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Abnormal Joint Loading During Gait in Persons With Hip Osteoarthritis Is Associated With Symptoms and Cartilage Lesions

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Abstract

BACKGROUND: Hip joint loading in persons with hip osteoarthritis (OA) is not well studied, and its associations with symptoms and lesions are unknown.

OBJECTIVES: To determine whether hip joint loading differs between people with and without radiographic hip OA, and to identify its associations with patients' symptoms and cartilage morphology.

METHODS: Forty-eight patients (28 male; mean ± SD age, 56.0 ± 12.2 years) with hip OA and 95 controls (40 male; age, 43.2 ± 13.6 years) participated in this cross-sectional analysis. Pelvic radiographs, questionnaires, magnetic resonance imaging (MRI), and gait analysis were conducted. The Hip disability and Osteoarthritis Outcome Score (HOOS) was used to assess symptoms. Cartilage morphology was graded on MRI scans using the Scoring Hip Osteoarthritis with Magnetic Resonance Imaging (SHOMRI) system. Biomechanical variables included peak external hip joint moment (Newton meters per kilogram) and moment impulses (Newton meters times milliseconds per kilogram) in all planes. Generalized estimating equations were used to compare the biomechanical characteristics between groups. In the patients with OA, associations of moment impulses with HOOS and SHOMRI scores were assessed with partial correlations.

RESULTS: The OA group exhibited higher peak external hip flexion and adduction moments ($P<.001$) and higher hip flexion, adduction, and external rotation moment impulses ($P = .001–.039$). Increased hip flexion moment impulses were correlated with worse HOOS subscale

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scores (r = −0.361 to −0.424, P<.05) and worse femoral SHOMRI grades (ρ = 0.256–0.315, P<.05). Increased hip external rotation moment impulses were correlated with worse femoral SHOMRI grades (ρ = 0.283–0.372, P<.05).

CONCLUSION: Persons with hip OA exhibited abnormally high hip joint loads during walking, and high loads were associated with worse self-reported symptoms and cartilage morphology.

Keywords

HOOS; increased hip moment; moment impulse; SHOMRI

Hip osteoarthritis (OA) is the leading cause of disability in middle-aged and elderly people, with a 4.2% prevalence of symptomatic hip OA and a 19.6% prevalence of radiographic hip OA in persons 50 years of age and older.[18] Moreover, the estimate of total hip replacements performed each year in the United States corresponds to 2.5 million individuals, with most of them for treatment of hip OA.[30]

While gait is one of the most common functional activities of daily living, gait assessments in the hip OA population, specifically related to joint kinetics, have not provided consistent results across studies. Most studies have reported reduced hip joint moments during walking in persons with hip OA,[5,6,9,25,40] and other studies have reported greater external hip flexion and hip adduction moments.[5,19] Because joint moments are largely dependent on walking speed, variation in controlling walking speed may have accounted for the differences in hip joint moments across studies. Assessment of 3-D hip joint kinetics under a controlled speed is crucial to provide a clear understanding of the effects of hip OA on lower extremity mechanics and to better understand the biomechanical factors associated with morphological hip pathologies. Furthermore, biomechanical parameters such as hip joint kinetics are potentially modifiable, offering researchers and clinicians an opportunity to develop disease-modifying treatment strategies.

Due to the weight-bearing nature of the hip joint, it is commonly hypothesized that mechanical overloading of the hip joint is related to the pathogenesis of hip pain and degeneration. A recent study[19] that examined the association of gait mechanics with hip OA using sagittal plane kinematics and magnetic resonance imaging (MRI) assessment of structural abnormalities found that greater peak hip flexion angle was associated with greater severity of femoral cartilage lesions.[19] However, clinical symptoms (eg, pain and function) are often not associated with imaging findings (eg, radiographic signs of OA and cartilage lesions shown on MRI scans).[1,2,12,39] It is unknown whether mechanical overloading of the hip joint is associated with patients' symptoms, and whether such an association exists in all 3 kinematic planes. Therefore, a combination of gait analysis, evaluation of patient symptoms, and MRI assessment may provide a better clinical understanding of the interactions between joint mechanical loading and degeneration.

The purpose of this study was (1) to determine whether 3-D external hip joint moments and moment impulses during walking differ between people with and without radiographic hip OA, and (2) to identify possible associations between hip joint loading and patients' self-reported clinical symptoms and MRI-based semi-quantitative measures of acetabular and femoral cartilage lesion abnormalities.

METHODS

The reporting of this study adheres to the Strengthening the Reporting of Observational Studies in Epidemiology guidelines.[37]

Participants

Participants were recruited from 2 longitudinal natural history cohort studies of people with hip OA; the data included in this study comprise the baseline cross-sectional assessment. The first study evaluated a single limb in persons with hip OA,[20] while the second study included both limbs (unpublished data). Participants in both studies were recruited from the community using flyers and advertisements. A screening X-ray was performed to determine the eligibility of the participants, as individuals with severe hip OA were excluded from the studies. Participants were excluded if any of the following criteria were present: (1) contraindications to MRI, (2) a Kellgren-Lawrence (KL) grade equal to 4, (3) previous hip trauma, (4) joint replacement of any lower extremity joint, (5) pain or existing OA at any other lower extremity joint, (6) self-reported inflammatory arthritis, and (7) any conditions that limit the ability to walk. Both cohort studies were conducted at the University of California San Francisco, but at different times (between 2011 and 2017). There were some additional differences in the functional testing performed that do not affect the current study. Given these numerous similarities, we combined the 2 cohorts for the current investigation.

