Rapidly Destructive Arthropathy of the Hip Joint in Patients With Rheumatoid Arthritis

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abstract

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This article describes 3 patients with rheumatoid arthritis in which hip joint conditions were similar to rapidly destructive arthropathy of the hip joint. The patients had the following additional characteristics: (1) age at diagnosis was comparatively older; (2) rheumatoid arthritis had an onset after age 40 years; (3) severe rheumatoid arthritis was present in >4 major joints; (4) long-term steroids and disease-modifying antirheumatic drugs were taken due to the high activity of rheumatoid arthritis; and (5) patients were underweight, with body mass indexes <20 kg/m².

Rapidly destructive arthropathy of the hip joint rarely occurs in osteoarthritis, avascular necrosis of the femoral head, and rheumatoid arthritis. Clinically, rapidly destructive arthropathy of the hip joint occurs in elderly patients who report severe pain but have a relatively preserved range of motion. Typical radiologic changes in rapidly destructive arthropathy of the hip joint are rapid destruction, resorption, or subluxation of the femoral head, destruction of the acetabulum, and minimal spur formation developing 6 to 12 months after symptom onset. Destruction of the hip joint in rheumatoid arthritis occurs in the sequence of depression, flattening, and loss, and commonly progresses gradually. When patients with rheumatoid arthritis report persistent and severe pain in the hip joint with no specific cause, rapidly destructive arthropathy of the hip joint must be identified through repetitive follow-up radiographic observations.

Figure: Intraoperative photographs showing the yellowish and turbid hip fluid that looks like pus (arrow) (A) and destruction of the femoral head (B).

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First reported by Postel and Kerboull in 1970, rapidly destructive arthropathy of the hip joint rarely occurs in patients with osteoarthritis, avascular necrosis of the femoral head, or rheumatoid arthritis. To date, the pathogenesis of rapidly destructive arthropathy of the hip joint has not been clarified, but several sources suggest a causal relationship, such as direct toxicity by drugs, subchondral osteonecrosis and ischemia, and immunological mechanisms mediated by cytokines. Clinically, rapidly destructive arthropathy of the hip joint occurs in elderly patients who report severe pain but show a relatively preserved range of motion. Typical radiologic changes are rapid destruction, resorption, or subluxation of the femoral head; destruction of the acetabulum; loss of joint space; and minimal osteophyte formation that develops 6 to 12 months after clinical symptoms occur. Rapidly destructive arthropathy of the hip joint should be distinguished from septic arthritis or neuropathic arthritis.

Rheumatoid arthritis of the hip joint occurs in 5% to 15% of rheumatoid arthritis cases, and most joint destruction progresses gradually. This article describes 3 cases of rheumatoid arthritis in which rapidly destructive arthropathy of the hip joint was present.

CASE REPORTS

Patient 1

A 67-year-old woman (body mass index, 19.9 kg/m²) presented with pain in the left hip joint and a limping gait that had begun 6 months previously. She had an 18-year history of rheumatoid arthritis and had been prescribed 5 mg of prednisolone daily, 400 mg of hydroxychloroquine daily, and 5 mg of methotrexate weekly. She had rheumatoid arthritis in the bilateral shoulder, elbow, wrist, and knee joints and in the right hip joint. One year previously, she had reported no pain in the left hip joint, and radiographs demonstrated normal findings in the left hip joint (Figure 1A).

Current radiographs showed destruction of the femoral head and acetabulum, loss of joint space, and minimal osteophyte formation in the left hip joint (Figure 1B). Body temperature was within the normal range, but no severe tenderness or skin rash was observed in the left buttock. Passive range of motion in the left hip joint was 0° of extension, 95° of flexion, 15° of internal rotation, 20° of external rotation, 0° of adduction, and 10° of abduction. Harris Hip Score was 52 points. Erythrocyte sedimentation rate was 23 mm/h, C-reactive protein was 7.95 mg/dL, and rheumatoid arthritis factor was 50 IU/mL. Joint aspiration, microbiological smear, and culture test results were negative.

A cementless total hip arthroplasty was performed. Histopathological testing revealed chronic synovitis with a foreign body reaction in the soft tissues, and bone tissue examination revealed loss of articular cartilage, osteonecrosis, fibrosis, and fatty degeneration. At follow-up examinations up to 1 year postoperatively, Harris Hip Score improved to 89 points (Figure 1C).

