Degeneration of lumbar paravertebral muscles in lumbar degenerative kyphosis with dynamic sagittal imbalance: A case-control study.

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Research Article

Keywords: lumbar degenerative kyphosis, dynamic sagittal imbalance, paravertebral muscle; fat infiltration, muscle muscularity

Posted Date: May 13th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-510603/v1>

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Abstract

Background: The aim of this study was to probe the degeneration of lumbar paravertebral muscles in lumbar degenerative kyphosis (LDK) with dynamic sagittal imbalance (DSI).

Method: A total of 132 patients with LDK were enrolled in the study. According to the Δ SVA of the full-spine lateral radiographs before and after walking, enrolled patients were divided into two groups: DSI group (31 cases) and control group (42 case). Lumbar magnetic resonance imaging examination was taken for each subject. Fat infiltration area (FIA) and muscle muscularity of multifidus (MF) and erector spinae (ES) were quantitatively measured through Image J software. Independent-sample t test were performed for comparison of quantitative variables between two groups. P value < 0.05 was considered statistically significant.

Result: DSI group had lower muscle muscularity both in ES and MF than control group. ES muscularity at L2 level was 0.42 ± 0.08 in DSI group and 0.82 ± 0.17 in control group. ($p = 0.016$). ES muscularity at L4 level was 0.36 ± 0.11 in DSI group and 0.76 ± 0.22 in control group. ($p = 0.001$). MF muscularity at L2 level was 0.17 ± 0.08 in DSI group and 0.36 ± 0.07 in control group. ($p = 0.001$). MF muscularity at L4 level was 0.34 ± 0.18 in DSI group and 0.48 ± 0.14 in control group. ($p = 0.001$).

DSI group had higher FIA both in ES and MF than control group. ES FIA at L2 level was 0.50 ± 0.17 in DSI group and 0.31 ± 0.10 in control group. ($p = 0.023$). ES FIA at L4 level was 0.55 ± 0.27 in DSI group and 0.34 ± 0.07 in control group. ($p = 0.001$). MF FIA at L2 level was 0.63 ± 0.22 in DSI group and 0.36 ± 0.12 in control group. ($p = 0.001$). MF FIA at L4 level was 0.76 ± 0.31 in DSI group and 0.40 ± 0.19 in control group. ($p = 0.001$).

Conclusions: LDK patients with DSI suffered lower muscle muscularity and higher FIA both in ES and MF compared to control group. Our study revealed that the weakness of the paravertebral muscles plays an important role in DSI process, targeted paravertebral muscle strengthening training may be a potentially effective treatment for this disease.

Introduction

Lumbar degenerative kyphosis (LDK) is a common problem in elderly population, especially in the Asian population[1, 2]. It is considered as sagittal imbalance due to marked loss of lumbar lordosis or lumbar kyphosis[3]. This disease brings immense burdens to the social economy and families.

Takemitsu et al. classified LDK into 4 types and revealed the pathology as disc narrowing, collapsed vertebral bodies due to osteoporosis, and lumbar paravertebral muscle atrophy[3]. Yagi et al. redefined LDK as drop body syndrome (DBS) due to tremendous increased sagittal vertical axis (SVA)[4, 5]. These diseases share similar characteristics of decreased lumbar lordosis or lumbar kyphosis, decreased cross-sectional area (CSA) and increased fatty infiltration area (FIA) of lumbar paravertebral muscles[2, 4].

Bae J firstly described dynamic sagittal imbalance (DSI) in flat back syndrome[6]. Zhou et al. proposed a novel classification for DSI and its quantitative diagnostic criteria: pre-walk SVA < 40 mm and post-walk SVA-pre-walk SVA $\geq$ 20 mm(Δ SVA) after 10-min walk[7]. DSI is also the character of LDK[2, 8]. However, in clinical practice we found that some LDK patients could maintain sagittal balance persistently during walking ('compensated' group), whereas some LDK patients complained about obvious stooping trunk after walking/activity ('decompensated' group).

These previous studies used 3D-gait analysis and lateral full-spine radiographs to examine detailed changes in spinal sagittal parameters in DSI[6, 7, 9]. However, to the best of our knowledge, the differences between 'compensated' group and 'decompensated' group in paravertebral muscle degeneration have not been studied before.