One hundred ninety-nine people were eligible for the study, with 13 people declining to participate. As lower extremity joint loading was the primary outcome in this study, those who did not undergo gait analysis were removed from the overall cohort for this particular study, which resulted in further excluding 43 potential participants, due to either their inability to perform the biomechanical task or technical problems during data analysis. In total, 48 patients (28 male; average ± SD age, 56.0 ± 12.2 years; body mass index [BMI], 24.3 ± 3.5 kg/m^2) with definite radiographic hip OA (27 patients with KL grade 2, 21 patients with KL grade 3) and 95 random controls without or with doubtful radiographic OA (40 male; average ± SD age, 43.2 ± 13.6 years; BMI, 24.0 ± 3.1 kg/m^2; 40 participants with KL grade 0, 55 participants with KL grade 1) were included in the final analysis, resulting in 71 hips in the OA group and 143 hips in the control group. Prior to testing, the study protocol was approved by the Institutional Review Board at the University of California San Francisco and all participants provided written informed consent.

Radiographic Imaging

To determine radiographic signs of hip OA, an anteroposterior weight-bearing pelvic radiograph was obtained. All radiographs were assessed using the KL grading system[16] by an experienced musculoskeletal radiologist. Study participants with mild to moderate hip OA were defined as those with KL grades of 2 or 3, while control participants without or with doubtful OA were defined as those with KL grades of 0 or 1.

Patient-Reported Symptoms

All participants completed the Hip disability and Osteoarthritis Outcome Score (HOOS). The HOOS questionnaire consists of 5 subscales: pain, other symptoms, function in daily living, function in sport and recreation, and hip-related quality of life. A normalized score (100 indicating no symptoms and 0 indicating severe symptoms) was calculated for each subscale. For the purpose of this study, the dimensions of pain and function in daily living were used to determine patients' self-reported symptoms. Patients who were enrolled bilaterally were asked to complete the HOOS questionnaire for both hips.

MRI Assessment

All imaging was performed using a 3.0-T MR750w scanner (General Electric, Boston, MA) with an 8-channel cardiac coil (General Electric). T2-based MRI scans were used to assess morphological abnormalities in the articular cartilage of the hip joint, using an arthroscopically validated, semi-quantitative scoring system (Scoring Hip Osteoarthritis with Magnetic Resonance Imaging [SHOMRI])[21,27] evaluated by an experienced, board-certified radiologist. High intra-reader and interreader agreement were previously reported in our group (intra-reader intraclass correlation coefficient [ICC] = 0.93–0.98; interreader ICC = 0.91–0.94).[21] The MRI protocol included intermediate-weighted, fat-suppressed, fast spin-echo sequences in the sagittal, oblique coronal, and oblique axial planes, with a repetition time of 2400 to 3700 milliseconds, an echo time of 60 milliseconds, a field of view of 14 to 20 cm, a matrix size of 288 × 224 pixels, and a slice thickness of 3 to 4 mm.

To assess cartilage morphology, the hip joint was divided into 10 anatomical subregions. Four subregions for acetabular cartilage (superolateral, superomedial, anterior, and posterior) and 6 subregions for femoral cartilage (lateral, superolateral, superomedial, inferomedial, anterior, and posterior) were utilized (FIGURE 1). The 10 subregions were graded separately for cartilage lesions on a 3-point scale, with 0 representing no lesion, 1 partial-thickness cartilage loss, and 2 full-thickness cartilage loss. The total cartilage SHOMRI grade was calculated for the femur and acetabulum separately, by adding the grades in the corresponding 6 and 4 subregions, respectively.

Gait Analysis

All biomechanical testing was performed in the Human Performance Center at the University of California San Francisco. Three-dimensional lower extremity position data were collected at 250 Hz using a 10-camera motion-analysis system (Vicon; Oxford Metrics, Yarnton, UK), while ground reaction force (GRF) data were collected with 2 in-ground force plates (Advanced Mechanical Technology, Inc, Watertown, MA) simultaneously at a rate of 1000 Hz.

Nineteen reflective markers were placed on the following bony landmarks: first and fifth metatarsal heads, medial and lateral malleoli, medial and lateral femoral epicondyles, greater trochanters, iliac crests, anterior superior iliac spines, and the L5-S1 junction. In addition, segment tracking was performed using marker clusters mounted on semi-rigid plastic plates placed on the lateral surfaces of the participants' thighs, shanks, and heel counters of the shoes. The participants wore nonrestrictive clothing and standardized athletic shoes (New

Balance 880; New Balance, Boston, MA) to minimize the impact of shoe types on gait biomechanics. Following a 1-second standing calibration trial, participants were asked to perform over-ground walking at a fixed speed of 1.35 m/s, as this is the mean of the average smooth-surface walking speeds of men and women.[29] Practice trials were permitted to allow participants to become familiar with the procedures. Five successful walking trials were obtained. A successful trial was defined as the foot of the tested limb falling within the borders of the force plate and the speed falling within ±5% of the fixed speed. Two timing gates were used to confirm the speed of each trial.

Marker position and GRF data were low-pass filtered with fourth-order, zero-lag Butterworth filters at cutoff frequencies of 6 Hz and 50 Hz, respectively, using Visual3D (C-Motion, Inc, Germantown, MD). A 7-segment musculoskeletal model consisting of the pelvis, bilateral thighs, shanks, and feet was created with Visual3D for each participant, using the respective standing calibration trial. The pelvic segment was modeled as cylinders, and the lower extremity segments were modeled as frusta of cones. The local orthogonal coordinate systems of the pelvis, thigh, shank, and foot segments were derived from the standing calibration trial. An unweighted least-squares method was used to estimate segment position and orientation.[34] Joint kinematics were calculated using a Cardan rotation sequence in an order of flexion/extension, abduction/adduction, and internal/external rotation, representing sagittal, frontal, and transverse plane rotations, respectively. Joint moments, expressed as external moments, were derived from standard inverse dynamics equations and normalized by body mass (Newton meters per kilogram). Joint moment impulses (Newton meters times milliseconds per kilogram) were calculated as the time integral of the joint moment. All gait kinetics were quantified during the stance phase of gait. The stance phase of gait was defined as from when the foot hit the ground and the vertical GRF was greater than 20 N (initial contact) to when the vertical GRF was less than 20 N (toe-off). Variables of interest included peak external hip moments and moment impulses in the sagittal, frontal, and transverse planes.

