Early MRI and intraoperative findings in rapidly destructive osteoarthritis of the hip: A case report

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1. Introduction

Rapidly destructive osteoarthritis (OA) of the hip generally occurs in elderly women and causes severe disability [1]. In 1970, Lequesne [2] proposed a standardized definition of rapid chondrolysis: chondrolysis >2 mm in 1 year, or 50% joint-space narrowing in 1 year and no evidence of other forms of rapidly destructive arthropathy. In the majority of cases, the disease is unilateral, without evidence of antecedent OA, osteonecrosis, neuropathy, infection, or inflammatory disease [3–5]. The etiology of rapidly destructive hip OA has not been clarified since the first report of the disease by Postel and Kerboull [3]. Yamamoto and Bullough [6] demonstrated that subchondral insufficiency fracture of the femoral head occurs in elderly women with osteoporosis. They suggested that insufficiency fracture resulting from osteopenia might lead to rapid breakdown of the hip joint [7]. However, it is still unclear why insufficiency fracture occurs in the subchondral area of the femoral head. We report herein a case of rapidly destructive OA of the hip in which we observed (1) a pattern of edema in the bone marrow on magnetic resonance imaging (MRI) and (2) extensive inversion of the acetabular labrum during surgery.

2. Case report

A 77-year-old woman presented with a 5-month history of left hip pain. She had no history of antecedent trauma, and she reported that the pain occurred without any cause after she awoke one morning. Plain radiographs obtained at the first office visit demonstrated no abnormality (center-edge angle [8]: 27°; acetabular roof angle [9]: 5°). Using the method of Tannast et al. [10], we found that her pelvis tilted posteriorly on supine radiographs and that it tilted more posteriorly, about 10.8°, on standing. Mean bone mineral density measured by dual X-ray absorptiometry was 0.888 g/cm² in the affected acetabulum, 0.869 g/cm² in the affected femoral head, and 0.608 g/cm² in the affected femoral neck. Mean urinary excretion of the cross-linked N-telopeptide of type I collagen for bone resorption markers was 39.0 nMBCE/mM Cr (reference range for postmenopausal women, 14.9–89.0 nMBCE/mM Cr).

REFERENCES

[1] Lequesne M. Rapid chondrolysis: definition, pathophysiology, and therapy. Clin Orthop Relat Res 1970;72:210–22.
[2] Yamamoto J, Bullough PW. Insufficiency fracture as a cause of rapid chondrolysis in the hip. J Bone Joint Surg Am 1988;70-A(5):719–22.
[3] Tannast M, Linser M, Hildebrandt R, et al. Quantitative assessment of acetabular anteversion in postoperative computed tomography and pelvis radiography. Acta Orthop Scand 2010;81(3):291–5.
Fig. 1. (A) Anteroposterior radiograph of the left hip at 3 weeks after the onset of pain. (B) In this radiograph obtained at 4 months after the onset of pain, it is apparent that joint-space narrowing of the superolateral portion of the femoral head had progressed rapidly.

Cr). The patient was treated with anti-inflammatory drugs (celecoxib, 100 mg, twice daily). Pain was present at rest and persisted at night. Eventually she was unable to walk because the pain was severe. Her height was 146 cm and weight was 48 kg. Her body mass index, 22.5 kg/m², was within the normal range. The range of motion in her left hip was 120° in flexion, 10° in extension, 30° in abduction, 20° in adduction, 30° in external rotation, and 5° in internal rotation. Preoperative blood tests showed no evidence of rheumatoid factor (including anticyclic citrullinated peptide antibody) or infection, and we found no evidence of Charcot joint or inflammatory arthropathies. Anteroposterior radiographs showed that joint-space narrowing of the superolateral portion of the femoral head progressed rapidly for only 4 months (Fig. 1).

In reporting in detail about MRI findings in rapidly destructive OA of the hip, Boutry et al. [11] and Sugano et al. [12] described its key features: joint effusion and bone marrow with an edema-like pattern in the femoral head and neck or acetabulum or both. MRI findings of this case obtained at 4 months after the first office visit were consistent with those in the reports by Boutry et al. and Sugano et al. Besides, we observed a focal low-intensity band on T₁-weighted images [6,13–15] (Fig. 2). However, neither tomosynthesis nor multiplanar reconstruction of computed tomography images showed any subchondral insufficiency fractures in

Fig. 2. Magnetic resonance images obtained 4 months after the onset of hip pain show joint effusion and a pattern of bone-marrow edema from the upper portion of the femoral head to the intertrochanteric region, with diffuse low intensity on a T₁-weighted image (A) and high intensity on the short τ inversion recovery (STIR) sequence (B). In addition, a small low-intensity band paralleling the articular surface in the femoral head is apparent on the T₁-weighted image that indicates subchondral insufficiency fracture (white arrow).

