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Longitudinal Assessment of MRI in Hip Osteoarthritis Using SHOMRI and Correlation with Clinical Progression

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Abstract

PURPOSE—To assess the evolution of MR imaging findings in normal volunteers and subjects with hip osteoarthritis (OA) over 1.5 years described by the semi-quantitative Scoring Hip OA with MRI (SHOMRI) scoring system and their correlation with the evolution of clinical parameters.

MATERIALS AND METHODS—Hip MRI studies of 18 subjects with (Kellgren-Lawrence (KL) score 2/3; mean age 54.4±11.2 years; 27.8% women) and 36 controls without radiographic OA (KL 0/1; mean age 43.7±12.8 years; 50.0% women) were assessed at baseline and after 1.5 years by using SHOMRI, and their clinical status was evaluated by using Harris Hip Score and Hip Disability and Osteoarthritis Outcome Score (HOOS). Imaging and clinical parameters at baseline and their change over time were compared between groups using Mann Whitney U and Fisher's exact tests. Spearman's rank correlations and generalized linear models adjusted for age, sex, BMI and KL were used to assess associations between imaging and clinical findings.

RESULTS—At baseline, OA subjects had significantly higher SHOMRI total scores than controls (median (IQR), 12.5 (6–19.5) vs. 7 (4–13.5); $p=0.024$). Over 1.5 years, only the progression rate of subchondral cysts was significantly higher in OA subjects than in controls (16.7 vs. 0.0%; $p=0.033$), while no significant differences were found for any of the other SHOMRI subscales.

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Baseline bone marrow edema pattern (BMEP) was significantly associated with worsening pain (HOOS subscale; $p=0.018$) and hip-related quality of life (HOOS subscale; $p=0.044$). Progression of subchondral cysts was significantly associated with worsening symptoms other than pain (HOOS subscale, $p=0.030$). Baseline KL did not significantly correlate with worsening of any clinical symptoms (each, $p>0.05$).

CONCLUSION—In this relatively young study population without or with mild to moderate radiographic hip OA, only minimal differences were found between groups regarding the progression of hip abnormalities as assessed by SHOMRI over 1.5 years. However, BMEP predicted clinical worsening and subchondral cyst progression was associated with worsening symptoms. Although longer follow-up periods are required, this suggests that SHOMRI is a useful tool to monitor hip abnormalities and their progression longitudinally.

1. Introduction

Osteoarthritis (OA) is the chronic joint disease with the highest prevalence in the United States with an increasing incidence due to the aging population and growing number of obese individuals [1, 2]. One in four people may develop symptomatic hip OA in his or her lifetime, causing pain and disability [3, 4]. While new therapeutic options have evolved for conditions associated with joint degeneration in younger patients such as femoroacetabular impingement [5], results from physical therapy and weight-loss are rather modest, and joint replacement surgery often remains the only available option for patients with primary hip OA [6]. Therefore, substantial research resources have been directed towards a better understanding of the underlying pathophysiology and new therapeutic approaches [7], as well as imaging modalities, that are able to detect early stages of the disease [8, 9]. Radiography is still commonly used to assess hip OA due to its widespread availability. Moreover, the Kellgren-Lawrence (KL) score and the measurement of joint space narrowing are assessed on radiographs and are considered as the best outcome measures for OA [10, 11].

However, radiography allows only for the evaluation of bone abnormalities and joint space narrowing typically seen in progressive stages of the disease. For the imaging of soft-tissue structures, which play a crucial role in the pathogenesis and development of clinical symptoms, MR imaging has emerged as an important tool. MRI of the hip remains challenging due to the complex geometry of the joint, its localization within the body, and the thin articular cartilage layers of the acetabulum and femur lying adjacent to one other [12-14]. In addition, to date, no dedicated hip coils exist resulting in reduced image quality when compared to other body regions such as the knee [12-14]. However, MRI is being used both in research and in daily clinical management more often [15], and the most recent Osteoarthritis Research Society (OARSI) Clinical Trials Recommendations [10] support its application in clinical studies.

To standardize reporting and to semi-quantitatively classify findings, the OARSI also advises the use of a semi-quantitative scoring system, such as the Hip OA MRI Scoring System (HOAMS) [16] or the Scoring Hip OA with MRI (SHOMRI) [17]. Of those, SHOMRI was specifically developed for the assessment of 3.0-T based MRI, and utilizes

sub-regional division of the acetabular and femoral cartilage, based on the geographic zone method established by the Arthroscopy Association of North America [18]. The SHOMRI scoring system showed good to excellent reproducibility and significant correlations with radiography-based OA grading. Several of its subscales describing different joint features, i.e. bone marrow edema pattern, subchondral cysts, articular cartilage, paralabral cysts and labrum pathologies showed significant correlations with clinical features [17]. This corresponds well with previous findings that changes to the subchondral bone and cartilage defects correlate with clinical symptoms [19-21].

However, SHOMRI has only been used in cross-sectional analyses so far [17, 22], and the progression of subscales over time and their clinical relevance remains unclear. To assess the value of this tool for future studies in the field of hip OA, and to estimate adequate follow-up periods, a longitudinal evaluation of the score is required. Therefore, the purpose of our study was to assess correlations between baseline imaging findings assessed by SHOMRI subscales and their evolution over 1.5 years with the evolution of clinical parameters, and to compare the progression of structural abnormalities in subjects with and without radiographic OA.

