Prosthetic Joint Infections

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KEYWORDS

- Prosthetic joint infection • Diagnosis • Suppressive antibiotics
- Irrigation and debridement • One-stage exchange arthroplasty
- Two-stage exchange arthroplasty

KEY POINTS

- The diagnosis of prosthetic joint infection is multifactorial, and is based on history, physical examination, serum laboratory tests, synovial fluid analysis, microbiology results, and biomarkers from the serum and synovial fluid.
- Patients with prosthetic joint infections who have high medical comorbidities that make them poor candidates for surgical intervention may be treated with suppressive, oral antibiotics.
- Acute prosthetic joint infections that occur less than 4 weeks after the initial surgery or 4 weeks within an acute hematogenous event may be treated with irrigation and debridement, although the success rate may not be as high as other treatment options.
- Chronic prosthetic joint infections that occur greater than 4 weeks after the initial surgery may be treated by 1-stage or 2-stage exchange arthroplasty.
- One-stage exchange arthroplasty is beneficial, because only 1 surgery is required, but the success rate may be lower than 2-stage exchange arthroplasty, which requires 2 procedures and a period with an antibiotic cement spacer and intravenous antibiotics.

INTRODUCTION

Periprosthetic joint infections (PJIs) are devastating complications after total joint arthroplasty, which lead to increased morbidity and mortality.[1] Treatment options for PJIs include medical management with chronic suppressive antibiotics, as well as surgical treatment with irrigation and debridement (I&D) for acute infections, and 1-stage or 2-stage exchange arthroplasty for chronic infections. The pros and cons of each surgical approach, along with the complications, postoperative care, and comparison of outcomes, are discussed in this article.

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DIAGNOSIS OF INFECTION

The diagnosis of infection requires a multifactorial approach. The patient must first be assessed by performing a complete history and physical examination. Signs of inflammation at an operative site include rubor (redness), tumor (swelling), calor (increased heat), dolor (pain), and functio laesa (loss of function), which may be manifest with decreased range of motion, pain with ambulation, and difficulty walking up and down stairs, when a previous problem did not exist. Patients may also present with evidence of a sinus tract, which is pathognomonic for diagnosis of a PJI. Radiographic analysis may show evidence of prosthetic loosening and bone loss if an indolent infection has been present for some time.

Diagnosing PJI is also performed by laboratory tests, looking at acute phase reactants like erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). However, these laboratory test results can be increased in any inflammatory condition and are not specific to PJI. Thus, a joint aspiration and analyzing the synovial fluid for white blood cell (WBC) count and neutrophil percentage (% PMN) and microbiology culture results can provide a more concrete diagnosis of PJI. It is recommended that a minimum of 3 cultures be taken for analysis and that the cultures be taken of tissue and not swabs.[2,3]

Using these factors, the Musculoskeletal Infection Society developed a definition of PJI based on major and minor criteria, of which the 2 major criteria are the presence of a sinus tract communicating to the implant and positive culture taken from separate samples. Patients may also meet 4 of 6 minor criteria to be diagnosed with a PJI: (1) increased ESR or CRP level, (2) increased synovial WBC count, (3) increased synovial % PMN, (4) purulence in the joint, (5) positive culture taken from 1 sample, and (6) greater than 5 neutrophils per high power field under a 400× magnified microscope.[4,5] These factors should be combined to determine the diagnosis of PJI, as shown in **Fig. 1**.

Newer biomarkers for determining infection are being investigated. Synovial fluid cytokines, such as interleukin 1β (IL-1β), IL-6, IL-8, tumor necrosis factor α (TNF-α), interferon δ, and vascular endothelial growth factor, are increased in inflammatory conditions such as PJI.[6] Also, synovial fluid biomarkers with antimicrobial function such as CRP, leukocyte esterase, α-defensin, human β-defensin-2 (HBD-2) and HBD-3, and LL-37 may be increased.[7–9] Serum biomarkers such as procalcitonin, IL-6, TNF-α, and soluble intercellular adhesion molecule-1 (sICAM-1) may also be elevated in PJI.[10]

MEDICAL TREATMENT OF INFECTION

The gold standard for treatment of infection is surgical management, as described later, but some patients have high medical comorbidities that make them poor candidates to undergo surgical intervention. These patients, or patients who refuse surgical treatment, can be managed with chronic suppressive antibiotics, which may not fully eradicate the infection but may reduce the bioburden enough so that the patient's innate immune system can continue to fight the infection. Rao and colleagues[11] reported that a combination of surgical debridement and short course (4–6 weeks) of intravenous antibiotics followed by chronic oral antibiotic suppression eradicated infection in 86% of patients at 5 years and in 69% of staphylococcal infections at 5 years, when using their organism-specific treatments.

SURGICAL TREATMENT OF INFECTION

The mainstay of PJI treatment is surgery. Depending on the chronicity of an infection, an acute PJI that presents less than 4 weeks since the index surgery or within 4 weeks