Statistical Analysis

Chi-square and independent *t* tests were used to compare demographics, HOOS subscale scores, and SHOMRI grades between the OA and control groups. Generalized estimating equations (GEEs) were used to evaluate differences in hip joint kinetics between groups. The GEE method is able to account for the correlation between the left and right hips in the same person.[22] The GEE analysis was performed with covariates of age, sex, and BMI, as these demographics are known to influence gait kinetics.[3]

In participants with hip OA, associations of hip moment impulses with HOOS scores and SHOMRI grades were assessed using partial Pearson (r) and Spearman (rho) correlation coefficients, respectively, adjusting for age, sex, and BMI. External hip moment impulses were analyzed but peak moments were not, because these variables showed strong correlations (r = 0.62–0.92) among one another and moment impulse was a more sensitive measure, based on our primary analysis. In addition, reducing the variables for correlational-based analyses helped to avoid potential type I error. Participants with missing data were excluded from the analysis. All analyses were performed using SPSS Version 23 (IBM Corporation, Armonk, NY), and the alpha value was set at .05.

RESULTS

Participant characteristics are presented in TABLE 1. Significant group differences were found for age ($P<.001$), HOOS function in daily living score ($P = .012$), and SHOMRI cartilage grades, as participants with hip OA were older, had worse HOOS function in daily living subscale scores, as well as greater severity of cartilage lesions in all subregions of the acetabulum and most subregions of the femur, except for the superomedial and inferomedial subregions ($P<.001$–.050). Representative images exhibiting cartilage morphology of a control participant and a patient with OA are presented in FIGURE 2.

When comparing hip joint kinetics between groups, the OA group exhibited significantly higher peak external hip flexion (OA, 0.97 ± 0.18 Nm/kg; control, 0.84 ± 0.30 Nm/kg; $\beta = -0.135$, $P<.001$) and adduction (OA, 0.97 ± 0.16 Nm/kg; control, 0.86 ± 0.27 Nm/kg; $\beta = -0.108$, $P<.001$) moments, as well as higher external hip flexion (OA, 121.42 ± 43.77 Nm · ms/kg; control, 99.76 ± 48.73 Nm · ms/kg; $\beta = -21.658$, $P = .009$), adduction (OA, 356.95 ± 69.01 Nm · ms/kg; control, 312.40 ± 107.58 Nm · ms/kg; $\beta = 44.552$, $P = .001$), and external rotation (OA, 22.94 ± 20.18 Nm · ms/kg; control, 15.85 ± 16.62 Nm · ms/kg; $\beta = -7.093$, $P = .039$) moment impulses (TABLE 2, FIGURE 3).

In the OA group, partial correlations revealed that among all the variables tested, only external hip flexion and external rotation moment impulses were associated with HOOS scores and SHOMRI grades; therefore, results were only presented for those 2 variables in TABLE 3. There were missing data from 2 participants for the outcomes of the HOOS and SHOMRI; those participants were excluded from further correlation analysis. When adjusted for age, sex, and BMI, increased external hip flexion moment impulses were correlated with worse scores on the HOOS pain ($r = -0.361$, $P = .003$) and function in daily living ($r = -0.424$, $P<.001$) subscales. For SHOMRI grading, increased external hip flexion moment impulses were correlated with greater severity of femoral cartilage lesions in the lateral ($\rho = 0.315$, $P = .009$) and superomedial ($\rho = 0.256$, $P = .035$) subregions, as well as total femoral cartilage grades ($\rho = 0.275$, $P = .023$), whereas increased hip external rotation moment impulses were correlated with greater severity of acetabular cartilage lesion in the posterior subregion ($\rho = 0.283$, $P = .019$), femoral cartilage lesions in the anterior ($\rho = 0.372$, $P = .002$) and posterior ($\rho = 0.313$, $P = .009$) subregions, and total femoral cartilage grades ($\rho = 0.334$, $P = .005$).

DISCUSSION

The primary purpose of this study was to evaluate whether participants with radiographic hip OA exhibited altered hip joint moments and moment impulses during walking compared to those without radiographic OA. In addition, we sought to assess the potential associations between hip joint kinetics, symptoms, and cartilage morphology. Our results revealed that peak external hip flexion and adduction moments were 11% to 13% higher in the hip OA group compared to the control group. In addition, hip flexion, adduction, and external rotation moment impulses were 12% to 30% higher in the hip OA group. Increased hip joint loading in participants with hip OA was found to be associated with self-reported hip joint pain and function, as well as with increased severity of hip joint cartilage lesions. These results support our hypotheses that patients with hip OA exhibit abnormal hip joint kinetics during walking and that these abnormal hip joint mechanics are associated with hip joint symptoms and cartilage morphology.

Our results are similar to previous studies that reported an average of 17% increased hip flexion[19] and adduction[5] moments in participants with hip OA. It is also not surprising to find higher hip moment impulses in all planes, as moment impulse is the time-based integral of the joint moment. By considering both the magnitude and duration of the joint moment, the moment impulse provides a cumulative measure of loading during each stride. This is clinically relevant because mechanical overloading has been known to lead to joint pain and degeneration,[14,28] which is further supported by our secondary analysis that higher moment impulses were associated with patients' symptoms and cartilage morphology.

