Bilateral rapidly destructive hip osteoarthritis: a case report

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ABSTRACT

Although hip osteoarthritis usually shows a slow progression, a rapidly destructive osteoarthritis is observed in approximately 10% of patients. We aimed to present a case with rapidly destructive osteoarthritis in bilateral hip joints. A 78-year-old male patient was admitted due to pain in hip joints. On examination, hip movements were minimally painful and limited. The patient was able to walk independently with a cane. When he re-applied six months later, hip movements were severely limited and painful. In plain radiographs, while a slight narrowing in hip joint space, sclerosis and minimal osteophyte had been observed at the first observation, extreme narrowing, subluxation, flattening of femoral head, increased sclerosis, resorption in femoral head and acetabulum were detected six months later. We consider that hip osteoarthritis in elderly people should be monitored at frequent intervals to assess both clinic and radiological progression.

Keywords: Rapidly destructive osteoarthritis; Hip

INTRODUCTION

Rapidly destructive osteoarthritis of hip is a rare syndrome characterized by rapidly progressing joint degeneration and destruction, without any specific diagnosis. Its synonyms are rapidly progressing destructive hip disease, Postel's disease, rapidly destructive arthropathy, atrophic osteoarthritis, and rapidly destructive coxarthrosis[1,2]. Although hip osteoarthritis usually shows a slow progression, rapidly destructive osteoarthritis is observed in approximately 10% of patients[3]. It was identified for the first time simultaneously by Lequesne *et al.*[4] and Roux *et al.*[5]. Within months, along with a rapid narrowing of joint space, symptoms of pain increase[1]. Lequesne has emphasized that a narrowing more than 2 mm in joint space and more than 50% loss of joint space within a year supports diagnosis[6].

We aimed to present a case with rapidly destructive osteoarthritis in bilateral hip joints.

CASE REPORT

A seventy eight year old male patient was admitted due to pain in hip joints and gait disturbance, which had been on-going for 2 years. His pain was increasing with movement and walking. In his history, he had undergone benign prostatic hyperplasia surgery, 5 years ago and coronary bypass surgery, 6 years ago. There was no history of other diseases (infectious, tumoral, rheumatologic or neuropathic). He was under treatment with paracetamol 2g/day. In musculoskeletal system examination, right hip flexion was 110 degrees, left hip flexion was 120 degrees, internal/external rotations were respectively 20/30 degrees, painful and limited in both hips. The patient, whom pain level was 6/10 by visual analog scale, was able to walk independently with a cane at the end of physical therapy and rehabilitation program. When he was admitted again six months later, due to increase in his hip pain, hip flexion was 45 degrees on the right hip and 30 degrees on the left, internal/external rotations were painful and limited at 0 degrees on the right hip and 10/0 degrees on the left. He had difficulty sitting position. Barthel Index of the patient was 25 which mean extremely dependent in his daily activities[7]. His pain level increased to 9/10. In

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FIGURA 1. A slight narrowing in hip joint space, increased sclerosis and minimal osteophyte were observed in pelvis radiography at the time of his first application

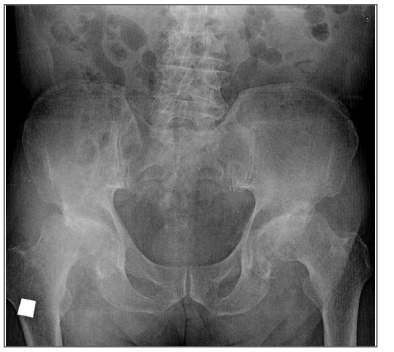

FIGURE 2. Severe narrowing in superior bilateral hip joint space, superolateral subluxation, flattening of femoral head, increased sclerosis, resorption in femoral head and acetabulum, and subchondral defect were detected in his radiography taken six months later

laboratory analyses, ESR was 34 mm/h, CRP 5,9 mg/L, Hb 13,9 g/dL, Hct 41,7%, free PSA 0,6 ng/mL, and other complete blood count and biochemical test results were normal. In his first radiological examinations, a slight narrowing in hip joint space, sclerosis, and minimal osteophyte was observed (Figure 1). Nevertheless, severe narrowing in superior bilateral hip joint space, superolateral subluxation, flattening of femoral head, increased sclerosis, resorption in femoral head and acetabulum and subchondral defect were detected in the radiography taken six months later (Figure 2). In magnetic resonance imaging, joint effusion, cystlike subchondral defects, femoral heads flattening, loss of height, superolateral subluxation, ankylosis and advanced degenerative findings were reported. Bilateral total hip arthroplasty was planned for the patient, who was consulted with the Orthopaedic Clinic. But the patient refused to have surgery.

