

Does Cartilage Degenerate in Asymptomatic Hips With Cam Morphology?

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Received: 8 May 2018 / Accepted: 12 December 2018 / Published online: 6 February 2019
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Abstract

Background The management of asymptomatic individuals with cam femoral morphology that predisposes their hips to femoroacetabular impingement has received little attention. Such hips may have subclinical articular damage;

This study was supported by The Canadian Institutes of Health Research (PB; MOP97778). One of the authors certifies that he (PB) has received or may receive personal fees, during the study period, in an amount of USD 10,000 to USD 100,000 from MicroPort (Arlington, TN, USA), personal fees in an amount of USD 10,000 to USD 100,000 from MatOrtho (Surrey, UK), personal fees in an amount of USD 10,000 to USD 100,000 from Zimmer Biomet (Warsaw, IN, USA), personal fees in an amount of USD 10,000 to USD 100,000 from Corin (Cirencester, UK), personal fees in an amount of USD 10,000 to USD 100,000 from Medacta (Castel San Pietro, Switzerland), and grants in an amount of USD 100,001 to USD 1,000,000 from Zimmer Biomet, all outside the submitted work.

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All ICMJE Conflict of Interest Forms for authors and *Clinical Orthopaedics and Related Research*® editors and board members are on file with the publication and can be viewed on request.

however, whether this cartilage damage will progress is unknown as is whether any particular bone morphologies are associated with this progression. Such knowledge could help determine the natural history and guide management of such individuals.

Questions/purposes The purpose of this study was to determine whether (1) asymptomatic hips with cam morphology are at risk of further cartilage degeneration (as evaluated by T1ρ); (2) T1ρ changes are predictive of symptom onset; and (3) bony morphologic parameters are associated with T1ρ signal changes.

Methods In a prospective, longitudinal study, 17 asymptomatic volunteers/hips (16 men; 33 ± 6 years) with cam morphology underwent two T1ρ MRI scans and functional assessment (WOMAC) at recruitment and at 4 years (range, 2–6 years). Volunteers were recruited from a previous study, which reported on the prevalence of cam morphology among asymptomatic individuals using hip MRI; cam morphology was defined as an α angle ≥ 60° anterolaterally and/or ≥ 50.5° anteriorly relative to the neck axis. The differences in T1ρ values (ΔT1ρ) and relative differences (%ΔT1ρ) were calculated as: ΔT1ρ = T1ρ_{Followup} – T1ρ_{Initial} and %ΔT1ρ = ΔT1ρ/T1ρ_{Initial}. A %ΔT1ρ > 17.6% was considered clinically important. Using CT data, femoral, acetabular, and spinopelvic parameters were measured. Whether ΔT1ρ and/or %ΔT1ρ was associated with any of the bone morphologic parameters was tested using Spearman's correlation coefficient. **Results** The global T1ρ in these asymptomatic hips with cam morphology remained unchanged between initial (mean, 35 ± 5 ms) and followup scans (mean, 34 ± 3 ms; p = 0.518). No differences with the numbers available in T1ρ values were seen initially between the anterolateral and posterolateral (34 ± 6 ms versus 33 ± 4 ms; p = 0.734)

regions; at followup, T1 values were higher posterolaterally (36 ± 5 ms versus 32 ± 5 ms; $p = 0.031$). The mean global $\Delta T1\rho$ was 1 ± 5 ms (95% confidence interval, -1 to +3 ms) and the mean global $\% \Delta T1\rho$ was $2\% \pm 13\%$. Two volunteers reported lower WOMAC scores; one patient exhibited a clinically important increase in $\% \Delta T1\rho$ (-26%). The degree of acetabular coverage correlated with $\% \Delta T1\rho$ ($\rho = 0.59-0.61$, $p = 0.002$); the lesser the acetabular coverage anterolaterally, the greater the corresponding area's T1ρ at followup.

Conclusions Although signs of posterolateral joint degeneration were detected, these were not generally associated with symptoms, and only one of the two volunteers with the onset of symptoms had a clinically important increase in $\% \Delta T1\rho$. We found that reduced acetabular coverage may increase the likelihood that preclinical cartilage degeneration will arise within 2 to 6 years; thereby reduced acetabular coverage should be considered when stratifying asymptomatic hips at risk of degeneration. Future studies should be performed with a larger cohort and include femoral version among the parameters studied.

Level of Evidence Level II, diagnostic study.

Introduction

Femoroacetabular impingement (FAI) is a dynamic process, which leads to abutment of the femoral head-neck junction against the acetabulum [33]. Variant morphologic features of the proximal femur (such as cam morphology [17, 26]) and/or acetabulum (pincer morphology) [16] are considered the principal factors contributing to FAI. Other factors such as spinal morphology [24] and pelvic motion [35] patterns also are believed to contribute. FAI can predispose people to osteoarthritis (OA) development; however, not all hips with radiographic features consistent with FAI are symptomatic or go on to develop degenerative changes [1, 22].

