CHAPTER 53

PYOGENIC ARTHRITIS: A Discussion and Clinical Case Presentation

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An acutely inflamed joint may be the result of a number of different conditions. These include: septic arthritis; gout; pseudogout; acute flares of systemic conditions, such as rheumatoid arthritis and systemic lupus erythematosus; Lyme arthritis; hemophilia associated hemarthrosis; Charcot joints; or even Reiter's syndrome.

Patients may possess more than one problem that could produce an acutely inflamed joint. Podiatric physicians can appreciate the dilemma of treating a patient with a known history of crystalline arthropathy who presents with an acutely inflamed joint. Each new presentation should be viewed independently. A thorough examination and history should be the basis for any further diagnostic interventions such as joint aspiration and venipuncture for laboratory analysis. This will reduce the likelihood of a septic joint being misdiagnosed and treated as a recurrent episode of gout.[1]

PATIENT POPULATION

Pyogenic arthritis may occur in any individual, but it is more prevalent in specific patient groups. These groups include those patients who are immunocompromised, have a history of previous damage to the joint(s), or have other non-specific conditions such as substance abuse.

The immunocompromised patient group may consist of individuals with rheumatoid arthritis, tissue transplants, and even steroid-dependent asthma. It is well recognized that corticosteroids may mask the physiologic signs of inflammation. These patients often present with vague symptomatology, normal temperature, and no leukocytosis. A high index of suspicion may prevent the clinician from dismissing a potentially septic joint as an acute rheumatoid flare. Sharp et al. recognized this fact and stated many of the patients with rheumatoid arthritis in whom the diagnosis of septic arthritis was established had been considered to have an exacerbation of the underlying condition prior to performing joint fluid studies that were carried out "to be safe."[2] Moreover, the patient with poorly controlled diabetes mellitus may mount a decreased response to the infection on a systemic as well as the cellular level.

It has frequently been reported in the literature that previous articular damage may predispose a joint to a septic event. Cartilage defects in a damaged joint allow organisms to become situated easier than in a joint with congruous surfaces. In a damaged joint, infection may spread into adjacent osseous structures due to the lack of protection normally provided by an intact layer of cartilage. It should be recognized that the damage may have been due to physical trauma or secondary to a disease process such as gout, pseudogout, rheumatoid arthritis, or neuropathic collapse. In addition, there is the potential for physical damage to a joint from needle aspirations. Intra-articular steroid injections can also damage the articular surfaces due to crystalizations.

A separate group of patients at risk for developing joint sepsis are those with a substance abuse problem. They include drug abusers and alcoholics, as seen in the case below. Each of these groups are predisposed to certain types of infections, determined by their habits and habitats.

Finally, patients with systemic illnesses are at risk for the development of joint sepsis. Hemotogenous spread of bacteria may occur secondary to other infections, including bacterial endocarditis, intravenous line sepsis, urinary tract infection, or overlying decubiti.

JOINT DISTRIBUTION

A review of the literature reveals that the joint most commonly affected by septic arthritis is the knee, followed by the hip. Evaluation of the incidence in the foot is difficult because these joints are often excluded from studies. Furthermore, septic arthritis in the foot is generally due to the contiguous spread of bacteria from neuropathic ulcerations or implantation such as puncture wounds, as opposed

to hematogenous seeding. A careful evaluation of the involved joint(s) may provide insight as to the probable infecting organism.

Staphylococcus aureus, is the most commonly isolated organism in septic arthritis, and causes a monoarticular pyarthrosis in 79% of cases. However, patients with rheumatoid arthritis are more likely to develop polyarticular involvement, apparently due to the use of systemic corticosteroids.[3] One example of organisms commonly causing polyarticular involvement is the Neisseria species. In fact, with these bacteria, multiple joints are involved 72% of the time.[2]

ROUTE OF INFECTION

The most common pathway for infection of a joint is through hematogenous seeding. This is of particular interest and concern in the pediatric patient, as the blood supply to the metaphysis is contiguous with the joint. If the infection is not recognized early, there is the potential for joint destruction and subsequent limb length discrepancy.

Of specific interest to podiatric physician are joint infections caused by direct inoculation. These infections may result from the implantation of bacteria by puncture wounds into the small joints of the foot. A similar process can develop following injury to the hand caused by fights or animal bites.

Finally, septic arthritis can develop due to osteomyelitis of a bone adjacent to the joint. An example of this would be septic arthritis of the knee acquired secondary to osteomyelitis of the tibia.

