

Lumbar Posterior Muscle and Local Kyphosis in Patients with Degenerative Thoracolumbar Kyphosis: A Case-Control Study

Shuai Xu

Peking University People's Hospital

Chen Guo

Peking University People's Hospital

Yan Liang

Peking University People's Hospital

Zhenqi Zhu

Peking University People's Hospital

Haiying Liu (✉ liuhaiying1131@sina.com)

Peking University People's Hospital

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Abstract

Purpose: To explore the relationship between thoracolumbar kyphosis (TLK), body mass index (BMI) and the content of lumbar posterior muscles in patients with degenerative thoracolumbar kyphosis (DTLK) combined with lumbar stenosis syndrome (LSS).

Methods: 126 patients with DTLK and LSS (DTLK group) and 87 patients with simple LSS (control group) were retrospectively included with well-matched demographics. TLK and lumbar lordosis (LL) were obtained on the X-ray of the whole spine. Lumbar crossing indentation value (LCIV) was introduced to evaluate the content of the lumbar muscles, which was measured from T12-L1 to L4-L5 at T2-MRI axial imaging. Three subgroups of normal weight, overweight and obesity were respectively divided into in both groups according to BMI. Three subgroups of increased LL, normal LL and decreased LL were also divided into in DTLK group.

Results: The mean LCIV (mLCIV) of the DTLK group was less than control group and LCIV showed an increasing trend in DTLK group from T12-L1 to L4-L5. Different from control group, there were no significances in gender and BMI distribution of mLCIV in DTLK group ($P>0.05$). LCIV in increased LL subgroup was larger than that of normal LL and less LL subgroup ($P<0.01$). There were no relationship between TLK and BMI in both groups. BMI was positively correlated with mLCIV in control group ($P=0.004$). TLK and LCIV were negatively correlated ($P<0.001$) in DTLK group with $LCIV=13.75-0.48\times TLK$.

Conclusion: LCIV in DTLK group was less than control group with no gender- and BMI-difference. LCIV and TLK were mutually predictable in DTLK with $LCIV=13.75-0.48\times TLK$.

Introduction

Degenerative thoracolumbar kyphosis (DTLK), as a class of adult spinal deformity (ASD), is a common degenerative spinal disease in the elderly [1, 2]. DTLK combined with lumbar stenosis syndrome (LSS) with a high exposure in the group can cause low back pain, lower limb radiation pain and intermittent claudication, having become a critical disease affecting the quality of life [3]. There are age- and gender-related differences in the severity of DTLK [4, 5] but the relationship between body mass index (BMI) and DTLK remains controversial. Kim et al.[1] found that muscle content but not BMI correlated to deformity degree. However, some reports shown that obesity was not only the risk factor associated with spinal degeneration, but played a role in promoting the progress of TLK.

The paraspinal muscle groups exert a vital importance in maintaining normal spine formation and it is proved close to the severity of deformity. The weakening of the strength of posterior muscle groups will upregulate the incidence of low back pain and muscle atrophy or damage can also affect the prognosis in this series [6–8]. A large number of studies have measured the paravertebral muscle component in spinal deformity or normal ones to assess the relationship between the sagittal parameters and paraspinal muscle content. The most common indicators are muscle cross sectional area (CSA) and fat

infiltration (FI) by MRI [9–11]. However, the overall approach is too complicated and lengthy to rapidly evaluate paraspinal muscle content. Thus, Takayama et al.[12] proposed the concept of lumbar crossing indentation value (LCIV), as the distance from spinous process to the apex of the surface of the paravertebral muscles on both sides, sharply simplifying the measurement process. In addition, the study confirmed that LCIV was highly relevant to CSA and FI and emphasized LCIV can represent muscle content in people over 50 years old. The characteristics of LCIV in the elderly without deformity has been depicted, but spinal malformation in DTLK patients may affect the distribution of paraspinal muscles and the depth of LCIV.

It showed a correlation between BMI and the paravertebral muscles content in non-deformity ones. Banno et al. [11] firstly confirmed that the CSA of multifidus and psoas are related to body weight, which should be taken into account. However, in DTLK with LSS, the relationship among TLK degree, BMI and LCIV needs to be explored. Therefore, this study faces to patients diagnosed DTLK with LSS to clarify the characteristics of paraspinal muscle content in this group and to explore the correlation between the TLK degree, BMI and LCIV.

