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Original Article

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Correlation of Paraspinal Muscle Mass With Decompensation of Sagittal Adult Spinal Deformity After Setting of Fatigue Post 10-Minute Walk

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Objective: The purpose of this study was to investigate the changes in spinopelvic parameters before and after the setting of muscle fatigue along with its correlation with pre-existing paraspinal and psoas muscle mass.
Methods: Single-center retrospective review of prospectively collected data was conducted on 145-adults with symptomatic loss of lumbar lordosis (LL). Radiographs were taken before and after walking for 10 minutes. Magnetic resonance imaging was used to calculate paraspinal muscle (PSM) cross-sectional area (CSA), mean signal intensity, fatty infiltration (FI), and lean muscle mass at thoracolumbar junction (T12) and lower lumbar level (L4). Psoas CSA was calculated at L3. Patients were divided into 2 groups namely compensated sagittal deformity (CSD) (SVA $\leq$ 4 cm, PT > 20°) and decompensated sagittal deformity (DSD) (SVA > 4 cm, PT > 20°) based on prewalk measurements.
Results: Initial mean SVA was 1.8 cm and 11 cm for CSD and DSD respectively ($p < 0.01$). After walking, significant deteriorations in SVA, PT–LL ($p < 0.01$) were observed in CSD without significant change in thoracic kyphosis (TK). All sagittal parameters in DSD deteriorated significantly. DSD group had significantly poorer PSM quality at T12 and L4 compared to CSD group. In CSD group, sagittal decompensation correlated with muscle quality, i.e., decreases in LL (ΔLL) correlated with CSA of PSM/vertebral body (VB) at L4 ($r = -0.412$, $p = 0.046$) while increases in TK (ΔTK) correlated with CSA of PSM/VB at T12 ($r = 0.477$, $p = 0.018$). ΔSVA and ΔPT correlated with FI at L4 ($r = 0.577$, $p = 0.003$ and $r = -0.407$, $p = 0.048$, respectively). DSD group, had weak correlations ($-0.3 < r < -0.1$) between changes in sagittal and PSM parameters.
Conclusion: PSM quality in adults with spinal deformity correlates with patients' ability to maintain an upright posture and sagittal decompensation after walking for 10 minutes.

Keywords: Deformity, Kyphosis, Fatigue, Paraspinal muscles

INTRODUCTION

The physiological sagittal alignment of the human spine is the most energy efficient position adapted by the spine while maintaining the erect posture. Sagittal imbalance causes accessory muscles to overwork in order to maintain the erect posture and thereby, inducing inefficient energy expenditure.[1] Thus, the skeletal muscles supporting the spine can mask the actual sagittal malalignment in some cases, while in others due to the magnitude of the deformity or reduced muscle strength, the muscles fail to compensate for the imbalance, leaving the deformity unmasked.

As the sagittal imbalance has direct bearing on the patient's functional outcome and quality of life,[2] it is important to understand if a patient is masking his/her sagittal imbalance by recruiting the supporting muscles. It has been observed that

when a person with sagittal malalignment exercises and exhausts the supporting muscle strength, the deformity is unmasked/exaggerated.[3] It is clear that patients with lower muscle strength will tend to have lower capacity for compensation and will have decompensated deformity while some patients can mask their sagittal imbalance owing to their muscle strength.

The purpose of this study was to investigate the changes in spinopelvic parameters before and after the setting of muscle fatigue along with its correlation with pre-existing paraspinal and psoas muscle mass.

MATERIALS AND METHODS

After the approval of the Institutional Review Board of Wooridul Spine Hospital (2021-05-WSH-005), retrospective review of prospectively collected radiological data in a series of 145 adults (age >18 years) who presented in outpatient clinic of a single specialty spine hospital, during the years 2015 to 2020 with symptomatic loss of lumbar lordosis ("flatback"; pelvic incidence minus lumbar lordosis >10°) without a major coronal deformity (Cobb angle <30°). We excluded patients with inadequate radiological examinations, history of vertebral compression fractures, spondylolysis, spondylolisthesis, symptomatic lumbar stenosis with neurogenic claudication or sciatica, idiopathic or neuromuscular scoliosis, Cobb angles >30°, hip joint disease, and Scheuermann's kyphosis.

