

Bilateral Rapidly Destructive Arthrosis Case Report and Review

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Abstract

Rapidly destructive osteoarthritis or rapidly destructive arthrosis (RDA) of the hip is an uncommon disorder of unknown etiology where there is a rapid destruction of both the acetabulum and the femoral head. The condition typically affects elderly females with normal or osteoarthritic hip joints, and presents with new-onset severe hip pain and dysfunction. It is thought to be caused by extremely rapid osteoarthritic changes leading to an impact of the femoral head into the acetabulum, with subsequent osteonecrosis and insufficiency fracture of the femoral head. Differential diagnosis should include those conditions known to potentially lead to rapid hip destruction, such as septic arthritis, metabolic bone diseases, autoimmune inflammatory arthritis, malignancy and classical osteonecrosis. Sequential X-rays in patients with fast worsening of hip symptoms and a high degree of clinical suspicion seem mandatory to avoid extensive joint destruction and facilitate better arthroplasty outcomes in these patients. In the present report we present a clinical case of bilateral RDA, and we offer a useful review for clinicians on the differential diagnosis of this condition and the main physiopathological mechanisms behind its occurrence.

Keywords: Rapidly destructive osteoarthritis; Hip pain; Dysfunction; Malignancy; Physiopathological mechanisms

Introduction

Rapidly destructive osteoarthritis or rapidly destructive arthrosis (RDA) of the hip is an uncommon disorder of unknown etiology where there is a rapid destruction of both the acetabulum and the femoral head [1]. The condition typically affects elderly females with normal or osteoarthritic hip joints, and presents with new-onset severe hip pain and dysfunction. In the present report we present a clinical case of bilateral RDA, the first described in the gulf countries (GCC).

Clinical case

A 73-year old lady, natural from Iraq, presented to a private clinic in Abu Dhabi in January 2010 with a two-week history of right hip pain of mechanical features, with no previous history of trauma or past fractures. The patient had a past medical history of long-standing hypertension, hypercholesterolemia, hypothyroidism, chronic atrial fibrillation, and severe aorta stenosis. Her regular medications included candesartan, simvastatin, levothyroxine, atenolol and dabigatran (since February 2012, previously on warfarin) (Figure 1A).

The pain progressed rapidly despite pain killers, in the absence of other joint involvement or systemic features. Magnetic Resonance Image (MRI) of right hip in August 2010 showed well delimited triangular area in superior pole of right femoral epiphysis, hypointense in T1-weighted sequence and hyperintense in T2-weighted sequence and in short tau inversion recovery (STIR) sequence, together with moderate joint effusion and active early erosions in external part of ipsilateral acetabular cavity (Figure 2).

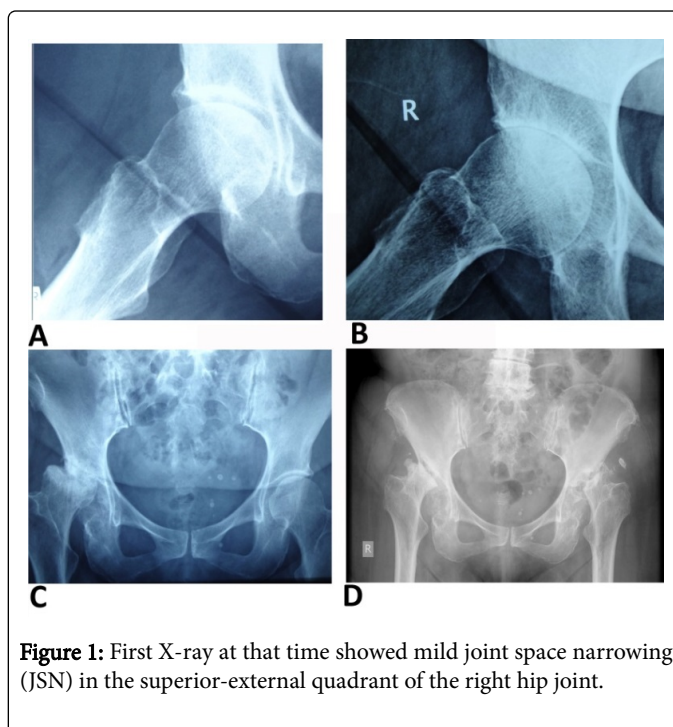


Figure 1: First X-ray at that time showed mild joint space narrowing (JSN) in the superior-external quadrant of the right hip joint.

