Alteration of Screw-home Movement in Patients With Knee Osteoarthritis: A Cross-sectional Study

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Abstract

Background:

Tibial rotation accompanying sagittal movement contains the phenomenon of screw-home movement of the knee, which plays an important role in knee stability during extension. This study aimed to investigate the alteration of screw-home movement in patients with knee osteoarthritis (OA).

Methods:

Participants (n =67) in this cross-sectional study were outpatients in the department of orthopedics of a general hospital and included patients with knee OA (n=31) and asymptomatic control subjects (n=36).Knee kinematic data were measured using an inertial measurement unit. The total tibial rotation was obtainedduring knee sagittal movement. The acquired angle of tibial rotation was divided into four periodseach extension and flexion. The total tibial rotation and the variation of each period were compared between the OA and control groups.

Results:

Significant differences arose between the OA and control groups in the total tibial rotation during knee extension and flexion(P< 0.001).The variation of tibial rotation was also significantly different between groups for all periods (P< 0.001; knee extensionat 70° to 45°,P = 0.014).

Conclusions:

We found a reduction in the total tibial rotation and loss of the screw-home movement in the unloaded OA knee. To normalize the screw-home movement, it is necessary to promote proper articular movement of the knee joint and suppress the hyperexcitability of the medial muscles.

Background

Although knee osteoarthritis (OA) is diagnosed by radiographic evidence of degenerative changes, it is difficult for a single definition to encompass all instances of OA [1]. Problematically, radiographic diagnosis of knee OA provides a weak association with symptoms and function [2]. A survey study found that 1,004 (14.6%) of 6,880 subjects experienced knee pain [3]. Of these patients with knee pain, 59% were diagnosed with knee OA by a physician, but only about 15% had radiographic stage (2–4) changes of knee OA. Therefore, a clinician diagnoses knee OA on the basis of clinical and/or radiological features [4] including the diagnostic criteria of pain, stiffness, age, bony tenderness, crepitus, and bony enlargement [5].

Structural changes in knee OA are thought to be caused by multiple pathways involving various risk factors [1]. The continuous formation and decomposition of the cartilaginous matrix are disrupted by these harmful influences, resulting in a reduction of total cartilage at the knee joint [6]. Direct measurement of articular cartilage has several potential advantages over indirect measurement via radiographs [7]. Several previous studies have used magnetic resonance imaging to investigate the relation between cartilage volume and knee OA [7–9]. A linear reduction in cartilage volume was associated with a narrowing of the joint space [7]. Articular cartilage loss (but not radiographic score) was a risk factor for undergoing a knee replacement, an intervention at end-stage knee OA [9]. However, cartilage volume is limited in detecting early knee OA due to swelling at the early stages of injury [10]. Causes of degenerative changes in articular cartilage are affected by the interrelation of mechanical, structural, and biological pathways [11].

The progression of knee OA has been associated with biomechanical disruption, which shifts the normal bearing region of the knee joint [12]. Thus, degenerative changes in cartilage can be affected by kinematic changes [11]. Several previous studies have attempted to predict or identify knee OA through an investigation of biomechanical changes [13–18]. Most biomechanical studies investigate the gait of patients with knee OA because gait is the most functional and common use of the lower extremities [14–18]. The main consequences for gait analysis, however, were increased knee adduction moment and internally rotated tibia [14, 16, 17, 19].

Tibial rotation accompanying sagittal movement contains the phenomenon of screw-home movement (SHM) of the knee, which plays an important role in knee stability during extension [20–22]. However, SHM is affected by weight-bearing or loaded conditions [23]. For example, external rotation occurred during the loading response phase, which was a reversal of the SHM [24]. To the best of our knowledge, only one previous study has evaluated the tibial rotation for the non-weighted bearing state in patients with knee OA [13]. However, their study reported tibial rotation only during the five-degree flexion position in a non-weight-bearing state, which was insufficient to demonstrate the dynamic state of SHM. It is difficult to determine how biomechanical changes are involved in soft tissue degenerative changes or compensation mechanisms [14].