The therapeutic goal in symptomatic FAI is to relieve pain, improve pain-free ROM, and halt the progression of cartilage degeneration [12]. Identifying hips in the early phases of chondrolabral damage aids timely surgical management before irreversible chondral degeneration and improves outcome (pain reduction and joint preservation) after treatment [14, 15]. Conversely, the management of asymptomatic individuals with abnormal bony features suggestive of FAI has received less attention. In recent studies [37], it has been shown that these asymptomatic patients may have compromised cartilage in a pattern similar to patients with symptomatic FAI despite the lack of symptoms [5]. Whether the cartilage damage will progress is unknown but is important because this knowledge could help determine the natural history of cam morphology and guide the management of such individuals.

Traditional imaging modalities have limited value in detecting early degenerative changes [18]. Advanced MRI techniques such as delayed gadolinium-enhanced MRI of

cartilage (dGEMRIC) [39, 45], T1ρ [40, 41], T2, and T2* [25] mapping allow probing of the biochemical content of cartilage tissue in vivo and have been shown to be sensitive to molecular changes seen in early OA. An inverse relation has been found between T1ρ relaxation time and proteoglycan content (an early marker of OA) [2, 47] in bovine and human cartilage samples of the knee [20, 38] and hip [40, 41].

The aims of this longitudinal, T1ρ MRI study were to determine whether (1) asymptomatic hips with cam morphology are at risk for further cartilage degeneration; (2) T1ρ changes are predictive of symptom onset; and (3) bony (acetabular, femoral, or spinal) morphologic parameters are associated with any of the T1ρ signal changes.

Patients and Methods

This is a prospective, single-center, institutional review board-approved study. Participants were recruited from a previous cross-sectional study of 200 asymptomatic hips [21] and have been described in previous reports [5, 6]. Of these hips, 28 (20 males) had a cam deformity and were asymptomatic. All were invited to participate in a study comparing the T1ρ signal between asymptomatic hips with cam morphology and controls [5]; 20 (71%) accepted the invitation. Inclusion criteria for this study were no previous hip pathology or surgery, no signs of OA based on radiologic (MRI) assessments at the time of the cross-sectional study, WOMAC score (> 90) [10] at initial MRI, and the presence of a cam deformity based on oblique axial imaging. The findings of the initial T1ρ imaging study compared with controls were previously reported [5]. Participants were mailed an Addendum to the Research Consent Form; those who agreed to further participation presented for a followup T1ρ MRI scan and functional outcome assessment at a minimum 2-year interval. Eighteen of the 20 hips, who volunteered for the initial study [5], presented for the second assessment at a mean interval of 4 years (range, 2–6 years). Motion artifacts were present in one hip, which could not be processed; the remaining 17 formed the study's cohort. A minimum scan interval of 2 years was deemed necessary. Most participants were men (16 of the 17) and the mean age was 33 years old (range, 27–45 years) at the time of the initial T1ρ (Table 1). The patients' mean body mass index was 25 kg/m^2 (range, $19-29 \text{ kg/m}^2$).

Bony Morphology

The alpha angle was measured on CT images of the radial series at the 1:30 and 3:00 positions. An alpha angle $> 60^\circ$

Table 1. Cohort demographics, interval between MRI scans, and WOMAC scores

Parameter	Mean (range)/number
Age (years)	33 (27–45)
Gender	
Male	16
Female	1
Side	
Right	6
Left	11
Interval (years)	4 (2–6)
WOMAC	
Initial	100 (95–100)
Followup	98 (87–100)
Δ WOMAC	-1 (-14 to 5)

Δ = delta.

at 1:30 and/or $> 50.5^\circ$ at 3:00 was defined as having a cam morphology [36]. The average alpha angle anterosuperiorly was 66° (range, 49° – 77°) and the average alpha angle anteriorly was 56° (range, 37° – 65°) [7, 36]. There were two hips with anterior cam lesions (3:00 location only), six hips with cam lesions located anterosuperiorly (1:30 location only), whereas nine hips had cam morphology at both the 1:30 and 3:00 locations.

Other parameters measured from CT, using validated software and methods [19], included the femoral neck-shaft angle [35], acetabular version and inclination, acetabular depth, acetabular subtended angles (measure of how much femoral head coverage the acetabulum provides along different points of its “clockface”) [13, 29], pelvic incidence, sacral slope, and pelvic tilt [30–32, 42] (Table 2). Subtended angles were measured around the weightbearing acetabular clockface starting anteriorly, at right angles to the anterior pelvic plane, equating to the 0° orientation. Thereafter, the superior acetabular aspect is at the 90° orientation and posterior acetabular rim is at 180° [19]. Subtended measurements were made at 30° increments around the acetabulum. Femoral version was measured according to Murphy et al. [34] using transverse slices at three different femoral locations: the center of the femoral head, the base of the femoral neck, and the condylar axis. Two observers (GG, GN) blinded to patient identity performed all measurements. Excellent interobserver coefficients (ICCs) were identified (ICC, 0.9–0.95; $p < 0.001$). Furthermore, one observer repeated the measurements for 10 participants and excellent ICCs (0.93–0.98) were identified.

