral conjunctivitis with purulent yellow discharge and clear rhinorrhea. Cardiovascular and respiratory examinations were unremarkable. She was able to eat and drink adequately and cried with tears, with no clinical evidence of significant dehydration. Bowel movements and urination were normal. There were no meningeal signs or other clinical findings suggestive of a central nervous system infection. The patient was fully immunized according to the national vaccination schedule, with the most recent vaccination, measles-mumps-rubella (MMR), administered 25 days before presentation. Prior to admission, her primary care physician had prescribed oral amoxicillin, which was initially continued after hospitalization.

During the following three days, the fever gradually resolved without escalation to intravenous antimicrobial therapy. Despite improvement in her temperature and preservation of her general condition and oral intake, the child subsequently refused to stand or walk and was unable to bear weight on the left lower extremity. There was no visible swelling, erythema, or increased local warmth over the affected hip or surrounding tissues. Given the recent viral illness, absence of high fever, and initially reassuring inflammatory parameters, transient synovitis was considered.

Although the patient's mother requested discharge following resolution of the fever, repeat laboratory investigations demonstrated marked thrombocytosis and an elevated erythrocyte sedimentation rate (ESR), prompting further evaluation. On physical examination, passive rotation of the left hip elicited significant discomfort and crying. Ultrasonography of the hip subsequently demonstrated joint effusion (Figure *1*). During this period, the patient's temperature again increased mildly to 37.8 °C.

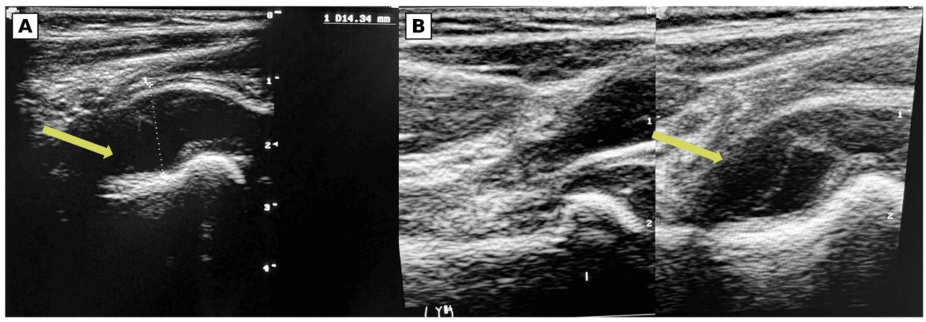


FIGURE 1: Hip ultrasonography demonstrating joint effusion

(A) Marked capsular distension with an anechoic fluid collection measuring approximately 14.3 mm. (B) Additional ultrasonographic views confirming intra-articular fluid surrounding the femoral head.

An additional source of irritability was identified when both the patient and her mother developed a pruritic rash that was particularly prominent at night. Dermatological assessment confirmed scabies, and appropriate treatment was initiated, resulting in substantial improvement in the child's generalized irritability. Nevertheless, the persistent refusal to bear weight and pain elicited by hip movement remained unexplained.

Following identification of the hip effusion, the differential diagnosis included septic arthritis, transient synovitis, osteomyelitis, and systemic inflammatory disease, including systemic juvenile idiopathic arthritis. There was no lymphadenopathy or hepatosplenomegaly. Neurological examination revealed no relevant abnormalities in the lower extremities. Additional laboratory investigations did not support a systemic inflammatory disorder. Serum albumin and total protein levels were within normal limits, procalcitonin was not elevated, and C-reactive protein (CRP) showed only a moderate elevation. Ferritin was mildly increased, which was interpreted as compatible with the ongoing inflammatory process rather than strongly suggestive of systemic juvenile idiopathic arthritis. Interleukin-6 was elevated at 54.6. In contrast to the relatively modest elevation of other inflammatory markers, the platelet count progressively increased during hospitalization (Table *1*). Table *2* and Table *3* list the inflammatory markers and biochemical findings.

