

## Clinical Study

Pelvic fixation and spinopelvic realignment accelerate hip osteoarthritis following long-segment fusion: a 5-year retrospective study<sup>1</sup>

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## Abstract

**BACKGROUND CONTEXT:** Spinopelvic fixation is commonly used to reduce the risk of distal adjacent segment disease in adult spinal deformity surgery. However, it may impose increased stress on the hip joint, potentially leading to hip osteoarthritis (OA). The concept of adjacent joint disease has been proposed to describe hip joint degeneration due to compensatory overload following rigid spinopelvic fixation, but longitudinal data supporting this are lacking.

**PURPOSE:** To investigate the prevalence and risk factors for the progression of hip OA after spinal fusion surgery, and to evaluate adjacent joint disease as a distinct pathological entity.

**STUDY DESIGN/SETTING:** Retrospective single-center cohort study with 5-year follow-up.

**PATIENT SAMPLE:** A total of 290 patients (580 hips) who underwent spinal fusion surgery between 2011 and 2018, and met inclusion/exclusion criteria.

**OUTCOME MEASURES:** Radiographic hip OA progression was defined by an increase in Kellgren–Lawrence (KL) grade at 5 years postoperatively. Spinopelvic parameters and hip morphology were also assessed.

**METHODS:** Radiographs were evaluated preoperatively, and at 1 month and 5 years postoperatively. Patients were divided based on the presence or absence of OA progression. Logistic regression was used to identify independent risk factors. A post hoc exploratory subgroup analysis was performed in patients with long fusion ( $\geq 6$  levels) and baseline KL grade 0–1.

**RESULTS:** In the full cohort, OA progression was observed in 13.8%, and new-onset OA in 10.8%. Risk factors for progression in the overall cohort included female sex (odds ratio (OR): 3.95,  $p=.0048$ ), pelvic incidence (PI) (OR: 1.04,  $p=.0012$ ), sacral slope (SS) correction (OR: 1.04,  $p=.018$ ), pelvic fixation (OR: 5.04,  $p=.0016$ ), and baseline KL grade (OR: 2.49,  $p=.00001$ ). In the long fusion subgroup ( $n = 122$ ; 244 hips), new-onset OA was observed in 21.7%. Within this subgroup, pelvic fixation (OR: 8.48,  $p=.0069$ ), larger SS correction (OR: 1.04,  $p=.027$ ), and higher PI (OR: 1.05,  $p=.0017$ ) remained significant predictors of hip OA.

FDA device/drug status: Not applicable.

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**CONCLUSIONS:** Spinopelvic fixation was associated with an increased risk of hip OA progression, especially in female patients and those with high PI or large SS correction. These findings support adjacent joint disease as a clinically relevant entity and suggest the need for surgical caution and postoperative strategies to protect the hip joint in vulnerable individuals. © 2026 Elsevier Inc. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

**Keywords:**

Adjacent joint disease; Adult spinal deformity; Hip osteoarthritis; Pelvic fixation; Pelvic incidence; Sacral slope

## Introduction

Spinal corrective fusion surgery for adult spinal deformities is performed at an increasing rate because of the aging population. Although the surgical procedure used to treat adult spinal deformity has improved severe lower back pain and quality of life, high rates of perioperative complications and the need for reoperation remain. A major complication of spinal fusion surgery is adjacent segment disease, which induces functional and neurogenic disorders [1].

To reduce the risk of distal segment disease, surgeons often include pelvic fixation during thoracolumbar fusion surgery [2]. However, recent studies have raised concerns that spinopelvic fixation may lead to the progression of hip osteoarthritis (OA) [3,4]. Proposed risk factors for this complication include female sex, preexisting hip OA, developmental dysplasia of the hip, and the use of pelvic fixation. Biomechanical studies suggest that pelvic fixation increases stress on the hip joint due to the loss of spinal and sacroiliac mobility, leading to compensatory motion at the hip [5,6]. Past papers previously proposed the concept of "adjacent joint disease" to describe hip degeneration resulting from compensatory overload after spinopelvic fixation surgery. However, no longitudinal studies have quantitatively validated this concept.

