RESEARCH ARTICLE

Gait Asymmetry Variation in Kinematics, Kinetics, and Muscle Force along with the Severity Levels of Knee Osteoarthritis

Junqing Wang, ME[1,2,3], Qinsheng Hu, PhD[2], Chenyu Wu, MB[2], Shiqi Li, MB[4], Qian Deng, MM[2], Ruoliang Tang, PhD[5], Kang Li, PhD[1], Yong Nie, PhD[2], Bin Shen, MD, PhD[2]

[1]West China Biomedical Big Data Center, Sichuan University West China Hospital and [2]Department of Orthopedics, Orthopedic Research Institute and National Clinical Research Center for Geriatrics, West China Hospital, West China Medical School, [3]Med-X Center for Informatics, [4]College of Electrical Engineering and [5]Sichuan University-Pittsburgh Institute (SCUPI), Sichuan University, Chengdu, China

Objective: Knee osteoarthritis (OA) patients exhibit greater gait asymmetry than healthy controls. However, gait asymmetry in kinematics, kinetics and muscle forces across patients with different severity levels of knee OA is still unknown. The study aimed to investigate the changes of gait asymmetry in lower limb kinematics, kinetics, and muscle force across patients with different severity levels of knee OA.

Methods: This is a cross-sectional study. From January 2020 to January 2021, 118 patients with symptomatic and radiographic medial knee OA were categorized into three groups using the Kellgren and Lawrence scale (mild: grade 1 and 2, n = 37; moderate: grade 3, n = 31; severe: grade 4, n = 50). During self-paced walking, marker trajectories and ground reaction forces data were recorded. Musculoskeletal simulations were used to determine gait kinematics, kinetics, and muscle force. One-way analysis of variance with Tukey's post-hoc test was used to evaluate group difference. Paired-sample t-test was used to compared the between-limb difference.

Results: In the Severe group, significantly greater asymmetry index in knee flexion/extension range of motion (45%) was observed with a greater value on the contralateral side ($p < 0.01$), compared to the Mild (15%) and Moderate (15%) groups. Significantly higher peak hip contact force (JCF) on the contralateral side was found in the Mild (more affected side: 3.80 ± 0.67 BW, contralateral side: 4.01 ± 0.58 BW), Moderate (more affected side: 3.67 ± 0.56 BW, contralateral side: 4.07 ± 0.81 BW), and Severe groups (more affected side: 3.66 ± 0.79 BW, contralateral side: 3.94 ± 0.64 BW) ($p < 0.05$). Significantly greater gluteus medius muscle force on the contralateral side was found in Mild (more affected side: 0.48 ± 0.09 BW, contralateral side: 0.52 ± 0.12 BW), Moderate (more affected side: 0.45 ± 0.10 BW, contralateral side: 0.51 ± 0.15 BW), and Severe groups (more affected side: 0.42 ± 0.15 BW, contralateral side: 0.47 ± 0.12 BW) ($p < 0.05$). The contralateral side showing significantly higher peak knee adduction moment and medial knee JCF was only observed in the Mild group ($p < 0.05$).

Conclusions: Gait asymmetry in kinematics and muscle forces increased from mild to severe knee OA. Asymmetrical gait pattern tends to transfer loads from the more affected side to the contralateral side. Peak hip JCF and gluteus medius muscle force can be used to detect this asymmetrical gait pattern in patients with knee OA, regardless of severity levels.

Key words: Gait Asymmetry; Gait Modification; Knee Osteoarthritis; Musculoskeletal Simulation

Address for correspondence Kang Li, PhD, West China Biomedical Big Data Center, Sichuan University West China Hospital, Chengdu, Sichuan Province, China 610041; Email: likang@wchscu.cn; Yong Nie, PhD, Department of Orthopedics, Orthopedic Research Institute and National Clinical Research Center for Geriatrics, West China Hospital, West China Medical School Sichuan University, Chengdu, Sichuan Province, China 610041; Email: nieyong1983@wchscu.cn
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Introduction

Knee osteoarthritis (OA) is a widespread joint condition that causes lower limb pain, instability, and muscle weakness.[1,2] To cope with these symptoms, individuals with knee OA often adopt strategies, e.g., reducing walking speed, lowering the pelvis to the contralateral side or other changes in multiplanar kinematics and kinetics of the lower limbs, resulting in modified gait patterns.[3,4] Unfortunately, these findings are closely related to the knee OA affected knee, while the understanding of the potential biomechanical changes on the contralateral side knee is still lacking. Evaluating the state of the contralateral knee may help prevent the development and progression of knee OA on the contralateral side.[3,5] Gait asymmetry which has been used to characterize and quantify the biomechanical differences between lower limbs can be used to evaluate bilateral limbs.[6] Therefore, it is important to calculate gait asymmetry when evaluating the progression of knee OA and its potential influence on the biomechanics of modified gait patterns.

