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Research article

Comparison of knee biomechanical characteristics during gait between patients with knee osteoarthritis and healthy individuals

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ABSTRACT

Objective: This study aim to quantify the differences in knee biomechanics during gait between knee osteoarthritis (KOA) patients and healthy individuals.
Methods: Twenty KOA patients (4 males and 16 females, 66.2 ± 7.7 years) and twenty controls (16 males and 4 females, 64.8 ± 5.4 years) were recruited for gait test using the motion capture system and force-platform system. The spatiotemporal parameters, knee kinematics and kinetics, and tibiofemoral contact force (TFCF) were calculated using an improved musculoskeletal model.
Results: KOA patients walked with reduced speed (48.6 %), stride length (32.9 %), stride height (33.0 %), time proportions of single-support phases (19.2 %), increased gait cycle time (31.0 %), time proportions of stance (8.5 %) and double-support phases (57.7–75.9 %). KOA patients had significant smaller peak flexion angle (29.1 %), flexion ROM (50.6 %) and peak flexion moment (90.2 %), greater peak adduction moment (KAM) (40.7 %), peak rotation moments (KRM) (50.0 %), KAM impulse (106.2 %) and KRM impulse (126.0 %). In proximodistal direction, greater medial TFCF impulse (238 %), total and medial first-peak TFCF (9.6 % and 15.2 %), and smaller lateral peak TFCF (33.3 %) and TFCF impulse (38.4 %) were found in KOA patients. Besides, significant differences were found in the total, medial and lateral peak TFCFs and TFCF impulses in mediolateral direction, and the medial and lateral TFCFs and TFCF impulses in anteroposterior direction.
Conclusions: Significant differences were found in the spatiotemporal parameters, knee kinematics and kinetics, and TFCF between the two groups. The results of this study have important implication for clinicians and rehabilitation physicians. These quantified biomechanical differences can provide data support for the personalized and quantified rehabilitation strategies, give suggestions for the exercises of KOA patients, help monitor disease, evaluate surgical treatment, and develop more effective preoperative planning and postoperative rehabilitation strategies.

1. Introduction

Knee osteoarthritis (KOA) is a prevalent, chronic, degenerative and multifactorial disease associated with pain, stiffness, and physical disability, and it is a significant public health problem in the word [1]. As the global population ages and obesity increases, the

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incidence, burden, and socioeconomic impact of KOA have been growing and are expected to continue growing [2]. Epidemiological study shows that the global prevalence of KOA was 16.0 % (13.4 % in Europe, and 19.2 % in Asia) and the global incidence was 203 per 10,000 persons, respectively [3]. Abnormal knee loading during gait has been designated as the main biomechanical factor for the initiation and progression of KOA [4]. Therefore, quantitative analysis of the biomechanical differences between patients with KOA and healthy individuals is significant for understanding the initiation of KOA, detecting the progression of KOA, and providing guidance for the treatments of KOA such as knee brace, gait modification and total knee replacement (TKR) surgery.

Gait analysis has become an important tool in the evaluation of KOA. In the past few years, several related researchers have been done to compare the gait biomechanical difference between patients with KOA and healthy controls. Aleena et al. [5] analyzed the total walking distance covered in the same amount of time, distance covered in each minute, walking speed and walking-related performance fatigability in patients with KOA as compared to healthy subjects. Li et al. [6] compared the walking speed, stride frequency, stride length, the time of gait cycle, and time proportion of double support phase between patients with KOA and healthy people. Both two studies only investigated the gait spatiotemporal parameters, but did not compared the kinematics, kinetics and tibiofemoral contact forces of knee joint. Yaguchi et al. [7] studied the differences in walking speed, stride length and foot kinematics in the sagittal plane during gait between patients with KOA and healthy controls. Dai et al. [8] investigated the differences in spatiotemporal parameters, knee joint angle in sagittal plane, hip joint angle in sagittal and coronal planes, and ankle joint angle in three anatomical planes between patients with KOA and femoral varus deformity as compared to healthy subjects. Ismailidis et al. [9] compared the walking speed, stride duration, stride length, cadence and knee flexion angle in patients with KOA and healthy group. However, further spatiotemporal, kinematics and kinetic characteristics of the knee joint were not analyzed in these papers. Barai et al. [10] compared the spatiotemporal, knee joint angle and moment between patients with mild KOA, patients with severe KOA and healthy controls. But the tibiofemoral contact force (TFCF), as the most important biomechanical evaluation parameters of KOA, was not involved in their study. Kumar et al. [11] investigated the walking speed, knee joint angle and moment in sagittal and coronal planes, and the knee contact loads in superior-inferior direction during gait in patients with KOA and healthy controls. However, the study did not discuss the knee joint angle and moment in three anatomical planes and the knee contact loads in three anatomical directions.

Because of the three-dimensional nature of knee joint, the biomechanical difference between patients with KOA and healthy individuals is manifested in three-dimensional space. Although the researches mentioned above have compared some biomechanical parameters of knee joint between patients with KOA and healthy individuals, no previous studies have systematically and comprehensively quantified the differences in gait spatiotemporal parameters, knee kinematics, knee kinetics, and knee contact forces in three-dimensional space.

The objective of this case-control study was to investigate and quantify the differences in the gait spatiotemporal parameters (self-selected walking speed, time of gait cycle, stride length, stride height and time proportion of each gait phase), knee kinematics (knee joint angle) and kinetics (knee joint moment) in three anatomical planes, and TFCF on the total, medial and lateral compartments in three anatomical directions during gait between patients with KOA and healthy individuals. It was hypothesized that many knee biomechanical parameters changed clearly and fewer parameters did not change during gait in patients with KOA compared to healthy individuals.

2. Methods

2.1. Study design, setting and ethics

A case-control comparative design was conducted to compare the gait spatiotemporal parameters, knee kinematics, knee kinetics and TFCF in patients with KOA and healthy individuals. All patients with KOA were recruited from the Joint Surgery Department, Honghui Hospital, Xi'an Jiaotong University, Xi'an, China, and the healthy controls were recruited from the local community via advertisements, from October 2023 to May 2024.

This study had ethical approval from the Ethics Committee of Honghui Hospital, Xi'an Jiaotong University (No: 202309006 and data of approval: October 9, 2023). All recruited participants provided written informed consent prior to the experiments.

2.2. Participants and sample size

The patients with KOA and healthy controls were assessed using the Kellgren-Lawrence (KL) grading scale by an orthopedic surgeon at our hospital based on standing lower-limbs-full-length anterior-posterior radiography [7]. The inclusion criteria for patients with KOA were as follows: (1) an age between 55 and 80 years; (2) osteoarthritis on the medial compartment of both knees with Kellgren-Lawrence (KL) grades 3–4 osteoarthritis [12,13]; (3) ability to walk independently without assistive devices. The inclusion criteria for healthy individuals were as follows: (1) an age between 55 and 80 years; (2) no history of pain, injuries and surgery that affected the lower limb; (3) ability to walk independently with no limitations. The exclusion criteria for all recruited participants were as shown: (1) any history of neuromuscular diseases that affected gait; (2) any history of rheumatoid arthritis; (3) any history of cardiovascular disorders that affected gait; (4) a BMI > 35 kg/m^2.

The sample size for this study was determined based on a previous study published by Yaguchi et al. [7]. According to the effect sizes reported by Yaguchi [7] and the results of the pilot study, to achieve power of 0.8 with α at 0.05, the sample size required for both the KOA group and healthy control group is thirteen. Thus, a total sample of forty participants, including twenty patients with KOA and twenty age-matched healthy individuals, was recruited for this study.

2.3. Instrumentation

As shown in Fig. 1, a ten-camera motion capture system (Vicon version 3.3, Vicon Motion Analysis Inc., Oxford, UK) was utilized to capture 3-dimensional (3D) kinematic data of participants. Thirty-seven hemispherical reflective markers (15 mm) were attached to the bony landmarks of the participants' full-body under the guidance of orthopedic surgeon according to our previously published marker setting [14,15]. The 3D coordinate values of all marker positions were recorded during data acquisition with a sampling frequency of 100 Hz. A three-force-platform system (BP 600600, AMTI, Watertown, MA, USA) embedded in the laboratory floor was utilized to capture 3D ground reaction forces (GRFs) with a sampling frequency of 1000 Hz. The two systems were connected to MX data conversion console through MX connection line to realize synchronous signal acquisition between two systems. The Vicon Nexus software (version 2.6.1, Vicon Motion Systems Ltd, Oxford, UK) was utilized for data acquisition on the PC. The 3D coordinate values of all marker positions and 3D GRFs during data acquisition were used for subsequent simulation calculations.

2.4. Procedures

Participants were instructed not to perform any strenuous physical activity 48h before evaluation. On the days of the experiment, the temperature of the gait laboratory was controlled at 26 ± 3 °C to avoid the influence of uncomfortable feeling on the participants' gait. Prior to data acquisition, 30 min warm-up period was used to familiarize the participant with the task. A 5 min rest followed, in which the reflective markers were attached. Then, the static data were collected with each participant standing on a single force plate with their feet placed shoulder-width apart. After the static standing trial was completed, each participant was asked to walk barefoot at a self-selected comfortable walking speed, stride length and stride height along the 15-m walkway with force platforms. For each participant, 20 repeated trials were performed to represent the mean. To remove the effect of fatigue, a 2 min rest period was provided between two trials.

