Biomechanical Gait Analysis in Patients with Osteonecrosis of the Femoral Head

Análise biomecânica da marcha de pacientes com osteonecrose da cabeça do fêmur

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Abstract

Objectives Although osteonecrosis of the femoral head is a prevalent condition, its effects on gait parameters have not been thoroughly studied and are not well-established in the current literature. The primary aim of the present study is to describe gait in patients with a diagnosis of osteonecrosis.

Methods This is a cross-sectional study. Nine patients diagnosed with osteonecrosis of the femoral head who were regularly followed-up at an outpatient clinic were selected for the present study and underwent gait analysis using Vicon Motion Capture Systems. Spatiotemporal data was obtained, and joint angles were calculated using an Euler angle coordinate system. Distal coordinate systems were used to calculate joint moments and force plates to obtain ground reaction forces.

Results Patients with osteonecrosis presented with slower velocity (0.54 m/s $\pm$ 0.19) and smaller cadence (83.01 steps/min $\pm$ 13.23) than healthy patients. The pelvic obliquity range of motion was of 10.12° $\pm$ 3.03 and rotation was of 18.23° $\pm$ 9.17. The mean hip flexion was of 9.48° $\pm$ 3.40. Ground reaction forces showed reduced braking and propelling forces. Joint moments were reduced for flexion and adduction (0.42 Nm/kg $\pm$ 0.2 and 0.30 Nm/kg $\pm$ 0.11, respectively) but the abduction moment was increased (0.42 Nm/kg $\pm$ 0.18).

Conclusions The present study showed that osteonecrosis of the femoral head presents compensatory gait mechanisms, with increased pelvic motion and decreased knee flexion to protect the hip joint. Decreased moments for hip flexion and adduction were also identified and muscle weakness for those groups may be correlated to the disease.

Keywords

- arthroplasty, replacement, hip
- biomechanical phenomena
- femur head necrosis
- gait analysis

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Resumo

Objetivos Embora a osteonecrose da cabeça do fêmur seja uma lesão prevalente, seus efeitos sobre os parâmetros da marcha não foram minuciosamente estudados e não estão bem estabelecidos na literatura atual. O objetivo principal do presente estudo é descrever a marcha em pacientes com osteonecrose.

Métodos Trata-se de um estudo transversal. Nove pacientes com diagnóstico de osteonecrose da cabeça do fêmur, sob acompanhamento regular em ambulatório, foram selecionados para o presente estudo e submetidos à análise da marcha com Vicon Motion Capture Systems. Os dados espaciais e temporais foram obtidos e os ângulos articulares foram calculados com o sistema de coordenadas angulares de Euler. Sistemas de coordenadas distais e plataformas de força foram utilizados para o cálculo de momentos articulares e de forças de reação ao solo, respectivamente.

Resultados Os pacientes com osteonecrose apresentaram menor velocidade (0,54 m/s $\pm$ 0,19) e menor cadência (83,01 passos/minuto $\pm$ 13,23) do que pacientes saudáveis. As amplitudes de movimento de obliquidade e rotação pélvica foram de 10,12° $\pm$ 3,03 e 18,23° $\pm$ 9,17, respectivamente. A média de flexão do quadril foi de 9,48° $\pm$ 3,40. O estudo das forças de reação ao solo revelou redução das forças de frenagem e propulsão. Os momentos articulares de flexão e adução caíram (0,42 Nm/kg $\pm$ 0,2 e 0,30 Nm/kg $\pm$ 0,11), mas o momento de abdução aumentou (0,42 Nm/kg $\pm$ 0,18).

Conclusões O presente estudo mostrou que a osteonecrose da cabeça do fêmur é associada a mecanismos compensatórios da marcha, com aumento da movimentação pélvica e diminuição da flexão do joelho para proteção da articulação do quadril. A redução dos momentos de flexão e adução do quadril também foi identificada e a fraqueza destes grupos musculares pode estar correlacionada à doença.

