

Evaluation of cartilage matrix disorders by T2 relaxation time in patients with hip dysplasia

T. Nishii M.D.†*, H. Tanaka M.D.‡, N. Sugano M.D.†, T. Sakai M.D.†,
T. Hananouchi M.D.† and H. Yoshikawa M.D.†

† Department of Orthopaedic Surgery, Osaka University Medical School E3, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan

‡ Department of Radiology, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan

Summary

Objective: Early detection of cartilage disorder in dysplastic hips is important in predicting subsequent progression of osteoarthritis and determining the appropriate timing of osteotomy surgery. We assessed the feasibility of T2 assessment using magnetic resonance (MR) imaging at 3 T for evaluating early changes in the acetabular and femoral cartilages for patients with hip dysplasia.

Methods: Sagittal T2 maps of the hip were obtained using 3 T MR imaging in 10 normal volunteers (14 hips) and in 23 patients (26 hips) with hip dysplasia at pre-arthritis stage (without osteoarthritis) or early-arthritis stage (with osteoarthritis at the Kellgren–Lawrence system of grade 1 or 2). T2 values and the visual appearance of T2 mapping, including gradient T2, low T2, and high T2 patterns, were compared at the superior zones of the acetabular and femoral cartilages among the normal, pre-arthritis, and early-arthritis groups.

Results: There were no significant differences in T2 values for both cartilages among the three groups. Regarding the visual appearance of T2 mapping for the acetabular cartilage, all hips in the normal group showed a gradient T2 pattern, while the pre-arthritis groups included six hips (43%) with a low T2 pattern, and the early-arthritis group showed either a low T2 pattern (33%) or a high T2 pattern (67%). The frequency of the gradient T2 pattern was significantly lower for dysplastic hips than for normal hips, in the acetabular and femoral cartilages ($P < 0.05$).

Conclusions: This preliminary study demonstrated the clinical feasibility of T2 assessment of hip cartilage using 3 T MR imaging. T2 mapping classification may enable the early detection of osteoarthritic degeneration and the detection of developmental disorders of cartilage matrix in patients with hip dysplasia.

© 2007 Osteoarthritis Research Society International. Published by Elsevier Ltd. All rights reserved.

Key words: Hip dysplasia, MR imaging, Cartilage, T2 relaxation time, Osteoarthritis, Cartilage matrix.

Introduction

Hip dysplasia is one of the major causes of hip osteoarthritis^{1,2}. Radiological evidence of dysplasia in hips without osteoarthritis is shown as a risk factor for the development of hip osteoarthritis in a prospective study³. Hips with moderate or severe degrees of dysplasia are likely to deteriorate progressively and eventually develop into terminal osteoarthritis with persistent symptoms and severely impaired function⁴. When effective surgical treatment such as osteotomy surgery is applied before osteoarthritic changes progress, reliable outcomes can be expected, with the prevention of osteoarthritic changes; however, delay of treatment following osteoarthritic involvement often results in unsatisfactory outcomes⁵. Early detection of cartilage disorders in dysplastic hips is important in predicting the subsequent progression of osteoarthritis and determining appropriate timing for osteotomy surgery.

Several imaging modalities are currently available to evaluate osteoarthritis of the hip, including plain radiography, arthrography, bone scintigraphy⁶, computed tomography (CT) arthrography⁷, and magnetic resonance (MR) imaging with and without arthrographic effect^{8,9}. Plain radiography is widely used for diagnosis and assessment of the severity of joint osteoarthritis, and showed significant correlation with hip cartilage thickness and volume¹⁰; however, several other reports have proposed inaccurate relationships between radiographic findings and the status of the articular cartilage^{11,12}. Recent investigations using CT arthrography⁷ and MR imaging with and without arthrographic effect^{7–9} achieved excellent visualization and sensitive detection of morphological changes in hip cartilage (thinning or defect); however, the diagnostic abilities of these modalities are limited for early cartilage disorders without change of cartilage thickness or volume, such as softening and surface fibrillation⁷. Disruption or alteration of the cartilage matrix such as a decrease in the concentration of proteoglycan and an increase in water content is found histologically in early changes of osteoarthritis¹³. It may be more effective to image cartilage matrix disorders or water content than to image cartilage morphological changes such as thickness and shape in detecting early changes of cartilage disorders with high sensitivity and accuracy.

*Address correspondence and reprint requests to: Takashi Nishii, M.D., Department of Orthopaedic Surgery, Osaka University Medical School E3, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. Tel: 81-6-6879-3552; Fax: 81-6-6879-3559; E-mail: nishii@ort.med.osaka-u.ac.jp

Received 24 December 2006; revision accepted 5 June 2007.

MR imaging techniques that have been proposed for sensitive evaluation of cartilage matrix changes include T2 relaxation time (T2), delayed gadolinium-enhanced MR imaging of cartilage (dGEMRIC), and T1 in the rotating frame (T1rho)¹⁴. Evaluation of T2 of the articular cartilage shows great potential for the quantitative assessment of collagen and water content^{15,16} and indicates clinical usefulness for knee imaging *in vivo*^{17,18}; however, to the best of our knowledge there have been no reports that assess hip cartilage by T2. This is because of difficulties involved in obtaining satisfactory image quality and in differentiating between the acetabular and femoral cartilages.

