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Original Article

Correlation between the severity of lumbar spinal stenosis and lumbar paraspinal muscle atrophy

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Abstract: Objective: To investigate the correlation between the severity of lumbar spinal stenosis (LSS) and the atrophy of lumbar paraspinal muscles. Methods: A retrospective analysis was conducted on 200 patients with LSS (stenosis group) and 60 individuals without lumbar spine disease (control group) treated at the Department of Orthopedics, Kunming Traditional Chinese Medicine Hospital, from January 2022 to October 2024. Using a 1.5T MRI system, we measured the total cross-sectional area (TCSA) and total fat-free cross-sectional area (TFCSA) of the multifidus, erector spinae, and psoas major muscles. Muscle atrophy was evaluated using the TFCSA/TCSA ratio, and its correlation with LSS severity was analyzed. Results: The stenosis group showed significantly lower TFCSA/TCSA ratios in the multifidus, erector spinae, and psoas major compared to controls ($P<0.05$). LSS severity was negatively correlated with the TFCSA/TCSA ratios of the multifidus ($r=-0.504$, $P<0.05$) and erector spinae ($r=-0.562$, $P<0.05$), but not with the psoas major ($P>0.05$). Similarly, the number of stenotic segments was negatively correlated with multifidus ($r=-0.381$) and erector spinae ($r=-0.420$) atrophy ($P<0.05$). TFCSA/TCSA ratios were significantly lower on the symptomatic side for all three muscles (all $P<0.05$). Conclusion: The severity and extent of LSS are significantly associated with atrophy of the multifidus and erector spinae, but not the psoas major. Greater muscle atrophy corresponds to a higher number of stenotic segments.

Keywords: Lumbar spinal stenosis, paraspinal muscles, muscle atrophy, correlation

Introduction

Lumbar spinal stenosis (LSS) is a common degenerative spinal disorder characterized by narrowing of the lumbar spinal canal, resulting in compression of the nerve roots and leading to symptoms such as lower back pain, radiating leg pain, and neurological deficits [1-3]. The pathogenesis of LSS is multifactorial, involving intervertebral disc degeneration, osteophyte formation, and facet joint hypertrophy [4]. Recent studies have increasingly focused on the impact of LSS on paraspinal muscles, particularly the association between muscle atrophy and stenosis severity [5].

The lumbar paraspinal muscles - including the multifidus, erector spinae, and psoas major - constitute the spine's active stabilizing system and are essential for lumbar movement and spinal stability [6]. The multifidus, the deepest of these muscles, originates from the transverse processes and inserts onto the spinous processes. It plays a major role in spinal extension, lateral flexion, and rotation, contributing more than two-thirds of spinal stabilization by rapidly generating force during dynamic activities [7]. The erector spinae, originating from the posterior sacrum, lumbar spinous processes, and thoracolumbar fascia, serves as a key extensor group. These muscles are among the first to exhibit degenerative changes in lumbar spine disorders, and their degeneration correlates strongly with age and intervertebral segment pathology [8]. The psoas major, originating from the lumbar vertebral bodies and transverse processes and inserting at the lesser trochanter of the femur, is involved in lumbar stabilization,

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hip flexion, and generating propulsive force during gait [9].

Recent research has further highlighted the role of paraspinal muscles in lumbar degenerative conditions. Wang et al. [8] reported significantly greater fatty infiltration in the multifidus at the herniated segment in patients with lumbar disc herniation compared to the contralateral side, with a positive correlation between fatty infiltration and chronic low back pain. FI et al. [10] identified degenerative spondylolisthesis as an independent risk factor for fatty infiltration in paraspinal muscles, with a strong association between multifidus degeneration and increased pelvic tilt angle. Moreover, moderate-to-severe canal stenosis may disrupt neural input to the multifidus, promoting muscle atrophy and fatty infiltration, thereby compromising spinal stability and negatively impacting postoperative outcomes [11]. These findings underscore a strong link between multifidus atrophy and degenerative lumbar disorders. However, the specific relationship between LSS severity and morphological or functional changes in lumbar paraspinal muscles remains inadequately explored.

