Title: Sagittal alignment and mobility of the thoracolumbar spine are associated with radiographic progression of secondary hip osteoarthritis

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Running title: Spinal impairment and hip OA progression

ABSTRACT

Objective:

To identify predictors of radiographic progression of hip osteoarthritis (OA) over 12 months among functional hip impairments and spinal alignment and mobility.

Design:

Fifty female patients with secondary hip OA, excluding those with end-stage hip OA, participated in this prospective cohort study. Joint space width (JSW) of the hip was measured at baseline and 12 months later. With radiographic progression of hip OA over 12 months (>0.5 mm in JSW) as dependent variable, logistic regression analyses were performed to identify predictors for hip OA progression among functional impairments of the hip and spine with and without adjustment for age, body mass index (BMI), and minimum JSW at baseline. The independent variables were hip pain, Harris hip score, hip morphological parameters, hip passive range of motion and muscle strength, and alignment and mobility of the thoracolumbar spine at baseline.

Results:

Twenty-one (42.0%) patients demonstrated radiographic progression of hip OA. Multivariable logistic regression analysis showed that larger anterior inclination of the spine in standing position (adjusted OR [95% CI], 1.37 [1.04–1.80]; P = 0.028) and less thoracolumbar spine mobility (adjusted OR [95% CI], 0.96 [0.92–0.99]; P = 0.037) at baseline were statistically significantly associated with radiographic progression of hip OA, even after adjustment for age, BMI, and minimum JSW.

Conclusions:

The findings suggest that spinal alignment and mobility should be considered when assessing risk and designing preventive intervention for radiographic progression of secondary hip OA.

Keywords: Hip osteoarthritis, Spine, Alignment, Mobility, Progression

INTRODUCTION

Prevention of hip osteoarthritis (OA) progression in the mild-to-moderate OA stage is a critical challenge. However, there is no evidence to suggest that conservative treatment slows hip OA progression. One of the reasons could be that the risk factors for hip OA progression remain to be fully elucidated. Hip OA progression seems to be multifactorial. Evidence supports that atrophic bone response and superolateral migration of the femur head are risk factors for hip OA progression, and there is also conflicting evidence that higher age, female sex, and a narrower joint space width (JSW) at baseline are associated with hip OA progression[1,2]. However, few modifiable risk factors in the conservative treatment have been found.

Inappropriate mechanical loading on the joint has been believed to be the modifiable risk factor of OA progression[3]. Overloading on the joint can be caused by joint impairment and/or excessive external loading[4]. Regarding joint impairment, for the hip joint, muscle weakness (e.g., weakness of the hip abductors and external rotators) changes the contact pressure distribution and increases the contact pressure at the lateral edge of the contact area[5]. Moreover, reduced range of motion (ROM) of the hip (e.g., reduced hip abduction) increases the hip contact force during walking[6]. These findings based on the numerical finite element analysis and simulation analysis suggest an adverse effect of hip impairment on articular tissues; however, no study has demonstrated the association between hip impairment and radiographic progression of hip OA.

Regarding external hip loading, daily cumulative hip moment, which is the product of hip joint moment impulse during the stance phase of gait and the mean number of steps per day, has been recently identified as a risk factor for radiographic progression of hip OA[7].This finding emphasizes the need for investigation of the association between mechanical factor and hip OA progression. However, other factors related to external hip loading causing hip OA progression have not been identified. Malalignment of the pelvis and spine is common in patients with hip OA[8] and can be an underlying factor of overloading on the hip during standing and walking because of the increasing moment arm of gravity force[9]. Furthermore, given that most daily activities (e.g., forward bending, sit-to-stand, and putting on socks) are achieved through a combination of hip and spinal motions in the hip-spine complex[10,11], a decrease in the mobility of the hip can lead to an increase in mechanical

stress of the spine, and vice versa. There is evidence that patients with low back pain (LBP) who have reduced hip ROM and positive provocative hip test show worse LBP-related function compared with patients with LBP who have no physical examination findings in the hip[12]. In the opposite direction, decreased spinal motion can cause a relative increase in hip motion and may also induce excessive mechanical loading on the hip during motion[13]. Such a pathological condition is known as a type of hip-spine syndrome[14]. Therefore, malalignment of the pelvis and spine and less spinal mobility may possibly affect hip OA progression.