DIAGNOSIS

Diagnosis of septic arthritis is often dependent on direct analysis of synovial fluid. This analysis should include culture and gram stain, glucose content, and if volume allows, a cell count.

TREATMENT OPTIONS

The appropriate treatment of septic arthritis usually involves parenteral antibiotic therapy. The choice of antibiotic is based on synovial fluid analysis, blood cultures, and the initial clinical impression. However, there is controversy regarding invasive treatment. Much of the literature supports serial joint aspirations, and advocates open arthrotomy only in cases where thick purulence can not be aspirated, or loculations are suspected. These recommendation are based upon poor results following open surgical drainage. However, the poor results noted in the literature include cases in which aspiration failed as the primary treatment before undergoing surgical intervention. Thus, the cause of the poor outcome may have resulted from the delay in surgical treatment, and from the surgical treatment itself.

Most authorities recommend surgical drainage of septic arthritis of the hip, especially in pediatric patients. The rationale is based on the difficulty of aspiration of the hip joint, as well as the high risk of subsequent osteomyelitis and growth disturbance.

CASE PRESENTATION

The patient concerned is a 50-year-old white male with a history of a previous injury to the right ankle sustained in 1991 in a motor vehicle accident. Treatment included casting, oral medications, and intra-articular injection therapy. The last injection was more than one year prior to the onset of the initial symptoms.

The patient's medical history was only significant for hypertension. Previous surgeries included an appendectomy, and removal of a submaxillary gland. The patient denied the use of tobacco or intravenous drugs and admitted only occasional intake of alcohol. In addition, the patient related that he was a monogamous homosexual.

The patient presented to the hospital for admission and surgical debridement of the right ankle following a previous outpatient joint aspiration. Gross purulence was obtained from the joint aspirate, and radiographic changes were noted on plain films that suggested pyogenic arthritis (Figs. 1-3).

Admission laboratory studies revealed an elevated white blood cell count of 18,500/mm3 with 75% PMNs, 15% bands, and 10% lymphocytes. Erythrocyte sedimentation rate (ESR) was 115mm/hour, and the chest x-ray revealed a left lower lobe pneumonia. Admission blood cultures were also obtained. All other pertinent laboratory values were relatively normal.

The patient was taken to the operating room where an anteromedial incisional approach was used to access the ankle joint (Fig. 4). After incision of the ankle joint capsule, a large amount of light brown seropurulent fluid was expressed from the

Figure 1. Preoperative AP radiograph of the right ankle. Note the lucency of the medial shoulder of the talus.

Figure 2. Preoperative lateral radiograph showing destruction of the superior surface of the talar dome.

Figure 3. Enlarged AP view illustrates subchondral lucency medially and centrally.

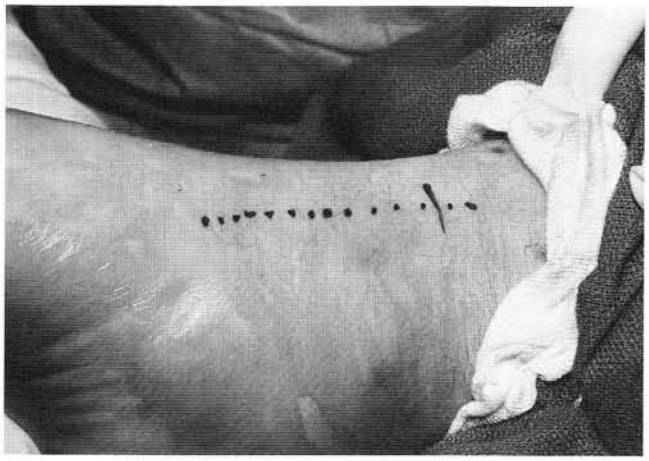

Figure 4. Proposed skin incision placed slightly posterior to the tibialis anterior tendon to avoid complications in wound healing.

joint (Fig. 5). Gram's stain of the fluid revealed many gram positive cocci in pairs and chains, and rare gram negative rods. Aerobic and anaerobic cultures were also obtained. Following aggressive irrigation of the joint, inspection of the articular surfaces revealed a moderate-sized undermined defect in the medial shoulder of the talus (Fig. 6). In view of the intraoperative and radiographic findings, bone biopsies of the talus (Fig. 7) and the tibia (Fig. 8) were obtained for microscopic examination.