To address this gap, we conducted a retrospective study evaluating clinical data from patients with LSS, using MRI to quantify the cross-sectional area of the multifidus, erector spinae, and psoas major. This study aims to clarify the correlation between paraspinal muscle atrophy and the severity, number of stenotic segments, and laterality of symptoms in LSS. Our findings may provide a valuable reference for optimizing clinical diagnosis and treatment strategies in patients with LSS.

Materials and methods

Study design

The sample size was calculated using PASS software, based on α=0.05, β=0.2, and an expected correlation coefficient of r=0.4 from previous studies [12]. A minimum of 48 subjects per group was required. Accounting for a 10-20% dropout rate and the hospital's admission volume, the final sample size was set at a minimum of 58 participants per group.

This retrospective study included 200 patients with LSS and 60 individuals without lumbar spine disease, treated at the Department of Orthopedics, Kunming Traditional Chinese Medicine Hospital between January 2022 and October 2024. LSS patients were categorized into three groups based on stenosis location: L3/4, L4/5, and L5/S1. Ethical approval was obtained from the Ethics Committee of Kunming Traditional Chinese Medicine Hospital (Approval No. 2024-018-01).

Inclusion Criteria: (1) First-time diagnosis of LSS based on criteria from Practical Orthopaedics (4th edited.) [13]: MRI showing dural sac compression, CT showing hypertrophied articular processes, and clinical findings consistent with imaging; (2) Single-level central or lateral recess stenosis; (3) Age 40-60 years; (4) BMI between 18 and 28 kg/m^2; (5) No history of hypertension, diabetes, or cardiovascular disease; (6) Complete lumbar MRI data; (7) Control group participants had no history or symptoms of lumbar disease, and normal MRI findings.

Exclusion Criteria: (1) Coexisting lumbar degenerative diseases (e.g., disc herniation, spondylolisthesis); (2) Neuromuscular disorders; (3) History of lumbar trauma, tuberculosis, or tumors; (4) Professional athletes or individuals engaged in strenuous physical labor; (5) Incomplete imaging data.

MRI

All participants underwent lumbar MRI using a 1.5T scanner (United Imaging Healthcare, Shanghai). Scans were acquired in the supine position with neutral lumbar alignment. Sagittal and axial T2-weighted images from T12 to S1 were obtained using the following parameters: TR/TE 2980/122.6 ms, matrix 208×320, slice thickness 4 mm, recovery time 3000-3600 ms, and echo time 87-114 ms.

Evaluation of lumbar paraspinal muscle atrophy

The TCSA and TFCSA of the multifidus, erector spinae, and psoas major were measured bilaterally at the L3/L4, L4/L5, and L5/S1 levels using Patrick's semi-quantitative method [14] (**Figure 1**). Image-Pro Plus was used for outlining and quantifying the TCSA and TFCSA. The degree of muscle atrophy was evaluated by calculating the TFCSA/TCSA ratio, with lower ratios indicating more severe fatty infiltration and muscle atrophy. Adobe Photoshop CS6 (Adobe Systems, San Jose, CA, USA) was used for qualitative image analysis.

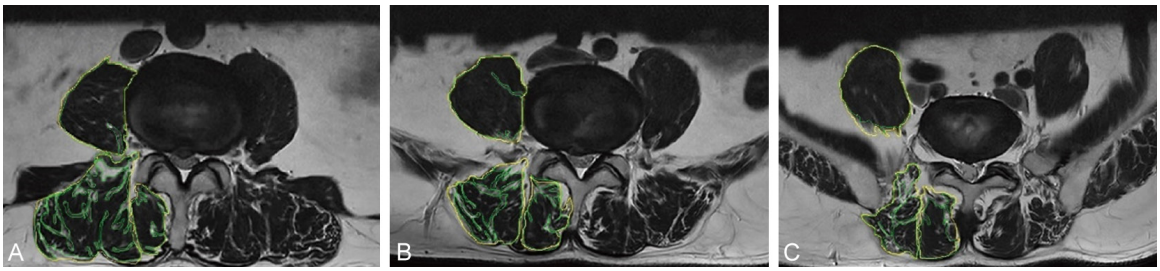

Figure 1. Measurement of TCSA and TFCSA at the L3/L4 (A), L4/L5 (B), and L5/S1 (C) intervertebral discs. Note: The yellow border outlined the transverse cross-sectional area (TCSA) of the multifidus, erector spinae, and psoas major muscles, while the green border outlined the fat-free multifidus cross-sectional area (TFSCA).

