CHAPTER 49

SEPTIC ARTHROPATHY

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INTRODUCTION

Septic arthritis is defined as inflammation of a joint caused by pus-forming bacteria.[1] Terming all incidence of joint sepsis as arthritis is excessive and inaccurate. Septic arthropathy is a more appropriate nomenclature for the acute presentation of joint sepsis, with arthritis reserved for the late-stage sequelae of joint sepsis resulting in joint destruction with long-term dysfunction. This article will use these terms accordingly.

Septic arthropathy is a relatively rare, but disabling disorder. Its incidence varies from 2 to 5 per 100,000 in the general population, increasing in rheumatoid arthritis patients to 28 to 30 per 100,000, and in patients with joint prosthesis to 40 to 68 per 100,000.[2] Patients with a history of preexisting arthritis, previous intravenous drug use, sexually transmitted diseases, diabetes mellitus, or other infections are at an increased risk for developing bacterial arthropathy. If the diagnosis and initiation of treatment of septic arthropathy is not made readily, permanent joint destruction and osteomyelitis may occur. Irreversible loss of joint function occurs in 25-50% of patients.[3] Septic arthropathy, particularly in the foot and ankle, poses a number of diagnostic challenges. These challenges and the recommended treatment will be reviewed in this article.

PATHOGENESIS

Joint sepsis develops from 4 conventional pathways. Most relevant to the foot and ankle practitioner is direct inoculation or implantation of organisms into a joint. This can occur from any number of situations including penetrating injuries ranging from the classic rusted nail to less common tooth-pick, biting injuries, bullet or knife-wounds, and other sharp objects.[4] Septic arthropathy from direct implantation can also occur in clinical scenarios including joint aspiration or injection of corticosteroid, particularly in the immunocompromised patient.[3]

Less common modes of extension include contiguous spread, postoperative and hematogenous spread. Most common in the diabetic population is the spread of infection from a contiguous source, occurring as a direct extension of an organism from an adjacent area of cellulitis, abscess, or osteomyelitis is seen more frequently in the foot and ankle than in more proximal joints.[5,6] Postoperative joint sepsis most commonly occurs in total joint replacements around the prosthetic joint.[5] Finally, joint sepsis can be spread by hematogenous spread, which involves the transfer of the infectious organism through the vasculature with implantation of the organism within the synovium or vascular beds of the articular surface.[5] This form is more commonly seen in children.[6]

CLINICAL PRESENTATION

Non-gonococcal septic arthropathy typically presents with the sudden onset of an erythematous, edematous, exquisitely painful joint with limited range of motion. Both children and adults present with fever in up to 80% of cases and with monoarticular involvement in 90% of cases.[2] Polyarticular septic arthropathy commonly occurs in immunocompromised hosts.[3] The knee, hip, and ankle joints are most frequently affected, with between 10 to 15% of cases involving the ankle.[7] Most patients with septic arthropathy have an acute presentation, but infections with neisseria, as well as mycobacterial and fungal infections may have a prolonged duration of symptoms.[2]

Among young adults in the US, disseminated gonococcal infection (DGI) is the most common form of septic arthropathy.[3] DGI commonly presents as migratory polyarthralgias, tenosynovitis, dermatitis, and fever.[3] In contrast to non-gonococcal infections, less than 50% of these have purulent joint effusions. The knees and wrists are most commonly affected.[3]

LABORATORY FINDINGS

In non-gonococcal bacterial arthropathy, both adults and children typically have an elevated ESR, but this test is noted to be elevated in a number of inflammatory processes. Children tend to have increased WBC count with elevated PMNs; by contrast, adults display little or no elevation of WBCs.[2]

Definitive diagnosis requires examination of synovial fluid from the inflamed joint. Clinical suspicion for septic arthropathy necessitates immediate joint aspiration. The location of the involved joint is usually apparent based on

clinical evaluation; however in the midfoot this can be difficult, and fluoroscopic or computed tomography (CT) guidance may be necessary.[5] A 22-gauge needle is recommended for aspiration in the foot and ankle, however an 18-gauge needle may be needed depending on the thickness of the aspirate.[6]