MR imaging at higher magnetic field strength (at 3 T or more) may provide improved image quality of the hip cartilage due to superior signal-to-noise contrast¹⁹. The objective of the present study is to assess the feasibility of T2 evaluation using MR imaging at 3 T in detecting early changes in the acetabular and femoral cartilages in patients with hip dysplasia.

Materials and methods

Ten normal volunteers (14 hips) and 23 patients with hip dysplasia (26 hips) were included in this study. Because patients with hip dysplasia are predominantly female²⁰, men were excluded from the study to prevent the potentially confounding influence of sex difference on T2 in the articular cartilage²¹. Exclusion criteria for volunteers were present and/or past experience of hip pain, stiffness, or gait disability. Hip dysplasia was defined by center-edge angle of Wiberg of 24° or less²² on anteroposterior radiographs. Inclusion criteria for dysplastic hips to the study were as follows: no previous hip surgery, Class I subluxation (less than 50%) according to the classification of Crowe *et al.*²³, and radiological osteoarthritis classification according to the Kellgren-Lawrence system²⁴ of grade 0 (no osteoarthritic finding), grade 1 (possible narrowing of joint space and/or osteophytes), or grade 2 (definite narrowing of joint space, definite osteophytes, and slight sclerosis). In the present study, osteoarthritis classification of grade 0 with radiological evidence of hip dysplasia was categorized as pre-arthritis stage, and grade 1 or 2 as early-arthritis stage. Institutional review board approval was obtained for this study, and all patients provided informed consent after the nature of the procedure had been fully explained.

The average ages of the volunteers and patients were 34 years (range, 23–51 years) and 40 years (range, 22–69 years), respectively. The average heights and weights were 163 cm (range, 153–171 cm) and 54 kg (range, 47–80 kg) in the volunteers, and 157 cm (range, 149–163 cm) and 53 kg (range, 42–80 kg) in the patients, respectively. The center-edge angle of the patients ranged from -20° to 24° (mean, 6.5°); there were 14 hips at the pre-arthritis stage (grade 0) and 12 hips at the early-arthritis stage (eight hips at grade 1 and four hips at grade 2). Patients had no pain in six hips and slight or moderate pain either while walking or after a long walk in 20 hips. The six asymptomatic hips were diagnosed as hip dysplasia during examination of the opposite symptomatic hips.

MR imaging of the hip was performed on a Signa 3 T MR scanner (GE Healthcare, WI, USA) using a flexible surface coil. The volunteers and patients were positioned supine with the hip in neutral position. Two-dimensional dual-echo spin-echo images were obtained with the following parameters: repetition time/echo time 1500 ms/10 and 45 ms; field of view 16 cm; matrix 512 × 256 interpolated to

512 × 512 with a resulting in-plane pixel resolution of 312.5 μm; 5 mm slice thickness, and two signals acquired for a total time of 13.5 min. Frequency encoding was head to foot across the hip joint, and the fat-suppression technique was used to minimize chemical shift artifact at the bone/cartilage interface. A single sagittal image passing through the center of the femoral head was obtained. When the imaging plane was lateral to the outer edge of the acetabular rim in the coronal scout view, the imaging plane was moved medially to be located within the acetabular rim. The sagittal plane was employed because cartilage disorder is often observed at the anterosuperior region of the acetabulum in arthroscopic studies of dysplastic hips²⁵. A single slice sequence was used to prevent inaccuracy of T2 measurement caused by magnetization transfer contrast from off-resonance radiofrequency irradiation found in multi-slice sequences^{26,27}.

The acetabular and femoral cartilages were manually segmented on the mid-sagittal image and the T2 value was calculated assuming a single exponential decay component. A color-coded T2 map of the cartilage was overlaid on the mid-sagittal image; low T2 values were represented in red while high T2 values were colored green or blue (Fig. 1). Regions of interest (ROIs) in the acetabular and femoral cartilages were defined at the weight-bearing area of the superior 20° range of the cartilage, from the cartilage surface to the basal area, and the average T2 value and visual appearance of T2 mapping within the ROIs were evaluated. The visual appearances of T2 mapping were classified into three patterns (Fig. 2): "gradient T2 pattern" for low T2 values at the deep cartilage area and high T2 values at the superficial cartilage area, which was considered representative of the spatial variation of normal knee cartilage²⁸; "low T2 pattern" for ROIs occupied predominantly by low T2 values up to the superficial cartilage area; and "high T2 pattern" for ROIs occupied predominantly by high T2 values even at the deep cartilage area. On assessment for each case, representative cases of the three mapping patterns (Fig. 2) were used as the reference atlas. Definitions of the ROIs were repeated three times by a single observer (TS) without knowledge of presence of hip dysplasia or the radiological osteoarthritis classification, and the T2 values of the ROIs were averaged. Inter-observer reliability between two observers (TN, TS) was assessed in the first 10 subjects, with a coefficient of variation of 2.5% for the acetabular ROI and 3.8% for the femoral ROI. Visual appearance of T2 mapping was interpreted blindly by two observers (TN, TS) independently without knowledge of presence of hip dysplasia or the radiological osteoarthritis classification. In general (95% of the cases in the acetabular cartilage and 93% of the cases in the femoral cartilage), there was agreement between the two observers. In the remaining cases, a consensus of opinion was obtained between the two observers. All imaging analysis was conducted using Beth Israel Deaconess Medical Center software for functional imaging of cartilage (Boston, MA, USA).

