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Thoracolumbar Slope Is Useful Parameter for Evaluating Health-Related Quality of Life and Sagittal Imbalance Aggravation in Adult Spinal Deformity: A Prospective Observational Cohort Study

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Objective: The purpose of the present study was to evaluate the natural course of primary degenerative sagittal imbalance (PDSI), its aggravating factors, and health-related quality of life (HRQoL) associated with various spinal alignment parameters (SAPs) in patients with PDSI who have not undergone surgery.
Methods: One hundred three participants volunteered to participate. The SAPs, including T1 pelvic angle (T1PA), thoracolumbar tilt, and thoracolumbar slope (TLS), were measured on whole-spine standing radiographs. The back and lumbar muscle volumes were measured. To determine HRQoL at baseline and at 2-year follow-up, face-to-face questionnaires were administered, which included visual analogue scale of the back and leg, physical component summary/mental component summary of 36-item Short Form Health Survey, Oswestry Disability Index (ODI), and Mini-Mental State Examination.
Results: Overall HRQoL measures had improved after 2 years of follow-up compared to baseline. PDSI aggravation was observed in 18 participants (26.1%). TLS, sagittal vertical axis (SVA), and T1PA were strongly correlated with each other. TLS, SVA, and T1PA were correlated with ODI score. Among them, TLS was most highly correlated with ODI score. TLS greater than -3.5° was a predicting factor for PDSI aggravation ($p = 0.034$; 95% confidence interval, 1.173–63.61; odds ratio, 8.636).
Conclusion: The present study implied that PDSI does not necessarily worsen with aging. TLS is an appropriate parameter for assessing the clinical situation in patients with PDSI. Furthermore, a TLS greater than -3.5° predicts PDSI aggravation; thus, TLS may be a useful parameter for predicting prognosis in PDSI.

Keywords: Sagittal imbalance, Health-related quality of life, Thoracolumbar slope, Adult spinal deformity, Spinal alignment parameter

INTRODUCTION

Primary degenerative sagittal imbalance (PDSI) is an adult spinal deformity that has become increasingly common in elderly patients, with an estimated prevalence rate of 20%–60% in that age group.[1,2] It causes severe back pain and functional disability. Several reports have described a significant relationship between PDSI and health-related quality of life (HRQoL).[3,4] With advances in surgical instruments and technology, surgical treatment of PDSI has become more widespread, and several

studies have reported that surgery improved HRQoL in patients with PDSI.[5-7] Additionally, to understanding sagittal imbalance, various sagittal alignment parameters (SAPs) have been discovered and applied clinically, including sagittal vertical axis (SVA), pelvic incidence (PI), pelvic tilt (PT), sacral slope (SS), T1 pelvic angle (T1PA), and thoracolumbar slope (TLS).[8-10] However, these SAPs are generally applied to evaluate patients immediately before and after surgery. In high-risk patients such as the elderly, deformity correction surgery is not usually recommended because it involves extensive surgery, long operation time and high blood loss, which lead to marked perioperative morbidity.

If clinicians better understand the natural course of the PDSI without surgical treatment, they may be able to predict sagittal imbalance aggravation, prevent further aggravation, and thus improve HRQoL in patients with PDSI. Therefore, the purpose of the present study was to evaluate the natural course of PDSI, its aggravating factors, and HRQoL associated with various SAPs in patients with PDSI who have not undergone surgery.

MATERIALS AND METHODS

1. Patient Population and Study Design

The study was approved by the Institutional Review Board of Chonnam National University Medical School Research Institute (approval number: CNUH-2016-127). Informed consent was obtained from all individual participants included in this study. This prospective longitudinal cohort study included a follow-up of PDSI cases for 2 years in patients with PDSI to determine which SAPs were associated with the natural course of PDSI and HRQoL.

