Natural history and aggravating factors of sagittal imbalance in marked sagittal deformity compared with mild to moderate sagittal deformity

A prospective cohort study

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Abstract

Sagittal imbalance is a multifactorial complex deformity that can arise from a variety of causes such as spinal stenosis, sarcopenia, vertebral fracture, and neuromuscular diseases. Furthermore, there is lack of research regarding spinal and general conditions that precede the development of sagittal imbalance. Our aim was to evaluate aggravating factors, such as natural history, for sagittal imbalance in a cohort comprising elderly individuals by conducting various examinations.

We recruited 96 participants who had a sagittal vertical axis (SVA) larger than 50 mm in a sagittal imbalance study. Finally, 69 participants were followed up and enrolled this study after 2 years. We evaluated full spine radiographs, magnetic resonance imaging (MRI), bone mineral density, and health-related quality of life from patients survey and analyzed factors associated with aggravation of sagittal imbalance. Aggravation was defined by an SVA > 30 mm and T1 pelvic angle (T1PA) > 3° in the third year compared to SVA and T1PA values of the first year.

Eighteen participants of the follow-up group had a sagittal imbalance aggravation. According to the deformity severity in the first-year evaluations, the marked deformity group (38 participants) defined as Schwab classification had 11 (28.9%) participants presenting with sagittal imbalance aggravation. These participants had larger mean values of Schwab sagittal modifiers and T1PA compared with the nonaggravation participants. Logistic regression analysis showed a higher pelvic incidence (PI) (OR = 1.201, 95% CI = 1.015–1.422, P = .033) and a small multifidus (MF) volume (OR = 0.991, 95% CI = 0.983–1.000, P = .043) correlated with sagittal imbalance aggravation.

From the follow-up group, 18 (26%) subjects of total 69 participants presented a deteriorated sagittal imbalance. A higher PI and smaller MF volume correlated with the aggravation of sagittal imbalance. We should consider that high PI and small MF volume are associated with aggravation of sagittal imbalance.

Abbreviations: BMD = bone mineral density, BMI = body mass index, CSA = cross-sectional area, HRQoL = health-related quality of life, KESICS = Korean elderly sagittal imbalance cohort study, LL = lumbar lordosis, MF = multifidus, MMSE = mini-mental state examination, MRI = magnetic resonance imaging, ODI = Oswestry Disability Index, PI = pelvic incidence, PT = pelvic tilt, SF-36 = Short Form -36 Health Survey, SS = sacral slope, SVA = sagittal vertical axis, T1PA = T1 pelvic angle, TK = thoracic kyphosis, VAS = visual analog scale.

Keywords: adult spinal deformity, oswestry Disability Index, sagittal imbalance, scoliosis Research Society-Schwab classification, short form health survey

1. Introduction

Over the past 2 decades, awareness of sagittal imbalance has increased. As a consequence, recognition of the importance of restoring sagittal balance in the surgical treatment of adult spinal deformity has also increased.[1] Because sagittal imbalance has a significant relation with health-related quality of life (HRQoL) deterioration and surgical outcome in symptomatic adults with degenerative spinal disorders, correction of sagittal imbalance is important for achieving good surgical outcomes and HRQoL.[2,3]

Sagittal imbalance of adult spinal deformity has various etiologies[4] and can develop gradually over many years. However, sagittal alignment does not deteriorate in all individuals as they get older.[2,4]

Because sagittal imbalance is a multifactorial complex deformity that can arise from a variety of causes such as spinal stenosis, sarcopenia, vertebral fracture, osteoporosis, body mass index (BMI), and neuromuscular diseases; therefore, it is very difficult to determine predisposing factors of an aggravating sagittal imbalance. Furthermore, there is lack of research regarding spinal and general conditions that precede the development of sagittal imbalance. Therefore, the purpose of this study was to evaluate the natural course and aggravating factors of sagittal imbalance associated with various spinal and general conditions in a Korean elderly cohort with sagittal imbalance.

2. Methods

The institutional review board of our institute granted approval for this study (approval number CNUH-2016-127).

This prospective cohort study is a longitudinal cohort study that follows over 2 years a group of sagittal imbalance individuals to determine aggravating factors of sagittal imbalance.