Material And Methods

1.1 Participants

The single-center retrospective study was performed from June 2015 to December 2018. Patients from our institution diagnosed with LSS and DTLK with surgical-indication were enrolled (DTLK group). Then, LSS patients need operation without spinal deformity were screened as control group. The study was approved by ethics committee in our institution and all participants have signed informed consent.

Inclusion criteria are (1) patients in DTLK group diagnosed as DTLK with LSS without coronal malformation; (2) control group patients with simple LSS; (3) complete and clear preoperative X-ray of the whole standing spine and thoracolumbar or lumbar MRI; (4) Age > 50 years. Exclusion criteria are (1) coronary imbalance or abnormalities; (2) incomplete or blurred imaging of X-ray or MRI; (3) diagnosis of idiopathic scoliosis, congenital scoliosis or other spinal deformities; (4) patients with lumbar spine fracture, tumor, infection or spondylolisthesis; (5) those who have undergone thoracolumbar surgery. Coronal scoliosis is Cobb angle > 10° and imbalance is defined as sagittal vertical axis (SVA) > 3 cm. Kyphosis severity is expressed by TLK (the angle between the upper end plate of T10 and the lower end plate of L2) and DTLK is TLK ≥ 15° caused by degeneration (Fig. 1 (A)).

1.2 BMI

BMI equals weight (kg) divided by the square of height (m^2). Patients were classified into normal weight (N), overweight (OW) and obesity (OB) according to BMI where $18.5 \leq N < 25.0$ (kg/m^2), $25.0 \leq OW < 30.0$ (kg/m^2) and $OW \geq 30.0$ (kg/m^2).

1.3 MRI

All subjects underwent MRI in a supine position using a 1.5-T scanner (Gyroscan; Philips Medical Systems, Washington, USA). Axial 5-mm slices were acquired using three-dimensional thick T2-weighted spin-echo axial scanning (TR: 3200 ms; TE: 102 ms; FOV: 180 mm; Matrix size: 512 × 512) from T12-L1 to L4-L5. The sagittal and axial images were uploaded to the PACS client software (Easy Vision IDS5, version 11.4; Philips, Hamburg, Germany). The axial images are obtained from the intervertebral space parallel to endplates.

1.4 Parameters measurement

TLK and lumbar lordosis (LL) are measured on whole spine X-ray. LL is the angle from upper endplate of L1 to upper endplate of S1 ($25^\circ \leq LL < 53^\circ$). T2 axial image is used for LCIV measurement (vertical distance from spinous process to the apex of paravertebral muscle on both sides (Fig. 1(B)). LCIV from T12-L1 to L4-L5 was separately measured while L5-S1 was excluded as the huge heterogeneity from occlusion of iliac crest and iliopsoas muscle attachment.

All parameters are measured by two persons independently and consistency is evaluated by intraclass correlation coefficient (ICC). ICC of TLK and LL are 0.86 and 0.79; ICC of each segment of LCIV is 0.90 (T12-L1), 0.78 (L1-L2), 0.78 (L2-L3), 0.83 (L3-L4) and 0.77 (L4-L5), which indicates all measurements are in good reliability.

1.5 Subgroup analysis

A subgroup analysis of gender (male, female) and BMI (N, OW, and OB) was performed to determine whether the gender and BMI have an effect on LCIV. Subgroup analysis was also performed for DTLK patients according to LL with larger LL group ($LL \geq 53^\circ$), normal LL group ($25^\circ \leq LL < 53^\circ$) and less LL group ($LL < 25^\circ$).

1.6 Statistical analysis

Dichotomous variables between groups are tested by χ^2 test or Fisher test. Independent sample t test is used for inter-group comparisons of measurement data while analysis of variance (ANOVA) analysis is used for comparisons of multiple measurements in the same group, which are also applied for comparison among subgroups. Pearson correlation analysis is used for correlation evaluation among TLK, BMI and LCIV and multiple linear regression is used to determine the influencing factors of LCIV. The software SPSS 22.0 (IBMC, Armonk, New York, USA) is used for statistical analysis and statistical differences is determined with $P < 0.05$.

Results

A total of 126 patients in DTLK group and 87 cases in control group were included. There were no statistical difference between the two groups in gender, age and BMI ($P > 0.05$). TLK in the DTLK group was 25.8 ± 10.1 (15.2–64.2)° (Table 1).