Previous studies that examined gait biomechanics in persons with hip OA have not been consistent about instructing their participants to maintain a specific speed during gait assessment, leading to great discrepancies in study results. It should be noted that patients with hip OA tend to (but not always) walk slower (average, 1.10 m/s) when compared to healthy controls (average, 1.35 m/s)[5,25,40]; nevertheless, the speed we chose was not a fast gait speed but a very comfortable, purposeful speed. Controlling for gait speed allowed us to make direct kinematic and kinetic comparisons, as it is known that gait speed will affect these dramatically.[3] It is beyond the scope of the present study to determine whether persons with hip OA, when instructed to walk at a preferred speed, would overload their hip joint or whether reduced walking speed may be a compensatory strategy to minimize loading and pain, as previously suggested.[4,32] Regardless, speed constraint might have limited the generalizability of our results, but also suggested that persons with hip OA, when instructed to walk at the same speed as healthy individuals, exhibit hip overloading.

The secondary purpose of the study was to determine the association of hip moment impulses with self-reported symptoms and MRI evidence of acetabular and femoral cartilage lesions. Similar to previous work, the hip OA group exhibited worse HOOS function in daily living scores and worse SHOMRI grades in acetabular and femoral cartilage.[19] Total SHOMRI grades revealed greater lesions within the entire acetabular and femoral cartilage areas in patients with OA. Nevertheless, subregional analysis provides insight into the localization of the lesions in these persons. The patients with hip OA exhibited greater severities of cartilage lesions in all subregions of the acetabulum, as well as most subregions of the femur, except for the superomedial and inferomedial subregions.

Similar to other hip pathology studies, our study found that in participants with hip OA, greater external hip flexion moment impulse was moderately associated with worse HOOS pain and HOOS function in daily living subscale scores.[31] The hip flexion moment impulse occurs during the first half of the stance phase, whereby the hip extensors are required to act eccentrically in order to absorb the external forces applied. Muscle strength deficits have been observed in persons with hip OA,[15,23] which may help to explain the greater external hip flexion moment impulse observed in the hip OA group in the current study. Because this was a cross-sectional study, we are unable to determine the causal relationship between mechanical overloading and presence of radiographic hip OA. Nevertheless, moderate correlations between mechanical overloading and self-reported symptoms correspond to earlier studies that hypothesized that overloading would lead to joint pain and even joint degeneration.[14,28]

In this cohort of patients with hip OA, severity of femoral cartilage lesions was weakly to moderately associated with external hip flexion and external rotation moment impulses. During gait, the primary weight-bearing portions of the hip joint are located at the anterior, posterior, and superior regions.[11,33,36] With the advantage of using subregional analysis,[10,24] the current study revealed that higher flexion moment impulses were associated with increased severity of femoral cartilage lesions in the lateral and superomedial regions, corresponding to the sites where high loading patterns are commonly observed within the hip joint. On the other hand, higher external rotation moment impulses were associated with increased severity of femoral cartilage lesions within the anterior and posterior regions of the hip joint, and with acetabular cartilage lesions in the posterior region. From an arthrokinematics standpoint, a joint moment applied in the transverse plane results in an anterior-to-posterior glide of the femoral head on the acetabulum.[26] More specifically, the higher external rotation moment impulse may be applying abnormal loading patterns to the anterior and posterior regions of the femoral head, which may result in increased severity of cartilage lesions within these specific regions in the hip OA group. Our findings suggest that abnormally higher hip joint loads may be risk factors for the development of hip OA, but longitudinal assessment is needed to further elucidate this relationship.

Identifying the biomechanical factors associated with persons with hip OA offers some direction for load modification in the presence of specific patterns of cartilage lesion development.[7,8,13,17,35] These biomechanical factors are potentially modifiable, offering researchers an opportunity to develop disease-modifying treatment strategies. For instance, a previous study reported that participants with radiographic hip OA exhibited higher peak hip flexion and lower peak hip extension during walking.[19,20] With higher peak hip flexion during early stance, it is likely that an increased moment arm at the hip joint results in this higher external hip flexion moment. Interventions that modify hip kinematics and joint loading may be beneficial for the hip OA population.

There are several limitations that need to be considered when interpreting the findings of the current study. Due to the cross-sectional design of the study, we are not able to elucidate whether 11% to 30% increased hip joint loading leads to hip cartilage degeneration and eventually OA, or whether the magnitudes exceeded clinically relevant differences. Longitudinal studies are needed to determine whether these biomechanical markers are risk factors for structural and symptomatic progression of hip OA. A fixed walking speed was used in this study, and it should be noted that patients with hip OA, when asked to walk at a self-selected speed, ambulated at a slower speed compared to healthy controls.[5,25,40] Also, patients with hip OA were older than the controls, which could have influenced biomechanical differences. Similarly, this holds true for the slight difference in sex distribution between groups. To minimize the confounding effects, age and sex were considered as covariates in all statistical analyses. Nevertheless, caution should be observed when considering these study results.[38] In addition, future work should assess the mechanical compensations that occur during gait at the knee and ankle joints, as the lower extremity is a linked system and these altered loading patterns at the hip joint may be associated with abnormal knee and ankle joint mechanics in patients with hip OA.

CONCLUSION

Patients with radiographic hip OA were older and exhibited higher peak external hip flexion and adduction moments, as well as higher hip flexion, adduction, and external rotation moment impulses, during walking when compared to healthy controls. In the OA group, joint overloading was moderately correlated with patients' self-reported clinical symptoms and cartilage lesions. The results of this study aid in identifying potential biomechanical factors that are associated with signs of hip joint cartilage degeneration and symptom progression. These biomechanical factors are potentially modifiable, offering researchers an opportunity to develop disease-modifying treatment strategies. Future work should focus on identifying gait-modification strategies to optimize hip joint loading, reduce onset/progression of cartilage degeneration, and aid in reducing associated symptoms of hip OA.