For subsequent musculoskeletal modelling and gait simulation, the standing lower-limbs-full-length anterior-posterior radiography was conducted by one radiographer for all participants. To ensure the patella was in the center of the femoral condyles, the participants were asked to stand in a forward knee position [16]. The radiographic data acquisition was conducted on the same day as the gait data acquisition.

2.5. Data processing

All modelling and simulation processes were performed in AnyBody software (AnyBody version 7.2, Aalborg, Denmark). An improved musculoskeletal model based on the generic musculoskeletal model (Lower Extremity, Plug-in-gait_Simple) in AnyBody software was employed to analyze the gait data. The generic musculoskeletal model was extracted from AnyBody Managed Model Repository (AMMR version 2.2.1) and the two legs of them were based on Twente Lower Extremity Model (TLEM version 2.1) [17,18]. The generic model was comprised of 15 body segments (skull, cervical spine, thorax, lumbar spine, pelvis, left and right thighs, shanks, patellas, talus and feet). The body segments were connected to each other by joints, such as 3-DOF spherical joints at the hip joint and 1-DOF (flexion-extension) revolute joints at the neck, tibiofemoral, patellofemoral, ankle and subtalar joints. 55 muscle-tendon-units (MTUs) represented by 160 Hill-type 1D string elements were utilized to actuate the musculoskeletal model. The detail about the generic musculoskeletal model could be found in Ref. [17].

To better fit our research, two improvements were made in our improved musculoskeletal model. Firstly, the knee and ankle joints

Fig. 1. Position of the reflective makers (**A** frontal view and **B** back view) and gait experiment (**C**).

of the generic musculoskeletal model were updated to 3-DOF in our model for better gait simulation analysis of patients with KOA. Secondly, the one-point contact knee joint of the generic musculoskeletal model was updated to the medial-lateral two-point contact knee joint for better calculation of the TFCF on the medial and lateral compartments. The locations of the medial and lateral contact points were obtained from the radiography of each participant. Based on the radiography, the minimal point-to-point distances between the points on the tibial plateau and opposing medial or lateral condyles were calculated. The contact points on each tibial plateau were identified based on a verified weighted center of proximity algorithm, and the accuracy of this algorithm was reported <3 mm [19,20]. Because of the lack of acquisition equipment, there were some assumptions about the contact of the knee joint in our model. Firstly, the medial and lateral contacts were assumed to be point-to-point contacts. Secondly, the contact centers were assumed to be fixed during gait and the same as those in the upright standing state.

The whole gait simulation process of the improved musculoskeletal model could be summarized as PI (parameter identification), MT (marker tracking) and ID (inverse dynamics). Firstly, PI was performed by changing the anthropometry of model so that it matched the participant as closely as possible, based on the differences of the 3D coordinate values between marker data of the static standing trial and the musculoskeletal model. The thigh length, shank length and pelvis width were further verified by comparing the values of the musculoskeletal model after PI with those measured by the radiography of each participant. Secondly, MT was utilized to track the marker trajectories during the gait process and calculate the gait spatiotemporal parameters and kinematics parameters of knee joint in three anatomical planes utilizing a set of Karush-Kuhn-Tucker optimality conditions. Finally, the GRFs and results of MT were inputted into the ID analysis to compute the knee joint moment in three anatomical planes and TFCF on the total, medial and lateral compartments in three directions.

To assess the predicted TFCF, the predicted values based on our musculoskeletal model in healthy individuals were compared with the corresponding values reported in previous studies [11,21,22]. Similar curve trends between predictions and reports in previous studies were found for the TFCF on the total, medial and lateral compartments [11,21,22]. Besides, the predicted peak TFCF on the total, medial and lateral compartments were close to previously values reported by Kumar et al. [11] (total: 3.16–4.17BW; medial: 2.12–2.61 BW; lateral: 0.95–1.65 BW), Winby et al. [21] (total: 3.2–4.9 BW; medial: 2.1–3.3 BW; lateral: 0.92–3.06 BW), and Richards et al. [22] (total: 3.19 ± 0.40BW; medial: 1.86 ± 0.29 BW; lateral: 1.35 ± 0.20BW). In general, the musculoskeletal modelling and gait simulation presented in this study was able to predict the TFCF with a reasonable accuracy.

2.6. *Statistical analysis*

The spatiotemporal parameters and knee joint angle were analyzed for each subject using all 15-m walkway data. The knee joint moment and TFCF were analyzed for each subject using the middle 3 gait cycle data of the 15-m walkway data, which had 3D GRFs data. All spatiotemporal parameters, knee angle, knee moment and TFCF during gait were expressed as mean ± standard deviation (SD). The curves of knee angle, knee moment and TFCF were normalized from 0 % to 100 % of the gait cycle (from heel strike to next heel strike of the same foot). The knee moment were expressed in unit of BW × Ht (Body weight × Height) and the TFCF were expressed in unit of BW (Body weight). The Shapiro-Wilk test was used to check the normal distribution of data. For parametric data, Student's *t*-test was used for comparison between patients with KOA and healthy individuals. For non-parametric data, the non-parametric Wilcoxon Signed-rank test was performed to detect statistically significant differences between patients with KOA and healthy individuals. A *p*-value of <0.05 was considered statistical significance for both parametric and non-parametric data. Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS version 16, SPSS Inc., Chicago, IL, USA) software.

3. Results

3.1. *Demographic and clinical characteristics of the study participants*

Twenty KOA patients and twenty healthy individuals were recruited in this study. Table 1 shows the demographic and clinical characteristics of the recruited participants, including sex, age, height, weight, BMI, KL grades and standing hip-knee-ankle angle.

Table 1
Demographic and clinical characteristics of the recruited participants.

	Patients with KOA	Healthy individuals
n	20	20
Sex (male: female)	4:16	6:14
Age (years)	66.2 ± 7.7	64.8 ± 5.4
Height (cm)	155.7 ± 6.4	158.1 ± 5.2
Weight (kg)	66.8 ± 8.7	65.7 ± 4.6
BMI (kg/m^2)	27.6 ± 3.2	26.3 ± 2.1
Kellgren-Lawrence (KL) grades	KL grade 3: 8 knee joints KL grade 4: 32 knee joints	KL grade 0: 40 knee joints
Standing hip-knee-ankle angle (deg)	171.3 ± 2.9	178.3 ± 1.7

3.2. Comparison of gait spatiotemporal parameters

Table 2 shows the comparison of the self-selected walking speed, time of gait cycle, stride length and stride height between patients with KOA and healthy individuals. It could be observed that the self-selected walking speed, stride length and stride height in patients with KOA were significantly lower than those in healthy individuals (self-selected walking speed: 0.609 ± 0.168 m/s vs. 1.185 ± 0.139 m/s, $p < 0.001$; stride length: 0.856 ± 0.238 m vs. 1.275 ± 0.090 m, $p < 0.001$; stride height: 0.150 ± 0.040 m vs. 0.224 ± 0.017 m, $p < 0.001$). The time of gait cycle was significantly larger in patients with KOA than in healthy individuals (1.417 ± 0.194 s vs. 1.082 ± 0.078 s, $p < 0.001$). Compared to the healthy individuals, the self-selected walking speed, stride length and stride height in patients with KOA reduced by 48.6 %, 32.9 % and 33.0 %, and the time of gait cycle increased by 31.0 %, respectively.

The comparison of the time proportion of each gait phase between patients with KOA and healthy individuals is shown in Fig. 2. Significant differences were observed between patients with KOA and healthy individuals: patients with KOA had larger time proportion of stance phase (64.90 ± 4.85 % vs. 59.79 ± 1.57 %, $p < 0.001$), smaller time proportion of swing phase (35.14 ± 4.08 % vs. 40.22 ± 1.55 %, $p < 0.001$) than healthy individuals. In the stance phase, the two double-legs support phases exhibited larger time proportions (first double-legs support phase: 16.75 ± 4.57 % vs. 9.52 ± 1.59 %, $p < 0.001$; second double-legs support phase: 15.39 ± 4.88 % vs. 9.76 ± 1.32 %, $p < 0.001$) and the single-leg support phase exhibited a smaller time proportion (32.73 ± 6.13 % vs. 40.50 ± 1.83 %, $p < 0.001$) in patients with KOA than healthy individuals. Compare to the healthy individuals, the time proportion of stance phase, first double-legs support phase, and second double-legs support phase in patients with KOA were significantly increased by 8.5 %, 75.9 % and 57.7 %, and the time proportion of swing phase and single-leg support phase were significantly decreased by 12.6 % and 19.2 %, respectively.

