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LETTER TO THE EDITOR

Letter to the Editor

Another Look: Is There a Flaw to Current Hip Septic Arthritis Diagnostic Algorithms?

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To the editor,

I read with interest the article by Uzoigwe in which the author critically assessed the current diagnostic algorithms recommended for differentiating between pediatric septic arthritis and transient synovitis in children [3]. The six evaluated studies show wide variation in the risk posed by each of the clinical (a body temperature $\geq$ 38.5°C and inability to bear weight) and laboratory (white blood cell count $> 12 \times 10^9$/L and erythrocyte sedimentation rate [ESR] $\geq$ 40 mm/hour) criteria included in the predictive scoring. Uzoigwe identified collinearity of the inflammatory predictors, inadequate sample size, and enrollment of an insufficient number of children with septic arthritis, as possible explanations for the incongruent between-studies results [3].

It should be noted, however, that the performance of any criterion for identifying diseased patients not only depends on the intrinsic properties of the investigated variable, but also on the reliability of the gold standard comparator. In the case of septic arthritis, the issue is complicated by the fact that, on average, in 33% (range, 16%–60%) of children with presumptive joint infections, the disease remains bacteriologically unconfirmed [2]. In full agreement with this concept, only 113 of 238 (47.5%) patients deemed to have septic arthritis in the studies reviewed by Uzoigwe grew an irrefutable pathogen in joint aspirates or blood cultures [3]. Therefore, children with symptoms of hip inflammation and synovial fluid (white blood cell count $> 50 \times 10^9$/L), but no positive isolation of bacteria from a normally sterile site were included in the "septic arthritis" category.

In recent years, *Kingella kingae*, a fastidious gram-negative member of the normal oropharyngeal flora has been recognized as the most common etiology of septic arthritis in children aged 6–36 months [4]. The organism requires inoculation of synovial fluid exudates into blood culture vials or the use of sensitive nucleic acid amplification assays for its detection [4], and it has been demonstrated that when these novel bacteriological methods are not used, many cases of joint infections caused by *Kingella kingae* will be missed and classified as "culture negative" septic arthritis [1, 4]. Additionally, children with *Kingella kingae* infections of the skeletal system frequently present with a mild clinical picture, requiring a high index of suspicion: the mean ± standard deviation body temperature is 38.4 ± 0.9°C, mean ESR is 44.1 ± 24.8 mm/hour, 21% of children have a blood white blood cell count $< 12 \times 10^9$/L, and 23% have less than 50×10^9/L white blood cells in the synovial fluid aspirate, mimicking transient synovitis [1, 4]. Because none of the studies evaluated by Uzoigwe employed blood culture vials nor nucleic acid amplification tests, it is entirely possible that

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many children with *K kingae* septic arthritis presenting with mild clinical symptoms, unimpressive laboratory markers of inflammation, and negative cultures, could have been misclassified as cases of "transient synovitis."

Although Uzoigwe is correct in that well-controlled and adequately powered studies are in need to constructing a reliable diagnostic algorithm aimed to differentiate between pediatric joint infection and benign synovitis, use of improved bacteriological methods is also crucial for defining a more accurate gold standard for pediatric septic arthritis.

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