We recruited volunteers who were older than 65 years, exhibited a stooping posture in daily living, but had not received medication or surgical treatment. Before enrolling subjects, we checked the whole-spine standing radiographs to confirm that the volunteers had SVA larger than 50 mm.[11] In total, 103 participants volunteered to participate. The exclusion criteria were as follows: (1) coronal deformity (Cobb angle > 10°); (2) less than 2 years of clinical or radiological follow-up; (3) history of spine, hip, or knee surgery; (4) prescription of pain medication; (5) history of Parkinson disease or other neuromuscular disorder; (6) presence of infection, fracture, or malignancy. Date of the following variables related to patient demographics were recorded at baseline and at the 2-year follow-up: age, sex, body mass index (BMI), bone mineral density, whole-spine standing radiograph, lumbar spine magnetic resonance imaging (MRI), and plasma clinical chemistry.

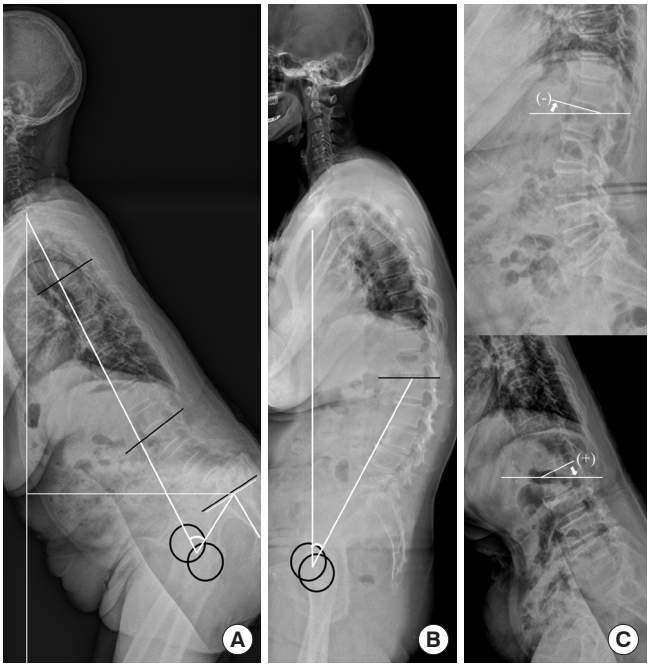

Fig. 1. Whole-spine standing radiograph. (A) T1 pelvic angle: angle between the lines from the centroid of T1 to the femoral head axis and from the femoral axis to the middle of the S1 endplate. (B) Thoracolumbar tilt: angle between a vertical reference line and the line connecting the midpoint of the L1 superior endplate to the center axis of the femoral heads. This is a positive value similar to pelvic tilt. (C) Thoracolumbar: angle between a horizontal reference line and the L1 superior endplate. This is a negative value similar to sacral slope.

2. Radiological and Clinical Evaluation

The following SAPs were measured on whole-spine standing radiographs based on Scoliosis Research Society–Schwab radiological classification: SVA, PI, PT, SS, lumbar lordosis (LL), and thoracic kyphosis (TK)[8]. Additionally, T1PA, TLS, and thoracolumbar tilt (TLT) were measured (Fig. 1).[9,11] Based on serial analysis, PDSI aggravation was defined as a T1PA increase of more than 3° and SVA increase of more than 30 mm compared to baseline value.[1]

The back and lumbar muscle volumes were measured based on the cross-sectional areas of the lumbar multifidus muscle (MF), lumbar erector spinae muscle (ES), and psoas muscle (PS). These measurements were obtained from MRI at the lumbar 4/5-disc level using the region of interest measurement tool of the picture archiving and communication system (M-view; INFINITT Healthcare, Seoul, Korea). Furthermore, fatty change in the lumbar muscle was measured according to the grading

