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External knee adduction and flexion moments during gait and medial tibiofemoral disease progression in knee osteoarthritis

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SUMMARY

Objective: Test the hypothesis that greater baseline peak external knee adduction moment (KAM), KAM impulse, and peak external knee flexion moment (KFM) during the stance phase of gait are associated with baseline-to-2-year medial tibiofemoral cartilage damage and bone marrow lesion progression, and cartilage thickness loss.

Methods: Participants all had knee OA in at least one knee. Baseline peak KAM, KAM impulse, and peak KFM (normalized to body weight and height) were captured and computed using a motion analysis system and six force plates. Participants underwent MRI of both knees at baseline and 2 years later. To assess the association between baseline moments and baseline-to-2-year semiquantitative cartilage damage and bone marrow lesion progression and quantitative cartilage thickness loss, we used logistic and linear regressions with generalized estimating equations (GEE), adjusting for gait speed, age, gender, disease severity, knee pain severity, and medication use.

Results: The sample consisted of 391 knees (204 persons): mean age 64.2 years (SD 10.0); BMI 28.4 kg/m^2 (5.7); 156 (76.5%) women. Greater baseline peak KAM and KAM impulse were each associated with worsening of medial bone marrow lesions, but not cartilage damage. Higher baseline KAM impulse was associated with 2-year medial cartilage thickness loss assessed both as % loss and as a threshold of loss, whereas peak KAM was related only to % loss. There was no relationship between baseline peak KFM and any medial disease progression outcome measures.

Conclusion: Findings support targeting KAM parameters in an effort to delay medial OA disease progression.

Introduction

Osteoarthritis (OA) is a leading contributor to chronic disability[1]. Twenty-three percent of US adults report doctor-diagnosed arthritis and 10% have arthritis-related activity limitations[2]. OA is the most common form of arthritis, frequently affecting the knee. The impact of knee OA in the US is likely to increase due to the aging population, obesity epidemic, and paucity of disease-modifying treatment. It is well accepted that an abnormal knee local mechanical environment can contribute to joint damage. Change in medial-to-lateral tibiofemoral load distribution and greater medial load are theorized to increase the risk of medial knee OA disease progression[3].

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Instrumented force-measuring knee implantation is the current gold standard method for measurement of medial knee load, but it is invasive and impractical. Knee load cannot be directly measured *in vivo* noninvasively. The external knee adduction moment (KAM) during the stance phase of gait has been characterized both as a determinant and a surrogate for dynamic medial knee load[3,4]. KAM reflects the medial-to-lateral joint load distribution[5] and has been associated with lower limb varus alignment[6], medial OA disease severity[7], and medial-to-lateral bone mineral density ratio[8]. Efforts have been directed toward developing and testing interventions that lower KAM with the ultimate goal of modifying disease course in medial tibiofemoral OA[9,10]. However, longitudinal evidence of an association between baseline KAM and subsequent medial disease progression comes from only a few studies with inconsistent findings[11–13].

Peak KAM during the stance phase potentially captures maximal medial joint load experienced at any one instant of time. KAM impulse is the time integral of KAM over the stance phase. By incorporating both load magnitude *and* duration, KAM impulse may provide a cumulative measure of KAM sustained during each step of walking. There is a theoretical rationale to support a role for both of these parameters in disease progression. Studies in recent years suggest that a reduction in KAM may be accompanied by a deleterious increase in the external knee flexion moment (KFM)[14,15]. However, whether KFM plays a role in knee OA disease progression in OA knees is unclear.

The objective of this study was to evaluate the association between baseline KAM and KFM parameters and subsequent medial tibiofemoral OA disease progression over 2 years. We hypothesized that in persons with knee OA, greater baseline peak KAM, KAM impulse, and peak KFM (each normalized to body weight and height) during the stance phase of gait are each associated with baseline-to-2-year worsening of medial tibiofemoral cartilage damage and bone marrow lesions, and with quantitatively measured cartilage thickness loss.

Methods

Sample

In this prospective, longitudinal, observational cohort study of knee OA, the MAK-3 Study (Mechanical Factors in Arthritis of the Knee-Study 3), participants were recruited from the community using advertising in periodicals, neighborhood organizations, letters to the Buehler Center on Aging, Health, and Society registry at Northwestern University, and via medical center referrals. Inclusion criteria were: definite tibiofemoral osteophyte presence [Kellgren/Lawrence (K/L) radiographic grade ≥ 2] in one or both knees; and Likert category of at least “a little difficulty” for 2 or more items in the WOMAC physical function scale. Exclusion criteria were: corticosteroid injection within previous 3 months; avascular necrosis, inflammatory arthritis, periarticular fracture, Paget's disease, villonodular synovitis, joint infection, ochronosis, neuropathic arthropathy, acromegaly, hemochromatosis, gout, pseudogout, osteopetrosis, or meniscectomy; or MRI exclusions. Approval was obtained from the Institutional Review Boards of Northwestern University and NorthShore University HealthSystem Evanston Hospital. All participants provided written consent.