Parameter	Day 1 (admission)	Day 3 (fever resolved; non-weight-bearing)	Day 5 (effusion on US; IV ceftriaxone started)	Day 7 (≈48 h of ceftriaxone)	Day 9 (switch to oral; discharge)	Reference range*
WBC (×10⁹/L)	12.7	11.6	14.88	11.93	8.7	6.0–17.0
Neutrophils (%)	50.4	43.6	58.5	44.2	34.5	20–50
Lymphocytes (%)	44.7	49.5	31	44.9	58.7	40–70
Hemoglobin (g/L)	99	94	98	92	91	105–135
Hematocrit (%)	29.4	28.5	27.1	26.4	28.4	33–39
Platelets (×10⁹/L)	217	514	629	890	741	150–450

TABLE 1: Serial complete blood count findings during the clinical course

Parameter	Day 1	Day 3	Day 5	Day 7	Day 9	Reference range
ESR (mm/h)	25	35	72	65	25	0–20
CRP (mg/L)	30.5	53.56	45.51	40.5	35.2	<5
Procalcitonin (ng/mL)	0.25	—	0.15	—	—	<0.5
Ferritin (ng/mL)	305.4	—	328	—	—	12–50
IL-6 (pg/mL)	54.6	—	—	—	—	<10

TABLE 2: Serial inflammatory markers during the clinical course

Parameter	Result	Reference range
Creatinine (µmol/L)	36.1	<88
AST (GOT) (U/L)	39.77	20–60
ALT (GPT) (U/L)	37.73	5–45
LDH (U/L)	343.3	120–300
Albumin (g/L)	38.95	35–50

TABLE 3: 3 Biochemical laboratory findings

AST (GOT), aspartate aminotransferase; ALT (GPT), alanine aminotransferase; LDH, lactate dehydrogenase

The combination of persistent inability to bear weight, painful restriction of hip movement, joint effusion, and elevated inflammatory markers raised significant concern for septic arthritis. Clinical assessment using the Kocher criteria further supported this possibility. However, the relatively mild systemic manifestations, absence of persistent high-grade fever, and modest inflammatory response were considered less typical of the classical presentation of *S. aureus* septic arthritis. Given the patient's age and clinical phenotype, *K. kingae* was considered a possible causative organism.

Intravenous ceftriaxone (100 mg/kg) was subsequently initiated. Within 36 hours of treatment, the fever completely resolved. Over the following 48 hours, the child's clinical condition and mobility progressively improved; she became increasingly active, was able to sit and play comfortably, and gradually began using the affected extremity. Serial laboratory investigations also demonstrated improvement in inflammatory parameters.

An attempt was made to establish a microbiological diagnosis. Blood cultures were obtained but remained negative. An oropharyngeal specimen was also collected and demonstrated bacterial colonization; however, pathogen-specific molecular confirmation of *K. kingae* could not be performed. At the time of the patient's hospitalization, a validated PCR assay or multiplex molecular panel including *K. kingae* was not available locally. Consequently, *K. kingae* infection could not be microbiologically confirmed, and the etiologic diagnosis remained presumptive, based on the patient's age, clinical presentation, laboratory profile, and response to appropriate antimicrobial therapy.

Serial ultrasonography of the affected hip demonstrated a marked reduction in the joint effusion in parallel with clinical improvement. Initially, joint aspiration was considered in consultation with orthopedic specialists; however, because the effusion was small and the patient demonstrated progressive clinical and laboratory improvement, the orthopedic team did not consider invasive sampling necessary.

As mobility improved and the child began bearing weight on the affected extremity, intravenous ceftriaxone was transitioned to an oral third-generation cephalosporin, with continued clinical and laboratory monitoring. The patient was subsequently discharged in good general condition. At discharge, she had substantially improved but had not yet completely returned to her baseline independent gait.

At follow-up five days after discharge, the child was afebrile, clinically well, and walking independently without apparent discomfort, including while playing with her favorite toy. The favorable clinical course,

normalization of systemic symptoms, progressive improvement in inflammatory markers, reduction of the hip effusion, and complete functional recovery supported the diagnosis of successfully treated bacterial septic arthritis.

Discussion

K. kingae is now recognized as a major cause of OAIs in young children, particularly between 6 and 36 months of age. Its importance was historically underestimated because of its fastidious growth and the low sensitivity of conventional culture methods. The introduction of nucleic acid amplification techniques, especially *K. kingae*-specific PCR, has substantially increased its detection and established the organism as a leading cause of septic arthritis in this age group [1,3,4,5]. These advances demonstrate how strongly the reported epidemiology of *K. kingae* depends on the diagnostic methods available [3,4,6,7].

