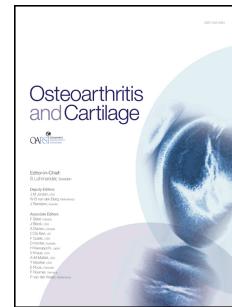


# Accepted Manuscript

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

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

3

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17

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25

26 **Running title:** Spinal impairment and hip OA progression

27

28

29

30

31

32 **ABSTRACT**

33

34 **Objective:**

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

37 **Design:**

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

46 **Results:**

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

53 **Conclusions:**

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

56

57

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

59

60

61 **INTRODUCTION**

62

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

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

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

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

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

105

106

## 107 PATIENTS AND METHODS

108

### 109 Patients

110

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

117

118 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

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

132

133 **Radiographic progression of hip OA**

134

135 The radiographic progression of hip OA was assessed with JSW in a digital supine  
136 anteroposterior radiograph of the pelvis obtained in a standardized manner by the same skilled  
137 radiology technicians. A negligible difference was found in radiographic parameters with regard to  
138 hip dysplasia and joint space width between supine and standing anteroposterior radiographs<sup>23,24</sup>.  
139 Therefore, we used radiograph in the supine position to improve image quality<sup>23</sup>. We used  
140 radiographs taken for general practice to avoid unnecessary exposure to radiation. To assess the  
141 change in JSW, the films at baseline and approximately 12 months later were paired by patients but  
142 blinded as to patient and sequence to the reader to avoid bias<sup>25</sup>. All radiographic measurements were  
143 performed by a single experienced examiner. Images were reviewed and measured using Centricity  
144 Enterprise Web, version 3.0 (GE Health care, Buckinghamshire, England). The JSW was measured in  
145 0.1 mm increments at three locations, namely, lateral margin of the subchondral sclerotic line, apical  
146 transection of the weight-bearing surface by a vertical line through the center of the femoral head,  
147 and medial margin of the weight-bearing surface bordering on the fovea. If the minimum JSW was

148 found aside from the three locations in the weight-bearing area, the JSW of the narrowest point was  
149 also recorded as a fourth measurement. Minimum JSW was defined as the smallest of the three or  
150 four measurements<sup>26</sup>. The radiographic progression of hip OA was defined as a reduction of >0.5 mm  
151 in JSW at any of the three or four locations<sup>27,28</sup>. The intrarater reliability [intraclass correlation (ICC)  
152 1,1] of JSW measurement for 20 randomly selected radiographs was 0.99. The MDC<sub>95</sub> (MDC at the  
153 95% confidence level) of the JSW in the current study was 0.39 mm.

154

155 **Morphological assessment of hip joint**

156

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

161

162 **Pain and Harris hip score**

163

164 The hip pain intensity and functional status of the patients were assessed at baseline using a  
165 100-mm visual analogue scale and Harris hip score. The hip pain intensity was questioned as the  
166 average hip pain during daily life in the last 3 months.

167

168 **Hip ROM and muscle strength**

169

170 A single examiner measured passive hip ROM and the maximal isometric hip muscle  
171 strength using a standard goniometer (Sakai medical Co., Ltd, Tokyo, Japan) and a handheld  
172 dynamometer ( $\mu$ TAS F-1; Anima Co., Ltd, Tokyo, Japan) at baseline in accordance with previous  
173 studies<sup>18,29,30</sup>.

174 Details of ROM and muscle strength tests were described elsewhere<sup>30</sup>. Briefly, hip ROM  
175 was measured in flexion, extension, abduction, adduction, and external and internal rotations. Hip  
176 flexion, abduction, and adduction ROM were measured in the supine position. Hip external and

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

192

### 193 **Spinal alignment and mobility**

194

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

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

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

217

218 (Fig. 1)

219

220 **Principal component analysis**

221

222 With regard to the morphological parameters in radiography, hip ROM, and hip muscle  
 223 strength, multiple variables were recorded because a single variable, which is associated with hip OA  
 224 progression, remains unclear. The number of variables in each category was then reduced while  
 225 retaining most of the variation in a coherent dataset by using principal component analysis. Principal  
 226 component (PC) accounting for less than 80% of cumulative contribution ratio with the eigenvalue  
 227 being <1.0 were retained for logistic regression analysis as factors. Variables in hip morphology and  
 228 variables in hip muscle strength were respectively combined into one PC while variables in hip ROM  
 229 were combined into two PCs. The contribution ratio was as follows: 75.8%, PC1 of hip morphology;  
 230 40.2%, PC1 of hip ROM; 22.4%, PC2 of hip ROM; 75.7%, PC1 of hip muscle strength (Table 1).