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REFERENCES

1. Barker K, Lamb SE, Toye F, Jackson S, Barrington S. Association between radiographic joint space narrowing, function, pain and muscle power in severe osteoarthritis of the knee. Clin Rehabil. 2004;18:793–800. 10.1191/0269215504cr754oa [PubMed: 15573836]
2. Bedson J, Croft PR. The discordance between clinical and radiographic knee osteoarthritis: a systematic search and summary of the literature. BMC Musculoskelet Disord. 2008;9:116. 10.1186/1471-2474-9-116 [PubMed: 18764949]
3. Chehab EF, Andriacchi TP, Favre J. Speed, age, sex, and body mass index provide a rigorous basis for comparing the kinematic and kinetic profiles of the lower extremity during walking. J Biomech. 2017;58:11–20. 10.1016/j.jbiomech.2017.04.014 [PubMed: 28501342]
4. Constantinou M, Barrett R, Brown M, Mills P. Spatial-temporal gait characteristics in individuals with hip osteoarthritis: a systematic literature review and meta-analysis. J Orthop Sports Phys Ther. 2014;44:291–303. 10.2519/jospt.2014.4634 [PubMed: 24450373]
5. Constantinou M, Loureiro A, Carty C, Mills P, Barrett R. Hip joint mechanics during walking in individuals with mild-to-moderate hip osteoarthritis. Gait Posture. 2017;53:162–167. 10.1016/j.gaitpost.2017.01.017 [PubMed: 28167387]
6. Eitzen I, Fernandes L, Nordsletten L, Risberg MA. Sagittal plane gait characteristics in hip osteoarthritis patients with mild to moderate symptoms compared to healthy controls: a cross-sectional study. BMC Musculoskelet Disord. 2012;13:258. 10.1186/1471-2474-13-258 [PubMed: 23256709]
7. Ewers BJ, Weaver BT, Sevensma ET, Haut RC. Chronic changes in rabbit retro-patellar cartilage and subchondral bone after blunt impact loading of the patellofemoral joint. J Orthop Res. 2002;20:545–550. 10.1016/S0736-0266(01)00135-8 [PubMed: 12038629]

8. Farrokhi S, Colletti PM, Powers CM. Differences in patellar cartilage thickness, transverse relaxation time, and deformational behavior: a comparison of young women with and without patellofemoral pain. Am J Sports Med. 2011;39:384–391. 10.1177/0363546510381363 [PubMed: 20962335]
9. Foucher KC. Sex-specific hip osteoarthritis-associated gait abnormalities: alterations in dynamic hip abductor function differ in men and women. Clin Biomech (Bristol, Avon). 2017;48:24–29. 10.1016/j.clinbiomech.2017.07.002
10. Gold SL, Burge AJ, Potter HG. MRI of hip cartilage: joint morphology, structure, and composition. Clin Orthop Relat Res. 2012;470:3321–3331. 10.1007/s11999-012-2403-7 [PubMed: 22723242]
11. Greenwald AS, Haynes DW. Weight-bearing areas in the human hip joint. J Bone Joint Surg Br. 1972;54:157–163. 10.1302/0301-620x.54b1.157 [PubMed: 4110978]
12. Hannan MT, Felson DT, Pincus T. Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. J Rheumatol. 2000;27:1513–1517. [PubMed: 10852280]
13. Ho KY, Hu HH, Colletti PM, Powers CM. Recreational runners with patellofemoral pain exhibit elevated patella water content. Magn Reson Imaging. 2014;32:965–968. 10.1016/j.mri.2014.04.018 [PubMed: 24906520]
14. Ho KY, Keyak JH, Powers CM. Comparison of patella bone strain between females with and without patellofemoral pain: a finite element analysis study. J Biomech. 2014;47:230–236. 10.1016/j.jbiomech.2013.09.010 [PubMed: 24188973]
15. Hurley MV. The role of muscle weakness in the pathogenesis of osteoarthritis. Rheum Dis Clin North Am. 1999;25:283–298. [PubMed: 10356418]
16. Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957;16:494–502. 10.1136/ard.16.4.494 [PubMed: 13498604]
17. Kessler MA, Glaser C, Tittel S, Reiser M, Imhoff AB. Volume changes in the menisci and articular cartilage of runners: an in vivo investigation based on 3-D magnetic resonance imaging. Am J Sports Med. 2006;34:832–836. 10.1177/0363546505282622 [PubMed: 16436539]
18. Kim C, Linsenmeyer KD, Vlad SC, et al. Prevalence of radiographic and symptomatic hip osteoarthritis in an urban United States community: the Framingham osteoarthritis study. Arthritis Rheumatol. 2014;66:3013–3017. 10.1002/art.38795 [PubMed: 25103598]
19. 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;33:527–534. 10.1002/jor.22781 [PubMed: 25678302]
20. Kumar D, Wyatt C, Lee S, et al. Sagittal plane walking patterns are related to MRI changes over 18-months in people with and without mild-moderate hip osteoarthritis. J Orthop Res. 2018;36:1472–1477. 10.1002/jor.23763 [PubMed: 29044677]
21. Lee S, Nardo L, Kumar D, et al. Scoring hip osteoarthritis with MRI (SHOMRI): a whole joint osteoarthritis evaluation system. J Magn Reson Imaging. 2015;41:1549–1557. 10.1002/jmri.24722 [PubMed: 25139720]
22. Liang K, Zeger SL. Longitudinal data analysis using generalized linear models. Biometrika. 1986;73:13–22. 10.1093/biomet/73.1.13
23. Loureiro A, Constantinou M, Diamond LE, Beck B, Barrett R. Individuals with mild-to-moderate hip osteoarthritis have lower limb muscle strength and volume deficits. BMC Musculoskelet Disord. 2018;19:303. 10.1186/s12891-018-2230-4 [PubMed: 30131064]
24. Mamisch TC, Zilkens C, Siebenrock KA, Bittersohl B, Kim YJ, Werlen S. MRI of hip osteoarthritis and implications for surgery. Radiol Clin North Am. 2009;47:713–722. [PubMed: 19631078]
25. Meyer CAG, Wesseling M, Corten K, et al. Hip movement pathomechanics of patients with hip osteoarthritis aim at reducing hip joint loading on the osteoarthritic side. Gait Posture. 2018;59:11–17. 10.1016/j.gaitpost.2017.09.020 [PubMed: 28968547]
26. Neumann DA. Kinesiology of the Musculoskeletal System: Foundations for Physical Rehabilitation. St Louis, MO: Mosby; 2002.
27. Neumann J, Zhang AL, Schwaiger BJ, et al. Validation of scoring hip osteoarthritis with MRI (SHOMRI) scores using hip arthroscopy as a standard of reference. Eur Radiol. 2019;29:578–587. 10.1007/s00330-018-5623-8 [PubMed: 29987419]