Previous studies have reported several characteristics regarding gait asymmetry in patients with knee OA. Gait asymmetry can be evaluated by comparing the two sides. Compared with the OA affected knee, the contralateral knee exhibits higher peak flexion angle,[3] peak vertical knee contact force,[7] peak vertical ground reaction force,[8] and hip and knee flexion moments.[3] In addition, gait asymmetry can also be quantified using asymmetry index,[6,9] making it easier to compare across different groups. For example, knee OA patients may exhibit greater asymmetry in foot posture index,[10] medial-lateral trunk acceleration,[11] knee flexion at initial contact,[9] peak knee flexion angle,[12] and peak knee flexion moment[12] compared to healthy individuals. However, gait asymmetry among knee OA patients with different severity levels is yet to be investigated. Asymmetry in quadriceps strength and trunk movement were closely related to an increased risk of developing knee OA and exerted impacts on lower limb function.[13] Therefore, in clinical settings, it is crucial for clinicians and physical therapists to understand how knee OA severity affects gait asymmetry and to evaluate the gait modification accompanied with asymmetry.

The purpose of the present study was to (1) evaluate knee OA patients for their gait asymmetry in kinematics, kinetics, and muscle forces and (2) investigate the associations between knee OA severity (i.e., mild, moderate, and severe) and the magnitude of gait asymmetry. We hypothesized that gait asymmetry in kinematics, kinetics, and muscle forces would increase along with the severity levels of knee OA.

Methods

Subjects

In this study, 118 patients with symptomatic medial knee OA were recruited from West China Hospital between January 2020 and January 2021 (Table 1). Ethics approval was obtained from the local ethics committee, and informed consent was provided by all patients (No. 268). Inclusion criteria included: (1) definitive radiographic diagnosis of knee OA in the medial tibiofemoral compartment in one or both knees defined as Kellgren and Lawrence (K&L) grade ≥ 1[14]; (2) knee pain most of the days within the past month; (3) ability to walk independently on a flat surface without external ambulatory assistance. Several exclusion criteria were also applied, including (1) medical history of lower limb surgery; (2) body mass index (BMI) greater than 30 kg/m^2; (3) rheumatoid arthritis; (4) other neuromuscular conditions or diseases that may potentially affect walking. In addition, 20 healthy adults without radiographic and symptomatic knee OA from the community were recruited as the healthy control (Table 1).

Knee OA patients were subsequently categorized into three groups based on the K&L grade of the more affected knee, including Mild group (Grade 1 and 2; n = 37), Moderate group (Grade 3; n = 31), or Severe group (Grade 4; n = 50). The lower limb alignment for each side was determined by measuring the angle between the mechanical axes of the femur and the tibia, as seen on a full-length standing radiograph.[15] Varus alignment was indicated by values less than 180°, while valgus alignment was indicated by values greater than 180°.[15]

Gait Analysis

The subjects walked barefoot along a 12-meter walkway at their self-selected pace. They were instructed to walk as naturally as possible and look straight ahead. A total of 28 retro-reflective markers were placed on the pelvis and lower limbs using the CAST lower body marker set.[16] Ground reaction force (GRF) data and marker trajectory data were collected using two Bertec force plates (Bertec, Columbus, OH, USA) embedded in the walkway synchronized with a 10-camera motion capture system (Oqus300, Qualisys, Gothenburg, Sweden). Data was recoded at 1000 Hz for GRF and 200 Hz for marker trajectory. Prior to the walking trial, participants completed a standing static trial, followed by three walking trails.[17]

Marker trajectory and GRF data were lowpass filtered using a fourth-order zero-lag Butterworth filter with cutoff frequencies of 6 Hz and 45 Hz, respectively,[18] and converted to a format compatible with OpenSim.