1. Radiographic Evaluation

All analyses were performed on preoperative full length, 36-inch exposure radiographs of the spine that extended from the base of the skull to the proximal femur in the anteroposterior and lateral planes. All radiographs were obtained with the patients standing and looking forward trying to maintain a horizontal gaze and with their arms flexed, hands placed on their clavicles without any support, and knees extended. After the first standing radiographs were completed, the patients were immediately asked to walk for 10 minutes at their usual walking speed in the same hallway (no inclination) of one clinic without resting on a chair or the walls. The manager of the radiology suite in the clinic who was not involved in image acquisition observed each patient for the entirety of the 10-minute walk. The walking time was exactly 10 minutes, as timed by the radiology supervisor. Immediately after the 10-minute walk, repeat radiographs were obtained.

Each patient's paraspinal muscles (PSMs) and psoas muscles were analyzed on prewalking MRIs. The cross-sectional areas (CSAs) of the psoas at L3 and of the PSM at T12 and L4 were analyzed on axial T2-weighted images (Figs. 1 and 2). The ratio of muscle CSA at each of these levels to the corresponding vertebral body's CSA was calculated. The mean signal intensity (SI) of the muscle was measured using a histogram (Fig. 3A, B). The fatty infiltration (FI) of the PSM was also determined with pseudo-color mapping at each level (Fig. 4) The lean muscle mass (LM) was calculated by extracting FI from CSA of PSM. An independent observer (spinal neurosurgeon in our hospital) measured the muscle parameters using computer-based picture-archiving communication system (PiView; INFINITT Co. Ltd., Seoul, Korea) and spinopelvic parameters were measured with Surgimap (Nemaris Inc., Methuen, MA, USA) software.

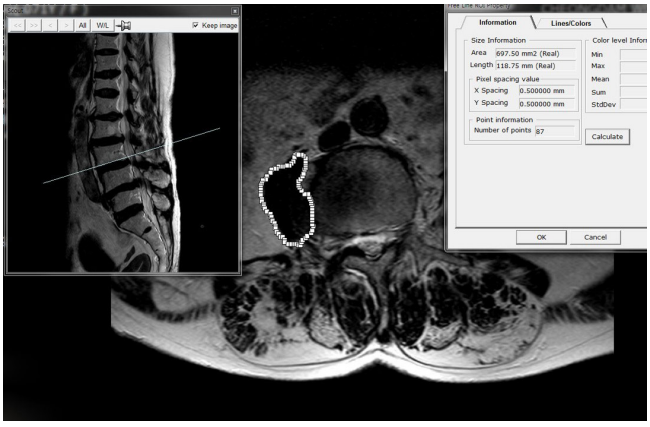

Fig. 1. Method to calculate psoas muscle cross-sectional area at L3 level based on T2-weighted magnetic resonance imaging sequences.

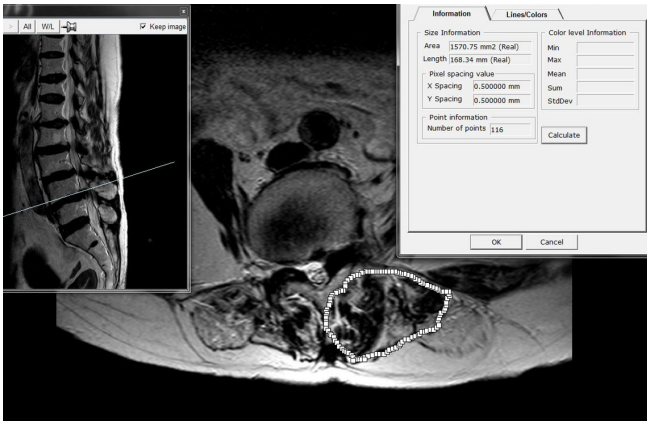

Fig. 2. Method to calculate paraspinal muscle cross-sectional area at L4 level based on T2-weighted magnetic resonance imaging sequences.

