Osteonecrosis of the Knee After an Intra-Articular Injection of Steroids

To the Editor: Aseptic or avascular osteonecrosis (AON) is a finding that is becoming ever more common in our daily clinical practice. It has been related to the treatment with systemic steroids, but only anecdotal cases have been published regarding its association to their intra-articular injection.[1-4] We present the case of 1 patient that presented this complication 1 week after receiving an injection of triamcinolone acetonide in the right knee.

A 67 year old female with a history of hypertension, who was treated with angiotensin converter enzyme (ACE) inhibitors presented a class II osteoarthritis of the both her knees during her routine outpatient visit. She referred chronic pain in both knees that had not suffered any modification in the past few months. Upon exploration a deformity of both knees was evident with a discreet hydroarthrosis of the right one.

Mobility was only limited in the last degrees of flexion. Because the patients' pain required a daily dose of 1200 mg of ibuprophen and only permitted her to walk about 1 km, infiltration with hyaluronic acid was proposed but, because she presented an effusion, it was decided to perform an arthrocenthesis and steroid infiltration before administering the hyaluronic acid. A week after the procedure she was admitted to the emergency room due to pain in the right knee, with functional impotence, without any fever nor worsening of her general conditions.

Upon exploration an effusion was seen, as well as a painful limitation on 90° flexion. On suspicion of septic arthritis an arthrocenthesis was performed and samples of synovial fluid were sent for a Gram stain and culture, with negative results. In addition, synovial fluid was analyzed under a polarized light microscope without any evidence of crystals. The patient did not improve with non-steroidal anti-inflammatory drugs (NSAID) and relative rest, making it necessary to ask for a magnetic resonance that showed an area of osteonecrosis of the internal femoral condyle (Figures 1 and 2). Treatment with NSAID was installed and weight bearing by the joint was avoided, with a progressive improvement of pain.

Osteonecrosis is the cell death of an area of bone as a result of the interruption of blood flow to that zone. It preferentially presents on the femoral head but can occur in several other localizations, especially the distal femur, the humeral head, and the small bones of the hand and the foot. Its most frequent causes are trauma, chronic abuse of alcohol, and systemic corticosteroid therapy and is considered idiopathic in an elevated percentage of cases.[5-8]

Other etiologic factors are recorded on Table. The association of osteonecrosis to the administration of systemic steroids has been seen for many years, and it is related to high doses or a total received dose and a longer time of exposure; it is estimated that an accumulation of 2 g could be the risk threshold and that for every 20 mg increment of the daily dose it increases in 5% the risk of

Etiology of Osteonecrosis

Aseptic osteonecrosis of an unknown cause
Traumatic
Humeral head fracture
Humeral head dislocation
Non-traumatic
Caisson disease (decompression syndrome)
Gaucher's disease
Sickle cell anemia
Radiotherapy
Aseptic osteonecrosis with a probable etiologic relationship
Traumatic
Minor trauma
Non-traumatic
Arteriosclerosis and occlusive vascular disease
Corticosteroids
Cushing's disease
Diabetes mellitus
Dyslipidemias
Acetabulum dysplasia
Excessive alcohol consumption
Fatty liver disease
Hyperuricemia and gout
Osteomalacia
Chronic pancreatitis
Pregnancy
Transplants
Systemic lupus erythematosus and other connective tissue diseases
Tumors
Coagulopathy

Figure 1. *Saggital cut in SE sequence potentiated in T1 in which a zone of irregular contour with a low signal intensity of the internal femoral condyle is seen.*

Figure 2. *Coronal cut in SE sequence potentiated in T2 in which a zone of low signal intensity corresponding to an area of osteonecrosis is seen, surrounded by a halo of signal increase of the bone marrow contiguous to the internal femoral condyle.*

presenting osteonecrosis; even then, this complication has been reported after 3 months since starting treatment with medium-high doses of steroid treatment.

Only anecdotal cases have been published after an intra-articular injection. In this respect there is some controversy because in several articles no greater frequency of AON has been found and in some articles this only happened in patients who had also received systemic steroids. Therefore, of the review of the literature that we have carried out in PubMed, MEDLINE, and Cochrane one can conclude that there is scarce evidence that intra-articular infiltration with steroids can cause osteonecrosis. In any case, the temporal relationship between the triamcinolone injection in our patient and the appearance of osteonecrosis leads us to establish a causal relationship that we find interesting enough to communicate.

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