DISCUSSION

Destructive arthropathy of the elderly involves many large joints in addition to the shoulders, and some patients also have erosive osteoarthritis of fingers[8]. Rapidly destructive hip osteoarthritis is a seldom seen form of hip osteoarthritis, mostly encountered in elderly postmenopausal women. Osteoarthritis is characterized by rapid chondrolysis in coxo-femural joint[9]. It is regarded as a variant, a severe form and an inflammatory phase of osteoarthritis[2,10]. In most recent studies, it is pointed out that subchondral insufficiency fracture in femoral head, which induces rapid chondrolysis, subchondral bone ischemia and cell necrosis all play a role in physiopathology of the disease[11-13]. It has been reported that patients who have subchondral insufficiency fracture are mostly osteopenic and osteoporotic[11,12]. In the study by Richette *et al.*[9] it has been remarked that systemic low bone mass does not play a role in rapidly destructive hip osteoarthritis physiopathology. Likewise, in the study by Okano *et al.*[14] it has been stated that there is no relation between rapidly destructive osteoarthritis of hip and osteoporosis. Although etiology of disease is not fully understood, it is asserted that mechanical stress, cartilage degeneration, and bone response may be responsible. If cartilage degeneration is slow, reparative sclerosis and osteophyte formation take place and result in joint stability and hypertrophic osteoarthritis. If cartilage degeneration is rapid, bone response is poor and results in atrophic or destructive osteoarthritis. Other factors that might be responsible for this disease are NSAIDs, increased osteoclasts and the presence of high levels of IL-6, IL-1 , and metalloproteinases at the sinovial fluid[2,8,10,13].

Rapidly destructive hip osteoarthritis is usually seen in women aged between 57 and 84 years. Presence of destruction in femoral head and/or acetabulum in radiographies taken 2 months and 4 years after the beginning of symptoms has been reported in studies[1,2,10]. Nguyen *et al.*[15] have reported an 81-year-old female patient, in whom total destruction of hip cartilage related to rapid and massive osteolysis of unilateral femoral head and acetabulum developed within a year. Amao Ruiz *et al.*[2] have reported a 66-year-old patient, who had a story of chronic obstructive pulmonary disease (COPD), streoid use and hip pain for a year, and admitted while on a wheelchair with the pain increased with-

in the last six months. They have shown destruction and resorption in both femoral head and acetabulum, flattening of femoral head and subchondral bone loss in radiographies taken six months later. The age of our patient (78 years) was consistent with the age group in literature, and similarly with the case of Amao Ruiz *et al.*[2] in particular, rapid development of bilateral hip destructive osteoarthritis within six months was observed. Findings similar to the cases of Nguyen *et al.*[15] and Amao Ruiz *et al.*[2], such as severe narrowing in bilateral hip joints, superolateral subluxation, flattening of femoral head, increased sclerosis, resorption in femoral head and acetabulum, and cystlike subchondral defect were detected.

Boutry *et al.*[16] indicated that the key MR imaging features of rapidly destructive hip osteoarthritis included joint effusion, bone marrow edema-like pattern in the femoral head and neck or acetabulum or both, femoral head flattening, and cystlike subchondral defects. Our patient's MR images, except for bone marrow edema-like pattern, showed similar findings with Boutry *et al*.

In a series of rapidly destructive hip osteoarthritis cases retrospectively studied by Batra *et al.*[10], non-specific serious degenerative changes along with marginal osteophyte were seen in a couple of cases, but primary osteonecrosis, pannus formation and crystal deposits were not detected. Similar to findings by Batra *et al.*[10], any finding suggesting subchondral ischemia, bone marrow edema, or osteonecrosis that are mentioned in literature was not detected in MR images of our patient.

For differential diagnosis of rapidly destructive hip osteoarthritis, primary osteonecrosis, rheumatoid arthritis, neuropathic arthropathy, or septic arthritis should be considered[10,17]. In literature hip joint replacement followed by a rehabilitation program is recommended for most patients[1,2,10]. Because of technical problems in surgery that are caused by acetabular defect early diagnosis is important[10]. In the study by Kuo *et al.*[1], it has been emphasized that in rapidly destructive hip osteoarthritis clinically and radiologically good results can be obtained by means of hybrid and cement-free total hip arthroplasty.

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