Palavras-chave

- artroplastia de quadril
- fenômenos biomecânicos
- necrose da cabeça do fêmur
- análise da marcha

Introduction

Osteonecrosis of the femoral head (ONFH) is a disease caused by the disruption of the blood supply to the femoral head, culminating in medullary bone tissue death and, subsequently, evolving to subchondral bone collapse and femoral head deformity.[1–9] It is a debilitating, progressive condition, with the potential for causing degenerative hip disease and functional loss.[1,2,10] The condition affects patients aged between 30 and 50 years old,[7,9] with an incidence of 10,000 to 20,000 cases per year in the USA,[7,10] and is responsible for 10% of all total hip arthroplasties (THAs) performed in the USA.[3]

Although the pathogenesis of ONFH is unclear,[4] it is considered a multifactorial disease in which both genetic and environmental factors play a role in its outcome.[5] Ischemia can arise from endothelial damage, thrombosis, increased intraosseous pressure, cytotoxic effects from medications such as corticosteroids, increasing osteocyte apoptosis, and traumatic events.[3] Considering its etiology, ONFH can be idiopathic, traumatic, and nontraumatic. Osteonecrosis due to trauma is generally related to femoral neck fractures, hip dislocations, or repetitive microtraumas. The nontraumatic risk factors can be cortisone therapy, alcohol abuse, blood dyscrasias (sickle cell disease, Factor V Leiden mutation, decreased protein C or S, increased blood lipoprotein), systemic lupus erythematosus, Gaucher disease, Caisson disease, and some less commonly documented factors such as human immunodeficiency virus infection.[4,6,9]

Patients are often asymptomatic in the early stages, progressing to pain in the groin or in the gluteal sulcus and decreased hip range of motion (RoM), particularly for internal rotation.[4,6,9] Early diagnosis is essential to prevent the progression to collapse. Radiographs are the first imaging methods obtained; however, these may be normal. If there is clinical suspicion with unaltered radiographs, a magnetic resonance imaging (MRI) exam should be performed since it is up to 100% sensitive for the diagnosis of ONFH.[2,3]

After the diagnosis, staging is used to define treatment. The Ficat and Arlet classification is the most widely used system in clinical settings. Radiographical signs are used to stage between 0 to IV in the Ficat and Arlet system.[11] The Steinberg classification system also uses radiographic criteria to classify mild, moderate, or severe stages according to the percentage of the femoral head affected. The Association Research Circulation Osseous (ARCO) system includes scintigraphy, MRI, and computed tomography (CT) scan findings that help to determine the position and total area of necrosis.[10,12]

Treatment choice is based on staging. Nonoperative treatments include oral medications (primarily bisphosphonates), hyperbaric chamber, extracorporeal shockwave therapy, and pulsed electromagnetic fields.[8,9] Operative treatment alternatives include core decompression, osteotomies, bone grafting, and THA. Hip arthroplasties are preferred due to their high success rate; however, there is a higher risk of aseptic loosening and infection in younger patients.[7,8]

Biomechanical gait analysis is one of the most reliable forms of assessing kinetic and kinematic gait parameters,[13] producing adequate and detailed data for evaluating patients with gait impairment diseases, their functional conditions, and compensatory mechanisms.[14]

Despite the high incidence, economic burden, and severe functional impairments of the disease, there is a paucity of studies evaluating its effect on gait parameters. The present cross-sectional study aimed to assess and describe gait parameters of patients with a diagnosis of ONFH, to compare this function with the existing literature on gait analysis of conditions with similar degenerative outcomes, as well as to describe a possible compensatory mechanism for identified gait deficits.

Methods

Participants

This was a cross-sectional study. It was approved by the Research Ethics Committee (47578621.0.0000.5404) of the institution. Nine patients who decided to participate were previously diagnosed with ONFH and were regularly followed-up at the Adult Hip Pathologies Outpatient Clinic at the clinical hospital were included. Patients who had previously undergone definitive surgical procedures to treat ONFH; had other pathologies such as neurological, syndromic, orthopedic, or lower limb deformities that could affect gait; had undergone previous procedures that affected gait; or had a recent lower limb trauma were excluded from the study. All participants gave their informed written consent for the present study.

Gait Analysis

Before gait analysis, a data collection form containing age, gender, and Ficat and Arlet and ARCO classifications were filled out according to clinical data, patient medical chart, and the most recent patient imaging in the hospital imaging system. The classifications were reviewed by the authors. Body mass was measured using a balance scale and was converted to Newtons for normalization.

