

Avascular Necrosis and Bone Infarcts of the Knee

Patrick Graham

Introduction

Avascular necrosis of bone (AVN), also known as osteonecrosis, ischemic necrosis, or aseptic necrosis, is a progressive pathological process in which compromise of the bony vasculature results in death of bone and bone marrow cells. This process eventually leads to bony collapse and destruction of the associated joint. Similarly, bone infarction involves the death of hematopoietic cells within the marrow. This is typically seen within the long bones and does not involve the weight-bearing surface. Given the marrow involvement, there may be a reduction in red blood cell production and there is potential for fat emboli in the acute phase of development. As the remainder of the cortex and weight-bearing aspects are left unaffected, this is not typically otherwise symptomatic (Jones & Mont, 2014; Karim, Cherian, Jauregui, Pierce, & Mont, 2015; Mont, Marker, Zywiell, & Carrino, 2011; National Institutes of Health [NIH], 2015).

Avascular necrosis of the knee, although rare, is the second most commonly affected site after the hip. This pathological finding is divided into three categories: spontaneous osteonecrosis of the knee (SONK), secondary (commonly referred to as atraumatic), and postarthroscopic. SONK is the most common form and typically seen in patients older than 50 years, with a reported incidence as high as 9.4%. Secondary osteonecrosis is most commonly seen in patients younger than 45 years (Jones & Mont, 2014; Karim et al., 2015; Mont et al., 2011).

Atraumatic etiologies include chronic alcohol abuse, cigarette smoking, long-term use of corticosteroids, sickle cell disease, systemic lupus erythematosus, chronic renal failure, human immunodeficiency virus (HIV) infection, pancreatitis, chronic hyperlipidemia, exposure to radiation, and thrombophlebitis. Still others are idiopathic. Chronic alcohol abuse and use of corticosteroids are associated with approximately 80% of atraumatic cases (Jones & Mont, 2014; Karim et al., 2015; Mont et al., 2011; NIH, 2015).

Case Presentation

A 37-year-old woman, with significant medical history for cardiac transplant times two with associated long-term steroid use, presented with near 3 years of

bilateral knee pain. She denied any injury or incident. She described a gradually worsening, aching, and throbbing type pain. Initially, she was responsive to over-the-counter medications. Eventually, she was requiring intermittent use of opioids and would have days that the pain would keep her from completing her activities of daily living. She was referred to physical therapy and noted improvement in her strength and flexibility, but the bouts of pain was persistent. She was then referred to orthopaedics for further evaluation.

Upon presentation was an overall well-appearing female in no apparent distress. She displayed a mildly antalgic gait, without use of an assistive device. Knees were without gross deformity, abrasions, discoloration, or abnormal warmth. There was a mild effusion of the right knee. She noted tenderness to palpation of medial and lateral joint lines of both knees. The medial joint lines, most notably on the right, displayed most focal tenderness. Range of motion was 0°–115° with mild crepitation. There was no appreciable joint laxity. Her strength was 5/5 in bilateral lower extremities, and she was found to be distally neurovascularly intact. Special testing was notable for a positive bounce home, patellar grind, and Clarke's sign.

Radiographs obtained at the time of evaluation included anteroposterior, lateral, and sunrise views of both knees (see Figure 1). These were evident for lucency with dense sclerotic rim within the medial femoral condyles. There was also subtle proximal tibial sclerosis. These findings were consistent with AVN of the femoral condyles and possible infarcts of the proximal tibia. With that, it was recommended the patient have magnetic resonance imaging (MRI) of both knees to further evaluate the extent of involvement and staging the areas of AVN (Jones & Mont, 2014; Karim et al., 2015; Mont et al., 2011). MRI findings were consistent with Stage III avascular necrosis and better defined the areas of infarction in the proximal tibias (see Figure 2).

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The author has disclosed no conflicts of interest.

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DOI: 10.1097/NOR.0000000000000631



FIGURE 1. Radiographs—anteroposterior, lateral, and sunrise views of both knees. Note lucency with dense sclerotic rim within the medial femoral condyles. Subtle proximal tibia sclerosis is also noted.

The patient returned after completion of MRI, and we had a detailed discussion about the findings and possible treatment options. Having been through her most recent cardiac transplant less than a year prior, she was not amenable to any surgical interventions. She also noted only mild symptoms at the time and so elected to proceed with intra-articular steroid injection. She was reportedly “pain free” 1 week after steroid administration and was planning to see a bone health endocrinologist to consider options for medical therapies.

Management

The management of avascular necrosis of the knee includes both conservative and surgical interventions depending on the severity of disease state and patient symptoms. Nonoperative/conservative interventions are primarily reserved for those patients who are asymptomatic

or with only mild, intermittent symptoms. This includes protected weight bearing, activity modification, use of analgesics (either orally or via intra-articular injection), and other pharmacotherapies such as bisphosphonates. Unfortunately, these measures will not halt the progression of disease but are effective in managing the associated symptoms in the interim (Jones & Mont, 2014; Karim et al., 2015; Mont et al., 2011; NIH, 2015).

