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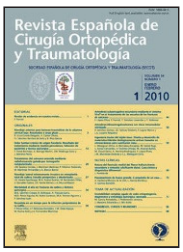

CASE REPORT

Transient migrant osteoporosis

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KEYWORDS
Hip;
Transient migrating osteoporosis;
Bone marrow oedema

Abstract
Purpose: To report on a case of transient migrant osteoporosis.
Clinical case: We present the case of a 50-year-old female who developed pain in the left hip, left knee and right ankle successively. Her hip MRI prompted a diagnosis of transient hip osteoporosis and successive migrations resulted in a diagnosis of transient migrating osteoporosis.
Results: Symptoms abated with conservative treatment comprising rest, analgesics, anti-inflammatories, non-weightbearing and physical therapy. Non-weightbearing relieved pain and prevented fatigue fractures. Medical treatment did not alter the course of the disease after 65 weeks.
Conclusion: Transient migrating osteoporosis is a rare, idiopathic and self-limiting disease. It is accompanied by migrating pain in the weight-bearing joints, as well as by diffuse periarticular radiographic osteopoenia and a pattern of bone marrow oedema seen on MRI. Treatment is conservative.

PALABRAS CLAVE
Cadera;
Osteoporosis transitoria migratoria;
Edema de médula ósea

Osteoporosis transitoria migratoria

Resumen
Objetivo: Presentar un caso de osteoporosis transitoria migratoria.
Caso clínico: Mujer de 50 años que desarrolla sucesivamente dolor en la cadera izquierda, la rodilla izquierda y el tobillo derecho. La resonancia magnética (RM) de cadera establece el diagnóstico de osteoporosis transitoria de cadera y las sucesivas migraciones establecen el diagnóstico de osteoporosis transitoria migratoria.
Resultados: La sintomatología remite con tratamiento conservador, con reposo, analgésicos, antiinflamatorios, descarga y fisioterapia hasta que los síntomas disminuyan. La descarga disminuye el dolor y previene la fractura por fatiga. El tratamiento médico no altera el curso de la enfermedad tras 65 semanas.

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Conclusión: La osteoporosis transitoria migratoria es rara, idiopática y autolimitada. Cursa con dolor migratorio en las articulaciones de carga, osteopoenia radiográfica difusa periarticular y patrón de edema medular óseo en la RM. Su tratamiento es conservador.

Introduction

Transient osteoporosis is a rare and idiopathic disease. It is characterised by joint pain, periarticular radiographic osteopoenia several weeks later, and spontaneous cure with no sequelae several months later. It was initially described in the hips of women during the third trimester of pregnancy.[1] It was later described with greater frequency in middle-aged men, which led to its labelling as transient hip osteoporosis. Publications also described transient osteoporosis in other areas: it was generally found affecting the weight bearing joints of the inferior limbs, leading to its current name: regional transient osteoporosis.

Between 10 and 40% of patients develop this condition in several joints either simultaneously or successively, in the same or contralateral limb (transient migrant osteoporosis).[2-5] The most common presentation is a proximal-distal extension along the limb.

At times, an intra-articular migration of the transient osteoporosis can be detected that affects different regions of the same joint. This evolution has been rarely described, above all in the knee. Even more rare cases of disseminated forms (polyarticular) are seen, with simultaneous and successive episodes in several joints.

Case report

Fifty-year-old woman with history of chronic back pain with frequent exacerbations in the form of a predominantly left-sided sciatica who came to the emergency room for acute, very intense lumbar pain irradiating to the left leg with two months evolution, which persisted even in repose and kept the patient from being able to sleep. Furthermore, she presented with the recent symptom of pain upon palpation of the left greater trochanter that increased with resisted hip abduction and rotations and that comprised one of the principal components of the pain suffered by the patient. There was no sign of motor or sensory deficits, we could not evaluate deep tendon reflexes, and plantar cutaneous reflexes were normal. Radiography showed a lumbosacral transition anomaly (S1 lumbarisation), pinching of the L4-L5 and less so of L5-S1, and lumbar scoliosis, without changes with respect to the previous exams; the left hip was normal. The patient was admitted with the diagnosis of left sciatica and left trochanteritis.