Table 1
Basic information between DTLK group and control group

| | DTLK group | Control group | P |
|------------------------|-----------------|-----------------|---------|
| Case | 126 | 87 | |
| Gender | | | 0.829 |
| Male | 35 | 23 | |
| Female | 91 | 64 | |
| Age, y | 68.0 ± 8.1 | 68.4 ± 8.3 | 0.711 |
| BMI, kg/m ² | 26.1 ± 3.6 | 26.3 ± 3.4 | 0.745 |
| N: 18.5–24.9 | 52 | 35 | |
| OW: 25–29.9 | 50 | 39 | |
| OB: ≥30 | 24 | 13 | |
| TLK, ° | 25.8 ± 10.1 | 6.7 ± 5.4 | < 0.001 |
| LL, ° | 36.6 ± 19.1 | 43.9 ± 11.3 | 0.001 |

Footnote: DTLK: degenerative thoracolumbar kyphosis; BMI: body mass index; N: normal; OW: overweigh; OB: obesity; TLK: thoracolumbar kyphosis; LL: lumbar lordosis

2.1 LCIV on DTLK and control group

LCIV in DTLK group was less than that of control group in the segment of T12-L1, L1-L2 and L2-L3 ($P < 0.01$), but not in the lower lumbar spine. The mean LCIV (mLCIV) of DTLK was less than control group ($P < 0.01$). LCIV showed an increasing trend in DTLK group from T12-L1 to L4-L5 with intra-group differences ($P < 0.01$) (Table 2).

Table 2
Comparisons on LCIV from T12-L5 between DTLK and control group

| LCIV, mm | DTLK group | Control group | P |
|----------|------------|---------------|---------|
| T12-L1 | 4.4 ± 5.4 | 9.8 ± 4.4 | < 0.001 |
| L1-L2 | 5.4 ± 5.7 | 10.0 ± 4.4 | < 0.001 |
| L2-L3 | 6.7 ± 5.9 | 9.8 ± 4.4 | < 0.001 |
| L3-L4 | 9.2 ± 6.2 | 10.0 ± 4.4 | 0.361 |
| L4-L5 | 13.7 ± 5.6 | 12.6 ± 5.2 | 0.202 |
| mLCIV | 7.9 ± 5.1 | 10.4 ± 3.9 | < 0.001 |

Footnote: LCIV: lumbar crossing indentation value; DTLK: degenerative thoracolumbar kyphosis;
mLCIV: mean LCIV from T12-L5

There was no gender difference in TLK in control group ($P = 0.924$), mLCIV and LCIV for all segments in male were larger than female group ($P < 0.05$) except T12-L1 level. In DTLK group, there was no gender-related difference in all segments and mLCIV besides T12-L1 segment ($P = 0.044$) (Table 3).

Table 3
Comparisons on LCIV between subgroups in terms of gender

| LCIV, mm | DTLK group | | | Control group | | |
|----------|------------|------------|-------|---------------|------------|-------|
| | Male | Female | P | Male | Female | P |
| T12-L1 | 2.4 ± 6.5 | 5.1 ± 4.9 | 0.044 | 11.0 ± 4.5 | 9.5 ± 4.3 | 0.198 |
| L1-L2 | 4.1 ± 7.1 | 5.8 ± 5.1 | 0.241 | 12.1 ± 4.5 | 9.3 ± 4.2 | 0.015 |
| L2-L3 | 7.4 ± 7.3 | 6.4 ± 5.4 | 0.527 | 12.3 ± 4.2 | 9.0 ± 4.1 | 0.003 |
| L3-L4 | 10.1 ± 9.2 | 8.9 ± 4.9 | 0.575 | 12.1 ± 4.6 | 9.3 ± 4.1 | 0.014 |
| L4-L5 | 15.2 ± 8.0 | 13.2 ± 4.6 | 0.265 | 14.9 ± 4.2 | 11.9 ± 5.4 | 0.028 |
| mLCIV | 7.8 ± 7.0 | 7.9 ± 4.3 | 0.979 | 12.5 ± 3.7 | 9.8 ± 3.8 | 0.008 |

Footnote: LCIV: lumbar crossing indentation value; DTLK: degenerative thoracolumbar kyphosis;
mLCIV: mean LCIV from T12-L5

TLK were of no differences with various BMI in both groups ($P = 0.605$ and $P = 0.464$). In control group, each LCIV and mLCIV were in differences among three subgroups by BMI ($P < 0.05$), where LCIV in N subgroup were less than OW subgroup from T12-L1 to L3-L4 ($P < 0.05$) while less than OB subgroup in all levels ($P < 0.01$). In DTLK group, LCIV in T12-L1 and L1-L2 were significant with various BMI ($P = 0.003$

and $P = 0.009$). Although mLCIV in N subgroup was smaller than OB subgroup, there was no statistical difference in mLCIV among subgroups ($P = 0.080$) (Table 4).

