European Spine Journal (2018) 27:2294–2302
https://doi.org/10.1007/s00586-018-5509-0

ORIGINAL ARTICLE

Radiological severity of hip osteoarthritis in patients with adult spinal deformity: the effect on spinopelvic and lower extremity compensatory mechanisms

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Received: 15 August 2017 / Revised: 9 January 2018 / Accepted: 30 January 2018 / Published online: 7 February 2018

Abstract

Purpose Sagittal spinal deformity (SSD) patients utilize pelvic tilt (PT) and their lower extremities in order to compensate for malalignment. This study examines the effect of hip osteoarthritis (OA) on compensatory mechanisms in SSD patients.

Methods Patients ≥ 18 years with SSD were included for analysis. Spinopelvic, lower extremity, and cervical alignment were assessed on standing full-body stereoradiographs. Hip OA severity was graded by Kellgren–Lawrence scale (0–4). Patients were categorized as limited osteoarthritis (LOA: grade 0–2) and severe osteoarthritis (SOA: grade 3–4). Patients were matched for age and T1-pelvic angle (TPA). Spinopelvic [sagittal vertical axis (SVA), T1-pelvic angle, thoracic kyphosis (TK), pelvic tilt (PT), lumbar lordosis (LL), pelvic incidence minus lumbar lordosis (PI-LL), T1-spinopelvic inclination (T1SPi)] and lower extremity parameters [sacrofemoral angle, knee angle, ankle angle, posterior pelvic shift (P. Shift), global sagittal axis (GSA)] were compared between groups using independent sample *t* test.

Results 136 patients (LOA = 68, SOA = 68) were included in the study. SOA had less pelvic tilt ($p = 0.011$), thoracic kyphosis ($p = 0.007$), and higher SVA and T1Spi ($p < 0.001$) than LOA. SOA had lower sacrofemoral angle ($p < 0.001$) and ankle angle ($p = 0.043$), increased P. Shift ($p < 0.001$) and increased GSA ($p < 0.001$) compared to LOA. There were no differences in PI-LL, LL, knee angle, or cervical alignment ($p > 0.05$).

Conclusions Patients with coexisting spinal malalignment and SOA compensate by pelvic shift and thoracic hypokyphosis rather than PT, likely as a result of limited hip extension secondary to SOA. As a result, SOA had worse global sagittal alignment than their LOA counterparts.

Research performed at Hospital for Joint Diseases, NYU Langone Medical Center, New York, NY.

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s00586-018-5509-0) contains supplementary material, which is available to authorized users.

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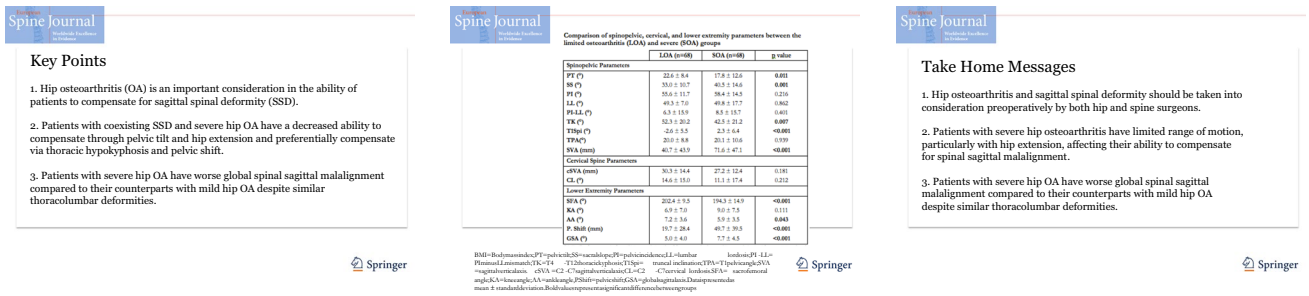
Graphical abstract These slides can be retrieved under Electronic Supplementary Material.