Gait analysis was performed using a 12-camera (Vero) Vicon motion capture system (Vicon, Oxford, United Kingdom) at 120 Hz for both kinetic and kinematic data, and two AMTI force plates built in a fixed 10-m-long walkway (Advanced Mechanical Technology Inc., Watertown, MA, USA) for ground reaction forces (GRFs). Three-dimensional (3D) reconstruction and data analysis were performed using The Motion Monitor xGen system (Innovative Sports Training Inc., Chicago, IL, USA). Seven four-marker sensor clusters were positioned in the sacrum, the right and left thighs, the right and left shanks, and the right and left foots of each participant, for segment selection. A stylus was used to digitize anatomical landmarks for the 3D reconstruction (►**Fig. 1**), segment, and joint center identification. The participants were then asked to walk at a comfortable pace over the walkway for 30 seconds and data were collected from 10 to 20 gait cycles for each patient. One value for each variable was collected every 10 milliseconds.

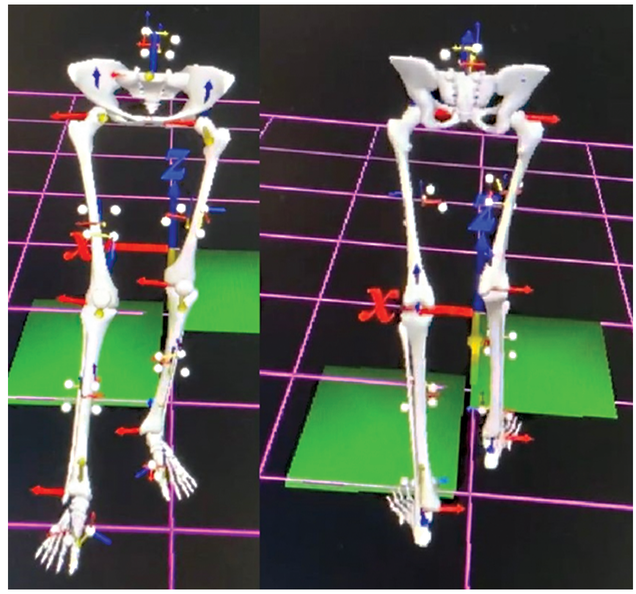

Fig. 1 Three-dimensional animation of gait during one trial with The Motion Monitor xGen system (Innovative Sports Training Inc., Chicago, IL, USA).

Joint centers and body segments were identified with The Motion Monitor (Innovative Sports Training Inc., Chicago, IL, USA) and segment mass and inertia were calculated using nonlinear regression equations. Euler angles were used to calculate joint angles, through a distal coordinate system, in a flexion-extension, abduction-adduction, and internal-external rotation sequence. Pelvic, hip, knee, and ankle angles data were collected bilaterally; however, only the affected limb was analyzed. The moment was calculated using inverse dynamics and was expressed in the distal segment coordinate system; division normalization was used for both moment and force data. The authors used an internal perspective to evaluate kinetic data and interpret gait function.

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY, USA). All data were evaluated for normality. One-sample *t*-tests were used to compare characteristics of peak values found in the literature for healthy participants.[14–16] Significance was established at $p < 0.05$.

Results

Nine participants who fulfilled the eligibility criteria were included in the present study, of which 66.7% ($n = 6$) had bilateral hip disease and 33.3% ($n = 3$) had unilateral hip disease. So, a total of 15 hips were analyzed. Six patients were men, and the mean age of all patients was 44.11 years old (± 14.8; $p = 0.101$). The average weight was 76.1 ± 17.6 kg, and the mean height was 166.6 ± 6.7 cm. The Ficat and Arlet classification was stage IV for 13 of the evaluated hips, stage

Table 1 Spatiotemporal linear kinematics of gait in patients with a diagnosis of osteonecrosis of the femoral head

	Reference*[14]	Mean	SD	*p-value*
Gait Velocity (m/s)	1.32	0.54	0.19	< 0.01
Cadence (steps/min)	99	83.01	18.23	0.015
Stride length (m)	1.21	0.78	0.24	< 0.01
Stride frequency (Hz)		0.69	0.15	
Cycle duration (s)	1.22	1.51	0.35	0.019

Abbreviation: SD, standard deviation.
*Reference for normal gait parameters found in the literature.

III for 1, and stage II for 1 hip evaluated. The ARCO classification was 4 for 13 of the hips, 3A for 1, and 1 for 1 hip. In sum, there was a significant preponderance of hips evaluated post femoral head collapse, which accounts for Ficat and Arlet stage IV and ARCO stage 4.