231

232 (Table 1)

233

234 **Statistical analysis**

235

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

246

247

## 248 RESULTS

249

250 Twenty-one (42.0%) of fifty patients demonstrated radiographic progression of hip OA.  
251 Baseline parameters of the patients are presented in Tables 2 and 3. The distribution of the minimum  
252 JSW at baseline in all patients was as follows: 11 (22.0%) in the apex, 6 (12.0%) in the lateral side,  
253 17 (34.0%) in the medial side, and 16 (32.0%) in the other area. In the progression group, the change  
254 in JSW was  $1.3 \pm 0.8$  mm, and the reduction of >0.5 mm in JSW was found in 5 (23.8%) in the apex,  
255 7 (33.3%) in the lateral side, 8 (38.1%) in the medial side, and 1 (4.8%) in other area.

256 In the univariable logistic regression analyses (Table 4), larger anterior inclination of the  
257 spine in standing position [OR (95% confidence interval): 1.36 (1.05–1.76)] and less spinal mobility  
258 [(0.96 (0.92–0.99)] at baseline were statistically significantly associated with radiographic  
259 progression of hip OA (Fig. 2). Multivariable logistic regression analyses with adjustment for age,  
260 BMI, and minimum JSW at baseline revealed that larger anterior inclination of the spine was  
261 statistically significantly associated with hip OA progression [1.37 (1.04–1.80); Table 4]. Less spinal  
262 mobility was also statistically significantly associated with hip OA progression [0.96 (0.92–0.99),  
263 Table 4]. No multicollinearity was found between variables, and no outlier defined as its residual

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

267

268 (Table 2)

269 (Table 3)

270 (Table 4)

271 (Fig. 2)

272

## 273 DISCUSSION

274

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

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

293 gait<sup>40</sup>. The anterior inclination of the spine is also a significant factor associated with poor physical  
294 activities in patients with hip OA<sup>8</sup>.

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

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

322 proximal femur<sup>13</sup>. Therefore, less spinal mobility can be a risk factor for hip OA progression through  
 323 the potential increase of mechanical load on the hip.

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

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

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

351 disc degeneration, and arthrosis, a low-dose X-ray imaging system may be suitable<sup>13</sup>. Despite the fact  
 352 that isokinetic muscle strength and total leg extensor power are also lower in patients with hip  
 353 OA<sup>51,52</sup>, muscle strength was only assessed in isometric contraction, with emphasis on easily  
 354 measurable parameters in common clinical practice. Therefore, the association between hip muscle  
 355 function and hip OA progression could not necessarily be declared. The potential limitation in the  
 356 generalizability of the findings in this study should also be acknowledged because our sample was  
 357 composed of female patients with secondary hip OA. The slight change in the alignment and  
 358 mobility of the spine found in this study may not create an adverse result in patients with primary hip  
 359 OA who have no morphological abnormality. We estimated the odds ratio despite the possibility of  
 360 underestimation or overestimation of the relative risk, when the event being modeled is not rare  
 361 (>10%)<sup>53</sup>. Therefore, there may be the discrepancy between the odds ratio and relative risk in this  
 362 study. Although the sample size required for multivariable analysis was satisfied, this study was an  
 363 exploratory study with a small sample size. Furthermore, the 12-month follow-up duration was short  
 364 though the yearly mean narrowing of the hip JSW has been reported as a risk factor for hastening  
 365 THA<sup>54</sup>. A longer follow-up period with a larger sample size could establish stronger relationship  
 366 between spine and/or hip impairments and hip OA progression.

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

372

373

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375

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 378 analyses.

379

380 **Author Contributions**

381

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

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

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401 **Competing interest**

402

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

404

405

406 **References**

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1   **Figure legends**

2

3   **Fig. 1.** Angle of the thoracic kyphosis, lumbar lordosis, sacral inclination, and spinal inclination  
4   were measured as spinal posture (A). Range of motion of the thoracolumbar spine in the sagittal  
5   plane was measured as spinal mobility (B).