3.3. Comparison of knee joint angle in three anatomical planes

Fig. 3 shows the comparison of knee angle curves, peak angle and range of motion (ROM) between patients with KOA and healthy individuals in three anatomical planes. In the sagittal plane, the knee flexion angle curves of patients with KOA and healthy individuals followed a similar pattern, as shown in Fig. 3I(A): a first peak of knee flexion (Peak 1) and a first peak of knee extension (Peak 2) in the stance phase, maximum knee flexion at middle of swing phase (Peak 3), and minimum knee flexion at the end of swing phase (Peak 4). There were significant difference in the magnitude of knee flexion angle between patients with KOA and healthy individuals throughout the whole gait cycle ($p < 0.001$). Fig. 3II(A) displays the comparison of peak knee flexion and ROM between patients with KOA and healthy individuals. It was obvious that patients with KOA had larger Peak 1 (22.96 ± 5.61 deg vs. 13.72 ± 5.91 deg, $p < 0.001$), Peak 2 (21.51 ± 6.82 deg vs. 4.79 ± 4.45 deg, $p < 0.001$) and Peak 4 (15.94 ± 5.20 deg vs. 2.42 ± 2.38 deg, $p < 0.001$), and smaller Peak 3 (48.56 ± 11.89 deg vs. 68.48 ± 4.08 deg, $p < 0.001$) than healthy individuals. The ROM of whole gait cycle, swing phase and stance phase in patients with KOA were significantly lower than those in healthy individuals (whole gait cycle or swing phase: 32.62 ± 11.98 deg vs. 66.06 ± 4.24 deg, $p < 0.001$; stance phase: 20.95 ± 9.87 deg vs. 33.63 ± 4.92 deg, $p < 0.001$). In the middle of stance phase, patients with KOA had very little variation in knee flexion angle compared to healthy individuals ($p < 0.001$) (Fig. 3I(A)). Compare to the healthy individuals, the values of Peak 1, Peak 2 and Peak 4 were significantly increased by 0.673 times, 3.491 times and 5.587 times, the value of Peak 3 was significantly decreased by 29.1 %, and the ROM of whole gait cycle/swing phase and stance phase were significantly decreased by 50.6 % and 37.7 %, respectively.

In the coronal plane, the knee adduction angle was significantly greater in patients with KOA throughout the whole gait cycle than in healthy individuals due to the inherent varus ($p < 0.001$) (Fig. 3I(B)). Similar to the knee flexion angle curves, we extracted the feature points of knee adduction angle curves, including the peak adduction angle in stance phase (Peak 1), peak knee abduction angle in stance phase (Peak 2), maximum knee adduction/abduction angle at middle of swing phase (Peak 3), and minimum knee adduction at the end of swing phase (Peak 4). It was evident that the Peak 1, Peak 2, Peak 3 and Peak 4 in patients with KOA were significantly greater than those in healthy individuals (Peak 1: 14.06 ± 4.43 deg vs. 1.54 ± 4.13 deg, $p < 0.001$; Peak 2: 12.57 ± 5.51 deg vs. −0.19 ± 2.69 deg, $p < 0.001$; Peak 3: 16.05 ± 6.47 deg vs. −3.93 ± 9.28 deg, $p < 0.001$; Peak 4: 10.58 ± 3.82 deg vs. −0.08 ± 2.69 deg, $p < 0.001$) (Fig. 3II(B)). However, there was no significant difference in the ROM of whole gait cycle, swing phase or stance phase between patients with KOA and healthy individuals (whole gait cycle or swing phase: 5.47 ± 4.80 deg vs. 6.61 ± 4.05 deg, $p > 0.05$; stance phase: 4.66 ± 2.59 deg vs. 1.73 ± 4.55 deg, $p > 0.05$).

In the transverse plane, no significant difference was seen when comparing the knee internal rotation angle in patients with KOA and healthy individuals during the 4.4 %–94.9 % gait cycle ($p > 0.05$) (Fig. 3I(C)). Significant difference existed only around the initial stance time (0–4.3 % and 94.8–100 % gait cycle, $p < 0.05$). We compared the peak angle, initial angle at 0 % gait cycle, and ROM of whole gait cycle, swing phase and stance phase between patients with KOA and healthy individuals, as shown in Fig. 3II(C). There were no significant differences in the peak angle (10.71 ± 6.13 deg vs. 9.81 ± 5.98 deg, $p > 0.05$) and ROM of whole gait cycle (11.23 ±

Table 2
Comparison of the self-selected walking speed, time of gait cycle, stride length and stride height between patients with KOA and healthy individuals.

	Patients with KOA	Healthy individuals	*p*-value
Self-selected walking speed (m/s)	0.609 ± 0.168	1.185 ± 0.139	$p < 0.001$
Time of gait cycle (s)	1.417 ± 0.194	1.082 ± 0.078	$p < 0.001$
Stride length (m)	0.856 ± 0.238	1.275 ± 0.090	$p < 0.001$
Stride height (m)	0.150 ± 0.040	0.224 ± 0.017	$p < 0.001$

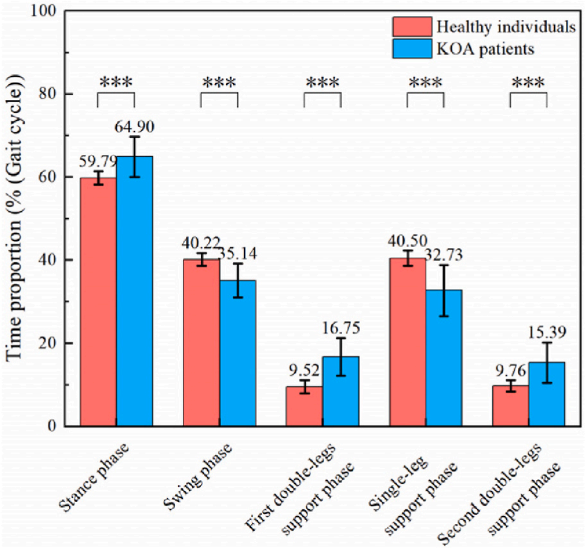

Fig. 2. Comparison of the time proportion of each gait phase between patients with KOA and healthy individuals. Asterisk *** represent significance level at 0.001.

5.34 deg vs. 9.41 ± 4.08 deg, $p > 0.05$), stance phase (6.91 ± 3.87 deg vs. 6.77 ± 3.81 deg, $p > 0.05$) and swing phase (11.23 ± 5.34 deg vs. 8.88 ± 3.67 deg, $p > 0.05$) between patients with KOA and healthy individuals. For the initial angle at 0 % gait cycle, a smaller value was observed in patients with KOA than in healthy individuals (−0.01 ± 3.59 deg vs. 4.52 ± 5.75 deg, $p = 0.003$).

3.4. Comparison of knee joint moment in three anatomical planes

The comparison of knee moment curves, peak moment and moment impulse between patients with KOA and healthy individuals in three anatomical planes is shown in Fig. 4. In the sagittal plane, knee flexion moment curves appeared to be very similar in patients with KOA and healthy individuals, with first peak knee flexion moment near heel strike (Peak 1), first peak knee extension moment near the end of first double-legs support phase (Peak 2), second peak knee flexion moment near the end of single-legs support phase (Peak 3), second peak knee extension moment near the end of stance phase (Peak 4), and third peak knee flexion moment during the terminal swing phase (Peak 5) (Fig. 4I(A)). The comparison of the peak knee moments between patients with KOA and healthy individuals is shown in Fig. 4II(A). It could be observed that the Peak 1, Peak 3 and Peak 5 in patients with KOA were significantly smaller than those in healthy individuals (Peak 1: 0.0055 ± 0.0034 BW × Ht vs. 0.0176 ± 0.0035 BW × Ht, $p < 0.001$; Peak 3: 0.0028 ± 0.0125 BW × Ht vs. 0.0287 ± 0.0106 BW × Ht, $p < 0.001$; Peak 5: 0.0057 ± 0.0019 BW × Ht vs. 0.0120 ± 0.0017 BW × Ht, $p < 0.001$). Compare to the healthy individuals, the Peak 1, Peak 3 and Peak 5 were significantly reduced by 68.8 %, 90.2 % and 52.5 %, respectively. No significant difference were existed in the Peak 2 and Peak 4 between patients with KOA and healthy individuals (Peak 2: −0.0065 ± 0.0063 BW × Ht vs. −0.0058 ± 0.0114 BW × Ht, $p = 0.589$; Peak 4: −0.0021 ± 0.0037 BW × Ht vs. −0.0059 ± 0.0034 BW × Ht, $p = 0.063$).

As shown in Fig. 4I(B), there was a difference in the pattern of knee adduction moment (KAM) curves between patients with KOA and healthy individuals. For the healthy individuals, the curves possessed two peaks (Peak 1 and Peak 3) and one valley (Peak 2) during the stance phase. But the relatively flat curves could be seen in patients with KOA during the middle of stance phase. During the swing phase, the KAM of both patients with KOA and healthy individuals tended to zero. We extracted the corresponding KAM of patients with KOA at the time points similar to the peak KAM of healthy individuals, and compared the values between patients with KOA and healthy individuals, as shown in Fig. 4II(B). It was evident that the Peak 1, Peak 2 and Peak 3 in patients with KOA were significantly greater than those in healthy individuals (Peak 1: 0.0401 ± 0.0089 BW × Ht vs. 0.0285 ± 0.0088 BW × Ht, $p < 0.001$; Peak 2: 0.0384 ± 0.0078 BW × Ht vs. 0.0122 ± 0.0055 BW × Ht, $p < 0.001$; Peak 3: 0.0366 ± 0.0084 BW × Ht vs. 0.0201 ± 0.0063 BW × Ht, $p < 0.001$). Compared to the values in healthy individuals, the Peak 1, Peak 2 and Peak 3 in patients with KOA increased by 0.41 times, 2.15 times and 0.82 times, respectively. In order to further analyze the difference of KAM between patients with KOA and healthy individuals, knee adduction moment impulse (KAMI), which was calculated by the integration of KAM over time, was utilized in our study. Since both the magnitude and duration of KAM were taken into account, KAMI could provide more comprehensive information of KAM. The comparison of KAMI between patients with KOA and healthy individuals is displayed in Fig. 4II(B). It could be seen that patients with KOA possessed greater KAMI than healthy individuals during stance phase and whole gait cycle (stance phase: 1.8301 ± 0.4017 BW × Ht × % gait cycle vs. 0.9175 ± 0.2868 BW × Ht × % gait cycle, $p < 0.001$; whole gait cycle: 1.8104 ± 0.3970 BW × Ht × % gait cycle vs. 0.8778 ± 0.2802 BW × Ht × % gait cycle, $p < 0.001$). Compared to the healthy individuals, the KAMI of stance phase and whole gait cycle in patients with KOA increased by 0.99 times and 1.06 times, respectively.