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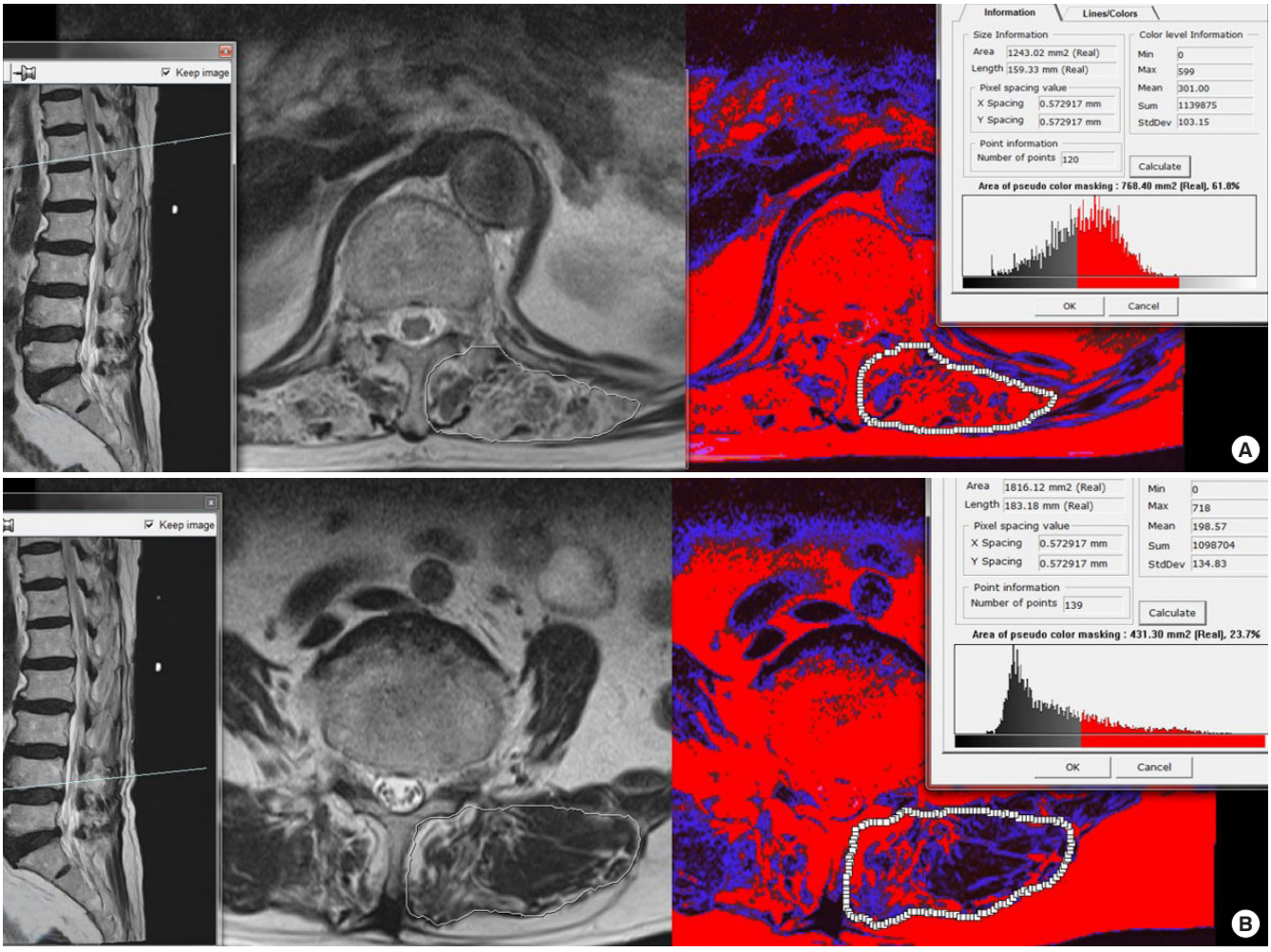

Fig. 3. (A) The calculation of mean signal intensity (SI) of paraspinal muscles at T12 level using histogram. (B) The calculation of mean signal intensity (SI) of paraspinal muscles at L4 level using histogram.

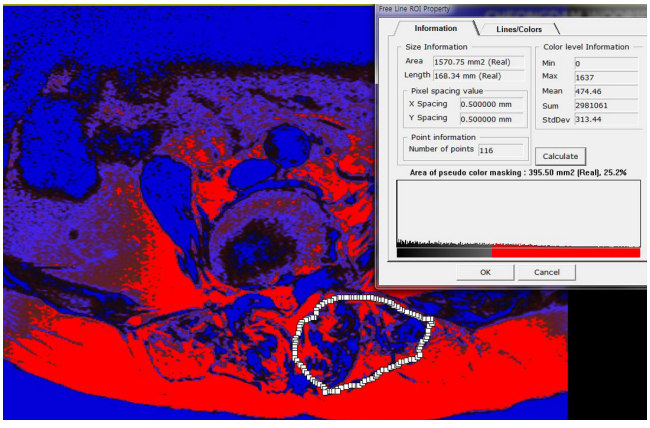

Fig. 4. The calculation of fatty infiltration (FI) of the paraspinal muscles using pseudo-color mapping technique.

2. Cohorts

The patients were divided into 2 groups based on their initial/prewalk C7–S1 SVA measurement. The first group with C7–S1 SVA ≤4 cm and PT >20° was termed as compensated sagittal deformity (CSD) while the second group was considered to have a decompensated sagittal deformity (DSD) with a C7–S1 SVA >4 cm and PT >20°.

