Case Report

Rapid Destruction of the Femoral Head After a Single Intraarticular Injection of Corticosteroid into the Hip Joint

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ABSTRACT. We describe a 50-year-old woman who had a 10-year history of left groin pain. She experienced rapid collapse of the femoral head during a 3-month period after a single injection of corticosteroid into the hip joint, in which osteonecrosis was histologically observed. (J Rheumatol 2006;33:1701–4)

Key Indexing Terms:
HIP CORTICOSTEROID OSTEONECROSIS

The concept of rapidly destructive arthrosis of the hip joint was proposed by Postel and Kerboull in 1970[1]. The condition is most commonly seen in elderly women with unilateral involvement. Radiographic characteristics are disappearance of the joint space followed by rapid joint destruction within 6 to 12 months. The majority of cases show no evidence of antecedent osteoarthritis, osteonecrosis, neuropathy, infection, or inflammatory disease[1-4]. Occasional cases diagnosed as osteonecrosis of the femoral head have been reported to undergo rapid joint destruction, similar to that seen in rapidly destructive arthrosis of the hip joint[5]. The majority of these reported cases were women who received high dose of corticosteroids as treatment for underlying conditions such as systemic lupus erythematosus, nephrotic syndrome, and renal transplant. A few such cases have also been reported after multiple intraarticular injections of corticosteroids[6,7].

We describe a case of rapid progression of the collapse in the femoral head during a 3-month period after a single injection of corticosteroid into the hip joint, in which osteonecrosis was histologically observed.

CASE REPORT

Clinical course. A 50-year-old woman had a 10-year history of left groin pain. She first presented at our hospital because of recent increase in the severity of pain. Her height was 163 cm and body weight was 78 kg. Body mass index indicated she was overweight (29.4 kg/m^2). There was no history of corticosteroid intake or alcohol abuse. Radiographs showed slight joint space narrowing at the superomedial portion of the left hip joint (Figure 1A). Singh Index on the affected left hip was considered grade 3, indicating the presence of osteopenia[8]. Two months later she returned because the hip pain gradually had got worse. Magnetic resonance imaging (MRI) at this time revealed an increase of joint fluid and iliopsoas bursitis in the left hip; however, no abnormality was noted throughout the femoral head on T1 weighted image (Figure 1B). On T2 weighted image, a high signal intensity lesion was noted in the left acetabulum (Figure 1C). One week later, an intraarticular injection of corticosteroids into the left hip joint was scheduled; at that time the hip was aspirated and clear yellow-tinged fluid was observed. Cell count, culture, and sensitivity test were performed, which all showed negative findings for infection. No crystals were observed in the joint fluid. Following injection of 1 cc Hypaque 60, Depo-Medrol (methylprednisolone acetate: 80 mg) and sensorcaine (0.5%) were injected. This corticosteroid injection improved the symptoms by about 50%, but fairly quickly the pain increased again and rapidly got worse, with no evidence of trauma.

Three months after the injection, radiographs showed a flat and sclerotic femoral head, which was subluxed superolaterally (Figure 1D). A blood examination revealed normal white blood cell count (7860 xxx), triglycerides (190 mg/dl), and a slight increase of cholesterol level (237 mg/dl, normal range 150–200). No abnormality was found in renal or liver functions, and no evidence of neuropathy or diabetes mellitus was noted. The range of motion in the left hip was 30° in flexion, 0° in extension, and other positions were not possible due to the severity of pain. The patient underwent total hip arthroplasty, when no purulent material was observed. Multiple culture examinations all were negative. One year after the operation, there was no evidence of infection or loosening.

Histological findings. The resected femoral head was markedly distorted with a saddle-shaped deformity. A 2.5 × 2 cm flap of the articular cartilage was attached to a small region of the anterior margin of the femoral head. On a mid-coronal cut section, a yellow, opaque, osteonecrotic lesion was observed only in the flapped lesion but not in the remaining femoral head.

Microscopically, the overlying cartilage showed cellular proliferation, but no evidence of infection or chondrolysis was noted. The bone attached to the overlying cartilage showed total necrosis of the bone trabeculae and bone marrow tissue (Figure 2A). There were a few round to oval shaped granulomatous lesions in the marrow space, where tiny fragments of bone were embedded in amorphous eosinophilic debris surrounded by epithelioid histiocytes and giant cells (Figure 2B). Vascular-rich granulation tissue was seen in the remaining femoral head, where appositional bone formation or callus formation was not obvious. No evidence of acute or chronic infection was noted.