Materials And Methods

A total of 132 patients with LDK were enrolled in the study between March 2018 and May 2020. (The detailed flowchart is shown in Fig. 1.) All enrolled patients with LDK were asked to take two sets of lateral full-spine standing radiographs at initiation and after a 10-min walk at their usual speed. Any kind of rest was prohibited in this 10-min walk test. Two well-trained spine surgeons would supervise each participant for the entirety of the 10-mins walk test[7]. Finally, 31 patients were included in the DSI group (Decompensated group).

Inclusion criteria[2, 7]: patients with LDK, pre-walk SVA < 40 mm and post-walk SVA-pre-walk SVA $\geq$ 20 mm(Δ SVA) after 10-min walk.

Exclusion criteria: spondylolysis/spondylolisthesis, congenital or neuromuscular scoliosis, post-traumatic kyphosis, hip joint or pelvic diseases, lower limb diseases such as osteoarthritis or ankle diseases, acute vertebral compression fracture, lumbar disc herniation/lumbar spinal stenosis with radicular symptoms, Scheuermann kyphosis and Cobb angle > 30°.

Among the remaining participants, in order to reduce bias because of obesity and age, the participants were well matched with the DSI patients based on body mass index (BMI) and age. Finally, a total of 42 participants were included in the control group (Compensated group). Numeric Rating Scales (NRS, 0–10) for low back pain and Oswestry Disability Index (ODI) scores were routinely collected. Written informed consent was obtained from all subjects. This study was approved by the Ethics Committee of local institution.

Paravertebral Muscle Evaluation

All subjects underwent lumbar MRI (magnetic resonance imaging) examination. MRI was performed by 1.5T Magnetom Vision scanners (Magnetom Symphony; Siemens, Berlin, Germany). The original T2-weighted axial images were imported into Image J (Version 1.52a. National Institutes of Health. USA) for analysis. The contours of the multifidus (MF) and erector spinae (ES) are outlined manually using Image

J. (Fig. 2.A) ES and MF were evaluated at the midlevel of L2 and L4 level bilaterally. The muscle fat infiltration area was estimated using the subcutaneous fat threshold as the standard[10, 11]. (Fig. 2.B) In order to compensate for the bias of individual body size, we introduced muscle muscularity[12], which was defined as the ratio of CSA of muscle to CSA of L4 vertebrae body. The FIA in each muscle was evaluated: the ratio of fat infiltration area to total CSA. CSA of muscle was calculated as the mean value of bilateral CSA. All data was assessed twice by two independent spinal surgeons, and the average value was calculated as the final result.

Statistical analysis

Statistical analysis was performed using SPSS (IBM Inc., Chicago, IL, US). The normality of the data was tested by the Shapiro–Wilk test firstly. Independent-sample t test were performed for comparison of quantitative variables between two groups. All data were presented as mean values $\pm$ SEM (standard error of the mean). P value < 0.05 was considered statistically significant.

Results

DSI group was comprised of 3 men and 28 women (age, 63.7 ± 4.8 years old; height, 159.8 ± 6.3 cm; and BMI, 25.0 ± 3.3). The control group was comprised of 2 men and 40 women (age, 63.5 ± 4.1 years old; height, 158.3 ± 6.4 cm; and BMI, 23.9 ± 3.4). There were no significant difference in age, height, or BMI between two groups.

There was either no significant difference in NRS for low back pain between two groups. Statistically significant difference was observed for ODI scores between both two groups, DSI group had higher ODI scores (17.7 ± 4.6) than the control group (14.7 ± 6.9) ($p = 0.02$). Detailed results are described in Table 1.

Table 1
Comparison of demographic data in DSI group and control group

Parameters	DSI (n = 31)	Controls (n = 42)	P value
Age(y)	63.7 ± 4.8	63.5 ± 4.1	0.545
BMI (kg/m ²)	25.0 ± 3.3	23.9 ± 3.4	0.176
Heights(cm)	159.8 ± 6.3	158.3 ± 6.4	0.638
NRS for back pain	2.6 ± 0.7	2.7 ± 0.6	0.342
ODI	17.7 ± 4.6	14.7 ± 6.9	0.002*
BM body mass index, ODI Oswestry Disability Index, NRS Numeric Rating Scales, * Statistical significance.			

DSI group had lower muscle muscularity both in ES and MF than control group. ES muscularity at L2 level was 0.42 ± 0.08 in DSI group and 0.82 ± 0.17 in control group. ($p = 0.016$). ES muscularity at L4 level

was 0.36 ± 0.11 in DSI group and 0.76 ± 0.22 in control group. ($p \leq 0.001$). MF muscularity at L2 level was 0.17 ± 0.08 in DSI group and 0.36 ± 0.07 in control group. ($p \leq 0.001$). MF muscularity at L4 level was 0.34 ± 0.18 in DSI group and 0.48 ± 0.14 in control group. ($p \leq 0.001$).