Therefore, the aim of this study was to investigate the prevalence of and risk factors for hip OA at 5 years after spinal fusion surgery, and to provide clinical evidence supporting adjacent joint disease as a distinct degenerative pathology associated with spinopelvic instrumentation.

## Methods

This retrospective study enrolled 413 patients who underwent spinal fusion surgery for lumbar lesion between January 2011 and December 2018. Pelvic fixations in this series were all performed with S2 alar-iliac (S2AI) screws, which traverse the sacroiliac joint. All patients provided informed consent before surgery, and the study protocol was approved by the institutional ethics committee of the author's institution. The follow-up period was 60 months after surgery. The exclusion criteria included pyogenic spondylitis, tumor, hemodialysis, Parkinson disease, steroid user, rheumatoid arthritis, achondroplasia, cerebral palsy, other combined spinal lesions at the cervical or thoracic level, and a history of hip surgery (Fig. 1).

## Radiographic assessment

A digital standing anteroposterior whole-spine radiograph including the hip joint was obtained by the same skilled technician using a standard protocol at preoperatively, 1 month and 5 years postoperatively. We defined the time point of 1 month after spinal fusion surgery as the baseline because the procedure changed the pelvis tilt and hip disposition as observed on X-rays. Sex was defined as biological sex (male or female) as recorded in the medical records. Gender identity was not assessed in this study. We measured the center edge (CE) angle [7,8] at 1 month after surgery, and Kellgren-Lawrence (KL) grade [9] at 1 month and 5 years postoperatively using the anteroposterior radiographic images. The CE angle was defined as angle between the perpendicular line of the center of the femoral head and the lateral edge of the acetabulum. The KL grade was rated as Grade 0 (no OA), Grade 1 (doubtful OA), Grade 2 (mild OA), Grade 3 (moderate OA), and Grade 4 (severe OA). This study also measured spinopelvic sagittal alignment parameters at preoperatively, 1 month and 5 years postoperatively. The lumbar lordosis (LL), sacral slope (SS), and pelvic incidence (PI) were also measured on the lateral radiographs. We also evaluated the lower instrumented vertebra (LIV) (lumbar/sacrum/pelvis). The CE angle, LL, SS, PI, and KL grade were independently evaluated by the first author (a board-certified orthopaedic surgeon with more than 10 years of experience) and the second author (an orthopaedic surgeon with 5 years of clinical experience). For intraobserver reliability, each author reevaluated a randomly selected subset of 60 patients after a 4-week interval to minimize recall bias. Weighted kappa statistics (linear weights) were calculated, yielding an interobserver  $\kappa$  of 0.90 and an intraobserver  $\kappa$  of 0.95 for KL grade. The reliability statistics using intraclass correlation coefficient values for CE angle, LL, SS, and PI for the interobserver (intraobserver) reliability were 0.91 (0.94), 0.85 (0.92), 0.86 (0.94), and 0.88 (0.95), respectively.

## Analysis 1

In Analysis 1, patients were divided into 2 groups according to radiographic progression of hip OA from baseline (1 month postoperatively) to 5 years. The Progression group (P1) included any hip that showed an increase of at least one KL grade or required total hip arthroplasty (THA) during follow-up, regardless of the baseline KL grade. This