Identifying the risk factor for hip OA progression from among hip impairments and spinal alignment and mobility is clinically useful because assessment of these impairments is generally recommended for patients with hip OA[15,16]. Additionally, these impairments can be quantitatively measured using goniometer, handheld dynamometer, and other easy-to-use instruments in a clinical setting[17–19]. This study aimed to identify predictors for radiographic progression of hip OA over 12 months among hip impairment and spinal alignment and mobility, which are clinically measurable and modifiable. We hypothesized that worsening of the spinal alignment and mobility as well as hip impairment is associated with radiographic progression of hip OA.

PATIENTS AND METHODS

Patients

Patients were selected from non-surgical outpatients in the Department of Orthopaedic Surgery at Kyoto University Hospital. Patients with secondary hip OA aged 20 years and older were enrolled continuously from April 2013 to March 2015. A total of 53 patients were eligible for inclusion in our study and were measured at baseline. Three patients were excluded from analysis because of missing measurement at 12 months later. In this prospective cohort study, we used the same cohort as in a previous study[7].

The inclusion criteria were as follows: (1) a diagnosis of preosteoarthritis (acetabular dysplasia with no other abnormal radiographic findings) or early (slight joint space narrowing [2 mm

or more] and abnormal subchondral sclerosis) or advanced-stage hip OA (marked joint space narrowing [less than 2 mm] with or without cysts or sclerosis) hip OA[20], and (2) ability to walk without any assistive device in daily life. The exclusion criteria were as follows: (1) patients with a baseline JSW of <0.5 mm, as >0.5 mm/year in JSW was defined as hip OA progression; (2) a history of previous hip surgeries (e.g., osteotomy, arthroplasty); and (3) neurologic, vascular, or other conditions that affect gait or activity of daily living. No patient with femoroacetabular impingement was noted in our cohort. Our sample was biased in gender (percentage of males; 7.1%), similar to previous reports on secondary hip OA (percentage of males; 7.6–9.2%)[21,22]. Therefore, only female patients were included in this study. Given that the degree of disease progression (minimum JSW) at baseline is a risk factor for hip OA progression[1,2], the side on which the radiographic OA change was more severe was used in the analysis for the patients with bilateral hip OA. All participants provided informed consent, and the protocol was approved by the Ethics Committee of the Kyoto University Graduate School and Faculty of Medicine (protocol identification number: E1683).

Radiographic progression of hip OA

The radiographic progression of hip OA was assessed with JSW in a digital supine anteroposterior radiograph of the pelvis obtained in a standardized manner by the same skilled radiology technicians. A negligible difference was found in radiographic parameters with regard to hip dysplasia and joint space width between supine and standing anteroposterior radiographs[23,24]. Therefore, we used radiograph in the supine position to improve image quality[23]. We used radiographs taken for general practice to avoid unnecessary exposure to radiation. To assess the change in JSW, the films at baseline and approximately 12 months later were paired by patients but blinded as to patient and sequence to the reader to avoid bias[25]. All radiographic measurements were performed by a single experienced examiner. Images were reviewed and measured using Centricity Enterprise Web, version 3.0 (GE Health care, Buckinghamshire, England). The JSW was measured in 0.1 mm increments at three locations, namely, lateral margin of the subchondral sclerotic line, apical transection of the weight-bearing surface by a vertical line through the center of the femoral head, and medial margin of the weight-bearing surface bordering on the fovea. If the minimum JSW was

found aside from the three locations in the weight-bearing area, the JSW of the narrowest point was also recorded as a fourth measurement. Minimum JSW was defined as the smallest of the three or four measurements[26]. The radiographic progression of hip OA was defined as a reduction of >0.5 mm in JSW at any of the three or four locations[27,28]. The intrarater reliability [intraclass correlation (ICC) 1,1] of JSW measurement for 20 randomly selected radiographs was 0.99. The MDC_{95} (MDC at the 95% confidence level) of the JSW in the current study was 0.39 mm.

Morphological assessment of hip joint

From the radiograph, Sharp angle, lateral center edge (CE) angle, acetabular head index (AHI), and acetabular roof obliquity (ARO) were measured to assess morphological abnormalities. These measurements are reliable and commonly used to diagnose dysplasia and hip OA[18]. The intrarater reliabilities [ICC (1,1)] for these measurements were 0.95 to 0.98.

Pain and Harris hip score

The hip pain intensity and functional status of the patients were assessed at baseline using a 100-mm visual analogue scale and Harris hip score. The hip pain intensity was questioned as the average hip pain during daily life in the last 3 months.

Hip ROM and muscle strength

A single examiner measured passive hip ROM and the maximal isometric hip muscle strength using a standard goniometer (Sakai medical Co., Ltd, Tokyo, Japan) and a handheld dynamometer (μTAS F-1; Anima Co., Ltd, Tokyo, Japan) at baseline in accordance with previous studies[18,29,30].