Keywords Sagittal spinal deformity · Hip osteoarthritis · Hip-spine syndrome · Compensatory mechanisms · Lower extremities · Global sagittal alignment

Abbreviations

AA	Ankle angle
AP	Anterior–posterior
BMI	Body mass index
GSA	Global sagittal angle
KA	Knee angle
LL	Lumbar lordosis
LOA	Limited osteoarthritis
OA	Osteoarthritis
P. Shift	Posterior pelvic shift
PI-LL	Pelvic incidence-lumbar lordosis mismatch
PSM	Propensity score matching
PT	Pelvic tilt
SFA	Sacrofemoral angle
SOA	Severe osteoarthritis
SSF	Sagittal spinal deformity
SVA	Sagittal vertical axis
T1SPi	T1-spinopelvic inclination
TK	Thoracic kyphosis
TPA	T1-pelvic angle

Introduction

Patients with coexisting hip osteoarthritis (OA) and sagittal spinal deformity (SSD) represent a unique diagnostic and surgical challenge. In hip-spine syndrome (HSS), end stage hip OA causes flexion contractures of the hip, which leads to compensatory changes of the lumbar spine, exacerbating symptoms and affecting activities of daily living [1]. Failure to recognize this close relationship between spine and hip pathology may delay treatment and lead to less than satisfactory outcomes of either hip or spine surgery [2].

Patients with sagittal malalignment increase pelvic tilt to compensate for a loss of lumbar lordosis and an anteriorly displaced center of gravity. In addition, regional compensatory mechanisms that occur in the thoracic, lumbar, and cervical spine aim to counteract the anterior truncal malalignment and maintain horizontal gaze [3]. More recently, the role of the lower extremities to compensate has become better understood, with hip extension, posterior pelvic translation, knee flexion, and ankle dorsiflexion utilized to maintain the gravity line over the ankle mortise [3–6].

Postural deformity, secondary to causes such as femoroacetabular joint pathology, may lead to different patterns of compensation [7–10]. Previous studies in the literature have observed that hip OA affects spinopelvic parameters but none have studied its effect on spinopelvic and lower extremity compensatory mechanisms [11, 12]. The purpose of this study is to evaluate the relationship between the severity of hip OA and spinal alignment. The hypothesis is that a worsening hip OA grade will be associated with limited compensatory increase in pelvic tilt and, subsequently, an increased dependence on other spinopelvic and lower extremity compensatory mechanisms.

Materials and methods

Study design and image acquisition

This is an institutional review board approved single-center retrospective study. A single-center database was reviewed retrospectively and patients were included for analysis if they presented a radiographic sagittal spinal deformity defined by one of the following criteria: sagittal vertical axis (SVA) ≥ 50 mm, pelvic tilt (PT) ≥ 25°, or thoracic kyphosis (TK) ≥ 60°. Patients were excluded if they had neurodegenerative disease, spinal stenosis, traumatic spine injury, or bilateral hip arthroplasty.

Our cohort included adult patients (≥ 18 years) with full-body, weight-bearing standing anterior–posterior (AP)

and lateral stereoradiography between 2013 and 2016. All included patients underwent low dose radiation [13], head to foot, biplanar standing stereoradiographic images (EOS imaging, Paris, France) [14, 15]. The protocol included a weight bearing free-standing position of comfort with arms flexed at 45° to avoid superimposition with the spine [16].

The severity of hip OA for each patient was graded using the Kellgren–Lawrence grading system (Table 1) on standing AP stereoradiographs [17, 18]. Patients were then categorized as having limited OA (LOA: grade 0–2) or severe OA (SOA: grade 3–4). Where different grades existed between sides, the patient was assigned the higher OA grade. Standard demographic information was recorded for each patient including age, gender, and body mass index (BMI).