Clinical symptoms of the hip were evaluated using the Western Ontario and McMaster Universities Osteoarthritis (WOMAC)²⁹ pain score at the time that MR imaging was conducted. When both hips were examined, WOMAC questionnaires were taken separately for the right and left hips. The WOMAC pain score was calculated as a summation of the scores ranging from 0 (no pain) to 4 (extreme pain) in response to each of five items (range of possible total score 0–20). T2 value and the visual appearance of T2 mapping for each ROI were compared among the normal,

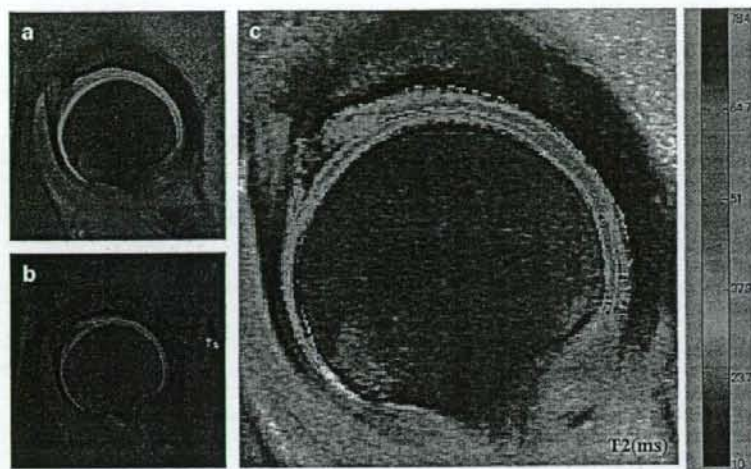


Fig. 1. Representative mid-sagittal MR images of a normal volunteer hip (a: 1500/10 ms, b: 1500/45 ms), and corresponding T2 map overlaid on the cartilage region (c). Superior and anterior directions of the hip are toward the top and left of each image, respectively.

pre-arthritic, and early-arthritic hips, using analysis of variance and the Fisher exact test. They were also compared between asymptomatic and symptomatic hips, using the nonparametric Mann-Whitney *U* test and the Fisher exact test. Between normal hips and dysplastic hips, and between asymptomatic hips and symptomatic hips, we calculated a sample size to detect a 10% difference of T2 value based

on a previous report comparing T2 value in healthy knees and osteoarthritic knees¹⁶. Fourteen hips or more in each group were sufficient to determine whether there was a significant difference (power > 0.8, $P < 0.05$). The relationship between WOMAC pain scores and T2 values was evaluated using the Spearman correlation coefficient. A *P* value of less than 0.05 indicated significance.

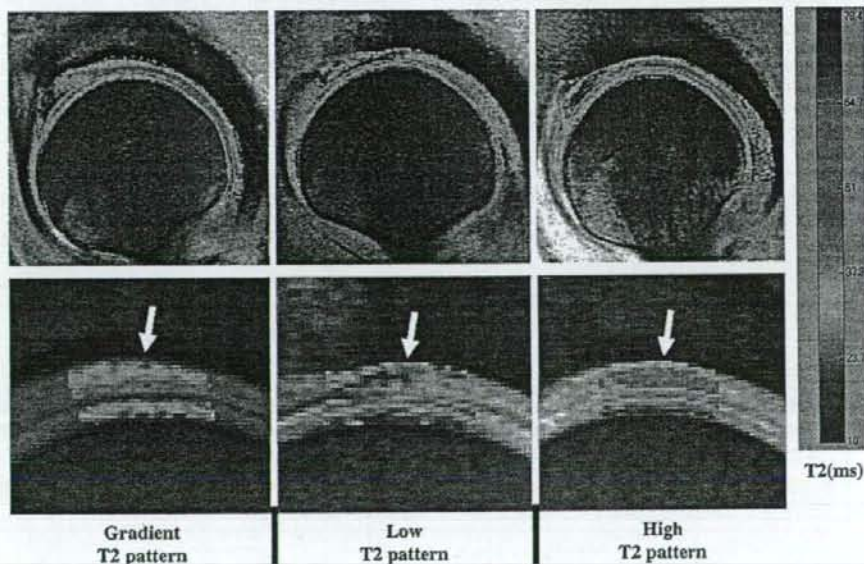


Fig. 2. Representative cases for the three patterns in visual appearance of T2 mapping (upper image) and their magnified images of the weight-bearing area with segmentation of the ROIs in the acetabular and femoral cartilages (lower image). Note T2 distribution within the ROIs of the acetabular cartilage (arrows). Gradient T2 pattern shows low T2 at the deep cartilage area and high T2 values at the superficial cartilage area; low T2 pattern shows predominantly low T2; and high T2 pattern shows predominantly high T2.