Musculoskeletal Modeling

A previously published and validated full-body musculoskeletal gait model[19] was implemented in OpenSim (v4.2).[20] This model was comprised of 18 segments, 21° of freedom, and 92 Hill-type muscle-tendon units. The Hip joints were modeled as ball-and-socket joints. Ankle and subtalar joints were modeled as hinge joints. The knee joints were modeled as hinge joints with 1° of freedom and the ability to resolve medial and lateral compartment contact forces. Furthermore, this model also allowed for adjustment in frontal plane knee alignment and the medial and lateral compartment contact locations.[19]

TABLE 1 Mean (SD) demographic data for the participating knee OA patients and healthy control

	Knee OA group			
	Mild(n = 37)	Moderate (n = 31)	Severe(n = 50)	Healthy control (n = 20)
Age (yr)	54.4 (12.9)	60.2 (10.2)	64.2 (6.5)	48.8 (3.2)
Height (cm)	160.0 (7.0)	157.5 (4.8)	159.2 (7.1)	157.5 (5.5)
Weight (kg)	61.9 (8.60)	60.5 (8.4)	64.9 (8.8)	58.6 (6.9)
BMI (kg/m^2)	24.2 (3.3)	24.3 (2.6)	25.6 (2.9)	23.6 (2.5)
Female, n (%)	30 (81.1)	25 (80.6)	40 (80)	16 (80)
K&L Grade (of the more affected knee)	I:4, II:33	III:31	IV: 50	0: 20
Alignment (of the more affected knee) (°)	178.8 (2.0)	177.8 (3.0)	169.7 (5.9)	179.4 (1.7)

Abbreviation: BMI, body mass index

After scaling the generic model to each subject's anthropometry, a subject-specific model was created using the lower limb alignment for that subject. Joint angles were determined by minimizing the errors between experimentally collected marker trajectories and virtual markers identified on the model at locations corresponding to the markers used in the gait trials. Then, a residual reduction algorithm[20] was used to improve the dynamic consistency of each simulated walking trial. Static optimization, a valid approach to estimate gait characteristics,[21] was employed to estimate muscle forces generated during the walking trial by minimizing the sum of squared muscle activations. Lastly, hip, knee (including the medial and lateral compartments), and ankle joint contact forces were calculated using the JointReaction Analysis tool in OpenSim.[22]

Data Post-Processing

Gait parameters were calculated using Visual3D™ software (C-Motion, Inc., Germantown, MD, USA), including gait speed, stance time, stride length, hip flexion/extension range of motion (RoM_{hip}), knee flexion/extension range of motion (RoM_{knee}), ankle flexion/dorsiflexion range of motion (RoM_{ankle}), peak knee flexion moment, and peak knee adduction moment (KAM). All parameters, except for gait speed and stride length, were obtained from the stance phase of the gait, which was defined as the period from heel strike (i.e., initial contact with vertical GRF > 20 N) to toe-off (i.e., vertical GRF < 20 N).[23]

Based on OpenSim simulations, peak muscle forces of gluteus maximus (GMax), gluteus medius (GMed), semimembranosus, biceps femoris long head (BFLH), rectus femoris, and gastrocnemius medialis during the stance phase were collected, along with the peak joint contact forces of the hip, knee (including medial and lateral knee contact force), and ankle, respectively. Muscle force and joint contact force data were then normalized against each subject's own body weight. For the Control group, mean values of the left and right side were calculated and used in future analysis.

Asymmetry index (ASI) was used to assess the asymmetry of lower limbs in knee OA patients, using the following formula[6]:

$$ASI = \frac{100 * |V_{affected} - V_{contralateral}|}{0.5 * |V_{affected} + V_{contralateral}|}$$

An ASI value of "0" indicates perfect symmetry between the two lower limbs, while a greater value of ASI indicates an increased magnitude of asymmetry. For healthy control, ASI values were also determined between the left and right legs.