Radiographic analysis

Spinopelvic, cervical and lower extremity parameters were measured on all radiographs using validated software (Surgimap, Nemaris, Inc., New York, USA) [19]. Global alignment parameters included sagittal vertical axis (SVA: horizontal offset from a C7 plumbline to the posterosuperior corner of S1), T1-pelvic angle (TPA: the angle between the line from the femoral head axis to the center of the T1 vertebra and the line from the femoral head axis to the middle of the S1 superior endplate) [20] and T1 spinopelvic inclination (T1SPi: the angle of a line from the bicoxofemoral axis to the center of T1 and a vertical line). Regional spinopelvic parameters included pelvic tilt (PT: angle between a line from the bicoxofemoral axis to the midpoint of the sacral endplate and a vertical line), sacral slope (SS), pelvic incidence (PI: angle between a line from the bicoxofemoral axis to the midpoint of the sacral endplate and the perpendicular to the sacral endplate), lumbar lordosis (LL: angle between the superior endplate of L1 and the superior endplate of S1), pelvic incidence minus lumbar lordosis (PI-LL) mismatch, and thoracic kyphosis (TK: Cobb angle between superior endplate of T4 and T12 vertebrae). Cervical spine parameters include: C2–C7 sagittal vertical axis (cSVA) and C2–C7 cervical lordosis (CL: C2–C7 Cobb angle) (Fig. 1).

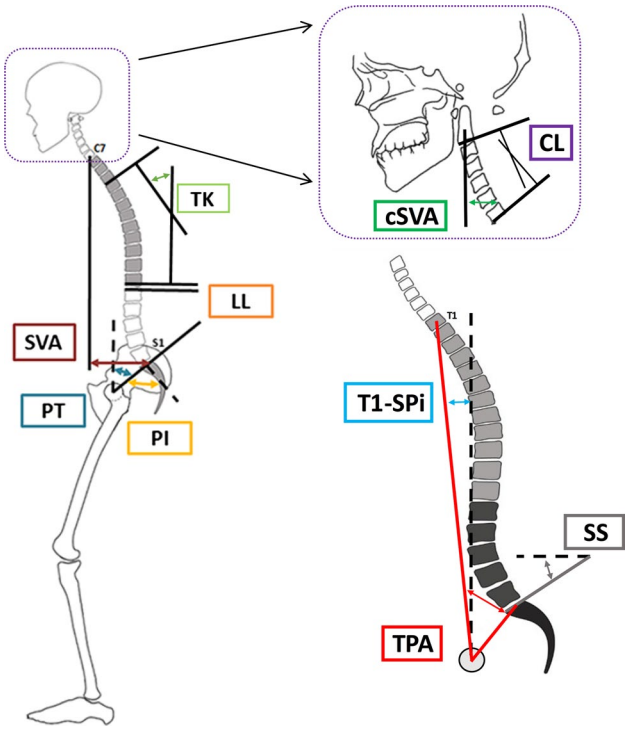


Fig. 1 Illustration of spinopelvic and cervical spinal radiographic alignment parameters and compensatory mechanisms

Lower extremity parameters (Fig. 2) include: sacrofemoral angle (SFA: the angle formed between the bicoxofemoral axis and the line tangent to the superior endplate of S1 and the line between the bicoxofemoral axis and the femoral axis), knee angle (KA: angle between the mechanical axis of the femur and the mechanical axis of the tibia), ankle angle (AA: angle between the mechanical axis of the tibia and a vertical line), posterior pelvic shift (P. Shift: the sagittal offset between the posterosuperior corner of the S1 endplate to the anterior cortex of the distal tibia), and global sagittal axis (GSA: the angle formed by a line from the midpoint of the distal femoral condyles to the center of C7, and a line from the midpoint between the distal femoral condyles to the posterior superior corner of the S1 endplate) [21].

Table 1 The Kellgren–Lawrence grading system

Grade		Description
0	No Osteoarthritis	No signs of osteoarthritis
1	Doubtful	Possible narrowing of the joint space medially, possible osteophytes around the femoral head
2	Mild	Definite narrowing of the joint space inferiorly, definite osteophytes, and slight sclerosis
3	Moderate	Marked narrowing of the joint space, slight osteophytes, some sclerosis and cyst formation, and deformity of the femoral head and acetabulum
4	Severe	Gross loss of joint space with sclerosis and cysts, large osteophytes, and marked deformity of the femoral head and acetabulum