Details of ROM and muscle strength tests were described elsewhere[30]. Briefly, hip ROM was measured in flexion, extension, abduction, adduction, and external and internal rotations. Hip flexion, abduction, and adduction ROM were measured in the supine position. Hip external and

internal rotation ROM were measured in the prone position. The hip extension ROM was measured in the supine position with the hip joints positioned at the edge of the treatment table and the contralateral hip was flexed to flatten the lumbar spine and stabilize the pelvis. A stabilization belt was applied across the pelvis and contralateral thigh during the ROM tests. The end of ROM was defined as the point at which the examiner felt a firm end feeling at which patient pain restricted further movement. The intrarater reliabilities [ICC (1,1)] for the ROM tests were 0.82 to 0.99. Hip muscle strength was measured in flexion, extension, abduction, and external and internal rotations. The patient's position for each muscle test was as follows: hip flexion, sitting on a treatment table with 90° of hip and knee flexions; hip extension, supine with 20° of hip flexion and 0° of knee flexion; abduction, supine with both hips in a neutral position; and hip rotations, prone with 90° of knee flexion. The pelvis or contralateral thigh was stabilized with a stabilization belt as appropriate. For each strength test, all patients performed two maximal trials for 3 s after few practice trials. The mean of the two trials was used for analysis. Each strength value and lever arm were converted into a ratio of torque to body weight (Nm/kg). The intrarater reliability [ICC (1,1)] for the muscle strength tests was 0.85 to 0.98.

Spinal alignment and mobility

Sagittal spinal alignment was measured using the Spinal Mouse (Index Ltd., Tokyo, Japan), a computer-aided, non-invasive device. Patients were asked to stand in a relaxed position and place arms along their sides. The device was guided along the midline of the spine, starting at the spinous process of C7 and finishing at S3, after sticking a small sticker on the spinous process of C7 and S3. The parameters measured using the Spinal Mouse were as follows (Fig. 1A): thoracic kyphosis angle (sum of 11 segmental angles from Th1/2 to Th11/12), lumbar lordosis angle (sum of 6 segmental angles from Th12/L1 to L5/S1), sacral inclination angle (angle between straight line from S1 to S3 and vertical line), and spinal inclination angle (angle between straight line from Th1 to S1 and vertical line). Spinal alignment was measured thrice in a row, and the mean value was used for analysis.

Spinal mobility was also measured using the Spinal Mouse. Patients were asked to sit on the

chair without back and to bend the spine forward and backward as much as possible. Measurements were carried out in a sitting position because maintaining spinal bending posture in a standing position is difficult for patients with hip OA. The pelvis was not fixed to avoid disturbance of natural movement. ROM was calculated in thoracolumbar spine (sum of 17 segmental angle changes from Th1/2 to L5/S1) (Fig. 1B). The mean value of the 3 measurements was used for analysis. The high reliability and validity of the Spinal Mouse were demonstrated in measurements of spinal alignment and ROM[17,19,31]. In this study, the intrarater reliabilities [ICC (1,1)] for spinal alignment measurements were 0.86 to 0.99, and the spinal mobility measurement was 0.95. The MDC_{95} of the spinal alignment measurements and the spinal mobility measurement was as follows: thoracic kyphosis, 3.06°; lumbar lordosis, 4.91°; sacral inclination, 5.71°; spinal inclination, 1.79°; and spinal mobility, 6.90°.

(Fig. 1)

Principal component analysis

With regard to the morphological parameters in radiography, hip ROM, and hip muscle strength, multiple variables were recorded because a single variable, which is associated with hip OA progression, remains unclear. The number of variables in each category was then reduced while retaining most of the variation in a coherent dataset by using principal component analysis. Principal component (PC) accounting for less than 80% of cumulative contribution ratio with the eigenvalue being <1.0 were retained for logistic regression analysis as factors. Variables in hip morphology and variables in hip muscle strength were respectively combined into one PC while variables in hip ROM were combined into two PCs. The contribution ratio was as follows: 75.8%, PC1 of hip morphology; 40.2%, PC1 of hip ROM; 22.4%, PC2 of hip ROM; 75.7%, PC1 of hip muscle strength (Table 1).

