

Rapid Acetabular Osteolysis Secondary to Subchondral Insufficiency Fracture

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ABSTRACT. A 93-year-old man presented with a one-month history of persistent left hip pain of sudden onset. At first visit, radiographs revealed a fracture line at the medial portion of the acetabulum with no displacement. Magnetic resonance imaging revealed bone marrow edema in the corresponding medial portion of the acetabulum. Radiographs obtained 2 months later showed rapid acetabular osteolysis with associated prominent migration of the femoral head into the acetabulum. Histology obtained from the hip joint was consistent with a subchondral insufficiency fracture with no evidence of massive chondrolysis. Our case was considered as a subchondral insufficiency fracture of the left acetabulum resulting in rapid acetabular osteolysis (protrusio acetabuli). (J Rheumatol 2007;34:592–5)

Key Indexing Terms:

OSTEOLYSIS

ACETABULUM

INSUFFICIENCY FRACTURE

The concept of rapidly destructive arthrosis of the hip joint was proposed by Postel and Kerboul in 1970¹. This disease is most commonly seen in elderly women with unilateral involvement. Radiographic characteristics are disappearance of the joint space followed by a rapid joint destruction within 6 to 12 months. The majority of cases show no evidence of antecedent osteoarthritis (OA), osteonecrosis, neuropathy, infection, or inflammatory disease¹⁻³. As to the etiology of rapidly destructive arthrosis, various theories have been proposed, including a variant of rheumatoid arthritis (RA) and osteonecrosis, idiopathic chondrolysis, apatite crystal deposition, drug toxicity, or abnormal immunoreaction⁴⁻⁸.

In the past decade, subchondral insufficiency fracture of the femoral head has been reported in elderly women with osteoporosis and also in renal transplant recipients^{9,10}. Some cases of subchondral insufficiency fracture have been reported to show rapid disappearance of the hip joint space and rapid joint destruction, such as that seen in rapidly destructive arthrosis. Therefore, we propose that subchondral insufficiency fracture of the femoral head could be one of the causes of rapidly destructive arthrosis of the hip joint^{11,12}. Recently, a

case of subchondral insufficiency fracture in both the femoral head and acetabulum has been reported to undergo rapid disappearance of the joint space¹³.

We describe the onset of rapid acetabular osteolysis and the progress of hip joint destruction, probably caused by a subchondral insufficiency fracture in the medial portion of the acetabulum.

CASE REPORT

A 93-year-old man had a one-month history of sudden-onset left hip pain. He was only able to walk minimally with use of a walker and was unable to negotiate stairs. He had no history of any antecedent trauma to the left hip joint. The range of motion in the left hip was 80° in flexion, 0° in extension, 20° in abduction, 10° in adduction, 25° in external rotation, and 10° in internal rotation. His height was 164 cm and body weight 75 kg. Body mass index indicated he was overweight (27.9 kg/m²). A blood examination revealed no abnormality in renal or liver functions. No evidence of RA, infection, or neuropathy was noted. Bone densitometry data were not available, although he was taking alendronate (70 mg/wk).

Three months before the onset of left hip pain, he had had a total knee replacement in the left side for a 12-month history of progressive knee pain, due to medial OA of the left knee. Before the knee surgery, he was only able to tolerate minimal ambulation, but after the knee surgery he could walk half a mile until the onset of left hip pain.

He also had a history of hypertension, prostatectomy for benign prostatic hypertrophy, myocardial infarction at the age of 58 years, Parkinson's disease, fracture of a vertebral body in the thoracic spine, and intertrochanteric fracture in the right hip about one year before the onset of left hip pain. He had no history of smoking, and alcohol consumption was a glass of wine nightly.

Clinical course. Radiographs, obtained at the first visit, showed a fracture line with no displacement in the medial portion of the left acetabulum on the lateral view; the fracture was not apparent on the anteroposterior view (Figures 1A, 1B). Magnetic resonance imaging (MRI) obtained at the same time revealed a bone marrow edema pattern in the corresponding medial portion of the acetabulum (Figure 1C). No abnormality was noted in the femoral head.

In the right hip, there was an intertrochanteric fracture treated with a telescoping screw and side plate, in which the greater trochanter is superiorly displaced lying at the level of the acetabular roof, and there was some heterotopic ossification.