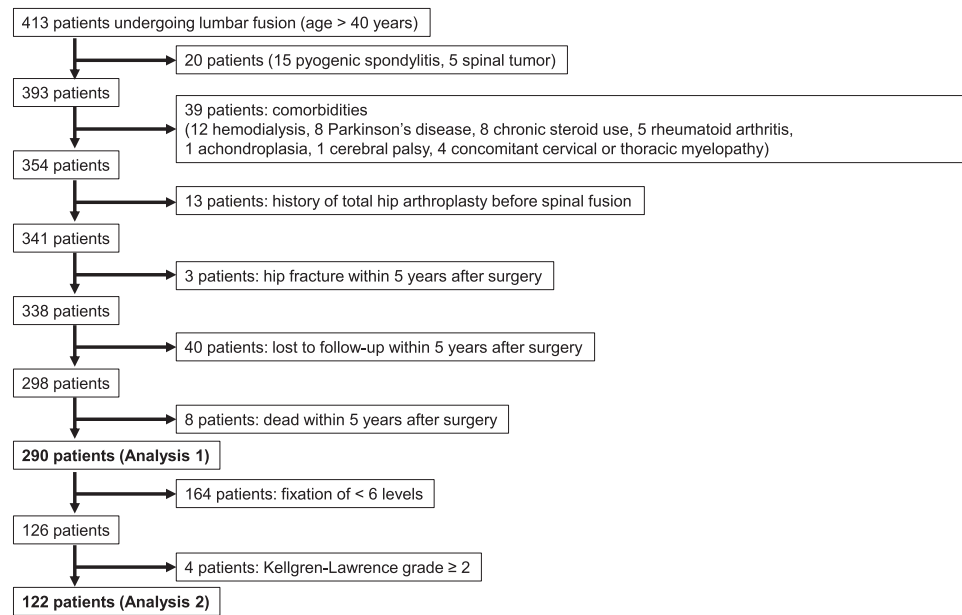


Fig. 1. Flow diagram of patient selection.

definition encompassed both hips with baseline KL 0–1 that progressed to  $\geq 2$ , as well as hips with baseline KL 2–3 that further worsened. The Nonprogression group (N1) consisted of hips that showed no increase in KL grade over the 5-year period. Because KL grades 0 and 1 are often difficult to distinguish radiographically, they were combined into a single baseline category for the purpose of this analysis.

### Analysis 2

In Analysis 2, we conducted an exploratory post hoc analysis limited to patients who underwent long fusion ( $\geq 6$  levels) and had baseline KL grade 0–1 to identify risk factors for *de novo* hip OA within this subgroup [10]. Progression was defined as development of KL grade  $\geq 2$  or having undergone THA at 5 years postoperatively. The patients were divided into 2 groups depending on the progression of hip pathology (Group P2) or not (Group N2) (Fig. 1).

### Statistical analysis

JMP Pro 14.1 (SAS Institute Inc., Cary, NC, USA) was used for all analyses. All numerical data are expressed as mean  $\pm$  standard deviation. A  $p$  value  $< 0.05$  was considered to be significant.

We compared values between the 2 groups using univariate regression analysis. The chi-square test was used to compare categorical data, and Student's  $t$ -test was used to compare continuous parameters. Multiple logistic regression analysis was performed to identify the factors that predicted the progression of KL grade.

## Results

### Analysis 1

The 290 patients included 80 men and 210 women. Their characteristics at the baseline are listed in Table 1. Their

Table 1  
Characteristics of the patients

|                                                               | N = 290 (580 hips)                     |
|---------------------------------------------------------------|----------------------------------------|
| Age (years)                                                   | 66.8 $\pm$ 10.9                        |
| Sex (male/female)                                             | 80/210                                 |
| BMI (kg/m <sup>2</sup> )                                      | 23.3 $\pm$ 3.7                         |
| LL at preoperation (degrees)                                  | 19.9 $\pm$ 22.3                        |
| SS at preoperation (degrees)                                  | 21.5 $\pm$ 13.5                        |
| PI at preoperation (degrees)                                  | 53.2 $\pm$ 12.5                        |
| Number of the fusion segments (segments)                      | 6.4 $\pm$ 4.0                          |
| LIV at lumbar fusion/sacrum fusion/pelvis fixation (patients) | 127/53/110                             |
| KL grade at 1 month after surgery (hips)                      | KL0-1:544, KL2:28, KL3:8               |
| KL grade at 5 years after surgery (hips)                      | KL0-1:480, KL2:68, KL3:16, KL4:7 THA:9 |

mean  $\pm$  standard deviation.

BMI, body mass index; LL, lumbar lordosis; SS, sacral slope; PI, pelvic incidence;.

LIV, lower instrumented vertebra; KL, Kellgren-Lawrence.