(Table 1)

Statistical analysis

Univariable and multivariable regression analyses with radiographic progression (yes/no) as a dependent variable were performed to identify the independent predictors of radiographic progression of hip OA. Multivariable logistic regression analyses were performed with the following independent variables: pain, HHS, PC1 of hip morphology, PC1 and PC2 of hip ROM, PC1 of hip muscle strength, spinal alignment, and spinal mobility. In addition, age, body mass index (BMI), and minimum JSW at baseline can be regarded as potential confounders[7,33–35] and were included in the multivariable analysis. Hip muscle strength was adjusted for age and minimum JSW because the values of muscle strength were already normalized by body weight. Variables correlated at absolute coefficients >0.7 were defined as multicollinearity[36]. A *P* value < 0.05 was considered statistically significant. SPSS version 24.0 (IBM Japan Ltd., Tokyo, Japan) was used for statistical analysis.

RESULTS

Twenty-one (42.0%) of fifty patients demonstrated radiographic progression of hip OA. Baseline parameters of the patients are presented in Tables 2 and 3. The distribution of the minimum JSW at baseline in all patients was as follows: 11 (22.0%) in the apex, 6 (12.0%) in the lateral side, 17 (34.0%) in the medial side, and 16 (32.0%) in the other area. In the progression group, the change in JSW was 1.3 ± 0.8 mm, and the reduction of >0.5 mm in JSW was found in 5 (23.8%) in the apex, 7 (33.3%) in the lateral side, 8 (38.1%) in the medial side, and 1 (4.8%) in other area.

In the univariable logistic regression analyses (Table 4), larger anterior inclination of the spine in standing position [OR (95% confidence interval): 1.36 (1.05–1.76)] and less spinal mobility [(0.96 (0.92–0.99)] at baseline were statistically significantly associated with radiographic progression of hip OA (Fig. 2). Multivariable logistic regression analyses with adjustment for age, BMI, and minimum JSW at baseline revealed that larger anterior inclination of the spine was statistically significantly associated with hip OA progression [1.37 (1.04–1.80); Table 4]. Less spinal mobility was also statistically significantly associated with hip OA progression [0.96 (0.92–0.99), Table 4]. No multicollinearity was found between variables, and no outlier defined as its residual

outside 3 standard deviations was found. Although only 21 patients were included in the progression group, even the final multivariable model (i.e., four independent variables) fulfilled the rule of a minimum of 5 events per variable[37].

(Table 2)

(Table 3)

(Table 4)

(Fig. 2)

DISCUSSION

This prospective cohort study revealed that larger anterior inclination of the spine and less spinal mobility rather than hip impairments were identified as predictors for the radiographic progression of hip OA over 12 months independent of age, BMI, and minimum JSW. This study is the first to suggest the association between functional decline of the spine, which is measurable and modifiable in clinical practice, and radiographic progression of hip OA.

In the sagittal balance of the spine in standing position, plumb line dropped from the center of C7 is generally located at the posterior superior corner of the 1st sacral vertebra in healthy individuals[38]. In individuals of the same age as the patients in the current study, gravity line is approximately located at the center of the hip joint in the sagittal plane in standing posture[39]. Given these findings, moment arm between gravity force and center of hip joint appears negligible in the sagittal plane in standing healthy individuals. This indicates that the internal hip joint moment generated by hip muscles and ligaments can be made small to maintain the sagittal balance. Thus, the mechanical load on the hip joint in the sagittal plane would be suppressed to be small for standard posture. The anterior inclination of the spine could increase the internal hip extension moment by the relative forward displacement of the upper body's center of mass with regard to the hip joint, consequently resulting in an increase in the mechanical load on the hip joint. Spinal inclination can also modify hip joint moment during gait. Healthy subjects with natural anterior inclination of the spine averaging 2.9° show increased hip moment for a longer time throughout the stance phase of

gait[40]. The anterior inclination of the spine is also a significant factor associated with poor physical activities in patients with hip OA[8].

The hip joint loading in the patients with hip OA is not necessarily larger than that in healthy individuals. Because the mean value of anterior inclination of the spine in healthy individuals, including the middle aged and elderly, ranged from 0.97° to 4.6°[41,42], the anterior spinal inclination of 2.8° in the progression group was not necessarily an abnormal displacement compared with the healthy individuals. It might be due to patients with mild-to-moderate hip OA because those with severe hip OA have larger forwardly inclined spine than that in healthy individuals[8]. Considering the report that hip contact force during gait is rather lower for patients with hip OA than the healthy subjects[43], only the magnitude of loading may not be an aggravating factor related to hip OA progression. In this study, we recruited patients with secondary hip OA; that is, most patients in this study have hip dysplasia. Patients with hip dysplasia generally have smaller cartilage contact area than those with normal joint, and in some cases, hip contact pressure is higher in patients with hip dysplasia than in healthy individuals[44]. Furthermore, a slight change in continuous and repetitive loading during standing and walking due to spinal inclination could damage the osteoarthritic cartilage because the cartilage quality is changed to lower static and dynamic compressive moduli even in the early pre-osteoarthritic stage[45]. Consequently, a slight inclination of the spine may facilitate the radiographic progression of hip OA. However, determining a clear cutoff value for the spinal inclination in the patients with hip OA is necessary in the future.