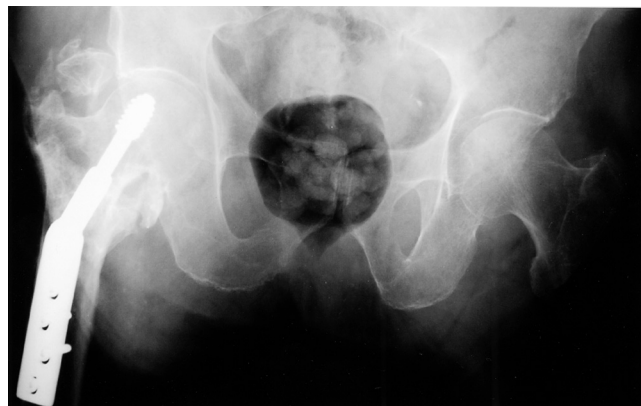
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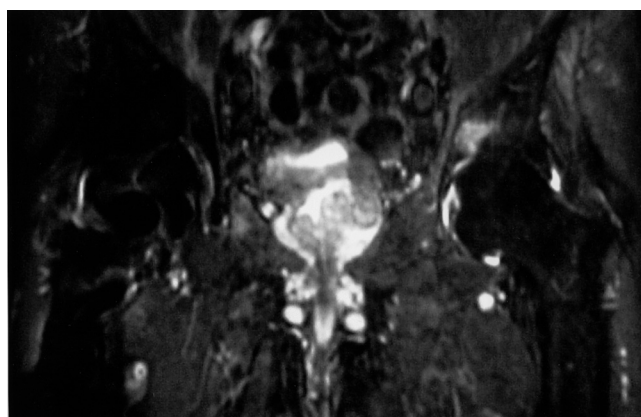
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A



B



C



D

Figures 1A to 1D. Anteroposterior radiograph obtained at the first visit shows no obvious changes (A), but on a lateral view there is a fracture line in the medial portion of the left acetabulum (arrow, B). C. Coronal proton-density-weighted image with fat suppression shows a high signal intensity in the corresponding medial portion of the acetabulum (TR/TE = 6216/22 ms). D. Radiograph obtained 3 weeks later shows destruction of the superomedial portion of the acetabulum, into which the femoral head is migrated. The joint space and shape of the femoral head are relatively preserved.

As treatment he was not to bear weight and was prescribed low dose oral propoxyphene napsylate, but the pain in the left hip worsened. Three weeks later, radiographs showed destruction of the superomedial portion of the acetabulum, into which the femoral head had migrated. Both the joint space and shape of the femoral head were relatively preserved (Figure 1D). Fractures of the inner wall of the acetabulum were observed on computerized tomography. On the radiographs obtained one month later, left hip joint destruction had progressed further, the joint space showed narrowing, and the femoral head had undergone marked deformity (Figure 1E). Because of the severe left hip pain, the patient underwent total hip arthroplasty.

Histological findings. The specimens obtained at total hip replacement showed fragmented articular cartilage, a distorted femoral head, and sclerotic synovium. The articular surface of the femoral head showed degenerative and proliferative changes with focal, irregular erosion and fissuration of the cartilage at the superolateral portion, but no evidence of massive chondrolysis was noted. The femoral head showed thickened bone trabeculae with associated fracture callus formation at the superior portion, with no evidence of primary osteonecrosis (Figure 1F). In the marrow space, there was a large number of round to oval-shaped granulomatous lesions, where tiny fragments of bone tissue were embedded in amorphous eosinophilic debris surrounded by epithelioid histiocytes and giant cells (Figure 1G). This finding has been

reported as a characteristic pathologic appearance in the rapid destruction of the joint¹¹. The synovium showed extensive hypertrophy due to a large amount of cartilaginous detritus (Figure 1H), but there was no evidence of synovitis suggesting RA. Thin, disconnected bone trabeculae indicative of osteoporosis were observed at the remaining intact area. These histological findings were consistent with a subchondral insufficiency fracture, resulting in a rapid destruction of the joint.

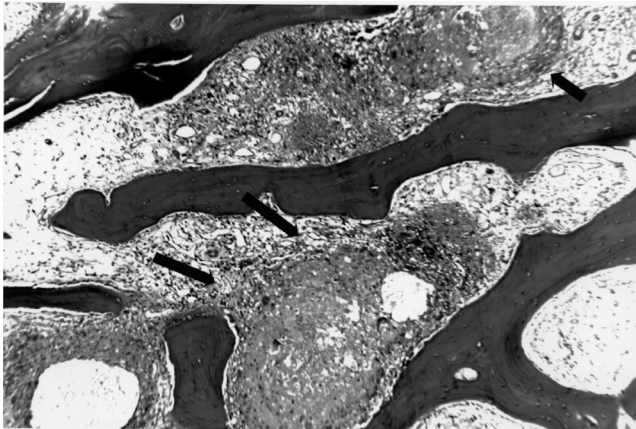
DISCUSSION

Clinically, several morbid conditions have been reported to be associated with rapid joint destruction, including articular chondrocalcinosis, apatite crystal deposition, neuropathy, infection, drug induced arthropathy, and a variant of osteonecrosis and RA⁴⁻⁸. We did not observe any of these conditions clinically or histopathologically.