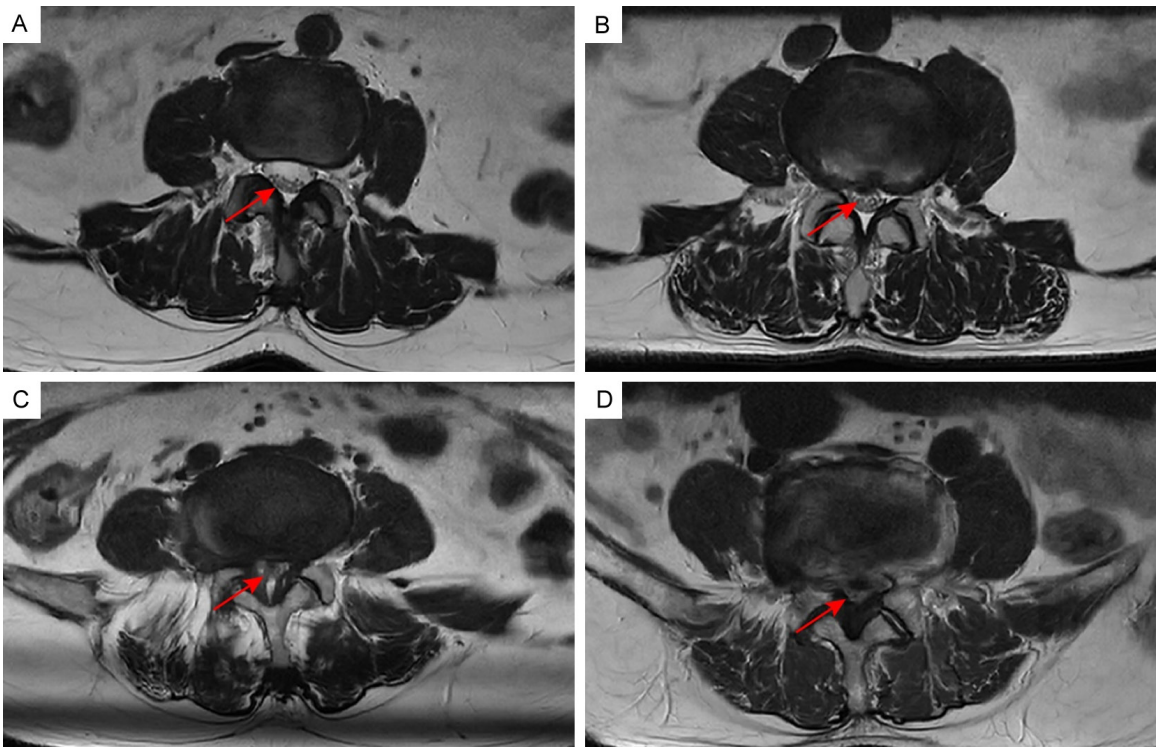

Figure 2. Representative MRI of lumbar spinal canal stenosis of differing degrees. Note: (A) No or mild stenosis; (B) Moderate stenosis; (C) Severe stenosis; (D) Extreme stenosis. The red arrow indicates the site of stenosis.

Grading of lumbar spinal stenosis

LSS severity was graded using the Schizas classification system [14], based on axial T2-weighted MRI.

No or mild stenosis: Uneven distribution of cerebrospinal fluid with visible dorsal cauda equina occupying less than half or most of the dural sac.

Moderate stenosis: Cauda equina fills the dural sac but individual rootlets remain distinguishable.

