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Disappearing Acts: The Many Causes of Rapidly Destructive Arthritis

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Most of the destructive joint disease necessitating arthoplasty is the result of an insidious and protracted process that occurs over the course of many years. However, there are a variety of diseases that may result in a rapid progressive deterioration of a joint. We describe both the common and less common etiologies of rapidly destructive arthropathy that one should consider, with imaging examples, and present discriminative factors when present.
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Trauma

Posttraumatic osteoarthritis (PTOA) refers to osteoarthritis that arises following a well-defined precipitating event causing impactful loading of a synovial joint, soft tissue tears (ligament, meniscus, or capsule), or joint dislocation. To varying degrees, PTOA occurs in 20%-50% of patients after trauma. It is estimated that trauma accounts for approximately 12% of all cases of osteoarthritis involving the knees, hips, or ankles.[1] PTOA tends to occur in relatively younger patients, as when compared with all patients with osteoarthritis. As compared with "typical" osteoarthritis, it has been estimated that patients present approximately 9 years earlier with hip PTOA, 10.4 years earlier with knee PTOA, and 14 years earlier with ankle PTOA.[1] Moreover, ankle PTOA not only occurs much earlier than would typically be expected, but also occurs only 5-12 years post injury.[2]

In addition to direct damage to articular cartilage and subchondral bone because of trauma, a number of inflammatory processes have been identified in the early injury phase.[3] This includes the release of proinflammatory cytokines (interleukin [IL]-1, IL-6, and IL-8), which stimulate the release of tumor necrosis factor α and other chemokines, as well as anti-inflammatory cytokines (IL-10 and IL-1RA), which attempt to attenuate the response. It has been postulated that with increasing injury severity, subsequent joint incongruity or instability, and certain genetic factors, there may be persistent activation of the proinflammatory cytokines and progression to PTOA.[4]

Patients often present with increasing pain and decreased function of the involved joint, as well as overall poor health status. Common injuries associated with PTOA include anterior cruciate ligament disruption, meniscal tears, and intra-articular fractures.[5-7] Specifically, intra-articular fractures have been reported to increase the risk of PTOA approximately 20-fold.[8] PTOA can affect any synovial joint, with the highest reported risk being the tibial plafond (up to 75%) and distal radius (up to 65%).[8] The tibial plateau, distal femur, distal humerus, and acetabulum all have reported risks of approximately 35%-44%.[8]

Altered biomechanics after injury increase the risk of progressive joint degeneration. In the acute trauma setting, imaging is useful to assess the degree of potential fracture comminution, articular surface involvement, and articular surface step-off. In general, computed tomography [CT] provides more detail regarding the morphology of fracture as compared with radiography, and it often leads to alterations in management.[9] However, surgical stabilization may not necessarily be protective against the development of PTOA.[10] Follow-up imaging would demonstrate typical features of osteoarthritis with evidence of prior traumatic injury. (Fig 1) PTOA can develop in as little as 2 years following high-energy intra-articular fractures.[11]

Septic Arthritis

Septic arthritis is a disabling and life-threatening disease that destroys the hyaline articular cartilage within a joint within a matter of days of onset.[12-19] Once the process of articular cartilage destruction begins, the presence of viable microorganisms is no longer necessary for ongoing joint destruction to take place because of proteolytic enzymes and acute inflammatory cells that cause synovial necrosis.[15,20]

Although most cases of septic arthritis are because of hematogenous seeding of a joint,[13,15,19-21] it can also occur because of direct inoculation or through spread of a local infection such as adjacent cellulitis, bursitis, or osteomyelitis.[15,20,22,23] The most common pathogens include *Staphylococcus aureus*, group B streptococcus, and gonococcal arthritis.[12,16-21,23-26]

Risk factors include advanced age, malignancy, diabetes mellitus, rheumatoid arthritis or connective tissue disorders immunosuppression, chronic liver or renal disease or both, history

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of joint replacement, skin infection, intravenous drug abuse, alcoholism, or prior intra-articular steroid injection.[13,15-23,26]

Patients often present with a painful, swollen joint(s), and less commonly with systemic symptoms such as fever, sweats, and rigors or chills.[14,15,18,22,23] These symptoms are, however, non-specific, and may be present with alternative forms of arthropathy, such as osteoarthritis or crystal-induced arthropathy.[13-17,20,22,26-28] The knee reflects the most common joint involved with septic arthritis, followed by the hip.[13,15,17-19,20,23,26]

A variety of laboratory markers are used in effort to establish the diagnosis of septic arthritis. The erythrocyte sedimentation rate and c-reactive protein have a high sensitivity in the setting of septic arthritis ($>$ 90%); however, these tests have a poor specificity.[14,17,26,28,29] The lack of an elevated serum erythrocyte sedimentation rate or c-reactive protein does not exclude the diagnosis of septic arthritis.[18] A positive blood culture may be the only source of microbiologic diagnosis in some instances of septic arthritis.[14,18,20,21,25]

Joint aspiration is an important component in the diagnostic work-up, in which the sample can be analyzed on a variety of levels, including white blood cell count and differential, glucose, protein, lactate dehydrogenase, Gram's stain, and culture.