Moreover, less spinal mobility was a statistically significant predictor for hip OA progression. The mobility of the thoracolumbar spine of the patients with hip OA tended to be less than that of healthy individuals of similar age (approximately 105°; sum of thoracic and lumbar spinal mobility in standing) [17]. Hip motion is accompanied by spinal motion in several activities of daily living[10,11]. Their coordinated motion can avoid regional concentration of mechanical load. The mechanical load of the one may be increased if the mobility of the other is decreased. It was revealed that the contribution of the hip motion relative to that of the lumbar spine motion was increased during sit-to-stand and stand-to-sit for subjects with LBP[11]. Moreover, during stand-to-sit, patients with stiff spine due to degenerative disc disease experience less spinal flexion and more hip flexion, which consequently potentially increasing the risk of impingement of the acetabular rim on the

proximal femur[13]. Therefore, less spinal mobility can be a risk factor for hip OA progression through the potential increase of mechanical load on the hip.

Evaluation and treatment for the standing spinal alignment and spinal mobility are generally included in clinical practice and randomized controlled trial for patients with hip OA[16,46] as the hip and spine are closely linked anatomically and functionally. Spinal alignment and spinal mobility can be measured quantitatively by non-invasive devices, such as the Spinal Mouse, with high reliability and validity[19,32]. The finding of this study has clinical significance in identifying predictors for the radiographic progression of hip OA from among the parameters that are clinically measurable and modifiable.

ROM and muscle strength of the hip at baseline tended to be wholly lower in the progression group than those of the no progression group. However, partially contrary to our hypothesis, no statistically significant association was found between hip impairments and hip OA progression. The values of ROM and muscle strength of the hip in this study coincided approximately with previous studies involving patients with mild-to-moderate hip OA, but the values were lower than the healthy individuals[47,48] and larger than the patients with end-stage hip OA[48,49]. Therefore, the hip impairments of patients with mild-to-moderate hip OA in this study may not be that progressive to raise critical problem in the mechanical environment of the hip. In a different perspective, pain and hip impairments can be improved by therapeutic exercise[50], but little evidence is available for the preventive effect of conservative treatment in radiographic progression of hip OA. Radiographic progression of hip OA is suggested to be more independent of the change of the hip impairments and pain. Further studies were warranted to investigate the association of hip impairments and pain with radiographic progression of hip OA.

This study has several limitations. The segmental alignment and mobility responsible for the whole spine were unclear because we did not use radiography or other imaging methods. Spinal inclination and mobility in the frontal and transversal planes were not measured despite the fact that lateral spinal inclination could also change hip loading. In addition, the anterior inclination of the spine in the progression group was small, but it was detectable with easy-to-use instruments, such as Spinal Mouse. These instruments are useful for assessing spinal alignment and mobility in the clinical setting. However, for detailed clinical assessment of the spine, such as segmental alignment,

disc degeneration, and arthrosis, a low-dose X-ray imaging system may be suitable[13]. Despite the fact that isokinetic muscle strength and total leg extensor power are also lower in patients with hip OA[51,52], muscle strength was only assessed in isometric contraction, with emphasis on easily measurable parameters in common clinical practice. Therefore, the association between hip muscle function and hip OA progression could not necessarily be declared. The potential limitation in the generalizability of the findings in this study should also be acknowledged because our sample was composed of female patients with secondary hip OA. The slight change in the alignment and mobility of the spine found in this study may not create an adverse result in patients with primary hip OA who have no morphological abnormality. We estimated the odds ratio despite the possibility of underestimation or overestimation of the relative risk, when the event being modeled is not rare (>10%)[53]. Therefore, there may be the discrepancy between the odds ratio and relative risk in this study. Although the sample size required for multivariable analysis was satisfied, this study was an exploratory study with a small sample size. Furthermore, the 12-month follow-up duration was short though the yearly mean narrowing of the hip JSW has been reported as a risk factor for hastening THA[54]. A longer follow-up period with a larger sample size could establish stronger relationship between spine and/or hip impairments and hip OA progression.