Functional Outcome

The WOMAC osteoarthritis score [11] was obtained for all patients at the time of the first and second scans. There was no difference with the numbers available in the WOMAC

Table 2. Bony parameters for the whole cohort

Parameter	Value (range)
Anterosuperior alpha angle (1:30) ($^\circ$)	66 (49–77)
Anterior alpha angle (3:00) ($^\circ$)	56 (37–65)
Neck-shaft angle ($^\circ$)	127 (123–134)
Femoral anteversion ($^\circ$)	10 (-18 to 24)
Acetabular abduction ($^\circ$)	48 (42–56)
Acetabular anteversion ($^\circ$)	10 (-3 to 22)
Anterior subtended angle ($^\circ$)	75 (69–83)
Superior subtended angle ($^\circ$)	84 (78–94)
Posterior subtended angle ($^\circ$)	72 (67–81)
Pelvic incidence ($^\circ$)	49 (42–56)
Sacral slope ($^\circ$)	44 (28–53)
Spinopelvic tilt ($^\circ$)	6 (-6 to 25)

Subtended angle reflects the degree of femoral head coverage the acetabulum provides: % femoral head coverage = (subtended angle/90) x 100.

score between initial (100; range, 95–100) and followup assessments (98; 87–100; $p = 0.534$). The difference between the two WOMAC scores was established (Δ WOMAC): Δ WOMAC = WOMAC_{Followup} - WOMAC_{Initial}.

MRI Protocol

Each participant underwent a hip MRI that was performed on a 1.5-T MRI scanner (Magnetom Symphone; Siemens Medical Solutions, Erlangen, Germany) using a body coil for signal transmission and a flexible four-channel surface coil for signal reception. Participants were positioned in a similar fashion for both scans: supine with the leg fixed in neutral rotation. T1 ρ -weighted data sets were obtained in the sagittal oblique plane, parallel to the acetabular fossa (see Appendix, Supplemental Digital Content, <http://links.lww.com/CORR/A134>).

Image Postprocessing and Data Analysis

The MR images were segmented and analyzed using an in-house, custom-written software program developed in MATLAB® (MathWorks Inc, Natick, MA, USA). A musculoskeletal imaging scientist (GM) performed the segmentation analysis of the MRI scans. The T1 ρ mapping and analysis protocol used in this study were evaluated in a recent publication, which showed strong interobserver, intraobserver, and inter-MR scan reliability [6]. The ICC and root mean square coefficient of variation were 0.965 and 4% (intraobserver), 0.953 and 4% (interobserver), and 0.988 (all $p < 0.001$) and 9% (inter-MR scan), respectively [6].

We used an oblique coronal localizer sequence to establish the transverse coverage of the hip that included the superior weightbearing acetabular surface. Seven or eight (depending on joint size) sagittal T1ρ image slices were analyzed, starting from the lateral sourcil margin and extending medially. The hyaline cartilage was segmented as one layer (acetabulum and femoral head together) using the first T1ρ-weighted data set (spin lock time = 12 ms). The matched, anatomic intermediate-weight image was used to facilitate delineation of the hyaline cartilage margins for segmentation, ensuring that the labrum and subchondral compact bone were excluded from the region of interest.

After segmentation, the joint was divided and subdivided into two 90° regions defined using a line drawn through the center and superior apex of the femoral head, creating an anterior and posterior zone. In each hip, the T1ρ relaxation zones were calculated for the whole surface analyzed (T1ρ_{complete}), the anterior (T1ρ_{anterior}) and posterior (T1ρ_{posterior}) halves, and six principal regions of interest (ROIs) as previously described [6]. To determine T1ρ in the six ROIs, sagittal slices were divided into lateral (slices 1-3), intermediate (slices 4-6), or medial (slices 7-8) zones. Thereafter, the ROI could be subdivided into six areas (anterolateral, anterointermediate, anteromedial, posterolateral, posterointermediate, and posteromedial; Fig. 1).

When the initial T1ρ signal changes were reported in this cohort [5], higher values were detected anterolaterally

in asymptomatic hips with cam FAI (33 ± 6 ms) compared with controls (29 ± 4 ms).

Because two MR scans were available for each patient in this study, we measured interpatient variability by calculating the individual patient's T1ρ signal differences and relative differences. The absolute differences in T1ρ values ($\Delta T1\rho$) were determined and were calculated as: $\Delta T1\rho = T1\rho_{\text{Initial}} - T1\rho_{\text{Followup}}$.

The relative differences ($\% \Delta T1\rho$) = $\Delta T1\rho / T1\rho_{\text{Initial}}$ were calculated as: $\% \Delta T1\rho = \Delta T1\rho / T1\rho_{\text{Initial}}$.

