Diagnosis of Periprosthetic Joint Infection Following Hip and Knee Arthroplasty

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KEYWORDS

• Periprosthetic • Joint • Infection • Diagnosis • Definition • Algorithm • Serology • Aspiration

KEY POINTS

- In an effort to establish clear diagnostic criteria for periprosthetic joint infections, this article proposes a modification of the currently established American Academy of Orthopaedic Surgeons algorithm.
- A stepwise approach should be undertaken, starting with history, physical examination, radiography, erythrocyte sedimentation rate, and C-reactive protein level.
- If the diagnosis is still unclear, joint aspiration with analysis of synovial leukocyte count, polymorphonuclear cell percentage, leukocyte esterase levels, and pathogen cultures should be obtained.
- In the case of indolent infections, newer diagnostic modalities, such as alpha-defensin or interleukin-6, show great potential to complement current techniques in future clinical practice.

INTRODUCTION

Total hip arthroplasty (THA) is one of the most successful operations in the history of orthopedic surgery.[1] Modeled on the low-friction arthroplasty introduced by Sir John Charnley in 1961, the modern THA has relieved pain and improved quality of life for millions of individuals worldwide.[2]

Total knee arthroplasty (TKA) gained popularity shortly after the advent of the modern THA, and, in 1972, Insall[3] introduced the total condylar prosthesis, which laid the framework for the modern TKA. Despite these developments, knee replacements were less successful than their hip counterparts because of complications with prosthetic design, and it was not until the 1990s that the total knee replacement was considered a successful operation.[4]

Presently, THA and TKA are among the most effective and widely performed surgical operations, with close to 1 million THAs and TKAs performed in the United States annually.[5,6] The number of joint replacements performed each year is also growing rapidly, with a 174% and 673% increase in annual procedures projected by 2030 for THA and TKA, respectively.[6] Given the considerable growth and success of total joint replacements over the past several decades, there has been a major focus on minimizing surgical complications in order to further improve long-term outcomes and drive down costs.

Periprosthetic joint infection (PJI) remains a major cause of failure in THA and TKA, despite an incidence of less than 2% in most national centers.[7,8] For both THA and TKA, PJI is the third leading cause of primary failure,[9,10] the

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leading cause of revision failure,[11,12] and the leading cause of early primary failure (<5 years),[10,13] which is a pertinent issue because PJI-associated revision results in a mortality 5 times greater than in revision following aseptic failure.[14] Furthermore, the financial burden of PJI is significantly greater than in uninfected cases, with a 76% and 52% increase in cost for infected THA and TKA, respectively.[15] With the significant added strain on patient outcomes and health care costs, the accurate and timely diagnosis of PJI is critical to the progressive improvement of modern arthroplasty.

DIAGNOSTIC CRITERIA FOR PERIPROSTHETIC JOINT INFECTION

The diagnostic requirements for PJI have been a source of uncertainty in the past, with conflicting criteria resulting in the inability to form a universal clinical definition. In 2011, the Musculoskeletal Infection Society proposed a unique set of PJI criteria[16] that, following further revision, was accepted by the US Centers for Disease Control and Prevention.[17] Of note, because of discrepancies in the magnitude of clinically meaningful biomarker increases in acute (<6 weeks) versus chronic (>6 weeks) PJI, the International Consensus Meeting (ICM) on PJI suggested specific biomarker threshold values that are reflected in the minor criteria of PJI[18] in the definition given later.

DEFINITION OF PERIPROSTHETIC JOINT INFECTION

Joint or bursa infections must meet at least 1 of the following criteria:

1. Two positive periprosthetic (tissue or fluid) cultures with matching organisms.
2. A sinus tract communicating with the joint (Fig. 1).
3. Having 3 of the following minor criteria:
   a. Increased serum C-reactive protein (CRP) level (>100 mg/L in acute PJI; >10 mg/L in chronic PJI) and erythrocyte sedimentation rate (ESR; not applicable to acute PJI; >30 mm/h in chronic PJI)
   b. Increased synovial fluid white blood cell (WBC) count (>10,000 cells/μL in acute PJI; >3000 cells/μL in chronic PJI) or ++ (or greater) change on leukocyte esterase test strip of synovial fluid
   c. Increased synovial fluid polymorphonuclear neutrophil percentage (PMN%) (>90% in acute PJI; PMN% >80% in chronic PJI)
   d. Positive histologic analysis of periprosthetic tissue (>5 neutrophils [PMNs] per high-power field [HPF])
   e. A single positive periprosthetic (tissue or fluid) culture

Fig. 1. Typical physical examination findings for PJI after TKA. Note the opening and drainage at the distal aspect of the incision.

Even with the establishment of firm diagnostic criteria, the ICM noted that, if clinical suspicion for PJI is high, further diagnostic evaluation should commence even if the criteria listed earlier are not met in full.[18] For this reason, risk stratification, based on patient history, physical examination, and joint radiographs, is critical to establishing a diagnosis in cases lacking a straightforward diagnosis.

Also of note, The Society of Unicondylar Research and Continuing Education suggested that these criteria, including ESR and CRP threshold values, can also be used in suspected PJI following unicompartmental knee arthroplasty (UKA), but that the aspiration biomarker thresholds in UKA can deviate significantly from the ICM values for TKA.[19]

DIAGNOSTIC MODALITIES IN SUSPECTED PERIPROSTHETIC JOINT INFECTION

Before constructing a stepwise approach to diagnosing PJI, it is essential to understand the clinical