



Research Article

Online ISSN (3219-2789)

The Illusion of Lumbar Multifidus Muscle Size: Gross vs. Contractile Differences at L4 in Young Adult Women with Non-Specific Chronic Low Back Pain

Siti Nuradriana Syafira Mohd Rizal¹ , Ardo Okilanda² , Rudyanto Rudyanto² , Nurul Athilah Muhammad Nasir¹ , Ahmad Rafshan Hazim Ahmad Risham¹ , Nur Syafiqah Elysya Amirudin¹ , Nuramnani Suffiya Rohim¹ , Dzulhafiz Zikrie Dzolkapli¹ , Siti Nurul Ezzah Che Man¹ , Sarah Syafieqah Anuwar¹ , Hashbullah Ismail¹ , Norasrudin Sulaiman¹ , Mazuin Mohd Razalli³ , Shariman Ismadi Ismail¹ , Rahmat Adnan^{1*}

¹Faculty of Sports Science and Recreation, Universiti Teknologi MARA, 40450 Shah Alam, Malaysia; ²Department of Coaching and Sports, Universitas Negeri Padang, Padang, West Sumatra, Indonesia; ³Faculty of Medicine, Universiti Teknologi MARA (UiTM), Al-Sultan Abdullah Hospital (HASA UiTM), Malaysia

Received: 14 July 2026; Revised: 4 September 2026; Accepted: 8 September 2026

Abstract

Background: Non-Specific Chronic Low Back Pain (NSCLBP) is associated with alterations in lumbar muscle morphology, fat infiltration, and reduced contractile tissue, which may affect function and clinical evaluation in younger populations when early structural changes may be underrecognized. **Objective:** To compare gross muscle (GM) and contractile muscle (CM) measurement-related differences in muscle composition at the L4 level in the upper and lower regions in young adults with NSCLBP. **Methods:** A cross-sectional observational study was conducted involving seven young adults with NSCLBP. Lumbar multifidus (LM) morphology was assessed using Dixon magnetic resonance imaging (MRI) at the upper and lower regions of the L4 vertebral level. Measurements included total cross-sectional area (CSA), fat CSA, muscle CSA, and muscle fat infiltration percentage (MFI%). **Results:** Significant differences were observed between GM and CM measurements across all variables ($p < 0.003$). GM values were consistently higher than CM for total CSA, fat CSA, muscle CSA, and MFI%. At the upper L4 level, total CSA differences ranged from 6.05 to 6.28, whereas larger differences were observed at the lower L4 level (9.06–10.66). MFI% and fat CSA were also significantly greater in GM measurements, indicating substantial contributions from non-contractile tissue. Differences were consistently more pronounced at the lower L4 region. **Conclusions:** GM measurements significantly overestimate LM morphology due to the inclusion of intramuscular fat. Distinguishing contractile from non-contractile tissue provides a more accurate assessment of muscle quality and may improve NSCLBP evaluation and management.

Keywords: Contractile muscle; Chronic low back pain; Cross-sectional area; Gross muscle measurement; Lumbar multifidus; Muscle fat infiltration.

وهم حجم العضلة القطنية متعددة الرغبات: إجمالي مقابل اختلافات الانقباض عند الفقرة الرابعة لدى النساء الشابات المصابات بالأم مزمن غير محدد في أسفل الظهر