A negative $\% \Delta T1\rho$ denotes a greater T1ρ value at followup, indicating a smaller proteoglycan content at followup (ie, further degeneration).

Outcome Measures

The T1ρ, $\Delta T1\rho$, and $\% \Delta T1\rho$ values were determined for all zones. It has been shown that the variability within the different regions in the joint is < 10% [40] and similarly the T1ρ difference from MR scans performed within a period of 2 weeks in the same individual can be up to 9% [6]. Therefore, 17.6% (1.96% x 9%) was considered clinically important.

Onset of symptoms was determined from the WOMAC score and if the score had deteriorated in the followup review. We tested the WOMAC score difference for correlation with the $\% \Delta T1\rho$; furthermore, we assessed whether participants with a clinically important reduction or

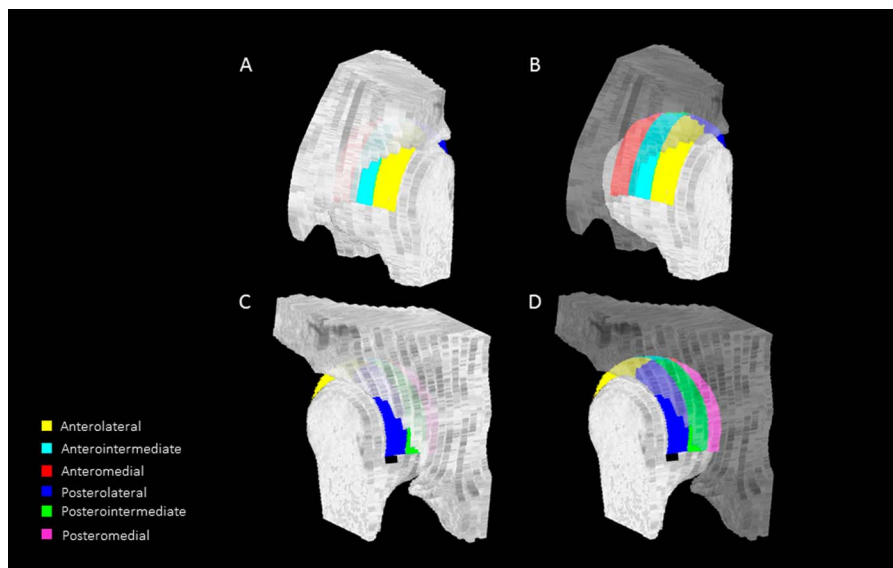


Fig. 1 A-D Three-dimensional reconstructed femoral acetabular joint showing the six cartilage regions analyzed in the study as color-coded overlays from different views. (A) AP oblique view. (B) AP oblique view with 75% acetabulum opacity. (C) Posteroanterior oblique view. (D) Posteroanterior oblique view with 75% acetabulum opacity.

increase in % Δ T1 ρ had greater Δ WOMAC compared with the rest of the group at followup.

Lastly, we tested whether the hips with a clinically important alteration in % Δ T1 ρ had different morphologic femoral, acetabular, or spinopelvic parameters compared with the rest of the hips. Whether Δ T1 ρ and/or % Δ T1 ρ were associated with any of the bone morphologic parameters was tested using correlation coefficients.

Sample Size Calculation

A power analysis based on published hip cartilage T1 ρ values of cam participants and healthy controls was performed to calculate the minimal number of hips needed for this study. In the anterolateral region, the area where cam impingement occurs, participants with a cam deformity showed significant higher cartilage T1 ρ values (33 ± 5 ms) compared with the control group (29 ± 4 ms) ($p = 0.039$) [6]. Based on these differences, using a power of 0.8 and alpha error (p) of 0.05, we estimated a minimum of 15 participants was needed for this study.

Statistical Analysis

Statistical analysis was performed with SPSS (Version 23; IBM Corp, Armonk, NY, USA). Nonparametric tests were used for analysis. Statistical significance was set at < 0.05 . The Mann-Whitney U and Kruskal-Wallis tests were used for scale data, whereas the chi-square and Fisher's exact tests were used for categorical data. Spearman's (ρ) correlation was used to determine whether any correlations existed for scale data. The Mann-Whitney U and Kruskal-Wallis tests were used for research question 1; Spearman's (ρ) correlation and Mann-Whitney U were used for research question 2, whereas Spearman's (ρ) correlation was used for research question 3.

