DIAGNOSIS: TRANSIENT OSTEOPOROSIS OF THE HIP

DISCUSSION

Transient osteoporosis of the hip, also known as (transient) bone marrow edema syndrome of the hip, is a self-limiting clinical entity of unknown cause, although almost certainly a vascular basis and possible overactivity of the sympathetic system exists. There is some controversy as to whether transient osteoporosis of the hip represents a very early, reversible stage of avascular necrosis (AVN) or is secondary to subchondral insufficiency fractures.

Although initially described during the 3rd trimester of pregnancy, transient osteoporosis of the hip tends to affect middle age men (40-55 year olds) with an M:F ratio of 3:1. Generally, only one hip is affected at a time. Recurrence in the same hip is possible. It has been described in patients with Vitamin D deficiency, testosterone deficiency, hyperthyroidism, hyperphosphatemia, corticosteroid therapy, vascular disease or osteogenesis imperfecta.

Typically, patients present with spontaneous onset of hip pain, usually progressive over several weeks. Patients generally do not have risk factors for avascular necrosis and do not go on to form avascular necrosis.

In some instances, patients can go on to develop similar changes in the opposite hip or in other joints; such cases should be referred to as regional migratory osteoporosis.

An insufficiency fracture is a possible complication.

TOH has three stages: the first stage involves bone edema which coincides with acute joint pain; the second stage involves resorption of the bone and the last stage is the resolution, which shows the self-limiting characteristic of the disorder.

Regarding medical imaging findings, plain radiographs are generally normal initially but usually become abnormal by 4-8 weeks following the onset of symptoms. Findings include:

- subchondral cortical loss involving femoral head and neck: virtually pathognomonic,
- often profound osteopenia of the femoral head and neck region
- joint effusion may be present
- joint space always preserved

MRI demonstrates bone marrow edema pattern involving the femoral head, neck, and even intertrochanteric region:

T1: decreased signal with loss of normal fatty marrow signal

T2: high signal, often heterogeneous.

Typically, skeletal scintigraphy demonstrates markedly increased homogeneous uptake in the femoral head; a finding which is seen well before osteopenia is seen on plain films.
Differential diagnosis

General imaging differential considerations include: avascular necrosis, characterized by subchondral low signal on T2 or T1 sequences (Figure 4); stress fracture, either due to repetitive overload in athletes, which mainly affects the femoral neck (calcar) or due to insufficiency, with a greater subchondral compromise in the femoral head (Figures 5 y 6); septic arthritis, joint effusion with synovitis and bone edema both in the femur and in the acetabulum.

Figure 4. A. Computed tomography. A linear crescent-shaped image at the subchondral level limiting the area of avascular necrosis. B and C. MRI T1-weighted and T2-weighted sagittal sequences. The same linear image is observed in the right femoral head, showing high signal intensity and bone edema.

Figure 5. MRI T2-weighted coronal and sagittal sequences and T1-weighted coronal sequence. A small cortical fracture of the calcar femorale with bone edema, suggesting stress fracture in an athlete patient.
Figure 6. MRI T1-weighted and T2-weighted coronal sequences. The peripheral subchondral area of the right femoral head shows high signal intensity in T2-weighted sequences, suggesting bone edema compatible with insufficiency fracture in a patient with osteopenia.