Kinematic data were analyzed linearly for gait speed, cadence, stride length, stride frequency and cycle duration, and angularly for joint angles. Results for kinematic analysis are assembled in ►**Tables 1**, **2** and **3**. Linear kinematics are presented in mean and standard deviation (S|D), and joint angles are described in mean, RoM, and SD for every joint movement.

Briefly, gait velocity was 0.54 ± 0.19 with a cadence of 83.01 ± 13.23. Pelvic obliquity reached a RoM of 10.12 ± 3.03 and rotation of 18.23 ± 9.17. Hip flexion mean was 9.48 ± 3.40 and RoM of 23.62 ± 7.56.

Kinetic data were analyzed for GRFs and joint moment. The GRFs were normalized for body weight (BW) and moment for body mass (kg).[13,17] The GRFs are presented in ►**Figura 2**, and joint moment data are presented in ►**Table 4**.

Discussion

Gait analysis is a helpful tool in evaluating patient functionality in osteomuscular diseases. Thus, physicians can assess a daily activity that can greatly impair quality of life, as well as invest in immediate nonoperative measures that can improve pain, functionality, and, eventually, surgical outcomes. Although several systems exist to evaluate hip performance,[18] they are dependent on patient self-evaluation and on physical examination and often lack objectivity.

Osteonecrosis of the femoral head is a fairly incident condition that can arise from several etiologies; however, very few studies evaluate its effect on gait, perhaps due to its similar clinical outcomes to hip osteoarthritis (OA). The present study described gait kinetics and kinematics and compared results with the relevant literature both in participants with ONFH and asymptomatic, healthy individuals.

Our study found smaller values of gait speed, cadence, and stride length, and larger cycle duration compared with those found by Cho et al.[19] in 39 participants with ONFH before

Table 2 Pelvic angular kinematics of gait in patients with a diagnosis of osteonecrosis of the femoral head with unilateral and bilateral disease and in the subject group overall

			Reference*[15]	Angle	SD	*p-value*
Unilateral	Pelvic Tilt	Mean	11.4	40.66	19.88	0.63
	Obliquity	Mean	0.1	5.56	4.64	0.89
		RoM	10.3	10.29	4.44	0.49
	Rotation	Mean	0	10.28	5.18	0.03
		RoM	12.5	15.70	5.94	0.22
Bilateral	Pelvic Tilt	Mean	11.4	35.13	16.34	0.08
	Obliquity	Mean	0.1	4.04	2.26	0.04
		RoM	10.3	10.04	5.20	0.45
	Rotation	Mean	0	11.70	9.66	0.02
		RoM	12.5	19.5	10.7	0.85
Overall	Pelvic Tilt	Mean	11.4	32.60	17.9	< 0.01
	Obliquity	Mean	0.10	4.55	4.68	< 0.01
		RoM	10.3	10.12	3.03	0.45
	Rotation	Mean	0	11.23	8.1	< 0.01
		RoM	12.5	18.23	9.17	0.05

Abbreviations: RoM, range of motion; SD, standard deviation.
*Reference for normal gait parameters found in the literature.

Table 3 Hip, knee and ankle angular kinematics of gait in patients with a diagnosis of osteonecrosis of the femoral head

			Reference*[15]	Angle	SD	*p-value*
Hip	Flexion	Mean	16.4	9.48	3.40	< 0.01
		RoM	43.6	23.62	7.56	< 0.01
	Extension	Mean	−7.4	7.29	3.66	< 0.01
		RoM	42.0	18.24	8.45	< 0.01
	Abduction	Mean	−8.0	5.04	2.50	< 0.01
		RoM	14.0	13.35	7.86	0.40
	Adduction	Mean	−0.4	4.01	2.14	< 0.01
		RoM	14.0	10.16	4.29	< 0.01
	Internal Rotation	Mean	0.7	5.29	2.58	< 0.01
		RoM	0.9	12.39	5.28	< 0.01
	External Rotaion	Mean	0.7	7.54	1.58	< 0.01
		RoM	0.9	17.36	3.47	< 0.01
Knee	Flexion	Mean	20.6	16.21	2.58	< 0.01
		RoM	61.2	51.9	6.69	0.02
Ankle	Dorsiflexion	Mean	29.4	4.31	1.62	0.004
		RoM	6.2	24.67	9.17	0.08
	Plantar Flexion	Mean	−29.4	5.05	1.51	0.026
		RoM	−6.2	25	6.61	0.041

Abbreviations: RoM, range of motion; SD, standard deviation.
*Reference for normal gait parameters found in the literature.