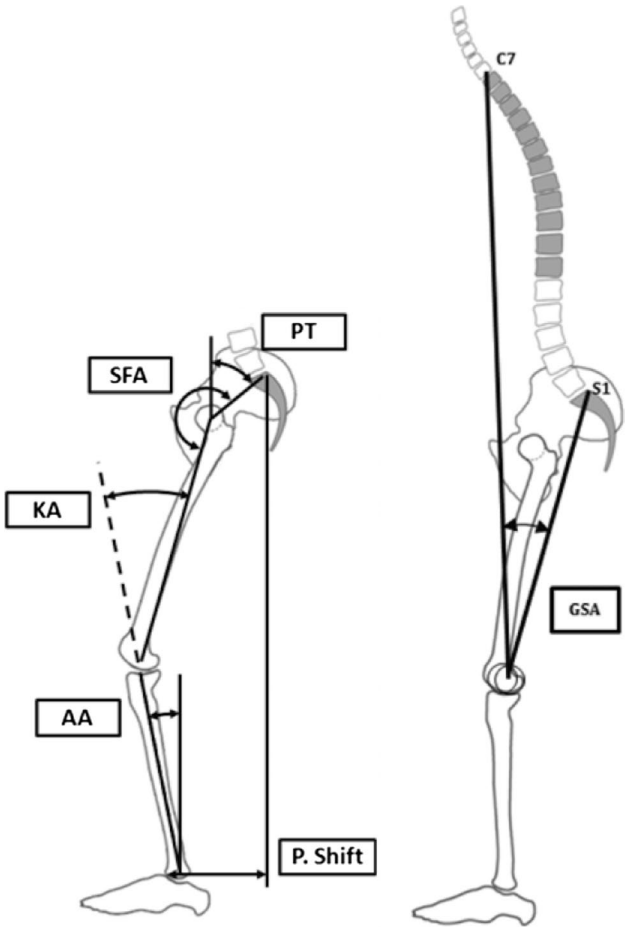


Fig. 2 Illustration of lower extremity radiographic parameters and compensatory mechanisms

Table 2 Comparison of spinopelvic, cervical, and lower extremity parameters between the limited osteoarthritis (LOA) and severe (SOA) groups

	LOA ($n = 68$)	SOA ($n = 68$)	p value
Spinopelvic parameters			
PT (°)	22.6 ± 8.4	17.8 ± 12.6	**0.011**
SS (°)	33.0 ± 10.7	40.5 ± 14.6	**0.001**
PI (°)	55.6 ± 11.7	58.4 ± 14.5	0.216
LL (°)	49.3 ± 7.0	49.8 ± 17.7	0.862
PI-LL (°)	6.3 ± 15.9	8.5 ± 15.7	0.401
TK (°)	52.3 ± 20.2	42.5 ± 21.2	**0.007**
T1Spi (°)	− 2.6 ± 5.5	2.3 ± 6.4	**< 0.001**
TPA (°)	20.0 ± 8.8	20.1 ± 10.6	0.939
SVA (mm)	40.7 ± 43.9	71.6 ± 47.1	**< 0.001**
Cervical spine parameters			
cSVA (mm)	30.3 ± 14.4	27.2 ± 12.4	0.181
CL (°)	14.6 ± 15.0	11.1 ± 17.4	0.212
Lower extremity parameters			
SFA (°)	202.4 ± 9.5	194.3 ± 14.9	**< 0.001**
KA (°)	6.9 ± 7.0	9.0 ± 7.5	0.111
AA (°)	7.2 ± 3.6	5.9 ± 3.5	**0.043**
P. Shift (mm)	19.7 ± 28.4	49.7 ± 39.5	**< 0.001**
GSA (°)	5.0 ± 4.0	7.7 ± 4.5	**< 0.001**

Data are presented as mean ± standard deviation. Bold values represent a significant difference between groups

BMI body mass index, *PT* pelvic tilt, *SS* sacral slope, *PI* pelvic incidence, *LL* lumbar lordosis, *PI-LL* PI minus LL mismatch, *TK* T4–T12 thoracic kyphosis, *T1Spi* truncal inclination, *TPA* T1 pelvic angle, *SVA* sagittal vertical axis, *cSVA* C2–C7 sagittal vertical axis, *CL* C2–C7 cervical lordosis, *SFA* sacrofemoral angle, *KA* knee angle, *AA* ankle angle, *P. Shift* pelvic shift, *GSA* global sagittal axis