To understand the mechanical factors of knee stability in patients with knee OA, herein we investigate the SHM changes in a group of these patients during dynamic movement.

Methods

Subjects

A total of 36 patients with symptomatic, medial-compartment knee OA were included in this cross-sectional study. Each subject had visited a local hospital between April 2019 and July 2019 in the Republic of Korea for complaints of knee pain. The diagnosis of knee OA was confirmed based on clinical and radiological features. Subjects were excluded from this study if they had undergone any major surgery or trauma to the lower limbs, limitation of motion in the knee joint, neuromuscular disorder, inflammatory arthritis, osteopetrosis, periarticular fracture, gout, history of stroke, or cardiovascular disease. Five OA patients were excluded because of limited movement in the knee and the previous stroke history. After assessing eligibility, 31 patients with OA were enrolled. Radiological severity in patients with knee OA was evaluated using Kellgren–Lawrence (KL) grades [25]. The visual analog scale (VAS) and Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) scores were used for clinical evaluation [2]. A group of 36 asymptomatic control subjects without a diagnosis of OA, rheumatoid arthritis, or a history of trauma or pain were recruited from the general population. This study was approved by the Institutional Review Board of the CM general hospital of the Republic of Korea (CMHCTC-19-001).

Data acquisition and analysis

Knee kinematic data were measured using an inertial measurement unit (IMU) (EBIMU24GV3, E2BOX, Seoul, Republic of Korea), consisting of a three-axis accelerometer (± 8 × *g*), magnetometer (± 16 Gs), and gyroscope (± 2,000°/s), at a frequency of 40 Hz. IMU data were collected from two sensors attached to the thigh (lateral side) and tibia shaft (proximal medial surface). The angles of knee flexion-extension and tibial rotation were calculated as the relative angles between the thigh and shank segments. Calculation of the joint angle was performed with the Kalman filter fusion algorithm using SMULeg software (SMULeg, XENART, Daejeon, Republic of Korea). Subjects performed knee flexion-extension movement to determine the physically meaningful axis of the joint in the local coordinate system of the sensor [26, 27]. The examiner manually moved the subject’s knee joint to full extension, where the joint angle was considered to be 0°. Positive values represent knee flexion and tibia external rotation in this study. A previous study demonstrated that the IMU and software used in this study were reliable motion analysis devices; compared with the motion capture system, the coefficients of multiple correlations were 0.959 and 0.803 for the sagittal and transverse planes, respectively [28].

The starting position was defined as sitting on a chair with the knee at 70° flexion without weight. The kinematic data was measured while each subject extended the knee from a starting position to a full extension for 2 s and repositioned back to the original position for 2 s (70° flexion, full extension, 70° flexion), assisted by a metronome. The total tibial rotation during knee extension was calculated by subtracting the rotation angle at the full extension from rotation angle at 70° flexion. Total tibial rotation during flexion was calculated by subtracting the rotation angle at 70° flexion from the rotation angle at full extension. The acquired tibial rotation angle during extension and flexion of the knee was divided into four periods each, according to the change in flexion-extension angle. These four periods were defined separately during every 15°-range of knee flexion-extension movement, except for the range between knee flexion at 70° and 45° and were as follows: (1) during knee extension, 70°–45°, 45°–30°, 30°–15°, and 15°–0° and (2) during knee flexion, 0°–15°, 15°–30°, 30°–45°, and 45°–70°.

Statistical analysis

An independent t-test was used to compare the difference of total tibial rotation during the knee sagittal movement (extension and flexion) and the variation in each period between the OA and control groups. All statistical analyses were performed using IBM SPSS version 22.0 software (IBM Corporation, Armonk, NY, USA). Statistical significance was set to $P < 0.05$.