Table 2

Comparison between the progression group (Group P1) and nonprogression group (Group N1)

|                                         | Group N1 (500 hips)      | Group P1 (80 hips)           | p value |
|-----------------------------------------|--------------------------|------------------------------|---------|
| Preoperation                            |                          |                              |         |
| Age (years)                             | 67.0 ± 9.8               | 69.6 ± 9.6                   | 0.012   |
| Sex (male/female)                       | 154/346                  | 6/74                         | <0.0001 |
| BMI (kg/m <sup>2</sup> )                | 23.3 ± 3.5               | 23.5 ± 4.4                   | 0.81    |
| LL (degrees)                            | 20.8 ± 22.1              | 14.3 ± 22.8                  | 0.014   |
| SS (degrees)                            | 22.0 ± 13.0              | 18.0 ± 16.4                  | 0.0095  |
| PI (degrees)                            | 52.5 ± 11.3              | 56.3 ± 15.3                  | 0.048   |
| PI-LL (degrees)                         | 30.6 ± 23.9              | 39.5 ± 24.9                  | 0.0008  |
| Postoperation (1 month after surgery)   |                          |                              |         |
| LL (degrees)                            | 31.8 ± 15.7              | 34.9 ± 15.1                  | 0.11    |
| SS (degrees)                            | 25.0 ± 10.4              | 25.6 ± 13.7                  | 0.40    |
| PI-LL (degrees)                         | 19.6 ± 19.1              | 18.5 ± 16.9                  | 0.89    |
| LL correction (degrees)                 | 11.1 ± 22.6              | 21.0 ± 22.1                  | 0.0002  |
| SS correction (degrees)                 | 5.6 ± 8.2                | 10.1 ± 11.1                  | 0.0016  |
| Number of fusion segments (segments)    | 6.2 ± 3.9                | 7.7 ± 4.2                    | 0.0026  |
| LIV=lumbar fusion (yes/no)              | 232/268 (46.4%)          | 22/58 (27.5%)                | 0.035   |
| LIV=sacrum fusion (yes/no)              | 98/402 (19.6%)           | 8/72 (10.0%)                 | 0.028   |
| LIV=pelvis fixation (yes/no)            | 170/330 (34.0%)          | 50/30 (62.5%)                | <0.0001 |
| CE angle (degrees)                      | 26.5 ± 5.2               | 25.1 ± 5.3                   | 0.056   |
| KL grade (grades)                       | KL0-1:480, KL2:19, KL3:1 | KL0-1:64, KL2:9, KL3:7       | <0.0001 |
| Final follow-up (5 years after surgery) |                          |                              |         |
| LL (degrees)                            | 30.2 ± 15.5              | 33.9 ± 14.4                  | 0.43    |
| SS (degrees)                            | 25.4 ± 10.3              | 25.3 ± 14.3                  | 0.92    |
| PI-LL (degrees)                         | 20.2 ± 19.5              | 19.0 ± 19.0                  | 0.72    |
| KL grade (grades)                       | KL0-1:480, KL2:19, KL3:1 | KL2:49, KL3:15, KL4:7, THA:9 | <0.0001 |

mean±standard deviation.

BMI, body mass index; LL, lumbar lordosis; SS, sacral slope; PI, pelvic incidence; LIV, lower instrumented vertebra; CE, center-edge; KL, Kellgren-Lawrence; THA, total hip arthroplasty.

average age was  $66.8 \pm 10.9$  years, and their average body mass index was  $23.3 \pm 3.7$  kg/m<sup>2</sup>. KL grade at 1 month postoperatively was grade 0–1 in 544 hips, Grade 2 in 28 hips, Grade 3 in 8 hips and Grade 4 in none of the hips. KL grade at 5 years postoperatively was grade 0–1 in 480 hips, grade 2 in 68 hips, Grade 3 in 16 hips, grade 4 in 7 hips, and 9 hips underwent THA. Finally, KL grade progression was observed in 80 hips (80/580 hips: 13.8%), and new onset of hip OA, which was defined as KL Grade 0–1 at 1 month postoperatively (baseline), and KL Grade 2–4 or undergoing THA at 5 years postoperatively was observed in 59 hips (59/544 hips: 10.8%).

Group P1 was related to female ( $p<.0001$ ), older ( $p=.012$ ), lower LL ( $p=.014$ ), SS ( $p=.0095$ ), and larger

PI ( $p=.048$ ) before surgery, larger correction of LL ( $p=.0002$ ) and SS ( $p=.0016$ ), larger number of the fusion segments ( $p=.0026$ ), and higher ratio of pelvic fixation as LIV ( $p<.0001$ ). Group P1 was also related to progressed KL grade at 1 month after surgery ( $p<.0001$ ) (Table 2).