6

7

8   **Fig. 2.** Distribution of anterior inclination of the spine in standing position (A) and spinal mobility  
9   (B) in each of the no progression (white) and progression groups (gray). Boxplots with upper and  
10   lower bars showing maximum and minimum values. Upper, middle, and lower lines in the box  
11   show 75th, 50th (median), and 25th centiles, respectively. The circle in the box shows the mean  
12   value. When adjusted for age, BMI, and minimum JSW at baseline, larger anterior inclination of  
13   the spine ( $P = 0.028$ ) and less spinal mobility ( $P = 0.037$ ) were statistically significantly associated  
14   with hip OA progression.

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

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

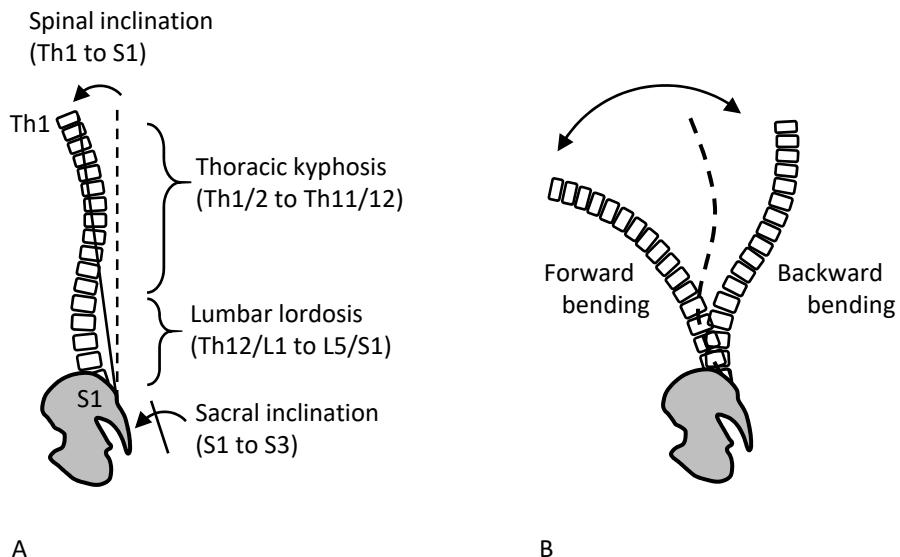
† PC1 of morphological parameters.

‡ PC1 of hip ROM.

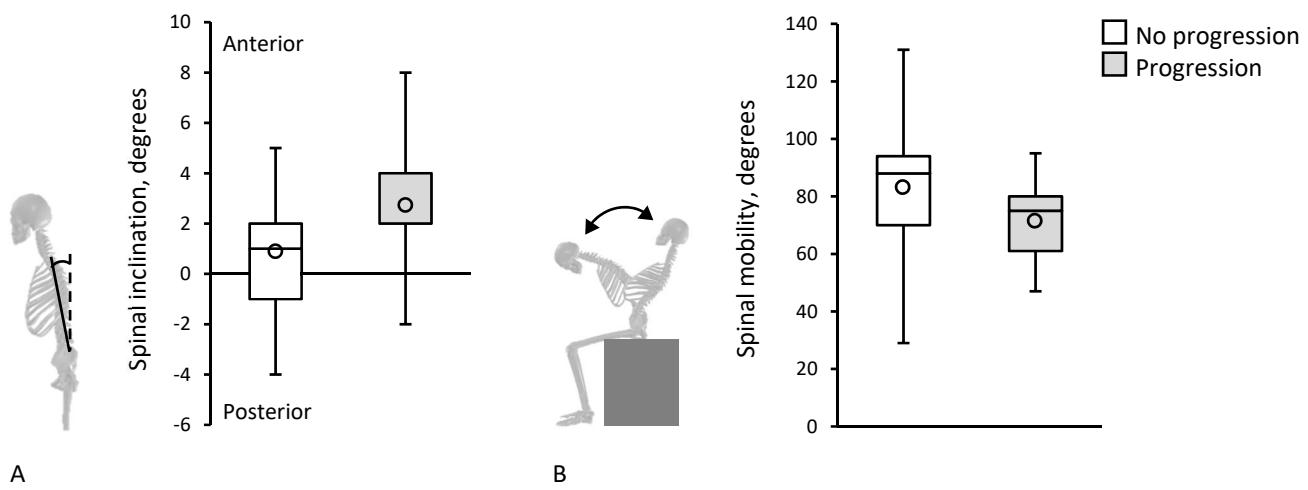
‡‡ PC2 of hip ROM.

§ PC1 of hip muscle strength.

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



**Fig. 1.**



**Fig. 2.**