

The Characteristic Knee Flexion Pattern of Walking Relates to Pathological Imaging of Bone Marrow Lesion and Articular Cartilage Degeneration in Knee Osteoarthritis in Japanese.

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INTRODUCTION: Osteoarthritis (OA) is considered a disease of the entire joint, involving whole joint tissue change. The bone-cartilage interface is a complex functional unit and biocomposite at the center of joint function and disease in which the individual components interact cooperatively and synergistically. Biomechanical and biological processes result in alterations in the composition, structure, and functional properties of this unit. Given the intimate contact between bone and cartilage, alterations of either tissue will modulate the properties and function of the other components. The causes of degenerative changes in knee OA(KOA) are complex and involve interrelated biological, mechanical, and structural pathways. While KOA is a complex disease with multiple phenotypes that can influence its initiation and progression, mechanical stress has become a primary consideration in assessing the nature of the disease. Recently, the importance of subgrouping KOA by pathological classification has been suggested for biomechanical analysis and treatment for KOA patients. Magnetic resonance imaging (MRI) allows the detection of the whole spectrum of pathological joint tissue changes. Clinically, Bone Marrow Lesions (BMLs) revealed by MRI is occasionally found in KOA patients, preceding the onset of articular cartilage degeneration. BMLs are indicators of KOA progression and are considered an important risk factor for structural deterioration. Recent studies of animal models indicate that compression stress to the knee joint induces subchondral bone remodeling. As such, we make a hypothesis that BMLs is formed by the mechanical stress resulting from a characteristic walking pattern different from articular cartilage degeneration. The objective of this study is to elucidate the relationship between types of pathological imaging of KOA (BMLs and articular cartilage degeneration) and the kinematic parameter of walking.

METHODS: Participants were divided into 5 groups. Normal adult group (NAG) were extracted from university students. Volunteers aged over 50 years and KOA patients were extracted and divided into OA risk group (OARG), BML group (BMLg), OA group (OAg) and End-stage OA group (EOAg) according to MRI and X-ray. OARG has healthy joint, BMLg has BMLs without severe cartilage degeneration (Kellgren-Lawrence grade (K-L) 4), OAg has cartilage degeneration (K-L 2,3) without BMLs, EOAg has BMLs and severe cartilage degeneration (K-L 4). MRI was obtained using a 3.0 Tesla MRI scanner (GE Health Care). BMLs was defined as poorly marginated areas of increased signal intensity in the epiphyseal marrow on proton density fat-suppressed 3D Fast Spin Echo images¹⁾. Exclusion criteria were a history of orthopedic disorder except KOA, a history of injuries of the knee joint, inability to walk on a treadmill, or MRI exclusions. Participants performed walking trials on the instrumented treadmill with force plates (Bertec) at their preferred walking speed. Using a three-dimensional motion analysis system (VICON), joint angles and joint moments were obtained from the coordinate information of markers and ground reaction force data. Using the optimal common shape technique (OCST), where an optimum rigid marker configuration for each segment using data from the calibration posture is formed to reduce the effects of skin elasticity. The hip joint center was then identified from the star-arc movements using the symmetrical center of rotation estimation (SCoRE), an algorithm to determine the center of rotation of joints from segment motion data²⁾. Functional knee axes were identified from the knee flexion-extension movements, using the symmetrical axis of rotation approach (SARA), which computes the axis of rotation in local segment-based coordinates for each segment²⁾. 15 gait cycles of each participant were extracted, and average data of the stance phase were calculated. Then, the average knee flexion angle of the stance phase (average KFA), the knee flexion angle of the initial contact (IC KFA) and the 1st peak (peak KFA), and the change of the knee flexion angle (KFA change; peak KFA - IC KFA) were calculated. Kruskal-Wallis test and Steel-Dwass test were used to compare the biomechanical variables between the different groups. The level of significance was chosen as $p < 0.05$. This study was approved by the ethics review committee of Saitama Prefectural University and Tokyo Metropolitan University.

RESULTS: Participants were divided NAG (20knees, age21.5, BMI21.1), OARG (6knees, age58, BMI21.9), BMLg (6knees, age70, BMI21), OAg (6knees, age54, BMI20.3) and EOAg (7knees, age73.75, BMI24.2). Average KFA was following; NAG=12.56, OARG=10.32, BMLg=9.56, OAg=12.73, EOAg=19.38. EOAg was significantly higher than other groups ($P < 0.01$), and OAg was significantly higher than BMLg ($P < 0.05$). IC KFA was following; NAG=8.60, OARG=8.42, BMLg=9.99, OAg=10.77, EOAg=21.25. EOAg was significantly higher than NAG. Peak KFA was following; NAG=15.65, OARG=16.79, BMLg=14.93, OAg=20.29, EOAg=21.25. The tendency that BMLg indicated lower peak KFA than OAg and EOAg was confirmed, however, there were no significant differences. The timepoint of peak KFA of EOAg was 0% even though that of other groups was 9~10% of the stance phase. KFA change was following; NAG=10.41, OARG=9.04, BMLg=5.04, OAg=9.67, EOAg=3.45. EOAg was significantly lower than NAG ($P < 0.05$). The tendency that BMLg indicated lower KFA change than NAG, OARG and OAg were confirmed, however, there was no significant differences.

DISCUSSION: Results of this study indicated that BMLg had lower KFA change, OAg had higher average/peak KFA, and EOAg had higher average/peak KFA and lower KFA change. Among the characteristic walking patterns shown by EOAg, BMLg denoted the same tendency of relatively low alteration in the angle of knee flexion, and OAg was similar in that it increased average KFA and its 1st peak. It is suggested that the difference in knee flexion pattern is related to the pathogenesis of BMLs and articular cartilage degeneration of the knee joint. The characteristic knee flexion pattern of BMLg has potentially led to regional high compression stress to the subchondral bone and articular cartilage of the knee joint and its effects to cause BMLs.

SIGNIFICANCE/CLINICAL RELEVANCE: This study provides preliminary insights to prevent the onset and degeneration of KOA.

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