In the first year, we recruited volunteers who were older than 65 years and exhibited a stooping posture in daily living. Before enrolling subjects, we checked the full spine radiograph to confirm that volunteers had a sagittal vertical axis (SVA) larger than 50 mm.[5,6] A total of 96 participants volunteered to participate (Fig. 1) and gave written consent for participation in this Korean elderly sagittal imbalance cohort study (KESICS). Exclusion criteria were age less than 65 years, previous spinal surgery, infection, malignancy, neuromuscular disease, bed-ridden state, or acute fracture.

During the first year, we examined the whole spine MRI, blood test, bone mineral density (BMD), visual analog scale (VAS), the Oswestry Disability Index (ODI), the Short Form -36 Health Survey (SF-36), and mini-mental state examination (MMSE). In the third year, we resumed the tests on the patients.

We measured the SVA, pelvic incidence (PI), pelvic tilt (PT), sacral slope (SS), lumbar lordosis (LL), thoracic kyphosis (TK), and T1 pelvic angle (T1PA), as well as the SRS-Schwab sagittal modifiers.[5,6] To evaluate competent muscles, we measured the cross-sectional area (CSA) of the multifidus (MF) muscle, erector spinae muscle, and psoas muscle at the lumbar 4/5-disc level (Fig. 2). We evaluated muscle fatty change using the Goutallier classification system.[7] Furthermore, we confirmed the whole lumbar disc degeneration using Pfirrmann grade,[8] canal stenosis,[9] foraminal stenosis,[10] old compression fracture, and spondylolisthesis in the thoracolumbar spine through whole spine MRI.

To our best knowledge, there is no definite value is defined as aggravation of sagittal imbalance. Therefore, in the third-year of follow-up, we defined aggravation of sagittal imbalance markers as increase of more than 3 degrees in T1PA and increase of more than 30 mm in SVA compared to values obtained the first-year evaluation regarding measurement error.

In the first year, we identified marked deformities and mild to moderate deformities utilizing the SRS-Schwab sagittal modifiers. Throughout the third year, we analyzed the natural history and aggravation of the sagittal imbalance according to deformity severity: marked deformity group vs mild to moderate deformity group.

All statistical analyses were performed using SPSS version 18.0 software (SPSS Inc., Chicago, Illinois, USA). Data analysis was conducted using the Mann–Whitney U test and the logistic regression analysis to compare results within the group. Statistical significance was defined as $P < .05$.