In conclusion, it was suggested that larger anterior inclination of the spine and less spinal mobility at baseline were associated with radiographic progression of hip OA defined by a cartilage thickness loss >0.5 mm in 12 months rather than hip impairments (i.e., lower ROM, muscle strength, and pain). Spinal alignment and mobility should be considered when classifying patients with higher risk of hip OA progression and designing treatment programs to slow hip OA progression.

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Author Contributions

HT: concept and design, obtaining of funding, analysis and interpretation of the data, drafting of the article, and final approval of the article. HT was the main investigator of this study, and performed all of the measurements.

HA: provision of study patients, analysis and interpretation of data, critical revision of the article for important intellectual content, and final approval of the article.

KG: provision of study patients, analysis and interpretation of data, critical revision of the article for important intellectual content, and final approval of the article.

KS: provision of study patients, analysis and interpretation of data, critical revision of the article for important intellectual content, and final approval of the article.

YK: provision of study patients, analysis and interpretation of data, critical revision of the article for important intellectual content, and final approval of the article.

NI: concept and design, obtaining of funding, analysis and interpretation of data, critical revision of the article for important intellectual content, and final approval of the article.

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Competing interest

There are no conflicts of interest to declare with regard to this study.

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Table 1. Principal component analysis for morphology, ROM, and muscle strength of hip (n = 50)

	PC1 (factor loading)	PC2 (factor loading)	Contribution ratio
Hip morphology			
Sharp angle, degrees	0.97	–	PC1: 75.8%
CE angle, degrees	0.92	–	
AHI	−0.87	–	
ARO, degrees	−0.71	–	
Hip ROM, degrees			
Flexion	0.59	−0.12	PC1: 40.2% PC2: 22.4%
Extension	0.63	0.36	
Abduction	0.74	0.17	
Adduction	0.77	−0.11	
Internal rotation	0.68	−0.64	
External rotation	0.28	0.87	
Hip muscle strength, Nm			
Flexion	0.85	–	PC1: 75.7%
Extension	0.90	–	
Abduction	0.85	–	
External rotation	0.85	–	
Internal rotation	0.90	–	

(Footnotes for Table 1)

Abbreviations: PC, principal component; ROM, range of motion; CE angle, center edge angle; AHI, acetabular head index; ARO, acetabular roof obliquity.

Contribution ratio: the fraction of the explained variance of extraction sums of squared loadings.

Table 2. Baseline demographic and hip-related parameters of study participants

	All patients* (n = 50)	No progression* (n = 29)	Progression* (n = 21)
Age, years	47.4 ± 10.7	46.6 ± 10.2	48.6 ± 11.6
Weight, kg	55.2 ± 10.2	54.2 ± 9.8	56.5 ± 10.9
Height, cm	156.9 ± 5.6	157.5 ± 6.8	156.1 ± 3.5
Body mass index	22.4 ± 4.1	21.8 ± 3.8	23.2 ± 4.4
Minimum JSW, mm	3.3 ± 1.4	3.7 ± 1.4	2.9 ± 1.4
Hip OA stage, n (%)			
Preosteoarthritis	14 (28.0%)	9 (31.0%)	5 (23.8%)
Early stage	24 (48.0%)	15 (51.7%)	9 (42.9%)
Advanced stage	12 (24.0%)	5 (17.2%)	7 (33.3%)
Pain (VAS), mm	42.0 ± 27.5	37.7 ± 1.4	47.9 ± 26.4
Harris hip score (total 100 points)	86.9 ± 9.9	87.9 ± 8.7	85.6 ± 11.4
Hip morphology			
Sharp angle, degrees	45.0 ± 6.5	45.6 ± 7.4	44.1 ± 4.8
CE angle, degrees	23.4 ± 11.5	22.0 ± 11.1	25.5 ± 12.1
AHI	73.8 ± 11.0	72.8 ± 10.7	75.2 ± 11.6
ARO, degrees	22.4 ± 7.9	22.8 ± 8.6	21.8 ± 7.0
Hip ROM, degrees			
Flexion	112.1 ± 14.7	113.6 ± 15.3	110.0 ± 14.0
Extension	11.6 ± 3.5	11.6 ± 3.2	11.5 ± 3.9
Abduction	22.9 ± 5.9	23.8 ± 6.1	21.7 ± 5.6
Adduction	15.4 ± 3.7	16.0 ± 3.6	14.7 ± 3.9
External rotation	24.0 ± 11.9	23.2 ± 11.8	25.1 ± 12.2
Internal rotation	41.2 ± 14.4	42.8 ± 16.0	39.0 ± 11.7
Hip muscle strength, Nm/kg			
Flexion	0.87 ± 0.25	0.91 ± 0.29	0.81 ± 0.19
Extension	1.46 ± 0.57	1.55 ± 0.66	1.34 ± 0.42
Abduction	0.75 ± 0.21	0.77 ± 0.20	0.73 ± 0.23
External rotation	0.36 ± 0.12	0.37 ± 0.13	0.35 ± 0.11
Internal rotation	0.32 ± 0.12	0.35 ± 0.14	0.29 ± 0.08

(Footnotes for Table 2)

Abbreviations: VAS, visual analogue scale; JSW, joint space width; CE angle, center edge angle; AHI, acetabular head index; ARO, acetabular roof obliquity; ROM, range of motion. * Values are mean ± standard deviation.

