REVIEW

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HALF A CENTURY OF KINGELLA KINGAE: A LITERATURE REVIEW FROM A PEDIATRIC RHEUMATOLOGY PERSPECTIVE

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Abstract. Objective: *To review the current knowledge on Kingella kingae infections in children from a pediatric rheumatology perspective, focusing on epidemiology, pathogenesis, clinical features, diagnostic methods, and treatment strategies.* ***Materials and methods:*** *A literature search was conducted using PubMed and Google Scholar (2010–2025) with keywords including "Kingella kingae", "pediatric rheumatology", "osteomyelitis", "septic arthritis", "spondylodiscitis", and "diagnostic techniques". Studies were selected based on clinical relevance, methodological quality, and contribution to understanding pediatric Kingella kingae infections, prioritizing recent reviews, original research, clinical guidelines, and seminal historical studies.* ***Results:*** *Kingella kingae has become a leading cause of osteoarticular infections in children under four years, often presenting with mild or afebrile symptoms and modest inflammatory markers. It can cause septic arthritis, osteomyelitis, spondylodiscitis, and tenosynovitis. Advances in nucleic acid amplification assays have improved detection over traditional culture methods. The RtxA toxin and outer membrane vesicles play key roles in pathogenicity. β-lactam antibiotics are highly effective, with short-course regimens increasingly favored. Remaining challenges include diagnostic delays, limited access to molecular testing, and a lack of standardized treatment guidelines.* ***Conclusion:*** *Early recognition of Kingella kingae infections is essential to prevent morbidity. Pediatric rheumatologists and orthopedics should maintain a high index of suspicion in children aged 6-48 months with subtle musculoskeletal symptoms. Further research is needed to refine diagnostic algorithms, optimize antibiotic regimens, and understand regional epidemiological variations.*

Key words: *Kingella kingae, pediatric rheumatology, osteomyelitis, septic arthritis, spondylodiscitis*

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INTRODUCTION

Kingella kingae (K. kingae) is a Gram-negative bacterium first isolated in 1960 by Elizabeth O. King at the Centers for Disease Control in Atlanta, GA [1]. It belongs to the Neisseriaceae family [2] and is a common component of the commensal oropharyngeal flora in young children [3]. Advances in culture techniques and molecular detection methods have revealed K. kingae as a frequent cause of

pediatric osteoarticular infections (POAIs) [4-6]. The estimated annual incidence of POAIs ranges from 1 to 13 cases per 100,000 children in developed countries, while in developing regions, it can reach up to 200 per 100,000 children [7]. These infections can disrupt bone development and lead to significant joint impairment [8]. Historically, Staphylococcus aureus was considered the predominant pathogen in POAIs [9]. However, recent advances in microbiological diagnostics have reshaped our understanding of the causative agents. The use of nucleic acid-based detection methods, such as real-time polymerase chain reaction (PCR), has significantly improved K. kingae identification. In recent decades, K. kingae has been detected in up to 82% of children under five years of age with septic arthritis (SA) [10, 11].

MATERIALS AND METHODS

This literature review aimed to synthesize current knowledge on pediatric *Kingella kingae* infections from a clinical and pediatric rheumatology perspective. Publications were included if they focused on K. kingae infections in children, addressed clinical features, diagnostics, or treatment strategies, and were published between January 2010 and May 2025. Review articles, original research, clinical guidelines, and historical studies were prioritized, while articles without abstracts or full-text access were excluded.

The literature search was conducted using PubMed and Google Scholar, with additional relevant publications identified through the reference lists of key articles. Keywords included: “Kingella kingae”, “pediatric rheumatology”, “osteomyelitis”, “septic arthritis”, “spondylodiscitis”, “tenosynovitis”, and “diagnostic techniques”. The initial search yielded 3,984 articles. Titles and abstracts were screened for relevance, followed by full-text review of potentially eligible studies, and 27 publications were ultimately selected that most closely aligned with the research focus.

Data were extracted from the studies regarding epidemiology, pathogenesis, clinical presentation, diagnostic approaches, and treatment strategies. Findings were summarized, with emphasis on clinically relevant insights for the pediatric practice. As this study involved only previously published literature, no ethical approval was required.

RESULTS AND DISCUSSION

Historical Perspective and Advances in Microbiology Techniques

The understanding of pediatric osteoarticular infections has evolved significantly over time. For decades, diagnosis relied on traditional culture-based methods to detect bacteria, mycobacteria, and fungi. However, culture-negative results were common, even when appropriate samples were collected. Studies indicate that 21% to 70% of septic arthritis (SA) cases and 24% to 68% of acute hematogenous osteomyelitis cases remained culture-negative despite thorough microbiological investigations [12, 13]. This posed challenges in selecting effective treatments, often necessitating an empirical approach without precise pathogen identification.