Radiographs are typically normal early in the disease.[15,17,22] However, if left untreated, the rapid cartilage destruction that ensues results in uniform and severe joint space narrowing. Periarticular demineralization of the bone adjacent to the infected joint develops with marginal erosions[15,22] (Fig 2). Magnetic resonance imaging (MRI) features of septic arthritis include joint effusion, synovial thickening, surrounding soft tissue edema, diffuse joint space narrowing, and adjacent bone marrow edema with bare area erosions.[15,22]

Neuropathic Osteoarthropathy

Neuropathic osteoarthropathy refers to the destruction of a joint associated with a neurosensory deficit.[30] The entity was first described by Sir William Musgrave in 1703[31]; however, the exact pathogenesis remains clouded. In the late 19th century, Jean-Martin Charcot recognized the association of arthropathy of the foot with syphilis, with tabes dorsalis reflecting the most common cause of neuropathic osteoarthropathy at the time.[31] Charcot postulated that damage to central nervous system trophic centers that controlled bone and joint nutrition resulted in a neuropathic joint. The term "Charcot joint" is still readily used to describe neuropathic osteoarthropathy.[30,31] Currently, diabetes mellitus is the overwhelming leading cause of neuropathic osteoarthropathy in the United States, with additional associations including syringomyelia, meningomyelocele, multiple sclerosis, trauma, alcoholism, and amyloidosis, to name a few.[31,32]

A variety of theories have been proposed to help explain the devastating effects of neuropathic osteoarthropathy. The neurotramatic theory holds that in the absence of normal protective sensory feedback, repetitive trauma results in progressive joint destruction.[30,32] The neurovascular theory states that in the absence of a neural stimulus to a limb, there is a loss of sympathetic tone that results in vasodilation and hyperemia,

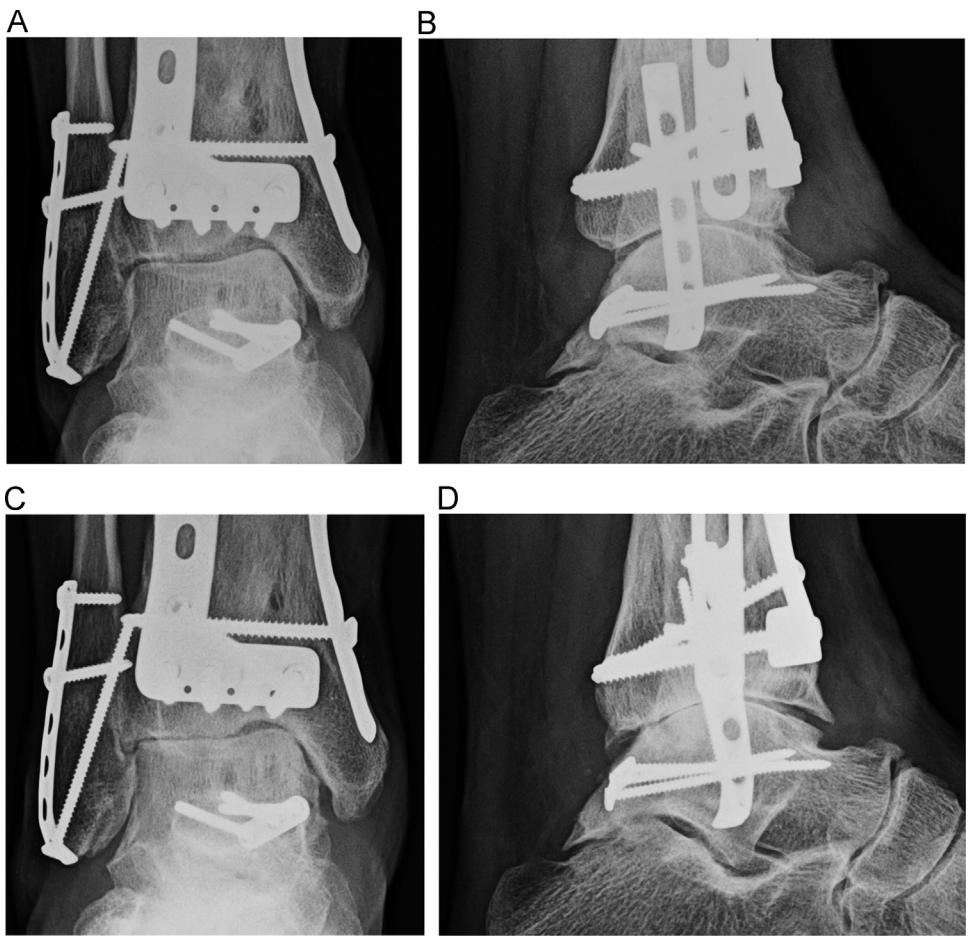

Fig. 1. Posttraumatic osteoarthritis. A 21-year-old woman with fractures of the distal tibia, distal fibula, and talus following fall, status-post surgical fixation. Frontal (A) and lateral (B) radiographs of the ankle from follow-up imaging December 2011 relative to January 2014 (C and D) demonstrate a rapid progression of osteoarthritis at the tibiotalar joint.

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