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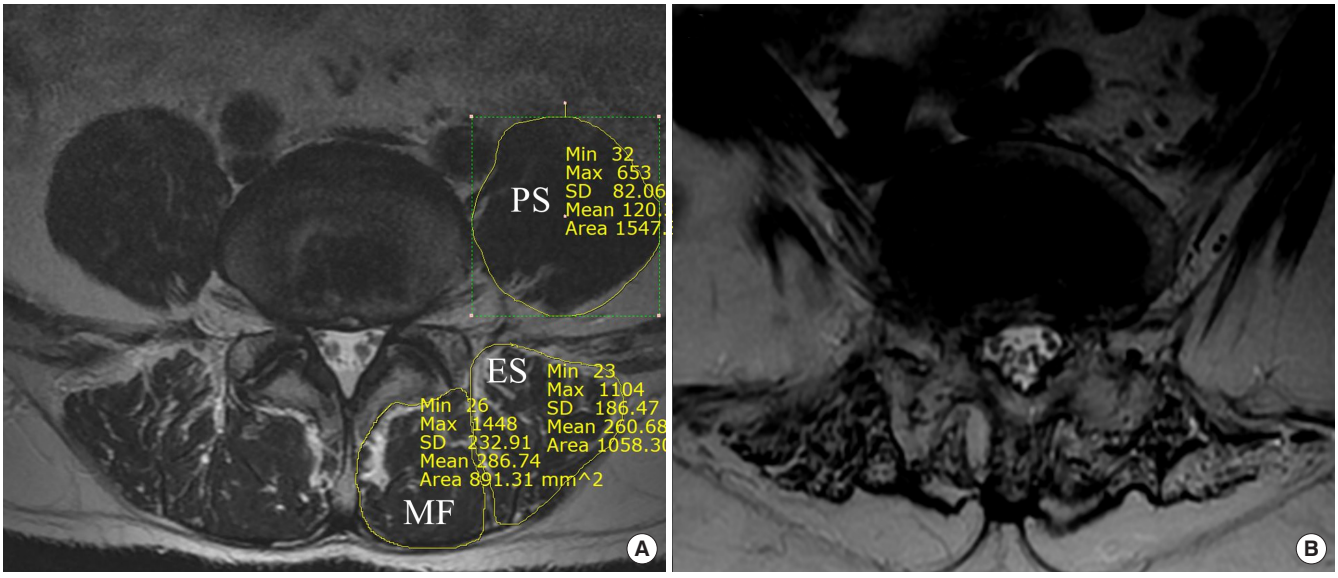

Fig. 2. Lumbar spine magnetic resonance imaging (MRI) at the L4/5-disc level. (A) The back and lumbar muscle volumes were measured based on the cross-sectional areas (CSAs) of the lumbar multifidus muscle (MF), lumbar erector spinae muscle (ES), and psoas muscle (PS). (B) Lumbar spine MRI at the L4/5-disc level showing Goutallier classification grade 4. The CSA of MF was 326 mm^2. SD, standard deviation.

system of Goutallier et al.[12] (Fig. 2).

Laboratory tests were performed on peripheral venous blood samples to determine the levels of the following parameters: total cholesterol, triglyceride, high-density lipoprotein, hemoglobin A1c, rheumatoid factor, vitamin D, osteocalcin, and C-terminal telopeptide.

To determine HRQoL at baseline and at 2-year follow-up, face-to-face questionnaires were administered, which included visual analogue scale of the back and leg, physical component summary (PCS)/mental component summary (MCS) of 36-item Short Form Health Survey (SF-36), Oswestry Disability Index (ODI), and Mini-Mental State Examination. In addition, after the participants had provided written consent for participation, they were educated about the etiology, natural course, and surgical treatment of PDSI; they underwent brief training on the importance of core and lumbar extensor muscle strengthening exercises (lumbar extension, hip extension, trunk flexion, and leg press exercise) and rest.[13-15]

3. Statistical Analysis

Data of continuous variables were expressed as mean ± standard deviation. The paired t-test was used to compare measurements taken at baseline and 2-year follow-up. The radiological and muscle parameters at 2-year follow-up were also compared between the aggravation and nonaggravation groups. Pearson correlation coefficient test was performed to analyze correlations between SAPs and HRQoL measures. Correlation strengths were interpreted according to the method described by Evans[16] ($r = 0.00–0.19$: very weak, $r = 0.20–0.39$: weak, $r = 0.40–0.59$: moderate, $r = 0.60–0.79$: strong, $r = 0.80–1.00$: very strong). Multivariate logistic regression analysis was used to identify predictive factors for PDSI aggravation. All statistical analyses were performed using the IBM SPSS Statistics ver. 26.0 (IBM Co., Armonk, NY, USA). A p-value of < 0.05 was considered significant.