Table 4
Comparisons on LCIV between subgroups in terms of BMI

| LCIV, mm | DTLK group | | | Control group | | |
|----------|---------------------------|--------------------------|----------------|------------------------------|---------------------------|----------------|
| | N | OW | OB | N | OW | OB |
| T12-L1 | $2.7 \pm 4.1^{\text{bb}}$ | $4.1 \pm 6.2^{\text{c}}$ | 7.7 ± 4.2 | $7.9 \pm 4.7^{\text{a,bb}}$ | $10.4 \pm 3.7^{\text{c}}$ | 13.2 ± 2.8 |
| L1-L2 | $3.4 \pm 4.3^{\text{bb}}$ | 5.6 ± 6.5 | 8.2 ± 4.8 | $8.2 \pm 4.9^{\text{a,bb}}$ | 10.6 ± 3.3 | 12.8 ± 3.9 |
| L2-L3 | $5.1 \pm 5.2^{\text{b}}$ | 7.0 ± 6.6 | 8.7 ± 5.2 | $8.0 \pm 4.3^{\text{a,bb}}$ | 10.6 ± 3.8 | 12.3 ± 4.3 |
| L3-L4 | 8.8 ± 5.4 | 9.3 ± 7.4 | 9.8 ± 5.0 | $8.2 \pm 4.0^{\text{a,bb}}$ | 10.8 ± 4.2 | 12.3 ± 4.6 |
| L4-L5 | 12.8 ± 5.5 | 14.0 ± 6.5 | 14.5 ± 4.0 | $10.8 \pm 5.5^{\text{bb}}$ | 14.1 ± 4.5 | 13.0 ± 5.2 |
| mLCIV | $6.6 \pm 4.2^{\text{b}}$ | 8.0 ± 6.0 | 9.8 ± 4.0 | $8.6 \pm 3.9^{\text{aa,bb}}$ | 11.3 ± 3.3 | 12.7 ± 3.6 |

Footnote: LCIV: lumbar crossing indentation value; BMI: body mass index; DTLK: degenerative thoracolumbar kyphosis; N: normal; OW: overweight; OB: obesity; mLCIV: mean LCIV from T12-L5

^a: statistical difference between N and OW in the same level ($P < 0.05$), ^{aa}: statistical difference between N and OW ($P < 0.01$), ^b: statistical difference between N and OB ($P < 0.05$), ^{bb}: statistical difference between N and OB ($P < 0.01$) c: statistical difference between OW and OB ($P < 0.05$)

There were 26 cases, 64 cases and 36 cases in larger, normal and less LL subgroup respectively in DTLK patients. Each LCIV and mLCIV were significant among three subgroups ($P < 0.01$) except for the T12-L1 segment. LCIV from L1-L2 to L4-L5 and mLCIV in increased LL subgroup was larger than that of normal LL and less LL subgroup ($P < 0.01$) (Fig. 2 and Fig. 3).

2.2 The influencing factor of LCIV

In control group, mLCIV positively correlated to BMI ($r = 0.328, P = 0.004$) but not to TLK. In DTLK group, TLK showed a negative correlation to mLCIV ($r = -0.480, P < 0.001$) but not to BMI. The integration of double group showed mLCIV positively correlated to BMI ($r = 0.201, P = 0.009$) while negatively to TLK ($r = -0.397, P < 0.001$).

mLCIV was seen as the dependent variable and variables ($P < 0.1$) were as independent variables. In control group, the independent influencing factor was BMI ($P < 0.01$) with $\text{mLCIV(mm)} = 0.33 \times \text{BMI}(\text{kg}/\text{m}^2)$ ($0 \leq \text{TLK} < 15^\circ$). In DTLK group, the independent influencing factor was TLK ($P < 0.001$) and mLCIV (mm) = $13.75 - 0.48 \times \text{TLK}^\circ$ ($\text{TLK} \geq 15^\circ$). The integration of the two group showed that mLCIV

was both determined by TLK and BMI where mLcIV (mm) = $5.41 + 0.22 \times \text{BMI} (\text{kg}/\text{m}^2) - 0.41 \times \text{TLK}({}^\circ)$ (Table 5).