The patient was treated with bed rest, analgesics, anti-inflammatories, corticosteroids, and neuromodulators, and an unsuccessful corticoanaesthetic infiltration was attempted in the greater trochanter. The bone scan demonstrated hypercaptation in the hip joint, the femoral head, and the trochanteric region, indicative of osteonecrosis (fig. 1). Blood and urine analyses came back normal. The hip MRI demonstrated extensive signal changes in the femoral head, neck, and metaphysis that was hypointense on T1 and hyperintense on T2, as well as a joint effusion that was compatible with the diagnosis of transient hip osteoporosis (fig. 2). The patient was discharged after 12 days with treatment (anti-inflammatories, calcium, vitamin D, and nasal calcitonin). Hip mobilisation was authorised, but no weight bearing.

The radiograph follow-up at 8 weeks showed regional osteoporosis and a bone densitometry indicated femoral osteopoenia (T: -2.12, Z: ?1.42) with normal levels in the lumbar spine (T: ?0.33 and Z: ?0.60).

At 24 weeks, the hip and lumbar pain had diminished markedly, but explained the intense referred pain in the internal interline of the left knee; the radiograph was normal. At 26 weeks there was also referred pain in the internal maleolus of the right ankle; the radiograph also showed up normal. The knee MRI showed degeneration of the posterior horn of the internal meniscus, inflammation of the lateral internal ligament, marked sign changes in the internal femoral chondyle, lateral tibial plateau, and fibular head consistent with focal oedema/bone bruises, and loss of cartilage in the patellar joints (fig. 2). A second scan showed severe hypercaptation in the right ankle indicative of osteochondritis or osteonecrosis, moderate-severe hypercaptation in the internal femoral chondyle of the left knee indicative of osteochondritis, and hypercaptation in

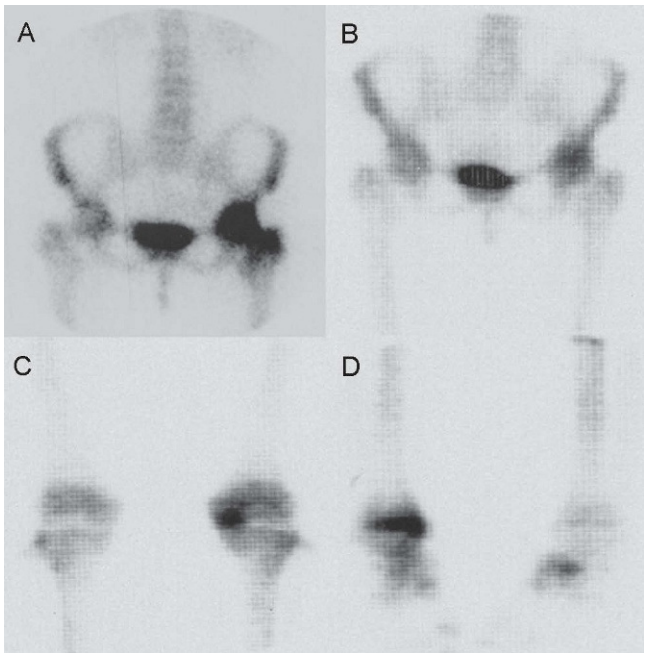

Figure 1 Sequence of bone scan images. A) Hips in the 1st gammagraphy. B) Hips in the 2nd gammagraphy. C) Knees in 2nd gammagraphy. D) Ankle in the 2nd gammagraphy.