Results

The average ages of the normal, pre-arthritis, and early-arthritis groups were 34 years (range, 23–51), 35 years (range, 22–50), and 45 years (range, 23–69), respectively. The mean height/weight of the three groups were 163 cm/54 kg, 156 cm/50 kg, and 157 cm/55 kg, respectively; there was no statistical difference of age and weight among the three groups, however, height of the normal group was significantly higher than the other two groups ($P < 0.05$). All hips in the normal group, four hips in the pre-arthritis group, and two hips in the early-arthritis group showed no pain, while the other 10 hips in the pre-arthritis group and 10 hips in the early-arthritis group showed slight or relatively mild pain with WOMAC pain scores ranging from 1 to 12 points.

There was no significant difference in T2 value at the defined superior ROI of the cartilage among the normal, pre-arthritis, and early-arthritis groups, both for the acetabular and femoral sides. On the acetabular cartilage ROI, the mean T2 values ± 1 standard deviation for the normal, pre-arthritis, and early-arthritis groups were $33.4 \text{ ms} \pm 4.5$, $32.0 \text{ ms} \pm 3.9$, and $37.1 \text{ ms} \pm 12.0$, respectively. On the femoral cartilage ROI, these values were $29.4 \text{ ms} \pm 3.0$, $29.0 \text{ ms} \pm 4.4$, and $28.0 \text{ ms} \pm 3.7$, respectively.

The visual appearance of T2 mapping for the acetabular ROI showed a different distribution of the three patterns among the normal, pre-arthritis, and early-arthritis groups (Fig. 3). On the acetabular ROI, all hips in the normal group demonstrated gradient T2 pattern, while six hips in the pre-arthritis group (43%) and four hips in the early-arthritis group (33%) demonstrated low T2 pattern. The remaining eight hips in the early group (67%) demonstrated high T2 pattern. Consequently, the frequency of the gradient pattern was significantly different between the normal (100%) and pre-arthritis/early-arthritis groups (31%) ($P < 0.0001$). On the femoral ROI, the frequency of the gradient pattern was also significantly different between the normal (93%) and pre-arthritis/early-arthritis groups (50%) ($P < 0.05$).

Comparing the 20 asymptomatic hips and 20 symptomatic hips, frequency of gradient pattern in the acetabular/femoral cartilages was significantly lower in the symptomatic hips (35%/40%) than the asymptomatic hips (75%/90%) ($P < 0.05$). However, there was no significant difference in T2 value between the asymptomatic and symptomatic hips, and there was no significant correlation between WOMAC pain scores and T2 values at the superior ROI of the cartilage.

Discussion

Degeneration of the articular cartilage in osteoarthritis is associated with concomitant changes in the extracellular matrix components that include disruption of collagenous architecture, depletion of proteoglycan, or an increase/decrease in water content, even at very early stages of the disease^{30,31}. There is a high expectation, based on numerous experimental and clinical studies, that assessment of T2 of the cartilage will become a potent surrogate of cartilage matrix changes such as these, as well as the associated loss of biomechanical function. Nieminen *et al.* observed an increase of T2 in the superficial zone of bovine cartilage following degradation of collagenous architecture by enzymatic treatment³². Lüsse *et al.* demonstrated a close correlation between the water content within the cartilage and T2 relaxation rates for human cartilage removed from the knee joint; the authors stated that the water content could be accurately estimated from the correlation of T2³³. Wayne *et al.* showed significant inverse correlations of T2 with proteoglycan content or cartilage stiffness, using porcine patella cartilage with depletion of proteoglycan matrix following enzymatic treatment¹⁵. For *in vivo* imaging of the knee joint, an increase in T2 was associated with aging^{17,34} and the involvement of osteoarthritis¹⁸, while a decrease in T2 was associated with the stress of running³⁵ and also with mechanical loading of the knee during MR imaging³⁶. Other than the knee joint, T2 assessment *in vivo* has also been performed for interphalangeal joint cartilage³⁷; however, to the best of our knowledge, T2 assessment of the hip joint has yet to be conducted.

Almost all hips of normal volunteers in the present study showed the gradient pattern of T2 mapping at the superior portion of the acetabular and femoral cartilages. This spatial variation, with T2 values increasing from the cartilage base toward the articular surface, is consistent with previous reports of normal knee cartilage T2 values *in vivo*^{17,28}. This T2 distribution was accounted for histologically by the physiological spatial distribution of water, collagen and proteoglycan, and by spatial differences in collagenous architecture¹⁷. High T2 values in the limited distance from the bone/cartilage interface were described in a detailed quantitative analysis of T2 variation of normal knee cartilage^{17,28}, but were not seen in the hip cartilages of the present study. T2 variations in these earlier studies of the knee were partly explained by chemical shift artifact and volume averaging artifact at the bone/cartilage interface^{17,28}. Average T2 values of the acetabular and femoral cartilages in

