Imaging of Posttraumatic Arthritis, Avascular Necrosis, Septic Arthritis, Complex Regional Pain Syndrome, and Cancer Mimicking Arthritis

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KEYWORDS

• Arthritis • Posttraumatic • Septic • Avascular • Necrosis • CRPS • Osteodystrophy

KEY POINTS

- Radiographic changes of posttraumatic arthritis typically reflect the underlying trauma to the joint and the resultant manifestations of osteoarthritis.
- Avascular necrosis imaging demonstrates the sequela of osseous ischemia, frequently complicated by collapse of articular surfaces and joint destruction.
- Septic arthritis should be a leading differential in acutely painful joint with effusion and increased levels of inflammatory markers; contrast MR imaging is helpful in the absence of radiographic findings.
- Several benign synovial tumors as well as malignant lesions can mimic arthritis; history is important in formulating differential diagnoses and recommending appropriate follow-up.
- Although complex regional pain syndrome remains a clinical diagnosis, bone scintigraphy may be used as a confirmatory test.

POSTTRAUMATIC ARTHRITIS

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Posttraumatic arthritis is a common form of secondary osteoarthritis (OA) that results from a prior insult to the joint.[1] Injuries may damage the structural integrity and change the mechanics of the joint, leading to early degenerative changes. The process is further accelerated by continued injury and factors predisposing to OA, such as excess body weight. Radiographic changes of posttraumatic arthritis typically reflect the underlying trauma to the joint and the resultant manifestations of OA. Treatment options range from conservative measures to joint arthroplasty, directed by the severity of injury, the level of physical activity, and long-term goals.

An estimated 10% to 15% of diagnosed cases of OA have a posttraumatic cause.[2] Although any joint may be involved, this condition most frequently affects weight-bearing joints, particularly the hip,

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knee, and ankle, which are the most susceptible to trauma and to the stresses imposed by ongoing functional demands. OA of the joints rarely affected by this disease, including the wrist and the elbow, has an underlying posttraumatic cause in most cases.[1]

The inciting event initiating or accelerating joint degeneration in posttraumatic arthritis is acute structural joint injury. The injury may be in the form of fracture, cartilage damage, meniscal or ligamentous damage, or a combination of these processes. The severity of joint degeneration and speed of onset are proportionate to the damage sustained in the inciting trauma.[2] Joint surface incongruity following an intra-articular fracture often leads to established arthritic change within a year. The potential for repair in hyaline cartilage is very limited, and the fibrocartilage subsequently deposited does not possess the same longevity or resilience.[3] This limitation leads to premature wear and denuding of the articular cartilage down to subchondral bone (**Fig. 1**).

The initial injury not only disrupts the structural integrity of the tissues but results in increased expression of inflammatory mediators, cartilage-degrading proteinases, and stress response factors that lead to additional injury and death of tissues, including those beyond the area of initial impact.[4] Mechanical stress can also induce production of reactive oxygen species that lead to oxidative stress, which impairs growth factor response and has adverse effects on the survival and functionality of the resident cell populations.[5,6] In vitro studies have shown that inhibition of reactive oxygen species decreased injury-induced chondrocyte death.[5]

Secondary injury results from chronic loading abnormalities caused by ligamentous, meniscal, and joint capsular damage, in addition to possible articular incongruity.[7] Joint instability alters the location where joint surfaces make contact, resulting in localized areas of cartilage degeneration in areas not typically loaded in the natural state.[8] Loss of anterior cruciate ligament (ACL) stability, for instance, causes increased medial femoral condyle translation on the tibial plateau, leading to altered wear patterns. Even in cases in which no surface injury or joint inflammation was appreciated following the initial injury, the existence of uncorrected ligamentous or capsule tears has resulted in posttraumatic arthritis over a period of years, likely stemming from increased joint instability.

The risk factors for arthritic initiation and progression in patients with posttraumatic OA are similar to those for idiopathic OA, suggesting that the systemic host factors and local biomechanical factors interact and compound. Obesity, female gender, and preexisting early-stage OA contribute significantly to adverse radiographic and clinical outcomes.[9] Additional patient risk factors include proprioceptive defects, calcium crystal deposition disease, and other disease states that may lead to disruption of normal joint structures or mechanics.

The symptoms of posttraumatic arthritis are joint pain, swelling, and decreased tolerance for

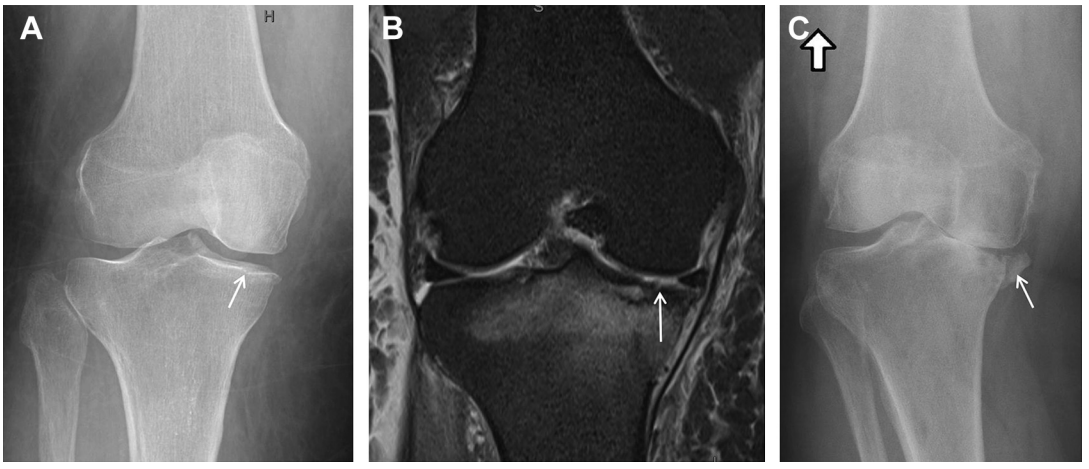

Fig. 1. (*A*) Anteroposterior (AP) radiograph of the right knee in a 60-year-old woman presenting after a fall shows subtle cortical irregularity along the medial tibial plateau (*arrow*). (*B*) Coronal long-recovery-time fat-saturated (FS) MR image confirms a nondisplaced fracture of the medial tibial plateau, with intra-articular extension and disruption of cartilage at the arrow. (*C*) AP radiograph of the right knee less than 2 years later shows severe joint space narrowing and subchondral sclerosis at the medial compartment, along with sequelae of the medial plateau fracture that include a displaced osseous fragment (*small arrow*).