3. Results

From a total of 96 participants, 27 participants were not included in the third-year follow-up period (Fig. 1). The causes of follow-up loss were nursing hospital admission, dementia, hip fracture, and refusal to participate in the follow-up evaluation. Therefore, 69 participants were enrolled in the third-year examinations.
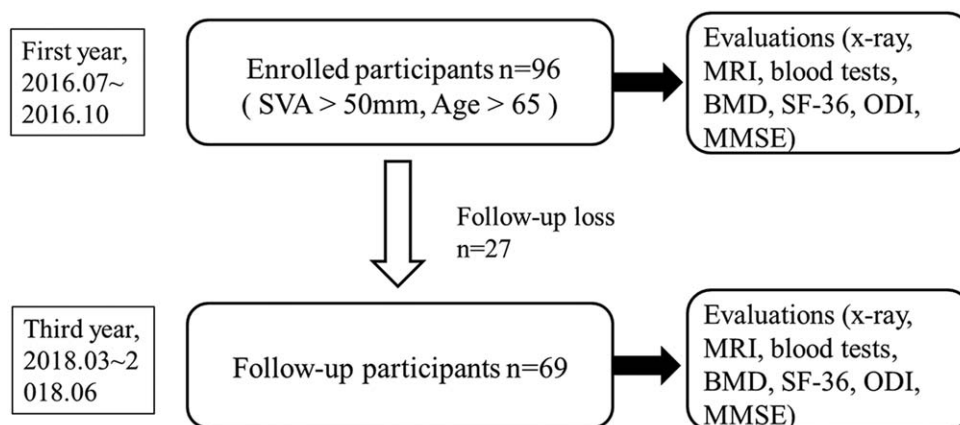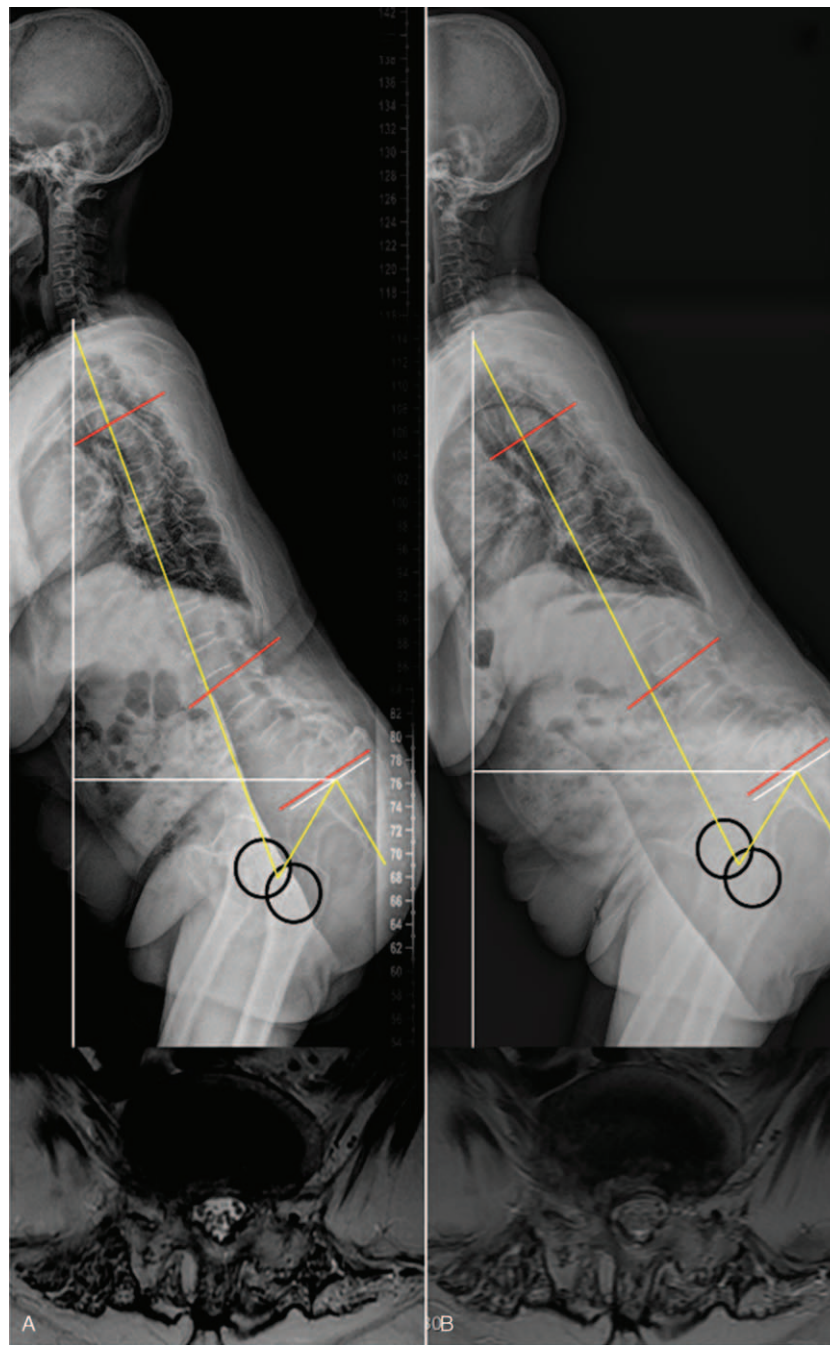

Figure 1. Participants enrolled in the prospective cohort study.

Figure 2. Lateral standing radiograph and lumbar MRI of a 73-year-old woman with sagittal imbalance. (A) Baseline (initial) image showing 49.3° T1PA, 30.1° PT, 69.8° PI, 37.3° SS, 1.9° TK, 12° LL and 208 mm SVA. Lumbar MRI at L4-5disc level showing Goutallier classification grade 4 (Cross sectional area of multifidus measured 326 mm2). (B) At 1 year after, follow-up, lateral standing radiograph showing aggravation of sagittal imbalance with 60° T1PA, 30.6° PT, 72.4° PI, 41.3° SS, 1.1° TK, 2.2° LL and 275 mm SVA.