2. Materials and Methods

2.1. Subjects

Subjects for a multidisciplinary hip study were enrolled from the community from September 2010 to November 2012. Each subject underwent MR-imaging including routine clinical and research imaging sequences, and clinical information and functional performance correlation were gathered with emphasis in evaluation of progression of OA. We included male and female subjects without history of hip surgery, knee or ankle OA (KL 2), severe hip OA (KL=4), femoroacetabular impingement (either clinical or imaging findings), inflammatory arthritis, hematochromatosis, sickle cell disease, hemoglobinopathy, presence of any condition other than OA which limits lower extremity function and mobility, or MRI contraindications (such as presence of cardiac pacemakers or metal implants, or the possibility of pregnancy).

For this analysis, all subjects with complete MRI studies and clinical information at baseline and at 1.5-year follow-up were included (n=54; mean age 47.2 ± 13.2 years; 42.6% women; mean BMI 24.0 ± 3.1 kg/m²), and were stratified in two groups according to their hip KL score (see below): Control subjects with a KL score of 0 or 1, and OA subjects with a KL score of 2 or 3. In controls, the side presenting pain (in n=4 subjects) or any other symptoms (in n=4 subjects) was evaluated; if no symptoms were present, the evaluated side was selected randomly. In subjects with a KL score >1, the side showing more severe radiographic changes at baseline was selected; if scores were equal on both sides, the more symptomatic side was selected.

Between baseline and 1.5-year follow-up, none of the subjects suffered from a relevant injury of the lower extremity according to questionnaires obtained at follow-up.

The Committee of Human Research at our institution approved this study and informed consent was obtained from all subjects prior to participation.

2.2. Imaging and Analysis

Radiographs Acquisition and Analysis—In all subjects, standing anterior-posterior radiographs were performed at baseline. For positioning, the feet were aligned with slight internal rotation. Settings included a focus-film distance of 40 inches, voltage of 80 kVp with automatic exposure using a GE Discovery 650 x-ray system (GE Healthcare, Waukesha, WI).

For inclusion and cohort assignment as well as for the identification of the more severely affected side, both hips were graded by one musculoskeletal radiologist with 23 years of experience (TML) using the Kellgren-Lawrence scoring system [23]. This classification consists of four grades: grade 0, normal; grade 1, doubtful narrowing of joint space and possible osteophytic lipping; grade 2, definite osteophytes and possible narrowing of joint space; grade 3, multiple osteophytes, definite narrowing of joints space, some sclerosis, and possible deformity of bone contour; grade 4, large osteophytes, marked narrowing of joint space, severe sclerosis, and definite deformity of bone contour. A KL score equal or greater than two was considered as radiographic OA.

MRI Acquisition—Hip MRI examinations were performed on a 3.0-T scanner (GE MR750; GE Healthcare) using an eight-channel flex coil (GE Healthcare). The MRI protocol included intermediate-weighted fat-suppressed fast spin-echo (FSE) sequences in a sagittal, oblique coronal and oblique axial orientation with a repetition time (TR) 2400–3700 ms, echo time (TE) 60 ms, slice thickness 4 mm, gap 4 mm, field of view 14–20 cm, matrix 288×224 and acquisition time of 3 min 5 s to 4 min 40 s per sequence. To achieve a reproducible position in all hip joints the feet were rotated inwards, forefeet were taped together and knees were supported.

MRI Analysis and SHOMRI Scoring—The radiologists performing SHOMRI were blinded to radiographic scores, clinical and functional information, time point, and MRI findings at the other time point, respectively. Initial consensus training sessions were previously performed by four experienced radiologists (BJS, SL, LN, and TML) to calibrate and standardize readings. The training included 26 studies, which were read in three sessions each separated by a two-week period. Subsequently the remainder of the studies was scored by three radiologists (BJS, SL, LN) independently. In case of discrepant or unclear readings, a consensus reading in the presence of the fourth radiologist (TML, board certified radiologist with 23 years of experience) was performed.

The SHOMRI scoring system has previously been described in detail [17, 22]. In short, eight features were assessed: articular cartilage loss (scored 0–2), bone marrow edema pattern (BMEP; scored 0–3), subchondral cysts (scored 0–2), labral abnormalities (scored 0–5), paralabral cysts (scored as present vs. not present), intra-articular bodies (scored as present vs. not present), joint effusion (scored as present vs. not present) and ligamentum teres abnormality (scored 0–3). For the evaluation of cartilage loss, BMEP and subchondral cysts, the femoral head and the acetabulum were divided into six and four sub-regions,

respectively, based on the geographic zone method established by the Arthroscopy Association of North America (Figure 1) [18]. Labral abnormalities were graded in four subregions. The subregions were then added for the total scores of the different structures, and the total SHOMRI score was calculated by adding all subscales. Any positive change (>0) between values at baseline and 1.5-years follow-up was rated as structural progression.

2.3. Clinical Scores

At both baseline and follow-up, self-reported functional and clinical assessment was performed by using two scores: The Hip Disability and Osteoarthritis Outcome Score (HOOS) consists of 40 items categorized in five subscales for pain, symptoms other than pain, function in activities of daily living (ADL), function in sport and recreation (Sport/Rec), and hip-related quality of life. Scores take into consideration the week prior to the assessment, and are summarized for each of the five subscales and transformed to a 0–100 scale (0 indicating extreme symptoms and 100 indicating no symptoms) [24–26].