Results

Detailed demographics and clinical characteristics of the subjects are summarized in Table 1. Patients with knee OA were comprised of 14 males and 17 females: 14 with right OA, and 17 with left OA. In these 31 knees, reader one graded 23 knees (74.2%) with KL grade 2, 5 knees (16.1%) with grade 3, and 3 knees (9.7%) with grade 4. The mean age of the OA group was 62.09 ± 9.67 (range 37–80). Their mean height, weight, VAS, and WOMAC were 162.09 ± 8.36 cm (range 148–178), 64.45 ± 11.84 kg (range 48–92), 5.29 ± 2.15 score (range 2–10), and 21.74 ± 10.91 (range 6–42), respectively.

Table 1
Demographic and clinical characteristics of the subjects.

Demographics and Clinical Characteristics	OA	Control
Gender (male/female)	14 / 17	21 / 15
Affected side (right/left)	14 / 17	–
Age (years)[a]	62.09 ± 9.67 (37–80)	23.69 ± 1.98 (21–28)
Height (cm)[a]	162.09 ± 8.36 (148–178)	167.58 ± 8.15 (153–184)
Weight (kg)[a]	64.45 ± 11.84 (48–92)	64.39 ± 11.66 (40–84)
Kellgren–Lawrence grade	II: 23, III: 5, IV: 3	–
VAS (0–10)[a]	5.29 ± 2.15 (2–10)	–
WOMAC (0–96)[a]	21.74 ± 10.91 (6–42)	–
Pain (0–20)[a]	4.45 ± 2.68 (1–12)	–
Stiffness (0–8)[a]	1.42 ± 1.26 (0–4)	–
Function (0–68)[a]	15.87 ± 8.64 (2–34)	–

[a] Mean ± standard deviation (range)

OA, osteoarthritis; VAS, visual analog scale; WOMAC, Western Ontario and McMaster Universities score.

The total tibial rotations during knee extension and flexion were − 0.39 ± 4.28 and − 1.61 ± 3.06, respectively, in the OA group (Table 2). For the control group, the total tibial rotation during knee extension and flexion were 12.28 ± 6.54 and − 12.06 ± 6.51, respectively (Table 2). Significant differences were observed between the OA and control groups in the total tibial rotation during knee extension and flexion ($P < 0.001$) (Table 2). Tables 3 provides the variation of the tibial rotation in each period during the knee sagittal movement of two groups. Significant differences arose between the two groups in the variation of tibial rotation for all periods ($P < 0.001$; knee flexion from 70° to 45° was $P = 0.014$) (Table 3 and Fig. 1).

Table 2
Comparison of total tibial rotation during knee extension and flexion between OA and control groups.

Knee movement	Total tibial rotation (°)		*P* value
	OA	Control	
Extension (knee flexion from 70° to 0°)	− 0.39 ± 4.28	12.28 ± 6.54	< 0.001
Flexion (knee flexion from 0° to 70°)	2.57 ± 4.61	− 12.06 ± 6.51	< 0.001

Mean ± standard deviation.

OA, osteoarthritis.

Positive values represent the external rotation of the tibia.

Table 3
Comparison of the variation of tibial rotation for each period between OA and control groups.

Knee movement	Period	Variation of tibial rotation (°)		*P* value
		OA	Control	
Extension	70°–45°	− 1.61 ± 3.06	0.29 ± 3.13	0.014
	45°–30°	0.29 ± 1.77	2.32 ± 1.98	< 0.001
	30°–15°	0.67 ± 1.67	4.05 ± 1.95	< 0.001
	15°–0°	0.25 ± 2.52	5.62 ± 3.22	< 0.001
Flexion	0°–15°	0.61 ± 1.99	− 3.52 ± 3.25	< 0.001
	15°–30°	0.61 ± 1.57	− 3.78 ± 2.64	< 0.001
	30° − 45°	0.43 ± 2.04	− 2.29 ± 1.48	< 0.001
	45°–70°	0.91 ± 2.54	− 2.47 ± 2.28	< 0.001

Mean ± standard deviation.

OA: osteoarthritis.

Positive values represent the external rotation of the tibia.