Fig. 4I(C) shows the comparison of knee internal rotation moment curves between patients with KOA and healthy individuals. The

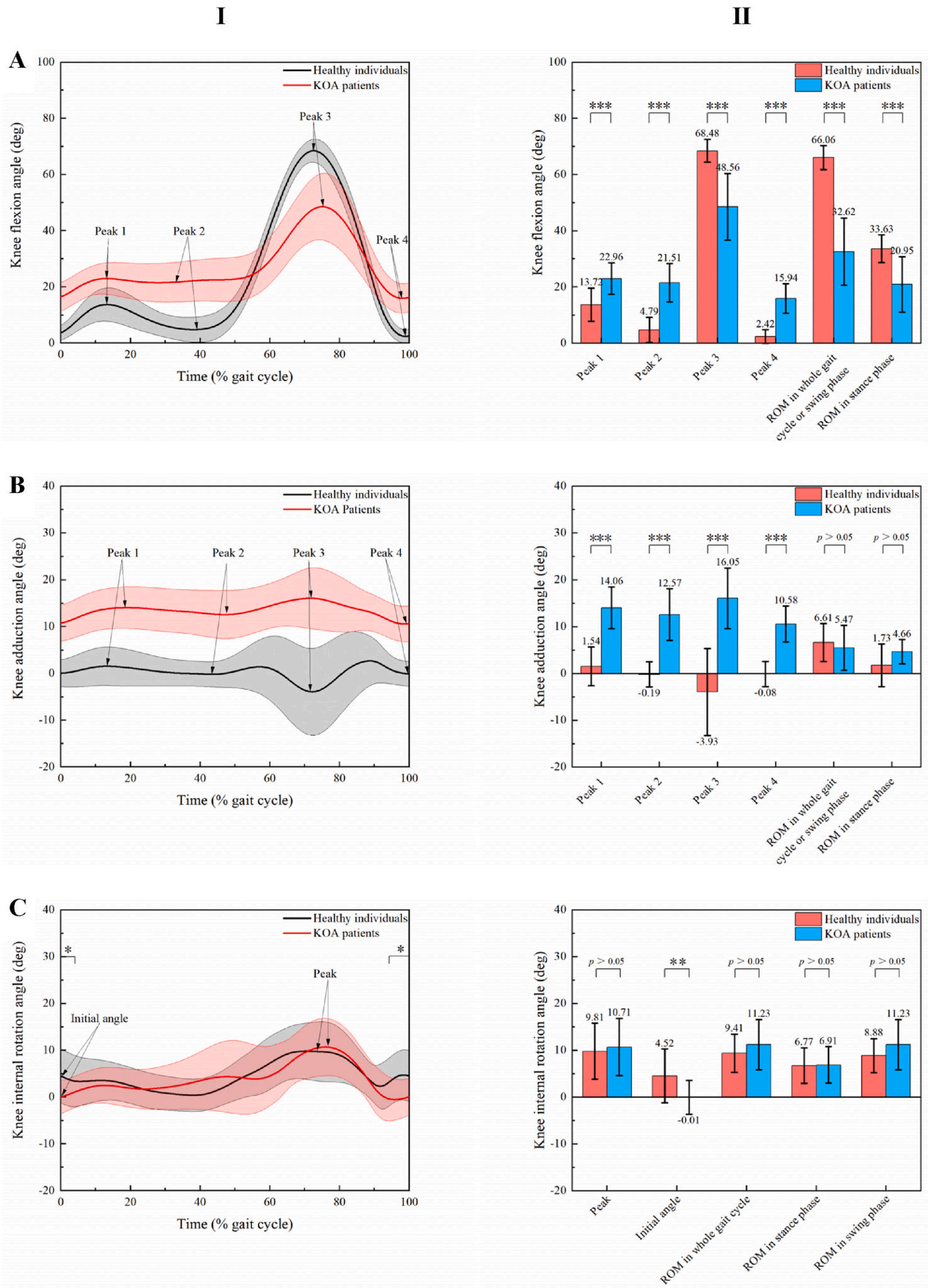

Fig. 3. Comparison of the knee angle-time curves (**I**), peak angle and range of motion (ROM) (**II**) between patients with KOA and healthy individuals in three anatomical planes, (**A**) knee flexion angle, (**B**) knee adduction angle and (**C**) knee internal rotation angle. Asterisk ** and *** represent significance level at 0.01 and 0.001 respectively.

curves appeared to be similar during the whole gait cycle, with minor peak knee internal rotation moment near heel strike (Peak 1), peak knee external rotation moment near the end of single-legs support phase (Peak 2) and zero value during the swing phase. The comparison of the peak moments and knee rotation moment impulse (KRMI) between patients with KOA and healthy individuals was

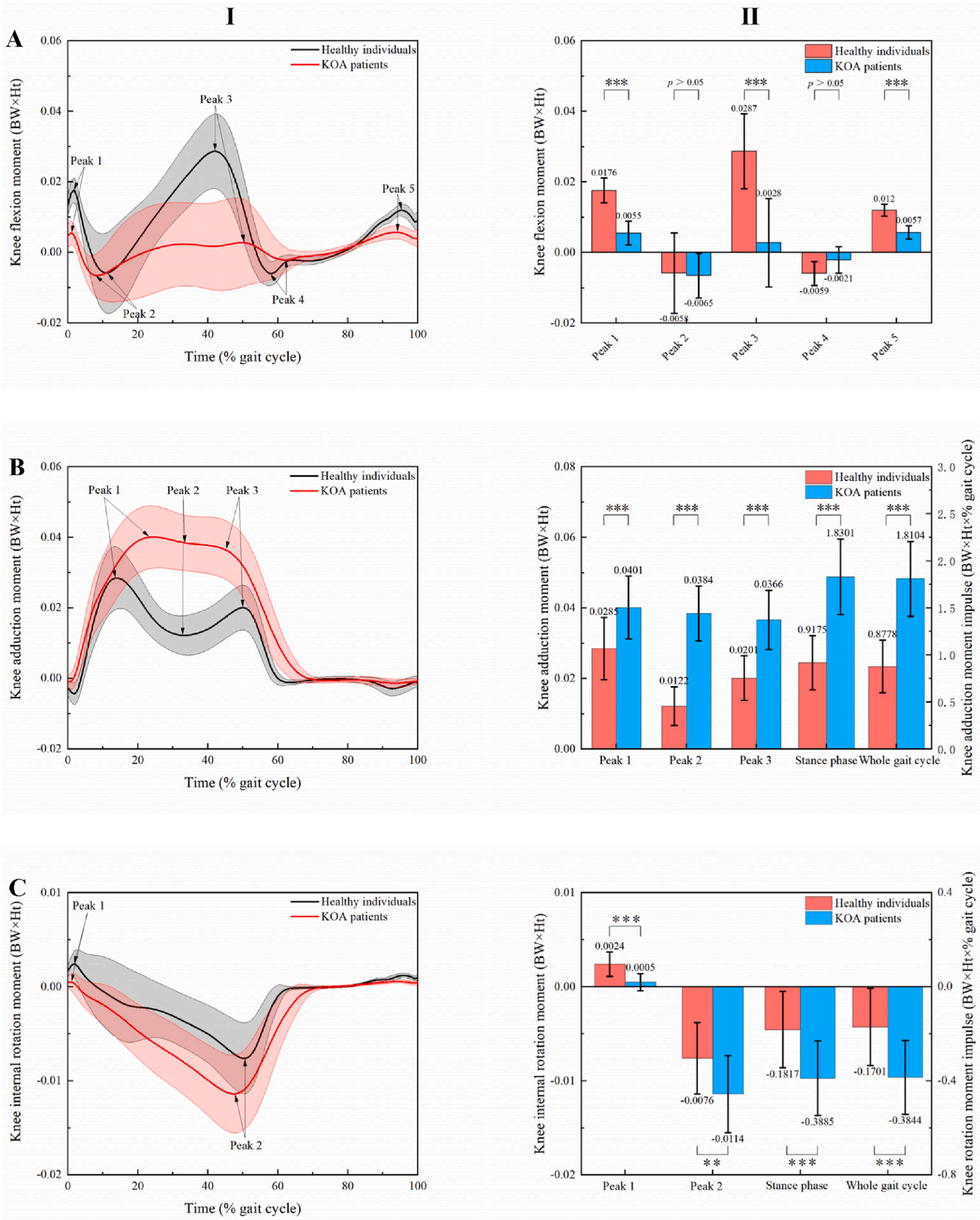

Fig. 4. Comparison of the knee moment-time curves (**I**), peak moment and moment impulse (**II**) between patients with KOA and healthy individuals in three anatomical planes, (**A**) knee flexion moment, (**B**) knee adduction moment, (**C**) knee internal rotation moment. Asterisk ** and *** represent significance level at 0.01 and 0.001 respectively.