The presentation of our 18-month-old patient was compatible with the clinical phenotype described for *K. kingae* infection. Following a recent febrile upper respiratory illness, she developed refusal to bear weight, painful restriction of hip movement, and ultrasonographic evidence of joint effusion. Despite significant functional impairment, systemic manifestations were relatively mild, procalcitonin was not elevated, CRP was only moderately increased, and her general condition remained relatively preserved. This pattern is characteristic of *K. kingae* OAIs, which often produce a less pronounced inflammatory response than infections caused by more virulent organisms such as *S. aureus* [6-9].

This relatively mild presentation can make *K. kingae* septic arthritis difficult to distinguish from transient synovitis. The Kocher criteria, originally developed to differentiate septic arthritis of the hip from transient synovitis, include fever >38.5 °C, inability to bear weight on the affected limb, ESR ⩾40 mm/h, and peripheral WBC count >12 × 10^9/L [6,7]. CRP >20 mg/L was subsequently added as an additional predictor in the modified criteria. Our patient demonstrated all of these predictors during her clinical course: she had documented fever up to 39-40 °C, persistent inability to bear weight on the affected extremity, an ESR that increased to 72 mm/h, a WBC count reaching 14.88 × 10^9/L, and a CRP concentration exceeding 50 mg/L. Thus, despite her relatively preserved general condition and modest systemic manifestations, her clinical and laboratory findings fulfilled the Kocher predictors and supported a diagnosis of septic arthritis rather than uncomplicated transient synovitis. These predictors were not all present at the same time in our patient, which is important when interpreting them. The high-grade fever (39-40 °C) occurred during the preceding febrile illness and had resolved before hip symptoms appeared. At the time the hip effusion was identified, her temperature was only 37.8 °C. At that point she fulfilled three of the four original Kocher criteria: inability to bear weight, ESR of 72 mm/h, and WBC count of 14.88 × 10^9/L. Her CRP of 45.51 mg/L also exceeded the 20 mg/L threshold of the modified criteria. Whether the earlier fever should be counted is uncertain, because it was temporally separate from the onset of joint symptoms. Without it, she met three of four original predictors and four of five modified predictors at the time of diagnosis. In the original derivation cohort, three predictors corresponded to a predicted probability of septic arthritis of approximately 93% [10]. This probability was lower in subsequent validation studies [11]. Therefore, although the criteria supported septic arthritis over uncomplicated transient synovitis, they did so with less certainty than would be implied by fulfilling all predictors simultaneously.

Nevertheless, the Kocher criteria should be interpreted cautiously in young children with suspected *K. kingae* infection. These criteria were developed before *K. kingae* was widely recognized as a major cause of OAI in this age group, and children with *K. kingae* septic arthritis may have a less pronounced febrile and inflammatory response than those infected with more virulent organisms such as *S. aureus*. Consequently, some children with *K. kingae* infection may fulfill fewer Kocher criteria despite having true septic arthritis [3,6-9]. Persistent focal findings, particularly refusal to bear weight, painful restriction of joint movement, and joint effusion, should therefore maintain clinical suspicion even when the systemic inflammatory response is relatively mild.

The clinical characteristics of our patient are broadly consistent with those reported in the literature on *K. kingae* OAIs. A systematic review of pediatric *K. kingae* musculoskeletal infections reported a mean age of approximately 18 months, closely matching the age of our 18-month-old patient, and found septic arthritis to be the most frequent musculoskeletal manifestation [7]. Previous series have also emphasized the relatively mild systemic presentation of *K. kingae* infection, with children often appearing well and demonstrating absent or modest fever and only moderate elevations in inflammatory markers [6,7]. In a series of PCR-confirmed *K. kingae* septic arthritis, the median age was 19 months, with a median WBC count of 12.4 × 10^9/L, ESR of 55 mm/h, and CRP of 24 mg/L [6]. Our patient similarly remained in relatively preserved general condition despite marked functional impairment, with refusal to bear weight and painful restriction of hip movement. Her inflammatory response was somewhat more pronounced, with CRP reaching 53.56 mg/L and ESR 72 mm/h, but remained compatible with the heterogeneous inflammatory profile described in *K. kingae* OAI [6,7]. A notable feature of this case was the progressive rise in platelet count, from 217 × 10^9/L at admission to a peak of 890 × 10^9/L on day 7, while CRP was already declining. This pattern is most consistent with reactive (secondary) thrombocytosis, which is by far the most common cause of an elevated platelet count in childhood. Infection is its leading trigger [9]. Reactive thrombocytosis is driven mainly by IL-6, which stimulates hepatic thrombopoietin production and, in turn, megakaryopoiesis. The elevated IL-6 level in our patient (54.6 pg/mL) is therefore mechanistically consistent with the