Surgical management includes options for joint-preserving versus joint-replacing procedures. Joint-preserving procedures include core decompression, osteotomy, bone marrow grafting, percutaneous drilling, as well as other various techniques of cartilage transplant or preservation. A lack of prospective randomized trials, as well as variable reported success rates of these surgeries, begs the question as to the true efficacy of these interventions. Ultimately, these interventions are aimed at postponing the progression of disease and improving quality of life

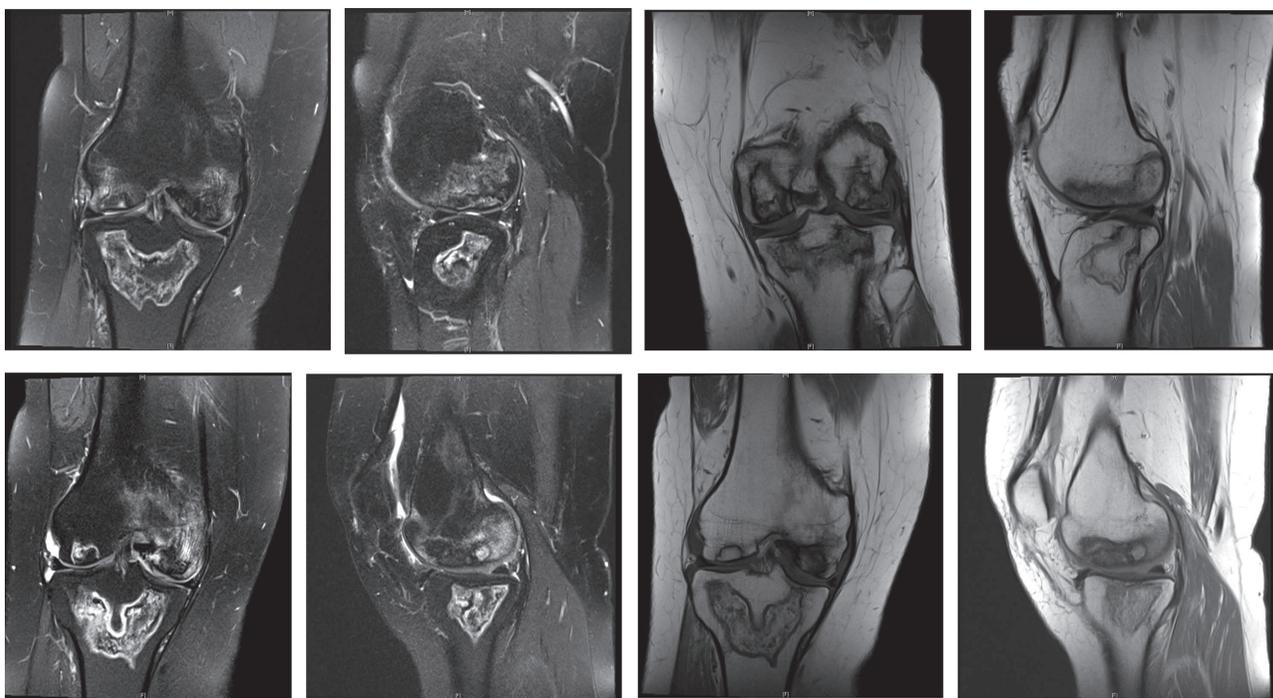


FIGURE 2. Magnetic resonance imaging of bilateral knees (left top, right bottom)—coronal and sagittal T2 with fat suppression, coronal and sagittal T1. Findings: Numerous areas of serpentine low T1, high T2 signal are noted throughout the femoral condyles, proximal tibial epiphysis, and metaphysis consistent with avascular necrosis. Reactive bone marrow edema is noted within the medial lateral femoral condyles. There is mild flattening of the weight-bearing portion of the lateral femoral condyle, with a small area of full-thickness cartilage loss measuring 4 mm. The medial femoral condyle articular surface is not flattened, and the overlying cartilage is intact. The tibial cartilage is unremarkable.

in the interim. The other surgical options are joint-replacing procedures and include uni-compartmental or total knee arthroplasty (Bugbee, Cavallo, & Giannini, 2012; Karim et al., 2015; Kraenzlin, Graf, Meier, Kraenzlin, & Friedrich, 2010; Marulanda, Seyler, Sheikh, & Mont, 2006).

Discussion

Although rare, the advanced practice provider should consider the possibility of avascular necrosis of the knee in any patient who presents with primary knee pain and noted risk factors as described earlier. This should also be a consideration for any patient who is postarthroscopy and continues to have symptoms out of proportion with stage of recovery. Advanced diagnostic imaging, either MRI or computed tomography, is certainly warranted for those with persistent knee complaints not consistent with radiographic findings (Jones & Mont, 2014; Karim et al., 2015; Mont et al., 2011; NIH, 2015).

Although conservative measures are effective in alleviating symptoms, allowing for better function and quality of life, it is important to communicate the progressive nature of avascular necrosis at the outset of treatment. This will better set expectation and prepare the patient for the likely necessity of surgery at some point in the course of treatment.

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