3. Statistical Analysis

Student t-tests were used to compare variables between groups. Pearson correlation coefficients were used to assess correlations between changes in sagittal radiographic parameters after walking 10 minutes and variable of muscles within each group. Multiple regression analysis was used to analyze predictive variables for changes of sagittal parameters. A p-value of <0.05 defined statistical significance. All analyses were performed using SPSS

Table 1. Comparison between spinopelvic parameters of initial standing versus 10 minutes after walking in sagittal compensated balance (n = 24) and imbalance (n = 121) groups

Variable	Compensated sagittal deformity (n = 24)			Decompensated sagittal deformity (n = 121)		
	Initial	10 min	p-value	Initial	10 min	p-value
PT	35.8 ± 9.2	31.2 ± 13.2	0.006*	34.2 ± 10.8	29.9 ± 11.3	< 0.001*
LL	-22.0 ± 21.5	-8.6 ± 25.4	< 0.001*	-7.9 ± 20.4	-1.3 ± 22.6	< 0.001*
TK	11.8 ± 14.2	12.2 ± 15.5	0.828	8.6 ± 14.8	12.0 ± 15.1	< 0.001*
PI–LL mismatch	32.8 ± 17.1	46.3 ± 23.3	< 0.001*	46.8 ± 20.7	53.4 ± 23.4	< 0.001*
SVA	18.0 ± 11.2	139.5 ± 90.6	< 0.001*	110.5 ± 61.8	208.2 ± 97.2	< 0.001*

Values are presented as mean ± standard deviation.
PT, pelvic tilt; LL, lumbar lordosis; TK, thoracic kyphosis; PI, pelvic incidence; SVA, sagittal vertical axis.
*p < 0.05, statistically significant differences.

ver. 14.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

One hundred forty-five patients (mean 68.1 ± 5.9 years old, 136 females) were included. Initial mean SVA was 1.8 cm for CSD and 11 cm for DSD (p < 0.01). After walking, significant deteriorations in SVA were observed, decreased PT minus LL was observed (p < 0.01) in CSD without significant change in TK, while all the sagittal parameters in DSD were significantly deteriorated (Table 1). Patients with DSD had significantly poorer muscle quality (i.e., less CSA, SI) in the PSM at the thoracolumbar junction and lower lumbar spine, compared to the patients with CSD. FI and LM were not different between the groups. DSD group had significantly lower value of BMD (p = 0.031) (Table 2).

In CSD patients, sagittal decompensation correlated with muscle quality, i.e., decreases in LL (ΔLL) correlated with CSA of PSM/VB at L4 (r = -0.412, p = 0.046), increases in TK (ΔTK) correlated with CSA of PSM/VB at T12 (r = 0.477, p = 0.018)). ΔSVA and ΔPT correlated with FI at L4 (r = 0.577, p = 0.003 and r = -0.407, p = 0.048, respectively) (Table 3). Multiple regression analysis showed that CSA of PSM at L4 was predictive of ΔLL (R^2 = 0.170, p = 0.046) and FI at L4 was predictive of ΔSVA (R^2 = 0.330, p = 0.003) and ΔPT (R^2 = 0.166, p = 0.048) in CSD. For DSD patients, there were weak correlations (-0.3 < r < -0.1) between changes in sagittal parameters and PSM FI, BMD (Table 4).

Table 2. Comparison demographic factors and parameters of paraspinal muscle degeneration in sagittal compensated balance (n = 24) and imbalance (n = 121) groups

Variable	Compensated sagittal deformity (n = 24)	Decompensated sagittal deformity (n = 121)	p-value
Age (yr)	66.7 ± 7.1	68.2 ± 5.7	0.230
BMD	-1.6 ± 1.2	-2.3 ± 1.4	0.031*
Psoas/VB	30.6 ± 7.8	31.0 ± 10.2	0.849
PSM/VB at T12	111.9 ± 27.8	92.8 ± 35.8	0.015*
PSM mean at T12	529.8 ± 1,106.2	279.8 ± 273.0	0.030*
PSM SI at T12	279.8 ± 503.1	153.1 ± 117.6	0.015*
FI T12	46.1 ± 20.8	42.4 ± 22.6	0.459
LM/VB T12	60.5 ± 28.2	53.9 ± 32.9	0.366
PSM/VB at L4	106.8 ± 30.9	93.8 ± 34.2	0.085
PSM mean at L4	435.9 ± 763.3	260.8 ± 184.5	0.027*
PSM SI at L4	266.2 ± 408.1	168.2 ± 110.2	0.024*
FI L4	39.1 ± 16.2	40.9 ± 18.8	0.663
LM/VB L4	64.9 ± 23.8	55.7 ± 28.6	0.149

Values are presented as mean ± standard deviation.
BMD, bone mineral density; VB, vertebral body; PSM, paraspinal muscle; SI, signal intensity; FI, fatty infiltration; LM, lean mass; CSA, cross-sectional area.
*p < 0.05, statistically significant differences.