Statistical analysis

Statistical analyses were performed using validated statistical software (SPSS, version 22.0, IBM, Armonk, NY, USA). Propensity score matching (PSM) was implemented to control for age and spinopelvic deformity (quantified by TPA) between the two groups. Cervical, spinopelvic, and lower extremity parameters were compared between the LOA and SOA groups using independent sample t tests. Mean values are represented as mean ± standard deviation. A p value < 0.05 was considered significant for all statistical analyses.

Results

Patient demographics

Nine hundred and ninety-seven SSD patients (LOA = 929, SOA = 68) met inclusion criteria. The mean ages of LOA and SOA were 64.0 ± 13.9 and 67.4 ± 10.2 years, respectively ($p = 0.05$), and the groups differed in TPA (25.3° ± 11.2° vs 20.1° ± 10.6°, $p < 0.001$). After matching for age and TPA with PSM, 136 patients (LOA = 68, SOA = 68) were included in the study for analysis. The mean age of the included patients was 67.6 ± 10.3 years (range 42.7–93.2 years), the BMI was 28.9 ± 7.1, and the majority of patients were female (58.1%). The LOA and SOA groups were adequately matched for age (67.7 ± 10.4 years vs 67.4 ± 10.2 years, $p = 0.853$) and did not differ in gender distribution (60.3% female vs 55.9% female, $p = 0.602$) or BMI (28.1 ± 6.6 vs 29.7 ± 7.5, $p = 0.193$). In SOA, 12 out of 68 patients (17.6%) had bilateral severe hip OA.

Radiographic analysis

The results of the analysis comparing spinopelvic, cervical, and lower extremity parameters between LOA and SOA are summarized in Table 2. LOA and SOA had similar pelvic morphology as evidenced by their PI (55.6 ± 11.7 vs 58.4 ± 14.5, $p = 0.216$) and were adequately matched for

their severity of thoracolumbar deformity according to TPA (20.0 ± 8.8 vs 20.1 ± 10.6, $p = 0.939$).

Despite similar TPA, the SOA group had significantly worse sagittal truncal malalignment as determined by both a significantly higher SVA (71.6 ± 47.1 vs 40.7 ± 43.9 mm, $p < 0.001$) and T1SPi (+ 2.3° ± 6.4° vs − 2.6° ± 5.5°, $p < 0.001$) than LOA. SOA patients had a smaller pelvic retroversion as measured by PT when compared to LOA (17.8° ± 12.6° vs 22.6° ± 8.4°, $p = 0.011$) and more SS (40.5° ± 14.6° vs 33.0° ± 10.7°, $p = 0.001$). SOA patients compensated more by thoracic hypokyphosis (42.5° ± 21.2° vs 52.3° ± 20.2°, $p = 0.007$). There was no difference in LL or PI-LL mismatch between the LOA and SOA groups ($p > 0.05$). There was also no difference between groups with respect to cervical spine compensation as determined by the CL and cSVA ($p > 0.05$).

When comparing lower extremity compensatory mechanisms, SOA had a significantly lower SFA (194.3° ± 12.4° vs 202.4° ± 9.5°, $p < 0.001$) and greater posterior pelvic shift to compensate for their sagittal malalignment (49.7 ± 39.5 vs 19.7 ± 28.4 mm; $p < 0.001$). There was also less ankle dorsiflexion in SOA versus LOA as indicated by a smaller AA (5.9° ± 3.5° vs 7.2° ± 3.6°, $p = 0.043$). KA was not significantly different between the SOA and LOA groups ($p > 0.05$). GSA, a novel angle sensitive to spine, pelvic, and lower extremity compensatory mechanisms, was higher in SOA compared to LOA (7.7° ± 4.5° vs 5.0° ± 4.0°, $p < 0.001$).