The synovial fluid from non-gonococcal bacterial arthropathy typically appears turbid or purulent.[2] A Gram's stain and culture of the fluid should be performed. Gram's stain is positive in 50% of cases;[1] and wound cultures yield a positive result in up to 90% of cases.[2] The synovial WBC count usually exceeds 50,000/mm[3] with greater than 75% PMNs. This, however, may also be found in rheumatoid arthritis and gout, which show similar elevations in WBC and PMNs.[2,8,9] Although not diagnostic, synovial fluid glucose is typically decreased, L-lactic acid levels elevated and protein levels are elevated as well in the presence of joint sepsis.[7,10] Blood cultures should also be obtained and are positive in 50-70% of cases of non-gonococcal septic arthropathy.[2]

The synovial fluid Gram's stain in gonococcal arthropathy is positive in less than 25% of cases and culture yields a positive result in 50%.[3] In DGI, blood cultures and skin lesions also frequently yield negative results.[3] However, genitourinary cultures are positive in up to 90% of patients with DGI and coupled with clinical suspicion are frequently relied upon for presumptive diagnosis.[3]

IMAGING

Imaging of a suspected septic joint begins with the radiograph. Symmetric soft tissue effusion around the effected joint is the earliest radiograph sign.[6] Such changes are most recognizable in the ankle joint, particularly on the lateral view.[6] The effusion may be the result of either thickened, inflammatory synovium or the presence of fluid.[5] With a small or tight joint capsule, a widening of the joint space may occur.[5] Occasionally gas may be demonstrated within the joint space, but a more common finding on plain films is articular cartilage destruction, which can occur within 2 to 3 days in septic arthropathy.[6] Articular erosions may begin centrally or along the margins of the joint.[3] The hallmark of septic arthritis is the loss of the white cortical line over a long contiguous segment; by contrast, inflammatory arthridities usually exhibit disruption along an eroded cortex.[5] In bacterial arthritis, these erosions are accompanied by a decrease in joint space.[5] If there is a delay in the diagnosis and initiation of treatment in bacterial arthropathy, the infection can spread along the margins of the bone resulting in fibrous or bony

ankylosis and the possibility of growth disturbances in children.[6]

Radionuclide imagining with three-phase bone scan may be useful in determining the extent of infection or with suspected osteomyelitis, but are of limited use in differentiating septic versus other inflammatory processes.[2,3,6] CT does have the ability to show subtle changes in the articular surfaces, but is usually reserved for aspiration guidance in the difficult to identify joints of the midtarsus.[6]

MR imaging should be used either to support the diagnosis of septic arthropathy or to help define the extent of involvement of the infectious process. Having a high sensitivity and low specificity, MRI is able to demonstrate changes consistent with septic arthropathy within 24 hours of onset; however, it is unable to differentiate septic fluid from nonseptic inflammatory fluid.[6] MR imaging is superior to other imaging modalities in its ability to demonstrate cartilage destruction.[5]

TREATMENT

After obtaining blood cultures, synovial fluid cultures and chemistries, treatment begins with the use of appropriate antibiotics, first empirically, then focused at the isolated organism (Table 1).

Local sensitivity patterns of pathogenic resistance should be considered when selecting the initial antibiotic(s). Local incidence of MRSA, as well as the clinical scenario must be taken into consideration. For example, in a patient with a penetrating injury caused by a rusty nail through the bottom of the shoe and sock, an