Results

No difference in T1 ρ values was seen in these asymptomatic hips with cam morphology on the baseline MRI in the anterolateral (34 ± 6 ms) and posterolateral (33 ± 4 ms) ROIs ($p = 0.734$); however, at followup, the T1 value was higher in the posterolateral (36 ± 5 ms) ROI compared with the anterolateral (32 ± 3 ms) ROI ($p = 0.031$). The global T1 ρ remained unchanged between the initial (mean, 35 ± 5 ms) and followup scans (mean, 34 ± 3 ms) ($p = 0.518$). For the whole cohort, there were no differences in the T1 ρ obtained between the initial and followup MRIs for

any of the six ROIs (Table 3). The mean global Δ T1 ρ was 1 ± 5 ms (95% confidence interval [CI], -1 to +3 ms). Interval time between scans ($\rho = 0.24$, $p = 0.36$) and age at initial scan ($\rho = 0.13$, $p = 0.610$) did not correlate with global Δ T1 ρ . Similarly, body mass index did not correlate ($\rho = -0.10$, $p = 0.721$) with global Δ T1 ρ . The mean global % Δ T1 ρ was $2\% \pm 13\%$. The % Δ T1 ρ showed an overall reduction in T1 ρ value (at followup) over the anterior half of the joint (9%; 95% CI, 2%–17%), which is contrary to an overall increase in the T1 ρ over the posterior half (-5%; 95% CI, -14% to 4%) at followup.

We observed a clinically important change in the anterolateral % Δ T1 ρ ($\geq 18\%$) in four hips; in two of these hips, the T1 ρ value increased at followup, whereas in the other two hips, the T1 ρ value was reduced at followup. The patient with the largest % Δ T1 ρ (-26%) was one of the two patients who reported a WOMAC score reduction (from 100 to 92; Fig. 2). We found that % Δ T1 ρ in the anterolateral ROI strongly and positively correlated ($\rho = 0.73$; $p < 0.001$) with the T1 ρ value in the anterolateral ROI of the baseline MR scan (Fig. 3).

The only bony morphologic parameter that correlated with T1 ρ value was the acetabular subtended angle. The anterior and superior acetabular subtended angles ($\rho = 0.59$ – 0.61 ; $p = 0.002$; Table 4) positively and moderately correlated with the % Δ T1 ρ seen over the corresponding ROIs (anterolaterally); the smaller the degree of coverage, the more negative the % Δ T1 ρ (ie, greater T1 ρ signal at followup scan; Fig. 4).

Discussion

Proximal femoral cam morphology has a prevalence of approximately 15% among all comers [21]. It can be associated with an increased risk of pain [27] and development of OA within 5 years of detection [46] in some volunteers, yet not all hips with a cam deformity will develop OA or symptoms [1, 22]. Recent studies have identified static/bony and dynamic parameters that increase the risk of a hip with a cam deformity being symptomatic [35, 43]. Identifying which hips are at risk of degeneration and intervening in a timely manner are paramount in the quest of joint preservation. Using patient symptomatology (in particular hip pain) certainly would be an obvious choice, but because this can be nonspecific with regard to disease state [8], evaluating biomarkers such as cartilage degenerative markers in the blood or advanced cartilage imaging with MRI might help identify at-risk hips. In this small, longitudinal study, we found little progression of degenerative changes as measured by T1 ρ ; only two of 17 individuals with previously asymptomatic cam morphology of the proximal femur developed a clinically important degree of

Table 3. T1ρ measurements for the whole cohort including the global and ROIs measurements

Parameter	Value ± SD (range)	p value
T1ρ global initial scan (ms)	35 ± 5 (28–46)	0.518
T1ρ global followup scan (ms)	34 ± 4 (28–41)	
%ΔT1ρ global	1 (95% CI, -7 to 9)	
T1ρ anterolateral ROI initial scan (ms)	34 ± 6 (26–45)	0.586
T1ρ anterolateral ROI followup scan (ms)	32 ± 3 (27–39)	
%ΔT1ρ anterolateral	3.3 (95% CI, -5 to 10)	
T1ρ anterointermediate ROI initial scan (ms)	34 ± 6 (27–44)	0.474
T1ρ anterointermediate ROI followup scan (ms)	32 ± 4 (24–40)	
%ΔT1ρ anterointermediate	4 (95% CI, -4 to 13)	
T1ρ anteromedial ROI initial scan (ms)	37 ± 6 (28–50)	0.290
T1ρ anteromedial ROI followup scan (ms)	34 ± 5 (23–42)	
%ΔT1ρ anteromedial	6 (95% CI, -3 to 16)	
T1ρ posterolateral ROI initial scan (ms)	33 ± 4 (27–41)	0.122
T1ρ posterolateral ROI followup scan (ms)	36 ± 5 (26–43)	
%ΔT1ρ posterolateral	-4.6 (95% CI, -16 to 7)	
T1ρ posterointermediate ROI initial scan (ms)	34 ± 5 (27–48)	0.218
T1ρ posterointermediate ROI followup scan (ms)	36 ± 5 (27–41)	
%ΔT1ρ posterointermediate	-6 (95% CI, -18 to 3)	
T1ρ posteromedial ROI initial scan (ms)	35 ± 6 (26–52)	0.414
T1ρ posteromedial ROI followup scan (ms)	36 ± 5 (28–44)	
%ΔT1ρ posteromedial	-6 (95% CI, -21 to 4)	
Ratio AP lateral initial scan (ms)	1.0 ± 0.2 (0.8–1.3)	0.034*
Ratio AP lateral followup scan (ms)	0.9 ± 0.1 (0.8–1.4)	
Δ ratio AP lateral	0.1 (95% CI, 0-0.2)	
Ratio AP intermediate initial scan (ms)	1.0 ± 0.1 (0.7–1.2)	0.067
Ratio AP intermediate followup scan (ms)	0.9 ± 0.1 (0.7–1.2)	
Δ ratio AP intermediate	0.1 (95% CI, 0-0.2)	
Ratio anteroposterior medial initial scan (ms)	1.1 ± 0.1 (0.8–1.4)	0.024*
Ratio AP medial followup scan (ms)	1.0 ± 0.1 (0.6–1.2)	
Δ ratio AP medial	0.1 (95% CI, 0-0.2)	