The Harris Hip Score consists of 10 items covering pain, function (containing daily activities and gait), absence of deformity, and range of motion, adding up to a maximum of 100 points (best possible outcome) [25], and has been validated in patients both after traumatic injury and with OA [27, 28]. For all scores, any negative change between baseline and follow-up, indicating worsening of symptoms, was considered as symptomatic progression.

2.4. Statistical Analysis

To compare subject characteristics, Fisher's exact test was used for categorical data, Student's t-test for numerical data, and the exact Mann-Whitney U test for nonparametric testing (exploratory data). Correlations between imaging and clinical parameters were assessed by using Spearman's rank correlation coefficient (primary data). To account for possible effects introduced by differences in demographic parameters and baseline OA severity, generalized linear models (GLM) adjusted for age, sex, BMI and KL were used to assess associations between SHOMRI subscales and clinical worsening with delta values of clinical scores as dependent variables (primary data).

Statistical analyses were performed with SPSS 23 (IBM, Armonk, NY), using a two-sided 0.05 level of significance.

2.5. Reproducibility

Intra-reader reproducibility of the SHOMRI scoring system in this population was assessed twice by two readers (BJS and LN) separately and blinded to each other's results by using 20 randomly selected studies from any of the two time points, with readings being separated by at least two weeks, respectively. To assess inter-reader reproducibility, the results of the first pass of readings by both readers were used. After all readings were recorded, intra-class correlation coefficients (ICC) [29] were calculated for SHOMRI total and subscales. *Intra-reader ICC* were as follows: SHOMRI total – 0.98 (95% confidence interval, 0.95–0.99) and 0.98 (0.94–0.99); cartilage – 0.90 (0.75–0.96) and 0.92 (0.79–0.97); BMEP – 0.98 (0.94–0.99) and 0.97 (0.91–0.99); subchondral cysts – 0.99 (0.98–0.99) and 0.99 (0.99–0.99); labrum – 0.97 (0.93–0.99) and 0.99 (0.96–0.99); paralabral cyst – 1.00 (1.00–1.00) and 1.00

(1.00–1.00); joint effusion – 0.88 (0.69–0.95) and 0.85 (0.65–0.90); loose body – 1.00 (1.00–1.00) and 1.00 (1.00–1.00); ligamentum teres abnormality – 0.94 (0.85–0.98) and 0.95 (0.90–0.97). *Inter-reader ICC* were as follows: SHOMRI total – 0.96 (0.89–0.98); cartilage – 0.89 (0.73–0.96); BMEP – 0.93 (0.82–0.97); subchondral cysts – 0.99 (0.98–0.99); labrum – 0.91 (0.79–0.97); paralabral cyst – 0.91 (0.77–0.96); joint effusion – 0.88 (0.69–0.95); loose body – 1.00 (1.00–1.00); ligamentum teres abnormality – 0.96 (0.89–0.98).

3. Results

3.1. Subjects

Overall, 54 subjects (mean age 47.2 ± 13.2 years; 42.6% women; mean BMI 24.0 ± 3.1 kg/m²) were included in this analysis. Of those, 36 were controls with a KL score of 0 or 1 (mean age 43.7 ± 12.8 years; 50.0% women; mean BMI 23.9 ± 3.2), and 18 were OA subjects with a KL of 2 or 3 (mean age 54.4 ± 11.2 years; 27.8% women; mean BMI 24.1 ± 2.8 ; Table 1). OA subjects were significantly older than controls ($p=0.004$).

Clinical parameters at baseline and their changes over 1.5 years (delta values) are shown in Table 1. At baseline, OA subjects showed significantly worse HOOS subscales for pain (median (IQR), 97.5 (78.5–100) vs. 100 (95–100); $p=0.041$) and function in sport and recreation (93.5 (81–100) vs. 100 (89–100); $p=0.044$) than controls.

At follow-up, worsening of HOOS subscales for pain (50.0 vs. 16.7%; $p=0.010$), symptoms other than pain (50.0 vs. 25.0%; $p=0.047$) and hip-related quality of life (50.0% vs. 22.2%; $p=0.038$) was significantly more often found in OA subjects than in controls. No other significant differences were found in delta values or progression rates of clinical parameters.

3.2. Baseline and Progression of Hip Abnormalities

Baseline SHOMRI subscales and their changes over 1.5 years (delta values) are shown in Table 2. Subjects with radiographic OA had a significantly higher baseline total SHOMRI than controls (median (IQR), 12.5 (6–19.5) vs. 7 (4–13.5); $p=0.024$). Also, OA subjects had significantly higher SHOMRI subscales for cartilage lesions (3 (1–8.5) vs. 1 (0–2.5); $p=0.007$), subchondral cysts (0 (0–1) vs. 0 (0–0); $p=0.025$) and Ligamentum teres abnormalities (1 (0–2) vs. 0 (0–1); $p=0.022$) than controls

At follow-up, progression of SHOMRI for subchondral cysts was significantly more often detected in OA subjects than in controls (16.7% vs. 0%; $p=0.033$; Figure 2). Progression rates of total SHOMRI scores did not significantly differ between groups (Figure 2).

3.3. Association of Imaging Findings and Clinical Features

Baseline Imaging Findings and Clinical Worsening—Of the baseline imaging parameters, the SHOMRI subscale for BMEP significantly correlated with delta HOOS for pain ($r=-0.37$; $p=0.007$; Table 3) and delta HOOS for hip-related quality of life ($r=-0.30$; $p=0.028$). Moreover, a statistical trend was found for the correlation between the baseline SHOMRI subscale for subchondral cysts and the delta of the Harris Hip Score ($r=-0.30$; $p=0.051$), suggesting both a correlation between the presence of BMEP and cysts and

progression of pain and symptoms. Of note, baseline KL scores did not significantly correlate with the change of any of the clinical scores (each, $p>0.05$).