Among 69 participants, 18 (26%) participants presented a deteriorated sagittal imbalance compared to the baseline. According to deformity severity in the first-year evaluations, the marked deformity group (38 participants) had 11 (28.9%) participants presenting with aggravation of the sagittal imbalance, while the mild to moderate deformity group (31 participants) had 7 (22.5%) participants with deteriorated sagittal imbalance (Table 1). In the marked deformity group, participants with an aggravation of sagittal imbalance had larger mean values of SVA, T1PA, PI, PT, and PI-LL compared with non-aggravation participants (Table 2). Fatty changes and muscle volume of paraspinal muscles were not statistically different between the aggravation group and nonaggravation group (Table 3). Pfirrmann grade for evaluating lumbar disc degeneration was similar between the 2 groups (Table 4). Lumbar spine central and foraminal stenosis grade, the number of spondylolisthesis, and old vertebral fractures were not different between the 2 groups (Table 5). SF-36, ODI, and MMSE in the

Table 1

Demographics of the marked deformity and mild to moderate deformity groups.

Mean (SD)	Marked DF (n=38)			Mild to moderate DF (n=31)		
	AG (n=11)	Non-AG (n=27)	P value	AG (n=7)	Non-AG (n=24)	P value
Age	69.91 (5.4)	70.63 (4.1)	.661	71 (2.7)	70.667 (3.8)	.834
Female (%)	10 (90.9)	24 (88.9)	.674	5 (71.4)	18 (75)	.600
Weight (kg)	54.18 (13.6)	56.56 (8.95)	.523	67.29 (7.4)	59.25 (8.2)	.033*
Height (cm)	148.64 (5.98)	15.56 (7.3)	.446	155.86 (5.27)	154.54 (8.98)	.717
BMI	25.04 (4.55)	24.90 (2.8)	.910	27.8 (4.03)	24.6 (2.96)	.127
BMD (FN)	−1.96 (0.74)	−1.46 (0.77)	.052	−1.6 (0.58)	−1.71 (0.81)	.672
Osteoporosis, n (%)	3 (27.3)	3 (11.1)	.329	0	4 (16.7)	.550

* Statistically significant difference by Mann–Whitney U test (P< .05).
AG = aggravation, BMD = bone mineral density, BMI = body mass index, DF = deformity, FN = femoral neck.

Table 2

Radiologic parameters of the marked deformity and mild to moderate deformity groups.

Mean (SD)	Marked DF (n=38)			Mild to moderate DF (n=31)		
	AG (n=11)	Non-AG (n=27)	P value	AG (n=7)	Non-AG (n=24)	P value
SVA, mm	197.88 (148.37)	103.15 (71.41)	.009*	100.19 (48.14)	44.35 (43.39)	.002*
T1PA, °	48.19 (10.41)	36.32 (10.89)	.001*	22.22 (8.15)	18.77 (7.29)	.340
PI, °	62.40 (8.78)	54.25 (10.75)	.030*	49.82 (5.23)	51.37 (10.89)	.610
PT, °	37.68 (7.77)	32.16 (8.82)	.032*	16.12 (7.07)	20.24 (7.53)	.211
SS, °	26.10 (10.11)	23.13 (10.0)	.421	32.05 (6.05)	31.37 (8.30)	.815
LL, °	11.30 (12.46)	17.57 (17.35)	.223	31.4 (9.09)	39.21 (2.06)	.164
TK, °	13.36 (17.92)	13.21 (14.21)	.980	21.28 (11.86)	26.68 (13.44)	.187
PI-LL, °	51.10 (13.98)	36.68 (16.45)	.012*	18.42 (12.40)	12.15 (20.71)	.334

* Statistically significant difference by Mann–Whitney U test (P< .05).
AG = aggravation, DF = deformity, LL = lumbar lordosis, PI = pelvic incidence, PT = pelvic tilt, SS = sacral slope, SVA = sagittal vertical axis, T1PA = T1 pelvic angle, TK = thoracic kyphosis.

aggravation and nonaggravation groups revealed similar mean values (Table 6). Aggravating factors of sagittal imbalance in marked deformity were high PI and a small volume of multifidus muscle assessed by using logistic regression analysis (Table 7).