28. Outerbridge RE, Dunlop JA. The problemof chondromalacia patellae. Clin Orthop Relat Res. 1975:177–196. 10.1097/00003086-197507000-00024 [PubMed: 1098819]
29. Perry J, Burnfield JM. Gait Analysis: Normal and Pathological Function. 2nd ed. Thorofare, NJ: SLACK; 2010.
30. Quintana JM, Arostegui I, Escobar A, Azkarate J, Goenaga JI, Lafuente I. Prevalence of knee and hip osteoarthritis and the appropriateness of joint replacement in an older population. Arch Intern Med. 2008;168:1576–1584. 10.1001/archinte.168.14.1576 [PubMed: 18663171]
31. Samaan MA, Schwaiger BJ, Gallo MC, et al. Joint loading in the sagittal plane during gaitis associated with hip joint abnormalities in patients with femoroacetabular impingement. Am J Sports Med. 2017;45:810–818. 10.1177/0363546516677727 [PubMed: 28006109]
32. Schmidt A, Meurer A, Lenarz K, et al. Unilateral hip osteoarthritis: the effect of compensation strategies and anatomic measurements on frontal plane joint loading. J Orthop Res. 2017;35:1764–1773. 10.1002/jor.23444 [PubMed: 27664397]
33. Sparks DR, Beason DP, Etheridge BS, Alonso JE, Eberhardt AW. Contact pressures in the flexed hip joint during lateral trochanteric loading. J Orthop Res. 2005;23:359–366. 10.1016/j.orthres.2004.08.019 [PubMed: 15734249]
34. Spoor CW, Veldpaus FE. Rigid body motion calculated from spatial co-ordinates of markers. J Biomech. 1980;13:391–393. 10.1016/0021-9290(80)90020-2 [PubMed: 7400168]
35. Subburaj K, Kumar D, Souza RB, et al. The acute effect of running on knee articular cartilage and meniscus magnetic resonance relaxation times in young healthy adults. Am J Sports Med. 2012;40:2134–2141. 10.1177/0363546512449816 [PubMed: 22729505]
36. von Eisenhart-Rothe R, Eckstein F, Müller-Gerbl M, Landgraf J, Rock C, Putz R. Direct comparison of contact areas, contact stress and subchondral mineralization in human hip joint specimens. Anat Embryol (Berl). 1997;195:279–288. 10.1007/s004290050047 [PubMed: 9084826]
37. von Elm E, Altman DG, Egger M, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Int J Surg. 2014;12:1495–1499. 10.1016/j.ijsu.2014.07.013 [PubMed: 25046131]
38. Watelain E, Dujardin F, Babier F, Dubois D, Allard P. Pelvic and lower limb compensatory actionsof subjects in an early stage of hip osteoarthritis. Arch Phys Med Rehabil. 2001;82:1705–1711. 10.1053/apmr.2001.26812 [PubMed: 11733886]
39. Yusuf E, Kortekaas MC, Watt I, Huizinga TW, Kloppenburg M. Do knee abnormalities visualised on MRI explain knee pain in knee osteoarthritis? A systematic review. Ann Rheum Dis. 2011;70:60–67. 10.1136/ard.2010.131904 [PubMed: 20829200]
40. Zeni J, Jr., Pozzi F, Abujaber S, Miller L. Relationship between physical impairments and movement patterns during gait in patients with end-stage hip osteoarthritis. J Orthop Res. 2015;33:382–389. 10.1002/jor.22772 [PubMed: 25492583]

KEY POINTS

FINDINGS:

Hip joint loads were compared between persons with hip osteoarthritis (OA) and controls. Patients with OA exhibited abnormally high hip joint loads during gait, and higher loads were associated with worse self-reported symptoms and cartilage morphology.

IMPLICATIONS:

Higher hip joint loads may be risk factors for the development of hip OA, but longitudinal assessment is needed to further elucidate this relationship.

CAUTION:

It should be noted that limitations existed within this cross-sectional analysis and unmatched study design. In addition, fixed walking speed was assessed during gait analysis, and joint kinetics in persons with hip OA might differ at self-selected speeds.