Patient 2

A 67-year-old man (body mass index, 16.9 kg/m²) presented with pain in the left hip joint and a limping gait that had begun 6 months previously. He had a 20-year history of rheumatoid arthritis and had been prescribed 10 mg of prednisolone daily, 400 mg of hydroxychloroquine daily, and 7.5 mg of methotrexate weekly. He had rheumatoid arthritis in the bilateral wrist and knee joints and the left hip joint.

A radiograph taken at the authors’ institution 7 months previously had revealed overall joint space narrowing, a subchondral cyst, and osteoporosis in the left hip joint (Figure 2A). During the current examination, destruction of the femoral head and acetabulum, loss of joint space, and minimal osteophyte formation were observed (Figure 2B). Computed tomography showed destruction of the femoral head and acetabulum (Figure 2C), and magnetic resonance imaging showed synovial hypertrophy of the left hip joint and severe cartilage erosion (Figure 2D).

Figure 1: Anteroposterior radiograph of the right hip joint of a 67-year-old woman with an 18-year history of rheumatoid arthritis taken at the onset of joint pain and showing joint space narrowing in the joint and sclerosis of the femoral head in the right side (A). Anteroposterior radiograph taken 1 year later showing >50% destruction of the left femoral head (B). Anteroposterior radiograph of the left hip joint taken 1 year after total hip arthroplasty (C).
Body temperature was in the normal range. Minor tenderness was observed but no skin rash or edema was found in the left buttck. Muscle atrophy was observed, with the thigh circumference reduced by 30 mm in comparison with the right side. Passive range of motion of the left hip joint was 0° of extension, 90° of flexion, 30° of internal rotation, 30° of external rotation, 0° of adduction, and 20° of abduction. Harris Hip Score was 56 points. Erythrocyte sedimentation rate was 75 mm/h, C-reactive protein was 41.85 mg/dL, and rheumatoid arthritis factor was 47 IU/mL. Joint aspiration, microbiological smear, and culture test results were negative.

A hybrid total hip arthroplasty was performed. At follow-up examinations up to 1 year postoperatively, Harris Hip Score improved to 92 points (Figure 2E).

**Patient 3**

A 65-year-old man (body mass index, 19.6 kg/m²) presented with pain in the right hip and a limping gait that had begun 3 months previously. He had an 18-year history of rheumatoid arthritis and had been prescribed 2 mg of triamcinolone daily, 20 mg of leflunomide (a tumor necrosis factor-a inhibitor) daily, and 12.5 mg of methotrexate weekly. He had rheumatoid arthritis in the bilateral elbow and wrist joints and in the left hip, left knee, and right ankle joint.

Computed tomography revealed destruction of the right femoral head and acetabulum (Figure 3A), and magnetic resonance imaging revealed synovial hypertrophy of the right hip joint and severe cartilage erosion (Figure 3B). Body temperature was in the normal range. Minor tenderness was observed but no skin rash or edema was found in the right buttck. Passive range of motion of the right hip joint was 0° of extension, 90° of flexion, 10° of internal rotation, 60° of external rotation, 10° of adduction, and 40° of abduction. The Harris Hip Score was 49 points. Erythrocyte sedimentation rate was 63 mm/h, C-reactive protein was 8.22 mg/dL, and rheumatoid arthritis factor was 70.1 IU/mL. Preoperative arthrocentesis was performed, obtaining approximately 40 cc of the aspirates (Figure 3C). When analyzed, the aspirates were visually red with mucinous properties, and white and red blood cells were 1300/mm³ and 35000/mm³, respectively. Microbiological smear and culture test results were negative.

A hybrid total hip arthroplasty was performed. A yellowish, turbid, pus-like fluid gushed from the joint intraoperatively (Figure 3D) and was accompanied by severe destruction of the femoral head (Figure 3E). At follow-up examinations up to 1 year postoperatively, Harris Hip Score improved to 92 points (Figure 3F).

**DISCUSSION**

Destruction of the femoral head by rheumatoid arthritis occurs in the following sequence: depression, flattening, and loss. It commonly progresses gradually. However, in the current cases, destruction...
of the hip joint and resorption of the femoral head were found within a short, 1-year time frame, and such findings showed a pattern similar to rapidly destructive arthropathy of the hip joint.\textsuperscript{1-5} Among patients with osteoarthritis, those who are overweight or elderly or who have lateral diseases or insignificant formation of the osteophyte are at a high risk for rapidly destructive arthropathy of the hip joint to occur as a secondary disease.\textsuperscript{6} The current patients had the following characteristics, in addition to the clinical findings presenting as rapidly destructive arthropathy of the hip joint: (1) age at diagnosis was comparatively older; (2) rheumatoid arthritis had an onset after age 40 years; (3) severe rheumatoid arthritis was present in >4 major joints; (4) long-term steroids and disease-modifying antirheumatic drugs were taken due to the high activity of rheumatoid arthritis; and (5) patients were underweight (all body mass indexes were <20 kg/m\textsuperscript{2}).