K. kingae is a fastidious microorganism, and classical culture methods have frequently failed to detect it [10]. Stahelin et al. [14] were the first to highlight the benefits of molecular techniques, specifically the universal 16S ribosomal DNA PCR method, in diagnosing K. kingae-induced culture-negative arthritis.

Since the early 2000s, nucleic acid amplification assays (NAAAs) targeting the 16S rRNA or species-specific genes have been introduced into clinical practice to detect fastidious bacteria in normally sterile body fluids and tissues. These assays enable rapid bacterial identification within hours, even in patients receiving antibiotic therapy [15].

Chometon et al. [15] reported that the use of species-specific PCR significantly improved the detection of K. kingae in osteomyelitis and arthritis cases, increasing identified cases from 17 to 39. NAAAs are increasingly replacing classical culture methods, allowing for more reliable pathogen identification. Real-time PCR amplification of drainage fluid samples has shown that K. kingae can be detected for up to six days after antibiotic initiation [16, 17].

However, a key limitation of PCR-based diagnostics is the inability to provide information on antimicrobial susceptibility, necessitating alternative methods for guiding targeted therapy.

K. kingae: A New Key Player in Pediatric Osteoarticular Infections

Since the early 2000s, the number of OAIs attributed to K. kingae has risen dramatically. This increase is largely due to the widespread use of nucleic acid amplification assays (NAAAs), which have provided definitive evidence that K. kingae is now the leading cause of OAIs in children under four years of age [3-5].

Today, K. kingae must be recognized as the primary pathogen responsible for hematogenous infections of bones, joints, intervertebral discs, and tendon sheaths in children aged 6 to 48 months. These findings highlight the previously underestimated role of K. kingae in pediatric OAIs and underscore the importance of improved diagnostic techniques [4, 5].

A longitudinal study examining average IgG levels against K. kingae found that they are initially elevated at birth but gradually decline, reaching their lowest levels at 6 to 7 months of age. These low levels persist until around 18 to 24 months, after which serum IgG levels gradually increase [18, 19]. Consequently, children between 6 and 48 months of age are more vulnerable to invasive K. kingae infections, including osteoarticular infections.

Coulin B et al. have conclusively demonstrated, through PCR-based diagnostics, that Kingella kingae is the predominant bacterial pathogen implicated in osteoarticular infections in young children (Figure 1) [20]. It is now identified as the causative pathogen in 52% to 93.8% of microbiologically confirmed cases [21, 22].

These findings challenge the long-standing belief that Staphylococcus aureus is the predominant bacterial pathogen in pediatric OAIs across all age groups. In children under four years old, K. kingae has largely replaced S. aureus as the primary causative agent worldwide [21]. However, S. aureus remains the leading pathogen in OAI cases among children over four years old and adolescents.

Interaction via Outer Membrane Vesicles

Gram-negative bacteria naturally produce outer membrane vesicles (OMVs) during growth. These vesicles contain various bacterial components, including DNA, RNA, peptidoglycan, and enzymes [23]. In 2011, Maldonado et al. [24] were the first to identify OMV production in nine different K. kingae strains. Based on their findings, they classified the strains into two groups: high and low OMV producers. Notably, all K. kingae isolates from joint infections belonged to the high OMV-producing group, suggesting that OMV production may enhance the bacterium's ability to cause septic arthritis. Since K. kingae primarily causes osteoarticular infections, researchers examined the effects of OMVs on human osteoblasts and synovial cells. They observed increased levels

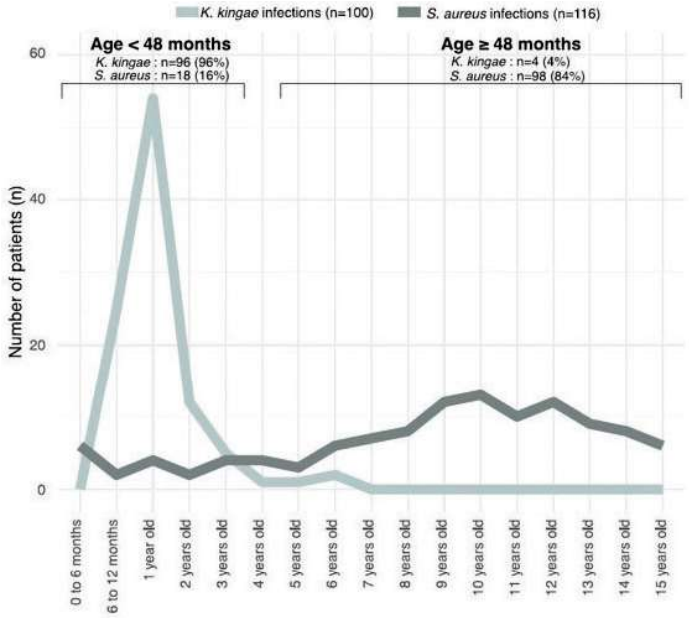

Fig. 1. Age Distribution of K. kingae and S. aureus Infections [20]