shown in Fig. 4II(C). For the peak moment, patients with KOA had significantly smaller peak knee internal rotation moment and greater peak knee external rotation moment than healthy individuals (Peak 1: 0.0005 ± 0.0009 BW × Ht vs. 0.0024 ± 0.0013 BW × Ht, $p < 0.001$; Peak 2: −0.0114 ± 0.0041 BW × Ht vs. −0.0076 ± 0.0038 BW × Ht, $p = 0.002$). For the moment impulse, the absolute values of KRMI in patients with KOA were significantly larger than those in healthy individuals (stance phase: −0.3885 ± 0.1586 BW × Ht × % gait cycle vs. −0.1817 ± 0.1628 BW × Ht × % gait cycle, $p < 0.001$; whole gait cycle: −0.3844 ± 0.1571 BW × Ht × % gait cycle vs. −0.1701 ± 0.1634 BW × Ht × % gait cycle, $p < 0.001$). Compared to the absolute values in healthy individuals, the Peak 1 in patients with KOA decreased by 79.2 %, and the Peak 2, KRMI of stance phase and KRMI of whole gait phase in patients with KOA increased by 0.50 times, 1.14 times and 1.26 times, respectively.

3.5. Comparison of tibiofemoral contact force in three anatomical directions

Figs. 5–7 show the comparison of TFCF on the total, medial and lateral compartments between patients with KOA and healthy individuals in three anatomical directions. The TFCF in proximodistal direction, the main component of TFCF, is shown in Fig. 5. For the TFCF on the total and medial compartments in proximodistal direction, there were differences in the curve trends between patients with KOA and healthy individuals (Fig. 5I(A) and Fig. 5I(B)). During the middle of stance phase, two peaks (Peak 1 and Peak 3) and one valley (Peak 2) could be found in the curves of healthy individuals, and only one peak (Peak 3) could be found in the curves of patients

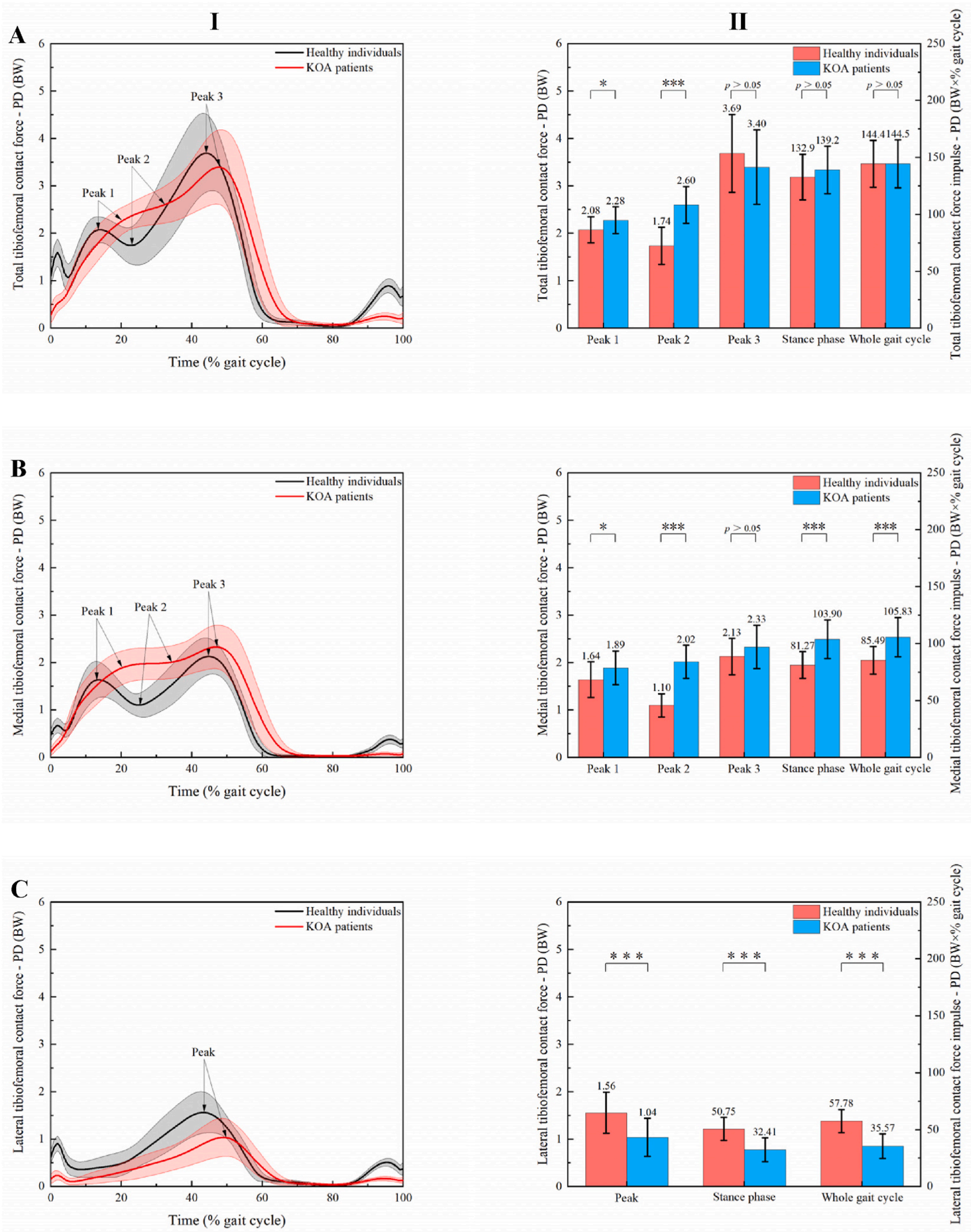

Fig. 5. Comparison of the tibiofemoral contact force-time curves (**I**), peak force and force impulse (**II**) between patients with KOA and healthy individuals in proximodistal (PD) direction, (**A**) total compartment, (**B**) medial compartment and (**C**) lateral compartment. Asterisk * and *** represent significance level at 0.05 and 0.001 respectively.

with KOA. We extracted the corresponding forces of patients with KOA at the time points similar to the Peak 1 and Peak 2 of healthy individuals, and compared the values between patients with KOA and healthy individuals, as shown in Fig. 5II(A) and Fig. 5II(B). It was obvious that patients with KOA had larger Peak 1 and Peak 2 than healthy individuals on the total (Peak 1: 2.28 ± 0.28 BW vs. 2.08 ± 0.28 BW, $p = 0.020$; Peak 2: 2.60 ± 0.39 BW vs. 1.74 ± 0.39 BW, $p < 0.001$) and medial compartments (Peak 1: 1.89 ± 0.36 BW vs. 1.64 ± 0.38 BW, $p = 0.023$; Peak 2: 2.02 ± 0.35 BW vs. 1.10 ± 0.25 BW, $p < 0.001$). But no significant difference could been seen in the Peak 3 on the total (3.40 ± 0.78 BW vs. 3.69 ± 0.82 BW, $p = 0.386$) and medial (2.33 ± 0.46 BW vs. 2.13 ± 0.38 BW, $p = 0.129$) compartments between patients with KOA and healthy individuals. Compared to the healthy individuals, the Peak 1 and Peak 2 in patients with KOA increased by 9.6 % and 49.4 % for the total compartment, and 15.2 % and 83.6 % for the medial compartment,