*Statistical significance; ROI = regions of interest; CI = confidence interval.

articular cartilage degeneration as measured by T1ρ, and of those, only one developed symptoms. In addition, we were able to demonstrate that biomechanical parameters other than the size of the cam morphology may be of importance; decreasing acetabular coverage anteriorly and laterally was associated with a decrease in T1ρ value content change in individuals with asymptomatic cam morphology.

Limitations to this study include the small sample size, which was predetermined by the number of volunteers who had a cam deformity in the prevalence study (n = 28) and the number that presented for the initial scans (n = 20). Such a small cohort raises the possibility of selection biases. Second, we did not obtain activity data for any volunteers; such data would have allowed us to determine whether any activity modification could be responsible for

the %ΔT1ρ detected. Third, the majority of volunteers were male (16 of 17) and therefore the findings of this study may not apply to female hips; nevertheless, cam-type FAI is more prevalent among males. Fourth, the bony morphologic assessments were made based on CT-based analysis and not per radiographic analysis, which is most commonly performed in clinical practice. However, this was performed to improve accuracy of the three-dimensional anatomic assessments. Lastly, a mean 4-year interval between MRI scans is not a long interval in the lifetime of the native hip, and therefore, longer surveillance and longitudinal studies are necessary. However, this work establishes key parameters in further determining what represents a pre- versus a clinical disease state in patients with cam morphology. Previous work has shown that the degenerative process in patients with cam morphology is

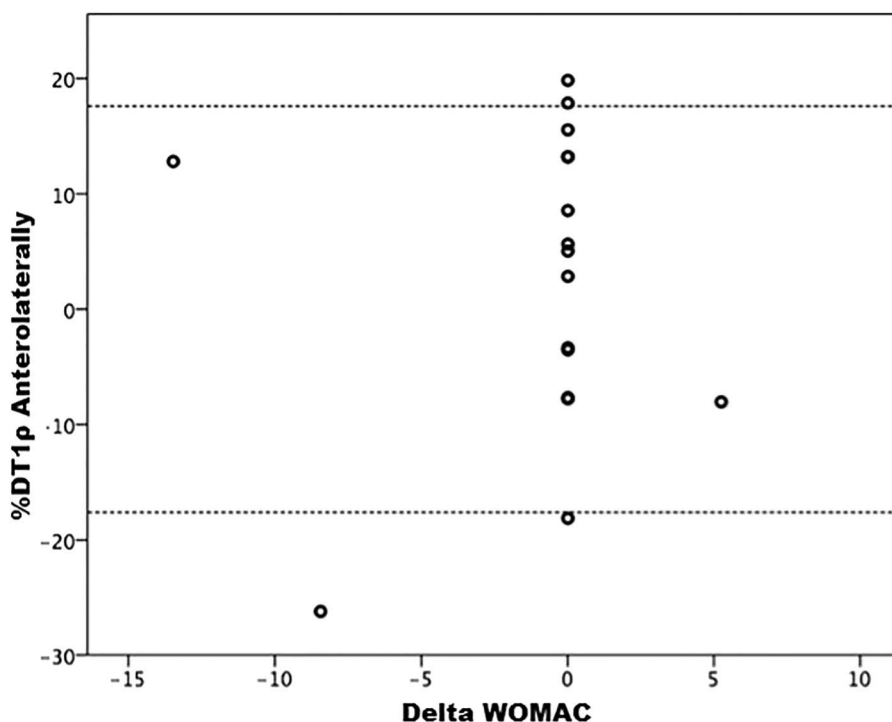


Fig. 2 Scatter box of $\% \Delta T1\rho$ seen in the anterolateral ROI (y-axis, plotted against Δ WOMAC). Dashed lines represent boundaries of significance ($\pm 17.6\%$ of change between scans).

complex with a variety of biomechanical and biologic processes involved [28, 35, 44].