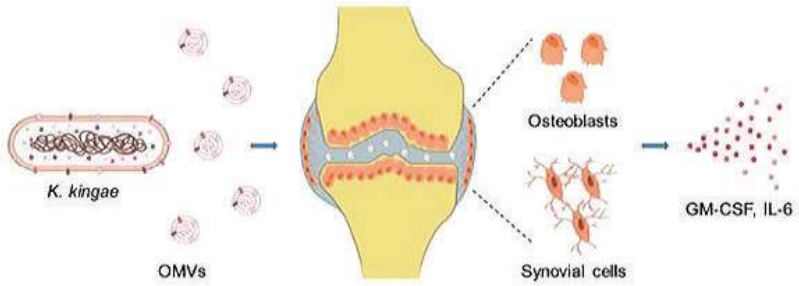

Fig. 2. The proposed mechanism of action of K. kingae OMVs in human bone [24]

of granulocyte-macrophage colony-stimulating factor (GM-CSF) and interleukins (IL) (Figure 2) [24]. However, it remains unclear whether these elevated cytokine levels result from OMV membrane components or the RtxA toxin.

Effects of Kingella kingae RtxA Cytotoxin on Host Cells

RtxA is a key virulence factor of Kingella kingae, belonging to a broad family of pore-forming RTX cytotoxins secreted by many Gram-negative pathogens. After binding to host cells, RtxA inserts itself into the cell membrane through an unexplored mechanism, forming pores. These pores trigger ion fluxes that disrupt normal cell physiology, ultimately leading to cell death. Although the exact role of RtxA in K. kingae pathogenesis remains poorly understood, recent findings suggest that the toxin may contribute to several key stages of infection. It may facilitate colonization of the upper respiratory tract, disrupt the respiratory epithelial barrier to promote bloodstream invasion, paralyze host innate immunity, and damage target tissues once the bacterium spreads to distant sites [25, 26].

Septic Arthritis due to Kingella kingae

Septic arthritis is the most common osteoarticular infection (OAI) caused by *Kingella kingae*. A number of studies reported that it accounts for approximately 55.3% to 82.8% of all K. kingae-related OAI cases [27-29]. The bacterium primarily affects large joints of the upper and lower extremities, such as the hip, knee, ankle, shoulder, and elbow [10, 28]. In contrast, infections of the hand or wrist are rare, with only a few reported cases [30-32].

In a large study of 335 children with OAI, Benoit Coulin et al. [20] identified 100 cases of K. kingae and 116 cases of Staphylococcus aureus infections (Figure 3) [20]. Their findings highlighted key differences between the two pathogens: S. aureus predominantly caused acute hematogenous osteomyelitis, whereas K. kingae was more frequently associated with septic arthritis. K. kingae infections most commonly affected the knee joint, while S. aureus had a more balanced distribution across different joints. S. aureus-induced osteomyelitis primarily affected the tibia and lower limbs. These findings underscore the distinct clinical presentations of K. kingae and S. aureus in pediatric osteoarticular infections.

Another study by Blais Cochard et al. [33] analyzed a large cohort of 497 patients with osteoarticular infections, identifying *Kingella kingae* in 249 cases. Among these, 33 cases involved K. kingae-related infections affecting the hand and/or wrist. The most common conditions observed were septic arthritis (69.7%) and tenosynovitis (51.2%).

Kingella kingae Osteomyelitis

K. kingae osteomyelitis primarily affects long bones such as the femur, tibia, humerus, radius, and ulna [3-5,19, 20]. However, it can also involve less commonly infected bones, including the sternum, clavicle, talus, and calcaneus [3-5,10].

This form of osteomyelitis accounts for approximately 15% to 31% of all K. kingae-related osteoarticular infections, with more than a quarter of cases occurring alongside septic arthritis [3, 5]. Notably, K. kingae osteomyelitis frequently affects the epiphysis and apophysis – sites that are rarely involved in infections caused by other pathogens [34, 35].