Table 5
Multiple linear regression analysis of mLcIV

| | Coefficient | Unstandardized | | Standardized | t | P value |
|---------------|-------------|----------------|-------|--------------|--------|---------|
| | | B | SE | Beta | | |
| DTLK group | Constant | 13.749 | 1.027 | | 13.386 | < 0.001 |
| | TLK | -0.183 | 0.036 | -0.480 | -5.131 | < 0.001 |
| Control group | Constant | 1.016 | 3.161 | | 0.321 | 0.749 |
| | BMI | 0.356 | 0.120 | 0.328 | 2.970 | 0.004 |
| All cases | Constant | 5.407 | 2.149 | | 2.516 | 0.013 |
| | TLK | 0.246 | 0.080 | 0.215 | 3.062 | 0.003 |
| | BMI | -0.127 | 0.022 | -0.413 | -5.872 | < 0.001 |

Notefoot: DTLK: degenerative thoracolumbar kyphosis; LCIV: lumbar crossing indentation value; SE: standard error; BMI: body mass index

Discussion

The maintenance of spinal sequence and sagittal balance have been fully realized close to paraspinal muscle groups, especially multifidus and erector spinae [6, 7, 13]. Yagi et al. [7] conducted a multicenter retrospective study with 60 ASD patients and found that CSA of multifidus and erector spina was less, significantly correlated with the sagittal spinal disorders. They found that the spine sequence and the paravertebral muscles are interacting. In fact, there was a interaction between paravertebral muscles and spinal sequence, especially in the elderly [13, 14]. In addition, Mannion et al. [15] found that the degeneration of paravertebral muscle is a secondary progress related to the severity of scoliosis in ASD by histochemical analysis.

For ASD patients, Banno et al [7] found that the malformation of sagittal parameters and lumbar spine were correlated with CSA of multifidus even if the weight and age were adjusted. While the muscle content in patients with thoracolumbar deformity was seldom focused on. The higher incidence and exposure rate in- and out-patient of DTLK with LSS in middle to old group forced it essential to indentify the relationship between TLK and paravertebral muscles [16]. However, usual methods for measuring paravertebral muscles were complicated and time-consuming, so many outpatient cases could not be measured in time. The concept of LCIV, innovated by Takayama et al., made the measurement easy and efficient, wide in use and with shorter learning-curve [12]. There were not many studies on body shape and sagittal deformity. Some reports showed obesity was a risk factor in promoting the occurrence of degenerative scoliosis while others believed that the absolute muscle composition related to sagittal

sequence instead of obesity presented by Fl [1, 17]. Our study found the muscle content with larger BMI was more than cases with lower BMI while the body shape was irrelevant to TLK in control group. Similarly, the degree of TLK in DTLK group did not correlate to BMI but to the content of paraspinal muscle.

Takayama et al. [12] found LCIV and CSA were highly correlated in all lumbar segments ($r = 0.708–0.789$) and LIV was negatively correlated with age, but there was no statistical difference among people over 50 years old. Our data showed that LCIV in DTLK group was less compared to normal ones. On the one hand, local kyphosis of the thoracolumbar and upper lumbar spine with DTLK approximated spinous processes to the skin in this range, even protruding over the surface. On the other hand, the posterior muscle groups got thinner under pressure with abnormal distribution by kyphotic bone structure, supporting the point that CSA of paraspinal muscle was in correlation to progressive kyphosis [9, 18]. Therefore, the intergroup difference on LCIV mainly concentrated on the upper lumbar spine and the lower region was almost comparable. The LCIV gradually increased from cranial to caudal vertebrae regardless of the DTLK or the control group, which mainly due to the sagittal lordosis in lumbar spine sine the anatomical structure enlarged the interval from spinous process to the apex of both muscle skin surface. In addition, the iliac crest and sacrum provided the attachment point of sacrospinous muscle and further thickened the paravertebral muscles in this area [12].

As the initial spinal-pelvic parameter, the loss or increase of LL will cause adjustment of pelvis rotation and proximal thoracolumbar kyphosis for compensating [19, 20]. In this study, the ratio of patients with increased and decreased LL were 20.6% and 28.6%, respectively. For those with larger LL, there is deepened skin grooves and higher apex of posterior muscles. While for lower LL ones, although surrounded by psoas and erector spinae, the content of paraspinal muscle decreased with spine kyphosis and less space for muscle attachment, together with the muscle strength of the lower back muscles significantly frail in patients with LL loss [7]. Takemitsu et al [21] measured the muscle content of the trunk in patients with LL kyphosis and found that the strength of posterior extensor muscle was significantly weaker than flexor muscle. Hongo et al. [18] indicated that posterior muscular strength was in correlation to LL loss or kyphosis in the elderly and strengthen lower back muscle could improve LL. Therefore, the restoration of LL by surgery or conservative treatment can increase the muscle volume and improve distribution. At the same time, it emphasizes the importance of strengthening back muscles to improve or maintain the sequence of the spine.