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Table 1

APPROPRIATE ANTIBIOTICS SHOULD BE FOCUSED AT THE ISOLATED ORGANISM

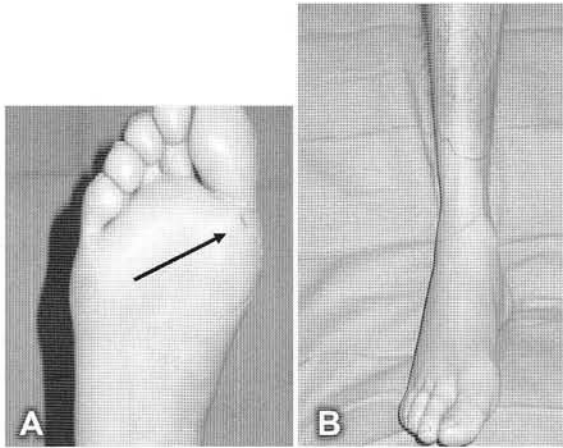| Organism	Population
S. Aureus	Overall
H. Influenzae	<2 years
Grp B Strep, Gram Negative Bacilli	<1 month
N. Gonorrhea	Adults <30 years
Strep Pneumoniae	Children with sickle cell
Salmonella	Children without sickle cell (unlike osteomyelitis)
Clostridium	Rare, but most common anaerobe

Figure 1A. Shows point of entry of rusty nail beneath the first metatarsal head 8 days prior. B. Presented to emergency room with painful erythematous, edematous right lower extremity. Noted on examination to be particularly taut over the first interspace.
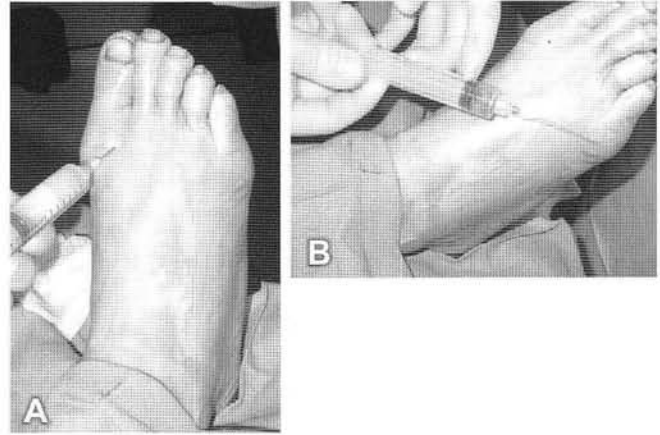

Figure 2A. The technique used to obtain an aspirate from within the first interspace. B. 2cc of fluid was obtained and sent for Gram's stain as well as culture and sensitivity.
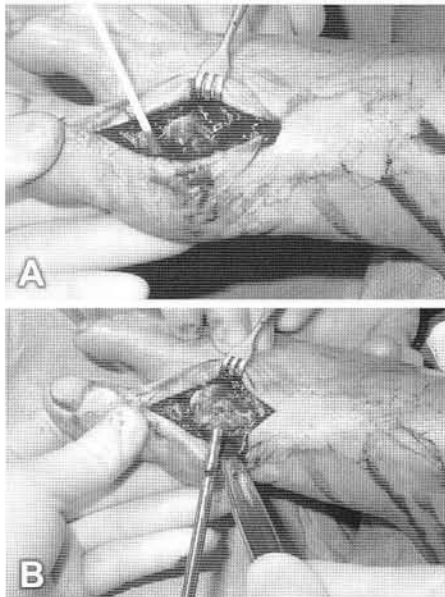

Figure 3A, B. Cultures and bone biopsy samples were taken intra-operatively. The bone biopsy sample was taken to measure the extent of the infection, and to guide the duration of antibiotic as well as surgical therapies.

antibiotic with pseudomonas coverage would be a necessity. Duration of antibiotic use varies depending on the clinical situation; however, soft tissue infections typically require 3-4 weeks of therapy with more extensive osseous infections requiring 6-8 weeks of therapy.[2]

In the event of prior surgeries and in the presence of implanted materials, the implants should be removed and future surgery planned following the appropriate antibiotic course.[3] Surgical drainage may be required in the presence of poor clinical response to antimicrobial therapy. There has been evidence that arthroscopic drainage allows visualization of joint tissue and effective lysis of adhesions with reduced healing and rehabilitation times;[2] however, traditional debridement remain the mainstay of therapy.

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