Statistical Analysis

The Shapiro–Wilk test was used to evaluate the normality of the data sets. Levene's test was used to check for equality of variance. Differences between the two lower limbs were determined using a paired-sample t-test or Wilcoxon signed-rank test, depending on the distribution of data. Differences

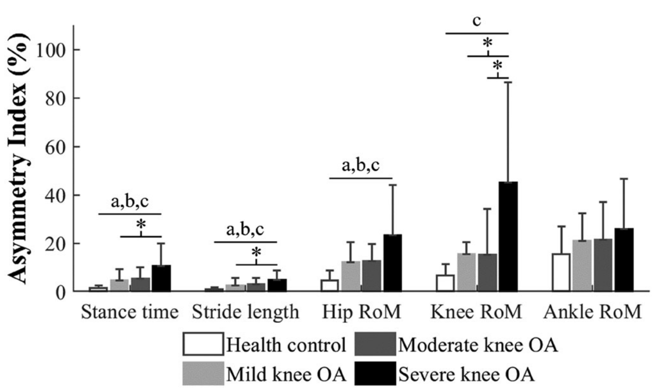

Fig. 1 Asymmetry index in kinematics. RoM: range of motion in the sagittal plane during stance phase. * indicates significant difference between knee OA groups ($p < 0.05$). (A) Mild knee OA was significantly different from healthy control ($p < 0.05$); (B) Moderate knee OA was significantly different from healthy control ($p < 0.05$); (C) Severe knee OA was significantly different from healthy control ($p < 0.05$)

among four subject groups (three patient groups and one control group) were analyzed using one-way analysis of variance (ANOVA) or the Kruskal–Wallis test. Tukey's post-hoc test or pairwise comparison with a Bonferroni correction was used for multiple comparisons across all subject groups. All statistical analyses were conducted using SPSS (v19, IBM Corp., Armonk, NY, USA). The significance level was set at $p < 0.05$.

Results

There was no significant difference in BMI between the knee OA patients and the healthy controls. Patients in the Severe group had a greater varus deformity ($169.7 \pm 5.9°$) compared to those in the Mild ($178.8 \pm 2.0°$) and Moderate groups ($177.8 \pm 3.0°$) ($p < 0.001$; Table 1). The asymmetry index (ASI) in kinematics and muscle force increased from mild to severe knee OA, but not in kinetics. No significant difference in ASI was observed between the Mild and Moderate groups.

Kinematics

The Severe group exhibited greater ASI in stance time and stride length ($p < 0.05$) compared to the Mild group. In addition, patients in the Severe group also had a significantly higher ASI in RoM_{knee} (45%) compared to the Mild and Moderate groups (15% respectively; $p < 0.001$). All three knee OA groups had greater ASI in stance time, stride length, and RoM_{hip} compared to healthy subjects ($p < 0.05$). Furthermore, the Severe group had a higher ASI in RoM_{knee} than the other groups ($p < 0.05$) (Figure 1). The Moderate and Severe groups both demonstrated significantly less stance time, RoM_{knee}, and RoM_{hip} on the more affected side compared to the contralateral side ($p < 0.05$) (Table 2).

Kinetics

No significant difference was observed in the ASI of kinetics across the three knee OA groups. All three knee OA groups had higher ASI in peak knee flexion moment, KAM, medial knee contact force (MCF), and lateral knee contact force (LCF) compared to healthy controls ($p < 0.05$) (Figure 2). Furthermore, significantly lower peak hip joint contact force on the more affected side compared to the contralateral side was observed in the Mild (more affected side: 3.80 ± 0.67 BW, contralateral side: 4.01 ± 0.58 BW), Moderate (more affected side: 3.67 ± 0.56 BW, contralateral side: 4.07 ± 0.81 BW), and Severe groups (more affected side: 3.66 ± 0.79 BW, contralateral side: 3.94 ± 0.64 BW) ($p < 0.05$). Only the Mild group exhibited significantly greater peak KAM (more affected side: 0.37 ± 0.15 N·m/kg, contralateral side: 0.43 ± 0.14 N·m/kg) and MCF (more affected side: 2.75 ± 0.82 BW, contralateral side: 3.18 ± 0.86 BW) on the more affected side ($p < 0.05$) (Table 3).