RESULTS

Among 103 participants, 34 were excluded from the study due to refusal of lumbar MRI check, nursing hospital admission, hip fracture, or loss to follow-up. Therefore, 69 participants with PDSI were enrolled.

After 2 years of follow-up, the overall HQRoL measures had improved when compared with those at baseline. Among them, ODI, PCS of SF-36, and MCS of SF-36 had improved significantly ($p = 0.047$, $p < 0.001$, and $p < 0.001$, respectively). Among SAPs, SVA, T1PA, and TLS had also improved significantly after 2 years of follow-up ($p = 0.007$, $p = 0.038$, and $p = 0.024$, respectively). Conversely, PT had increased, and TK had decreased significantly after 2 years of follow-up ($p = 0.003$ and $p < 0.001$, respectively), meaning a compensatory change. These results confirmed that PDSI is not necessarily aggravated by the aging

Table 1. Patient clinical and laboratory characteristics

Variable	Baseline (n = 69)	2-Year F/U (n = 69)	p-value†
Clinical characteristics			
Age (yr)	70.6 ± 4.00	-	
Female sex	57 (82.6)	-	
BMI (kg/m^2)	24.29 ± 2.77	24.77 ± 2.87	0.34
BMD (g/cm^2)	-1.49 ± 0.88	-1.59 ± 0.80	0.043*
VAS back	7.67 ± 2.63	7.43 ± 2.24	0.068
VAS leg	7.16 ± 2.93	7.02 ± 2.81	0.059
ODI	20.58 ± 7.35	19.63 ± 7.22	0.047*
SF-36 PCS	36.18 ± 13.72	26.83 ± 11.77	< 0.001*
SF-36 MCS	47.64 ± 15.49	40.14 ± 14.81	< 0.001*
MMSE	26.16 ± 3.22	25.77 ± 3.42	0.064
Laboratory characteristics			
Cholesterol (mg/dL)	193.90 ± 47.04	181.84 ± 40.14	0.016*
TG (mg/dL)	136.26 ± 89.43	142.65 ± 65.06	0.536
HDL (mg/dL)	55.77 ± 12.50	55.32 ± 12.54	0.683
HbA1c (%)	5.87 ± 1.09	5.96 ± 0.93	0.278
RF (IU/mL)	6.69 ± 11.53	7.11 ± 14.73	0.495
Vitamin D (ng/mL)	21.66 ± 8.13	18.09 ± 8.65	0.003*
CTX (ng/mL)	0.42 ± 0.22	0.49 ± 1.33	0.649
Osteocalcin (ng/mL)	21.30 ± 8.48	18.12 ± 7.89	0.002*

Values are presented as mean ± standard deviation or number (%).
F/U, follow-up; BMI, body mass index; BMD, bone mineral density; BMI; VAS, visual analogue scale; ODI, Owestry Disability Index; SF-36, 36-item Short Form Health Survey; PCS, physical component summary; MCS, mental component summary; MMSE, Mini-Mental State Examination; TG, triglyceride; HDL, high-density lipoprotein; HbA1c, hemoglobin A1c; RF, rheumatoid factor; CTX, C-terminal telopeptide.
*$p < 0.05$, statistically significant differences. †A comparison of mean values between the baseline and 2-year follow-up.