Discussion

Maintenance of spinal balance relies on the complex interplay between cervical, thoracic, lumbar spine and the pelvis in conjunction with the lower extremities while minimizing muscle exertion [22]. Failure to maintain a "conus of economy" leads to significant pain and disability [23, 24]. Patients with positive sagittal alignment progressively compensate with pelvic tilt, lordosis of mobile spinal segments and via their lower extremities to maintain this upright posture, horizontal gaze, and gait ability [25]. Patients with SOA have limited range of motion at the hip joint, particularly hip extension due to flexion contracture of the hip. Previous studies have shown the effect of hip OA on spinopelvic parameters, but this is the first study to our knowledge that examines global, regional, and lower extremity compensation for sagittal deformity in patients with concomitant hip OA.

With regard to hip and pelvic compensatory mechanisms, we identified that patients with SOA had worse global sagittal alignment than their LOA counterparts. These SOA patients had significantly less hip extension and less PT, and the authors suggest hip flexion contracture as the pathomechanical cause. Despite the two groups having similar TPA, SOA had higher SVA and T1Spi due to the inability to adequately recruit PT. In fact, in these TPA-matched cohorts, SOA met criteria for deformity in SVA (71.6 mm) and T1SPi (2.3°) where the LOA group did not (SVA = 40.7 mm, T1SPi = − 2.6°). A higher GSA was also noted in SOA due to less hip extension and increased PT. These patients did attempt to compensate by decreasing their TK, but in this low-mobility region only minimal compensation can be expected. Severity of hip OA may be a significant factor in patients who maintain a high SVA and low PT despite sufficient correction of thoracolumbar deformity as measured by TPA [9]. Further imaging and clinical analysis of these patients' hip pathology are required to understand this phenomenon. Figure 3 shows an SOA patient with less PT than an LOA counterpart with similar PI and TPA.

Posterior pelvic shift is a radiographic measurement used to quantify the axial distance of the pelvis from the ankle [4, 26]. P. Shift allows the body to maintain the gravity line over the foot, and is suggested to be recruited after pelvic tilt [27]. Patients with SOA had significantly more P. Shift compared to patients with LOA in this study. This difference in P. Shift can be attributed to limited hip extension and PT. This is consistent with previous work regarding compensatory mechanisms [27]. Although larger SVA, TPA and GSA have been shown to correlate with P. Shift [21, 26], this study also demonstrates that variations in SVA, GSA and P. Shift may be driven by hip flexion deformity. Differences were also observed at the ankle joint; patients with severe OA had a smaller AA indicating less dorsiflexion. This likely occurs in tandem with P. Shift to enable compensation for less hip extension. Knee flexion is a known compensatory mechanism in the setting of SSD [28] and helps permit P. Shift, however, although KA was higher in SOA, it was not a significant means of compensation affected by hip OA in this study.

Prior studies have attempted to evaluate the spinopelvic parameters in patients with hip OA but without SSD. Okuda et al. reported that patients with severe hip OA have larger LL and SS compared to control subjects of a similar age group due to anterior pelvic tilt; however, this present study showed no difference in LL [12]. Other changes in spinopelvic parameters in patients with severe osteoarthritis have been reported in the literature, namely higher SS and less PT [8, 11, 12, 29]. Yoshimoto et al. compared patients with hip OA to those with lumbar back pain. They reported that hip arthritis patients had a higher PI, LL, SS, and lower PT. Our study also reported higher SS and a lower PT in patients with hip osteoarthritis; however, PI was similar between groups, allowing analysis of compensatory mechanisms without the confounding effect of a larger PI. These named studies did not analyze a population that matched spinal deformity criteria. Similar to our study, Weng et al. reported that