Quantitative gait analysis

Kinematic data were collected at 120 Hz, using an 8-camera, Eagle Digital Real-Time motion measurement system from Motion Analysis Corporation (MAC). At a sampling rate of 960 Hz, ground reaction forces and moments were measured with six AMTI (Advanced Mechanical Technology Inc., Watertown, MA, USA) force platforms embedded flush with the floor as participants walked along a 10.7 × 1.2 m walkway. An experienced technician placed external passive reflective markers, using the modified Helen Hayes full-body marker set[16] (bilaterally on acromion process tip, lateral humeral epicondyle, between radius and ulna styloids, anterior superior iliac spine, superior sacrum at L5/sacral interface, lower thigh, along flexion/extension rotation axis at lateral femoral condyle, lower leg, along flexion/extension rotation axis at lateral malleolus, posterior calcaneus, foot center between second and third metatarsals). To closely match usual daily walking, each participant wore his/her own comfortable athletic or walking shoes and walked at a self-selected comfortable speed without using assistive devices (no participant habitually used assistive devices). A minimum of five trials having clean foot strikes on the force platforms for the left and right feet were acquired, with rest between trials. OrthoTrak gait analysis software (MAC) was used to calculate 3-D joint angles, moments, and temporal–spatial parameters. Inverse dynamics were used to compute 3-D external joint moments. Baseline predictors of peak KAM (% body weight*height), KAM impulse – the area under the KAM-time curve (seconds*% body weight*height), and KFM (% body weight*height) were calculated using custom Matlab programs. Gait speed was measured within the quantitative gait analysis; the 5-trial average was used.

While KAM normalization is widely accepted and established, to address the possibility that the absolute (i.e., non-normalized) KAM parameter values differed in pattern of association with the outcomes, we evaluated the correlation between normalized and non-normalized values, and, in sensitivity analyses, the association between non-normalized KAM parameters and outcomes.

MRI acquisition and semi-quantitative assessment of cartilage damage and bone marrow lesion progression

At baseline and 2-year follow-up, magnetic resonance images (MRI) of both knees were obtained in all participants, using a commercial knee coil and 1 of 2 whole-body scanners, 3T Verio or 1.5T Avanto (both Siemens Healthcare, Erlangen, Germany); the same scanner was used at both evaluations. The protocol included coronal T1-weighted spin-echo (SE) [TR/TE/FOV/Matrix/Slice thickness = 3 s/20 ms/14 cm, 256 × 256, 3 mm at 3T; TR/TE/FOV/Matrix/Slice thickness = 3 s/18 ms/14 cm, 256 × 256, 3 mm at 1.5T], and sagittal axial, and coronal fat-suppressed proton density-weighted turbo spin echo sequences [TR/TE/Turbo Factor/FOV/Matrix/Slice thickness = 500 ms/11 ms/7/12 cm, 320 × 320, 3 mm at 3T; TR/TE/Turbo Factor/FOV/Matrix/Slice thickness = 600 ms/11 ms/7/12 cm, 320 × 320, 3 mm at 1.5T].

Following a detailed reading protocol, each knee was scored using the Whole-Organ MRI Score (WORMS) method[17], by one of two expert musculoskeletal radiologists. Baseline and 2-year scans were evaluated as pairs, with known chronology as suggested for longitudinal studies in knee OA[18], but blinded to all other data. Two medial weightbearing femoral condylar subregions (central and posterior) and three medial tibial plateau subregions (anterior, central, and posterior) were each scored separately for cartilage morphology and bone marrow lesions. At each subregion, cartilage morphology was scored: 0 (normal thickness and signal); 1 (normal thickness, increased signal on T2-weighted images); 2 (solitary, focal, partial or full-thickness defect ≤ 1 mm in width); 3 (multiple areas of partial-thickness loss or grade 2 lesion > 1 mm, with areas of preserved thickness); 4 (diffuse, >75%, partial-thickness loss); 5 (multiple areas of full-thickness loss, or full-thickness lesion > 1 mm, with areas of partial-thickness loss); and 6 (diffuse, >75%, full-thickness loss). Subchondral bone marrow

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