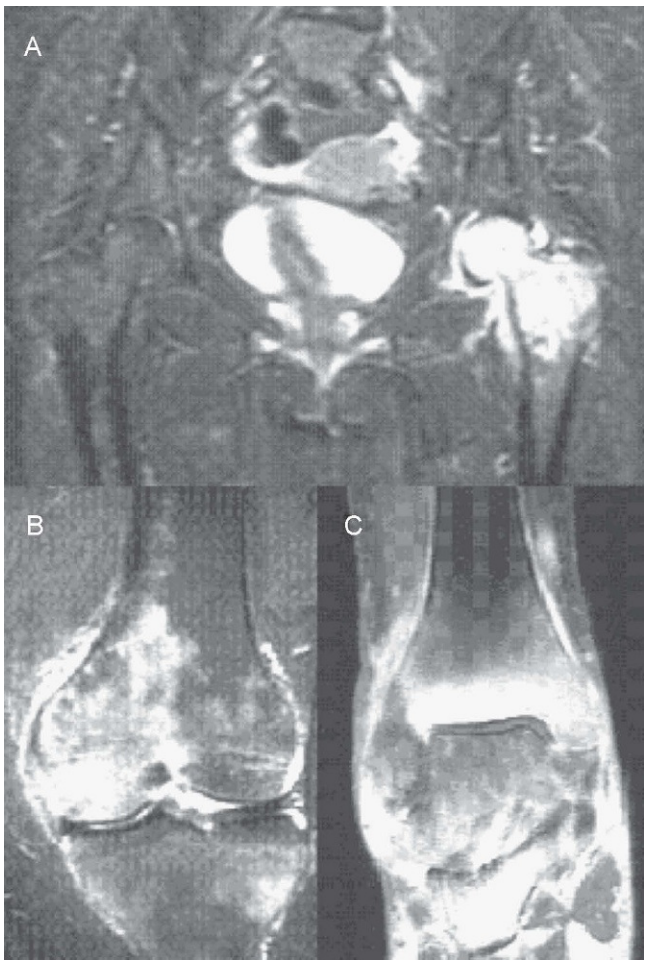

Figure 2 Sequence of MRI images. A) Hips. B) Left knee. C) Right ankle.

the left femoral head that was diminished in comparison to the previous scan (fig.1). At this point, the patient was diagnosed with transient migratory osteoporosis.

At 34 weeks, the referred pain in the left knee was occasional, but pain in the right ankle was intense. At 39 weeks, the right ankle experienced very intense pain with signs of inflammation, significant hobbling, and severe functional impotence, necessitating two canes for walking. The MRI of the ankle showed extensive inflammation concentrated in the tibial maleolus with mild presence in the talar dome, degenerative tibiotalar arthropathy, marked diffuse soft tissue edema, patchy osteoporosis affecting the talus, navicular, and wedge, and muscular atrophy (fig. 2). We prescribed functional rehabilitation. At 52 weeks, following an improvement in the ankle pain, it was increased, and weekly oral biphosphonate was prescribed.

At 65 weeks the patient was almost asymptomatic. Medical treatment was suspended except for the analgesics when needed and the biphosphonate, and she was discharged. We used densitometry for follow-up during 8 months. At this point, she had not presented any relevant pain in the left hip, left knee, or right ankle, and continued taking the biphosphonate. The densitometry showed femoral osteopoenia (T: −1.49, Z: −0.56, ↑ up: 103%) and lumbar osteopoenia (T: −1.25, Z: −0.33, ↓: 9.5%). The back pain had worsened, but the femoral pain had improved, demonstrating the recuperation from transient osteoporosis of the hip.

At present, 6 years later, she presents no symptoms attributable to transient migrant osteoporosis.

Discussion

Transient osteoporosis is rare, but is mentioned with ever-increasing frequency and is probably underdiagnosed.[3-6] It is usually localised in the weight-bearing joints of the leg: the hip (70%), knee, ankle, and foot.[6] Transient osteoporosis of the hip affects middle-aged adults (40-60 years), primarily men (3:1) and pregnant women who are around their third trimester.[1,4-8] It can be bilateral, simultaneous, or sequential in one third of patients.