To minimize influence of demographic parameters, generalized linear models (GLM) adjusted for age, sex, BMI, and KL were calculated. The baseline SHOMRI subscale for BMEP was significantly associated with both worsening of HOOS subscales for pain (Beta=0.690 (95% confidence interval, 0.464–0.913); $p=0.018$) and hip-related quality of life (Beta=0.613 (0.380–0.987); $p=0.044$).

Evolution of Imaging Findings and Clinical Worsening—Changes of SHOMRI subscale for subchondral cysts correlated significantly with changes of HOOS symptoms other than pain ($r=-0.30$; $p=0.030$; Table 3); and the incidence of a new paralabral cyst at follow-up significantly correlated with the change of HOOS for activities of daily living ($r=-0.30$; $p=0.030$).

A GLM adjusted for age, sex, BMI, and KL showed a significant association between delta values of the SHOMRI subscale for subchondral cysts and worsening of HOOS symptoms other than pain (Beta=0.224 (95% confidence interval, 0.058–0.859); $p=0.029$).

Evolution of Imaging Parameters in Hips with Versus Without Pain or Symptoms at Baseline—In addition to the tested associations, the evolution of hip imaging findings was specifically assessed in subjects with versus without clinical symptoms at baseline. In generalized linear models adjusted for age, sex, BMI and KL, no significant associations between the delta values of total SHOMRI or any of the subscales and the presence or absence of HOOS hip pain at baseline were found (each, $p>0.05$). Analogously, no significant associations between delta SHOMRI values and the presence or absence of HOOS other symptoms than pain were found (each, $p>0.005$).

4. Discussion

In this study, we analyzed the evolution of hip joint abnormalities over 1.5 years as characterized by the semi-quantitative SHOMRI scoring system [17], and its correlation with clinical parameters. Over 1.5 years, progression of absolute values of SHOMRI total scores and subscales as well as the Harris Hip Score and HOOS subscales was moderate. However, rates of subjects showing any progression of SHOMRI total were substantial (39% over both groups). Moreover, 50% of the OA subjects showed progression of HOOS subscales for pain, symptoms other than pain and hip related quality of life.

Several imaging parameters that have been previously suggested to correlate with clinical features have been assessed at baseline and follow up:

At baseline, more than half of the subjects in both groups had cartilage abnormalities, which is congruent with the findings presented by Lee et al. [17] in their cross-sectional analysis of SHOMRI. However, no significant associations with worsening of any of the clinical features was found, which is in contrast to the cross-sectional analysis by Lee et al. reporting correlations between cartilage lesions and hip symptoms other than pain and activity of daily living subscales. This might be due to the only minimal SHOMRI cartilage progression over

1.5 years in both subject groups, suggesting a longer follow-up period might be necessary to pick up cartilage changes. That no correlations were found between cartilage lesions and the HOOS subscale for pain, is congruent with the cross-sectional analysis by Lee et al. and might suggest that due to the lack of nociceptors, cartilage defects do not immediately generate pain, but might rather be associated with altered biomechanical loading and thus joint degeneration over a longer period of time [30, 31].

Almost all of the subjects had labrum abnormalities, which is also consistent with the findings by Lee et al. and another study in asymptomatic subjects without hip pain, trauma or surgery in their history, that reported a high prevalence of labral tears (69%) [32]. Although labral abnormalities thus seem to be common in subjects without or with only mild hip joint degeneration, the clinical relevance especially of low-grade findings remains unclear. Consequently, at this point we cannot conclude whether SHOMRI might be too sensitive for low-grade abnormalities, or whether the depicted findings are of relevance: they have been shown to correlate with histologically proven labrum degeneration [33], and they might develop into more severe labral pathologies, which can increase joint friction and thus accelerated cartilage degeneration and OA [34]. We therefore believe a longer follow-up period might be necessary to understand the long-term evolution of labral abnormalities.

The extent of baseline BMPEP was associated with worsening of hip pain and hip-related quality of life. This is consistent with findings by a previous cross-sectional analysis in subjects with advanced hip OA, showing that the amount of BMPEP correlates significantly with hip pain [20]. Additionally, the progression of subchondral cysts over 1.5 years significantly correlated with the progression of symptoms other than pain. This confirms recent findings suggesting patients with subchondral cysts have greater disease severity and pain [21]. Of note, it has previously been shown that presence of BMPEP and subchondral cysts are strongly associated in OA [35-37]. Overall, our longitudinal findings emphasize the relevance of imaging findings in subchondral bone for the evolution of hip OA.

Also, the incidence of a new paralabral cyst at follow-up significantly correlated with a worsening of the HOOS subscale for activities of daily living, while baseline SHOMRI for labral pathologies or its progression did not show significant correlations with any of the clinical scores. The latter finding is consistent with the literature, reporting a high prevalence of labral abnormalities in asymptomatic subjects [32]. Paralabral cysts, on the other hand, correlate with labral abnormalities [38, 39], though, our findings suggest that they might be more specific regarding clinical relevance.

Interestingly, no correlations were found between baseline KL scores and development of clinical parameters. Although a KL score of three has previously shown to be a predictive factor for total knee replacement [40], this seemed not to apply to comparatively modest changes of clinical outcome.