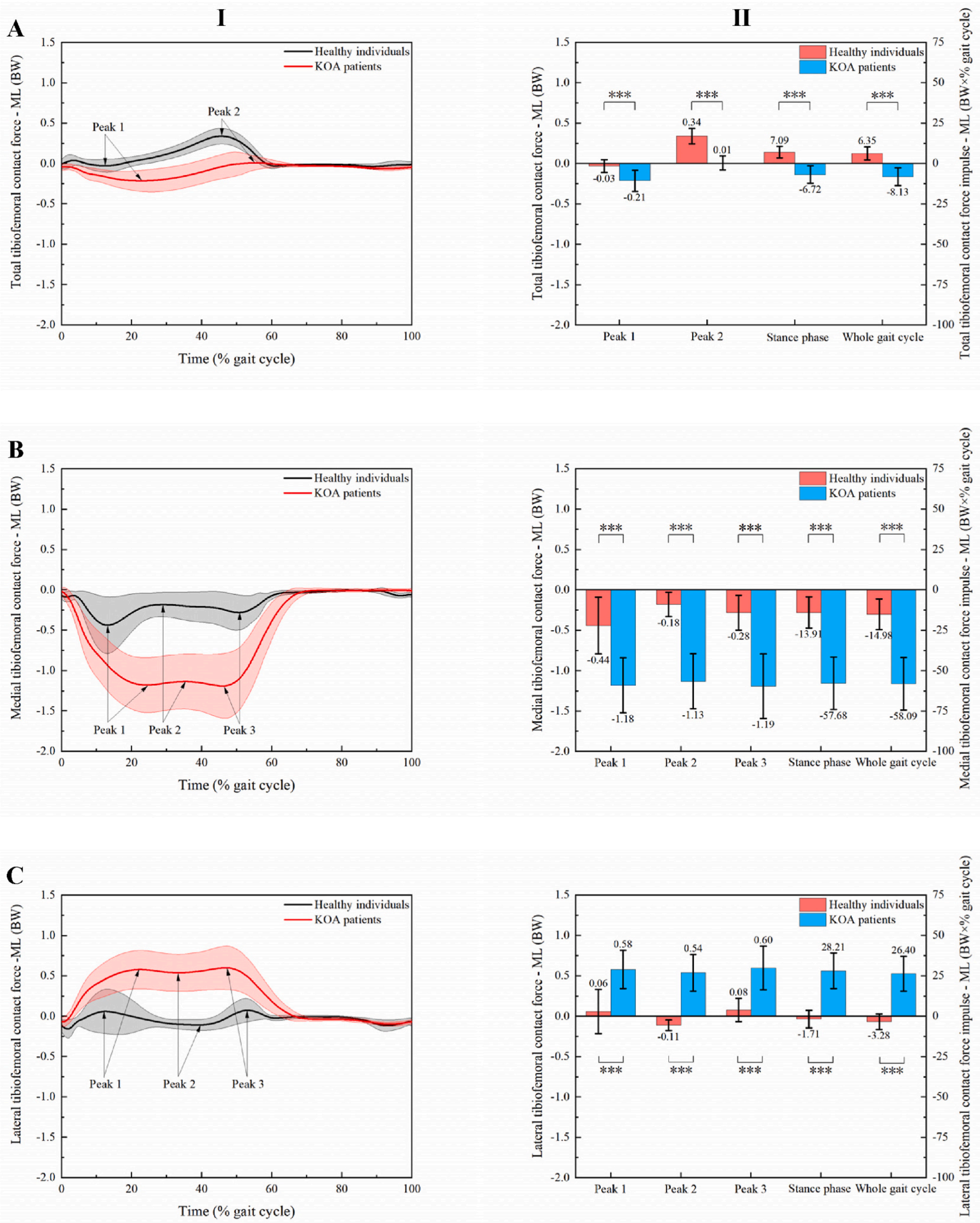

Fig. 6. Comparison of the tibiofemoral contact force-time curves (**I**), peak force and force impulse (**II**) between patients with KOA and healthy individuals in mediolateral (ML) direction, (**A**) total compartment, (**B**) medial compartment and (**C**) lateral compartment. Asterisk *** represent significance level at 0.001.

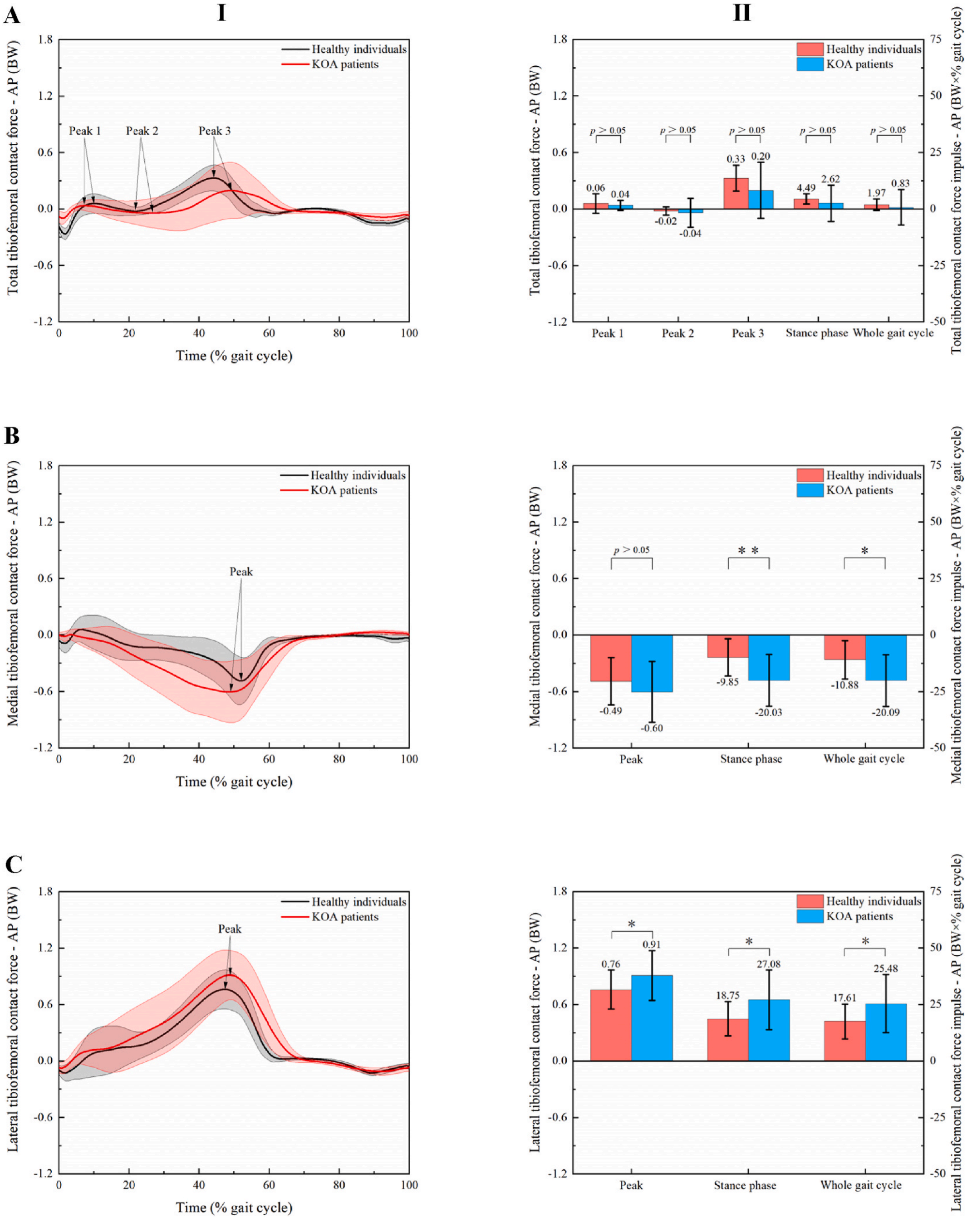

Fig. 7. Comparison of the tibiofemoral contact force-time curves (**I**), peak force and force impulse (**II**) between patients with KOA and healthy individuals in anteroposterior (AP) direction, (**A**) total compartment, (**B**) medial compartment and (**C**) lateral compartment. Asterisk * and ** represent significance level at 0.05 and 0.01 respectively.

respectively. To comprehensively analyze the difference of TFCF between patients with KOA and healthy individuals, the TFCF impulse (TFCFI) of stance phase and whole gait cycle were calculated in our study. It was evident that there was no significant difference in the TFCFI of the total compartment during the stance phase (139.21 ± 20.74 BW × % gait cycle vs. 132.94 ± 20.05 BW × % gait cycle, $p = 0.279$) and whole gait cycle (144.53 ± 21.03 BW × % gait cycle vs. 144.40 ± 20.59 BW × % gait cycle, $p = 0.822$) between patients with KOA and healthy individuals. However, the TFCFI of the medial compartment was significantly greater in patients with KOA than those in healthy individuals during the stance phase (103.90 ± 16.99 BW × % gait cycle vs. 81.27 ± 11.73 BW × % gait cycle, $p < 0.001$) and whole gait cycle (105.83 ± 17.11 BW × % gait cycle vs. 85.49 ± 12.08 BW × % gait cycle, $p < 0.001$). Compared to the healthy individuals, the TFCFI of stance phase and whole gait cycle in patients with KOA increased by 27.8 % and 23.8 %, respectively. For the TFCF on the lateral compartment, similar single-peak curves were observed in both patients with KOA and healthy individuals during the middle of stance phase (Fig. 5I(C)). The peak TFCF in patients with KOA was significantly smaller than that in healthy individuals (1.04 ± 0.40 BW vs. 1.56 ± 0.44 BW, $p < 0.001$). For the TFCFI of the lateral compartment, patients with KOA had significantly smaller values than healthy individuals during both stance phase (32.41 ± 10.51 BW × % gait cycle vs. 50.75 ± 10.17 BW × % gait cycle, $p < 0.001$) and whole gait cycle (35.57 ± 10.77 BW × % gait cycle vs. 57.78 ± 10.17 BW × % gait cycle, $p < 0.001$). Compared to the healthy individuals, the peak TFCF, TFCFI of stance phase and TFCFI of whole gait cycle in patients with KOA reduced by 33.3 %, 36.1 % and 38.4 %, respectively.