The multiple logistic regression analysis showed that the risk factors for the progression of KL grade were female (odds ratio (OR): 3.95, 95% confidence interval (CI): 1.34–11.6,  $p=.0048$ ), PI (OR: 1.04, 95% CI: 1.01–1.06,  $p=.0012$ ), SS correction (OR: 1.04, 95% CI: 1.01–1.07,  $p=.018$ ), pelvic fixation (OR: 5.04, 95% CI: 1.81–14.1,  $p=.0016$ ), and KL grade at 1 month after surgery (OR: 2.49, 95% CI: 1.67–3.70,  $p=.00001$ ) (Table 3).

Table 3

Factors associated with progression of KL grade at 5 years after surgery (multiple logistic regression analysis)

|                                              | Odds ratio | 95% confidence interval | p value |
|----------------------------------------------|------------|-------------------------|---------|
| Age (+1 year)                                | 1.02       | 0.98–1.06               | 0.33    |
| Sex (male/female) (1=female)                 | 3.95       | 1.34–11.6               | 0.0048  |
| PI at preoperation (+1 degree)               | 1.04       | 1.01–1.06               | 0.0012  |
| LL correction (+1 degree)                    | 1.00       | 0.98–1.02               | 0.87    |
| SS correction (+1 degree)                    | 1.04       | 1.01–1.07               | 0.018   |
| Number of fusion segments (+1 segment)       | 0.89       | 0.79–1.03               | 0.11    |
| LIV=pelvis fixation (1= yes)                 | 5.04       | 1.81–14.1               | 0.0016  |
| KL grade at 1 month after surgery (+1 grade) | 2.49       | 1.67–3.70               | 0.00001 |

PI, pelvic incidence; LL, lumbar lordosis; SS, sacral slope; LIV, lower instrumented vertebra; KL, Kellgren-Lawrence.

Table 4

Comparison between the progression group (Group P2) and nonprogression group (Group N2)

|                                               | Group N2 (191 hips) | Group P2 (53 hips)           | p value |
|-----------------------------------------------|---------------------|------------------------------|---------|
| Preoperation                                  |                     |                              |         |
| Age (years)                                   | 68.4 ± 8.4          | 70.3 ± 7.0                   | 0.092   |
| Sex (male/female)                             | 34/157              | 2/51                         | 0.019   |
| BMI (kg/m <sup>2</sup> )                      | 23.1 ± 3.0          | 23.4 ± 5.3                   | 0.46    |
| LL (degrees)                                  | 9.7 ± 21.6          | 4.8 ± 19.2                   | 0.17    |
| SS (degrees)                                  | 16.0 ± 12.8         | 14.5 ± 12.2                  | 0.19    |
| PI (degrees)                                  | 51.6 ± 10.4         | 56.9 ± 14.4                  | 0.013   |
| PI-LL (degrees)                               | 42.0 ± 20.3         | 49.2 ± 20.3                  | 0.016   |
| Postoperation (1 month after surgery)         |                     |                              |         |
| LL (degrees)                                  | 39.4 ± 11.2         | 42.0 ± 9.1                   | 0.22    |
| SS (degrees)                                  | 25.9 ± 7.9          | 27.5 ± 7.0                   | 0.34    |
| PI-LL (degrees)                               | 8.9 ± 10.9          | 8.8 ± 7.7                    | 0.93    |
| LL correction (degrees)                       | 29.8 ± 21.5         | 36.5 ± 19.5                  | 0.054   |
| SS correction (degrees)                       | 10.1 ± 12.5         | 13.6 ± 12.0                  | 0.046   |
| Change of PI (degrees)                        | −3.8 ± 8.8          | −6.0 ± 8.3                   | 0.042   |
| Number of fusion segments (segments)          | 10.4 ± 2.2          | 10.6 ± 2.9                   | 0.66    |
| LIV=sacrum fusion(yes/no)                     | 13/178 (6.8%)       | 1/52 (1.9%)                  | 0.13    |
| LIV=pelvis fixation (yes/no)                  | 158/33 (82.7%)      | 52/1 (98.1%)                 | 0.0008  |
| CE angle (degrees)                            | 26.5 ± 5.3          | 26.1 ± 5.3                   | 0.61    |
| KL grade (grades)                             | KL0-1:191           | KL0-1:53                     | 0.49    |
| Final follow-up (5 years after surgery)       |                     |                              |         |
| LL (degrees)                                  | 34.0 ± 13.0         | 37.8 ± 10.5                  | 0.054   |
| SS (degrees)                                  | 22.9 ± 9.0          | 26.6 ± 9.3                   | 0.018   |
| PI-LL (degrees)                               | 17.9 ± 14.4         | 17.0 ± 16.8                  | 0.88    |
| Change of PI (degrees) from 1month to 5 years | 3.5 ± 8.7           | 5.2 ± 7.0                    | 0.14    |
| KL grade (grades)                             | KL0-1:191           | KL2:33, KL3:14, KL4:4, THA:2 | <0.0001 |