The recently published OARSI Clinical Trials Recommendations [10] suggest the use of a semi-quantitative scoring system such as SHOMRI [17], HOAMS [16], or the Hip Inflammation MRI Scoring System (HIMRISS) for active lesions [41] in clinical trials on hip OA subjects. So far, these scoring systems were developed and analyzed cross-

sectionally, and their performance in detecting progression of hip abnormalities over time still needed to be evaluated. In the knee, similar scoring systems such as the Whole-Organ Magnetic Resonance Imaging Score (WORMS) [42], have proven their feasibility and value in large longitudinal studies [15].

In this first longitudinal study using a semiquantitative hip scoring system, prevalence of baseline pathologies was nearly 100% both in subjects with and without radiographic hip OA. Progression rates of subscales for different joint structures varied between groups, however, significant differences were only found for subchondral cysts. This may be due to the following reasons: First, the study population was relatively healthy with only 33% of the subjects presenting a KL score of 2 or 3; and second, the follow-up period of 1.5 years was relatively short. Moreover, sample size was limited with only 54 subjects in total. Also, due to community-based enrollment, subjects with radiographic OA were significantly older than controls. Due to the already relatively small sample size, we decided not to match subjects for demographic parameters. This might have influenced different progression rates of both, imaging and clinical factors, in the groups, since OA is known to be a disease of the aging population [43]. However, subjects with OA were still relatively young with a mean age of 54. In addition, we used statistical models adjusted for baseline demographic parameters, to validate associations between imaging findings and the evolution of clinical scores.

Another limitation of this study is that no arthroscopic or surgical reference standards could be acquired due to the relatively healthy subjects and innate invasiveness of such procedures. Thus, sensitivity and specificity of SHOMRI assessed with a standard of reference other than radiography remain unknown. A study in subjects undergoing MR at 1.5-T and subsequent arthroscopy reported a sensitivity of 86–93% and specificity of 72–88% for cartilage defects and a sensitivity of 96–97% and specificity of 33% for labral tears [44]. We assume that values might be higher in studies performed at 3.0-T, however, this remains unproven both for SHOMRI and other semi-quantitative scoring systems.

In addition, progression rates for clinical scores were based on any negative change being registered as progression, not considering more differentiated thresholds such as minimal detectable change (MDC) or minimal clinically important difference. We decided to work without such thresholds, since MDC for HOOS subscales have only been validated for patients undergoing arthroscopy and with severe OA, and no data exists to date for patients with or without mild OA [45, 46]. Future studies therefore should encompass the recruitment of more advanced stages of OA, and longer observational periods.

5. Conclusions

In conclusion, in this young study population without or with mild to moderate radiographic hip OA, only minimal differences were found between groups regarding the progression of hip abnormalities as assessed by SHOMRI over 1.5 years. Although progression of clinical scores was modest, several significant associations were found between baseline and changes of SHOMRI subscales and worsening of clinical parameters. This supports the

validity of SHOMRI in its first longitudinal assessment and suggests that it is a useful tool for the evaluation of MRI findings in future longitudinal trials on hip OA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

- [1]. Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. *Arthritis Rheum.* 2008; 58:26–35. [PubMed: 18163497]
- [2]. Helmick CG, Felson DT, Lawrence RC, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I. *Arthritis Rheum.* 2008; 58:15–25. [PubMed: 18163481]
- [3]. Murphy LB, Helmick CG, Schwartz TA, et al. One in four people may develop symptomatic hip osteoarthritis in his or her lifetime. *Osteoarthritis Cartilage.* 2010; 18:1372–1379. [PubMed: 20713163]
- [4]. Nho SJ, Kymes SM, Callaghan JJ, Felson DT. The burden of hip osteoarthritis in the United States: epidemiologic and economic considerations. *J Am Acad Orthop Surg.* 2013; 21(Suppl 1):S1–6. [PubMed: 23818185]
- [5]. Philippon MJ, Stubbs AJ, Schenker ML, Maxwell RB, Ganz R, Leunig M. Arthroscopic management of femoroacetabular impingement: osteoplasty technique and literature review. *Am J Sports Med.* 2007; 35:1571–1580. [PubMed: 17420508]
- [6]. Srikanth VK, Fryer JL, Zhai G, Winzenberg TM, Hosmer D, Jones G. A meta-analysis of sex differences prevalence, incidence and severity of osteoarthritis. *Osteoarthritis Cartilage.* 2005; 13:769–781. [PubMed: 15978850]
- [7]. Hawker GA, Stanaitis I. Osteoarthritis year in review 2014: clinical. *Osteoarthritis Cartilage.* 2014; 22:1953–1957. [PubMed: 25456292]
- [8]. Link TM, Stahl R, Woertler K. Cartilage imaging: motivation, techniques, current and future significance. *Eur Radiol.* 2007; 17:1135–1146. [PubMed: 17093967]
- [9]. Roemer FW, Guermazi A. Osteoarthritis Year in Review 2014: imaging. *Osteoarthritis Cartilage.* 2014; 22:2003–2012. [PubMed: 25456295]
- [10]. Gold GE, Cicuttini F, Crema MD, et al. OARSI Clinical Trials Recommendations: Hip imaging in clinical trials in osteoarthritis. *Osteoarthritis Cartilage.* 2015; 23:716–731. [PubMed: 25952344]
- [11]. Siebelt M, Agricola R, Weinans H, Kim YJ. The role of imaging in early hip OA. *Osteoarthritis Cartilage.* 2014; 22:1470–1480. [PubMed: 25278058]
- [12]. Chang CY, Huang AJ. MR imaging of normal hip anatomy. *Magn Reson Imaging Clin N Am.* 2013; 21:1–19. [PubMed: 23168179]
- [13]. Link TM. MR imaging in osteoarthritis: hardware, coils, and sequences. *Radiol Clin North Am.* 2009; 47:617–632. [PubMed: 19631072]
- [14]. Link TM, Schwaiger BJ, Zhang AL. Regional Articular Cartilage Abnormalities of the Hip. *AJR Am J Roentgenol.* 2015; 205:502–512. [PubMed: 26295636]