There is a gender-difference in muscle density and CSA in normal populations [4, 22], where the male have greater muscle content than female group, consistent with our data on LCIV in terms of sex. However, this study showed no significance in muscle content in DTLK group, considering the loss of muscle content and lower muscle density in the elderly weakened the gender discrepancy. Consequently, the muscle content and composition must be valued in patients with DTLK or potential DTLK, regardless of aged male or female.

In shows that increased load on spine will lead to loss of intervertebral-space height and reduction of gravity absorbing of disc. Compression on the spine by obesity prolonged, inflammation will be caused through the release of fat cell factors and affect spinal degeneration [23, 24]. In addition, the weakened paravertebral muscles of obese patients and inadequate capacity of maintaining upright will deteriorate intervertebral disc and facet. Wang et al. [3] believed that BMI closely correlated to the formation and progress of degenerative scoliosis, especially BMI $\geq 25.57 \text{ kg/m}^2$ while Kim et al. [25] proposed ambiguous attitude. In this study, neither TLK in DTLK group or in control group were not affected by BMI, consistent with the points of Kim et al. There is a significance in muscle content between N and OB subgroup in control group, indicating that the "obese" figure was also accompanied with "compact" muscle in obese patients and the strength of muscle content was to confront hyperload on spine with higher BMI [17]. The muscle content was comparable with various BMI but related to TLK severity in DTLK, which mainly because the muscle content of DTLK was more affected by kyphosis than BMI. Meanwhile, the muscle content and strength got lost instead of fat component in DTLK, more obvious in those with larger BMI [26], which was also proved by MRI and intraoperative dissection [27].

The study firstly clarified the characteristics of BMI and paraspinal muscle distribution in DTLK patients by a case-control study. It emphasized that paraspinal muscle need be paid attention to for DTLK and function excising or rehabilitation training on paraspinal muscle after surgery was necessary for promoting the reconstruction of sagittal spinal sequence [28]. Then, the introduction of the LCIV simplified the measurement procedure of paraspinal muscle, saving time and cost for outpatient doctor to make a preliminary judgment and for the patient to perform a self-check [12, 29]. In addition, the study quantified the relationship between TLK, BMI and LCIV. For DTLK patients, measurement on TLK and the muscle content can roughly predict each other, which was valuable for primary diagnosis and taking further measures on DTLK therapy. There were some limitations: Firstly, the measurement of LCIV is not as accurate as CSA, especially for muscle tissue with much fat. Then, the age range so large (50-87y) with no stratification although LCIV was proved stable over 50y that will cause reporting bias. Furthermore, the conclusion was only suitable for patients DTLK with LSS, may not for other types of deformity such as ankylosing spondylitis, Scheuermann's disease or coronal scoliosis. Finally, it was a retrospective study and the points needed to be consolidated by prospective cohort with large sample.

Conclusion

The LCIV in patients with DTLK combined with LSS was less than control group and the LCIV gradually increased from the upper to lower lumbar spine. In control group, the content of paraspinal muscle enlarged in aged LSS patients as BMI increased and LCIV in male was larger than females, while the DTLK group was of no gender-difference. The LCIV in DTLK group was richer in patients with increased LL. The degree of TLK was not related to BMI but to the content of the paravertebral muscle. For patients with DTLK and LSS, it was predictable between paraspinal muscle content and the degree of TLK.

Abbreviations

degenerative thoracolumbar kyphosis: DTLK

adult spinal deformity: ASD

lumbar stenosis syndrome:LSS

body mass index: BMI

muscle cross sectional area: CSA

lumbar crossing indentation value; LCIV

sagittal vertical axis: SVA

lumbar lordosis: LL

intraclass correlation coefficient: ICC

Declarations

Ethics approval and consent to participate:

This study has obtained ethics approval and consent of the ethics committee in our hospital.

Consent for publication:

Not applicable

Availability of data and material:

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

Competing interests:

The authors declare that they have no competing interests.