Fig. 3. Tomosynthesis (A) and multiplanar reconstruction of computed tomography (B) evaluation for subchondral fracture were performed. No obvious subchondral fracture was found in either image.
any slices (Fig. 3). Nonspecific accumulation in the left hip was apparent on bone scintigraphy. On the basis of both clinical and radiological findings, our diagnosis was rapidly destructive hip OA [3–5,16]. Because of her severe pain, the patient underwent total hip arthroplasty. During surgery, we found that the anterosuperior surface of the acetabular cartilage was covered extensively with the inverted labrum (Fig. 4A). Also, the location of the inverted labrum was mostly consistent with the bone-marrow edema lesions in the femoral head and the acetabulum. Proliferation of the synovial tissue was not remarkable. However, a macroscopic defect and degeneration of the anterolateral portion of cartilage on the acetabulum and femoral head were obvious (Fig. 4B and C). The resected labrum included lots of fragments of articular cartilage (Fig. 4D). There were no crystal deposits, such as pyrophosphate crystals or uric acid crystals, in either the pathology specimens or the synovial fluid. There was not extensive subchondral necrosis with a deep wedge-shaped lesion as seen in primary osteonecrosis, including that caused by corticosteroid intake or alcohol abuse [17]. In addition, we could not find extensive subchondral fracture, which would have indicated insufficiency fracture. We aspirated bone-marrow blood from the femoral neck intraoperatively. We looked for the presence of MMP-2, MMP-3, and MMP-9 in peripheral blood and bone-marrow blood because activation of those proteases has been suggested [18,19] as an indicator of rapidly destructive OA. Although the levels of all three proteases were within normal limits (MMP-2: 694 ng/mL; MMP-3: 25.5 ng/mL; MMP-9: 20 ng/mL) in peripheral blood obtained 1 day before the surgery, the level of MMP-9 was extremely high in bone-marrow blood (1000 ng/mL).

Fig. 4. (A) The labrum at the anterosuperior portion (arrowheads) was obviously inverted and widely covered the lunate surface of the acetabulum. The asterisk indicates the fossa acetabuli. ANT = anterior; POST = posterior. (B) Intraoperative photograph showing a defect of the acetabular cartilage just under the inverted labrum after resection of the labrum. (C) Intraoperative photograph showing an anterosuperior cartilage defect of the femoral head. (D) Photomicrograph obtained from the inverted labrum included lots of fragments of articular cartilage.

Fig. 5. (A) A hemipelvis with a tilt within the normal range. (B) A hemipelvis with posterior tilting. The distance between the free margin of the acetabular labrum to the femoral head–neck junction (arrows) is longer than that in a normal pelvis (i.e., distance B > distance A). (C) The free margin of the acetabular labrum is more likely to be trapped by the femoral head into the intra-articular space in a posteriorly tilted pelvis than in a normal pelvis because in the tilted pelvis, the distance where the free margin of the acetabular labrum runs across the surface of the femoral head is longer when the hip joint bends. (D) Abnormal stress caused by inversion of the acetabular labrum to the superolateral portion of the femoral head and lateral part of the acetabulum.
3. Discussion