Kingella kingae Spondylodiscitis

Beyond arthritis and osteomyelitis, spondylodiscitis is one of the most frequent invasive Kingella kingae

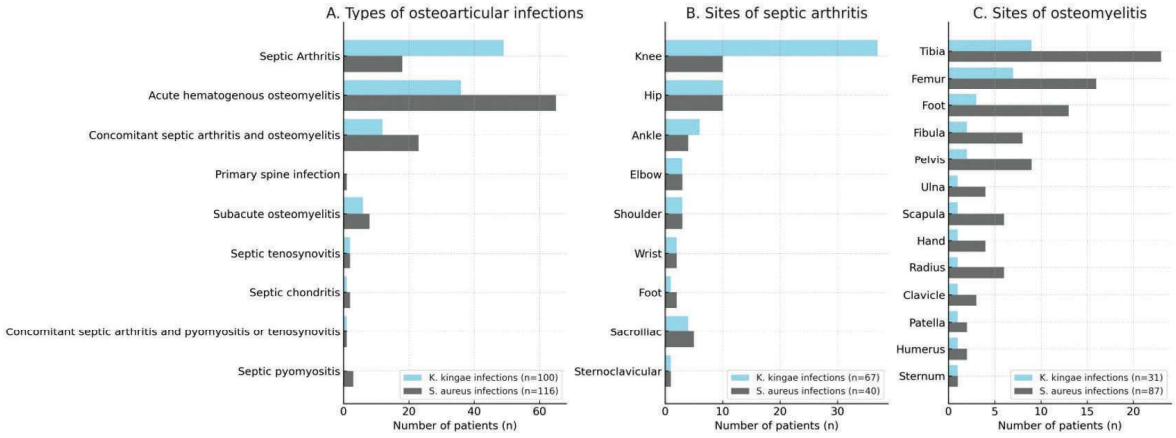

Fig. 3. Types of infection caused by S. aureus and K. kingae [20]

infections in children under four years old. Childhood spondylodiscitis represents a continuum of primary pyogenic spinal infections, ranging from discitis to spondylodiscitis and vertebral osteomyelitis, sometimes accompanied by soft tissue abscesses [36-38]. Three primary clinical forms of spondylodiscitis have been identified based on age: neonatal form – the most severe type, occurring in infants under six months, often associated with Staphylococcus aureus sepsis and multiple infectious sites; infantile form – the most common, affecting children between six months (when maternally derived immunity declines) and four years (this age group accounts for 80% of childhood spondylodiscitis cases, with K. kingae suspected to be the leading cause); and older childhood form – occurring in children over four years, this type is more frequently associated with fever and systemic illness, with S. aureus being the predominant pathogen responsible for vertebral osteomyelitis [39, 41].

A study by Moez Chargui et al. [42] examined 32 children with spondylodiscitis and found that fewer than 10% had a fever exceeding 38 °C at admission. Additional findings included: elevated ESR (> 20 mm/h) in 81.3%, abnormal platelet counts in 56.3%, normal white blood cell (WBC) counts (< 12,000/mm³) in 34.4% of cases, and normal C-reactive protein (CRP) levels (≤ 10 mg/L) in 31.3%. Notably, K. kingae was detected in almost 90% of confirmed cases using an oropharyngeal swab PCR test targeting the RTX toxin gene.

The mean delay in diagnosing spondylodiscitis was three weeks (range: 2-62 days), aligning with previous reports of delays from 4 to 6 months [37, 43, 44]. A number of studies have reported that the lumbar spine is the most commonly affected region, with more than 50% of pediatric spondylodiscitis cases occurring in this area [37, 39, 41].

Challenges in Diagnosing Kingella kingae Osteoarticular Infections

K. kingae osteoarticular infections (OAI) typically present with mild clinical symptoms and a moderate inflammatory response, making them difficult to identify using standard OAI diagnostic criteria. Most affected children appear in good overall condition and primarily exhibit musculoskeletal symptoms, such as limb favoring, limping, or refusal to bear weight.

Recent studies of large patient cohorts indicate that only 10% to 33% of children with K. kingae OAI have a fever exceeding 38 °C at admission [3, 19-21]. Additionally, most patients exhibit normal or near-normal WBC counts and CRP levels. A Swiss study of 151 patients reported a mean WBC count of 11.9×10⁹/L, with only 11.2% showing an elevated count. Although CRP levels were elevated (> 10 mg/L) in 61% of cases, the median CRP value was just 15.5 mg/L [3]. Several studies suggest that erythrocyte sedimentation rate (ESR) and platelet counts (> 400,000) may be the most sensitive inflammatory markers in K. kingae OAI cases [3-5, 23].