Severe stenosis: Rootlets are indistinct, and the dural sac presents as a uniform gray area with absent cerebrospinal fluid signal but preserved posterior epidural fat.

Extreme stenosis: In addition to indistinct rootlets, posterior epidural fat is absent (**Figure 2**).

Statistical analysis

Data were analyzed using SPSS 27.0 (IBM Corp., Armonk, NY, USA). GraphPad Prism was used for image processing. Normality was

Table 1. Comparison of Baseline characteristics between groups

Variables	Control group (n=60)	Narrow group (n=200)			χ^2/F	*P*
		L3/4	L4/5	L5/S1		
Sex (n, %)					0.190	0.979
Male	31 (51.67)	25 (48.08)	39 (51.32)	37 (51.39)		
Female	29 (48.33)	27 (51.92)	37 (48.68)	35 (48.61)		
Age (years, $\bar{x} \pm s$)	50.88±5.38	50.63±5.74	49.38±5.56	49.44±5.46	1.298	0.275
Educational level (n, %)					1.935	0.926
Primary school	9 (15.00)	10 (19.23)	15 (19.74)	10 (13.89)		
Junior high school	36 (60.00)	28 (53.85)	39 (51.32)	43 (59.72)		
High school and above	15 (25.00)	14 (26.92)	22 (28.95)	19 (26.39)		
BMI ($\bar{x} \pm s$, kg/m^2)	24.06±2.44	23.94±3.04	24.44±2.65	23.88±2.74	0.619	0.603
Disease duration (month, $\bar{x} \pm s$)	-	11.63±2.60	12.20±2.50	11.26±2.83	2.332	0.100
History of diabetes (n, %)					0.691	0.875
Yes	18 (30.00)	12 (23.08)	20 (26.32)	19 (26.39)		
No	42 (70.00)	40 (76.92)	56 (73.68)	53 (73.61)		
History of hypertension (n, %)					4.161	0.245
Yes	34 (56.67)	39 (75.00)	48 (63.16)	46 (63.89)		
No	26 (43.33)	13 (25.00)	28 (36.84)	26 (36.11)		
History of coronary heart disease (n, %)					1.751	0.626
Yes	28 (46.67)	25 (48.08)	30 (39.47)	28 (38.89)		
No	32 (53.33)	27 (51.92)	46 (60.53)	44 (61.11)		

assessed using the Shapiro-Wilk test and homogeneity of variance using Levene's test.

Normally distributed data with homogeneous variance are expressed as mean ± standard deviation ($\bar{x} \pm s$). Inter-group comparisons were performed using one-way ANCOVA with Bonferroni correction for post-hoc analysis.

Non-normally distributed data are presented as median (interquartile range), and inter-group differences were assessed using the Wilcoxon test.

Categorical data are presented as counts and percentages and analyzed using the chi-square test.

Pearson correlation analysis was used to assess the relationships between the number and severity of stenotic segments and TFCSA/TCSA ratios for the multifidus, erector spinae, and psoas major. A *P* value <0.05 was considered statistically significant.

Results

Comparison of baseline characteristics between groups

There were no statistically significant differences in gender, age, education level (primary school, junior high school, high school, college and above), BMI, medical history, or disease duration between the control group and the stenosis group (L3/4, L4/5, and L5/S1) (all *P*>0.05), indicating good comparability between the two groups (**Table 1**).

Comparison of TFCSA/TCSA ratios between groups

The average TFCSA/TCSA ratios of the multifidus, erector spinae, and psoas major muscles on the symptomatic side were significantly lower in the stenosis group compared to the control group (all P<0.05), indicating more pronounced muscle atrophy in patients with lumbar spinal stenosis (**Table 2**).