One convincing cause that has been proposed for rapidly destructive hip OA is subchondral insufficiency fracture of the femoral head [6,7,20,21]. In the case reported here, we could not find an obvious subchondral insufficiency fracture pathologically. However, MRI showed a focal low-intensity band paralleling the articular surface in the femoral head in T1-weighted image and a pattern of bone-marrow edema not only in the femoral head but also in the lateral side of the acetabulum. We speculate that the subchondral fracture was so small, as seen in Fig. 2A, that we could not detect it in our pathology specimens. Meanwhile, as Boutry et al. [11] noted, if the presence of a subchondral fracture can explain joint destruction, then the mechanism of rapid chondrolysis in rapidly destructive hip OA remains uncertain. Yamamoto and Bullough [7] suggested that fragments of the articular cartilage, with or without attached subchondral bone tissue, are frequently observed in the marrow space. This would seem to indicate that subchondral fracture occurs prior to the loss of articular cartilage. We assume that their theory is based on the fact that if articular cartilage disappeared because of chondrolysis prior to the development of a subchondral insufficiency fracture, then fragments of articular cartilage, with or without attached subchondral bone tissue, should not be in bone in the marrow space. However, in our patient, we found no fragments of articular cartilage or subchondral bone in the marrow space before collapse of the femoral head. Thus, another factor may be involved in rapid joint-space narrowing, which is an initial symptom of rapidly destructive hip OA. Before femoral-head collapse in our patient, the inverted labrum contained various sizes of cartilage fragments, but the femoral-head marrow space did not. This would seem to suggest that inversion of the labrum is involved in joint-space narrowing. If a patient with fragile articular cartilage also has an inverted labrum, the articular cartilage might be easily peeled. Solomon et al. [22,23] proposed that if cartilage degeneration is slow and bone response is good, reparative sclerosis and osteophyte formation will result in joint stability and hypertrophic OA. Conversely, if cartilage degeneration is rapid and bone response is poor, as may be seen in elderly patients, atrophic or destructive OA will occur. Similarly, we speculate that although degenerative cartilage will prevail over the labrum even if the labrum inverts into the articular surface in young patients, an inverted labrum will prevail over degenerative cartilage in elderly patients, resulting in detritic synovitis with large fragments of articular cartilage. It has already been reported that an inverted acetabular labrum causes hip OA [24,25]. Whether joint-space narrowing is rapid or not may be related to the quality of articular cartilage. In this case, we found the acetabular labrum to be inverted; however, the inversion mechanism is unclear. Watanabe et al. reported that posterior pelvic tilt may be one of the factors that contribute to the development of rapidly destructive hip OA [21]. We found that our patient had posterior pelvic tilt as well. We propose a mechanism of inversion: in posterior tilting, the distance between the free margin of the acetabular labrum to the femoral head–neck junction is longer than in a normal pelvis. That potentially increases the chance that the free margin of the acetabular labrum is sucked into the joint space when persons with tilted pelvises bend at the hip (Fig. 5A–C). Posterior pelvic tilt in rapidly destructive hip OA may involve two factors: an inversion of the acetabular labrum and an increase in contact force through the inverted labrum. As shown in Fig. 5D, articular cartilage at the site of labral inversion receives most of the stress. The concentration of stress on the subchondral bone may lead to a fracture in the femoral head andacetabulum beneath the cartilage. In fact, the relationship between the inverted labrum and subchondral insufficiency fracture of the femoral head has recently been demonstrated [26]. In this case, a bone-marrow edema pattern was observed on MRI, not only in the femoral head but also in the lateral side of the acetabulum. Besides, intraoperative findings showed an extensively inverted labrum lying between the intensity changes on magnetic resonance images.

Conflict of interest

None.

Funding

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Ethical approval

We report about a single case that did not require ethical approval. The manuscript is not a clinical study.

Author contributions

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version for publication. Dr. Fukui had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study conception and design: Fukui. Acquisition of data: Fukui and Fukushima. Analysis and interpretation of data: Fukui, Kaneuji, Matsumoto.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Key learning points

- Magnetic resonance imagings from the present case, which involved rapidly destructive hip osteoarthritis, showed a bone-marrow edema pattern not only in the femoral head but also in the acetabulum.
- The concentration of stress on the subchondral bone due to inversion of the acetabular labrum may lead to fracture of the femoral head and acetabulum beneath the cartilage.
- Inversion of the acetabular labrum may be a mechanism of rapidly destructive hip osteoarthritis.

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References

[1] G.W. Bock, A. Garcia, M.H. Weisman, P.A. Major, D. Lyttle, P. Haghighi, et al., Rapidly destructive hip disease: clinical and imaging abnormalities, Radiology 186 (1993) 461–466.
[2] M. Lequesne, Rapid destructive coxarthritis, Rhumatologie 2 (1970) 51–63 (In French).
[3] M. Postel, M. Kerboull, Total prosthetic replacement in rapidly destructive arthritis of the hip joint, Clin. Orthopaedics Relat. Res. 72 (1970) 138–144.
[4] Z.S. Rosenberg, S. Shankman, G.C. Steiner, D.K. Kastenbaum, A. Norman, M.G. Lazansky, Rapidly destructive osteoarthritis: clinical, radiographic, and pathologic features, Radiology 182 (1992) 213–216.
[5] L.R. Irwin, J.A. Roberts, Rapidly progressive osteoarthrosis of the hip. J. Arthroplasty 13 (1998) 642–646.
[6] T. Yamamoto, P.G. Bullough, Subchondral insufficiency fracture of the femoral head: a differential diagnosis in acute onset of coxarthrosis in the elderly, Arthritis Rheumatol. 42 (1999) 2719–2723.
[7] T. Yamamoto, P.G. Bullough, The role of subchondral insufficiency fracture in rapid destruction of the hip joint, Arthritis Rheumatol. 43 (2000) 2423–2427.