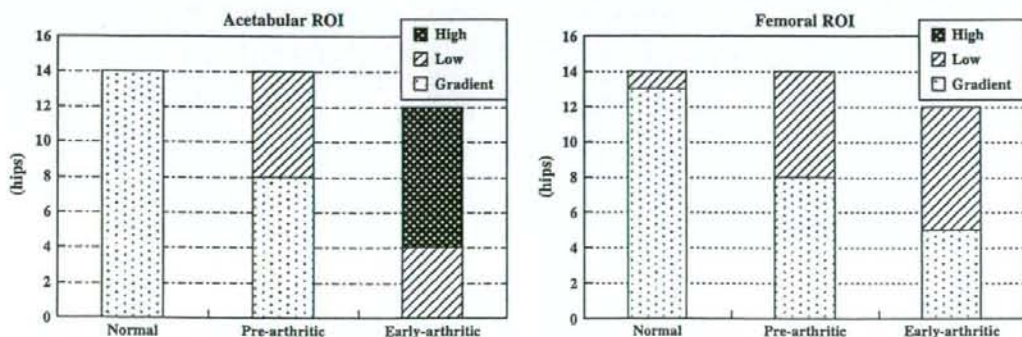


Fig. 3. Distribution of gradient T2 pattern, low T2 pattern and high T2 pattern in the normal, pre-arthritis, and early-arthritis groups, for the acetabular ROIs and femoral ROIs.

the healthy female volunteers (29–33 ms) of the present study were relatively low compared with those observed in the knee cartilages of healthy women of a similar age (40–60 ms) using 3 T MR imaging³⁴. A previous cadaveric study revealed that mean cartilage thickness of the hip joint is significantly thinner than the thickness of cartilage in the knee joint, and that thinner cartilage is correlated with a higher compressive stiffness of the cartilage³⁵. Considering that compressive stiffness of the cartilage is significantly related to water content or proteoglycan content^{39,40}, the difference in average T2 values between our study and a previous knee study may partly reflect the physiological differences of those matrix components at the two sites.

The favorable results of the present study might partly result from the superior hardware capability of 3 T MR imaging. MR imaging of the hip cartilage has been difficult at conventional magnetic field strengths (≤ 1.5 T) because of the relatively thin structure of the acetabular or femoral cartilages, their location deep inside the body, and the close contact between the acetabular and femoral cartilages⁴¹. MR imaging with high magnetic field strength improves image quality and precision because of the superior signal-to-noise ratio and spatial resolution^{19,42–44}. If the cartilage thickness of the hip joints is generally assumed to range from 1 to 3 mm^{38,45}, the MR images at 3 T in the present study contain approximately 3–10 pixels across each of the acetabular and femoral cartilages. We consider that this high-resolution imaging with high signal-to-noise ratio might be effective in classifying T2 mapping patterns and differentiating between the acetabular and femoral cartilages. However, additional studies to compare diagnostic accuracy and reproducibility between MR images at 3 T and at conventional 1.5 T are necessary to determine true advantages of using 3 T on imaging of hip cartilage.

In contrast to the normal hips of volunteers in the present study, the hips in the early-arthritis groups showed a high frequency of the high T2 pattern in the acetabular cartilage. The tendency of an increase in T2 associated with arthritic involvement agrees with previous results concerning high T2 in patients with knee osteoarthritis¹⁸. Abnormal elevation of T2 is accounted for pathologically by an increase in water content and water mobility associated with a decrease in proteoglycan content and disruption of the collagen network^{18,32}. Predominant occurrence of high T2 pattern in the acetabular cartilage agrees with previous arthroscopic findings²⁵ showing high frequency of cartilage disorder in the superior acetabular cartilage at early-arthritis stages of hip dysplasia. An interesting finding of the present study is the high frequency of the low T2 pattern in the acetabular and femoral cartilage of the pre- and early-arthritis group. There are several possible explanations for this specific pattern of T2 mapping. First, the cartilage in dysplastic hips is prone to increasing biomechanical stress at the weight-bearing area due to reduced contact area between the opposing surfaces⁴⁶. Previous interventional studies of the knee cartilage showed that axial loading by mechanical loading apparatus and cyclic compressive load by running exercise had a T2-shortening effect, presumably due to the loss of water content or an increase in collagen fiber anisotropy^{35,36}. The long-term biomechanical environment of elevated stress distribution in dysplastic hips could lead to a different quantity and distribution of the cartilage matrix compared to normal hips, leading to different mapping of the low T2 pattern. Second, the articular cartilaginous structure of the hip progressively changes during postnatal developmental periods. In dysplastic hips, the inverted or hypertrophic labrum may cover the outer surface

of the acetabular articular cartilage after birth and may subsequently constitute a portion of the acetabular cartilage after the childhood developmental periods are completed^{47–49}. Because the labrum has a considerably different extracellular matrix structure from the hyaline cartilage, with poor glycoaminoglycan and disorganized collagen fibrils⁴⁹, acetabular cartilage with a mixture of original labral components might provide a low T2 pattern in T2 mapping. Hip of the early-arthritis group might present with either low T2 or high T2 pattern in the acetabular cartilage, depending on severity of involvement of degenerative changes. Although further follow-up of hips with high and low T2 patterns is needed to determine the clinical relevance of these specific patterns, it is tempting to suggest that T2 assessment may not only provide early detection of osteoarthritic degeneration but also enable the detection of developmental pathological disorders of the cartilage matrix that may lead to disorders of biomechanical function on load-bearing in cases of hip dysplasia.