Correlation between stenosis severity and TFCSA/TCSA ratios

Patients in the stenosis group were classified into four subgroups based on the Schizas classification. The TFCSA/TCSA ratios of the multifidus and erector spinae showed a significant downward trend with increasing stenosis severity (both P<0.05), while no significant differences were observed for the psoas major (P>0.05) (**Table 3**). Pearson correlation analysis revealed that stenosis severity was negatively correlated

Table 2. Comparison of TFCSA/TCSA ratios between groups ($\bar{x} \pm s$)

	Control group (n=60)	Narrow group (n=200)	*t*	*P*
Multifidus TFCSA/TCSA (%)	78.71±6.30	68.13±8.73	10.368	<0.001
Erector spinae TFCSA/TCSA (%)	77.13±7.00	62.90±8.42	11.917	<0.001
Psoas major TFCSA/TCSA (%)	78.84±6.29	69.09±11.22	8.587	<0.001

TCSA: Total cross-sectional area, TFCSA: total fat-free cross-sectional area.

Table 3. Comparison of TFCSA/TCSA ratios among different degrees of spinal canal stenosis ($\bar{x} \pm s$)

	No or mild stenosis (n=29)	Moderate stenosis (n=46)	Severe stenosis (n=73)	Extreme stenosis (n=52)	*F*	*P*
Multifidus TFCSA/TCSA (%)	75.08±3.87	71.88±6.88	67.18±8.55[a,b]	62.28±8.25[a,b,c]	22.561	<0.001
Erector spinae TFCSA/TCSA (%)	70.13±7.76	67.81±6.81	61.03±7.89[a,b]	57.15±4.96[a,b,c]	31.614	<0.001
Psoas major TFCSA/TCSA (%)	71.48±14.89	70.44±11.40	68.43±11.17	67.50±8.38	1.095	0.352

Note: Compared with no or mild stenosis, [a]*P*<0.05; compared with moderate stenosis, [b]*P*<0.05; compared with severe stenosis, [c]*P*<0.05. TCSA: Total cross-sectional area, TFCSA: total fat-free cross-sectional area.

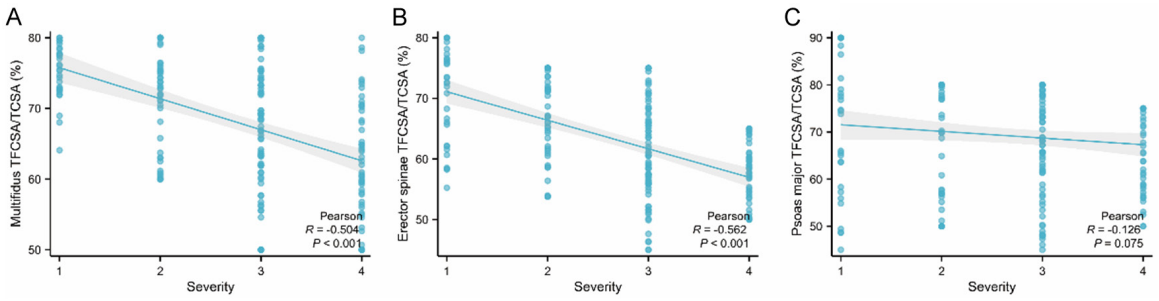

Figure 3. Correlation between Stenosis Severity and TFCSA/TCSA Ratios. Note: (A) Multifidus TFCSA/TCSA (%); (B) Erector spinae TFCSA/TCSA (%); (C) Psoas major TFCSA/TCSA (%). TCSA: Total cross-sectional area, TFCSA: total fat-free cross-sectional area. ***P<0.001.

with TFCSA/TCSA ratios of the multifidus (r=-0.504, P<0.05) and erector spinae (r=-0.562, P<0.05), but not with the psoas major (P>0.05) (**Figure 3**).

Correlation between number of stenotic segments and TFCSA/TCSA of ratios

Among the 200 patients in the stenosis group, 133 (66.5%) had single-segment stenosis, 34 (17%) had two-segment stenosis, and 33 (16.5%) had three-segment stenosis. The TFCSA/TCSA ratios of the multifidus and erector spinae significantly decreased with increasing numbers of stenotic segments (P<0.05), while no significant differences were observed in the psoas major (P>0.05) (**Table 4**). Pearson correlation analysis confirmed a negative correlation between the number of stenotic segments and the TFCSA/TCSA of the multifidus (r=-0.381, P<0.05) and erector spinae (r=-0.420, P<0.05), indicating that atrophy in these muscles worsens as the number of affected segments increases (**Figure 4A**, **4B**). No significant correlation was found for the psoas major (P>0.05) (**Figure 4C**).