DISCUSSION

The spinal alignment is the consequence morphology of the vertebra and the discs as well as the forces acting on them. The compressive forces acting on the spine ae countered by the vertebrae as well as the discs. However, as the disc degenerates with advancing age, there is an imbalance of the forces acting on the spine which may result in kyphosis (asymmetric disc degeneration in sagittal plane) or scoliosis (asymmetric disc degeneration in coronal plane) or kypho-scoliosis.[4] The body tries to compensate for this imbalance with various mechanisms such as discopathies, retrolisthesis, changes in the pelvic tilt, knee flexion, and the use of paraspinal muscular forces to improve overall sagittal alignment, in order to maintain center of gravity

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Table 3. Correlation between paraspinal muscle parameters and changes of sagittal parameters after 10 minutes walking in overall, compensated sagittal deformity group

Variable	Change in sagittal spine deformity parameters									
	ΔPT		ΔLL		ΔTK		ΔSVA		ΔPI–LL	
	Correlation coefficient	p-value	Correlation coefficient	p-value	Correlation coefficient	p-value	Correlation coefficient	p-value	Correlation coefficient	p-value
Age	-0.084	0.684	-0.145	0.480	0.182	0.374	-0.081	0.693	-0.145	0.480
BMD	0.281	0.183	-0.323	0.124	0.418	0.042*	-0.288	0.173	-0.323	0.124
Psoas	0.185	0.386	-0.078	0.718	0.167	0.437	-0.117	0.585	-0.078	0.718
PSM/VB at T12	-0.105	0.626	-0.352	0.092	0.477	0.018*	-0.139	0.517	-0.352	0.092
PSM mean at T12	0.042	0.845	0.189	0.378	0.065	0.762	0.254	0.231	0.189	0.378
FI T12	0.291	0.168	-0.042	0.847	-0.183	0.391	-0.257	0.224	-0.042	0.847
PSM area at L4	-0.134	0.534	-0.245	0.249	0.221	0.300	-0.145	0.500	-0.245	0.249
PSM/VB at L4	-0.170	0.428	-0.412	0.046*	0.324	0.123	-0.252	0.234	-0.412	0.046*
PSM mean at L4	0.054	0.802	0.159	0.459	0.085	0.692	0.299	0.281	0.159	0.459
FI L4	0.407	0.048*	-0.342	0.102	0.191	0.372	-0.577	0.003*	-0.342	0.102

PT, pelvic tilt; LL, lumbar lordosis; TK, thoracic kyphosis; SVA, sagittal vertical axis; PI, pelvic incidence; BMD, bone mineral density; PSM, paraspinal muscle; VB, vertebral body; FI, fatty infiltration.
*$p < 0.05$, statistically significant differences.

Table 4. Correlation between paraspinal muscle parameters and changes of sagittal parameters after 10 minutes walking in overall, decompensated sagittal deformity groups

Variable	Change in sagittal spine deformity parameters									
	ΔPT		ΔLL		ΔTK		ΔSVA		ΔPI–LL	
	Correlation coefficient	p-value	Correlation coefficient	p-value	Correlation coefficient	p-value	Correlation coefficient	p-value	Correlation coefficient	p-value
Age	-0.003	0.976	0.078	0.378	-0.095	0.283	-0.043	0.624	0.078	0.378
BMD	0.137	0.170	-0.203	0.041*	0.008	0.940	-0.235	0.017*	-0.203	0.041*
Psoas	0.175	0.055	0.049	0.591	-0.051	0.581	-0.016	0.864	0.049	0.591
PSM area at T12	0.047	0.612	-0.131	0.153	-0.137	0.135	-0.192	0.036*	-0.131	0.153
PSM/VB at T12	0.006	0.950	-0.207	0.023*	-0.142	0.121	-0.164	0.072	-0.207	0.023*
PSM mean at T12	-0.067	0.466	-0.093	0.315	-0.024	0.791	0.031	0.739	-0.093	0.315
FI T12	-0.092	0.342	-0.026	0.785	-0.040	0.677	0.031	0.745	-0.026	0.785
PSM area at L4	-0.036	0.695	-0.081	0.376	-0.035	0.705	-0.033	0.723	-0.081	0.376
PSM/VB at L4	-0.062	0.503	-0.168	0.065	-0.040	0.662	-0.049	0.591	-0.168	0.065
PSM mean at L4	-0.184	0.043*	-0.063	0.492	-0.029	0.752	0.116	0.205	-0.063	0.492
FI L4	-0.077	0.427	-0.079	0.414	0.058	0.549	-0.021	0.827	-0.079	0.414