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Authors' contributions:

Conceptualization: Haiying Liu, Shuai Xu; Data Curation: Haiying Liu, Shuai Xu, Chen Guo; Formal Analysis: Chen Guo, Shuai Xu; Investigation: Yan Liang, Chen Guo; Methodology: Shuai Xu; Yan Liang, Chen Guo; Project Administration: Haiying Liu, Chen Guo; Resources: Shuai Xu; Chen Guo;

Software: Shuai Xu, Chen Guo; Validation: Yan Liang, Chen Guo; Visualization: Haiying Liu; Writing & Editing: Haiying Liu, Shuai Xu, Chen Guo, Yan Liang. Finally, We acknowledge Houshan Lv who contributed towards the study by making substantial contributions to the design and the acquisition of data.

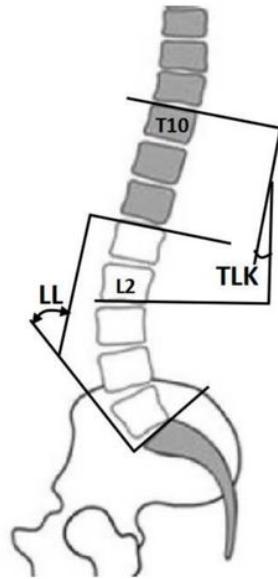
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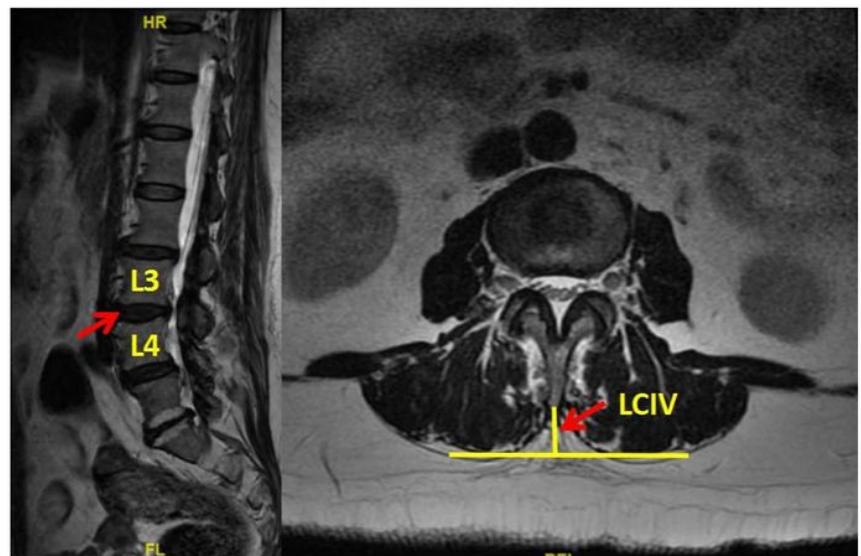
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Figures



1A



1B

Figure 1

(A) the definition of TLK and LL; (B) the definition of LCIV. TLK:thoracolumbar kyphosis; LL: lumbar lordosis; LCIV: lumbar crossing indentation value