Discussion

The present study demonstrated the alteration of SHM in patients with knee OA. The total tibial rotation in these patients was decreased compared with that in normal subjects during sagittal movement of the knee joint. The normal group showed external rotation while approaching the terminal knee extension. In addition, internal rotation was accompanied by an early period of flexion movement. By contrast, patients with OA showed reduced tibial rotation during identical sagittal movements. The tibiofemoral contact location is the thickest region of articular cartilage when the intact knee is at full extension [11]. A previous study reported that the junction of the knee joint moved to a thinner region of cartilage in the internally rotated tibia associated with anterior cruciate ligament (ACL)-deficient knee [12]. Decreased SHM in knee OA patients increases stress on the cartilage and accelerates the loss of cartilage. Thus, we considered that SHM is a biomechanical phenomenon for moving to a stable position on the articular surface.

SHM was defined as the tibial rotation when close to terminal extension [20, 21]. In the current study, patients with knee OA had different patterns of tibial rotation compared with those of normal subjects. The normal group showed a relatively large tibial rotation close to terminal extension. However, the group of patients with knee OA had no significant tibial rotation during the sagittal movement. In a gait analysis, the knee OA group had smaller external rotation moment during the early stance compared with that of asymptomatic subjects [16]. Knee extension should be accompanied by external rotation at an early stance phase, but the effects of moment direction were to reduce the magnitude of SHM in patients with knee OA. In patients with ACL deficiency, the tibia remained internally rotated throughout the stance phase [12, 19]. The result of gait analysis in patients with ACL injury was comparable to those in knee OA patients. ACL injury was an important factor leading to degenerative changes in the knee joint [29, 30]. Andriacchi et al. [11] attributed the initiation of knee OA after ACL injury to rotational changes. Although 74% of the patients in the present study were in KL grade 2, an early stage of diagnosis of knee OA, their SHM was markedly reduced. Therefore, the reduction of SHM is hypothesized to have a greater effect on the development than the progression of arthritis.

SHM is influenced by the surrounding soft tissues of the knee in addition to the geometry and shape of the articular surfaces [31]. The occurrence of SHM has been attributed to the larger medial femoral condyle and medial meniscus, rather than to the lateral condyle and meniscus [32]. Ligaments and capsules guide the movement path along with the joint geometry and shape [23, 33]. The posterolateral bundle of the ACL, collateral ligaments, and capsules restricted the longitudinal rotation at the full extension position [31, 34]. When the knee is at almost full extension, the ACL contributes to the posterior translation of the tibia, and, paradoxically, the ACL restricts external rotation [31]. Therefore, we believe that the main factor in the occurrence of SHM is the geometry and shape of the articular surfaces of the knee joint.

OA patients have an increased laxity of the medial compartment compared with that of healthy subjects with no evidence of knee OA [18]. While walking, the co-contraction of the medial muscles (vastus medialis–medial gastrocnemius) increased to compensate for the laxity of the medial compartment. Previous *in vitro* studies reported that tibial rotation was influenced differently by various quadriceps loads, regardless of hamstring loads on the medial or lateral side [35]. The tibial external rotation was smallest during quadriceps loads with medial hamstrings at a close-to-terminal extension. Thus, the co-

contraction of the medial muscles seems to reduce the SHM due to the anatomical location of muscle attachments. The limitation of this study is that the number of patients was insufficient to identify the changes according to knee OA grade; also, because inertial sensors are attached to the skin, they are affected by skin movement.

Conclusions

We found a reduction in the total tibial rotation and loss of the SHM in the unloaded OA knee. We suggest two main causes of tibial rotational changes in OA patients. First, changes in soft tissues, including the ACL, decrease the posterior translation of the tibia. The first cause is that the joint surface movement in patients with OA is insufficient to move to a stable position due to changes in soft tissues, including the ACL. In particular, this cause may be related to the loss of the SHM phenomenon when close to terminal extension. Second, increased medial muscle activation compensates for medial laxity, which may affect the reduction in the total amount of tibial rotation. Therefore, we reasoned that for the rehabilitation of the knee OA, it is necessary to promote proper joint surface movement of the knee joint and to suppress the hyperexcitability of the medial muscles.