Rapidly destructive arthropathy of the hip joint rarely occurs in patients with osteoarthritis,\textsuperscript{2,3} avascular necrosis of the femoral head,\textsuperscript{4} or rheumatoid arthritis.\textsuperscript{5} A differential diagnosis is needed to exclude septic arthritis and neuropathic arthritis. In the current cases, erythrocyte sedimentation rate and C-reactive protein were elevated in the general hematological assay, but the physical examinations showed no clinical findings, such as fever, tenderness, or flare on the lesions. Plain radiographs showed no bony destruction with unclear boundaries and secondary osteophyte formation, unlike septic arthritis, which usually presents with bone changes; magnetic resonance imaging revealed that the inflammatory lesion was limited to inside the joint, but no inflammatory findings were revealed in the bone marrow. In addition, suppurative inflammation was not found in the analysis of the aspirates from arthrocentesis. Therefore, it was possible to eliminate septic arthritis. Kim et al\textsuperscript{9} reported that fatty joint effusion in an osteonecrotic hip may look like pus and be erroneously interpreted as leukocytes in an automated cell count; a microscopic evaluation of the joint fluid smear should be performed. In the current cases, microscopic evaluation results from the joint fluid smear were negative.

Neuropathic arthritis does not have accompanying pain as one of its characteristics, but rather involves a history of syphilis, diabetes mellitus, syringomyelia, or spinal cord injury. Moreover, the locations where the disease occurs are determined by its cause, but the frequency in the hip joint is relatively low. The current

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\caption{Coronal computed tomography scan (A) and magnetic resonance image (B) of the right hip joint of a 65-year-old man with an 18-year history of rheumatoid arthritis showing >50\% destruction of the right femoral head. Hip joint aspiration was performed under fluoroscopic guidance (C). Intraoperative photographs showing the yellowish and turbid hip fluid (arrow) that looks like pus (D) and destruction of the femoral head (E). Anteroposterior radiograph of both hips taken 1 year after total hip arthroplasty (F).}
\end{figure}
patients had clinically excruciating pain but no neurological symptoms, making it possible to eliminate neuropathic arthritis. Rapidly destructive arthropathy of the hip joint is known to have a good prognosis without major complications, such as infection.\textsuperscript{1,3} In the current cases, the patients had good prognoses with no complications after total hip arthroplasty.

The etiology of rapidly destructive arthropathy of the hip joint is still undetermined, but authors have suggested various associative factors.\textsuperscript{1,5,6,10} Postel and Kerboull\textsuperscript{1} reported that direct drug toxicity caused rapidly destructive arthropathy of the hip joint, whereas Yoshino et al\textsuperscript{3} reported that long-term prescription steroid use in doses $>10,000$ mg was associated with rapidly destructive arthropathy of the hip joint in patients with rheumatoid arthritis. In the current cases, steroids and disease-modifying antirheumatic drugs were administered, but the exact total administration volume was unknown.

Mitrovic and Riera\textsuperscript{10} reported that subchondral osteonecrosis and ischemia play important roles in the onset of rapidly destructive arthropathy of the hip joint. In the current cases, the aforementioned findings were observed, but the causes for subchondral osteonecrosis and ischemia may vary; it was not possible to directly identify the cause of the rapidly destructive arthropathy of the hip joint among these cases. Komiya et al\textsuperscript{5} performed molecular and biological research on the causal factors of rapidly destructive arthropathy of the hip joint and reported that prostaglandin, interleukin-IB, and metalloproteinase-2 and -3 could facilitate the occurrence of rapidly destructive arthropathy of the hip joint. However, in the current cases, molecular and biological research could not be attempted.

Rapidly destructive arthropathy of the hip joint is an indication for total hip arthroplasty, but only exact differentiation from other diseases makes it possible to obtain a satisfactory result. In addition, to identify and understand the cause of the disease, further immunological study and analysis on the synovial fluid and synovial membrane may be required. Further study on the association with rheumatoid arthritis is required with more cases and longer follow-up.

The current cases of rapidly destructive arthropathy of the hip joint occurred in association with rheumatoid arthritis. When patients with rheumatoid arthritis report persistent and severe pain in the hip joint with no specific reason, rapidly destructive arthropathy of the hip joint should be monitored with repetitive follow-up radiographic observations.

**REFERENCES**