DSI group had higher FIA both in ES and MF than control group. ES FIA at L2 level was 0.50 ± 0.17 in DSI group and 0.31 ± 0.10 in control group. ($p = 0.023$). ES FIA at L4 level was 0.55 ± 0.27 in DSI group and 0.34 ± 0.07 in control group. ($p \leq 0.001$). MF FIA at L2 level was 0.63 ± 0.22 in DSI group and 0.36 ± 0.12 in control group. ($p \leq 0.001$). MF FIA at L4 level was 0.76 ± 0.31 in DSI group and 0.40 ± 0.19 in control group. ($p \leq 0.001$). Detailed results are described in Table 2.

Table 2
Comparison of FI and muscle muscularity in DSI group and control group

	Levels	Muscles	DSI (n = 31)	Controls(n = 42)	P value
FIA	L2	MF	0.63 ± 0.22	0.36 ± 0.12	$\leq 0.001^*$
		ES	0.50 ± 0.17	0.31 ± 0.10	0.023*
	L4	MF	0.76 ± 0.31	0.40 ± 0.19	$\leq 0.001^*$
		ES	0.55 ± 0.27	0.34 ± 0.07	$\leq 0.001^*$
Muscle muscularity	L2	MF	0.17 ± 0.08	0.36 ± 0.07	$\leq 0.001^*$
		ES	0.42 ± 0.08	0.82 ± 0.17	0.016*
	L4	MF	0.34 ± 0.18	0.48 ± 0.14	$\leq 0.001^*$
		ES	0.36 ± 0.11	0.76 ± 0.22	$\leq 0.001^*$
FIA: fat infiltration area, ES erector spinae, MF multifidus, *Statistical significance.					

Discussion

LDK was characterized by degenerative loss of lumbar lordosis or flatback syndrome without a history of prior spinal surgery[3]. It is more frequently reported in Asian population than in western countries[2, 13]. Some researchers argued that LDK is caused by unique life styles such as the prolonged crouched posture during agricultural work and performing activities of daily living on the floor[13]. Typically, LDK is categorized as 'N type' according to SRS-Schwab ASD classification[14].

DSI was first systematically reported by Bae[6], he introduced the method of taking two sets of lateral full-spine standing radiographs at initiation and after a 10-min walk to assess DSI. In this study, different compensatory changes of sagittal parameters were studied in 'compensated' group and 'decompensated'

group, respectively. Recently, Zhou et al. investigated more detailed changes of sagittal parameters in 'decompensated' group[7]. Moreover, Lee found that the good compensation of the pelvis determines the outcome of surgery in DSI[9]. In a word, DSI is a dynamic process which is associated with different changes of sagittal parameters. Although the above studies speculated that there was weakness of paravertebral muscles, no further investigation was did.

The compensatory mechanisms for sagittal imbalance are very complex, which include reduction in TK, hyperextension in lumbar, retroversion of pelvis, and lower extremities-related compensatory mechanisms[15, 16]. All those mentioned above compensatory mechanisms depend on contraction and interaction between gluteus, quadriceps femoris, iliopsoas and paravertebral muscles, etc[17].

Paravertebral muscles have long been viewed as important factors in maintaining sagittal balance. MF and ES have gained more attention for their functions. Anatomically, MF and ES connect the lumbar spine to the pelvis, MF is usually divided into two layers, the superficial multifidus is responsible for lumbar extension and the deep multifidus for intersegmental stability[18, 19]. Whereas the ES play a key role in extending the spine and maintaining spinal balance against body weight[20]. Moreover,

previous studies have demonstrated that changes in spino-pelvic parameters is strongly associated with a decrease in the mass of the MF, ES as well as psoas[19, 21, 22]. Once the muscles degenerate, it will inevitably affect the compensation mechanism, which will lead to the sagittal imbalance.

There is no doubt that the spine sequence is the primary factor affecting the sagittal balance of the spine. In LDK, in addition to the sagittal imbalance caused by marked loss of lumbar lordosis or lumbar kyphosis itself, the fatigue of back extensor muscles would further accelerate sagittal imbalance[11, 23]. The results of biomechanical experiments and surface electromyography have confirmed the significantly increased activity in the paravertebral muscles of LDK[2, 24, 25]. For LDK patients, better paravertebral muscle function is needed to maintain ideal spinal balance.

