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Case report 733

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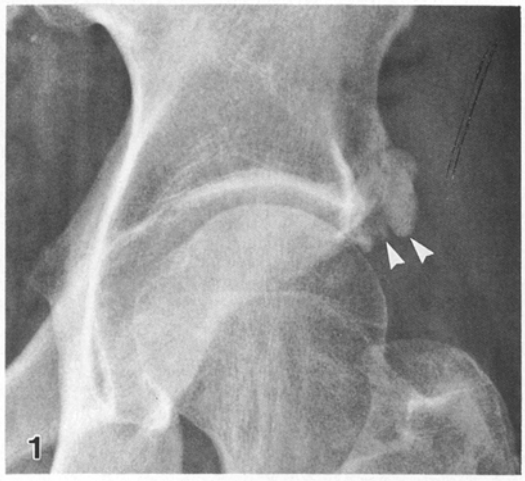
Radiological studies



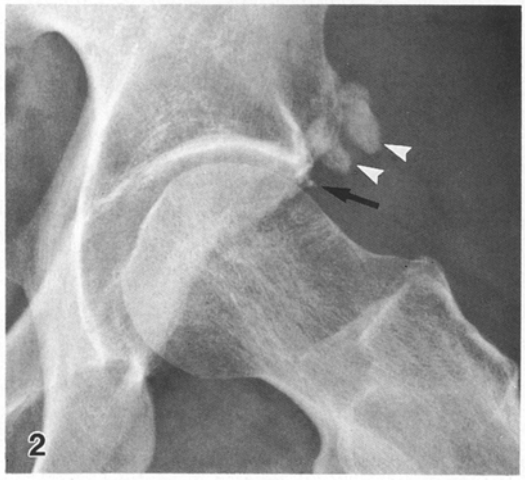


Fig. 1. Coned-down anteroposterior view of the left hip showing calcific densities projected lateral to the acetabulum (*white arrowheads*)

Fig. 2. Frog-leg projection of the left hip showing two calcifications adjacent to anterior inferior iliac spin (AIIS) (*white arrowheads*). Incidentally noted is a small os acetabulum (*black arrow*)

Clinical information

This 45-year-old woman presented to a local family physician with pain in the left hip. Figures 1 and 2 are coned-down anteroposterior (AP) and frog-leg projections of the left hip. No history of a previous injury was obtained, and all laboratory findings were unremarkable.

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Diagnosis: Calcific tendinitis of the origin of the medial and lateral heads of the rectus femoris muscle and the anterior inferior iliac spin (AIIS).

The differential diagnosis would include recent or old chronic avulsion fractures of the AIIS. Incidently noted is an os acetabulum.

Discussion

Duplay's clinical description of the periarthritis scapulo-humeralis syndrome in 1872 was the first published case of calcific tendinitis from deposition of hydroxyapatite crystals [6]; the designation "periarthritis calcarea" was given to the syndrome first by Gondos in 1953 when he described the radiographic findings in the wrist [5].

The most common anatomic location for calcific tendinitis is the supraspinatus muscle in the glenohumeral joint, although any joint can be affected. The disorder affects both men and women and is most common between 40 and 70 years of age [11]. The hip is estimated to be involved by the disorder in approximately 5.4% of people over 15 years old [4, 12]. Reported sites for deposition of calcium in the hip joint include the gluteus maximus tendon [2], the gluteus medius tendon near the greater trochanter, the gluteus medius tendon near the acetabulum, the bursa between gluteus medius and greater trochanter, and the bursa between gluteus medius and greater trochanter, and within the joint capsule itself [4]. However, calcific tendinitis within the rectus femoris tendon at its origin is rarely encountered.

The first description of rectus femoris calcification was in 1967 by King and Vanderpool who reported two patients, one with an acute injury to the hip and one with the gradual onset of pain in the hip with calcific deposits above the superior portion of the acetabulum at the insertion of the rectus femoris muscle [10]. The diagnosis of calcific tendinitis was made, as in our case, by the anatomic location and the characteristic radiological appearance. King and Vanderpool stressed the importance of the frog-leg projection in demonstrating the calcifications on plain films. Baudrillard et al. reported four patients with a history of acute trauma and calcifications adjacent to the AIIS [1]. They attributed these findings to posttraumatic enthesopathy from rupture of the rectus femoris tendon, while other authors have reported the development of posttraumatic exostoses at the AIIS after acute trauma [7]. In the current case, the medial and lateral heads of the origin of the rectus femoris muscle from the AIIS are demonstrated.

Therapy for this lesion is usually nonsteroidal antiinflammatory medication, although some authors have reported the use of radiotherapy in the treatment of the disease with resolution of the patient's symptoms [10] and even of the calcifications.

Os acetabuli, separate ossification centers for the acetabulum, and paraacetabular periarthritis calcarea may also occur near this anatomic site [8, 9]. However, these calcifications are usually smaller, have a more irregular appearance, and are located below and slightly inferior to the AIIS. They may also be associated with a deformity of the superior portion of the acetabulum. These lesions are also generally asymptomatic and require no therapy [8, 9].

In *summary*, calcific tendinitis at the site of origin of the rectus femoris muscle is a rare lesion. Presumed to be formed by deposition of hydroxyapatite crystals, this entity may be confused with other lesions such as os acetabuli or posttraumatic abnormalities. The characteristic location and appearance of the calcifications in a symptomatic patient with no history of trauma should allow diagnosis and subsequent symptomatic therapy.

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