[8] G. Wiberg, Studies on dysplastic acetabulum and congenital subluxation of the hip joint with special reference to the complications of osteoarthrosis, Acta Chirurgica Scand. 83 (1939) 29–38.

[9] W.K. Massie, M.B. Howorth, Congenital dislocation of the hip, J. Bone Joint Surg. Am. 32 (1950) 519–531.

[10] M. Tannast, S.B. Murphy, F. Langlotz, S.E. Anderson, K.A. Siebenrock, Estimation of pelvic tilt on anteroposterior X-rays — a comparison of six parameters, Skeletal Radiol. 35 (2006) 149–155.

[11] N. Boutey, C. Paul, X. Leroy, D. Fredoux, H. Migaud, A. Cotten, Rapidly destructive osteoarthritides of the hip: MR imaging findings, AJR Am. J. Roentgenol. 179 (2002) 657–663.

[12] N. Sugano, K. Ohzono, T. Nishii, T. Sakai, K. Haraguchi, H. Yoshikawa, et al., Early MRI findings of rapidly destructive coxopathy, Magn. Reson. Imaging 19 (2001) 47–50.

[13] T. Yamamoto, Subchondral insufficiency fractures of the femoral head, Clin. Orthop. Surg. 4 (2012) 173–180.

[14] S. Ikenura, T. Yamamoto, G. Motomura, Y. Nakashima, T. Mawatari, Y. Iwamoto, MRI evaluation of collapsed femoral heads in patients 60 years old or older: differentiation of subchondral insufficiency fracture from osteonecrosis of the femoral head, AJR Am. J. Roentgenol. 195 (2010) W63–W68.

[15] T. Yamamoto, Y. Iwamoto, R. Schneider, P.G. Bullough, Histopathological prevalence of subchondral insufficiency fracture of the femoral head, Ann. Rheumatic Dis. 67 (2008) 150–153.

[16] C.J. Menikes, F. Simon, F. Delrieu, M. Forest, F. Delbarre, Destructive arthropathy in chondrocalcinosis articularis, Arthritis Rheumatic 19 (1976) 329–348.

[17] T. Yamamoto, T. Yamauchi, K.B. Lee, P.G. Bullough, A clinicopathologic study of osteonecrosis in the osteoarthritic hip, Osteoarthritis Cartilage 8 (2000) 303–308.

[18] S. Komiya, A. Inoue, Y. Sasaguri, K. Minamitani, M. Morimitsu, Rapidly destructive arthropathy of the hip: studies on bone resorptive factors in joint fluid with a theory of pathogenesis, Clin. Orthop. Relat. Res. 284 (1992) 273–282.

[19] K. Ogawa, M. Mawatari, M. Komine, M. Shigematsu, M. Kitajima, A. Kukita, et al., Mature and activated osteoclasts exist in the synovium of rapidly destructive coxarthrosis, J. Bone Miner. Metab. 25 (2007) 354–360.

[20] T. Yamamoto, K. Takabatake, Y. Iwamoto, Subchondral insufficiency fracture of the femoral head resulting in rapid destruction of the hip joint: a sequential radiographic study, AJR Am. J. Roentgenol. 178 (2002) 435–437.

[21] W. Watanabe, E. Itoi, S. Yamada, Early MRI findings of rapidly destructive coxarthrosis, Skeletal Radiol. 31 (2002) 35–38.

[22] L. Solomon, C.M. Schnitzerl, J.P. Browett, Osteoarthritis of the hip: the patient behind the disease, Ann. Rheumatic Dis. 41 (1982) 118–125.

[23] L. Solomon, C.M. Schnitzler, Pathogenic types of coxarthrosis and implications for treatment, Arch. Orthopaedic Trauma Surg. 101 (1983) 259–261.

[24] W.H. Harris, R.B. Bourne, I. Oh, Intra-articular acetabular labrum: a possible etiological factor in certain cases of osteoarthrosis of the hip, J. Bone Joint Surg. Am. 61 (1979) 510–514.

[25] J.W. Byrd, K.S. Jones, Osteoarthritis caused by an inverted acetabular labrum: radiographic diagnosis and arthroscopic treatment, Arthroscopy 18 (2002) 741–747.

[26] K. Fukui, A. Kaneuji, M. Fukushima, T. Matsumoto, Inversion of the acetabular labrum triggers rapidly destructive osteoarthrosis of the hip: representative case report and proposed etiology, J. Arthroplasty 29 (2014) 2468–2472.