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Spontaneous osteonecrosis of the knee

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Abstract

Spontaneous osteonecrosis of the knee presents with acute onset of severe, pain in elderly patients, usually female and usually without a history of trauma. Originally described as idiopathic osteonecrosis, the exact etiology is still debated. Evidence suggests that an acute fracture occurs as a result of chronic stress or minor trauma to a weakened subchondral bone plate. The imaging characteristics on MR reflect the age of the lesion and the symptoms. More appropriate terminology may be "subchondral insufficiency fracture of the knee" or "focal subchondral osteonecrosis".

Keywords: SONK; Osteonecrosis; Spontaneous; Knee; Insufficiency fracture; Osteochondral fracture

1. Introduction

Osteonecrosis is defined as ischemic death of the cellular constituents of bone and bone marrow. It is a relatively common condition that has been known for millennia and has been demonstrated in Egyptian mummies [1]. Despite this, there is a lack of understanding of the disease and its treatment.

Osteonecrosis is found most commonly in the femoral head, followed by the knee and the humeral head. The true incidence of osteonecrosis is unknown, but involvement of the knee is believed to account for approximately 10% of all cases [2,3]. Osteonecrosis of the knee involves the distal femur, proximal tibia, and one or both condyles or plateau [4].

Secondary osteonecrosis is associated with predisposing factors including corticosteroid use, rheumatoid arthritis, alcohol consumption, sickle-cell disease, systemic lupus erythematosus, Caisson's disease and Gaucher's disease [2,4–7]. Idiopathic or spontaneous osteonecrosis of the knee (SONK) was first described by Ahlback et al. [8] in 1968, in the medial femoral condyle in 40 patients (6 men and 34 women) with an average age of 70 years. Involvement of the medial tibial plateau is less common and was described in 1976 by D'Angelijan et al. [9].

There are significant differences between the two forms of osteonecrosis [4]. Patients with predisposing factors generally had an insidious onset of mild or vague pain. Those without predisposing factors experienced sudden, severe pain, which frequently persisted at rest [4,8,10–13], this latter presentation being more typical of SONK [4,7,8,10–12,14].

Location and size of lesions also differed [4]. Lesions are usually unilateral in SONK with a strong predilection for the medial compartment. Most cases involve the medial femoral condyle [10,11,13,15–17] but the ipsilateral tibial plateau, alone or in conjunction with femoral condyle involvement, may be seen [4,10,11,13,17]. In secondary osteonecrosis, bilateral involvement of the knee joint is seen in 30–80% of patients [2,4,10,15,18–21] and the lateral femoral condyle is affected in 60% [2,4,10,21]. Multifocal osteonecrosis with simultaneous involvement of knees, hips and shoulders is also seen [2,4,21].

The histopathological appearances of the two types of osteonecrosis differ [15,18]. In a study of bone specimens removed at total knee replacement, specimens from patients with SONK had lesions that involved only a small area of the subchondral bone. In the specimens from patients with secondary osteonecrosis, the lesion was wedge-shaped and larger. MRI studies confirmed that lesions attributed to secondary osteonecrosis are significantly larger than those attributed to SONK (Fig. 1). In secondary osteonecrosis MR shows a demarcation rim at the border between necrotic and viable bone [4,22,23]. In SONK, a focal subcortical low signal intensity is seen on both T1 and T2 weighted images [23] (Figs. 2 and 3).

Patients with SONK are more likely to have osteoarthritis and meniscal lesions. SONK typically develops in elderly patients, most of whom are women, whereas secondary osteonecrosis

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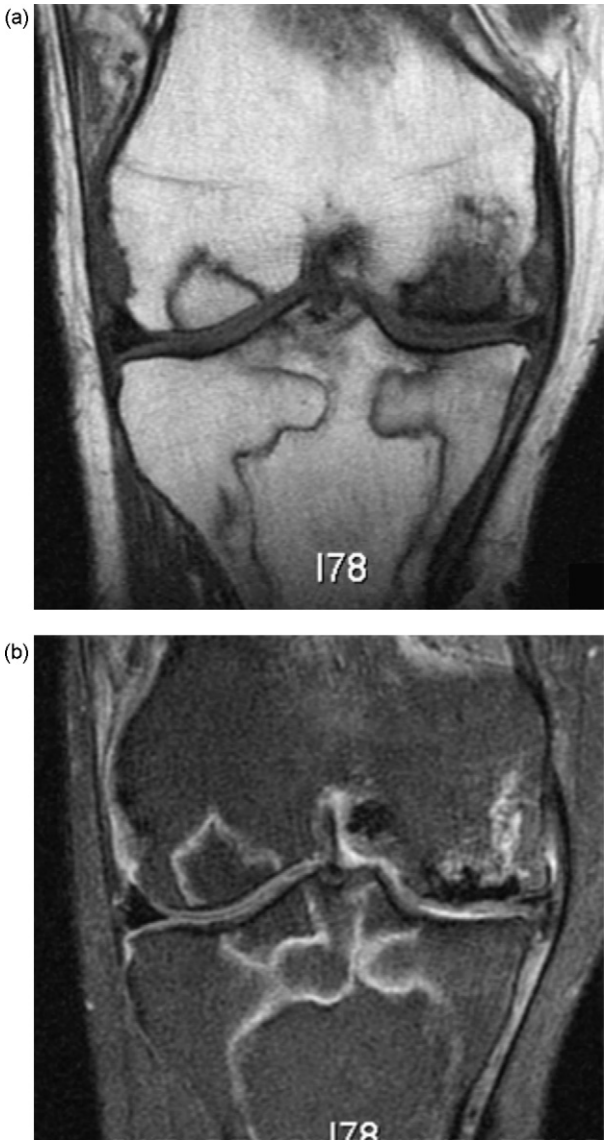