Comparison of TFCSA/TCSA ratios between symptomatic and contralateral sides

The TFCSA/TCSA ratios of the multifidus, erector spinae, and psoas major were significantly lower on the symptomatic side compared to the contralateral side (*P*<0.05), indicating more severe muscle atrophy on the affected side (**Figure 5**).

Discussion

In this study, the severity of muscle atrophy in the multifidus, erector spinae, and psoas major was assessed by calculating the TFCSA/TCSA ratio at the L3/4, L4/5, and L5/S1 levels in 200 LSS patients. Stenosis severity was graded

Table 4. Comparison of TFCSA/TCSA of ratios among different numbers of stenotic segments ($\bar{x} \pm s$)

	1 - segment stenosis (n=133)	2 - segment stenosis (n=34)	3 - segment stenosis (n=33)	*F*	*P*
Multifidus TFCSA/TCSA (%)	70.3±8.03	66.06±9.03[a]	61.54±7.43[a,b]	16.722	<0.001
Erector spinae TFCSA/TCSA (%)	65.45±8.37	58.75±6.90[a]	56.89±4.46[a,b]	22.710	<0.001
Psoas major TFCSA/TCSA (%)	67.74±12.21	68.39±9.28	67.20±8.56	0.760	0.469

Note: Compared with 1 - segment stenosis, [a]*P*<0.05; compared with 2 - segment stenosis, [b]*P*<0.05. TCSA: Total cross-sectional area, TFCSA: total fat-free cross-sectional area.

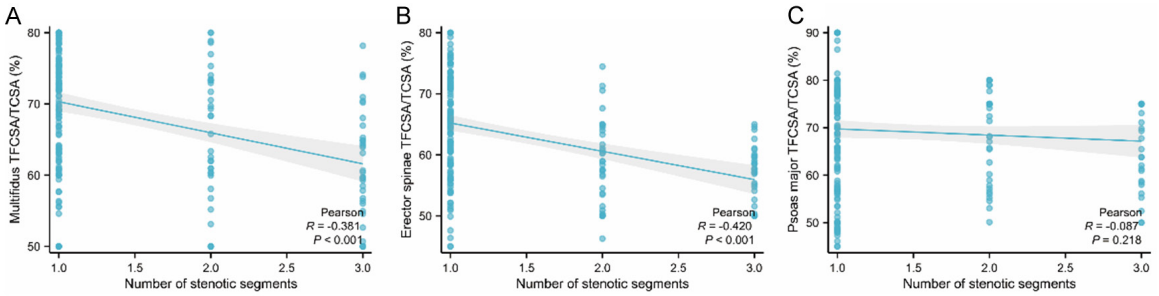

Figure 4. Correlation between number of stenotic segments and TFCSA/TCSA of Ratios. Note: (A) Multifidus TFCSA/TCSA (%); (B) Erector spinae TFCSA/TCSA (%); (C) Psoas major TFCSA/TCSA (%). TCSA: Total cross-sectional area, TFCSA: total fat-free cross-sectional area.