Table 2. Patient radiological and muscle characteristics

Variable	Baseline (n = 69)	2-Year F/U (n = 69)	p-value†
Radiological characteristics			
SVA (cm)	12.32 ± 6.38	9.85 ± 9.20	0.007*
PI (°)	56.53 ± 11.30	57.28 ± 9.99	0.574
PT (°)	26.99 ± 11.81	29.90 ± 10.63	0.003*
SS (°)	29.61 ± 12.10	27.37 ± 9.76	0.069
LL (°)	25.36 ± 20.72	25.50 ± 20.43	0.94
TK (°)	30.35 ± 23.68	19.53 ± 15.81	< 0.001*
T1PA (°)	32.13 ± 13.94	30.68 ± 14.31	0.038*
TLS (°)	2.97 ± 15.36	0.69 ± 16.27	0.024*
TLT (°)	7.32 ± 10.34	8.80 ± 9.48	0.062
Muscle characteristics			
Goutallier grade of MF	2.29 ± 0.96	2.32 ± 0.95	0.159
Goutallier grade of ES	2.10 ± 0.88	2.12 ± 0.87	0.321
Goutallier grade of PS	2.15 ± 0.82	2.16 ± 0.80	0.321
CSA of MF (mm^2)	713.10 ± 208.50	704.90 ± 211.69	0.306
CSA of ES (mm^2)	1,704.3 ± 1,080.5	1,688.8 ± 1,077.8	0.249
CSA of PS (mm^2)	2,364.5 ± 420.8	2,323.6 ± 420.8	0.217
Aggravation of sagittal imbalances	-	18 (26.1)	-

Values are presented as mean ± standard deviation or number (%).
F/U, follow-up; SVA, sagittal vertical axis; PI, pelvic incidence; PT, pelvic tilt; SS, sacral slope; LL, lumbar lordosis; TK, thoracic kyphosis; T1PA, T1 pelvic angle; TLS, thoracolumbar slope; TLT, thoracolumbar tilt; MF, multifidus; ES, erector spinal muscle; PS, psoas muscle; CSA, cross-sectional area.
*$p < 0.05$, statistically significant differences. †A comparison of mean values between the baseline and 2-year follow-up.

process (Tables 1, 2). PDSI aggravation was observed in 18 participants (26.1%). When analyzed by dividing into the aggravation group and nonaggravation group, HRQoL measures were poorer in the aggravation group, although the mean values showed no significant difference between the 2 groups. Most SAPs also revealed poor values in the aggravation group, but only SVA, T1PA, and TLS differed significantly ($p < 0.001$, $p = 0.019$, and $p = 0.008$, respectively). After the exclusion of SVA and T1PA, which are used to define PDSI aggravation, TLS was the only SAP that differed significantly between the 2 groups ($p = 0.008$). Fatty changes and volume of the back and lumbar muscles were not significantly different between the 2 groups (Table 3).

The relationship between HRQoL measures and SAPs was assessed at baseline and at 2-year follow-up (Table 4). TLS, SVA, and T1PA were strongly correlated with each other. TLS showed a strong correlation with SVA ($r = 0.782$, $p < 0.001$ at baseline; $r = 0.737$, $p < 0.001$ at 2-year follow-up) and T1PA ($r = 0.670$, $p < 0.001$ at baseline; $r = 0.747$, $p < 0.001$ at 2-year follow-up). T1PA showed a strong correlation with SVA ($r = 0.672$, $p < 0.001$ at baseline; $r = 0.695$, $p < 0.001$ at 2-year follow-up). TLS, SVA, and T1PA were correlated with ODI score at baseline. Among them, TLS was most highly correlated with ODI score ($r = 0.336$, $p < 0.005$). TLS and T1PA were correlated with ODI score at the 2-year follow-up. Among them, TLS most highly correlated with ODI score ($r = 0.374$, $p = 0.002$).