Table 3

Muscle characteristics of the aggravation group and noaggravation group.

Mean (SD)	AG (n=18)	Non-AG (n=51)	P value
Goutallier grade of MF	2.39 (0.916)	2.24 (10.23)	.521
Goutallier grade of ES	2.24 (0.913)	1.97 (0.823)	.213
Goutallier grade of Psoas	2.29 (0.867)	2.00 (0.707)	.148
CSA of MF, mm^2	656.21 (179.98)	767.03 (239.01)	.378
CSA of ES, mm^2	1789.13 (1419.84)	1567.55 (289.47)	.412
CSA of Psoas, mm^2	2301.45 (454.94)	2361.24 (382.28)	.570

AG = aggravation, CSA = cross sectional area, ES = erector spinae muscle, MF = multifidus muscle, Psoas = psoas muscle.

Table 4

Pfirrmann grade for evaluating disc degeneration between aggravation and nonaggravation groups.

Level, mean (SD)	AG (n=18)	Non-AG (n=51)	P value
L1/2	2.32 (0.962)	2.79 (1.207)	.076
L2/3	2.68 (0.989)	3.10 (0.860)	.074
L3/4	2.74 (1.057)	3.17 (0.848)	.074
L4/5	2.95 (0.985)	3.38 (1.083)	.093
L5/S1	2.79 (1.189)	3.07 (1.438)	.387

AG = aggravation, L = lumbar spine, S = sacrum.

4. Discussion

Recently, many studies and concepts regarding spine sagittal balance have emerged.[3,5,11] Sagittal imbalance is correlated to HRQoL and disability significantly regardless of spinal surgery.[2,3] Sagittal balance should be considered when spinal surgery is required. The natural history of sagittal imbalance has not been well known due to a lack of prospective cohort studies.

Table 5

Spinal diseases in the aggravation and non-aggravation groups.

Disease, mean (SD)	AG (n=18)	Non-AG (n=51)	P value
Central stenosis grade	1.95 (0.769)	1.97 (0.823)	.926
Foraminal stenosis grade	1.79 (1.143)	1.69 (1.039)	.714
Spondylolisthesis, n (%)	11 (28.9)	13 (44.8)	.207
Old vertebral fracture, n (%)	16 (42.1)	11 (39.3)	1.000

AG=aggravation.

Table 6

Findings of SF-36, ODI, and MMSE in aggravation and non-aggravation groups.

Mean (SD)	AG (n=18)	Non-AG (n=51)	P value
SF-36, PCS	29.02 (13.78)	23.97 (7.81)	.077
SF-36, MCS	41.19 (14.20)	38.75 (15.69)	.501
ODI	19.15 (7.60)	20.26 (6.75)	.530
MMSE	25.64 (3.00)	25.93 (3.95)	.728

AG = aggravation, MCS = mental component score, MMSE = mini-mental state examination, ODI = Oswestry disability index, PCS = physical component score, SF-36 = Short Form-36 Health Survey.

Table 7

Aggravating factors of sagittal imbalance in the marked deformity group.

	OR	95% CI	P value
SVA	1.050	0.975–1.131	.119
PT	1.438	0.914–2.261	.116
PI	1.201	1.015–1.422	.033*
PI-LL	1.091	0.965–1.232	.164
T1PA	0.999	0.996–1.001	.310
CSA of MF	0.991	0.983–1.000	.043*
CSA of ES	1.009	1.000–1.018	.146
CSA of Psoas	1.005	0.996–1.015	.267

* Statistically significant difference by logistic regression analysis ($P < .05$).
CI = confidence interval, CSA = cross sectional area, ES = erector spinae muscle, LL = lumbar lordosis, MF = multifidus muscle, OR = odds ratio, PI = pelvic incidence, Psoas = psoas muscle, PT = pelvic tilt, SVA = sagittal vertical axis, T1PA = T1 pelvic angle.

