Letters to the Editor

Avascular necrosis of the knees and ulcerative colitis

Key words: Avascular necrosis. Osteonecrosis. Ulcerative colitis. Inflammatory bowel disease.

Dear Editor,

Avascular necrosis (AN), also called osteonecrosis, is the death of bone tissue due to a lack of blood supply. AN has been infrequently described in patients with inflammatory bowel disease (IBD) (1-7) and even more rarely in cases of ulcerative colitis (4-6). We present a case of AN in a patient with ulcerative colitis.

Case report

A 41-year-old man with a past history of ulcerative colitis was referred to our rheumatology unit because of bilateral gonalgia. Thirteen years before, when he was diagnosed as having IBD, colonoscopy showed active and extensive ulcerative pancolitis (E3 according with Montreal classification); biopsies of the mucosa confirmed the diagnosis. Corticosteroids, aminosalicylates and ciclosporin were required to achieve disease control. Actually, he was maintained on stable medication with mesalazine with occasional ulcerative colitis flare-ups that responds to oral corticosteroids. In the last two years, he complained of mechanical pain in both knees. On examination, he showed signs of arthritis of the right knee, without effusion. Spinal mobility showed a normal range and provocative sacroiliac joint stress maneuvers were negative. Also, strength, sensitivity, muscle bulk and tone were

normal and he had no cutaneous or ocular involvement. The routine laboratory data (including ESR and C-reactive protein) showed a normal complete blood count and coagulation parameters. ANA was positive at a titre of 1:160 with a diffuse granular pattern, while anticardiolipin antibodies and lupus anticoagulant were both negatives. Thyroid function, iron status and proteinogram were all within the normal range. Plain radiographs of the elbows, shoulders, hips and sacroiliac joints were normal. However, plain knee radiographs demonstrated sclerotic and irregular lesions compatible with bone infarctions and degenerative changes, most pronounced in the right knee (Fig. 1). With the diagnosis of AN of the knees we started aspirin, simvastatin, calcium and cholecalciferol. At present the patient remains asymptomatic.

Fig. 1. Plain radiograph. Right knee. Frontal and lateral projections. Epiphysis of the right knee with mixed radiopaque-radiolucent texture and altered trabecular pattern. Radiological findings suggestive of bone infarctions and degenerative changes.

Discussion

AN is a complication classically reported in patients receiving corticosteroid therapy. The condition is found most commonly in the femoral head, but in some cases it is a multifocal process (2). The overall rate of AN in Crohn's disease patients is estimated at 0,5% (3), but the incidence in ulcerative colitis patients would be much lower. Controversy exists regarding the role of corticosteroids in AN. However, today it is recognized that IBD predispose patients to AN. Some studies suggest that IBD lowers the threshold of corticosteroids (lower doses per day and for shorter time) needed to induce AN (2,5). Also, AN may occur in patients with IBD who have not received any corticosteroid therapy (1) suggesting that other pathogenic factors must be involved, such as hypercoagulability (5) or parenteral nutrition with infused lipids (7).

A high index of suspicion for the early diagnosis of AN is required. In patients with IBD who have received corticosteroid therapy and referred articular pain a magnetic resonance imaging or radionuclide bone scan could allow early diagnosis (5). In advanced stages of AN, with flattening of the articular surface or collapse, total joint replacement surgery is the main alternative. Other conservative procedures should be reserved for selected cases.

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