Considering the whole cohort, the mean global $\% \Delta T1\rho$ was 1%, demonstrating that on average very little change in the overall articular cartilage health took place, although we did detect regional differences (reduction in $T1\rho$ value anteriorly, increase in $T1\rho$ value posteriorly). In two cross-sectional, observational studies of asymptomatic hips, nonuniform $T1\rho$ values have been described with increasing $T1\rho$ values detected from anterior to posterior [40, 45]. In this longitudinal cohort, similar $T1\rho$ values were seen at the baseline scan between the anterolateral (34 ms) and posterolateral (33 ms) ROIs; however, at followup, the $T1\rho$ values were higher for posterolateral (36 ms) compared with anterolateral (32 ms). This positive change (increased $T1\rho$ signal) in the posterolateral zone would suggest arthritis progression within regions other than the anterolateral zone. The cause of these findings calls for further studies; for example, it could represent arthritis progression secondary to (1) impingement in the superoposterior region, which would typically occur in 90° of flexion in a hip with a cam anterosuperiorly; or (2) microinstability–anterior impingement could lead to counter-coup injury posteriorly, a mechanism precipitated by lesser degrees of acetabular cover or lastly secondary to the action of the inflammatory mediators within the hip with cam FAI [23]. It is evident that further (in vitro and in vivo) studies are needed

that would address these mechanical and molecular hypotheses. Such studies would study the areas of hip impingement with three-dimensional modeling work and identify potential microinstability of the hip, correlating the findings with cartilaginous mapping. Additionally, an animal model of the disease could help us determine what contributes to the changes and in what topographic order. To study the contribution of inflammation, histologic and immunologic analysis from the synovial fluid and other regions of the joint from patients in the various stages of the pathologic process would be necessary.

With only two patients who developed symptoms over the relatively short followup interval (2–6 years) in this study, we were not able to determine whether $T1\rho$ changes were predictive of symptom onset. Whether asymptomatic individuals with cam morphology should be under surveillance is a matter of debate. However, this group of patients already exhibited signs of degeneration compared with normal individuals [5], and it would appear that for the most part they still remain asymptomatic within 5 years. This is consistent with other reports showing that certain patients with cam morphology can remain free of arthritic symptoms [3]. The findings of this cohort would argue that surveillance with the newest generation MRI modalities is not currently indicated in patients within 5 years of followup. One of the two volunteers with a clinically important reduction in the proteoglycan content (as per $\% \Delta T1\rho$)

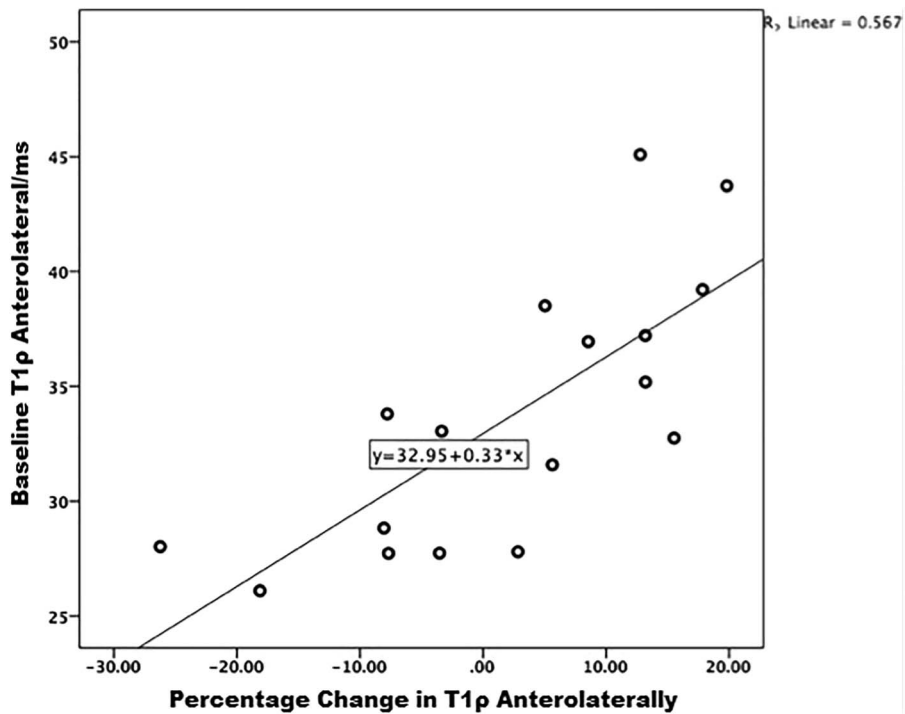


Fig. 3 Scatterplot of baseline T1ρ in the anterolateral ROI plotted against the percentage change in T1ρ in the anterolateral ROI.

was a patient with a decreased WOMAC score. In this cohort, there were two volunteers with a reduction in WOMAC score by followup, and only one showed a clinically important increase in T1ρ (%ΔT1ρ of -26% in anterolateral ROI). What factors might cause the onset of hip pain is unclear, and certainly one could argue that

patient symptomatology probably is the most effective way to monitor those patients. For the asymptomatic individual with cam morphology, our current understanding of the clinical relevance of the observed T1ρ signal changes and their relationship with patient-reported outcome measures necessitates further investigation. Furthermore, such studies should include and compare the various advanced MR techniques (dGEMRIC, T2, T1ρ) as part of crossvalidation because they probe different biologic changes that occur in cartilage degeneration.