Our study differs from previous cross-sectional studies regarding the correlation between sagittal imbalance and HRQoL with disability.[1,2,4] In our study, to evaluate the natural course and aggravation of sagittal imbalance, we recruited participants aged more than 65 years and who had an SVA of more than 50 mm. We conducted a follow up at 2 years without administering treatment. From the 69 participants of the follow-up, 18 (26%) participants presented a deteriorated sagittal imbalance. According to deformity severity evaluations from the first year, the marked deformity group (38 participants) had 11 (28.9%) subjects exhibiting aggravation of the sagittal imbalance and the mild to moderate deformity group (31 participants) had 7 (22.5%) participants who presented deteriorated sagittal imbalance. The prevalence of aggravation of sagittal imbalance was not significantly different between the marked deformity group and the mild to moderate deformity group. However, in the marked deformity group, participants with severe sagittal deformity presented a more aggravated sagittal imbalance. Furthermore, we found that a higher PI and smaller MF volume were correlated to the aggravation of the sagittal imbalance.

Progressive loss of lumbar lordosis results from hypertrophic facet joint arthritis, disc degeneration, bone remodeling with bony spurs, vertebral compression fracture, and atrophy of extensor muscles. Progressive lumbar kyphosis results in a progressive development of a global sagittal imbalance.[4,12] In our study, we evaluated causes of sagittal imbalance such as central stenosis, foraminal stenosis, disc degeneration, vertebral compression fracture, and characteristics of muscles; no significant difference was observed between the aggravation and nonaggravation of sagittal imbalance groups. Because all participants already had a sagittal deformity, there was no significant difference regarding the aggravation of sagittal imbalance. The definition of aggravation of sagittal imbalance remains unclear, as there are no reference and criteria for evaluation of the deterioration of sagittal imbalance. Previous studies reported the standard deviation of T1PA was maximum 1.70 degrees.[13] The measurement of SVA was more reliable than that of PI-LL and PT among SRS-Schwab sagittal modifiers because of exclusion of the 2 femoral heads. Therefore, we defined the aggravation of sagittal imbalance as increase of more than 3 degrees in T1PA and increase of more than 30 mm in SVA compared to the baseline as sufficient criteria.[6,13]

Previous studies have suggested that increasing the PI can be related to the progressive loss of LL with age.[14,15] Increase in PT

indicates that pelvic retroversion appears before SVA changes positively.[16] In this study, a higher PI, higher PT, and higher PI-LL in the aggravation group of marked deformity were found compared with the nonaggravation group. The PI-LL mismatch is one of the main causes of sagittal imbalance.[17] Kim et al[18] stated that PI-LL mismatch is associated with pathologic changes and not the normal aging process of the spine. Among these parameters, we found that a higher PI is an aggravating factor of sagittal imbalance.

We investigated the cross-sectional area of the MF, ES, and psoas muscles at the L4/L5 level based on the result that the total back extensor strength is significantly affected by lumbar extensors muscle rather than by the thoracic extensors muscle.[19] Banno et al[20] found that the CSA of the MF muscle was significantly associated with all spinopelvic parameters in elderly patients with adult spinal deformity. Furthermore, the MF muscle has been identified to be the key back muscle for stabilizing the lumbar segments and maintaining lumbar lordosis.[21] In this context, the smaller CSA of MF muscle became one of the aggravating factors of sagittal imbalance.

This study has some limitations. First, it did not include a large number of subjects. Due to a relatively small number of subjects, our results cannot be generalized the aggravation of the sagittal imbalance. Further investigations of aggravating factors utilizing a larger number of subjects will be performed in the future. Second, the follow-up period of this study may be too short to evaluate the natural history and aggravation of sagittal deformity. A longer follow-up cohort study is needed.

5. Conclusion

Among 69 participants who underwent follow-up, 18 (26%) participants presented with deteriorated sagittal imbalance. According to the deformity severity assessment in the first-year evaluations, the marked deformity group (38 participants) had 11 (28.9%) participants with aggravation of sagittal imbalance. A higher PI and smaller MF volume correlated with the aggravation of sagittal imbalance. We should consider that high PI and small MF muscle volume are associated with the aggravation of sagittal imbalance.

Author contributions

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