Muscle Forces

The Severe group had significantly greater ASI in peak forces of the GMed, GMax, rectus femoris, and gastrocnemius

TABLE 2 Mean (SD) kinematic data between lower limbs for knee OA patients

	Mild knee OA			Moderate knee OA			Severe knee OA			Healthy control
	More affected side	Contralateral side	Statistical value (*p* value)	More affected side	Contralateral side	Statistical value (*p* value)	More affected side	Contralateral side	Statistical value (*p* value)	
Speed (m/s)		0.90 (0.25)			0.76 (0.20)			0.56 (0.20)		1.23 (0.12)
Stance time (s)	0.75 (0.11)	0.76 (0.12)	−0.83 (0.410)	0.80 (0.12)	0.83 (0.13)	−3.77 (<0.001)[a]	0.95 (0.22)	1.03 (0.26)	269 (<0.001)[a]	0.63 (0.04)
Stride length (m)	1.07 (0.22)	1.08 (0.21)	293 (0.381)	0.98 (0.15)	0.99 (0.15)	−0.54 (0.595)	0.82 (0.20)	0.81 (0.19)	681 (0.678)	1.24 (0.11)
Hip flexion/extension RoM (°)	35.5 (5.4)	36.8 (6.7)	292 (0.378)	33.0 (5.8)	34.8 (6.3)	−2.18 (0.037)[a]	29.9 (7.5)	33.3 (7.1)	−2.98 (0.004)[a]	40.5 (4.6)
Knee flexion/extension RoM (°)	34.6 (6.2)	36.5 (7.3)	244 (0.107)	32.2 (8.1)	35.3 (5.2)	−3.08 (0.004)[a]	21.4 (9.7)	30.2 (8.0)	−6.75 (<0.001)[a]	40.0 (4.7)
Ankle flexion/ dorsiflexion RoM (°)	16.0 (4.8)	17.0 (5.3)	232 (0.072)	14.8 (3.1)	15.2 (3.1)	1.59 (0.123)	15.9 (5.9)	16.5 (4.7)	−0.81 (0.423)	14.4 (4.1)

Abbreviation: RoM, range of motion in stance phase.; *Note*: Statistical value refers to the t-value of paired-sample t-test or the v-value of Wilcoxon signed-rank test.; [a] Indicates significant difference between the more affected side and the contralateral side ($p < 0.05$).

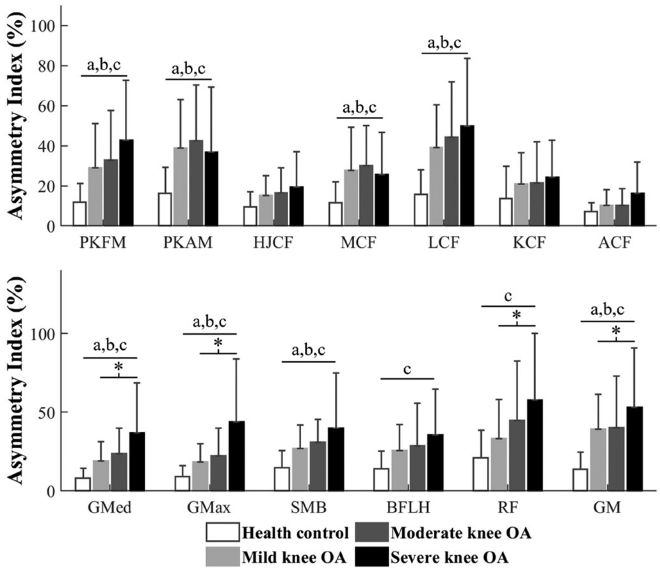

Fig. 2 Asymmetry index in kinetics. PKFM: peak knee flexion moment; PKAM: peak knee adduction moment; HCF: peak hip contact force; MCF: peak medial knee contact force; LCF: peak lateral knee contact force; KCF: knee contact force; ACF: peak ankle contact force. GMed: gluteus medius; GMax: gluteus maximus; SMB: Semimembranosus; BFLH: Biceps femoris long head; RF: rectus femoris; GM: gastrocnemius medialis. * indicates significant difference between knee OA groups ($p < 0.05$). (A) Mild knee OA was significantly different from healthy control ($p < 0.05$); (B) Moderate knee OA was significantly different from healthy control ($p < 0.05$); (C) Severe knee OA was significantly different from healthy control ($p < 0.05$)

medialis compared to the Mild group ($p < 0.05$). When compared to healthy controls, all three knee OA groups demonstrated higher ASI in peak forces of the GMed, GMax, semimembranosus, and gastrocnemius medialis ($p < 0.05$). Additionally, the Severe group showed greater ASI in peak forces of the BFLH and rectus femoris compared to healthy controls ($p < 0.01$) (Figure 2). The more affected side exhibited a significantly lower peak force of the GMed compared to the contralateral side in patients with mild (more affected side: 0.48 ± 0.09 BW, contralateral side: 0.52 ± 0.12 BW), moderate (more affected side: 0.45 ± 0.10 BW, contralateral side: 0.51 ± 0.15 BW) and severe knee OA (more affected side: 0.42 ± 0.15 BW, contralateral side: 0.47 ± 0.12 BW) ($p < 0.05$). Patients in the Severe group had significantly lower peak forces of the GMax, BFLH, and rectus femoris in the affected side compared to the contralateral side ($p < 0.05$) (Table 4).