- [15]. Guermazi A, Roemer FW, Haugen IK, Crema MD, Hayashi D. MRI-based semiquantitative scoring of joint pathology in osteoarthritis. *Nat Rev Rheumatol*. 2013; 9:236–251. [PubMed: 23321609]
- [16]. Roemer FW, Hunter DJ, Winterstein A, et al. Hip Osteoarthritis MRI Scoring System (HOAMS): reliability and associations with radiographic and clinical findings. *Osteoarthritis Cartilage*. 2011; 19:946–962. [PubMed: 21550411]
- [17]. Lee S, Nardo L, Kumar D, et al. Scoring hip osteoarthritis with MRI (SHOMRI): A whole joint osteoarthritis evaluation system. *J Magn Reson Imaging*. 2014
- [18]. Ilizaliturri VM Jr, Byrd JW, Sampson TG, et al. A geographic zone method to describe intra-articular pathology in hip arthroscopy: cadaveric study and preliminary report. *Arthroscopy*. 2008; 24:534–539. [PubMed: 18442685]
- [19]. Baum T, Joseph GB, Arulanandan A, et al. Association of magnetic resonance imaging-based knee cartilage T2 measurements and focal knee lesions with knee pain: data from the Osteoarthritis Initiative. *Arthritis Care Res (Hoboken)*. 2012; 64:248–255. [PubMed: 22012846]
- [20]. Taljanovic MS, Graham AR, Benjamin JB, et al. Bone marrow edema pattern in advanced hip osteoarthritis: quantitative assessment with magnetic resonance imaging and correlation with clinical examination, radiographic findings, and histopathology. *Skeletal Radiol*. 2008; 37:423–431. [PubMed: 18274742]
- [21]. Tanamas SK, Wluka AE, Pelletier JP, et al. The association between subchondral bone cysts and tibial cartilage volume and risk of joint replacement in people with knee osteoarthritis: a longitudinal study. *Arthritis Res Ther*. 2010; 12:R58. [PubMed: 20356405]
- [22]. Kumar D, Wyatt C, Chiba K, et al. Anatomic correlates of reduced hip extension during walking in individuals with mild-moderate radiographic hip osteoarthritis. *J Orthop Res*. 2015
- [23]. Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthritis. *Ann Rheum Dis*. 1957; 16:494–502. [PubMed: 13498604]
- [24]. Klassbo M, Larsson E, Mannevik E. Hip disability and osteoarthritis outcome score. An extension of the Western Ontario and McMaster Universities Osteoarthritis Index. *Scand J Rheumatol*. 2003; 32:46–51. [PubMed: 12635946]
- [25]. Nilsson A, Bremander A. Measures of hip function and symptoms: Harris Hip Score (HHS), Hip Disability and Osteoarthritis Outcome Score (HOOS), Oxford Hip Score (OHS), Lequesne Index of Severity for Osteoarthritis of the Hip (LISOH), and American Academy of Orthopedic Surgeons (AAOS) Hip and Knee Questionnaire. *Arthritis Care Res (Hoboken)*. 2011; 63(Suppl 11):S200–207. [PubMed: 22588745]
- [26]. Nilsson AK, Lohmander LS, Klassbo M, Roos EM. Hip disability and osteoarthritis outcome score (HOOS)--validity and responsiveness in total hip replacement. *BMC Musculoskelet Disord*. 2003; 4:10. [PubMed: 12777182]
- [27]. Harris WH. Traumatic arthritis of the hip after dislocation and acetabular fractures: treatment by mold arthroplasty. An end-result study using a new method of result evaluation. *J Bone Joint Surg Am*. 1969; 51:737–755. [PubMed: 5783851]
- [28]. Hoeksma HL, Van Den Ende CH, Rooday HK, Heering A, Breedveld FC. Comparison of the responsiveness of the Harris Hip Score with generic measures for hip function in osteoarthritis of the hip. *Ann Rheum Dis*. 2003; 62:935–938. [PubMed: 12972470]
- [29]. Shrout PE, Fleiss JL. Intraclass correlations: uses in assessing rater reliability. *Psychol Bull*. 1979; 86:420–428. [PubMed: 18839484]
- [30]. Dieppe PA, Lohmander LS. Pathogenesis and management of pain in osteoarthritis. *Lancet*. 2005; 365:965–973. [PubMed: 15766999]
- [31]. Hunter DJ, McDougall JJ, Keefe FJ. The symptoms of osteoarthritis and the genesis of pain. *Rheum Dis Clin North Am*. 2008; 34:623–643. [PubMed: 18687276]
- [32]. Register B, Pennock AT, Ho CP, Strickland CD, Lawand A, Philippon MJ. Prevalence of abnormal hip findings in asymptomatic participants: a prospective, blinded study. *Am J Sports Med*. 2012; 40:2720–2724. [PubMed: 23104610]
- [33]. Kanazaki S, Yamamoto A, Nakamura S, Osawa M, Matsushita T. Magnetic resonance imaging findings compared with histological findings of the labrum in hip osteoarthritis. *Skeletal Radiol*. 2015; 44:767–775. [PubMed: 25616616]