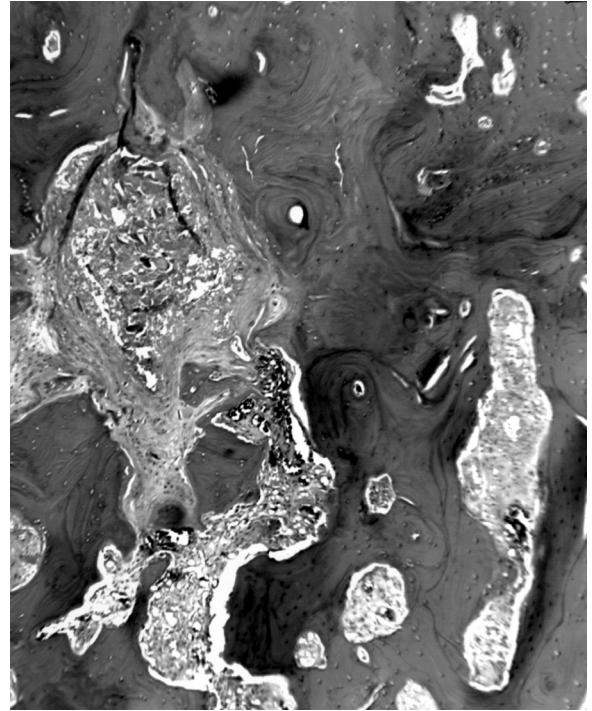
Our initial diagnosis of subchondral insufficiency fracture of the acetabulum was based on the radiographic evidence of a fracture supported by published characteristics of the insufficiency fracture, including old age, overweight, acute onset



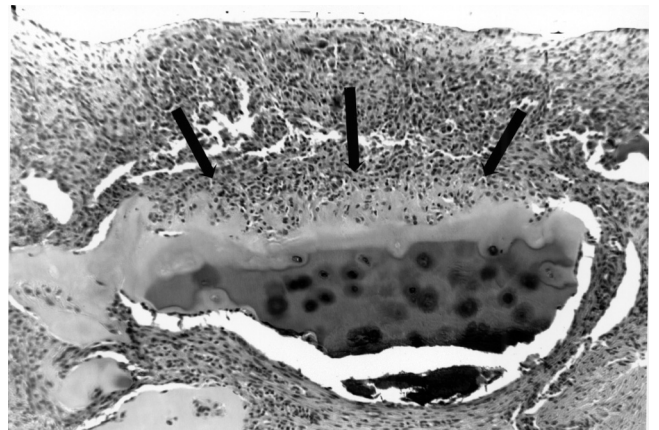
E



G



F



H

Figures 1E to 1H. On the radiograph one month later, left hip joint destruction has progressed. Joint space narrowing is observed and the femoral head has undergone marked deformity. F. The surface of the resected femoral head shows thickened bone trabeculae with associated fracture callus formation at the superior portion. There is no evidence of primary osteonecrosis (hematoxylin and eosin; original magnification $\times 100$). G. In the marrow space, large numbers of round to oval-shaped granulomatous lesions are noted, where tiny fragments of bone tissue are embedded in amorphous eosinophilic debris surrounded by epithelioid histiocytes and giant cells (arrows) (hematoxylin and eosin; original magnification $\times 100$). H. Synovium shows a cartilaginous detritus containing a large fragment of articular cartilage (arrows) (hematoxylin and eosin; original magnification $\times 200$).

of hip pain, bone marrow edema on MRI, and histologic evidence of a fracture^{9-12,14}. Since radiographs show no obvious changes in the early phase of subchondral insufficiency fracture, MRI examination would be of help for the detection of subchondral fracture^{15,16}.

It is our hypothesis that the etiology of the fracture resulted from his increased daily activity after his total knee replacement and associated minor trauma on the hip joint, which probably had been osteoporotic before the knee surgery due to the secondary osteoporosis based on immobility and reduced physical activity. Shear force due to axial loading

applied to the acetabulum may have played some role in the fracture on the medial aspect.

Rapid hip joint destruction was seen within 2 months after the onset of hip pain, and was at first predominantly in the acetabulum, resulting in rapid osteolysis. The impact of the femoral head on the fractured acetabulum as a result of daily sitting or walking could have led to the further fracture of the acetabulum as well as of the femoral head. However, the mechanism of rapid joint destruction is multifactorial. Many factors seem to play an important role in the pathogenesis of rapid joint destruction, including increased levels of bone resorptive enzymes and synovitis resulting from the initial fracture, as well as the use of antiinflammatory drugs, being overweight, and degree of osteoporosis^{11,12,17-19}.

It has been suggested that chondrolysis is an important factor in the etiology of rapid joint destruction^{1,2,17-19}. But we believe that rapid cartilage destruction in this case was traumatic and not the result of chondrolysis based on the following: (1) Clinically, chondrolysis is generally a severe event, in which the cartilage over most of the articular surface is necrotic. In our case, histopathologic examinations revealed preserved viable articular cartilage except for the area of cartilage loss on the superior surface; (2) fragments of the articular cartilage with or without attached subchondral bone tissue were frequently observed in the marrow space as well as in the synovium, as shown in Figure 1H. This would seem to indicate that subchondral fracture occurred prior to the loss of articular cartilage.

Some cases of subchondral fracture in the femoral head were reported to cause rapid destruction of the hip joint^{1-3,11,12}. We believe that little has been written on the pathology of the acetabulum in cases of arthritis^{13,20}. Since the etiology of rapidly destructive arthrosis of the hip joint is still unknown, investigations of the acetabular side may help elucidate the pathogenesis of rapidly destructive arthrosis of the hip joint.

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