Abbreviations

OA

Osteoarthritis; SHM:Screw-home movement; KL grade:Kellgren-Lawrence grade; VAS:Visual analog scale; WOMAC:Western Ontario and McMaster universities osteoarthritis index; IMU:Inertial measurement unit; ACL:Anterior cruciate ligament

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of the CM general hospital of the Republic of Korea (CMHCTC-19-001), and all participants provided written informed consent prior to commencement of the study.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and analyzed during the current study are available through the corresponding author on reasonable request.

Competing interests

The authors declare that they have no conflict of interest.

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Authors' contributions

JJ and JH contributed to the conception and design of the work. JJ, JH, SHL, and SC recruited the participants and collected the data. JJ, JH, DL, JY, and JK performed the data analysis and interpretation. All authors contributed to writing, as well as the review and approval of the final version of the manuscript.

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References

1. Roos EM, Arden NK. Strategies for the prevention of knee osteoarthritis. Nat Rev Rheumatol. 2016;12(2):92.
2. Roos M, Klässbo LS, Lohmander EMWOMAC, Osteoarthritis Index. Reliability, validity, and responsiveness in patients with arthroscopically assessed osteoarthritis. Scand J Rheumatol. 1999;28(4):210–5.
3. Hannan MT, Felson DT, Pincus T. Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. J Rheumatol. 2000;27(6):1513–7.
4. Heidari B. Knee osteoarthritis diagnosis, treatment and associated factors of progression: part II. Caspian J Intern Med. 2011;2(3):249–55.
5. Altman R, Asch E, Bloch D, Bole G, Borenstein D, Brandt K, Christy W, Cooke T, Greenwald R, Hochberg M. Development of criteria for the classification and reporting of osteoarthritis: classification of osteoarthritis of the knee. Arthritis Rheumatism: Official Journal of the American College of Rheumatology. 1986;29(8):1039–49.
6. Michael JW, Schlüter-Brust KU, Eysel P. The epidemiology, etiology, diagnosis, and treatment of osteoarthritis of the knee. Deutsches Arzteblatt International. 2010;107(9):152.

7. Cicuttini FM, Wluka AE, Forbes A, Wolfe R. Comparison of tibial cartilage volume and radiologic grade of the tibiofemoral joint. Arthr Rhuem. 2003;48(3):682–8.
8. Ding C, Cicuttini F, Scott F, Boon C, Jones G. Association of prevalent and incident knee cartilage defects with loss of tibial and patellar cartilage: a longitudinal study. Arthr Rhuem. 2005;52(12):3918–27.
9. Cicuttini FM, Jones G, Forbes A, Wluka AE. Rate of cartilage loss at two years predicts subsequent total knee arthroplasty: a prospective study. Ann Rheum Dis. 2004;63(9):1124–7.
10. Ding C, Cicuttini F, Jones G. How important is MRI for detecting early osteoarthritis? Nat Clin Pract Rheumatol. 2008;4(1):4–5.
11. Andriacchi TP, Mündermann A, Smith RL, Alexander EJ, Dyrby CO, Koo S. A framework for the in vivo pathomechanics of osteoarthritis at the knee. Ann Biomed Eng. 2004;32(3):447–57.
12. Andriacchi TP, Briant PL, Bevill SL, Koo S. Rotational changes at the knee after ACL injury cause cartilage thinning. Clin Orthop Relat Res. 2006;442:39–44.
13. Nagao N, Tachibana T, Mizuno K. The rotational angle in osteoarthritic knees. Int Orthop. 1998;22(5):282–7.
14. Astephen JL, Deluzio KJ, Caldwell GE, Dunbar MJ. Biomechanical changes at the hip, knee, and ankle joints during gait are associated with knee osteoarthritis severity. Journal of orthopaedic research. 2008;26(3):332–41.
15. Chang A, Hayes K, Dunlop D, Song J, Hurwitz D, Cahue S, Sharma L. Hip abduction moment and protection against medial tibiofemoral osteoarthritis progression. Arthr Rhuem. 2005;52(11):3515–9.
16. Landry SC, McKean KA, Hubley-Kozey CL, Stanish WD, Deluzio KJ. Knee biomechanics of moderate OA patients measured during gait at a self-selected and fast walking speed. J Biomech. 2007;40(8):1754–61.
17. Mündermann A, Dyrby CO, Andriacchi TP. Secondary gait changes in patients with medial compartment knee osteoarthritis: increased load at the ankle, knee, and hip during walking. Arthr Rhuem. 2005;52(9):2835–44.
18. Lewek MD, Rudolph KS, Snyder-Mackler L. Control of frontal plane knee laxity during gait in patients with medial compartment knee osteoarthritis. Osteoarthritis cartilage. 2004;12(9):745–51.
19. Vincent KR, Conrad BP, Fregly BJ, Vincent HK. The pathophysiology of osteoarthritis: a mechanical perspective on the knee joint. PM&R. 2012;4(5):3–9.
20. Hallen L, Lindahl O. The “screw-home” movement in the knee-joint. Acta Orthop Scand. 1966;37(1):97–106.
21. Piazza SJ, Cavanagh PR. Measurement of the screw-home motion of the knee is sensitive to errors in axis alignment. J Biomech. 2000;33(8):1029–34.
22. De Palma AF. Diseases of the knee: management in medicine and surgery: Lippincott; 1954.
23. Wilson DR, Feikes J, Zavatsky A, O'connor J. The components of passive knee movement are coupled to flexion angle. J Biomech. 2000;33(4):465–73.