Quantitative assessment of T2 values within the defined ROI failed to show significant differences among the three groups, although average T2 values of the acetabular ROI for the pre-arthritis group were relatively low and those for the early-arthritis group were relatively high, as compared with the normal group. There is considerable variation in T2 values along the cartilage depth in response to physiological non-uniform distribution of extracellular matrix in normal cartilage^{17,28}. Degenerative change of matrix components in the early phase is likely to occur in a superficial or small localized area¹⁷. Average T2 values of bulk ROI from the cartilage base to the articular surface might be insufficiently sensitive to detect small T2 changes in a regional area, requiring the use of other quantitative methods such as comparison of T2 profile curves as a function of normalized distance from the bone/cartilage interface to the cartilage surface^{17,28}, or the enhancement of abnormal T2 adjusted by standard T2 value distributions within the cartilage at each pixel.

Pain assessment using the WOMAC score was not correlated with T2 assessment in the hip cartilage at the superior zone. This absence of correlation may partly reflect the relatively mild level of hip pain in the present study. In addition, there are many potential sources of hip pain other than disorders of the articular cartilage, including labral tear, synovitis, ganglionic cyst, and loose body⁵⁰. Previous arthroscopic studies for dysplastic hips at the pre-arthritis stage indicated a high correlation between hip pain and labral tear⁵¹. The status of labral disorders might have a stronger influence on pain severity than the status of articular cartilage disorders investigated in the present study.

There are several limitations in the present study. First, the pulse sequences available in this study meant that T2 values were calculated from two echoes. In many previous studies, T2 was calculated from more than two echo images and the initial echo image obtained from a multi-echo sequence was excluded in calculating T2 to minimize T2 inaccuracy caused by stimulated echoes^{17,28,37}; however, a previous study that used a dual-echo spin-echo sequence for T2 assessment successfully achieved significant differences between the knee cartilages of healthy subjects and those of patients with osteoarthritis¹⁸. Given the similarities of the gradient pattern of T2 mapping in the present study to previous findings in knee cartilage^{17,28}, we consider that T2 assessment using a dual-echo spin-echo sequence allowed reliable assessment of the extracellular matrix in the hip cartilage. Second, reliability of T2 assessment was influenced both by reproducibility of acquisition of

MR images and reproducibility of T2 calculation such as definition of ROI or judgment of visual appearance of T2 mapping patterns. Acceptable reproducibility of T2 calculation was obtained in this study with inter-observer reliability ranging from 2.5% to 3.8%. However, reproducibility of acquisition of MR images by scanning repeatedly was not evaluated, and it is unknown how variations in acquisition of MR images influenced the outcomes. Third, this was a feasibility study that conducted comparison of T2 values and mapping patterns between the normal hips and dysplastic hips only at the superior zone, where assessment is particularly important for dysplastic hips, based on biomechanical condition and assessment of osteoarthritis progression. However, additional care should be taken in interpreting T2 values in further studies to assess other anterior or posterior areas of the hip cartilage. Collagen fibril orientation of the cartilage against the static magnetic field differs considerably between the anterior, superior, and posterior regions because of the strongly curved structure of the articular cartilage of the hip. Assessment of T2 values may be significantly influenced by the variations in collagen fibril orientation associated with cartilage positions⁵². Finally, the number of normal volunteers and patients with hip dysplasia was small. The subjects were limited to female gender, and predominantly young subjects were examined both in volunteers and patients, partly due to the low frequency of pre-arthritis or early-arthritis stages in older patients with hip dysplasia. Previous reports showed that T2 in the knee joint was influenced significantly by age¹⁷ and insignificantly by gender²¹; however, it is unknown whether these factors influence the T2 of hip cartilage. Further studies are required to explore the degree of influence of age, gender, and other relevant factors on hip cartilage T2.

In summary, this preliminary study reveals that T2 assessment using 3 T MR imaging shows promise in the early detection of osteoarthritic degeneration and in the detection of developmental pathological disorders of cartilage matrix in patients with hip dysplasia. A combination of T2 assessment and other quantitative assessment techniques such as dGEMRIC⁵³, which is sensitive to cartilage proteoglycan content, may enable further detailed assessment of fundamental cartilage disorders in patients with dysplastic hips and enhance the early detection of the degeneration of hip cartilage.

Acknowledgments

This work was partly supported by Grant-in-Aid for Scientific Research, the Ministry of Education, Science and Culture in Japan, and Grant of Japan Hip Research Foundation, Inc.