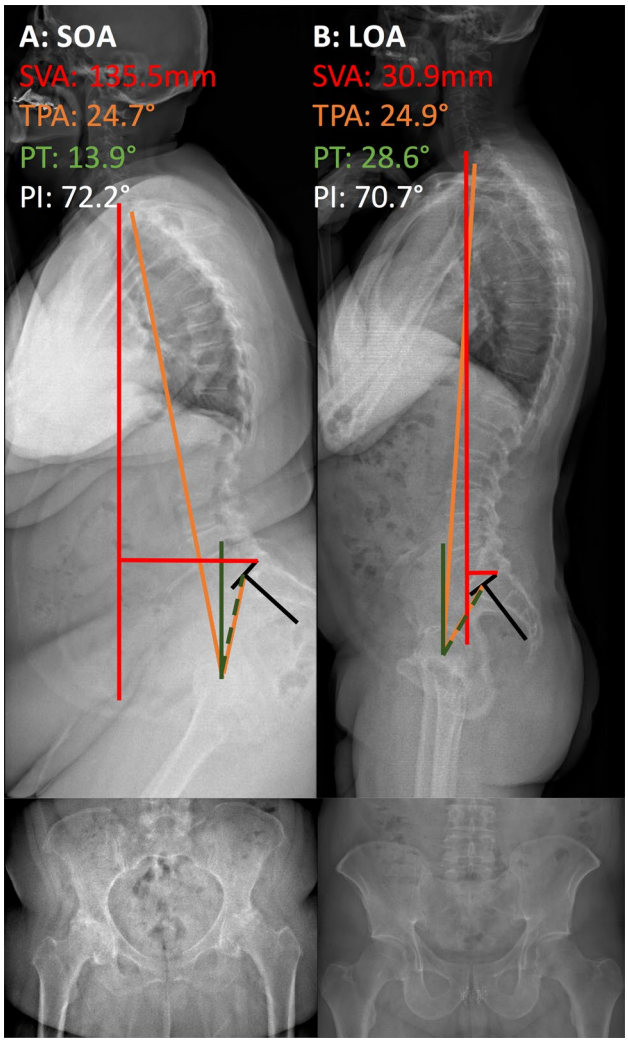


Fig. 3 Radiographs of two patient with similar Pelvic Incidence (PI) and spinopelvic deformity as measured by T1-Pelvic Angle (TPA). Note the higher Sagittal Vertical Axis (SVA) and low pelvic tilt (PT) in the patient with Severe hip OA (SOA-left) when compared to the patient with low grade hip OA (LOA)

patients with hip OA had higher SS and T1SPi, less PT, and a smaller pelvic femoral angle indicating more hip flexion, when compared to asymptomatic controls [8]. It is important to emphasize that these studies were analyzed in patients without SSD. In evaluation of hip-spine disease, the whole spine must be accounted for because spinal malalignment is a main driver of spinopelvic parameters. Global alignment, BMI, and age have been shown to affect the ability to compensate for thoracolumbar deformity, and were hence controlled for in our study to prevent bias in examining the effect of hip OA on compensatory mechanisms.

Whether patients with concomitant severe hip OA and SSD should undergo spinal fusion or total hip arthroplasty (THA) first is still being debated. There tends to be reciprocal reductions in compensatory mechanisms for SSD following surgical correction [30]; however, in certain patients, pelvic compensation does not change following surgery. Ferrero et al. reported a subset of patients with positive sagittal malalignment who had a lack of compensatory pelvic retroversion as evidenced by low baseline PT (High SVA, low PT) [9]. After surgical correction, the patients with a low PT had improvements in SVA and PI-LL mismatch, but no significant changes in PT. This limitation of compensatory pelvic retroversion may be explained by coexisting hip OA. Previous THA studies have examined reciprocal changes in spinopelvic alignment with THA [31–35], several of these studies reporting no significant change in PT [31, 33–35]. One study examined patients with concomitant unilateral hip OA and lower back pain and stated that post-THA that a contributor to spinopelvic misalignment was an antalgic posture developed by patients to keep the spine vertical [36]. The applicability of these studies in the setting of spinal deformity is limited by the absence of SSD, measurement of PT by the anterior pelvic plane, and using supine radiographs. Weng et al. demonstrated that patients had reduced T1Spi and femoral inclination and greater pelvic femoral angle 1 year after THA, showing increased hip extension and improved sagittal balance; however, there were no differences in LL, SS, PI, or PT [32]. Figure 4 demonstrates a patient who experienced a reduction in SVA and increase in PT post-THA. Although THA may allow patients more hip extension with increased mobility at the hip, PT still may not change. If SSD is not present, there will be minimal change in pelvic parameters post-THA because SSD is the main driver of spinopelvic compensation. PT may not significantly change after THA in the absence of SSD due to the lower PT values required to maintain upright posture, although improved hip extension following THA logically would allow greater PT compensation in the setting of sagittal malalignment. Conversely, SSD correction has been shown to alter acetabular anteversion in patients with pre-existing THA, with potential implications for implant stability [37, 38]. For this reason, in patients with coexisting thoracolumbar spinal misalignment and SOA, it may be reasonable to perform spinal deformity correction prior to THA to reduce the risk of iatrogenic instability. Consideration for dual mobility bearings may also help reduce the risk of THA dislocation in these high risk spinal deformity patients [39].