Following incision and drainage, the wound was packed with 1/2" plain sterile gauze, and a dry sterile dressing was applied (Fig. 9). Intravenous antibiosis was initiated postoperatively using Timentin 3.1 g every 6 hours.

On the first postoperative day, the CBC showed a WBC of 12.3 with 87% PMNs, 0 bands, 9% lymphocytes, and 4% monocytes. Due to the patient's poor dentition, the infectious disease specialist ordered an echocardiogram to rule out subacute bacterial endocarditis as the cause of the septic ankle joint and pneumonia. The echocardiogram results could not rule out mitral valve vegetations and revealed a slight mitral regurgitation. The intraoperative cultures revealed heavy growth of alpha hemolytic strep after 24 hours.

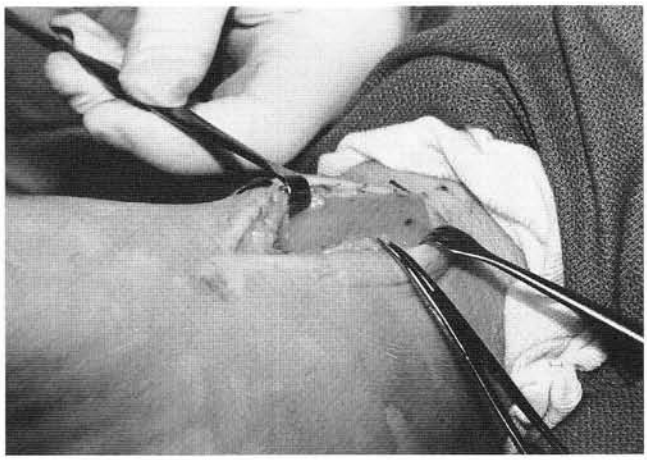

Figure 5. Gross purulence noted with incision of the ankle joint capsule.

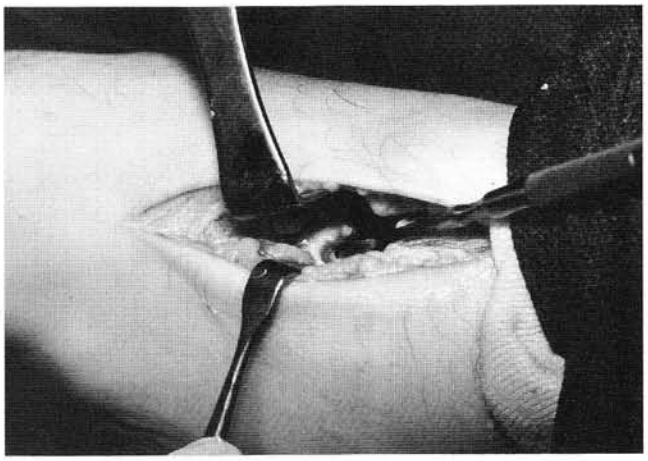

Figure 6. Probing the talar defect with a Freer elevator revealed a cavity approximately 6mm in depth.

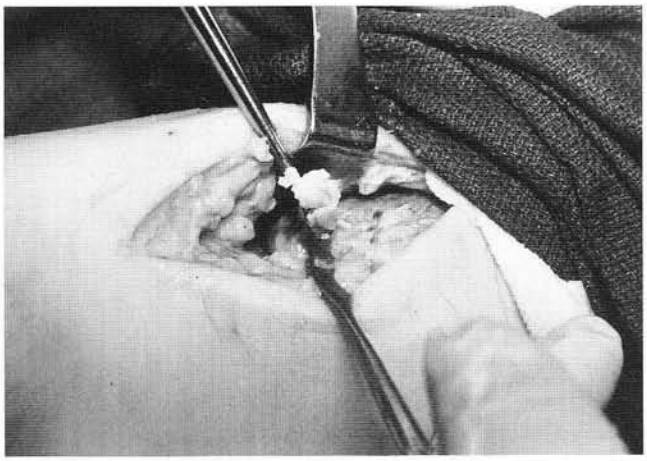

Figure 7. Curetted osteochondral talar fragments for pathologic examination.

