

Case report

Rapidly destructive osteoarthritis of the hip: Case presentation

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ABSTRACT

The rapidly destructive osteoarthritis of the hip is a variant of osteoarthritis that is characterized by hip pain during one to twelve months and rapid destruction of the femoral head, in absence of infectious, neurologic, metabolic or inflammatory disease. We present the case of a 66-year old male with severe COPD that referred pain in both hips and was unable to walk. In the radiography of the pelvis complete destruction of both femoral heads was observed. The patient did not have abnormalities in blood tests that were related to infectious, neurologic or metabolic diseases. The patient underwent bilateral total hip replacement with prosthesis and physical therapy later. The patient functional status greatly improved with this treatment.

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Artrosis rápidamente destructiva de cadera: a propósito de un caso

RESUMEN

La osteoartritis rápidamente destructiva de cadera es una variante de la osteoartritis caracterizada por dolor a nivel de cadera de uno a 12 meses de evolución asociada a una rápida destrucción de la cabeza femoral en ausencia de infección, enfermedad neurológica, metabólica e inflamatoria. Presentamos el caso de un varón de 66 años con EPOC severo que refería dolor en ambas caderas y dificultad para deambular de 6 meses de evolución. En la radiografía de pelvis se apreciaba la destrucción de ambas cabezas femorales. No tenía alteración analítica relacionada con enfermedad infecciosa, metabólica, inflamatoria y neurológica. El paciente fue intervenido quirúrgicamente sustituyendo ambas caderas por prótesis y posteriormente tratado con rehabilitación física presentando una notable mejoría.

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Palabras clave:

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Introduction

Rapidly destructive hip osteoarthritis is a rare syndrome of unknown aetiology, different from that of aseptic necrosis of the femoral head, which compromises the femoral head and acetabulum with the disappearance of the femoral head.¹ It was first described in 1957 by Forestier.¹

It is characterized by hip pain with an evolution from 1 to 6 months, associated with rapidly progressive atrophic bone destruction of the femoral head and acetabulum. This is evident in radiological studies, in the absence of signs of sepsis, neurological disease, metabolic or inflammatory symptoms.^{1,2}

Case report

We report the case of a 66-year old male with a history of severe COPD with bronchial hyperresponsiveness and emphysematous bullae in both lung apices, who was following oxygen therapy (15 h per day) at home combined with bronchodilators, diuretics and steroids (prednisone 10 mg) on alternate days. The patient had been admitted several times for exacerbations of COPD. Since the beginning of 2008 he had referred pain in the central lumbar region and in both hips upon ambulation. For this reason he was treated at the rheumatology and rehabilitation service. Complementary explorations were conducted which included a pelvic radiograph (Figure 1) and a lumbosacral CT which showed spondylolysis with spondylolisthesis of L5 and osteoarthritis of the hip respectively. The symptoms were attributed to these processes.

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Figure 1. The initial pelvic radiograph shows a narrowing of the joint space and subchondral sclerosis.



Figure 2. Destruction of both femoral heads and acetabulas.

Six months after the tests, the patient attended the physical medicine and rehabilitation service in a wheelchair, being unable to stand and referring intense pain in both hips. Further imaging studies and laboratory tests were requested.

The radiograph of the pelvis (Figure 2) showed the destruction of both femoral heads and acetabulas. The most significant findings of the blood test were: leukocytes $9.5 \times 10^3/\text{mm}^3$ (48.4% neutrophils), Hb 12 mg/dl, Hct 36, platelets $355 \times 10^3/\text{mm}^3$, ESR 32 mm/h, glucose 86 mg/dl, urea 44 mg/dl, creatinine 0.88 mg/dl, cholesterol 190 mg/dl, triglycerides 116 mg/dl, phosphorus 4.2 mg/dl, total calcium 9.5 mg/dl, PT activity 106%, INR 0.97, thromboplastin ratio 1.10, protein C 1.22, protein S 0.73, antithrombin III 0.99 U/l. Antinuclear antibodies, antiphospholipid antibodies and rheumatoid factor were negative, as were the serologies for hepatitis B, C, and HIV.

The patient underwent surgery for joint replacement. No crystals were observed in the synovial fluid and cultures for bacteria and fungi were negative. Anatomopathological studies of the extracted bone and synovial tissue removed (Figures 3 and 4) showed severe degenerative joint disease, avascular bone necrosis and chronic synovial inflammation.

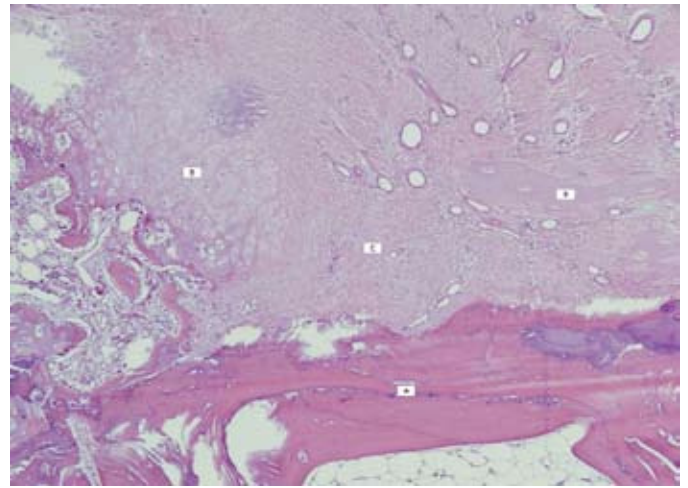


Figure 3. Areas of uninjured bone tissue (A), necrotic bone tissue (B) and cartilage (C).