subsequent platelet rise. Because megakaryocyte maturation takes several days, platelet counts typically peak about one to two weeks into an inflammatory illness. This lags behind CRP and often coincides with clinical improvement, as in our patient. The platelet count therefore functions as a delayed acute-phase marker. In this case, it was the persistent thrombocytosis, together with the rising ESR, that prompted re-evaluation of a child whose fever had resolved and whose discharge had been requested. This led to recognition of the hip effusion. The teaching point is that a rising platelet count and ESR in a child with persistent focal musculoskeletal findings should not be dismissed as a non-specific post-viral phenomenon, even when the child appears well. The concurrent mild anemia (hemoglobin 91-99 g/L) was consistent with anemia of inflammation. Coexisting iron deficiency, which can also contribute to thrombocytosis, could not be excluded because ferritin is itself an acute-phase reactant. Reactive thrombocytosis in children rarely causes thrombotic complications and does not usually require antiplatelet therapy. In our patient, the platelet count began to fall by day 9. Importantly, the difficulty in obtaining microbiological confirmation in our case also parallels previous reports, in which conventional cultures frequently failed to identify *K. kingae* and molecular testing substantially increased diagnostic yield [6,7]. The patient's progressive clinical and functional recovery following appropriate β-lactam therapy is likewise consistent with the generally favorable outcomes reported for adequately treated *K. kingae* OAI [7].

Oropharyngeal colonization with *K. kingae* is most frequent during the same age period in which invasive disease occurs, with reported colonization rates approaching 10%-12% during the second year of life. Viral respiratory infections may disrupt the oropharyngeal mucosa, facilitating bloodstream invasion and subsequent hematogenous spread to bones and joints [3,7]. The preceding respiratory illness in our patient therefore adds epidemiological plausibility, although it cannot establish the causative organism.

The principal limitation of this case is the absence of microbiological confirmation. Blood cultures remained negative, and joint aspiration was not performed because the effusion was small and the child showed rapid clinical improvement. However, conventional culture has limited sensitivity for *K. kingae*, while inoculation of synovial fluid into blood culture bottles and, particularly, molecular testing substantially improve detection. Molecular studies have identified *K. kingae* in specimens that remained negative by conventional culture, demonstrating how reliance on routine microbiological methods may underestimate its contribution to pediatric OAIs [4,6,7].

This limitation is particularly relevant in Georgia. To the best of our knowledge, no pediatric *K. kingae* OAI has previously been reported from the country. This absence should not be interpreted as evidence that the organism is absent from the Georgian pediatric population. *K. kingae*-specific PCR is not routinely available in Georgia, making its true prevalence and contribution to pediatric OAIs impossible to determine. Some cases currently classified as culture-negative septic arthritis or osteomyelitis-and potentially some cases with an atypically prolonged course initially considered transient synovitis-may therefore represent unrecognized *K. kingae* infection. Similar increases in detection have been observed internationally following the introduction of molecular diagnostic methods [6,7]. In Georgia, however, this possibility remains hypothetical until systematic molecular testing becomes available.

The antimicrobial characteristics of *K. kingae* are also clinically relevant. The organism is generally susceptible to β-lactam antibiotics, including third-generation cephalosporins, such as ceftriaxone, whereas vancomycin and clindamycin do not provide reliable activity against this Gram-negative pathogen [4,7]. Empirical treatment of pediatric septic arthritis often prioritizes coverage of *S. aureus*, including methicillin-resistant strains. Awareness of *K. kingae* is therefore important when selecting empirical therapy in young children and may also help avoid unnecessarily prolonged anti-MRSA treatment once other pathogens have been reasonably excluded.

Our patient showed a rapid response after initiation of intravenous ceftriaxone, with resolution of fever, progressive improvement in mobility, reduction of the hip effusion, and eventual restoration of independent walking. This favorable course is consistent with appropriately treated *K. kingae* OAI [6,7]. However, response to ceftriaxone is not pathogen-specific and cannot establish the etiological diagnosis. The case should therefore be regarded as presumed rather than microbiologically confirmed *K. kingae* septic arthritis.

The importance of this case extends beyond the individual patient. It highlights a diagnostic gap in settings where molecular testing for *K. kingae* is unavailable and emphasizes the need for greater awareness among pediatricians, orthopedic surgeons, infectious disease specialists, and microbiologists. Introducing *K. kingae*-specific PCR or validated molecular assays into the investigation of culture-negative pediatric OAIs in Georgia could improve pathogen identification, support more targeted antimicrobial therapy, reduce unnecessary antibiotic exposure and investigations, and, importantly, help establish the true epidemiology of *K. kingae* infection in the country.