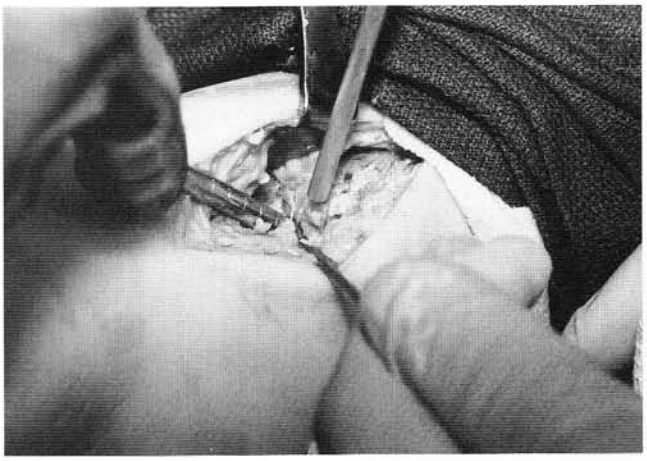

Figure 8. Harvesting of bone from tibia just proximal to joint for pathologic examination.

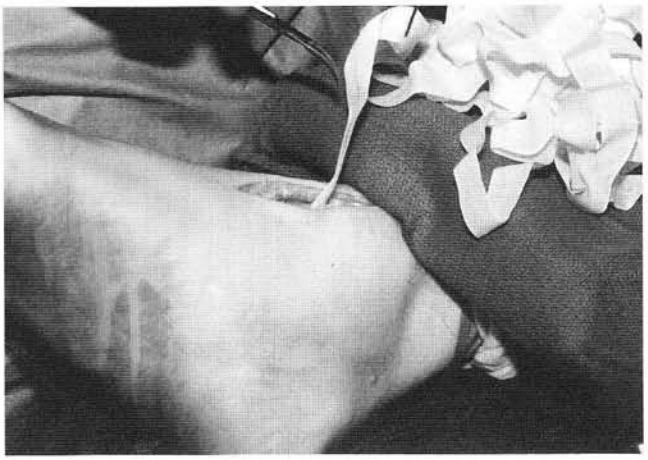

Figure 9. Packing of the wound with plain sterile packing.

On the second postoperative day, admission blood cultures revealed growth of alpha hemolytic strep. A dressing change was performed, and a moderate amount of brown drainage was present on the packing and dressing. The talus appeared grayish-brown around the osteocartilaginous defect. Although the wound margins bled freely and edema was markedly decreased, no granulation tissue was noted.

Postoperative day three revealed a WBC of 9.3 with 82% polys 12% lymphs and 5% monos. Infectious disease changed the antibiotic to IV Vancomycin due to the marginal clinical response of the wound (Fig. 10), as well as the sensitivities to the isolates which were identified as Strep pneumonia from the blood and wound cultures. Pathologic examination of bone biopsies showed evidence of acute osteomyelitis of the talus, but demonstrated no changes in the distal tibia consistent with infection. T-cell counts and HIV testing were also performed despite a low index of suspicion.

On day seven, the ESR was repeated and found to be 15 mm/hour. Final MIC/MBC information was returned and the Vancomycin was

Figure 10. Note residual hyperpigmentation in the retromalleolar space.

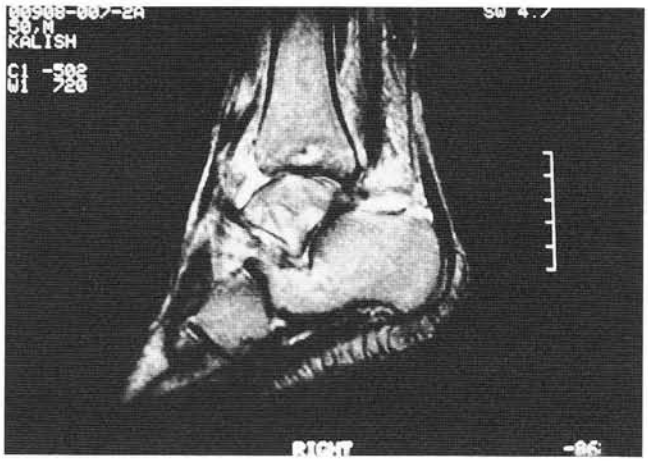

Figure 11. Follow-up MRI showing a high signal intensity at the tibial biopsy site, and talar involvement unchanged since surgery.

replaced with Zinacef. On the eighth postoperative day, the patient began to develop hallucinations and disorientation. For two days prior he had expressed great anxiety. Psychiatry consultation revealed that the patient routinely drank three shots of liquor and six beers a night. A diagnosis of late-stage alcohol withdrawal was made and treatment was instituted with Ativan and Haldol.

The wound appearance continued to improve over the next several days, and the patient was discharged after placement of a PICC line (peripherally inserted central catheter). Six weeks of parenteral antibiotic therapy for acute osteomyelitis/septic arthritis of hematogenous origin was administered.