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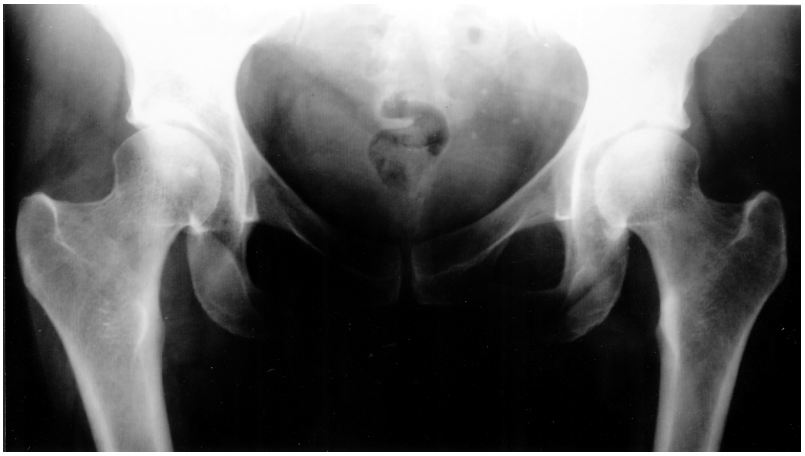

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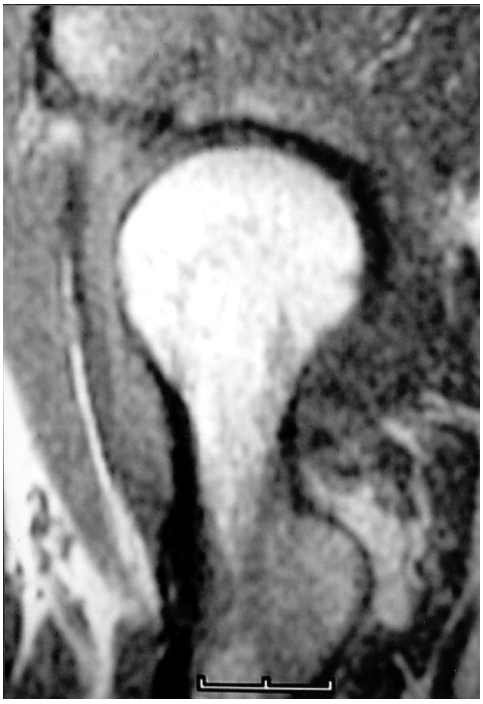

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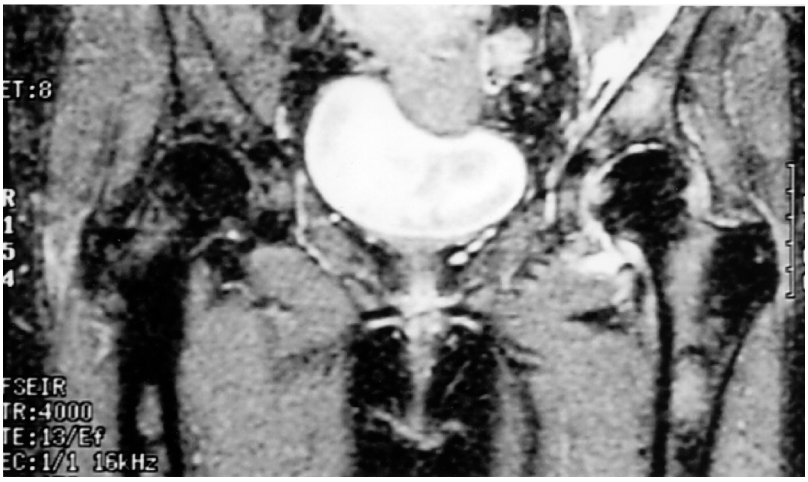

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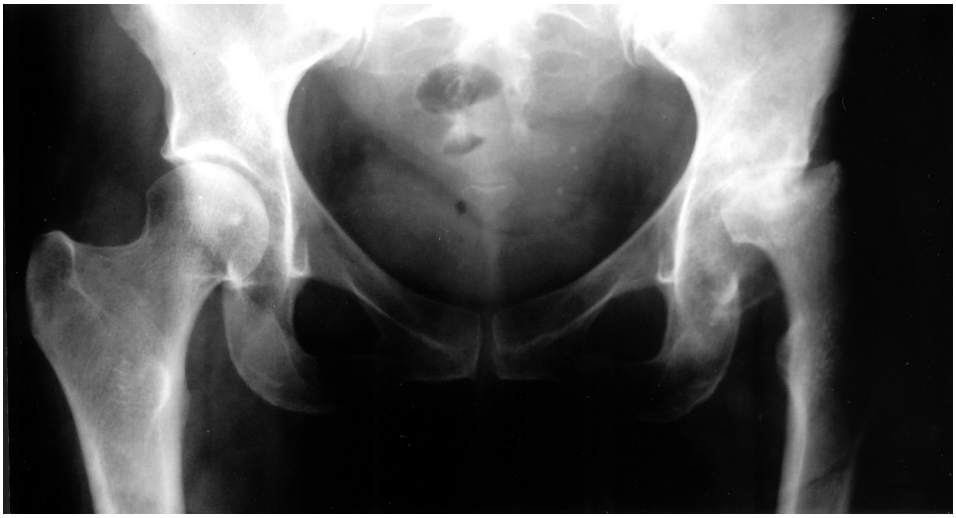