Fig. 1. 59-year-old female with asthma and HIV infection. MRI of the knee obtained for knee pain. (a and b) Coronal T1 and T2-weighted images show the typical irregular contoured, well demarcated lesions within the marrow and subchondral bone, representing areas of necrosis. Note the dark signal margin between the normal and abnormal bone (arrows). The area of necrosis in the medial femoral condyle is fairly large.

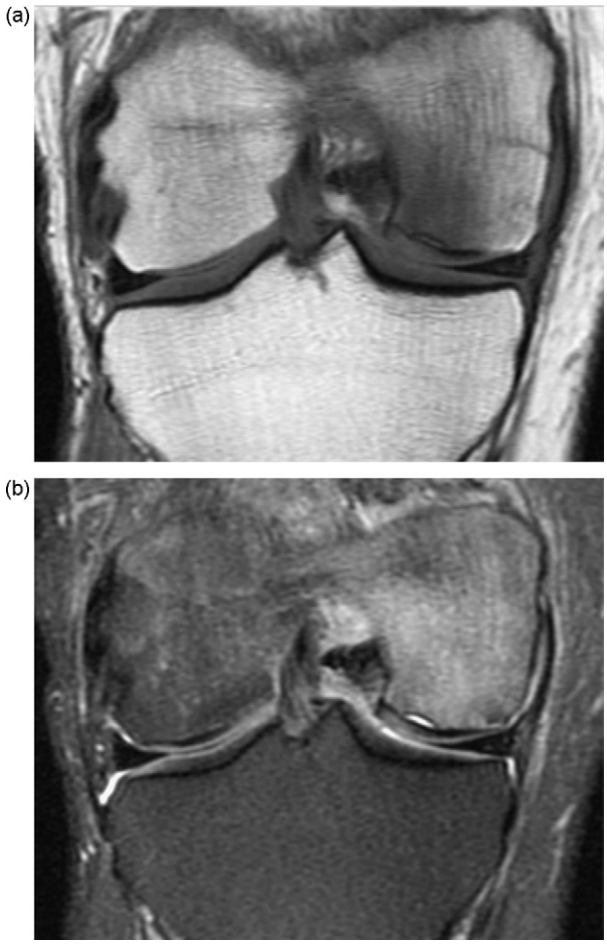

Fig. 2. MR images of the knee in a 59-year-old male with instability and acute knee pain. (a) Coronal T1-weighted image shows dark signal replacing the marrow in the medial femoral condyle. A small subchondral fracture is seen. (b) Coronal T2-weighted image with fat saturation shows edema in the medial femoral condyle. A fluid filled cleft (fracture) is seen in the subchondral bone with intact adjacent cartilage (arrow).

can develop at any age and has no gender bias [4,24]. This led to the suggestion that the two conditions have different pathogenic mechanisms, with secondary osteonecrosis having a metabolic etiology and SONK having a local, mechanical etiology [4,12,25].

2. Pathogenesis of osteonecrosis

It is generally accepted that osteonecrosis follows ischemia due to vascular compromise although the possible causes of ischemia are under debate. Microvascular disruption in the subchondral bone may lead to bone marrow edema, which, in the confines of the bony environment, could worsen ischemia and result in osteonecrosis. In the case of corticosteroid-related necrosis, the development of osteonecrosis could be due to decreased circulation and ischemia associated with drug-induced increase in size of the marrow fat cells [19,26]. Other possible contributors to the etiopathogenesis of secondary osteonecrosis are coagulopathies, fat emboli, and thrombi formation [19,26].

The pathogenesis of SONK remains controversial. Several theories have been proposed as causes, including meniscal tears and/or chondromalacia associated with osteoarthritis [14,25,27–30], prior arthroscopic surgery [14], and insufficiency fractures associated with osteoporosis [4,15,25,27,28].

The association between SONK and meniscal lesions in the same compartment has been noted by several authors [4,8,15,17,29]. For example, medial meniscal tears were associated with “spontaneous” osteonecrosis in 21 (78%) of 27 knees examined by arthrography [29]. They suggested that stress concentration over the edge of the meniscal fragment may result in ischemic necrosis of the femoral condyle and that early detection and treatment of medial meniscal tears in older patients might prevent the late changes of “spontaneous” osteonecrosis.