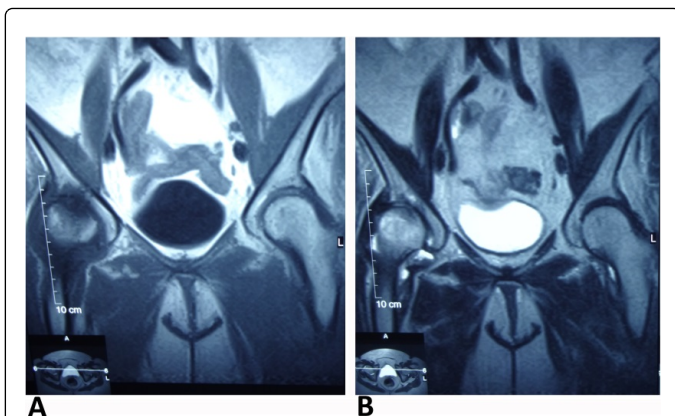


Figure 2: Active early erosions in external part of ipsilateral acetabular cavity.

The patient didn't respond to conservative therapy, suffering fast worsening of the hip pain and function. Screening for osteoporosis was undertaken in February 2011 by dual-X-ray-absorptiometry, showing severe osteopenia in lumbar spine. She was started, then, on oral weekly alendronate and oral calcium and vitamin D supplementation. Next X-ray in October 2010 showed severe JSN in the right hip joint and normal left hip joint appearance (Figure 1B). The patient was offered hip arthroplasty, but concerns aroused regarding her cardiovascular morbidity, and consequently conservative management remained. Early 2011, the patient started to suffer the first symptoms in the left hip, while pain and dysfunction in the right hip progressed to pain at rest and severe limping. New X-ray in February 2012 demonstrated JSN in left hip and remarkable flattening of right femoral head secondary to insufficiency fracture (Figure 1C). Given the high impact on patient's quality of life, elective total hip replacement was planned after heart valve replacement (bioprosthetic). One month after the heart surgery, the patient was admitted with lower limbs oedema and bilateral pleural effusion. Her transthoracic ECHO showed an ejection fraction of left ventricle of 55%, severe tricuspid regurgitation and estimated pressure at pulmonary artery (PAP) of 59 mmHg. Angiovascular computerized tomography (CT) of chest, nuclear ventilation/perfusion scan, doppler ultrasound of lower limbs, and angiogram of coronary arteries were normal. She was discharged on diuretic therapy with progressive clinical improvement of her heart failure episode. However, patient suffered progression of her bilateral hip syndrome. In March 2013, radiological findings included extensive destruction of both femoral heads and acetabuli (Figure 1D). The patient was then referred to orthopaedic surgery at our centre, who sought rheumatology advice for the screening of possible underlying conditions. In the rheumatology clinic, the patient did not present with symptoms or signs suggestive of systemic or infective disease. Peripheral white blood cell count, red cell count, platelets, electrolytes, renal function, and liver function test did not show remarkable findings; bone metabolism parameters (serum calcium and phosphorus, alkaline phosphatase, parathormone), thyroid-stimulating hormone, total cholesterol, glycosylated haemoglobin, iron profile, and inflammatory markers (C-reactive protein and erythrocyte sedimentation rate) were within normal limits; serum protein electrophoresis, rheumatoid factor, cyclic citrullinated peptid antibodies, antinuclear antibodies, anticardiolipin antibodies, lupus anticoagulant, anti-beta-2-glycoprotein antibodies, and nuclear

antigen antibodies were negative (except very mild increase of anti-SSB and anti-double strand DNA antibodies, which was not consistent when repeated). Activated partial time of thromboplastin (APTT) was moderately prolonged as expected in the context of dabigatran treatment, while international normalized ratio (INR) was within normal limits. Midstream urinalysis didn't show red cells, white cells, proteins or casts and 24 hproteinuria was 0.080 g/day. Chest XR showed mild cardiomegaly, while lungs and pleura appeared clear. All screening tests for infections (tuberculosis quantiferon, syphilis, brucella, HIV, HBV and HCV serologies) were negative. Despite cardiology evaluation found the patient fit enough to undergo surgery with new ECHO showing PAP of 32 mmHg, hip CT scan objectivised extensive destruction of both acetabuli (Figure 3), making the bilateral hip replacement a great technical challenge.



Figure 3: Hip CT scan objectivised extensive destruction of both acetabula.