Table 3. Baseline spine-related parameters of study participants

	All patients* (n = 50)	No progression* (n = 29)	Progression* (n = 21)
Spinal posture, degrees			
Thoracic kyphosis	42.6 ± 9.6	42.2 ± 9.9	43.1 ± 9.4
Lumbar lordosis	30.4 ± 10.6	30.4 ± 10.7	30.4 ± 10.7
Sacral inclination (+; anterior)	14.7 ± 7.1	14.1 ± 6.8	15.6 ± 7.5
Spinal inclination (+; anterior)	1.7 ± 2.6	0.9 ± 2.3	2.8 ± 2.7
Spinal mobility, degrees			
Thoracolumbar spine	78.7 ± 17.7	83.6 ± 19.2	71.9 ± 12.8

(Footnotes for Table 3)

* Values are mean ± standard deviation.

Table 4. Univariable and multivariable logistic regression predicting the progression of hip osteoarthritis (n = 50)

	Crude OR (95% CI)	*P* value	Adjusted OR (95% CI)*	*P* value
Age, years	1.02 (0.97 to 1.08)	0.499	–	–
Body mass index	1.09 (0.94 to 1.25)	0.261	–	–
Minimum JSW, mm	0.68 (0.45 to 1.03)	0.066	–	–
Hip OA stage	1.58 (0.71 to 3.51)	0.263	–	–
Pain (VAS), mm	1.01 (0.99 to 1.04)	0.198	1.01 (0.98 to 1.03)	0.619
Harris hip score	0.97 (0.92 to 1.04)	0.426	0.99 (0.93 to 1.06)	0.837
Hip morphology				
PC1†	1.28 (0.71 to 2.29)	0.408	1.28 (0.69 to 2.35)	0.443
Hip ROM, degrees				
PC1‡	0.70 (0.39 to 1.25)	0.225	0.91 (0.41 to 1.99)	0.806
PC2‡‡	1.26 (0.71 to 2.26)	0.432	1.14 (0.61 to 2.14)	0.689
Hip muscle strength, Nm				
PC1§	0.70 (0.38 to 1.28)	0.245	0.85 (0.43 to 1.68)	0.636
Spinal posture, degrees				
Thoracic kyphosis (+; kyphosis)	1.01 (0.95 to 1.07)	0.741	1.01 (0.94 to 1.08)	0.807
Lumbar lordosis (+; lordosis)	1.00 (0.95 to 1.06)	0.995	1.00 (0.94 to 1.06)	0.895
Sacral inclination (+; anterior)	1.03 (0.95 to 1.12)	0.468	1.02 (0.94 to 1.11)	0.628
Spinal inclination (+; anterior)	1.36 (1.05 to 1.76)	0.020	1.37 (1.04 to 1.80)	0.028
Spinal mobility, degrees				
Thoracolumbar spine	0.96 (0.92 to 0.99)	0.029	0.96 (0.92 to 0.99)	0.037

(Footnotes for Table 4)

Abbreviations: OR, odds ratio; 95% CI, 95% confidence interval; VAS, visual analogue scale; JSW, joint space width; ROM, range of motion.

† PC1 of morphological parameters.

‡ PC1 of hip ROM.

‡‡ PC2 of hip ROM.

§ PC1 of hip muscle strength.

* Adjusted for age, BMI, and minimum JSW at baseline.