24. Kim HY, Kim KJ, Yang DS, Jeung SW, Choi HG, Choy WS. Screw-home movement of the tibiofemoral joint during normal gait: three-dimensional analysis. Clinics in orthopedic surgery. 2015;7(3):303–9.
25. Cho HJ, Chang CB, Yoo JH, Kim SJ, Kim TK. Gender differences in the correlation between symptom and radiographic severity in patients with knee osteoarthritis. Clinical Orthopaedics Related ResearchÂ®. 2010;468(7):1749–58.
26. Seel T, Raisch J, Schauer T. IMU-based joint angle measurement for gait analysis. Sensors. 2014;14(4):6891–909.
27. Takeda R, Tadano S, Natorigawa A, Todoh M, Yoshinari S. Gait posture estimation using wearable acceleration and gyro sensors. J Biomech. 2009;42(15):2486–94.
28. Jeon JW, Lee DY, Yu JH, Kim JS, Hong J. Detection and Quantification of Screw-Home Movement Using Nine-Axis Inertial Sensors. The Journal of Korean Physical Therapy. 2019;31(6):333–8.
29. Matsumoto H, Seedhom BB, Suda Y, Otani T, Fujikawa K. Axis location of tibial rotation and its change with flexion angle. Clinical Orthopaedics Related ResearchÂ®. 2000;371:178–82.
30. Goodfellow J, O'Connor J. The mechanics of the knee and prosthesis design. J Bone Joint Surg Br. 1978;60-B(3):358–69.
31. Wilson D, Feikes J, Oâ€™connor J. Ligaments and articular contact guide passive knee flexion. J Biomech. 1998;31(12):1127–36.
32. Yagi M, Wong EK, Kanamori A, Debski RE, Fu FH, Woo SL. Biomechanical analysis of an anatomic anterior cruciate ligament reconstruction. Am J Sports Med. 2002;30(5):660–6.
33. Buckland-Wright J, Lynch J, Dave B. Early radiographic features in patients with anterior cruciate ligament rupture. Ann Rheum Dis. 2000;59(8):641–6.
34. Roos H, Adalberth T, Dahlberg L, Lohmander LS. Osteoarthritis of the knee after injury to the anterior cruciate ligament or meniscus: the influence of time and age. Osteoarthritis Cartilage. 1995;3(4):261–7.
35. Victor J, Labey L, Wong P, Innocenti B, Bellemans J. The influence of muscle load on tibiofemoral knee kinematics. Journal of orthopaedic research. 2010;28(4):419–28.