mean±standard deviation.

BMI, body mass index; LL, lumbar lordosis; SS, sacral slope; PI, pelvic incidence; LIV, lower instrumented vertebra; CE, center-edge; KL, Kellgren-Lawrence; THA, total hip arthroplasty.

## Analysis 2

The 122 patients included 18 men and 104 women. The new onset of hip OA (Group P2) was observed in 53 hips (53/244 hips: 21.7%). Group P2 was related to female ( $p=.019$ ), larger PI ( $p=.013$ ), larger PI-LL mismatch ( $p=.016$ ), SS correction ( $p=.046$ ), higher ratio of pelvic fixation as LIV ( $p=.0008$ ), and larger SS at 5 years after surgery ( $p=.018$ ) (Table 4).

To identify independent risk factors for hip OA, we performed a multivariable logistic regression analysis focusing on preoperative or surgically modifiable parameters. As a result, PI (OR: 1.05, 95% CI: 1.02–1.08,  $p=.0017$ ), SS correction (OR: 1.04, 95% CI: 1.01–1.07,  $p=.027$ ), and pelvis fixation (OR: 8.48, 95% CI: 1.08–66.8,  $p=.0069$ ) were identified as significant predictors (Table 5).

## Discussion

This study reported on the prevalence of and risk factors for the progression of hip OA at 5 years after spinal fusion surgery. In a multivariate logistic regression model focused on baseline patient characteristics, female, PI, SS correction, pelvic fixation, and higher postoperative KL grade at 1 month after surgery were identified as independent risk factors (Analysis 1). Analysis 1 of the overall cohort demonstrated that smaller preoperative LL/SS and greater corrections of these parameters, which are characteristics commonly associated with long fusions, were related to hip OA progression. These findings suggest that long-fusion patients represent a particularly high-risk subgroup. Based on this rationale, Analysis 2 was designed to specifically examine the long-fusion cohort to determine the factors

Table 5

Factors associated with progression of KL grade at 5 years after surgery (multiple logistic regression analysis)

|                                   | Odds ratio | 95% confidence interval | p value |
|-----------------------------------|------------|-------------------------|---------|
| Sex (male/female) (1=female)      | 3.59       | 0.81–15.9               | 0.051   |
| PI at preoperation (+1 degree)    | 1.05       | 1.02–1.08               | 0.0017  |
| PI-LL at preoperation (+1 degree) | 1.01       | 0.99–1.02               | 0.64    |
| SS correction (+1 degree)         | 1.04       | 1.01–1.07               | 0.027   |
| LIV=pelvis fixation (1= yes)      | 8.48       | 1.08–66.8               | 0.0069  |

LL, lumbar lordosis; SS, sacral slope; PI, pelvic incidence; LIV, lower instrumented vertebra.



most responsible for hip OA progression, rather than to simply compare short versus long fusion constructs. PI, SS correction, and pelvic fixation remained significant risk factors in long spinal fusion subgroup (Analysis 2).