References

- Harris WM. Etiology of osteoarthritis of the hip. *Clin Orthop* 1986;213:20-33.
- Nakamura S, Ninomiya S, Nakamura T. Primary osteoarthritis of the hip joint in Japan. *Clin Orthop* 1989;241:190-6.
- Lane NE, Lin P, Christiansen L, Gore R, Williams EN, Hochberg MC, *et al.* Association of mild acetabular dysplasia with an increased risk of incident hip osteoarthritis in elderly white women: the study of osteoporotic fractures. *Arthritis Rheum* 2000;43:400-4.
- Hasegawa Y, Iwata H, Mizuno M, Genda E, Sato S, Miura T. The natural course of osteoarthritis of the hip due to subluxation or acetabular dysplasia. *Arch Orthop Trauma Surg* 1992;111:187-91.
- Anwar MM, Sugano N, Matsui M, Takaoka K, Ono K. Dome osteotomy of the pelvis for osteoarthritis secondary to hip dysplasia: an over five-year follow-up study. *J Bone Joint Surg Br* 1993;75-B:222-7.
- McCrae F, Shouls J, Dieppe P, Watt I. Scintigraphic assessment of osteoarthritis of the knee joint. *Ann Rheum Dis* 1992;51:938-42.
- Nishii T, Tanaka H, Nakanishi K, Sugano N, Miki H, Yoshikawa H. Fat-suppressed 3D spoiled gradient-echo MRI and MDCT arthrography of articular cartilage in patients with hip dysplasia. *AJR Am J Roentgenol* 2005;185:379-85.
- Nishii T, Sugano N, Sato Y, Tanaka H, Miki H, Yoshikawa H. Three-dimensional distribution of acetabular cartilage thickness in patients with hip dysplasia: a fully automated computational analysis of MR imaging. *Osteoarthritis Cartilage* 2004;12:650-7.
- Schmid MR, Nötzli HP, Zanetti M, Wyss TF, Hodler J. Cartilage lesions in the hip: diagnostic effectiveness of MR arthrography. *Radiology* 2003;226:382-6.
- Zhai G, Cicuttini F, Srikanth V, Cooley H, Ding C, Jones G. Factors associated with hip cartilage volume measured by magnetic resonance imaging: the Tasmanian Older Adult Cohort Study. *Arthritis Rheum* 2005;52:1069-76.
- Fife RS, Brandt KD, Braunstein EM, Katz BP, Shelbourne KD, Kalasinski LA, *et al.* Relationship between arthroscopic evidence of cartilage damage and radiographic evidence of joint space narrowing in early osteoarthritis of the knee. *Arthritis Rheum* 1991;34:377-82.
- Felson DT. The course of osteoarthritis and factors that affect it. *Rheum Dis Clin North Am* 1993;19:607-15.
- Buckwalter JA, Mankin HJ. Articular cartilage: degeneration and osteoarthritis, repair, regeneration, and transplantation. *Instr Course Lect* 1998;47:487-504.
- Burstein D, Gray ML. Is MRI fulfilling its promise for molecular imaging of cartilage in arthritis? *Osteoarthritis Cartilage* 2006;14:1087-90.
- Wayne JS, Kraft KA, Shields KJ, Yin C, Owen JR, Disler DG. MR imaging of normal and matrix-depleted cartilage: correlation with biomechanical function and biochemical composition. *Radiology* 2003;228:493-9.
- Liess C, Lüsse S, Karger N, Heller M, Glüer CC. Detection of changes in cartilage water content using MRI T2-mapping *in vivo*. *Osteoarthritis Cartilage* 2002;10:907-13.
- Mosher TJ, Dardzinski BJ, Smith MB. Human articular cartilage: influence of aging and early symptomatic degeneration on the spatial variation of T2- preliminary findings at 3T. *Radiology* 2000;214:259-66.
- Dunn TC, Lu Y, Jin H, Ries MD, Majumdar S. T2 relaxation time of cartilage at MR imaging: comparison with severity of knee osteoarthritis. *Radiology* 2004;232:592-8.
- Gold GE, Suh B, Sawyer-Glover A, Beaulieu C. Musculoskeletal MRI at 3.0 T: initial clinical experience. *AJR Am J Roentgenol* 2004;183:1479-86.
- Inoue K, Wichart P, Kawasaki T, Huang J, Ushiyama T, Hukuda S, *et al.* Prevalence of hip osteoarthritis and acetabular dysplasia in French and Japanese adults. *Rheumatology (Oxford)* 2000;39:745-8.