Approximately three weeks after the initial surgery, the wound had improved to the point that delayed primary closure was planned. MRI examination was performed to check for signs of continuing infection (Fig. 11). However, no evidence of continued infection was noted and delayed primary closure was performed without complication.

DISCUSSION

This case illustrates an example of group B streptococcal arthritis. The causative organism seemed odd at first, since strep pneumonia is rarely a cause of joint sepsis in the general population. The picture only became clear with elucidation of social history (alcoholism). The case is of particular interest as the diagnosis of pneumonia and possible bacterial endocarditis were gained from the complete infection work-up. The key piece of clinical information in this case was the history of alcohol abuse, as strep pneumonia causing septic arthritis in a healthy individual is rare. It is also interesting to note that the patient fell into several risk categories including previous joint damage, previous intra-articular steroid injections, and alcohol abuse.

The successful diagnosis of septic arthritis is based on a high index of suspicion and early joint aspiration and fluid analysis. The importance of an accurate and complete history and physical examination cannot be over-stressed. This information will guide the initial therapy, and serve as an early predictor of the infecting organisms.

Appropriate therapy consists of intravenous antibiotics and appropriate joint drainage. The latter may be accomplished with serial aspiration, open surgical techniques, or arthroscopy. The particular case presented shows the complication of partial destruction of the talus due to a delay in presentation by the patient.

The patient is one year post-infection and is doing well in spite of losing approximately 15-20% of the articular surface of the talus.

REFERENCES

1. Baer PA, Tenenbaum J, Fam AG, Little H: Coexistent septic and crystal arthritis. Report of four cases and literature review. *J Rheumat* 13:604-607, 1986.
2. Sharp JT, Lidsky MD, Duffy J, Duncan MW: Infectious arthritis. *Arch Intern Med* 139:1125-1130, 1979.
3. Kelly PJ: Bacterial arthritis in adults. *Orth Clin North Am* 6: 973-981, 1975.
4. Obeid EM: Neisseria subflava causing septic arthritis of the ankle in a child. *J Infect Dis* 27:100-101, 1993.
5. Parisien JS, Shaffer B: Arthroscopic management of pyarthrosis. *Clin Orthop* 275:243-247, 1992.
6. Vartian C, Lerner PI, Shales DM, Copalakrishna KV: Infections due to Lancefield Group B Streptococci. *Medicine* 64:75-88, 1985.
7. Grippo GN, Genovese MN, Piccora R: Monarthritic Gonococcal arthritis involving the calcaneocuboid joint. *J Am Podiatr Med Assoc* 80:91-96, 1990.
8. Roca R, Yashikawa TT: primary skeletal infections in heroin users: A clinical characterization, diagnosis, and therapy. *Clin Orthop* 144:238-248, 1979.
9. Hirsch E, Sherman M, Lenet MD: Gonococcal arthritis - A case report. *J Am Podiatr Med Assoc* 79:190-194, 1989.
10. Herndon WA, Knauer S, Sullivan JA, Gross RH: Management of septic arthritis in children. *J Ped Orth* 6:576-578, 1986.
11. Barker CS, Symmond DP, Scott DL, Bacon PA: Joint sepsis as a complication of sero-negative arthritis. *Clin Rheumatol* 4:51-54, 1985
12. Ang-Fontz GZ, Rozboril MB, Thompson GR: changes in non-gonococcal septic arthritis: Drug abuse and MRSA. *Arthritis Rheum* 28:210-213, 1985.
13. Ho G, Gadbow JJ, Glickstein SL: Hemophilus Influenza septic arthritis in adults. *Sem Arthritis Rheum* 12:314-321, 1983.
14. Baker SB, Robinson DR: Sympathetic joint effusion in septic arthritis. *J Am Med Assoc* 240: 1989.
15. Lemont H, McCarthy D: Gonococcal arthritis: A case report. *J Am Podiatr Assoc* 70:279-282, 1980.
16. Gordon SL, Maisels MJ, Robbions WJ: Multiple joint infections with Enterobacter cloacae. *Clin Orthop* 125:136-138, 1977.
17. Kelly PJ, Martin WJ, Coventry MB: Bacterial arthritis in the adult. *J Bone Joint Surg* 52:1595-1602, 1970.
18. Brown PW; Arthrocentesis for diagnosis and therapy. *Surg Clin North Am* 49:1269-1278, 1969.