Osteonecrosis of the Knee Due to Hypertriglyceridemia and Hyperuricemia: A Case Report

Hipertrigliseridemi ve Hiperürisemiye Bağlı Gelişen Diz Osteonekrozu: Olgu Sunumu

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Abstract

Osteonecrosis is a pathologic process which results with the death of bone and bone marrow elements. The main predisposing factors (trauma, glucocorticosteroids, alcoholism and connective tissue disorders) should be carefully sought but osteonecrosis can also be idiopathic in origin. We report a patient who developed osteonecrosis of the knee with hypertrigliceridemia and hyperuricemia. Knee osteonecrosis combined with hypertriglyceridemia is a rare condition. This case draws attention to the association between osteonecrosis and metabolic disorders. *(Rheumatism 2007; 22: 114-6)*

Key words: Knee, osteonecrosis, hypertriglyceridemia

Özet

Osteonekroz, kemik ve kemik iliği elemanlarının ölümüyle sonuçlanan patolojik bir süreçtir. Temel predispozan faktörlerin (travma, glukokortikoid kullanımı, alkolizm ve bağ dokusu bozuklukları) araştırılması gerekmekle birlikte osteonekroz idiopatik de olabilir. Bu çalışmada, hipertrigliseridemi ve hiperürisemiye bağlı dizinde osteonekroz gelişen bir vaka sunulmuştur. Hipertrigliseridemiye bağlı diz osteonekrozu nadir görülen bir durumdur. Bu vaka sunumu ile osteonekroz ve metabolik bozukluklar arasındaki ilişkiye dikkat çekilmiştir. *(Romatizma 2007; 22: 114-6)*

Anahtar kelimeler: Diz, osteonekroz, hipertrigliseridemi

Introduction

Osteonecrosis is a pathologic process which results with the death of bone and bone marrow elements. Spontaneous osteonecrosis of the knee was first reported as a distinct clinicopathological entity by Alback et al. (1) in 1968. This entity is believed to be an important but underestimated cause of osteoarthritis of the knee. The main predisposing factors (trauma, glucocorticosteroids, alcholism and connective tissue disorders) should be carefully sought but osteonecrosis can also be idiopathic. Hypertrygliceridemia and hyperuricemia are rare causes of osteonecrosis. To our knowledge our case is the second reported case in the literature with hypertrygliceridemia and hyperuricemia associated with osteonecrosis of the knee (2). The purpose of this report is to draw attention to the association between osteonecrosis and metabolic diseases.

Case

A 48 year old man had increasing left knee pain without a history of trauma. Weight bearing aggravated the pain and the pain was worse at night. He was referred to our department two months after the onset as his symptoms got worse.

His medical history was unremarkable. There was no use of alcohol, cigarettes or corticosteroids. He did not give any history of a rheumatologic, metabolic or vascular disease.

On physical examination, there was localized tenderness on the left medial femoral condyle. The range of motion was limited at 110 degree of flexion. He had patellar instability at the left knee. No synovitis and effusion was present. Routine laboratory studies were normal except uric acid (8.5 mg/dl) and triglyceride (431 mg/dl). Radiography was normal. Magnetic resonance imaging

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(MRI) showed the area with decreased signal intensity on the T1-weighted images involving both the femoral condyles being more intense at the medial condyle. The T2-weighted images showed a low signal intensity in the central lesion, with a high-intensity about the margin caused by the bone marrow edema surrounding the lesion defined as osteonecrosis involving both condyles of the femur (Figure 1).

The patient was initially treated conservatively by avoiding weight-bearing and using anti-inflammatory medications. He was consultated to Endocrinology Department for the increased levels of uric acid and triglyceride. He received phenofibrate 200 mg/day and a diet program for 6 months. On follow-up his complaints resolved slowly and he functioned fairly well-with limited symptoms 6 months after the onset. His uric acid and triglyceride levels decreased to 6.3 mg/dl and 257 mg/dl respectively.

Discussion

Most common localization of osteonecrosis is the femoral head, followed by the humeral head, the knee and the small bones of the wrist and foot (3). Idiopathic osteonecrosis of the knee is typically a disease of the elderly characterized by severe knee pain of sudden onset, unilateral involvement and restriction of the lesions generally to one femoral condyle or tibial plateau, especially in the medial compartment of the joint. Secondary osteonecrosis generally occurred in younger patients and frequently has an insidious onset with mild or vague pain, the total compartment of the knee was often involved, and the lesions are generally larger than lesions arising spontanously. The signs and symptoms of our case presented secondary osteonecrosis.There are significant differences between idiopathic and secondary osteonecrosis, especially with regard to clinical presentation, location and the MRI of the lesions. These differences are probably due to a difference in the pathogenic mechanism (4).

Traumatic and vascular theories have been suggested as a causative factor of osteonecrosis of the knee, but the precise etiology still remains speculative (5). There are some hypotheses to explain ischemia and necrosis mechanism in non traumatic osteonecrosis. Primitive vascular problems can lead to coroner disease of the bone such as diabetes mellitus, alcoholism, hyperlipidemia (6). Experimental studies suggest that hypertrophy of the marrow cells, micro embolic phenomena, and lipid-induced osteocyte necrosis, alone or in combination, may cause osteonecrosis. In this case, the reason of osteonecrosis was considered that it is a lipid-induced osteocyte necrosis caused by high levels of triglyceride and uric acid. It is taught that such events may result in local inflammatory exudates or vascular impedance, or both. Either may increase the hydrostatic pressures in bone with the potential to limit blood flow and to magnify the initial insult. Vascular insult on the arterial side, the venous side, or directly on the bone and marrow sinusoids may lead to the bone ischemia. Bone microcirculation is confirmed within an unexpandable compartment and thus, an increase in the bone marrow pressure can cause bone ischemia (5).