PT, pelvic tilt; LL, lumbar lordosis; TK, thoracic kyphosis; SVA, sagittal vertical axis; PI, pelvic incidence; BMD, bone mineral density; PSM, paraspinal muscle; VB, vertebral body; FI, fatty infiltration.
*$p < 0.05$, statistically significant differences.

and horizontal gaze.[5,6] Barrey et al.[5] classified 3 types for the sagittal alignment in adult spinal deformities (ASDs): balanced, balanced with compensatory mechanisms, and imbalanced. The compensatory mechanisms can be intraspinal or extraspinal in the latter 2 groups. Intraspinal mechanisms include pelvic retroversion, thoracic or adjacent lumbar hyperextension, retrolisthesis at the immediate adjacent level in the lumbar spine, and cervical hyperlordosis. Extraspinal mechanisms include knee and hip flexion and extension at the ankle.

We published in 2017, that there is a change in sagittal spinal

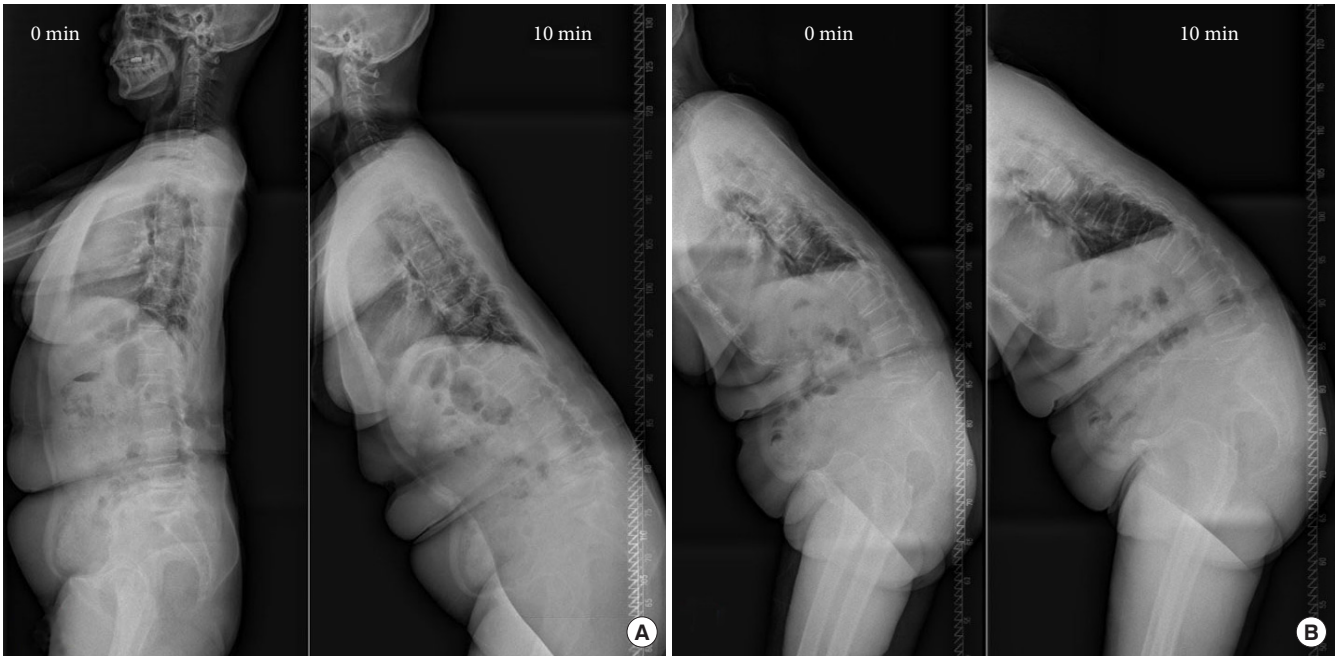

Fig. 5. (A) Lateral full-length spine radiograph of a patient with compensated sagittal spinal deformity at rest and after walking for 10 minutes. (B) Lateral full-length spine radiograph of a patient with decompensated sagittal spinal deformity at rest and after walking for 10 minutes.

alignment after 10-minute walking in compensated/uncompensated ASDs after setting of fatigue (Fig. 5A, B).[3] In the current study, we went further to investigate the correlation of paraspinal and psoas muscle mass on the changes in sagittal spinal alignment before and after setting of fatigue.