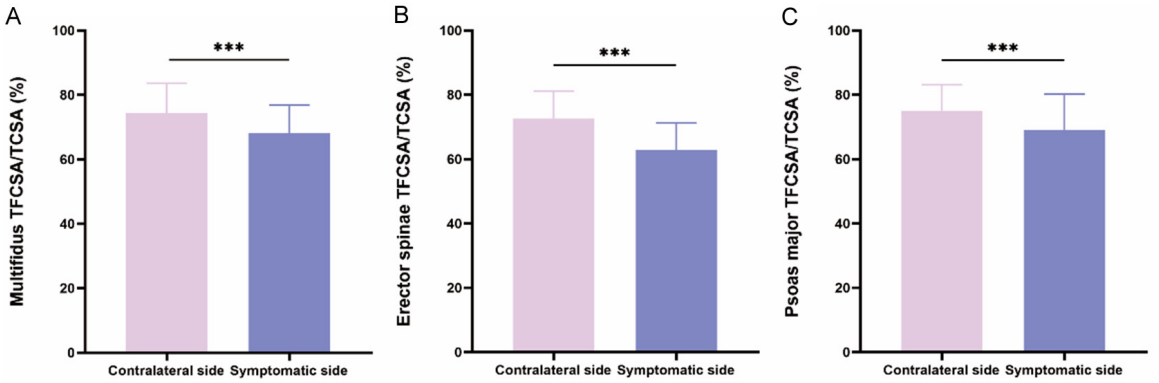

Figure 5. Comparison of TFCSA/TCSA ratios between the symptomatic and contralateral sides. Note: (A) Multifidus TFCSA/TCSA (%); (B) Erector spinae TFCSA/TCSA (%); (C) Psoas major TFCSA/TCSA (%). TCSA: Total cross-sectional area, TFCSA: total fat-free cross-sectional area.

using the Schizas classification system based on axial T2-weighted MRI images. The TFCSA/TCSA ratios of all three muscles were significantly lower in the stenosis group than in the control group, indicating greater muscle atrophy at stenotic levels. Moreover, both stenosis severity and the number of stenotic segments were negatively correlated with the TFCSA/TCSA ratios of the multifidus and erector spinae. In addition, these muscles exhibited more pronounced atrophy on the symptomatic side compared to the contralateral side. These findings provide novel insights into LSS pathogenesis and may inform clinical decision making.

The lumbar paraspinal muscles play a vital role in maintaining spinal stability and facilitating motion [15]. Among them, the multifidus is essential for segmental stabilization and lumbar extension [16]. Owing to its unique anatomical structure - attaching directly to the lumbar vertebrae - it contracts preferentially during spinal imbalance to preserve alignment and minimize vertebral displacement. It regulates ten-

sion across 1-3 vertebral segments to maintain physiological lumbar lordosis, a function closely related to its fiber composition [17]. Notably, the multifidus is rich in type I (slow-twitch) fibers, which are critical for postural control [18].

The erector spinae, comprising the spinalis, longissimus, and iliocostalis muscles, extends along the posterior spine and is primarily responsible for trunk extension and maintaining an upright posture. Each segment contributes differently: cervical spinalis aids head movement, while the lumbar longissimus and iliocostalis govern lumbar extension and lateral bending [19].

The psoas major, located anterior to the transverse processes and vertebral bodies, joins with the iliacus to form the iliopsoas. It functions primarily in hip flexion. When the lower limbs are fixed, bilateral contraction causes spinal flexion, whereas unilateral contraction facilitates lateral flexion and rotation. Together with the other paraspinal muscles, it helps maintain dynamic balance and mobility of the lumbar spine [20].

Consistent with previous studies [14], our findings confirm more pronounced paraspinal muscle atrophy in patients with LSS, evidenced by significantly lower TFCSA/TCSA ratios. Schönnagel et al. [11] similarly reported degenerative changes in these muscles, reinforcing the link between LSS and paraspinal muscle deterioration. However, our results further suggest that atrophy is not confined to the posterior paraspinal muscles but may also involve the psoas major, though possibly via distinct mechanisms.

Multifidus and erector spinae atrophy may result from multiple interrelated factors. Both muscles are innervated by the posterior rami of the spinal nerves, making them vulnerable to denervation atrophy due to nerve root compression in LSS [21]. Pain, a common LSS symptom, often leads to restricted activity and subsequent disuse atrophy [22]. Additionally, chronic inflammation and local ischemia may promote fatty infiltration, further reducing the functional muscle mass [23].

In contrast, the psoas major, primarily responsible for hip and lower limb movement, receives innervation from the lumbar plexus rather than directly from the spinal canal [24]. Thus, its atrophy may result more from decreased physical activity secondary to chronic pain, rather than direct nerve compression. This distinction supports the hypothesis that psoas major atrophy in LSS patients is primarily related to systemic disuse rather than local neuropathic changes.