In present study, we found that patients in the control group also have a certain degree of paravertebral muscle degeneration, but DSI group suffered lower muscle muscularity and higher FIA both in ES and MF than control group. High FI rate and low muscle muscularity always represent decreased muscle power and poor fatigue resistance[25, 26]. In control group, strong muscle power and good fatigue resistance of paravertebral muscles keep the spine always in good balance permanently. Whereas in DSI group, decreased quantity and quality of paravertebral muscles lead to sagittal imbalance quickly even in 10-min walk. The representative cases are shown in Fig. 3 and Fig. 4. Therefore, our study revealed that the weakness of the paravertebral muscles plays an important role in the DSI process, which remind spinal surgeons that targeted muscle strengthening training may be an effective treatment for LDK with DSI. Previous studies have proved that targeted muscle strengthening training can effectively prevent spinal sagittal imbalance and improve ODI[19, 25].

In summary, DSI is the result of the continuous interaction of the spinal sequence and the paravertebral muscle. A bad spinal sequence such as LDK only represent a strong propensity to fatigue for

paravertebral muscles, which could accelerate sagittal imbalance. Another decisive factor is that the strength and fatigue resistance of paravertebral muscles. Good muscle function could combat this propensity to fatigue, which would keep sagittal balance persistently.

The present study has some limitations that need further discussion and investigation. First, an inherent limitation of this study is the sample size. Small sample size limited the ability to conduct more detailed groups for analysis. Second, the compensatory mechanisms of lower extremities have not been taken into account in this study. Full-body radiographs and 3D gait analysis are needed to evaluate the effect of lower limbs in further plan. Finally, the potential degeneration of other muscles involved in pelvic compensation such as gluteus and iliopsoas were not explored. MR of the pelvis and lower limbs are needed for further exploration in next step.

Conclusions

LDK patients with DSI suffered lower muscle muscularity and higher FIA both in ES and MF compared to LDK patients without DSI. Our study revealed that the weakness of the paravertebral muscles plays an important role in the DSI process, targeted paravertebral muscle strengthening training may be a potentially effective treatment for this disease.

List Of Abbreviations

LDK, lumbar degenerative kyphosis; DBS, drop body syndrome; SVA, increased sagittal vertical axis; CSA, cross-sectional area; FIA, increased fatty infiltration area; DSI, dynamic sagittal imbalance; Δ SVA, post-walk SVA-pre-walk SVA; BMI, body mass index; NRS, Numeric Rating Scales; ODI, Oswestry Disability Index; MRI, magnetic resonance imaging; MF, multifidus, ES, erector spinae

Declarations

Acknowledgements

The authors thank the Department of Imaging in Shandong Provincial Hospital affiliated to Shandong First Medical University for providing the image data of patients.

Funding

This article did not receive any funding.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Authors' contributions

JS and GW thought out the study, participated in its design and

coordination. NR carried out this study, did the statistics and drafted the manuscript. XC polished the manuscript. YL collected the image data of patients. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The participants provided written informed consent to participate in this research. The subjects' rights and interests are protected well in the whole process. The research has been approved by Medical Ethics Committee of Shandong Provincial Hospital affiliated to Shandong First Medical University

Consent for publication

All patients involved had given informed consent.

Competing interests

The authors declare that they have no competing interests.

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Figures

Figure 1

Flow chart of study processing.

Figure 2

A: The contours of the multifidus, erector spinae were outlined manually using Image J. B: Muscle fat infiltration (FI) area was estimated using the subcutaneous fat threshold as the standard.

Figure 3

Case 1. Female, 64 years old. A:Lateral full-spine standing radiographs at initiation shows normal SVA(SVA=9.4mm).B:SVA after 10-minute walk test remains normal (SVA=13.1mm, Δ SVA=3.7mm).C&D:No obvious fat infiltration and paravertebral muscle degeneration was found at L2 and L4 levels

Figure 4

Case 2. Female, 60 years old. A: Lateral full-spine standing radiographs at initiation shows normal SVA. (SVA=-15.7mm) B:Lateral full-spine standing radiographs after 10-minute walk test shows dynamic increased SVA (SVA=90.1mm , Δ SVA=105.8mm) C&D :Severe fat infiltration and paravertebral muscle degeneration at both L2 and L4 levels.