A reduction of muscle CSA and presence of FI are considered as indicators of muscle degeneration.[7] We used the CSA of iliopsoas and PSMs (after deducting the area of FI from overall CSA) at thoracolumbar and lower lumbar level as a measure to quantify the muscle mass. The precise definition of the muscle quality is the amount of force generated by each volumetric unit of muscle tissue. However, there is no consensus as to how measure this force generated/strength.[8] Although the relationship between the muscle mass and muscle strength is not linear,[9] CSA is considered as a reliable indicator for the evaluation of the muscle strength in the maintenance of sagittal spinal balance.[10]

Yagi et al.[6] studied the effect of paraspinal and iliopsoas muscles in the maintenance of the sagittal balance in degenerative lumbar scoliosis patients. They recruited 60 patients each of lumbar canal stenosis and lumbar degenerative scoliosis. They found that the multifidus and iliopsoas areas were significantly smaller in the scoliotic patients. Also, multifidus CSA was correlated with progression of kyphosis at the unfused thoracic vertebrae in scoliosis group. In our study, we excluded patients with more than 30° coronal deformity and mainly focused with the sagittal plane deformity. Similar to abovementioned study we found significantly lower mass in the paraspinal and psoas muscle in DSD group as compared to CSD group.

PSM atrophy has been associated with multitudes of spinal disorders like lumbar canal stenosis, isthmic spondylolisthesis, facet arthropathy, degenerative lumbar kyphosis.[11] Moreover, it is also associated with worse postsurgery outcomes.[11] The most important part of our study was exaggerating the deformity by nullifying the role of the compensatory muscular forces, thereby unmasking the pivotal role played by the paraspinal and iliopsoas muscles in compensating the sagittal spinal deformity.

The average age in the entire study population was 68.1 years (66.7 and 68.2 years in CSD and DSD, respectively) which corresponds to existing literature of ASD which affects mainly the old persons. Cho et al.[12] studied the relationship between BMD and sagittal imbalance in elderly and found that there was no statistically significant correlation between the two. However, Mika et al.[13] demonstrated that decreased BMD influences TK but despite decreased BMD, if the back muscles are sufficiently strong, spinal deformities do not occur. The BMD in our study was significantly lower in DSD group as compared to CSD group and most of the parameters measuring muscle mass at thoraco-

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lumbar as well as lower lumbar level were significantly lower in DSD group. This may indicate an association between the BMD, PSM mass, and severity of sagittal imbalance.

Takahashi et al.[14] studied the correlation of back muscles and knee extensors with the compensatory mechanism of sagittal alignment in community-dwelling elderly persons. They found that thoracic kyphosis demonstrated a negative correlation with back muscle strength and positive correlation with vertebral fracture. Back muscle strength was important for the decrease in thoracic kyphosis, and knee extensor strength was associated with pelvic tilt. In our study, we focused on back and psoas muscle mass and its correlation with sagittal alignment. In our study, there was no significant change in thoracic kyphosis after 10-minute walk in CSD group while the change in DSD was significant. In order to nullify the individual variation in the muscle mass and body size, we used CSA/VB as a measure for comparison. We found that the change in thoracic kyphosis correlated with PSM/VB at T12 level in CSD group, implying that the PSM mass at thoracolumbar junction was an important determinant affecting changes thoracic kyphosis.

In the CSD group, after a 10-minute walk, there was a significant derangement of spinopelvic parameters which included lumbar lordosis (reduced from -22.0 to -8.6), PI–LL mismatch (increased from 32.8 to 46.3), pelvic tilt (reduced from 35.8° to 31.2°), and the SVA (increased from 18.0 mm to 139.5 mm). The change in thoracic kyphosis was not significant. These findings clearly suggest that as the activity causes muscles to fatigue, the compensatory effect of the muscular activity on sagittal balance of the spine is lost and the spinopelvic parameters derange. The findings were similar in DSD group which included derangement of lumbar lordosis (reduced from -7.9 to -1.3), PI–LL mismatch (increased from 46.8 to 53.4), pelvic tilt (reduced from 34.2° to 29.9°), SVA (increased from 110.5 mm to 208.2 mm), and thoracic kyphosis (increased from 8.6° to 12.0°) with statistically significant derangement in all the spinopelvic parameters in this group. However, in DSD group the parameters were already abnormal to begin with, which further deranged after the 10-minute walk. In the DSD group, significantly poorer muscle quality (i.e., less CSA, SI) was observed at the thoracolumbar junction and lower lumbar spine. The changes in the sagittal parameters after 10 minutes walk and the PSM MRI parameters at T12 and L4 were not significantly correlated in this group as muscle quality was already poor. Hence, it had little contribution in maintaining the sagittal alignment at rest in these patients.