- [34]. Song Y, Ito H, Kourtis L, Safran MR, Carter DR, Giori NJ. Articular cartilage friction increases in hip joints after the removal of acetabular labrum. *J Biomech.* 2012; 45:524–530. [PubMed: 22176711]
- [35]. Li G, Yin J, Gao J, et al. Subchondral bone in osteoarthritis: insight into risk factors and microstructural changes. *Arthritis Res Ther.* 2013; 15:223. [PubMed: 24321104]
- [36]. Crema MD, Roemer FW, Zhu Y, et al. Subchondral cystlike lesions develop longitudinally in areas of bone marrow edema-like lesions in patients with or at risk for knee osteoarthritis: detection with MR imaging--the MOST study. *Radiology.* 2010; 256:855–862. [PubMed: 20530753]
- [37]. Link TM, Li X. Bone marrow changes in osteoarthritis. *Semin Musculoskelet Radiol.* 2011; 15:238–246. [PubMed: 21644197]
- [38]. Rakhra KS. Magnetic resonance imaging of acetabular labral tears. *J Bone Joint Surg Am.* 2011; 93(Suppl 2):28–34. [PubMed: 21543685]
- [39]. Magee T, Hinson G. Association of paralabral cysts with acetabular disorders. *AJR Am J Roentgenol.* 2000; 174:1381–1384. [PubMed: 10789799]
- [40]. Gossec L, Tubach F, Baron G, Ravaud P, Logeart I, Dougados M. Predictive factors of total hip replacement due to primary osteoarthritis: a prospective 2 year study of 505 patients. *Ann Rheum Dis.* 2005; 64:1028–1032. [PubMed: 15640268]
- [41]. Jaremko JL, Lambert RG, Zubler V, et al. Methodologies for semiquantitative evaluation of hip osteoarthritis by magnetic resonance imaging: approaches based on the whole organ and focused on active lesions. *J Rheumatol.* 2014; 41:359–369. [PubMed: 24241486]
- [42]. Peterfy CG, Guermazi A, Zaim S, et al. Whole-Organ Magnetic Resonance Imaging Score (WORMS) of the knee in osteoarthritis. *Osteoarthritis Cartilage.* 2004; 12:177–190. [PubMed: 14972335]
- [43]. Felson DT, Lawrence RC, Dieppe PA, et al. Osteoarthritis: new insights. Part 1: the disease and its risk factors. *Ann Intern Med.* 2000; 133:635–646. [PubMed: 11033593]
- [44]. Mintz DN, Hooper T, Connell D, Buly R, Padgett DE, Potter HG. Magnetic resonance imaging of the hip: detection of labral and chondral abnormalities using noncontrast imaging. *Arthroscopy.* 2005; 21:385–393. [PubMed: 15800516]
- [45]. Naylor JM, Hayen A, Davidson E, et al. Minimal detectable change for mobility and patient-reported tools in people with osteoarthritis awaiting arthroplasty. *BMC Musculoskelet Disord.* 2014; 15:235. [PubMed: 25015083]
- [46]. Kemp JL, Collins NJ, Roos EM, Crossley KM. Psychometric properties of patient-reported outcome measures for hip arthroscopic surgery. *Am J Sports Med.* 2013; 41:2065–2073. [PubMed: 23835268]

Figure 1.

Coronal 2D fast spin-echo intermediate-weighted sequence of the left hip of a 50-year-old male with hip OA showing regional subdivision of acetabulum and femoral head according to SHOMRI and the following findings: Full-thickness defects in the superolateral acetabular cartilage (arrow), the lateral femoral cartilage (#), and a partial thickness defect in the superolateral femoral cartilage (*). Labral tear (dashed arrow).

Figure 2.

Progression rates of SHOMRI total and subscales for the different cohorts illustrating higher progression rates in subjects with OA and borderline OA subjects. * Significantly higher progression rate of SHOMRI for subchondral cysts in OA subjects compared to controls ($p=0.033$).

Table 1

Baseline demographic parameters and baseline clinical scores and their evolution over 1.5 years of subjects without (KL 0/1) versus with radiographic evidence of hip osteoarthritis (KL 2/3)