Blais Cochard et al. [33] reported 497 patients with OAI and identified K. kingae in 249 cases. The pathogen was detected using various methods, including oropharyngeal swab PCR (93.8%), PCR in bone or joint fluid (92.9%), bone or fluid culture (13.3%), and blood culture (10%). In children with culture-confirmed K. kingae arthritis, synovial fluid analysis frequently reveals low leukocyte counts. As a result, the commonly used diagnostic threshold of 50,000 WBC/mL in synovial fluid aspirates – typically applied to bacterial arthritis – may fail to detect K. kingae infections, making it an unreliable criterion in such cases [27].

Additionally, K. kingae arthritis often presents with mild symptoms, lack of fever, and minimal or no elevation of acute-phase reactants, failing to meet standard diagnostic criteria for septic arthritis. Alarmingly, Kocher's predictive algorithm suggests that approximately 75% of children with culture-confirmed K. kingae septic arthritis of the hip would likely be misclassified as having transient synovitis [45, 46].

These findings emphasize the need for improved diagnostic approaches to accurately detect K. kingae infections and prevent misclassification.

Radiologic Investigations in Kingella kingae Osteoarticular Infections

Osteomyelitis caused by *Kingella kingae* often presents with distinctive radiological features due to the pathogen's relatively low virulence. Compared to osteomyelitis caused by more aggressive pathogens, K. kingae infections tend to have a longer symptom duration before hospital admission and are associated with lower fever. As a result, K. kingae osteomyelitis is frequently overlooked or underestimated in its early stages, potentially progressing into subacute osteomyelitis.

Radiologically, K. kingae osteomyelitis may manifest as lytic bone lesions. Notably, the epiphysis and apophysis, rarely affected by other pathogens, are frequently involved in K. kingae infections [34, 35]. Because of this pattern, pediatric rheumatologists often consider lytic lesions in children under four years old as highly suggestive of subacute osteomyelitis due to K. kingae.

Magnetic resonance imaging (MRI) is widely regarded as an essential tool for diagnosing and managing osteoarticular infections, providing high accuracy in

distinguishing *Kingella kingae* OAI from those caused by Gram-positive cocci [47]. Key MRI findings suggestive of K. kingae infection include mild soft tissue reaction, absence or minimal bone reaction, and epiphyseal cartilage involvement.

Antibiotic Susceptibility and Treatment of Kingella kingae Osteoarticular Infections

The treatment of *Kingella kingae* osteoarticular infections typically involves intravenous antibiotic administration. K. kingae is highly susceptible to β-lactam antibiotics, including ampicillin and second- and third-generation cephalosporins [10, 48]. Reports of β-lactamase-producing K. kingae strains are extremely rare, with only a few cases documented, including three isolates from Iceland [49].

Given this susceptibility profile, K. kingae arthritis diagnosed by PCR can be confidently treated with a β-lactam antibiotic such as amoxicillin, allowing for an easy transition from intravenous to oral therapy.

The recommended duration of antibiotic treatment varies based on the type of infection – from 2 to 3 weeks for K. kingae arthritis, 3 to 6 weeks for osteomyelitis, and from 3 to 12 weeks for spondylodiscitis [10].

Future Research Directions and Knowledge Gaps

Despite growing recognition of *Kingella kingae* as a pediatric pathogen, key gaps remain. Most data comes from high-income countries, while incidence, clinical patterns, and colonization in low- and middle-income regions, including Bulgaria, are largely unknown. Diagnostic approaches, antibiotic regimens, and treatment durations vary internationally, and long-term outcomes are poorly studied. Important research questions include: the prevalence and clinical presentation of K. kingae infections in under-studied regions; how different diagnostics and antibiotic regimens affect outcomes; the main virulence factors and transmission dynamics; long-term functional and developmental consequences; and whether rapid or point-of-care diagnostics can improve early detection and treatment. Addressing these gaps will enhance prevention, diagnosis, and management worldwide.

CONCLUSION

Kingella kingae osteoarticular infections are a significant yet frequently underdiagnosed cause of skeletal infections in young children. Their mild clinical presentation and moderate inflammatory response often complicate early detection. However, advancements in molecular diagnostic techniques, particularly nucleic acid amplification tests, have greatly enhanced identification rates. Despite the pathogen‘s virulence, K. kingae OAI generally responds well to appropriate antibiotic therapy, leading to favorable outcomes when treated promptly. Increasing clinician awareness and refining diagnostic strategies are crucial for ensuring early recognition and effective management of this important pediatric pathogen.

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