The CSA of PSM at T12 and L4 level was significantly lower in DSD than CSD group suggesting the patients in whom the muscle mass was lower could not have compensated their deformity using PSMs while patients with higher muscle mass could compensate for the deformity. This also means that the patients in whom the PSM mass is lower are unlikely to show compensation and will have a decompensated deformity. On the other hand, patients with better muscle mass will show compensation of the deformity. This emphasizes the need to have strategies for extensive preoperative physiotherapy program in order to improve PSM mass before a patient with sagittal spinal deformity is operated. This will help in compensating the deformity of the patient at rest and also, this will have better outcome for the patients in the postoperative period with improved sagittal balance.

The changes in some of the sagittal parameters in CSD group had correlation with the parameters measuring muscle quality. For example, PSM/VB at T12 correlated with the change in thoracic kyphosis, suggesting that reduced muscle mass at T12 level can result in thoracic decompensation in the form of increased kyphosis. PSM/VB at lower lumbar level (L4) correlated significantly with the change in the lumbar lordosis and changes in PI–LL mismatch. This means that the reduction of PSM mass at L4 level may result in lumbar decompensation by reduction of lordosis as well as abnormal pelvic alignment. Also, FI at L4 level significantly correlated with changes in pelvic tilt as well as changes in SVA indicating that FI at L4 can predict decompensation of SVA and PT.

In the DSD group, the paraspinal as well as psoas muscle SA and FI did not correlate significantly with the changes in sagittal parameters. Thus, the changes of sagittal parameters after walking were less related to muscle status in DSD. As already degenerated muscles are causes of initial decompensation of DSD, their roles in maintaining upright position are insignificant. This emphasizes the fact that since the muscle quality is poorer and the sagittal balance is already abnormal at rest in these patients, the remaining muscle mass doesn't have bearing on how much more the decompensation will occur after the 10-minute walk.

These observations can be extrapolated for the clinical practice, whereby a patient with CSD will benefit significantly more by strengthening the PSMs by undergoing physiotherapy, while the benefit of muscle strengthening on sagittal alignment in decompensated patients may be limited. Nonetheless, as muscle and body balance training and maintenance of spinal sagittal alignment can lead to prevention of fall in elderly patients,[15] we believe that this should become a part of treatment strategy in

all the elderly patients with sagittal imbalance.

There are some limitations of our study. As muscle fatigue is a dynamic factor, whether patient walked before coming to clinic and whether the patient was examined in the morning or in evening when the fatigue might have set in due to the activities throughout the day may bring about some variation. However, we had standardized the 10-minute duration for all the patients whereby bringing uniformity to the evaluation. We also have not included the coronal plane malalignment and assessment for the parameters of lower limb in compensation of the deformity. We did not measure muscle strength using hand grip as a baseline for comparing the muscle strength amongst the patients as the muscle volume alone may not perfectly correlate with muscle strength. In addition, we could not include the effect of compensation by the lower limbs in our study as we used 36-inch radiographs which cannot accommodate the entire skeleton starting from basiocciput up to the legs. However, with recent availability of full-length standing radiographs using EOS imaging, this type of study can become feasible in future. Also, a single spinal neurosurgeon measured the radiological parameters using computer-based software, which can be regarded as a limitation. Lastly, we did not include quality of life parameters, as the focus of this study was mainly on radiological parameters. However, we calculated PI–LL mismatch and SVA which are known to correlate well with their impact on health-related quality of life parameters and thus can indirectly indicate the patients' disability.

CONCLUSION

PSM quality in adults with spinal deformity correlates with patients' ability to maintain an upright posture and sagittal decompensation after walking for 10 minutes. As such, patients who present with compensated sagittal deformities may benefit from dedicated exercise programs to maintain PSM quality/strength so as to prevent sagittal decompensation.

CONFLICT OF INTEREST

The authors have nothing to disclose.

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