Parameter		Kellgren/Lawrence score (KL)		P-value *
		KL 0/1	KL 2/3	
Group size (n)		36	18	
Female sex (n; % of group)		18 (50.0%)	5 (27.8%)	.120
Age (years; mean $\pm$ SD)		43.7 $\pm$ 12.8	54.4 $\pm$ 11.2	.004
BMI (kg/m ² ; mean $\pm$ SD)		23.9 $\pm$ 3.2	24.1 $\pm$ 3.2	.834
Hip side (left; n; % of group)		18 (50.0%)	9 (50.0%)	1.000
Baseline Harris Hip Score	(Median; IQR)	100 (96–100)	97 (88.5–100)	<i>0.057</i>
Baseline HOOS pain score	(Median; IQR)	100 (95–100)	97.5 (78.5–100)	0.041
Baseline HOOS other symptoms	(Median; IQR)	97.5 (90–100)	95 (83.5–100)	0.284
Baseline HOOS activities of daily living	(Median; IQR)	100 (97–100)	95 (83.5–100)	0.114
Baseline HOOS sports	(Median; IQR)	100 (89–100)	93.5 (81–100)	0.044
Baseline HOOS quality of life	(Median; IQR)	100 (81–100)	90 (81–100)	0.160
Evolution of Harris Hip Score	Delta values (median; IQR)	0 (–0.5–1)	0 (–6.5–1.5)	0.652
	Worsening (n; % of group)	7 (24.1%)	6 (37.5%)	0.494
Evolution of HOOS pain score	Delta values (median; IQR)	0 (0–0)	–1.5 (–7.5–8.5)	0.562
	Worsening (n; % of group)	6 (16.7%)	9 (50%)	0.010
Evolution of HOOS other symptoms score	Delta values (median; IQR)	0 (–3.5–5)	–2.5 (–10–5)	0.295
	Worsening (n; % of group)	9 (25.0%)	9 (50.0%)	0.047
Evolution of HOOS activities of daily living	Delta values (median; IQR)	0 (0–0)	0 (–3–2)	0.419
	Worsening (n; % of group)	7 (19.4%)	8 (44.4%)	<i>0.053</i>
Evolution of HOOS sports	Delta values (median; IQR)	0 (0–0)	0 (–18.5–9.5)	0.724
	Worsening (n; % of group)	8 (22.2%)	6 (33.3%)	0.512
Evolution of HOOS quality of life	Delta values (median; IQR)	0 (0–0)	–3.5 (–15.5–14)	0.502
	Worsening (n; % of group)	8 (22.2%)	9 (50.0%)	0.038

* Fisher's exact test was used for categorical data presence/absence of abnormalities or progression yes/no), Student's t test for numerical data, and the exact Mann-Whitney U test for nonparametric testing. Worsening of clinical scores is any change <0 of absolute Harris Hip Score or HOOS subscales over time.

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Table 2

Total SHOMRI at baseline and selected subscales and their delta values and progression rates over 1.5 years in subjects without (KL 0/1) versus with radiographic OA (KL 2/3)

Parameter		Kellgren/Lawrence score (KL)		P-value*
		KL 0/1	KL 2/3	
Baseline SHOMRI total	Values (median; IQR)	7 (4–13.5)	12.5 (6–19.5)	0.024
Baseline SHOMRI cartilage	Values (median; IQR)	1 (0–2.5)	3 (1–8.5)	0.007
Baseline SHOMRI BMEP**	Values (median; IQR)	0 (0–0)	0 (0–0.5)	0.278
Baseline SHOMRI subchondral cysts	Values (median; IQR)	0 (0–0)	0 (0–1)	0.025
Baseline SHOMRI labrum	Values (median; IQR)	5.5 (3–9.5)	6 (5–10)	0.085
Baseline SHOMRI Lig. teres	Values (median; IQR)	0 (0–1)	1 (0–2)	0.022
Evolution of SHOMRI total	Delta values (median; IQR)	0 (0–1)	0 (0–2)	0.615
Evolution of SHOMRI cartilage	Delta values (median; IQR)	0 (0–0)	0 (0–0)	0.509
Evolution of SHOMRI BMEP**	Delta values (median; IQR)	0 (0–0)	0 (0–0)	0.535
Evolution of SHOMRI subchondral cysts	Delta values (median; IQR)	0 (0–0)	0 (0–0)	0.112
Evolution of SHOMRI labrum	Delta values (median; IQR)	0 (0–0)	0 (0–0)	0.593
Delta SHOMRI ligamentum teres	Values (median; IQR)	0 (0–0)	0 (0–0)	0.408

* Fisher's exact test was used for categorical data (presence/absence of abnormalities or progression yes/no), and the exact Mann-Whitney U test for nonparametric testing. Abnormality present applies to any SHOMRI sub-score >1. SHOMRI progression is any change >0 over time.

** Bone-marrow edema pattern.

Table 3

Selected correlations between baseline Kellgren-Lawrence score, SHOMRI total and subscales at baseline and follow up, and change of clinical parameters of 1.5 years. Complete correlations in supplemental material.

		Delta Harris Hip Score	Delta HOOS pain	Delta HOOS other symptoms	Delta HOOS activities of daily living	Delta HOOS sport/rec	Delta HOOS quality of life
Baseline Kellgren- Lawrence	Correlation Coefficient	-.168	-.087	-.068	-.079	-.012	-.023
	Sig. (2- tailed)	.271	.531	.623	.570	.932	.870
Baseline SHOMRI BMEP	Correlation Coefficient	.002	-.365 *	-.064	-.241	-.131	-.299 *
	Sig. (2- tailed)	.990	.007	.648	.079	.345	.028
Delta SHOMRI ligamentum teres	Correlation Coefficient	-.117	-.185	-.012	-.062	.076	-.306 *
	Sig. (2- tailed)	.445	.181	.934	.654	.587	.024
Delta SHOMRI subchondral cysts	Correlation Coefficient	.118	-.182	-.296 *	-.096	.044	-.197
	Sig. (2- tailed)	.440	.188	.030	.490	.754	.153
Delta SHOMRI paralabral cyst	Correlation Coefficient	-.199	-.155	-.063	-.296 *	-.204	-.239
	Sig. (2- tailed)	.190	.262	.651	.030	.139	.081

* Significant at p<0.05