Table 4. Correlations tested for the different bony parameters and the %ΔT1ρ in the anterolateral ROI

Bony parameter	Correlation with %ΔT1ρ in AL ROI
Anterosuperior alpha angle (1:30) (°)	ρ = -0.1, p = 0.680
Anterior alpha angle (3:00) (°)	ρ = -0.1, p = 0.745
Neck-shaft angle (°)	ρ = -0.1, p = 0.637
Femoral anteversion (°)	ρ = 0.1, p = 0.748
Acetabular abduction (°)	ρ = 0.2, p = 0.437
Acetabular anteversion (°)	ρ = -0.1, p = 0.888
Anterior subtended angle (°)	ρ = 0.6, p = 0.012*
Antero-superior subtended angle (°)	ρ = 0.6, p = 0.017*
Superior subtended angle (°)	ρ = 0.6, p = 0.024*
Posterior subtended angle (°)	ρ = 0.5, p = 0.034*
Pelvic incidence (°)	ρ = 0.1, p = 0.648
Sacral slope (°)	ρ = -0.3, p = 0.323
Spinopelvic tilt (°)	ρ = 0.2, p = 0.432

*Correlations of significance; ρ = Spearman's rho; AL = anterolateral; ROI = region of interest.

The only morphologic parameter that showed a positive correlation with T1ρ was the degree of acetabular coverage when there was greater coverage anteriorly; we detected a smaller increase in %ΔT1ρ. Two cohort studies [4, 9] reported a protective role of acetabular coverage reflected through the center-edge angle (a radiographic measure of the degree of femoral head coverage the acetabulum provides) on the degree of cartilage damage in cam-type FAI.

In conclusion, in this small longitudinal study, we found that only two of 17 individuals with asymptomatic cam morphologies developed clinically important progression of cartilage degeneration as measured by T1ρ, and of those, only one developed symptoms. By studying several bony parameters, we found that decreasing acetabular coverage, particularly anterosuperiorly, was associated with increased articular cartilage degeneration as measured by T1ρ. Reduced acetabular coverage may increase the likelihood that preclinical cartilage degeneration will arise within 2 to 6 years. Future

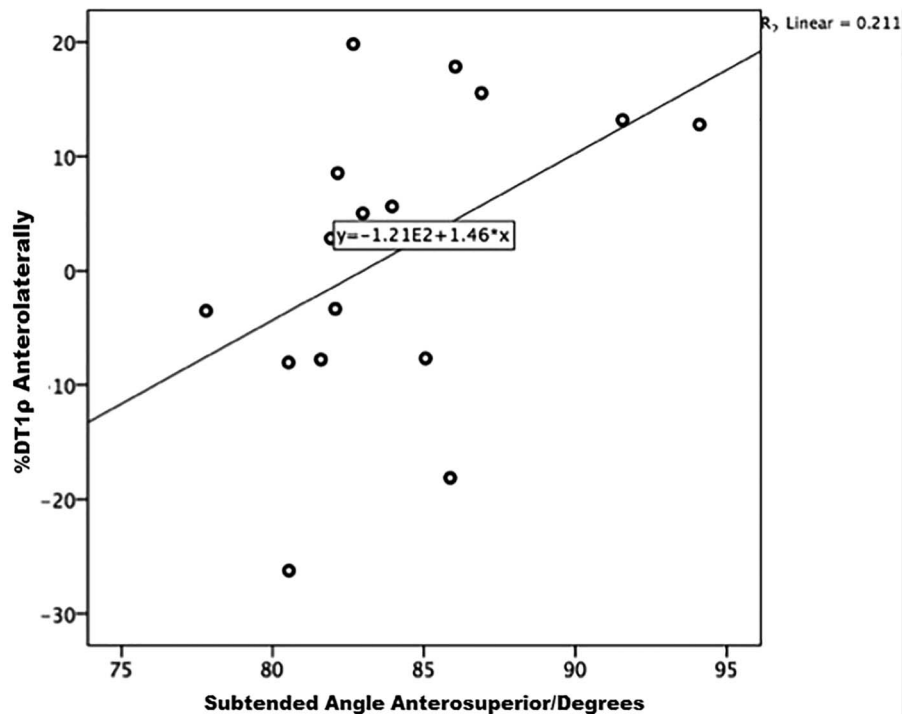


Fig. 4 Scatterplot of %ΔT1ρ seen in the anterolateral ROI (y-axis) plotted against subtended angle of acetabulum anterosuperiorly. Dashed line represented correlation detected.

longitudinal studies should include larger cohorts and the study of subclinical degeneration should perhaps include histologic and immunologic assessments of the hip's structures.

Acknowledgments We thank Andrew Speirs and Geoff Ng for their contributions to the study methodology. We also acknowledge The Hans K. Uthoff Scholarship Program for providing financial support to graduate students involved in this work.

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