Discussion

We present a case of bilateral RDA, to our knowledge, the first ever reported from the GCC region. Rapid destructive arthrosis was first described in 1970 by Postel and Kerboull [2] as an uncommon condition of unknown etiology where there is a rapid destruction (6-12 months) of both the acetabulum and the femoral head. In one longitudinal study published in 2006, RDA was defined by a loss of more than 50% of the joint space at the narrowest point between two evaluations 1 year apart [3]. More recently, Richette and colleagues defined RDA as a severe hip pain developed within the last two years and with an annual rate of JSN of >1mm [1]. Regardless of the exact definition used, the key concept is that radiological changes in RDA are possible to occur in very advanced osteoarthritis (OA), albeit the progression is extremely fast compared with the usual slowly progressive OA (SPOA) [4].

Our patient presented the most important imaging features proposed for this condition [4,5]: joint effusion, bone marrow oedema in femoral head and acetabulum, and femoral head flattening. The condition typically affects elderly females with normal or osteoarthritic hip joints [1,3,4-6]; despite unilateral involvement is more common in RDA[4], bilateral cases have been previously reported [4,7].

Although the etiology of this disorder still remains unclear, several mechanisms have been postulated in the physiopathology of the condition. Osteonecrotic changes and insufficiency fractures are seen both in MRI studies [5] and histological examinations [7], and thought to be caused by extremely rapid osteoarthritic changes leading to an impact of the femoral head into the acetabulum. The role of low bone mineral density (BMD) in the pathogenesis of RDA has also been investigated as a differential feature from SPOA, as osteoarthritic changes have been generally related to a higher BMD compared to

general population [8,9]. A 7-year prospective study [6] compared BMD measured by dual-x-ray absorptiometry in non-hip sites between RDA cases and SPOA cases among candidates for total hip replacement. The investigators did not find significant differences in mean BMD values among both groups. Similar results had been reported previously [1]. The value of cartilage and bone degradation markers to differentiate RDA from usual SPOA has also been investigated. Degradation markers of type I collagen (serum cross-linking C-terminal telopeptide) [10] and type II collagen (urinary crosslinking C-terminal telopeptide) [11] have shown to be increased in RDA patients in comparison to controls with SPOA, reflecting the fast degradation of bone and cartilage, respectively, in RDA. Likewise, serum and synovial levels of metalloproteinases, which seem to play an important role in the extracellular matrix degradation in patients with OA, have been found to be significantly higher in patients with RDA than in those with SPOA [12]. Such markers could be helpful in early diagnosis of RDA in OA patients with fast progressing symptoms; however there is no sufficient data to establish laboratory standard thresholds. Other investigators have recently reported that high osteoclastic activity in synovial fluid differentiates RDA patients from those with osteonecrosis of femoral head or developmental dysplasia of the hip [13].

Differential diagnosis should include those conditions known to potentially lead to rapid hip destruction, such as septic arthritis, metabolic bone diseases hyperthyroidism, hyperparathyroidism, Cushing syndrome, autoimmune inflammatory arthritis (rheumatoid arthritis, ankylosing spondylitis, and systemic lupus erythematosus), malignancy and classical osteonecrosis. Less common causes that occur with severe destruction of the hip include osteolytic syndromes (e.g., Gorham disease) and osteochondritis dissecans. Thrombophilic profile should be thorough in order to rule out antiphospholipid syndrome (primary or secondary) and other hypercoagulability syndromes shown to be involved in the physiopathology of some aggressive cases of osteonecrosis, particularly when more than one site is affected. High prevalence of prothrombotic abnormalities, for example, has been observed in multifocal osteonecrosis, which involves 3%-10% of patients diagnosed with osteonecrosis and it's defined by the involvement of 3 or more anatomic sites [14]. In our patient, antiphospholipid profile was negative, and the APTT was prolonged in the context of her anticoagulant. Further coagulation studies would have probably required withdrawing the anticoagulant for a proper investigation. However, we thought that it was not clinically justified given her high risk of thrombosis and the presence of long-term oral anticoagulation that made the hypercoagulability mechanism unlikely to be the origin of her hip destruction.

There is no specific treatment for patients suffering from RDA. A longitudinal study assessing the role of weight-bearing elimination and fluoroscopic-guided intra-articular glucocorticoid injection didn't find a reduction in the need for total arthroplasty in patients with RDA compared to those with SPOA [3].

Conclusion

Rapid destructive arthrosis is an uncommon condition that can lead to rapid destruction of the hip joint. High speed of JSN and early

involvement of acetabulum are typical. Sequential XRs in patients with fast worsening of hip symptoms and a high degree of clinical suspicion seem mandatory to avoid extensive joint destruction and facilitate better arthroplasty outcomes in these patients.

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