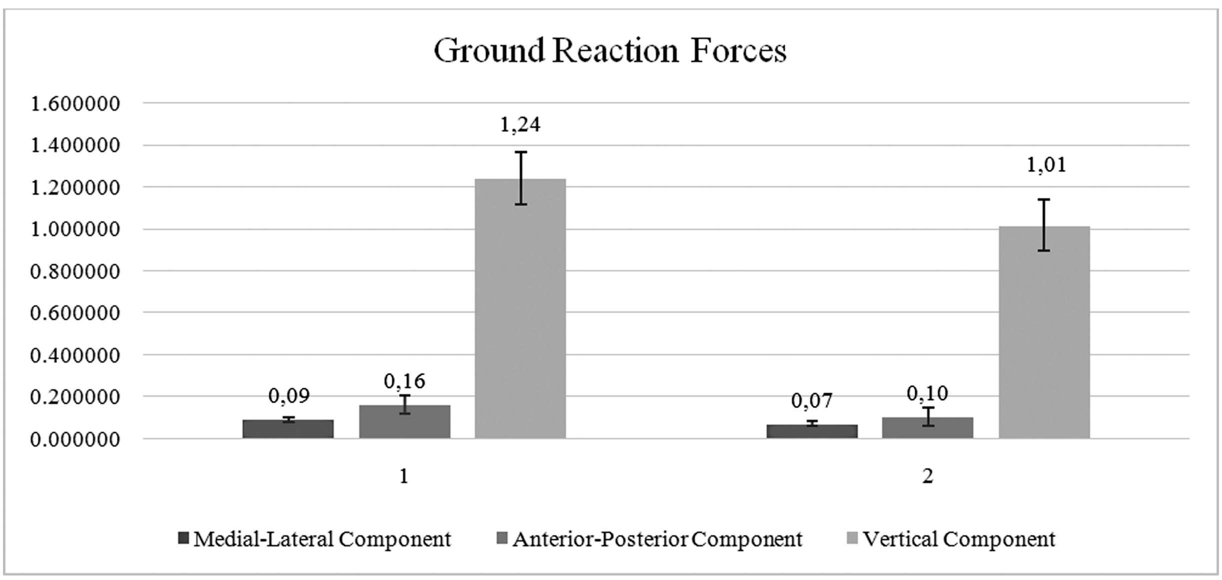

Fig. 2 Means and standard deviations for ground reaction forces (GRFs) (N/BW) in patients with osteonecrosis of the femoral head. 1. Average mean GRF in the second peak stance. 2. Average mean GRF in the first peak stance. Standard deviation for medial-lateral (ML) component of 0.01 ($p = 0.29$), anteroposterior (AP) component of 0.04 ($p = 0.015$) and vertical (V) component of 0.12 ($p = 0.34$).

THA. All spatiotemporal data were compared with those found by Holden et al.,[14] showing significantly minor averages for both distance and temporal parameters in healthy individuals. While those results were expected in comparison with physically fit participants, differences found between both ONFH groups may arise from the disease severity in patients analyzed in the present study. Slower velocity is commonly found in degenerative hip conditions with decreased joint RoM and pain.

Overall, the pelvic angular parameters were abnormally different to those found in healthy participants by Otayek et al.,[15] particularly regarding pelvic mean upwards obliquity and in mean and RoM for rotation. However, these parameters were similar to those observed in the ONFH group by

Table 4 Joint moments in patients with osteonecrosis of the femoral head

		Reference*[16]	Mean	SD	*p-value*
Hip flexion / Extension moment (Nm/kg)	Loading response	0.92	0.42	0.20	< 0.01
	Terminal stance	0.65	0.51	0.20	0.06
	Terminal Swing		0.34	0.09	
Hip abduction / Adduction moment (Nm/kg)	Initial single support	0.24	0.42	0.18	< 0.01
	Middle single support		0.28	0.09	
	Terminal single support	0.83	0.30	0.11	< 0.01

Abbreviation: SD, standard deviation.
*Reference for normal gait parameters found in the literature.

Cho et al.,[19] which may signify a compensatory mechanism to minimize the effects of decreased hip RoM in gait. When comparing groups of patients with ONFH, those with unilateral disease have isolated increased mean pelvic rotation, whereas those with bilateral hips affection have an increase in both mean pelvic obliquity and mean pelvic rotation.