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Figure 1. A. Radiograph at the first visit reveals slight joint space narrowing at the superomedial portion of the left hip joint. Singh Index was considered grade 3, since there was a break in the continuity of the principal tensile trabeculae. B. No abnormality is seen in the femoral head on sagittal section of T1 weighted MR image (TR/TE: 500/13) 1 week before the corticosteroid injection. A low intensity band is not observed. C. On T2 weighted image (TR/TE: 4000/13), an increase of joint fluid can be seen, while no abnormality is visible in the femoral head. A high signal intensity lesion is visible in the left acetabulum. D. Radiograph obtained 3 months after the corticosteroid injection shows a flat and sclerotic femoral head with a saddle-shaped deformity, which is subluxed superolaterally. Severe joint space narrowing is also seen.

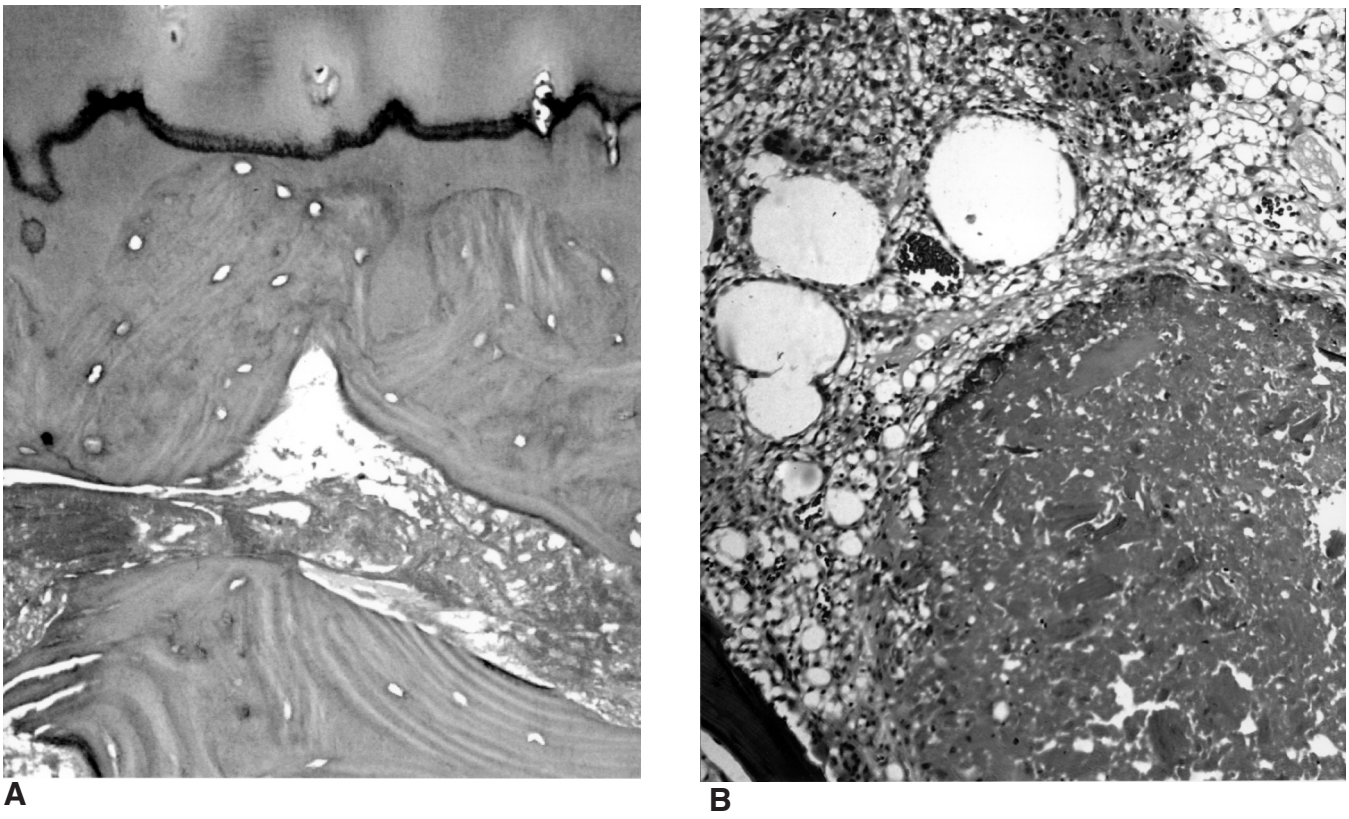

Figure 2. A. The bone attached to the overlying cartilage undergoes total necrosis of bone trabeculae and bone marrow tissue (H&E; original magnification ×200). B. Granulomatous lesions are observed in the marrow space of the remaining femoral head, where tiny fragments of bone are embedded in amorphous eosinophilic debris surrounded by epithelioid histiocytes and giant cells. No evidence of acute or chronic infection is noted (H&E; original magnification ×200).

Table 1. Summary of cases of osteonecrosis after intraarticular injection of corticosteroids.

Reference	Patient Age & Gender	Drug	Dose, mg × No. (total)	Injection Site	Period*, mo	ON	Histology	Outcome
6	42 F	Triamcinolone	40 × 2 (80)	Bilateral shoulder	6	Bilateral hips	Biopsy (ON)	NA
	78 F (RA**)	Triamcinolone	40 × 8 (740)***	Shoulder, knee, ankle	5	Bilateral hips & shoulders	Done (L FH:ON)	L THA
7	67 F	Triamcinolone	40 × 6 (408)****	Bilateral knee	13	Bilateral femoral condyles & tibial plateau	NA	NA
Present report	50 F	Methylprednisolone	80 × 1 (80)	L hip	3	L hip	Done (ON)	Rapid collapse & THA

* From the first corticosteroid injection until the radiographic confirmation of osteonecrosis. ** History of seronegative rheumatoid arthritis. *** Oral prednisone (420 mg) was also taken. **** Oral prednisone (168 mg) was also taken. No.: Number of injections; ON: osteonecrosis; L: left; THA: total hip arthroplasty; FH: femoral head; NA: not available.

DISCUSSION