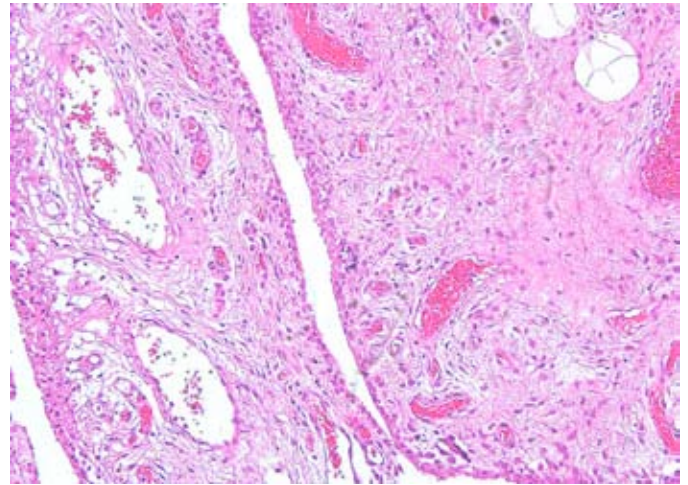


Figure 4. Synovial area with granulation tissue and chronic inflammation.

Prostheses were placed in both hips. The patient recovered without complications and subsequently received rehabilitation treatment based on pain control, recovery of the joint balance of both hips, improvement of muscular balance with isotonic and isometric exercises of quadriceps and gluteal muscles, rehabilitation of transfers and an early start of ambulation.

After surgical and rehabilitative treatment, the patient improved markedly until the pain practically disappeared. He maintains a correct articular balance of the prosthetic joints and walks with the aid of canes with few restrictions.

Discussion

Rapidly destructive hip osteoarthritis is a type of osteoarthritis which was first described by Forestier in 1957 and later became known as atrophic osteoarthritis, rapidly progressive osteoarthritis, destructive osteoarthritis and Postel osteoarthritis.^{1,2} Laquesne defined it as a narrowing of the articular space at a rate of 2mm/year or loss of articular space greater than 50% in 1 year.³

Its aetiology is not fully understood but it appears to involve three factors: mechanical stress, cartilage degeneration and bone response. If the cartilage degeneration is slow, then reparative

sclerosis and osteophyte formation take place, resulting in joint stability and hypertrophic osteoarthritis; if cartilage degeneration is rapid, then bone response is poor, resulting in atrophic or destructive osteoarthritis. Recently, subchondral bone ischemia and cell necrosis have been described as important factors in the pathogenesis. Other factors include NSAIDs, increased osteoclasts and the presence of high levels of IL6, IL1B and metalloproteinases at the synovial fluid level.³

The few published series have not described the use of steroids as a factor favouring rapidly destructive osteoarthritis since they excluded patients who received corticosteroid treatment, with the exception of 2 patients who received oral and intra-articular corticosteroids.^{3,4} Furthermore, information about the relationship between respiratory diseases or other pathologies causing hypoxemia and rapidly destructive osteoarthritis is virtually nonexistent. It could be assumed that the steroid treatment and the respiratory disease in this patient favoured joint disease, but more studies would be required.

The histological study is similar to the findings of osteoarthritis and secondary avascular necrosis, distinguished from idiopathic avascular osteonecrosis by the presence of necrotic areas alongside viable areas and areas of bone repair in bone tissue.^{3,4}

Rapidly destructive hip osteoarthritis affects mainly women between 57 and 84. The few studies published on this entity describe chronic hip pain of 12 months evolution or less, and the disappearance of the femoral head on radiographs at between 2 months and 4 years from the symptoms onset.^{3,4} In the presently described case, the symptoms lasted for approximately 1 year and the destruction of the hip was revealed in the radiographs after 6 months.

In the initial stages, the radiological images show narrowing of the joint space, subchondral bone sclerosis and cysts, and small or absent osteophytes. In advanced stages there is resorption of the femoral head and acetabulum and it is not unusual to observe a flattening of the femoral head and subchondral bone loss in areas which support weight.⁵ MRI scans show femoral head deformity, loss

of cartilage, a variable amount of synovial fluid, diffuse bone oedema and subchondral bone sclerosis.⁵

The diagnosis of rapidly destructive hip osteoarthritis is based on clinical, laboratory, radiological and histopathological findings, always after discarding the most common infectious, metabolic, and chronic inflammatory processes and neuropathies. It is suspected by clinical and radiology data and confirmed by the study of osteoarticular tissue samples taken before or during surgical intervention.⁶

The medical literature suggests that most patients with this condition may be candidates for hip joint replacement and subsequent rehabilitation. Thus, in a short period of time, patients may regain optimal performance with a short hospital stay, and similarly, achieve psychological and social reintegration into activities of daily living which they followed before the intervention.⁷

Conclusion

Rapidly destructive hip osteoarthritis is an uncommon, clinical and anatomical variant of osteoarthritis which should be suspected in patients with chronic hip pain who present clinical and radiological features characteristic of osteoarticular destruction.

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