In Japan, a nationwide cohort study reported 5-year rates of 1.1% in men and 3.0% in women for OA progression, and 2.8% in men and 4.2% in women for new-onset OA [11]. Although this provides a useful reference, such population-based data may not directly reflect the demographics of surgical patients. Therefore, future studies with a matched nonfusion control cohort will be essential to more accurately estimate the incremental risk attributable to spinal fusion. Compared to these rates, our results may suggest that spinal fusion—particularly with pelvic fixation—accelerates hip OA. A novel concept: "adjacent joint disease"—a pathological condition in which adjacent mobile joints, such as the hip joint, degenerate due to compensatory motion following spinal or sacroiliac joint (SIJ) fusion [3,5,6]. As the global number of spinal fusion surgeries continues to rise, spinopelvic fixation is increasingly used to address distal junctional kyphosis and provide stronger distal anchorage [12]. However, rigid pelvic fixation may alter spinopelvic biomechanics and increase hip joint stress, thereby contributing to OA development [5,6]. This mechanism mirrors the pathophysiology of adjacent segment disease, in which fusion-induced stiffness leads to compensatory overload in adjacent mobile segments [13,14]. Furthermore, in cases of larger deformity correction, the distribution of contact stress across the hip joint may change, potentially altering hip biomechanics and thereby contributing to OA progression. It should be noted that all pelvic fixations in our series used S2AI screws, which traverse the sacroiliac joint and substantially restrict its motion. Since the SIJ plays a critical role in transferring spinal loads to the lower extremities [15], its immobilization by pelvic fixation likely increases biomechanical demand on the hip joint.

In the present study, we further identified that a larger SS correction, and higher PI was associated with increased risk of hip OA. A pelvis with a larger PI reportedly transmits greater mechanical forces to the lumbar spine [16–19]. Fixation of the lumbopelvic construct in such individuals may lead to transmission of greater forces toward the hip joint and contributing to hip OA. In addition, a persistently large SS may reflect a fixed anteversion of pelvis, which enhances the likelihood of femoroacetabular impingement (FAI) during hip flexion, in which repetitive impingement leads to labral damage and subsequently causes cartilage degeneration, ultimately resulting in radiographic progression of OA. Supporting this concept, a previous ultrasonographic study showed that patients with spinopelvic fixation had a higher prevalence of intra-articular abnormalities suggestive of FAI, including labral tears, capsular thickening, and cartilage thinning than nonpelvic fixation [20]. These findings align with past paper that pincer-type FAI in patients with high SS resulted in anterior joint space narrowing

under axial load [21]. Likewise, recent paper also discussed that spinopelvic fixation reduced compensatory pelvic and spinal motion, predisposing patients to anterior hip stress and degenerative changes [22]. Collectively, these results support the hypothesis that altered spinopelvic alignment—particularly increased SS and SIJ fusion—creates a biomechanical environment that promotes anterior femoroacetabular conflict and subsequent OA.

Interestingly, although previous studies linked long fusion to hip OA [23–28], we did not identify long fusion as an independent risk factor in our multivariable analysis. However, this result should be interpreted with caution, as our model evaluated the number of fused segments on a per-segment basis (+1 segment), which may not fully capture the increased risk associated with larger differences in construct length (eg, 4 vs 8 segments). This discrepancy may be due to differing inclusion of sacral versus pelvic fixation. Spine-to-sacrum fusion increases SIJ stress [29–31], and recent studies report that preoperative SIJ laxity—a surrogate for loss of mobility postfixation—also correlates with OA risk [22]. In addition to surgical factors, we identified known patient-related risk factors: female sex [32], and higher baseline KL grade [33,34]. These findings reaffirm that patients with anatomical or degenerative hip vulnerabilities are at increased risk. Accordingly, they should be monitored more closely following spinal surgery, particularly when pelvic fixation is involved.

From a clinical perspective, meticulous surgical planning is warranted for patients at high risk of hip OA (Fig. 2). Spinopelvic fixation may be avoided in selected cases, particularly in females, or individuals exhibiting early degenerative changes. Preoperative ultrasonographic assessment may aid in detecting subclinical intra-articular pathology, including FAI [20]. In cases who secure pelvic anchorage is required, such as in severe osteoporosis, postoperative rehabilitation may include strategies to avoid excessive hip flexion—such as deep squatting or low chair transfers—to reduce anterior acetabular stress and mitigate OA progression.