Conclusions

This case describes presumed *Kingella (K.) kingae* septic arthritis in an 18-month-old child. The diagnosis was supported by her age, a preceding respiratory illness, mild systemic features despite marked hip impairment, a joint effusion, reactive thrombocytosis, and a prompt response to ceftriaxone. However, microbiological confirmation was not obtained. Other bacterial pathogens partially treated by prior

amoxicillin, atypical transient synovitis, reactive arthritis, and early osteomyelitis cannot be excluded, so the case remains suspected rather than confirmed. To our knowledge, no *K. kingae* osteoarticular infection (OAI) has previously been published from Georgia. Greater clinical awareness and wider availability of *K. kingae*-specific PCR are needed to confirm future cases and define the organism's local epidemiology.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ia Khurtsilava, Sophiko Osiashvili, Ekaterine Gozalishvili, Darejan Kanjaradze, Teimuraz Mikeladze

Acquisition, analysis, or interpretation of data: Ia Khurtsilava, Sophiko Osiashvili, Ekaterine Gozalishvili, Darejan Kanjaradze, Teimuraz Mikeladze

Drafting of the manuscript: Ia Khurtsilava, Sophiko Osiashvili, Ekaterine Gozalishvili, Darejan Kanjaradze, Teimuraz Mikeladze

Critical review of the manuscript for important intellectual content: Ia Khurtsilava, Sophiko Osiashvili, Ekaterine Gozalishvili, Darejan Kanjaradze, Teimuraz Mikeladze

Supervision: Ia Khurtsilava, Sophiko Osiashvili, Ekaterine Gozalishvili, Darejan Kanjaradze, Teimuraz Mikeladze

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Pääkkönen M: Septic arthritis in children: diagnosis and treatment. Pediatric Health Med Ther. 2017, 8:65-8. 10.2147/PHMT.S115429
2. Cochard B, Gurbanov E, Bazin L, et al.: Pediatric osteoarticular Kingella kingae infections of the hand and wrist: a retrospective study. Microorganisms. 2023, 11:2123. 10.3390/microorganisms11082123
3. Yagupsky P: Kingella kingae: carriage, transmission, and disease. Clin Microbiol Rev. 2015, 28:54-79. 10.1128/CMR.00028-14
4. Centers for Disease Control and Prevention (CDC): Osteomyelitis/septic arthritis caused by Kingella kingae among day care attendees—Minnesota, 2003. MMWR Morb Mortal Wkly Rep. 2004, 53:241-3.
5. McElvania TeKippe E: The brief case: Kingella kingae septic arthritis in a 14-month-old child . J Clin Microbiol. 2016, 54:513-5, 822. 10.1128/JCM.02649-15
6. El Houmami N, Ceroni D, Codjo Seignon K, et al.: Acute septic arthritis of the knee caused by Kingella kingae in a 5-year-old Cameroonian boy. Front Pediatr. 2017, 5:230. 10.3389/fped.2017.00230
7. Williams N, Cooper C, Cundy P: Kingella kingae septic arthritis in children: recognising an elusive pathogen. J Child Orthop. 2014, 8:91-5. 10.1007/s11832-014-0549-4
8. Wong M, Williams N, Cooper C: Systematic review of Kingella kingae musculoskeletal infection in children: epidemiology, impact and management strategies. Pediatric Health Med Ther. 2020, 11:73-84. 10.2147/PHMT.S217475
9. Kocher MS, Zurakowski D, Kasser JR: Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinical prediction algorithm. J Bone Joint Surg Am. 1999, 81:1662-70. 10.2106/00004623-199912000-00002
10. Kocher MS, Mandiga R, Zurakowski D, Barnewolt C, Kasser JR: Validation of a clinical prediction rule for the differentiation between septic arthritis and transient synovitis of the hip in children. J Bone Joint Surg Am. 2004, 86:1629-35. 10.2106/00004623-200408000-00005
11. Caird MS, Flynn JM, Leung YL, Millman JE, D'Italia JG, Dormans JP: Factors distinguishing septic arthritis from transient synovitis of the hip in children. A prospective study. J Bone Joint Surg Am. 2006, 88:1251-7. 10.2106/JBJS.E.00216