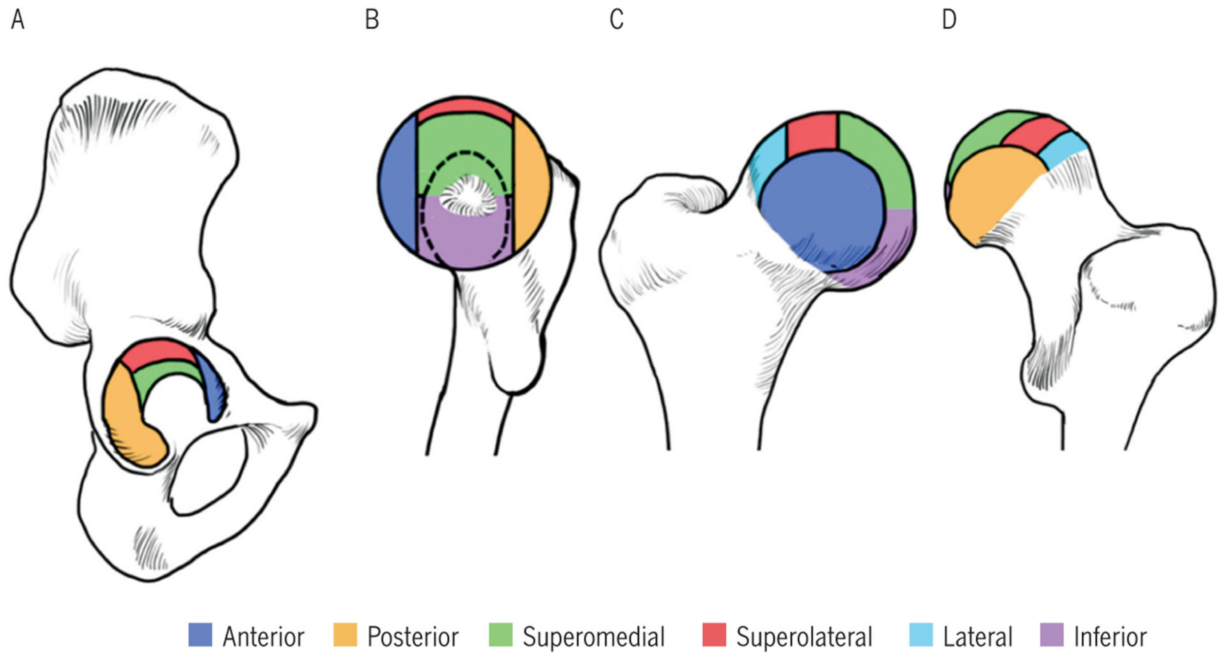

FIGURE 1.
Hip joint subregions for the grading of Scoring Hip Osteoarthritis with Magnetic Resonance Imaging. (A) Acetabular joint subregions seen from the lateral aspect. Femur joint subregions seen from (B) the medial aspect, (C) the anterior aspect, and (D) the posterior aspect. Reproduced with permission from Kumar et al.[19]

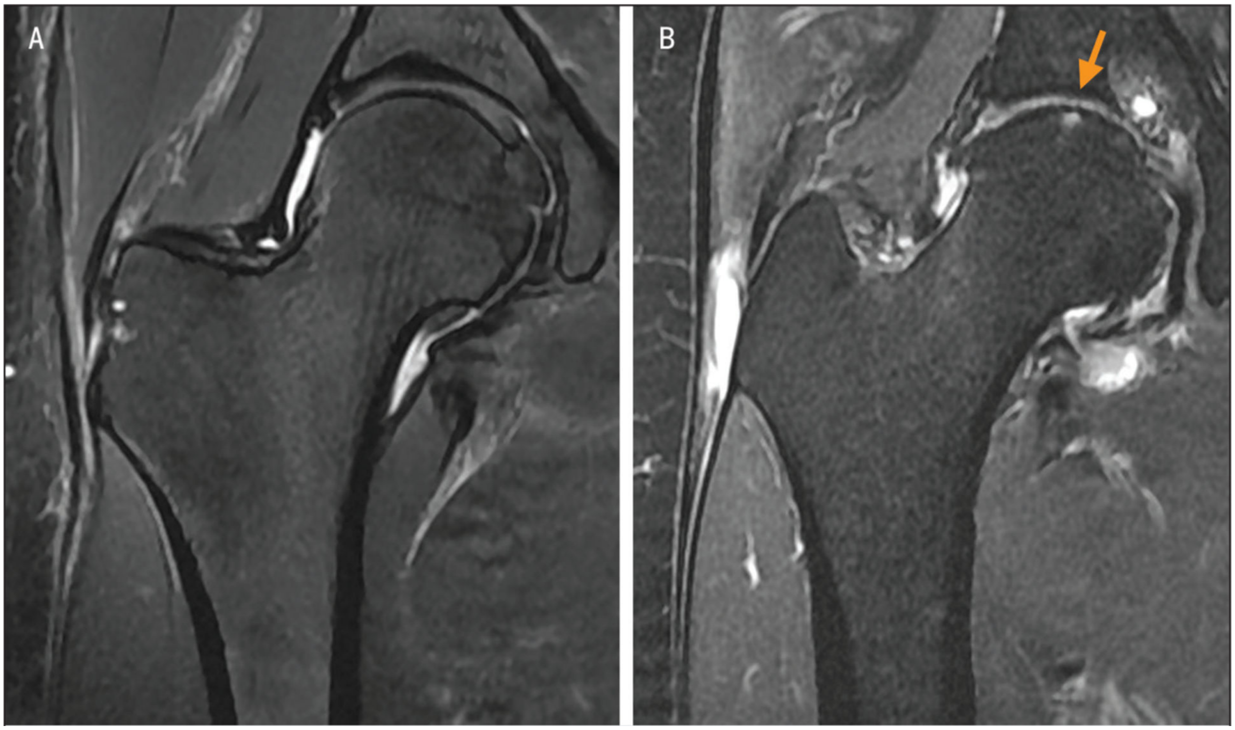

FIGURE 2.

Examples of coronal clinical images acquired on the right hip of (A) a control participant with no cartilage lesions and (B) a participant with osteoarthritis and acetabular and femoral cartilage thinning (arrow), with a femoral-head bone marrow edema pattern, osteophytes, and labral tearing.