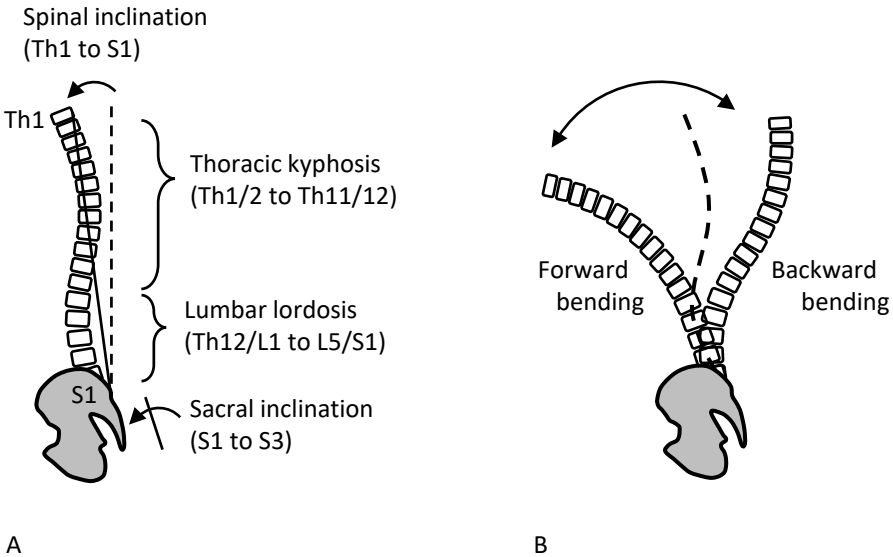
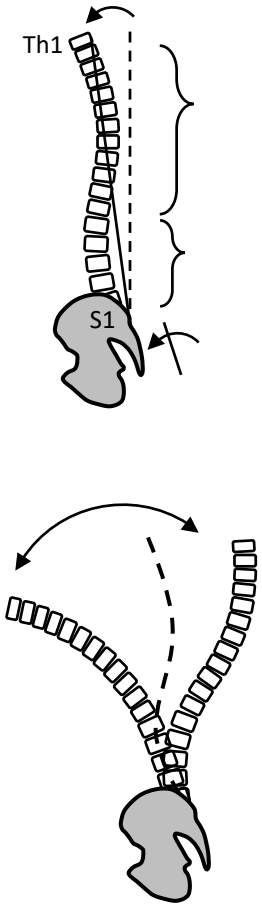

Spinal inclination
(Th1 to S1)
Th1
Thoracic kyphosis
(Th1/2 to Th11/12)
Lumbar lordosis
(Th12/L1 to L5/S1)
S1
Sacral inclination
(S1 to S3)
Forward
bending
Backward
bending
A
B

Fig. 1.


Th1
S1

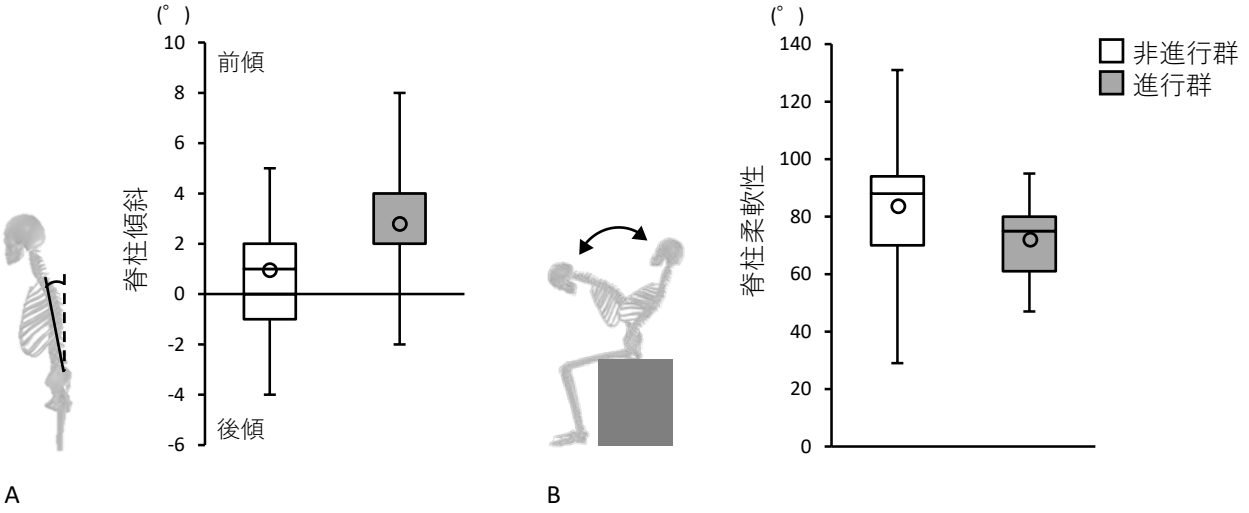

(°)
10
8
6
4
2
0
-2
-4
-6
前傾
後傾
脊柱傾斜
(°)
140
120
100
80
60
40
20
0
脊柱柔軟性
非進行群
進行群
A
B

Fig. 2.