Multivariate logistic regression analysis showed that TLS greater than -3.5°, which is the mean value in patients with PDSI who require surgical treatment due to clinical symptoms,[11] was a predicting factor for PDSI aggravation ($p = 0.034$; confidence in-

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Table 3. Radiologic, muscle parameters, and HRQoL measures in the aggravation and nonaggravation group

Variable	AG	Non-AG	p-value†
No. of patients (%)	18 (26.1)	51 (73.1)	
Female sex	15 (83.3)	42 (82.4)	0.874
BMI (kg/m^2)	26.13 ± 4.46	24.98 ± 3.88	0.328
BMD (g/cm^2)	-1.58 ± 1.81	-1.46 ± 1.24	0.325
HRQoL measures			
VAS back	7.61 ± 2.33	7.28 ± 1.94	0.061
VAS leg	7.63 ± 2.52	6.99 ± 2.73	0.052
ODI	20.11 ± 7.28	18.28 ± 7.05	0.374
SF-36 PCS	29.41 ± 10.75	25.92 ± 12.07	0.224
SF-36 MCS	42.97 ± 14.47	39.14 ± 14.94	0.234
MMSE	25.78 ± 2.88	25.76 ± 3.63	0.789
Radiological parameters			
SVA (cm)	15.99 ± 12.72	7.68 ± 6.47	< 0.001*
PI (°)	58.36 ± 9.77	56.89 ± 10.14	0.662
PT (°)	29.94 ± 12.32	29.88 ± 10.09	0.832
SS (°)	28.41 ± 9.05	27.01 ± 10.05	0.571
LL (°)	19.11 ± 14.90	27.76 ± 21.73	0.084
TK (°)	16.44 ± 15.95	20.64 ± 15.77	0.396
T1PA (°)	38.09 ± 16.02	28.07 ± 12.83	0.019*
TLS (°)	8.38 ± 13.05	-2.02 ± 16.53	0.008*
TLT (°)	4.84 ± 10.58	10.20 ± 8.75	0.054
Muscle parameters			
Goutallier grade of MF	2.28 ± 0.75	2.34 ± 1.03	0.635
Goutallier grade of ES	2.05 ± 0.87	2.14 ± 0.89	0.487
Goutallier grade of PS	2.11 ± 0.83	2.18 ± 0.80	0.505
CSA of MF (mm^2)	708.00 ± 234.97	702.78 ± 207.22	0.702
CSA of ES (mm^2)	2,101.7 ± 2011.7	1,543.2 ± 317.4	0.332
CSA of PS (mm^2)	2,326.6 ± 462.7	2,327.5 ± 412.5	0.707

Values are presented as mean ± standard deviation or number (%).
HRQoL, health-related quality of life; AG, aggravation; BMI, body mass index; BMD, bone mineral density; BMI; VAS, visual analogue scale; ODI, Owestry Disability Index; SF-36, 36-item Short Form Health Survey; PCS, physical component summary; MCS, mental component summary; MMSE, Mini-Mental State Examination; SVA, sagittal vertical axis; PI, pelvic incidence; PT, pelvic tilt; SS, sacral slope; LL, lumbar lordosis; TK, thoracic kyphosis; T1PA, T1 pelvic angle; TLS, thoracolumbar slope; TLT, thoracolumbar tilt; MF, multifidus; ES, erector spinal muscle; PS, psoas muscle; CSA, cross-sectional area.
*p < 0.05, statistically significant differences. †A comparison of mean values between the aggravation and nonaggravation group.

Table 4. Pearson correlation coefficient of sagittal alignment parameters and health-related quality of life measures at baseline and 2-year follow-up

Variable	TLS	SVA	T1PA	ODI	SF-36 PCS	SF-36 MCS
Baseline						
TLS	-	0.782*	0.670*	0.336*	-0.223	-0.136
SVA		-	0.672*	0.250*	-0.236	-0.224
T1PA			-	0.293*	-0.234	-0.129
ODI				-	-0.714*	-0.531*
SF-36 PCS					-	0.672*
SF-36 MCS						-
2-Year follow-up						
TLS	-	0.737*	0.747*	0.374*	-0.181	-0.102
SVA		-	0.695*	0.234	-0.156	-0.200
T1PA			-	0.370*	-0.201	-0.159
ODI				-	-0.779*	-0.688*
SF-36 PCS					-	0.730*
SF-36 MCS						-

TLS, thoracolumbar slope; SVA, sagittal vertical axis; T1PA, T1 pelvic angle; ODI, Owestry Disability Index; SF-36, 36-item Short Form Health Survey; PCS, physical component summary; MCS, mental component summary.
*p < 0.05, statistically significant differences.