الخلاصة

يرتبط ألم أسفل الظهر المزمن غير المحدد (NSCLBP) بتغيرات في شكل عضلات أسفل القطن، وتسرب الدهون، وانخفاض نسيج الانقباض، مما قد يؤثر على الوظيفة والتقييم السريري لدى الأشخاص الأصغر سناً عندما تكون التغيرات الهيكلية المبكرة غير ملحوظة بشكل كافٍ. **الهدف:** مقارنة الفروق المتعلقة بقياس العضلات الكلية (GM) والعضلات الانقباضية (CM) في تركيب العضلات على مستوى L4 في المناطق العليا والسفلى لدى الشباب المصابين ب NSCLBP. **الطرائق:** أجريت دراسة رصدية مقطعية شملت سبعة شباب مصابين ب NSCLBP. تم تقييم شكل العضلة القطنية متعددة الألياف (LM) باستخدام تصوير الرنين المغناطيسي ديكسون (MRI) في المناطق العليا والسفلى من مستوى الفقرة L4. شملت القياسات المساحة العرضية الكلية (CSA)، CSA والدهون، ونسبة تسرب الدهون في العضلات (MFI%). **النتائج:** لوحظت فروق ذات دلالة إحصائية بين قياسات GM و CM عبر جميع المتغيرات ($p < 0.003$). كانت قيم GM أعلى باستمرار من CM بالنسبة لإجمالي CSA، CSA والدهن، CSA والعضلات، و MFI%. في المستوى العلوي L4، تراوحت الفروق الإجمالية في CSA بين 6.05 و 6.28، بينما لوحظت فروقات أكبر في المستوى السفلي (9.06–10.66). كما كانت نسبة MFI% و CSA للدهون أكبر بشكل ملحوظ في قياسات المعدل وراثياً، مما يشير إلى مساهمات كبيرة من الأنسجة غير المنقبضة. كانت الفروقات أكثر وضوحاً باستمرار في المنطقة السفلية L4. **الاستنتاجات:** تبلغ قياسات الوراثة بشكل كبير في تقدير شكل النسيج العضلي بسبب وجود الدهون داخل العضلات. التمييز بين الأنسجة الانقباضية وغير الانقباضية يوفر تقييماً أكثر دقة لجودة العضلات وقد يحسن تشخيص ومعالجة NSCLBP.

*Corresponding author: Rahmat Adnan. Faculty of Sports Science and Recreation, Universiti Teknologi MARA, 40450 Shah Alam, Malaysia; Email: rahmatadnan@uitm.edu.my

Article citation: Mohd Rizal SS, Okilanda A, Rudyanto R, Nasir NAM, Ahmad Risham ARH, Amirudin NSE, Rohim NS, Dzolkapli DZ, Che Man SNE, Anuwar SS, Ismail H, Sulaiman N, Razalli MM, Ismail SI, Adnan R. The Illusion of Lumbar Multifidus Muscle Size: Gross vs. Contractile Differences at L4 in Young Adult Women with Non-Specific Chronic Low Back Pain. *Al-Rafidain J Med Sci.* 2026;11(2):172-177. doi: <https://doi.org/10.54133/ajms.v11i2.3268>

© 2026 The Author(s). Published by Al-Rafidain University. This is an open access journal issued under the CC BY-NC-SA 4.0 license (<https://creativecommons.org/licenses/by-nc-sa/4.0/>).



INTRODUCTION

Low back pain persisting over 12 weeks is classified as chronic (CLBP). It is the leading cause of disability worldwide. Specific structural pathology is absent in most cases, classified as non-specific (NSCLBP). NSCLBP is treated worldwide using interdisciplinary methods that emphasize function restoration over pain relief [1]. Nevertheless, important morphological and functional alterations occur in the lumbar paraspinal muscles. Lower back muscles frequently fail to "fire" before the body

starts limb or trunk motions due to delayed contraction and decreased endurance [2]. Lumbar Multifidus (LM) are constantly involved. A cycle of muscle atrophy, fatty infiltration, and impaired neuromuscular control is a common manifestation of significant morphological and functional changes in the LM. When combined, these alterations weaken the stability of the spine and are the main cause of persistent low back pain [3]. Their impairment is linked to persistent pain and reduced spinal stability. The deep core stabilizers, including the LM and transversus abdominis, are central to this stabilizing