In this study, LSS patients were categorized into four groups - mild, moderate, severe, and extreme - according to the Schizas classification. The results demonstrated a progressive decrease in the TFCSA/TCSA ratios of the multifidus and erector spinae with increasing stenosis severity. A significant negative correlation was observed between stenosis severity and the TFCSA/TCSA of the multifidus and erector spinae. These findings are consistent with those of Xia et al. [14], who similarly reported that greater spinal canal stenosis is associated with more severe fatty infiltration and muscle atrophy. Our study extends these observations by quantitatively mapping muscle degeneration across distinct stenosis grades, thereby reinforcing the clinical significance of paraspinal muscle deterioration in LSS progression.

Several mechanisms may underlie this phenomenon. Severe spinal stenosis may chronically compress the cauda equina or nerve roots, leading to denervation atrophy in the affected muscles. Elevated intraspinal pressure could also reduce local blood flow, exacerbating ischemic damage and degeneration. Kim et al. [25] provided electrophysiological evidence of chronic neurogenic changes, particularly in the multifidus, resulting from prolonged nerve root compression in LSS patients.

Interestingly, the TFCSA/TCSA ratio of the psoas major did not significantly differ among the four stenosis subgroups, supporting the hypothesis that its atrophy is less directly related to LSS pathology. Instead, as Hou et al. [26] suggest, psoas muscle atrophy may reflect systemic deconditioning due to chronic pain and reduced mobility rather than direct neural injury. This highlights the importance of distinguishing localized paraspinal degeneration from broader muscular deconditioning in LSS patients, which has implications for individualized rehabilitation strategies.

Our study also found that the number of stenotic segments was negatively correlated with the TFCSA/TCSA of the multifidus and erector spinae, suggesting that multi-segmental stenosis contributes cumulatively to muscle degeneration. This is likely due to increased mechanical instability, greater neural compromise, and higher compensatory load on the paraspinal musculature [27, 28]. Moreover, patients with multi-level stenosis typically exhibit more complex clinical manifestations, longer disease duration, and greater functional impairment, which may further accelerate muscular deterioration.

Additionally, we observed that the TFCSA/TCSA ratios of the multifidus, erector spinae, and psoas major were significantly lower on the symptomatic side compared to the contralateral side. This corresponds with clinical presentations of unilateral or asymmetric lower limb pain, numbness, or weakness in LSS. Potential explanations include greater nerve compression on the symptomatic side leading to denervation, reduced limb usage due to pain causing disuse atrophy, and localized inflammatory or hemodynamic alterations promoting muscle degeneration [29].

These findings have important clinical implications. TFCSA/TCSA ratios, particularly for the multifidus and erector spinae, may serve as auxiliary imaging biomarkers to assess LSS severity and guide treatment planning. In patients with marked paraspinal muscle atrophy, decompression surgery may need to be complemented by targeted rehabilitation to restore muscle function and improve outcomes. Core stability and paraspinal strengthening exercises could help mitigate further atrophy, enhance function, and improve quality of life.

This study does have some limitations. As a retrospective design, it is susceptible to selection and information biases. Muscle atrophy was evaluated solely through imaging, without incorporating functional measures such as electromyography or strength testing. Future prospective studies should include dynamic functional assessments and explore molecular mechanisms (e.g., inflammation, oxidative stress) underlying muscle atrophy in LSS.

In conclusion, this study demonstrates a strong association between lumbar paraspinal muscle atrophy and both the severity and extent of lumbar spinal stenosis. The multifidus and erector spinae muscles are most prominently affected, suggesting that muscle degeneration plays a key role in the progression of LSS. These findings provide new insights into the pathophysiology of LSS and underscore the clinical importance of muscle assessment. Future research should focus on elucidating the underlying mechanisms of muscle degeneration and developing targeted interventions to optimize LSS management.

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Disclosure of conflict of interest

None.

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