Table 5. Multivariate analysis of factors predicting primary degenerative sagittal imbalance aggravation

Variable	No. of preservations (%)	Multivariate analysis		
		p-value	95% CI	OR
TLS (°)				
< -3.5	3/30 (10)	0.034	1.173–63.61	8.636
≥ -3.5	15/39 (38.5)			
T1PA (°)				
< 30	6/36 (16.7)	0.442	0.041–4.010	0.408
≥ 30	12/33 (36.4)			
SVA (cm)				
< 9.5	5/30 (16.7)	0.057	0.943–46.466	6.618
≥ 9.5	14/39 (35.9)			
BMI (kg/m^2)				
< 25	9/32 (28.1)	0.512	0.411–5.945	1.564
≥ 25	9/37 (24.3)			

CI, confidence interval; OR, odds ratio; TLS, thoracolumbar; T1PA, T1 pelvic angle; SVA, sagittal vertical axis; BMI, body mass index.

terval, 1.173–63.61; odds ratio, 8.636). A BMI greater than 25 kg/m^2, which is the criterion for overweight; an SVA greater than 9.5 cm, which is the criterion for marked deformity[8]; and a T1PA greater than 30° were not significant predictors for PDSI aggravation (Table 5). No variables other than TLS were significantly associated with PDSI aggravation, including volume and

fatty change of back muscle, BMI, or laboratory values.

DISCUSSION

PDSI is understood as the cumulative result of degenerative changes and the natural progression of aging.[11,17] In a longitudinal study of age-related changes in sagittal spinal alignment in 237 individuals over a 4-year period, Oe et al.[18] reported that SAPs deteriorated with age, including SS, PT, LL, T1 slope, cervical lordosis, and SVA in the seventh-decade female group. Many elderly individuals experience mild sagittal imbalance, but PDSI does not deteriorate in all individuals with aging.[4,17,19] Interestingly, in the present study, overall SAPs had not deteriorated, and overall HRQoL measures had improved after 2 years of follow-up compared to baseline. Although, we included a smaller number of individuals and our follow-up period was shorter than that in the study of Oe et al.,[18] the result that the PDSI has not deteriorated is significant. Participants were not given any special treatment, but only at the beginning of the study, they were educated about the etiology, natural course, and surgical treatment of PDSI; they were also trained briefly on the importance of core and lumbar extensor exercises and rest. Although it is difficult to find out the evidence of PDSI improvement only with this study, it is assumed that PDSI aggravation can be prevented by education, and further, prevalence can likely be reduced.

