Vertebral Fracture should be the Last Straw for Sagittal Decompensation in Adult Spinal Deformity; a Mean 13.6-year Longitudinal Study of Natural Course

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INTRODUCTION:

The prevalence of adult spinal deformity (ASD) is reported to be around 30% in the aging society, which disproportionately rises to 60% among the elderly population. ASD has been gathering medical and socioeconomic attentions, and majority of studies have been recommending surgical treatment despite with high complication rate and medical cost for major spinal interventions. Physicians should properly manage this common degenerative condition, however, the natural course of ASD has not been well-documented. ASD is associated with predominantly kyphotic spinal alignment, usually starts with decrease in lumbar lordosis (LL), which is compensated by pelvis and lower extremity joints to maintain global sagittal balance. Pelvic tilt (PT) increases within the limit of pelvic incidence (PI), and sagittal vertical axis (SVA) is maintained until the late stage. ASD patients with forward shift in SVA were associated with worse clinical and radiographic outcomes in recent studies, and worsening SVA was regarded as sagittal decompensation. Purpose of this study was to investigate radiographic and clinical factors associated with the worsening SVA, using a mean 13.6-year longitudinal cohort of adult volunteers with normal SVA at baseline radiographs.

METHODS:

Community-dwelling adult female volunteers were recruited from population register and subjected to upright entire spine radiographs and clinical evaluations performed by designated orthopaedic physicians and physical therapists. This study was a part of our ongoing longitudinal cohort study which has been approved by institutional review board. Standardized radiographic measurements included thoracic kyphosis (TK), LL, PT, PI, and SVA. SVA was classified as normal (<40mm), grade I (40-95mm), and grade II (>95mm), according to SRS-Schwab ASD classification. Degenerative spondylolisthesis (DS; >5% slip), degenerative lumbar scoliosis (DLS; >10°), and radiographic vertebral fracture (VF; diagnosed by quantitative and semi-quantitative methods) were also recorded. Clinical evaluations included isometric muscle strength of trunk flexor/extensor, quadriceps femoris, gluteus maximus, and iliopsoas; range of motion (ROM) of hip extension, internal/external rotation, knee flexion/extension, ankle dorsiflexion, active back extension (BET) from prone position; short form 36 physical component score (PCS), and visual analog scale (VAS) for back and knee pain. We also evaluated ambulatory kyphosis, which was a difference of trunk inclination angles between standing and walking, using surface markers and digital camcorder. Statistical analyses were performed using analysis of variance (ANOVA) and multiple regression analysis, and a p-value of less than 0.05 was used for significant difference.

RESULTS:

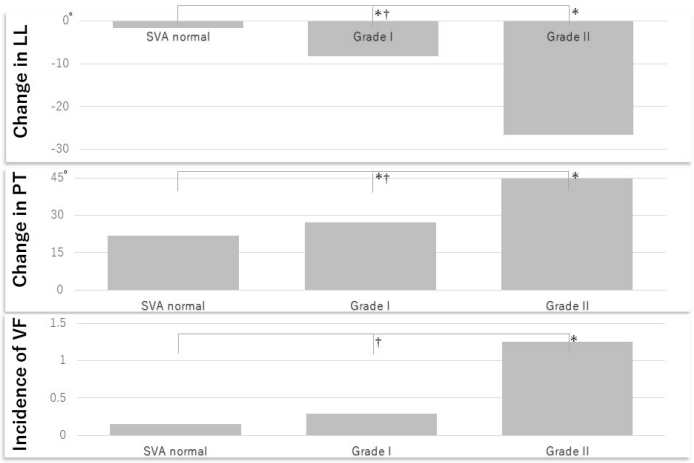
A final total of 115 volunteers with normal SVA at baseline were included and followed. Their mean age was 56.3±9.9 years at baseline and 69.9±5.8 years at the final follow-up, and mean follow-up period was 13.6±9.0 years. Longitudinal changes in radiographic parameters were as follows; TK 31.3 ±11.6° to 29.5±14.2°, LL 44.3±10.8° to 39.6±13.8°, PT 19.6±6.7° to 24.9±10.7°, PI 53.4±9.5° to 52.3±11.0°, and SVA 2.2±22.6mm to 26.7±32.7mm. SVA changed to grade I in 35 (30%) and to grade II in 5 (4%). Change in SVA was significantly associated with changes in LL (p<.0001), PT (p<.0001), and the number of VF (p=.0027, figure1). Multiple regression analysis including all radiographic parameters, occurrence of DS, DLS, VF, and muscle-joint parameters showed VF was the only significant factor related to the worsening of SVA. At baseline, subjects who later became SVA grade I-II exhibited worse BET (normal/grade I/II; 13.4/11.2/4.3cm, p=.0002) and ambulatory kyphosis (3.1/4.5/5.2°, p=.0433).

DISCUSSION AND CONCLUSION:

Sagittal decompensation leads to global sagittal imbalance, which has a significant relation with health-related quality of life deterioration. Sagittal imbalance in ASD patients could arise from a variety of causes such as spondylosis, osteoporosis, sarcopenia, pathological obese, spinal stenosis, and neuromuscular diseases. Most of the patients with ASD show compensatory mechanism to maintain upright spinal posture, and SVA deteriorates at the last stage of ASD indicating exhausted compensatory sources. Current study showed SVA maintained in 75 (65%) of subjects after 13.6 years and SVA shift was significantly related with the incidence of VF; 26% in grade I SVA and 60% in grade II SVA at the final follow-up. Subjects who later became SVA grade I-II have already exhibited substantial lumbopelvic compensation at baseline; lumbar joint contracture expressed by decrease in BET by 16 in grade I and 68% in grade II indicating prolonged lumbar kyphosis, and pelvic compensation expressed by increased ambulatory kyphosis (dTIA) by 45% in grade I and 68% in grade II. BET and dTIA could be useful as predictive factors for the development of SVA shift, and the importance of physical parameters should be emphasized under the circumstances of prolific static radiographic evaluations of ASD. Vertebral fracture might be inflicting kyphotic burden in already advanced and maximally-compensated ASD patients, which leads to sagittal decompensation with marked global malalignment. Management of osteoporosis to prevent vertebral fracture should be sound and adequate advice for elderly patients with spinal deformity. Limitation of this study

includes limited number of participants to acquire enough statistical power, and the onset of SVA shift and the timing of VFs are not clear. This is one of the longest observational studies of natural course of ASD, and still counting number of participants might provide further critical data for this heterogeneous, multifactorial deformity.

Figure 1



(P<0.05;∗ vs.normal, † vs.Grade II)