In another study,[19] hip mean flexion and RoM were greater in patients with ONFH, as well as in healthy participants.[15,20] Our study found significantly greater mean hip extension, abduction, and adduction, although, for the latter two, the RoM was smaller than in the unaffected hips in other studies; however, for abduction RoM, that was insignificant.[15] As for rotation, our study found greater mean internal and external rotation in patients than those observed by Otayek et al.[15] in unaffected individuals, but similar values to those observed by Cho et al.[19] in other participants with ONFH and in a healthy control group. Limited hip motion is often associated with ONFH, particularly after femoral head collapse and initially for internal rotation. Although the evaluated group in the present study comprised most patients with an advanced degenerative disease, which could limit RoM on its own, limited hip angular values might also arise from patient pain.[4]

Knee mean flexion and range were slightly reduced when compared with kinematics in physically fit participants. Ankle dorsiflexion and plantarflexion were also slightly decreased; however, it is insignificant for ankle dorsiflexion RoM.[15,20] As reported by Bejek et al.,[21] when evaluating participants with OA, increased pelvic movement and reduced knee joint motion could mean an adjusting mechanism to maintain overall speed and stride length, as well as to protect the affected hip joint from excess motion and subsequent pain.

In kinetic data, GRFs were evaluated for stance phases of gait. Participants presented with a bimodal aspect of stance. The average impact peak (first peak) neared the BW of the participants, showing little muscle component in generating this reaction force, which is to be expected. The average active peak (second peak) was above BW at a 1.24 Nm/BW, translating to push-off during gait. The detected values were significantly similar to those found by Nilsson et al. during walking at slow speed in healthy participants, indicating effective muscle activity during push-off.[16] There was no significant difference in medial-lateral GRF in comparison with normal hips. Braking and propulsive GRF translate to the anteroposterior component obtained from the force plates. The braking and propulsive second peaks were significantly smaller than the value observed in healthy individuals.[16] These values may be a consequence of the slower gait speed observed, since propulsive force is in direct proportion to gait velocity and is consistent with a tendency of preserving the hip joint.

The hip joint moment was analyzed in all affected hips. Flexion moments in loading response and terminal swing, as well as abduction moments for all single support phases, were smaller than those found by Cho et al. in other participants with ONFH.[19] As previously mentioned, this may be due to the higher severity of the evaluated cases in the present study, although the gravity classifications are not disclosed in the aforementioned article, neither are the normalization methods. Regarding healthy participants, all data were smaller for flexion moment. There was no significant difference in maximum extension moment, which occurs during terminal stance, from that found by Moisio et al.[17] There is a significant muscle weakness for flexion, particularly the rectus femoris and the iliacus, which participate most actively during gait, especially in loading response and swing phases.[22] In contrast, the hip extension moment is quite similar to the ONFH group evaluated by Cho et al.,[19] but it was slightly decreased when compared with healthy individuals.[17] Regarding abduction moment, which was higher than what was found by Moisio et al.,[17] the present study shows a good function for the gluteus medius, which acts during the initial single support phases of gait.[22] In contrast, adduction moment was significantly reduced when compared with normal hip motion.[17] A stronger abduction strength may arise in patients with ONFH from the necessity to stabilize the pelvis during gait, since it is apparent that pelvic motion may be one of the main compensatory mechanisms in ONFH gait.

Conclusion

Overall, ONFH participants showed significantly slower time-distance parameters in comparison with healthy individuals. In angular kinematics, joint angles are generally decreased for ONFH hip mean and RoM; compensatory mechanisms appeared to be present. There was an increased pelvic movement for both obliquity and rotation, and decreased knee flexion, which appeared to decrease hip motion and reduce pain in order to maintain spatiotemporal parameters. The observed GRFs were compatible with a bimodal vertical GRF in healthy participants. Braking and propulsive forces were significantly smaller and might be related to a slower walking speed. In the present study, the patients were found to have increased muscle weakness for hip flexor and adductor muscles; however, increased abduction strength in single support phases was observed when compared with unaffected participants.

The present study focused primarily on patients with ONFH post femoral head collapse and further studies could focus on a more varied group of disease stages. Future studies could also evaluate gait parameters before and after nonoperative and operative treatments.

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Conflict of Interests
The authors have no conflict of interests to declare.

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