This study has several limitations. First, it was a retrospective case–control design, and most patients in the pelvic fixation group were female patients, who are inherently at higher risk for hip OA. Higher KL grades were also more prevalent in female patients. Second, the 5-year follow-up period may be insufficient to fully assess long-term OA progression. Interestingly, although previous studies linked long fusion to hip OA [23–28], we did not find long fusion to be an independent risk factor. This discrepancy may be due to differing inclusion of sacral versus pelvic fixation. Spine-to-sacrum fusion increases SIJ stress [29–31], and recent studies report that preoperative SIJ laxity—a surrogate for loss of mobility postfixation—also correlates with OA risk [22]. Third, we were unable to evaluate other potential risk factors such as physical activity [34]. Larger studies with extended follow-up are needed to clarify the

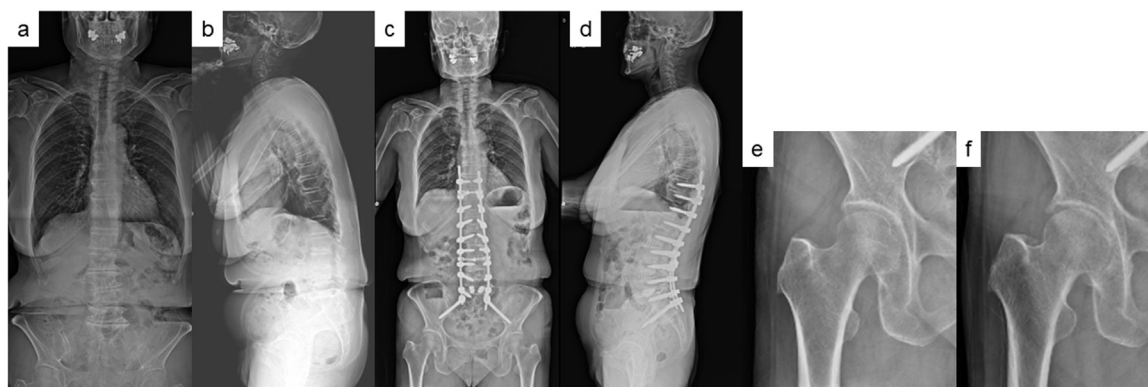


Fig. 2. Representative case. A 76-year-old woman underwent spinopelvic corrective fusion surgery from T10 to the pelvis. There was no hip lesion 1 month after surgery, but she experienced right hip pain 5 years later. A standing X-ray showed that the joint space had decreased and the KL grade had progressed for 5 years.

relationship between spinal fusion and hip OA. Motion analysis studies suggest that spinopelvic fixation increases hip joint stress during daily activities involving deep flexion, such as standing up or stair climbing [4], but this mechanism remains underexplored. Finally, all participants were Japanese. As PI tends to be lower in Asian populations, anatomical differences may limit the generalizability of our findings. Further multinational studies are needed to validate these results.

## Conclusion

This 5-year retrospective study demonstrated that spinopelvic fixation was associated with a higher risk of hip OA progression following spinal fusion surgery, particularly in female patients and those with high PI or large SS correction. These findings provide clinical support for the concept of "adjacent joint disease", wherein rigid fixation of the lumbopelvic complex imposes compensatory stress on the hip joint, contributing to degenerative changes. Long fusion was not identified as an independent risk factor in our analysis; however, the possibility that larger construct lengths may increase the risk of hip OA progression cannot be excluded and warrants further investigation. Surgical planning should therefore account for individual patient anatomy and risk profiles, especially in those with early OA.

## Declaration of competing interest

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

## CRediT authorship contribution statement

**Takuhei Kozaki:** Project administration, Methodology, Data curation, Conceptualization. **Takahiro Kozaki:** Writing – review & editing, Data curation. **Hiroshi Hashizume:** Writing – review & editing, Conceptualization. **Hiroshi Iwasaki:** Supervision. **Shunji Tsutsui:**

Supervision. **Masanari Takami:** Supervision. **Keiji Nagata:** Supervision. **Yuyu Ishimoto:** Supervision. **Masatoshi Teraguchi:** Supervision. **Daisuke Nishiyama:** Supervision. **Daisuke Fukui:** Supervision. **Manabu Yamanaka:** Supervision. **Ryo Taiji:** Supervision. **Shizumasa Murata:** Supervision. **Yuta Kaimochi:** Data curation. **Yuta Yamamoto:** Data curation. **Taiki Hayashi:** Data curation. **Hiroshi Yamada:** Writing – review & editing, Project administration, Methodology, Conceptualization.

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