PDSI causes back pain and functional disability that leads to severe quality of life disturbance. Indeed, several reports have described a significant relationship between PDSI and HRQoL.[3,4] Along with these perceptions, various SAPs are being discovered to evaluate PDSI and HRQoL in patients. Schwab et al.[8] reported that sagittal imbalance severity, assessed using SVA, PT, and PI minus LL (PI–LL), was correlated with HRQoL measures. Banno et al.[20] found that T1PA was correlated with HRQoL measures. However, because the SAPs are usually applied in surgical planning to restore the ideal global spinal alignment, and because they are related to pre- or postoperative HRQoL, questions have arisen concerning their clinical suitability in patients with PDSI who have not undergone surgical treatment. In addition, global SAPs that require spinopelvic alignment, such as SVA and T1PA, may involve measurement errors because it is difficult to obtain clear visualization of both femoral heads, the S1 endplate, and the T1 vertebral body using whole-spine lateral radiography.[3] Moreover, SAPs that are measured using Cobb angle through 2 or more endplates are more likely to have erroneous measurements.[21] Recently, Moon et al.[10,11] reported novel SAPs of the thoracolumbar junction, including TSL and TLT, categorizing them as thoracolumbar junction orientation (TLJO). To measure TLS, whole-spine lateral radiography is not required, and because only the L1 endplate needs to be clearly identified, measurement errors are reduced and the reliability is high (Fig. 1C). Moon et al.[10,11] did not reveal an association between TLJO and HRQoL in patients with PDSI who had not undergone surgical treatment, but they argued that TLJO was correlated with spinopelvic alignment and global SAPs in various clinical situations. Similarly, in the present study, TLS was correlated with global SAPs such as SVA and the T1PA. In particular, TLS was correlated strongly with SVA at baseline and 2-year follow-up ($r = 0.782$ and $r = 0.737$, respectively, $p < 0.001$), as well as with T1PA at baseline and 2-year follow-up ($r = 0.670$ and $r = 0.747$, respectively, $p < 0.001$). In addition, TLS was significantly correlated with those at baseline and 2-year follow-up HRQoL measures in this study, and the correlations between the HRQoL were stronger than those for SVA and T1PA.

PDSI is a multifactorial complex spinal deformity that can arise from various causes such as spinal stenosis, sarcopenia, osteoporosis, vertebral fracture, high BMI, and neuromuscular diseases. As such, it is difficult to clearly identify the predictive factors for aggravation in patients with PDSI. Several studies have attempted to predict the PDSI aggravation using various SAPs, but most have focused on identifying predictive factors associated with postoperative PDSI aggravation.[3,10,20] Moreover, in high-risk patients such as the elderly, deformity correction surgery cannot be easily recommended because perioperative morbidity is greater in such patients. A comprehensive understanding of the natural course of the PDSI without surgical treatment may be able to predict the aggravation of sagittal imbalance, prevent further aggravation, and thus improve HRQoL in patients with PDSI. Several prospective obervatinoal cohort studies have been carried out in patients with PDSI, but no predictive factors for PDSI aggravation have been identified.[17,18] Similarly, we previously reported that marked sagittal imbalance is associated with small MF volume, high PI, and working for a long time in a crouched posture, as do agricultural workers, but we failed to identify predictive factors for PDSI aggravation.[1,22] The present study was the first to reveal that TLS greater than -3.5° is a predictive factor for PDSI aggravation in patients who have not undergone surgical treatment ($p = 0.034$), regardless of various other SAPs. In their retrospective study, Moon et al.[10,11] revealed that TLS of -3.5° is the mean value in patients with PDSI who require surgical treatment due to clini-

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cal symptoms and that proximal junctional kyphosis can be reduced when the postoperative TLS change is less than 9.4°. Based on these results, they argued that TLS is a useful parameter that could be used as a guidelines in sagittal realignment surgery. Consequently, TLS may be a useful and highly reliable additional global SAP to evaluate PDSI in various clinical situations in asymptomatic, non-surgical patients and in those requiring correction surgery.

This prospective cohort study had some limitations. First, we found no evidence for improvement in overall SAPs or HRQoL. Second, because of the relatively small number of subjects, our results cannot be generalized to all patients with PSDI aggravation. Further investigations utilizing a larger number of subjects should be performed to reduce bias. Third, the follow-up period may have been too short to evaluate PDSI aggravation and a longer follow-up cohort study will be necessary.

CONCLUSION

The present study implied that PDSI does not necessarily worsen with aging, even though it is a multifactorial complex spinal deformity associated with the natural progression of aging. Moreover, TLS is more accessible and more strongly correlated with HRQoL than other SAPs. As such, it is an appropriate parameter for assessing the clinical situation in patients with PDSI. Furthermore, a TLS greater than -3.5° predicts PDSI aggravation; thus, TLS may be a useful parameter for predicting prognosis in PDSI.

CONFLICT OF INTEREST

The authors have nothing to disclose.

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