Fig. 6 shows the comparison of TFCF between patients with KOA and healthy individuals in mediolateral direction. The TFCF on the total, medial and lateral compartments appeared to be similar curve pattern between patients with KOA and healthy individuals, with two peaks (Peak 1 and Peak 2) during stance phase for the total compartment, two peaks (Peak 1 and Peak 3) and one valley (Peak 2) during stance phase for the medial and lateral compartments, and near-zero TFCF during swing phase for all of the total, medial and lateral compartments. For the total compartment, greater absolute value of Peak 1 (−0.21 ± 0.13 BW vs. −0.03 ± 0.08 BW, $p < 0.001$) and smaller Peak 2 (0.01 ± 0.09 BW vs. 0.34 ± 0.10 BW, $p < 0.001$) could been seen in patients with KOA than healthy individuals, as shown in Fig. 6(A). The TFCFI of stance phase and whole gait cycle in patients with KOA was negative compared to the positive values in healthy individuals (stance phase: −6.72 ± 5.45 BW × % gait cycle vs. 7.09 ± 3.60 BW × % gait cycle, $p < 0.001$; whole gait cycle: −8.13 ± 5.55 BW × % gait cycle vs. 6.35 ± 4.12 BW × % gait cycle, $p < 0.001$). As shown in Fig. 6(B), all of the peak TFCF and TFCFI were negative on the medial compartment. Compared to the healthy individuals, the absolute values of Peak 1, Peak 2, Peak 3, TFCFI of stance phase and TFCFI of whole gait cycle in patients with KOA increased by 1.68 times (−1.18 ± 0.34 BW vs. −0.44 ± 0.35 BW, $p < 0.001$), 5.28 times (−1.13 ± 0.34 BW vs. −0.18 ± 0.15 BW, $p < 0.001$), 3.25 times (−1.19 ± 0.40 BW vs. −0.28 ± 0.22 BW, $p < 0.001$), 3.15 times (−57.68 ± 16.19 BW × % gait cycle vs. −13.91 ± 9.71 BW × % gait cycle, $p < 0.001$) and 2.88 times (−58.09 ± 16.19 BW × % gait cycle vs. −14.98 ± 9.42 BW × % gait cycle, $p < 0.001$), respectively. The TFCF of the lateral compartment was shown in Fig. 6(C). Greater Peak 1 (0.58 ± 0.24 BW vs. 0.06 ± 0.27 BW, $p < 0.001$), Peak 2 (0.54 ± 0.23 BW vs. −0.11 ± 0.07 BW, $p < 0.001$), Peak 3 (0.60 ± 0.27 BW vs. 0.08 ± 0.14 BW, $p < 0.001$), TFCFI of stance phase (28.21 ± 10.91 BW × % gait cycle vs. −1.71 ± 5.49 BW × % gait cycle, $p < 0.001$) and TFCFI of whole gait cycle (26.40 ± 10.81 BW × % gait cycle vs. −3.28 ± 4.74 BW × % gait cycle, $p < 0.001$) could be found in patients with KOA than healthy individuals.

The comparison of TFCF between patients with KOA and healthy individuals in anteroposterior direction was shown in Fig. 7. Two peaks (Peak 1 and Peak 3) and one valley (Peak 2) could be found in the TFCF curves of the total compartment and one peak could be found in the TFCF curves of the medial and lateral compartments for both patients with KOA and healthy individuals. As shown in Fig. 7II(A), there were no significant differences in the Peak 1 (0.04 ± 0.05 BW vs. 0.06 ± 0.10 BW, $p = 0.386$), Peak 2 (−0.04 ± 0.15 BW vs. −0.02 ± 0.04 BW, $p = 0.324$), Peak 3 (0.20 ± 0.30 BW vs. 0.33 ± 0.14 BW, $p = 0.095$), TFCFI of stance phase (2.62 ± 8.07 BW × % gait cycle vs. 4.49 ± 2.33 BW × % gait cycle, $p = 0.290$) and TFCFI of whole gait cycle (0.83 ± 7.90 BW × % gait cycle vs. 1.97 ± 2.56 BW × % gait cycle, $p = 0.530$) of the total compartment between patients with KOA and healthy individuals. For the medial compartment, no significant difference was found in the peak TFCF (−0.60 ± 0.32 BW vs. −0.49 ± 0.25 BW, $p = 0.361$) between patients with KOA and healthy individuals (Fig. 7II(B)). However, the absolute values of TFCFI in patients with KOA increased by 1.03 times for stance phase (−20.03 ± 11.43 BW × % gait cycle vs. −9.85 ± 8.25 BW × % gait cycle, $p = 0.004$) and 0.85 times for whole gait cycle (−20.09 ± 11.45 BW × % gait cycle vs. −10.88 ± 8.52 BW × % gait cycle, $p = 0.010$) compared to healthy individuals, respectively. Fig. 7(C) shows the comparison of TFCF between patients with KOA and healthy individuals on the lateral compartment. Compared to the healthy individuals, the peak TFCF (0.91 ± 0.26 BW vs. 0.76 ± 0.21 BW, $p = 0.041$), TFCFI of stance phase (27.08 ± 13.19 BW × % gait cycle vs. 18.75 ± 7.58 BW × % gait cycle, $p = 0.020$) and TFCFI of whole gait cycle (25.48 ± 12.85 BW × % gait cycle vs. 17.61 ± 7.72 BW × % gait cycle, $p = 0.020$) in patients with KOA increased by 19.7 %, 44.4 % and 44.7 %, respectively.

4. Discussion

Analyses of the knee biomechanical differences during gait in patients with KOA versus healthy individuals are critical to understanding the initiation of KOA, detecting the progression of KOA, and providing guidance for the treatments of KOA. For this purpose, an optical motion capture system and an improved musculoskeletal model were utilized in this study. To the best of our knowledge, this is the first systematical and comprehensive study investigating the differences in spatiotemporal parameters, knee joint angle and moment in three anatomical planes, and TFCF on the total, medial and lateral compartments in three anatomical directions during gait between patients with KOA and healthy individuals.

The results of spatiotemporal parameters comparison were concordant with our hypothesis. Our finding showed that patients with KOA had significantly reduced self-selected walking speed, stride length, stride height and the time proportions of swing phase and single-leg support phase, and significantly increased time of gait cycle and time proportions of stance phase and double-legs support phase, compared to healthy individuals. Similar results in self-selected walking speed [5–11], stride length [6–10], time of gait cycle [6,9,10], time proportion of stance phase [10], double-leg support phase [6,8] were found in previous studies. The differences in stride height between patient with KOA and healthy people have rarely been studied. Possibly because all patient with mild and severe KOA included, no significant difference in the time proportions of single-leg support period and time of gait cycle between patient with KOA and healthy people were found in the study by Dai et al. [8]. As we all know, knee pain during walking and lack of gait stability are the main characteristics of KOA [23,24]. This study speculates that patients with KOA may reduce joint pain and increase gait stability by changing their gait spatiotemporal parameters. The body's compensatory adaptation during gait may be manifested by increasing the time of gait cycle and time proportions of stance phase and double-legs support phase, and decreasing the walking speed, stride length, stride length, and the time proportions of swing phase and single-leg support phase.

Consistent with our hypothesis, there were significant differences in peak knee flexion angle, peak knee adduction angle and ROM of knee flexion joint between the two groups. However, no significant differences were found in peak knee rotation angle and ROM of knee adduction and rotation angle between the two groups. Dai et al. [8] found that patients with KOA walked with smaller peak knee flexion angle, peak knee extension angle and ROM of knee flexion angle. McCarthy et al. [25] reported that patients with KOA had significant lower ROM of knee flexion angle during stance and swing than healthy subjects. Ismailidis et al. [9] observed that lower peak knee flexion angle during stance and swing as well as lower ROM during load acceptance, terminal stance and swing were existed in patients with KOA compared to healthy controls. Barai et al. [10] reported that there were significant decrease in peak knee flexion angle and ROM of knee flexion angle between the two groups. In brief, the results in our study are in good agreement with the recent literature on the differences of knee kinematics in sagittal plane. Few studies have investigated the differences of knee kinematics in coronal and transverse planes. Similar to our results, greater knee adduction angle and knee internal rotation angle at heel strike were found in the study by Barai et al. [10]. But unlike our results, a smaller ROM of knee adduction angle in patients with KOA was observed in their study. This inconsistency in results may be due to the differences in the severity of recruited patients with KOA. To our knowledge, the differences in peak knee adduction angle, peak knee internal rotation angle and ROM of knee internal rotation angle between patient with KOA and healthy people have rarely been reported. Overall, we speculate that the significant change in knee kinematics is sought to be a compensation strategy of patient with KOA in response to pain and stiffness of knee.