Discussion

The present study evaluated the effects of varying degrees of knee OA severity on gait asymmetry in kinematics, kinetics, and muscle forces. The current findings suggest that increased gait asymmetry in kinematics and muscle forces

TABLE 3 Mean (SD) kinetic data between lower limbs for knee OA patients

	Mild knee OA			Moderate knee OA			Severe knee OA			
	More affected side	Contralateral side	Statistical value (p value)	More affected side	Contralateral side	Statistical value (p value)	More affected side	Contralateral side	Statistical value (p value)	Healthy control
Peak knee flexion moment (N·m/kg)	0.41 (0.16)	0.45 (0.15)	243 (0.103)	0.34 (0.12)	0.38 (0.16)	−1.61 (0.118)	0.30 (0.13)	0.34 (0.16)	530 (0.302)	0.55 (0.10)
Peak knee adduction moment (N·m/kg)	0.37 (0.15)	0.43 (0.14)	−2.29 (0.028)[a]	0.42 (0.16)	0.43 (0.16)	−0.15 (0.883)	0.57 (0.23)	0.55 (0.18)	0.49 (0.628)	0.37 (0.07)
Peak hip contact force (BW)	3.80 (0.67)	4.01 (0.58)	484 (0.045)[a]	3.67 (0.56)	4.07 (0.81)	387 (0.005)[a]	3.66 (0.79)	3.94 (0.64)	2.23 (0.03)[a]	4.03 (0.72)
Peak knee contact force (BW)	3.20 (0.93)	3.41 (0.87)	477 (0.059)	2.99 (0.58)	3.33 (1.24)	292 (0.399)	2.65 (0.64)	2.84 (0.68)	793 (0.135)	4.05 (0.92)
Peak MCF (BW)	2.75 (0.82)	3.18 (0.86)	536 (0.005)[a]	2.71(0.79)	3.05 (1.07)	335 (0.09)	3.38 (0.96)	3.25 (0.94)	553 (0.417)	3.23 (0.59)
Peak LCF (BW)	1.01 (0.29)	1.01 (0.32)	349 (0.976)	0.88 (0.35)	0.92 (0.32)	0.61 (0.546)	0.70 (0.29)	0.75 (0.34)	697 (0.569)	1.27 (0.24)
Peak ankle contact force (BW)	4.71 (1.02)	4.78 (0.86)	398 (0.492)	4.75 (0.77)	4.72 (0.76)	−0.28 (0.779)	3.93 (0.82)	4.08 (0.76)	1.35 (0.182)	6.10 (1.27)

Abbreviations: LCF: Lateral knee contact force; MCF: Medial knee contact force.; *Note*: Statistical value refers to the t-value of paired-sample t-test or the v-value of Wilcoxon signed-rank test.; [a] Indicates significant difference between the more affected side and the contralateral side ($p < 0.05$).