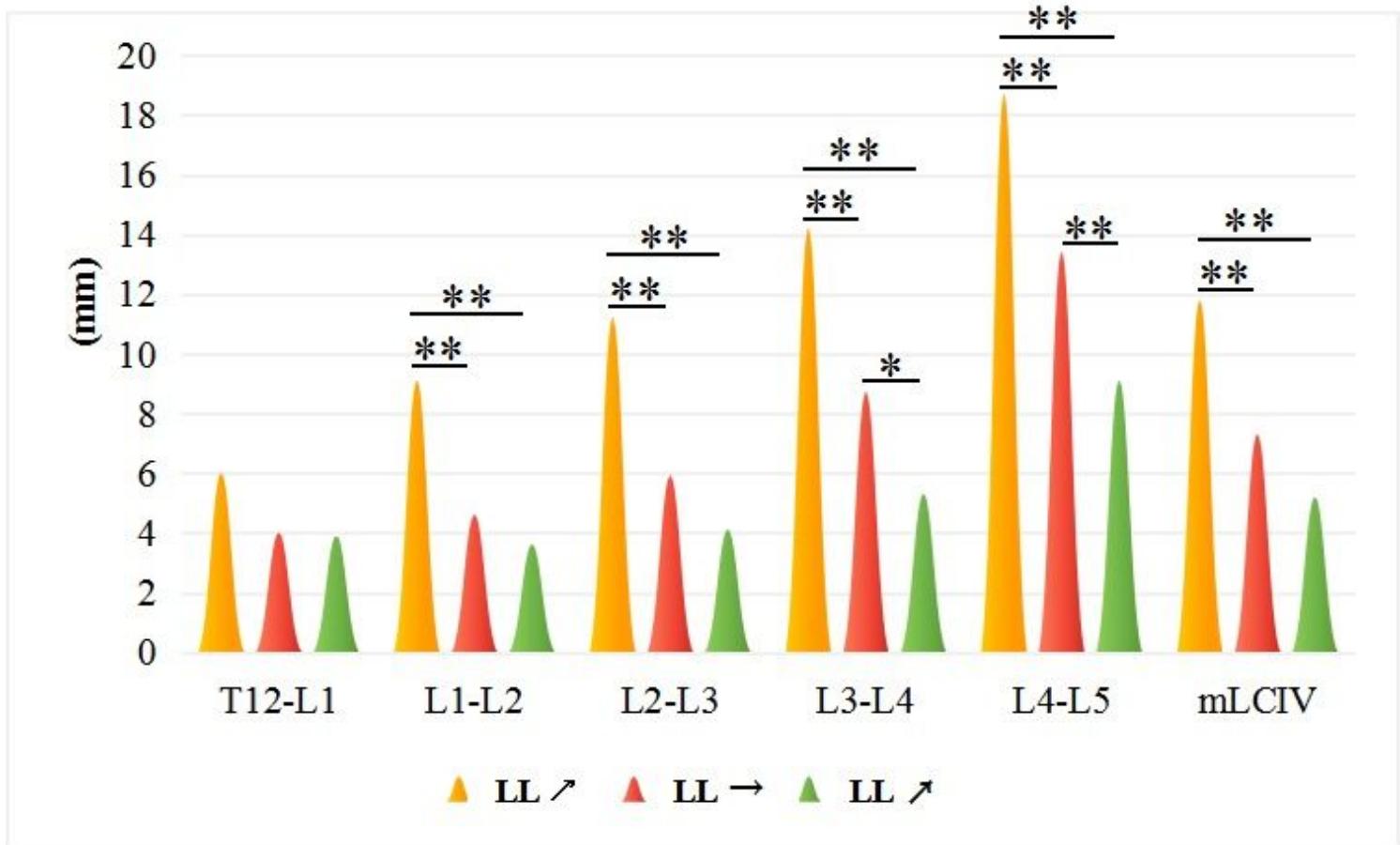
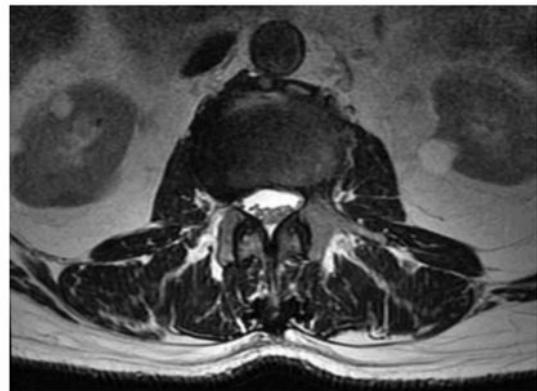


Figure 2

LCIV in increased LL and decreased LL subgroup in DTLK group. *: statistical difference between variables ($P<0.05$); **: statistical difference between variables ($P<0.01$)



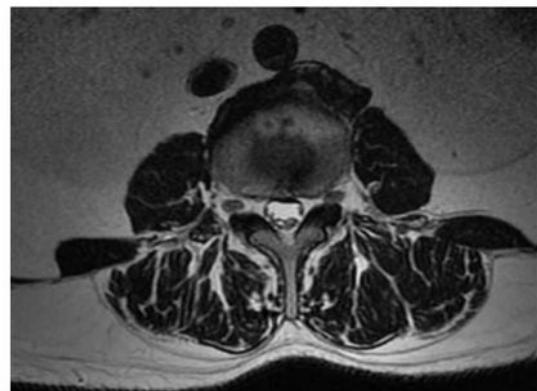
3A



3B



3C



3D



3E



3F

Figure 3

The cases with DTLK in less LL, normal LL and larger LL subgroups respectively. (A-B) 73-year-old male, BMI=26.4kg/m², TLK=26.3°, LL=5.9°, LCIV in L3-4 was 7.2mm, mLcIV was 5.8mm; (C-D) 68-year-old male, BMI=26.5kg/m², TLK=28.2°, LL=32.1°, LCIV in L3-4 was 11.2mm, mLcIV was 7.5mm; (E-F) 68-year-old male, BMI=25.8kg/m², TLK=29.8°, LL=61.9°, LCIV in L3-4 was 5.2mm, mLcIV was 12.0mm