KAM, as a surrogate measure for knee load distribution, have been linked to the onset, progression and severity of KOA [22,26]. In agreement with the results of our study, greater peak KAM (Peak 1) and minimum KAM in mid-stance (Peak 2) were found in the study by Barai et al. [10], greater peak KAM in the first half of stance (Peak 1) and peak KAM in the second half of stance (Peak 3) were observed in the study by Kumar et al. [11], and greater peak KAMs (Peak 1, Peak 2 and Peak 3) were reported in the study by Baliunas et al. [23], respectively. However, higher mid-stance KAM (Peak 2) and smaller peak KAM (Peak 1 and Peak 3), which is inconsistent with our results, was observed in the study by Astephen et al. [27]. It is well-known that the peak KAM is a speed-dependent measure and may be reduced at slower speeds [28]. we speculate that the reason for the inconsistency may be that patients with KOA had lower self-selected walking speed than healthy individuals. Since both the magnitude and duration of KAM were taken into account, KAMI was compared in our study. Our finding showed that patients with KOA had greater KAMI during stance phase and whole gait cycle. The results in our study are in good agreement with the study by Thorp et al. [29], where the KAMI increased with the Kellgren-Lawrence (KL) grade. Knee flexion-extension moment was associated with the development of knee pain and also commonly used as a surrogate measure for knee load distribution [22]. Our finding showed that the peak knee flexion moments (Peak 1, Peak 3 and Peak 5) were significantly smaller in patients with KOA, while the peak knee extension moments (Peak 2 and Peak 4) were not significantly different. The comparison results of knee joint moment in sagittal plane between patients with KOA and healthy individuals were inconsistent in previous studies. Barai et al. [10] reported that the peak knee flexion moment and peak knee extension moment decreased significantly in patients with KOA than those in healthy individuals. Astephen et al. [27] found that patients with KOA had smaller peak knee flexion moment and smaller early stance knee extension moment. Kumar et al. [11] observed that the peak knee flexion moments in the first half of stance and the second half of stance between patients with KOA and healthy controls were not significantly different. Baliunas et al. [23] found that none of the four KAM (initial knee moment, early midstance knee moment, terminal stance knee moment and preswing knee moment) was significantly different between patient with KOA and healthy subjects. We speculate that the reduced knee flexion moment may be a compensatory mechanism of limiting knee flexion. In addition, we suspect that these inconsistent results may be due to population differences in race, strength, body weight, walking speed, as well as other variations in the selection criteria between the studies. Knee internal rotation moment has rarely been studied in previous studies. In our study, patients with KOA had significantly smaller peak knee internal rotation moment and greater peak knee external rotation moment than healthy individuals. Unlike our study, Barai et al. [10] observed there was no significant difference in the peak knee internal rotation moment between the two groups, and Astephen et al. [27] reported patients with KOA had a significant decreased peak knee internal rotation moment than healthy subjects. In order to further analyze the differences of knee moment in transverse plane, our study is the first to compared the knee rotation moment impulse during stance phase and whole gait cycle between the two groups. Our results showed that the knee external rotation moment impulse in patients with KOA were significantly larger than those in healthy individuals.

Increased abnormal knee load distribution is associated with progression of KOA [22]. Because the contributions of the surrounding muscles and soft tissues are not included, knee joint moment represents the net moment around the knee joint and is not a strong predictor for the knee load [21]. To further understand the mechanisms related to initiation and progression of KOA, the TFCF on the total, medial and lateral compartments in three anatomical directions during gait were compared in this study. In the proximodistal direction, our finding showed that patients with KOA had significant greater first peak TFCF (Peak 1) and minimum TFCF (Peak 2) during the middle stance phase on the total and medial compartments, and smaller values on the lateral compartment. But for the second peak TFCF (Peak 3), there were no significant difference between patients with KOA and healthy individuals on the total and medial compartments, and a smaller value for patients with KOA on the lateral compartment. The comparison results of TFCF in proximodistal direction between patients with KOA and healthy individuals were inconsistent in previous studies. Yamagata et al. [30] found that patient with severe KOA had a greater medial TFCF and medial shift of TFCF. Moura et al. [31] reported that the first peak and second peak TFCF on the total compartment was greater in patients with KOA than those in healthy subjects. Possibly because all patient with mild and severe KOA included, Kumar et al. [11] reported that the loading was no significant different between the two groups but OA subjects had greater medial load and smaller lateral load. To further comprehensively analyze the difference of TFCF between patients with KOA and healthy individuals, the TFCF impulse of stance phase and whole gait cycle were calculated in our study for the first time. Our finding showed that patients with KOA had greater TFCF impulse on the medial compartment and lower TFCF impulse on the lateral compartment compared to healthy individuals. But there was no significant difference in the TFCF impulse of the total compartment between the two groups. To our knowledge, no previous studies have compared the TFCF between patients with KOA and healthy individuals in mediolateral and anteroposterior directions. In our study, there were significant differences in the peak TFCF and TFCF impulse of the total, medial and lateral compartments in the mediolateral direction, and the medial and lateral compartments in the anteroposterior direction. But the peak TFCF and TFCF impulse of the total compartment was no significant different between the two groups in the anteroposterior direction. Overall, our study suggests that comparing TFCF in three anatomical directions would provide more comprehensive information for analyzing the changes of knee biomechanical properties caused by KOA.

A number of limitations associated with this study are worth discussing. Firstly, only medial KOA patients with Kellgren-Lawrence grades 3–4 KOA were recruited in this study. In the future, the patients with lateral compartment KOA and Kellgren-Lawrence grades 1–2 KOA will be recruited, and the relationship between knee biomechanical characteristics and the severity of KOA will be analyzed. Secondly, the knee functional assessment, such as KSS and WOMAC scores, was not included in this study. Knee pain, function, stiffness, etc., can be systematically assessed in this way. Thirdly, the subject-specific geometries of bones, cartilages and meniscus, detailed ligament models, and natural deformable contact conditions were not considered in the musculoskeletal model of this study. Increasing the personalization of the musculoskeletal model will be done in future. Finally, only gait experiment was carried out in this study. Because the different motions possessed different kinematics and kinetics, the results of this study were only suitable for gait and not for other motions. Quantitative comparing the knee biomechancial characteristics between patients with KOA and healthy individuals during other motions, such as sit-stand-sit and stair climbing, will be studied in the future.

The results of this study have important implication for clinicians and rehabilitation physicians. Getting the knee biomechanical properties back is the ideal target of various treatments of KOA. Firstly, These quantified biomechanical differences between patients with KOA and healthy controls can provide data support for the personalized and quantified rehabilitation strategies, such as the smart knee orthosis, which is our target research product. Based on the differences of knee adduction moment, tibiofemoral contact force, knee flexion moment, etc., between the two groups, the smart knee orthosis can provide quantified orthopedic moment for the patient's knee joint, and then slow the progression of KOA. Secondly, the results of our study have some suggestions for the exercises of KOA patients. Compared to healthy individuals, smaller peak knee flexion angle and knee flexion ROM were observed in the patient with KOA. Reasonable knee stretching exercise can help strengthen the flexibility of knee joint, and then improve the knee flexion ROM. Besides, greater peak TFCF and TFCF impulse on the medial compartment, and smaller peak TFCF and TFCF impulse on the lateral compartment were found in the patients with KOA. Targeted strength training for the muscles around the knee joint can help to reduce the load on the knee medial compartment and improve the stability of knee joint. Finally, the quantified differences between patients with KOA and healthy controls can help monitor disease, evaluate surgical treatment, and develop more effective preoperative planning and postoperative rehabilitation strategies, which can improve the effectiveness of knee functional recovery and then improve their overall quality of life.

5. Conclusion

In this study, the knee biomechanical differences during gait between patients with KOA and healthy controls were investigated. Based on the experimental and simulation results, the main conclusions can be summarized as follows.

1. Patients with KOA walked with reduced self-selected walking speed, stride length, stride height, time proportion swing phase and time proportion of single-leg support phase, and increased gait cycle time, time proportion of stance phase and time proportion of double-legs support phase, compared to healthy individuals.
2. The peak knee flexion angle and knee flexion ROM of the patients with KOA were smaller than those of the healthy controls, while the peak knee adduction angle was greater in the patients with KOA. Besides, no significant differences were found in the peak knee rotation angle, knee adduction ROM and knee rotation ROM between the two groups.
3. Patients with KOA had significant lower peak knee flexion moment, while greater peak knee adduction moment (KAM), peak knee rotation moments (KRM), KAM impulse and KRM impulse.
4. In proximodistal direction, greater first-peak TFCF on the total and medial compartments and TFCF impulse on the medial compartment, smaller peak TFCF and TFCF impulse on the lateral compartment were found in the patients with KOA. However, there were no significant differences in the second-peak TFCF on the total and medial compartments and TFCF impulse on the total compartment. In mediolateral direction, greater absolute values of the peaks and TFCF impulses on the total, medial and lateral compartments were found in the patients with KOA. In anteroposterior direction, the patients with KOA had significant greater peak TFCF on the lateral compartment and greater absolute values of the TFCF impulses on the medial and lateral compartments. But no significant differences were found in the peak TFCF and TFCF impulse on the total compartment and peak TFCF on the medial compartment.

In conclusion, significant differences were found in the gait spatiotemporal parameters, knee kinematics and kinetics, and tibiofemoral contact force during gait between patients with KOA and healthy individuals. Quantitative differences between the two groups will be significant for comprehensive understanding the gait biomechanics of KOA and providing guidance for the treatment of KOA.

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Ethics statement

The studies involving human participants were approved by the Ethics Committee, Honghui Hospital, Xi'an Jiaotong University (No: 202309006 and data of approval: October 9, 2023). This study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. All participants provided their Written Informed Consent to participate in this study and to publish their data.

Data availability statement

All data used in this study are available from the corresponding author on a reasonable request.

CRediT authorship contribution statement

Li Zhang: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Rui Ma:** Writing – original draft, Validation, Software, Resources, Methodology, Formal analysis, Data curation. **Hui Li:** Writing – review & editing, Supervision, Resources, Methodology, Conceptualization. **Xianjie Wan:** Writing – review & editing, Resources, Methodology, Data curation. **Peng Xu:** Writing – review & editing, Supervision, Funding acquisition, Resources, Project administration, Conceptualization. **Aibin Zhu:** Writing – review & editing, Supervision, Project administration, Conceptualization. **Pingping Wei:** Writing – review & editing, Software, Methodology.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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