Figures

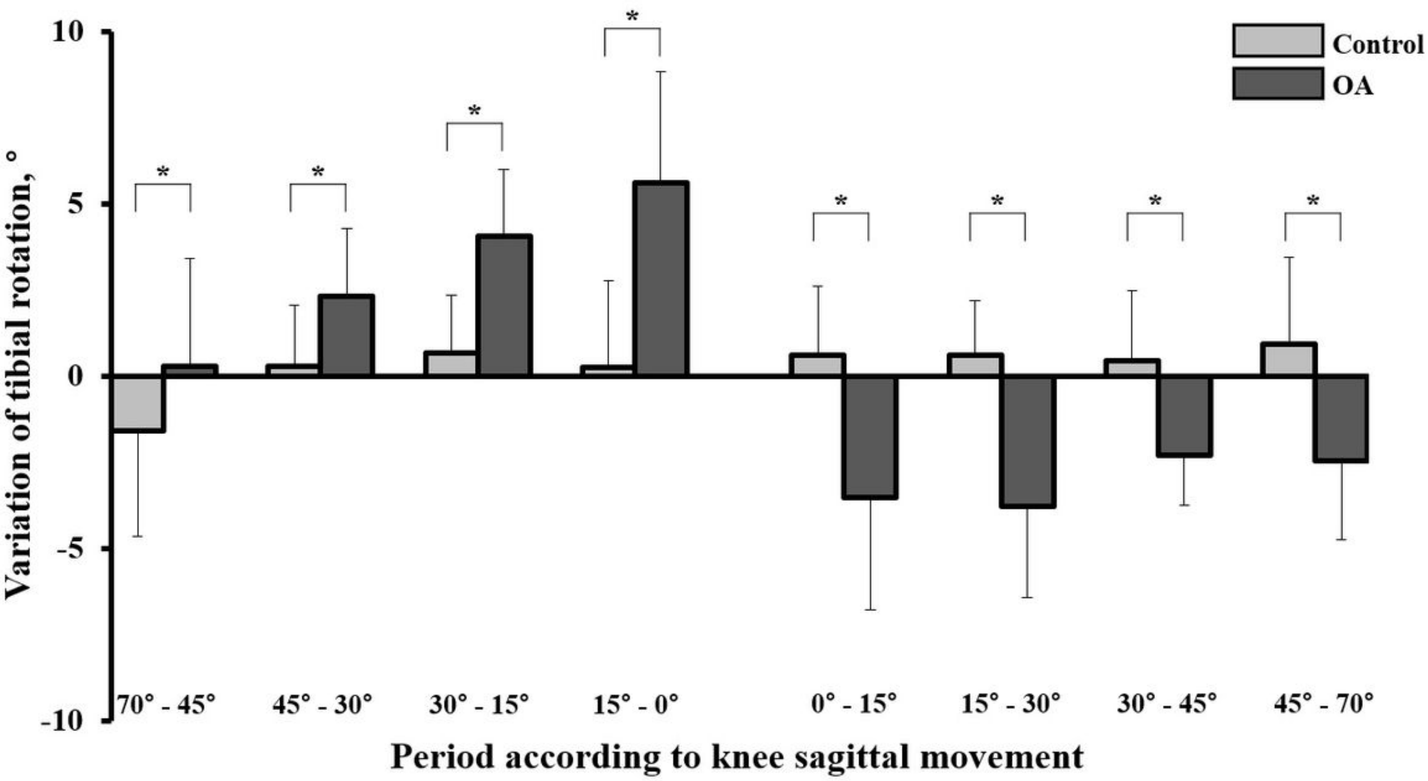

Figure 1

Variation of tibial rotation for each period in the knee osteoarthritis (OA) and control groups. Positive values represent the external rotation of the tibia. The asterisk (*) indicates a significant difference between the OA group and the control group ($P < 0.001$; knee flexion from 70° to 45° was $P = 0.014$).