The radiographs are usually normal during the early course of the disease. Later they may show a subchondral radiolucent area in the affected femoral condyle or the tibial plateau. As the disease progresses, a large area of osteonecrosis may be seen. In the late stage, severe destruction with collapse of the femoral condyle occurs, leading to secondary osteoarthritis of the knee.

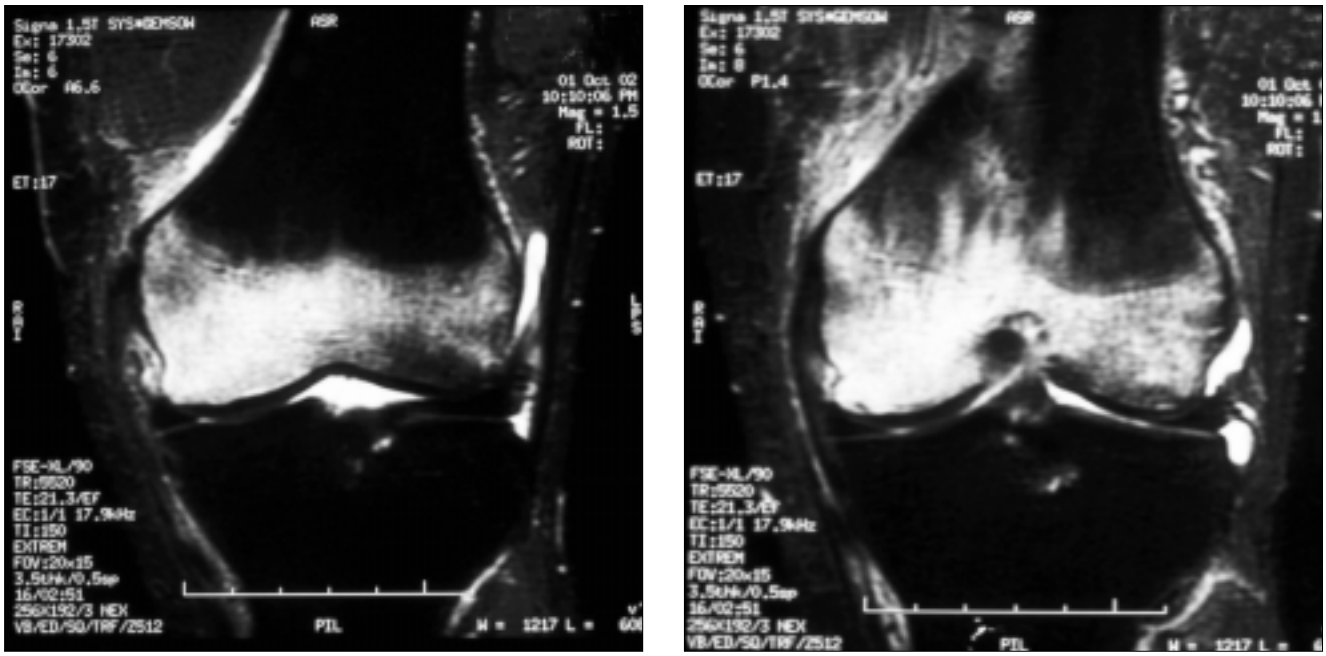

Figure 1-2. MRI images of the knee presenting bone marrow edema (osteonecrosis) of the condyles

Magnetic resonance imaging has improved the early diagnosis of osteonecrosis as radiographs maybe normal in the initial stages (3). In this case the radiographs were normal while the MRI scans showed the osteonecrosis (Figure 1). It has provided enhanced ability to visualize the bone marrow and the distinguished necrotic tissue from viable segments with a high level of specify. Magnetic resonance imaging is a totally noninvasive investigation and provides more information than the radioisotope bone scan. It can be also helpful in determining prognosis concerning the eventual natural course of osteonecrosis of the knee (5). Skai et al. (7) studied MRI evaluation of steroid or alcohol related osteonecrosis of the femoral condyle and conclude that the extent of the necrotic lesion on both the mild coronal and mild sagittal planes is of importance for the prognosis of osteonecrosis of the femoral condyle.

Prognosis of osteonecrosis of the knee depends on radiographic size of lesion, ratio of size of the lesion to the size of the condyle, and stage of the lesion (5).

In the early stages of the disease, non-operative treatment is indicated and many patients if diagnosed early, have a benign course with a satisfactory pain relief and a good knee function. Conservative treatment, consisting of anti-inflammatory medication, analgesics, and protected weight-bearing, is essential until the size of the lesion has been defined. In this case the patient received conservative treatment, medical treatment and diet program for the etiologic reason. It took 6 months for him to recover. This may take as long as six months for a large lesion. If the lesion is small, it will do well and no surgical treatment is required (1). In patients with advanced stage of disease, surgery is recommended (5,8).

As a result of this case report, clinicians should keep in mind that metabolic diseases should be a reason for osteonecrosis and detailed examination should be done.

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