TABLE 4 Mean (SD) muscle force between lower limbs for knee OA patients

	Mild knee OA			Moderate knee OA			Severe knee OA			
	More affected side	Contralateral side	Statistical value (*p* value)	More affected side	Contralateral side	Statistical value (*p* value)	More affected side	Contralateral side	Statistical value(*p* value)	Healthy control
GMed (BW)	0.48 (0.09)	0.52 (0.12)	−2.10 (0.042)[a]	0.45 (0.10)	0.51 (0.15)	140 (0.034)[a]	0.42 (0.15)	0.47 (0.12)	−2.03 (0.048)[a]	0.44 (0.06)
GMax (BW)	0.45 (0.14)	0.48 (0.18)	−1.83 (0.076)	0.44 (0.22)	0.47 (0.25)	170 (0.13)	0.49 (0.25)	0.57 (0.20)	406 (0.026)[a]	0.35 (0.10)
Semimembranosus (BW)	0.41 (0.20)	0.39 (0.19)	395 (0.517)	0.37 (0.16)	0.37 (0.16)	236 (0.824)	0.38 (0.19)	0.42 (0.20)	449 (0.07)	0.59 (0.14)
BFLH (BW)	0.21 (0.11)	0.20 (0.11)	413 (0.357)	0.18 (0.09)	0.18 (0.08)	208 (0.444)	0.16 (0.07)	0.20 (0.11)	364 (0.008)[a]	0.39 (0.05)
Rectus femoris (BW)	0.64 (0.27)	0.64 (0.22)	0.037 (0.97)	0.53 (0.27)	0.59 (0.26)	−1.00 (0.325)	0.41 (0.28)	0.55 (0.25)	279 (<0.001)[a]	0.92 (0.50)
Gastrocnemius medialis (BW)	1.23 (0.70)	1.34 (0.64)	−1.33 (0.192)	1.03 (0.47)	1.20 (0.69)	209 (0.456)	0.57 (0.41)	0.69 (0.41)	−1.96 (0.056)	1.84 (0.51)

Abbreviations: BFLH, Biceps femoris long head; GMed, gluteus medius; GMax, gluteus maximus.; *Note*: Statistical value refers to the t-value of paired-sample t-test or the v-value of Wilcoxon signed-rank test.; [a] Indicates significant difference between the more affected side and the contralateral side ($p < 0.05$).

may be linked to the worsening severity of knee OA, from mild to severe. However, the current study did not find evidence that the severity of knee OA has an effect on the between-limb differences in gait kinetics.

Kinematics

The current study found that knee OA patients had a higher degree of gait asymmetry in stance time and stride length, as the severity of their knee OA worsens. Patients with severe knee OA exhibit greater gait asymmetry in stance time compared to those with mild knee OA. During self-paced walking, those with severe knee OA often shorten the contact time of their more affected side in an attempt to alleviate the pain, contributing to the observed increase in asymmetry of stance time.[24]

Regarding lower limb joint RoM, knee OA patients exhibited increased gait asymmetry based on their grades of knee OA conditions, particularly the RoM_{knee}. Patients with severe knee OA had substantially greater gait asymmetry in RoM_{knee}, compared to other groups. More confined RoM_{knee} is commonly seen as a result of pain and deficiency associated with knee OA.[25] Additionally, limited RoM_{knee} has been linked to a varus knee alignment rather than a neutral one.[26] For patients with severe knee OA, the more affected side knee joint experiences pain and greater varus angles, leading to reduced RoM_{knee} and increased gait asymmetry. This result supports previous finding that the more affected knee shows higher peak knee flexion angle than the contralateral knee.[3] With respect to RoM_{hip}, all knee OA patients exhibited greater gait asymmetry than healthy controls, as the more affected side hip had a lower RoM_{hip}. This decrease in RoM_{hip} may be due to the deficiency in the hip joint motions. These findings further suggest that the impact of knee OA extend beyond the knee joint and could potentially affect other adjacent joints, such as the hip joint.[14] However, patients with mild knee OA demonstrated no between-limb kinematic difference, indicating that between-limb differences in this group may not be evident based on kinematic parameters.

Kinetics

Although no significant difference regarding gait asymmetry in joint loading was observed across all three knee OA groups, all patients had greater gait asymmetry in peak knee flexion moment, KAM, MCF, and LCF compared to healthy controls. These results are consistent with previous study that knee OA patients exhibit higher gait asymmetry in peak knee flexion moment than healthy controls.[12] Our findings indicate that asymmetry in knee joint loadings may be closely linked to the ongoing degeneration at the knee joint. Interestingly, only the Mild group exhibited greater peak KAM and MCF on the contralateral side, which could be due to the knee alignment changes associated with different levels of severity. Literature suggests that larger varus knee angles are associated with increased KAM,[27] and that knee joint loading can be transferred from the lateral compartment to the medial compartment in severe varus deformity,[28] resulting in increased MCF. For patients with mild knee OA with relatively neutral knee alignments, the greater peak KAM and MCF on the contralateral side may be results of reducing the pain on the more affected side by shifting loads from the more affected side to the contralateral side.[3,5] Patients with severe knee OA demonstrated more severe varus deformity in both lower limbs, resulting in greater peak KAM and MCF in both sides. Therefore, the effect of the selected gait modification where the contralateral side bears more loading may be reduced in the presence of the larger varus angles in both sides, since the more affected knee may also bear the load. The current findings are consistent with previous studies that found gait modifications to be effective in reducing knee loading only in patients with less severe knee OA.[29] Therefore, the between-limb differences could be observed with peak KAM and MCF at the mild stage of knee OA. However, for those with severe varus deformity, these two parameters may not reflect the between-limb differences and instead reveal the load on the medial knee.[27]