This study has several limitations to consider, including the reliability of the Kellgren–Lawrence grading system; however, previous studies have demonstrated its intra- and inter-user reliability [40]. Due to the heterogeneous nature of spinal deformity, there may be other confounding factors that were not adequately controlled such as neuromuscular disease and frailty. Additionally, we did not have sufficient statistical power to assess whether or not the laterality (or bilaterality) of hip OA has an effect on spinopelvic compensatory mechanisms in SSD. We are also unable

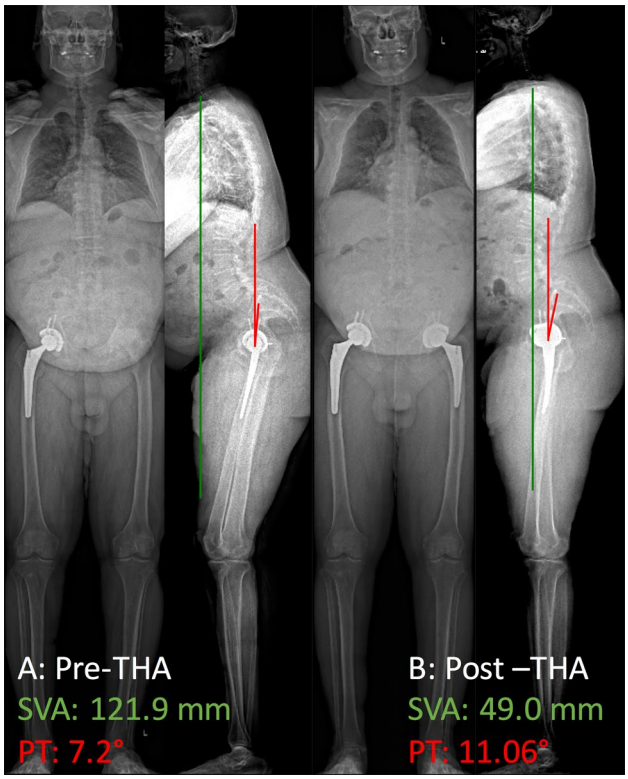


Fig. 4 AP and lateral radiogaphs of the same patient pre-(left) and post-(right) total hip replacement (THA). Note the increase in pelvic tilt THA with reduction in SVA and correction of positive sagittal malalignment

to report whether spine-specific health-related quality of life (HRQoL) measures are influenced by hip pathology when controlling for thoracolumbar deformity as increased pain may impact alignment. In a recent study, however, Ochi et al. examined the association between hip-specific HRQoL scores and sagittal spinopelvic parameters found that THA patients with preoperatively imbalanced sagittal alignment had poorer postoperative outcomes [41].

This study demonstrates that SSD patients with severe hip OA (Kellgren–Lawrence grades 3 and 4) have a significantly decreased ability to compensate for fixed spinal sagittal deformity through pelvic tilt and hip extension; consequently, they preferentially compensate via thoracic hypokyphosis and pelvic shift when compared to patients with less severe hip disease (Kellgren–Lawrence grade 0–2). Despite this, patients with severe hip OA have worse global spinal sagittal malalignment than their counterparts with mild OA despite similar thoracolumbar deformity. Hip OA is an important etiologic factor in compensation for SSD, and should be considered by both hip and spine surgeons.

Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

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