21. Mosher TJ, Collins CM, Smith HE, Moser LE, Sivarajah RT, Dardzinski BJ, *et al.* Effect of gender on *in vivo* cartilage magnetic resonance imaging T2 mapping. *J Magn Reson Imaging* 2004;19:323-8.
22. Fredensborg N. The CE angle of normal hips. *Acta Orthop Scand* 1976;47:403-5.
23. Crowe JF, Mani VJ, Ranawat CS. Total hip replacement in congenital dislocation and dysplasia of the hip. *J Bone Joint Surg Am* 1979;61-A:15-23.
24. Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. *Ann Rheum Dis* 1957;16:494-502.
25. Noguchi Y, Miura H, Takasugi S, Iwamoto Y. Cartilage and labrum degeneration in the dysplastic hip: generally originates in the anterosuperior weight-bearing area: an arthroscopic observation. *Arthroscopy* 1999;15:496-506.
26. Maier CF, Tan SG, Hariharan H, Potter HG. T2 quantitation of articular cartilage at 1.5 T. *J Magn Reson Imaging* 2003;17:358-64.
27. Yao L, Gentili A, Thomas A. Incidental magnetization transfer contrast in fast spin-echo imaging of cartilage. *J Magn Reson Imaging* 1996;6:180-4.
28. Smith HE, Mosher TJ, Dardzinski BJ, Collins BG, Collins CM, Yang QX, *et al.* Spatial variation in cartilage T2 of the knee. *J Magn Reson Imaging* 2001;14:50-5.
29. Bellamy N, Buchanan WW, Goldsmith CH, Campbell J, Stitt LW. Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. *J Rheumatol* 1988;15:1833-40.
30. Mankin HJ, Dorfman H, Lippie L, Zarins A. Biochemical and metabolic abnormalities in articular cartilage from osteo-arthritic human hips. II. Correlation of morphology with biochemical and metabolic data. *J Bone Joint Surg Am* 1971;53-A:523-37.
31. Panula HE, Hyttinen MM, Arokoski JP, Langsjö TK, Peltari A, Kiviranta I, *et al.* Articular cartilage superficial zone collagen birefringence reduced and cartilage thickness increased before surface fibrillation in experimental osteoarthritis. *Ann Rheum Dis* 1998;57:237-45.
32. Nieminen MT, Töyräs J, Rieppo J, Hakumäki JM, Silvennoinen J, Helminen HJ, *et al.* Quantitative MR microscopy of enzymatically degraded articular cartilage. *Magn Reson Med* 2000;43:676-81.
33. Lüsse S, Claassen H, Gehrke T, Hassenpflug J, Schunke M, Heller M, *et al.* Evaluation of water content by spatially resolved transverse relaxation times of human articular cartilage. *Magn Reson Imaging* 2000;18:423-30.
34. Mosher TJ, Liu Y, Yang QX, Yao J, Smith R, Dardzinski BJ, *et al.* Age dependency of cartilage magnetic resonance imaging T2 relaxation times in asymptomatic women. *Arthritis Rheum* 2004;50:2820-8.
35. Mosher TJ, Smith HE, Collins C, Liu Y, Hancy J, Dardzinski BJ, *et al.* Change in knee cartilage T2 at MR imaging after running: a feasibility study. *Radiology* 2005;234:245-9.
36. Nag D, Liney GP, Gillespie P, Sherman KP. Quantification of T₂ relaxation changes in articular cartilage with *in situ* mechanical loading of the knee. *J Magn Reson Imaging* 2004;19:317-22.
37. Lazovic-Stojkovic J, Mosher TJ, Smith HE, Yang QX, Dardzinski BJ, Smith MB. Interphalangeal joint cartilage: high-spatial-resolution *in vivo* MR T2 mapping - a feasibility study. *Radiology* 2004;233:292-6.
38. Shepherd DE, Seedhom BB. Thickness of human articular cartilage in joints of the lower limb. *Ann Rheum Dis* 1999;58:27-34.
39. Armstrong CG, Mow VC. Variations in the intrinsic mechanical properties of human articular cartilage with age, degeneration, and water content. *J Bone Joint Surg Am* 1982;64-A:88-94.
40. Kempson GE, Muir H, Swanson SA, Freeman MA. Correlations between stiffness and the chemical constituents of cartilage on the human femoral head. *Biochim Biophys Acta* 1970;215:70-7.
41. Hayes CW, Balkissoon AA. Magnetic resonance imaging of the musculoskeletal system. II. The hip. *Clin Orthop* 1996;322:297-309.
42. Gold GE, Han E, Stainsby J, Wright G, Brittain J, Beaulieu C. Musculoskeletal MRI at 3.0 T: relaxation times and image contrast. *AJR Am J Roentgenol* 2004;183:343-51.
43. Eckstein F, Charles HC, Buck RJ, Kraus VB, Remmers AE, Hudelmaier M, *et al.* Accuracy and precision of quantitative assessment of cartilage morphology by magnetic resonance imaging at 3.0T. *Arthritis Rheum* 2005;52:3132-6.
44. Fischbach F, Bruhn H, Unterhauser F, Ricke J, Wiener G, Felix R, *et al.* Magnetic resonance imaging of hyaline cartilage defects at 1.5T and 3.0T: comparison of medium T2-weighted fast spin echo, T1-weighted two-dimensional and three-dimensional gradient echo pulse sequences. *Acta Radiol* 2005;46:67-73.
45. Kurrat HJ, Oberländer W. The thickness of the cartilage in the hip joint. *J Anat* 1978;126:145-55.
46. Hipp JA, Sugano N, Millis MB, Murphy SB. Planning acetabular redirection osteotomies based on joint contact pressure. *Clin Orthop* 1999;364:134-43.
47. Tachdjian MO. Congenital dysplasia of the hip. In: Wickland Jr EH, Ed. *Pediatric Orthopedics*. Philadelphia: Saunders WB 1990;Volume I:297-312.
48. Dunn PM. Perinatal observations on the etiology of congenital dislocation of the hip. *Clin Orthop* 1976;119:11-22.
49. Ponseti IV. Morphology of the acetabulum in congenital dislocation of the hip. Gross, histological and roentgenographic studies. *J Bone Joint Surg Am* 1978;60-A:586-99.
50. Baber YF, Robinson AH, Villar RN. Is diagnostic arthroscopy of the hip worthwhile? A prospective review of 328 adults investigated for hip pain. *J Bone Joint Surg Br* 1999;81-B:600-3.
51. Suenaga E, Noguchi Y, Jingushi S, Shuto T, Nakashima Y, Miyaniishi K, *et al.* Relationship between the maximum flexion-internal rotation test and the torn acetabular labrum of a dysplastic hip. *J Orthop Sci* 2002;7:26-32.
52. Xia Y. Magic-angle effect in magnetic resonance imaging of articular cartilage: a review. *Invest Radiol* 2000;35:602-21.
53. Kim YJ, Jaramillo D, Millis MB, Gray ML, Burstein D. Assessment of early osteoarthritis in hip dysplasia with delayed gadolinium-enhanced magnetic resonance imaging of cartilage. *J Bone Joint Surg Am* 2003;85-A:1987-92.