Clinically, several morbid conditions have been reported to be associated with rapid joint breakdown, including articular chondrocalcinosis, apatite crystal deposition, neuropathy, infection, drug induced arthropathy, and a subset of rheumatoid arthritis[1-4]. In our case, none of these conditions was observed. Subchondral insufficiency fracture has been proposed as an etiology of rapid joint destruction[4]; however, we found no definite evidence of subchondral fracture, since the majority of the subchondral lesion had disappeared. Final histological diagnosis was rapidly destructive arthrosis with associated osteonecrosis of undetermined etiology. The development of osteonecrosis of the femoral head following multiple injection of corticosteroid into the joint has been reported in a few cases (Table 1)[6,7]; however, a relationship with a single injection of corticosteroids into the hip joint has not been described.

Many factors have been suggested as the cause of

osteonecrosis[9,10]. Our patient had no history of corticosteroid intake or alcohol abuse, and a blood coagulation examination including fibrinolysis and liver function tests were all normal. Since MRI showed no evidence of osteonecrosis in the hip at the time of intraarticular injection, osteonecrosis observed in this case might have occurred shortly after the steroid injection or might have developed as part of the pathophysiology of joint destruction. Hirota, *et al* reported a significant relationship between risk of the development of osteonecrosis and daily mean dose of corticosteroid (≥ 16.6 mg in terms of prednisolone) or the highest daily dose (≥ 80 mg in terms of prednisolone)[11]. A recent experimental study showed that the rate of osteonecrosis after the use of methylprednisolone was higher than that after use of triamcinolone[12]. The dose and type of corticosteroid used in this case (80 mg methylprednisolone) might have influenced the development of osteonecrosis.

Since the initial radiographs showed slight narrowing of the joint space, osteoarthritis might have been the predisposing factor for the longterm left groin pain. In addition, as a high-signal lesion was noted in the acetabulum, a bone marrow edema related to microfracture of the acetabulum as well as iliopsoas bursitis might have had some relation with the hip pain.

In this case, all we really know is that the patient, who had a long history of groin pain, developed rapid destruction of the femoral head. Although the precise etiology is unknown, the case appears to indicate that rapid destruction of the hip joint may have had some relationship with the intraarticular injection of corticosteroids.

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