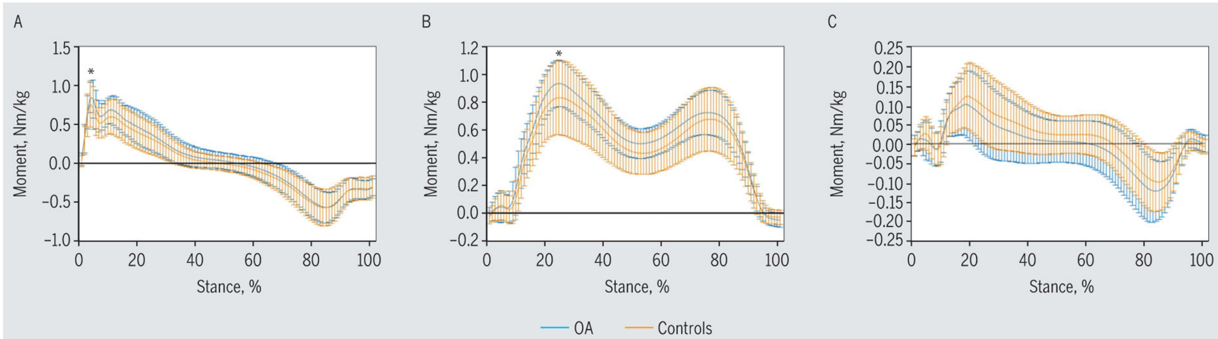

FIGURE 3.

Hip external joint moments during the stance phase of walking in the (A) sagittal, (B) frontal, and (C) transverse planes for the OA group and control group. (A) Positive values indicate flexion and negative values indicate extension. (B) Positive values indicate adduction and negative values indicate abduction. (C) Positive values indicate internal rotation and negative values indicate external rotation. *Significant difference between groups. Abbreviation: OA, osteoarthritis.

TABLE 1

Demographics, Patient-Reported Symptoms, and MRI Evidence of Acetabular and Femoral Cartilage Lesions in the OA and Control Groups*

	OA (n = 48, n = 71 hips)	Control (n = 95, n = 143 hips)	*P* Value
Sex, n†			.111
Male	28	40	
Female	20	55	
Age, y	56.0 ± 12.2	43.2 ± 13.6	<.001‡
BMI, kg/m^2	24.3 ± 3.5	24.0 ± 3.1	.568
HOOS, %			
Pain	86.2 ± 18.3	91.5 ± 14.5	.062
Function in daily living	88.8 ± 17.9	95.2 ± 11.1	.012‡
SHOMRI†			
Acetabular (0–8)	1.9 ± 1.8	0.8 ± 1.1	<.001‡
Femoral (0–12)	2.4 ± 2.6	0.9 ± 1.7	<.001‡

Abbreviations: BMI, body mass index; HOOS, Hip disability and Osteoarthritis Outcome Score; OA, osteoarthritis; MRI, magnetic resonance imaging; SHOMRI, Scoring Hip Osteoarthritis with Magnetic Resonance Imaging.

*Values are mean ± SD unless otherwise indicated.

†Chi-square analysis.

‡Significant (P<.05).

TABLE 2

Hip Joint Kinetics During the Stance Phase of Walking in Hips With and Without Radiographic Osteoarthritis*

	OA (n = 48, n = 71 hips)	Control (n = 95, n = 143 hips)	β	*P* Value
Hip moment, Nm/kg				
Flexion	0.97 ± 0.18	0.84 ± 0.30	−0.13	<.001†
Extension	−0.59 ± 0.18	−0.59 ± 0.22	<0.01	.951
Adduction	0.97 ± 0.16	0.86 ± 0.27	−0.10	<.001†
Internal rotation	0.13 ± 0.07	0.14 ± 0.08	<0.01	.481
External rotation	−0.14 ± 0.06	−0.11 ± 0.06	0.02	.072
Hip moment impulse, Nm · ms/kg				
Flexion	121.42 ± 43.77	99.76 ± 48.73	−21.65	.009†
Extension	99.71 ± 49.28	102.36 ± 48.56	2.64	.767
Adduction	356.95 ± 69.01	312.40 ± 107.58	−44.55	.001†
Internal rotation	21.71 ± 17.26	25.42 ± 22.68	3.71	.271
External rotation	22.94 ± 20.18	15.85 ± 16.62	−7.09	.039†

Abbreviation: OA, osteoarthritis.

*Values are mean ± SD unless otherwise indicated. All analyses were performed with covariates of age, sex, and body mass index. Positive values indicate peak hip flexion, adduction, and internal rotation moments.

†Significant (P<.05).

TABLE 3

Associations of Hip Moment Impulses With the HOOS and SHOMRI*

	Hip Flexion Moment Impulse		Hip External Rotation Moment Impulse	
	Partial Correlation	***P* Value**	**Partial Correlation**	***P* Value**
HOOS				
Pain	$r = -0.361$	.003†	$r = 0.194$	.116
Function in daily living	$r = -0.424$	<.001†	$r = 0.200$	.105
SHOMRI				
Acetabular				
Superolateral	$\rho = 0.175$	.153	$\rho = 0.206$	.092
Superomedial	$\rho = -0.001$	.992	$\rho = -0.009$	.940
Anterior	$\rho = 0.074$	.547	$\rho = 0.182$	.138
Posterior	$\rho = 0.198$	.105	$\rho = 0.283$	.019†
Total	$\rho = 0.124$	.312	$\rho = 0.204$	.096
Femoral				
Lateral	$\rho = 0.315$	.009†	$\rho = 0.216$	.077
Superolateral	$\rho = 0.011$	.927	$\rho = 0.187$	.127
Superomedial	$\rho = 0.256$	.035†	$\rho = 0.156$	.203
Inferomedial	$\rho = 0.140$	.254	$\rho = 0.156$	.204
Anterior	$\rho = 0.280$	.021	$\rho = 0.372$	.002†
Posterior	$\rho = 0.217$	.075	$\rho = 0.313$	.009†
Total	$\rho = 0.275$	.023†	$\rho = 0.334$	.005†

Abbreviations: HOOS, Hip disability and Osteoarthritis Outcome Score; SHOMRI, Scoring Hip Osteoarthritis with Magnetic Resonance Imaging.

*All analyses were performed with covariates of age, sex, and body mass index.

†Significant (P<.05).