Transient osteoporosis is idiopathic, but several aetiopathogenic theories have been proposed.[1-7] No known factors for predisposition exist except for pregnancy.[6,8] It has been associated with vertebral osteoporosis and systemic osteoporosis.[6] It is self-limiting[1,2,4-9] and remits following birth or abortion in pregnant women.[4]

Transient osteoporosis progresses with periarticular pain that increases with weight and improves with rest. The impacts on function can be severe, but pain and clinical findings can be scarce, a characteristic discrepancy. The symptoms reach a plateau and gradually revert with no sequelae in the first year after appearance.[1,2,4-7,9] The bones afflicted with transient osteoporosis can have fatigue fractures, above all subcapital fractures in transient osteoporosis of the hip during pregnancy,[5,7] that although rare, present a more serious complication.

Initial radiographs are normal, and at 3-8 weeks, radiographic diffuse osteopoenia appears around the joint with preservation of the articular space.[1,2,4-10] The bone scan gives an early indication within the first week of marked hypercaptation, both diffuse and homogeneous. It allows for the detection of migration and monitoring of the lesions, above all in transient migratory osteoporosis.[2,4,5,7-10]

An MRI shows the diminished sign intensity in the bone marrow of T1 and diffuse increase in T2, indicating bone marrow edema, without signs of osteonecrosis and joint effusion.[4,6,8-10] Exploration is preferred, as it is highly sensitive and can detect positive results within 48 hrs of the onset of symptoms.[5] This allows the exclusion of other illnesses as the cause of the condition and facilitates monitoring of the patient's progression. The bone marrow oedema is a non-specific finding that can be seen in other entities as well.[6,8-10] The densitometry shows localised bone demineralisation. Electromyography is generally normal. Laboratory tests also tend to be normal.[2,6-8] Joint liquid presents no alterations and cultures are negative.[8,9] The synovial biopsy can show a minimal non-specific chronic inflammation.[2,7,8] Pressure inside the bone marrow is elevated and interosseous venography exhibits reflux and stasis.[10]

A bone biopsy can turn out to be non-specific: it shows a varying combination of bone marrow oedemas, inflammatory changes, bone reabsorption, bone formation, fibrosis, osteoporosis, and medullar necrosis, but no bone necrosis.[2,4,7,10]

According to most authors, this tactic should not be used since the disease is self-limiting, requiring a conservative treatment, whereas the bone biopsy is a radical procedure.

Upon reversion of the disease, bone density is recovered and complementary explorations are performed within a few months.[1,2,4,7-10]

The differential diagnosis between bone marrow oedema syndrome, transient osteoporosis, and reflex sympathetic dystrophy, which heal spontaneously, and other aggressive conditions with long-term sequelae is essential. In this diagnosis, the clinical history, physical exam, localisation and extension of the lesions and laboratory results. Avascular necrosis is the principal differential diagnosis, since an early surgical treatment if of crucial importance.[4,5,8]

The treatment of transient osteoporosis is generally conservative, with rest, analgesics, anti-inflammatories, and unloading and physical therapy until the symptoms diminish.[4,5,7,9] Unloading diminishes pain and helps prevent stress fractures. Medical treatment does not alter the course of the illness.[5] Decompressive surgery of the femoral head in bone marrow oedema syndrome and transient osteoporosis of the hip immediately alleviates pain and increases mobility, significantly shortens the duration of the disease, allows a rapid return to work, and implies no complications around the operation and eliminates the progression of an avascular necrosis, and thus must be considered when symptoms are intense, prolonged, and discapacitating.[8,10] Transient migratory osteoporosis, episodes in other joints cannot be predicted.[5]

Südeck's algodystrophy, sympathetic reflex dystrophy, complex regional pain syndrome, bone marrow oedema syndrome, transient osteoporosis (regional or migratory), and avascular necrosis have similar symptoms, are idiopathic and present bone marrow oedema in the MRI, and as such, can make up different components of the same disease[9] that some authors describe as all falling under the syndrome of bone marrow oedema.[10]

Transient migratory osteoporosis is rare, idiopathic, and self-limiting. It progresses with migratory pain in weight-bearing joints, diffuse periarticular radiographic osteopoenia and a pattern of bone marrow oedema in the MRI. Treatment is conservative.

Conflict of interest

The author affirms having no conflicts of interest.

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