With respect to the hip joint contact force, all knee OA patients had greater peak values on the contralateral side compared to the more affected side, suggesting that gait modification may reduce load at the hip joint, regardless of knee OA severity. For the ankle joint contact force, no significant difference in gait asymmetry was observed across four groups or between two limbs of knee OA patients. These findings suggest that knee OA patients may modify their walking patterns to redistribute loads from the more affected side to the contralateral side.[3] However, this mechanism may be limited to the hip and knee joints.

Muscle Force

This study showed that gait asymmetry in muscle forces increased with the severity levels of knee OA. Our observations support the previous finding that quadriceps strength asymmetry is associated with increased gait asymmetry.[13,30] Patients with severe knee OA showed greater gait asymmetry in peak forces of the GMax, GMed, rectus femoris, and gastrocnemius medialis was found in patients with severe knee OA compared to those in the Mild group. This gait asymmetry may be due to greater peak muscle forces found on the contralateral side compared to the more affected sides. This greater muscle force can lead to increased load at the knee and hip joints on that side, and potentially worsen the OA on the contralateral joints.[3,5]

It is worth noting that the peak muscle force of GMed was consistently higher on the contralateral side compared to the more affected side across all three knee OA groups, in line with the peak hip contact force. The between-limb difference in peak hip contact force may be due to the contribution of the GMed to hip movement. These results may suggest that the hip joint plays an important role in gait modification between two lower limbs in varying degrees of knee OA severity. This gait modification can be observed with the peak hip contact force and GMed muscle force, regardless of the severity of knee OA.

Strengths and Limitations

The strength of our study was that the results were quantified using gait asymmetry, which may help evaluate bilateral limbs. This study also has several limitations. Firstly, gait speed was not considered a covariate.[31] Although gait speed has been reported to impact lower limb biomechanics,[32] gait speed is inherently linked to the worsening of knee OA and changes with knee OA severity.[31,33] Secondly, the musculoskeletal model used did not include ligaments, which may lead to an underestimation of knee contact force. While a significant lateral collateral ligament contribution to joint stability in the frontal plane has been predicted using models, these same models also predicted lateral compartment unloading, which would tend to induce greater ligament strain.[34] In addition, considering the cross-sectional study design, the current findings may not be applicable to improve the understanding of the causal relationships between these biomechanical parameters and the severity levels of knee OA. Future longitudinal studies are needed to better understanding these relationships. Lastly, a lower limb emphasized marker set was used in this study, rather than a full-body marker set. Since trunk lean and trunk movement asymmetry have been reported in knee OA patients,[13,35] investigations of gait asymmetry using a full-body marker set may provide deeper insights into the specific gait modification strategy employed by knee OA patients with varying severity levels.

Conclusion

Increased gait asymmetry was observed in kinematics and muscle forces, but not in kinetics with the worsening severity level of knee OA. This increased gait asymmetry accompanies modified gait pattern that patients distribute more load to the contralateral side. The gait modification accompanied with asymmetry may be detectable by monitoring the peak hip contact force and GMed muscle force, regardless of knee OA severity. In clinical settings, peak hip contact force and GMed muscle force may be used to evaluate gait asymmetry in patients with knee OA.

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Conflicts of Interest

The authors declare no conflict of interest.

Authors' Contributions

Conception and design: Junqing Wang, Kang Li, and Yong Nie.
Collection and assembly of data: Junqing Wang, Yong Nie.
3D gait and musculoskeletal analysis: Junqing Wang, Shiqi Li.
Analysis and interpretation of the data: Junqing Wang, Qinsheng Hu, Kang Li, and Yong Nie.
Drafting and critical revision of the article: Junqing Wang, Qinsheng Hu, Kang Li, Yong Nie, Ruoliang Tang, Qian Deng, Chenyu Wu, and Bin Shen.

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