function. Impaired neuromuscular control and core stabilizer muscle dysfunction are the main deficits associated with chronic pain and decreased spinal stability. Muscles such as the LM and transversus abdominis frequently experience delayed activation, atrophy, and fatty infiltration in people with persistent low back pain (LBP) [4]. Direct inter-segmental control and stabilization are provided by small, deep muscles that traverse the spine [5]. Paraspinal muscle imaging has attracted growing interest in phenotyping and treatment planning in spinal conditions [6,7]. Findings remain inconsistent across studies; however, [7] identified three main sources of inconsistency. Mechanisms underlying muscle changes are often overlooked. Confounding factors and imaging protocols also vary substantially between studies. Muscle atrophy, hypertrophy, and sarcopenia are examples of biological causes of muscle changes that are frequently overlooked in favor of merely noting changes in mass or function [8]. When research claims that "protocols vary substantially" and "mechanisms underlying muscle changes in the LM are rarely considered," it highlights a significant scientific barrier to comprehending low back pain. Researchers and physicians continue to disagree on how and why this vital spinal stabilizer deteriorates in individuals with LBP when literature reports that techniques differ, and underlying mechanisms of LM alterations are rarely taken into account [9]. It means that instead of completely comprehending the cause or developing reliable, standardized methods to measure it, researchers frequently simply note that the muscle is degrading [10]. Each proposed mechanism produces a distinct spatial distribution and time course of change. Reflex inhibition causes localized atrophy at a single segment in the acute phase [7]. Denervation results from nerve root compression. For instance, disc herniation and spinal stenosis are common causes. It produces atrophy and fatty infiltration across multiple levels on the affected side [11]. Inflammatory mechanisms cause localized changes that become more diffuse over time. Disuse induces bilateral multilevel atrophy and fat accumulation [6,11]. The multifidus innervation explains this level dependence. All fibers from a single vertebra are innervated by the corresponding nerve root. Even though multifidus fibers are long and span across multiple joints, all the fascicles that pull on a single spinous process receive signals exclusively from the nerve root of that specific vertebra. Changes can therefore extend across up to five levels [7]. Age, sex, and physical activity are established confounders requiring analytical or design control [7,13]. MRI is the gold standard for paraspinal muscle assessment. When evaluating paraspinal muscles, including the multifidus and erector spinae, MRI is regarded as the gold standard. It offers unparalleled soft-tissue contrast and high-resolution imaging, enabling medical professionals to accurately assess muscle health without using radiation [14]. It offers superior resolution and measurement reliability with excellent soft tissue contrast [7,15]. Quantitative measures include total muscle CSA, functional CSA, and fat

CSA. Chemical shift fat-water imaging is the contemporary standard for composition assessment [6,15]. T1- and T2-weighted images remain widely used and provide reliable measures when validated against biopsy findings [7]. A critical distinction exists between GM CSA and CM measurements. GM CSA encompasses both contractile tissue and non-contractile components such as intramuscular fat [13,16]. The distinction resembles judging a battery's remaining charge from its physical size. External dimensions say nothing about internal capacity. Individuals with NSCLBP often show a reduction in CM tissue despite relatively preserved GM size [13,16]. This limitation is probably reflected in studies that show negligible gross size differences between NSCLBP and healthy controls. Conventional CSA cannot detect changes in myofibrillar density, fibrosis, or intramuscular fat [16]. Relying solely on GM CSA therefore risks overestimating muscle integrity [13]. Level-specific measurement is critical, as fatty infiltration at a single level is not representative of the entire lumbar musculature. Average measures may obscure clinically meaningful localized changes [7,16]. The L4 level is of particular relevance. Evidence from MRI-based studies consistently identifies L4-L5 as the most sensitive site for detecting degenerative and atrophic changes in the multifidus, due to its higher load-bearing role and increased range of motion [6,14]. Atrophy or fatty infiltration at this level compromises intervertebral control and shifts the stabilizing load to superficial muscles. Morphological changes may occur early, even in younger individuals. Altered muscle composition can precede observable reductions in gross size [16,17]. This study therefore compares GM and CM measurements at L4 in young individuals with NSCLBP. When comparing young people to healthy controls, GM size measurements at L4 frequently reveal either no discernible difference or only slight atrophy [16]. Only the active, lean muscular tissue's volume or fat cross-sectional area (fCSA) [18]. The aim is to determine whether conventional gross assessments overestimate muscle integrity and obscure meaningful compositional changes at this level.

METHODS

Study design and participants

This observational study is to examine differences between GM CSA measurements at the UL L4 and LL L4 of the lumbar spine level in individuals with NSCLBP. The study was conducted in accordance with the Declaration of Helsinki and received ethical approval from the UiTM Research Ethic Committee (REC/02/2026(ST/MR/43). All participants provided written informed consent prior to enrollment. A total of seven young participants (n= 7) with NSCLBP were recruited with a history of low back pain lasting longer than 12 weeks and presented without any specific underlying spinal pathology. Inclusion criteria for the CLBP group were 1) low back pain

persisting for more than 12 weeks, 2) pain intensity of at least 3/10 on the Numerical Rating Scale, and 3) age between 20 and 30 years. Inclusion criteria for the HC group include 1) no history of low back pain in the preceding 12 months, 2) no chronic musculoskeletal condition, and 3) age-matched to the CLBP group. Exclusion criteria for both groups include prior lumbar surgery, spinal deformity, active infection, malignancy, and contraindications to MRI.

Baseline clinical measurement

Demographic and clinical characteristics were recorded for all participants at baseline. These included age, body mass index (BMI), and symptom duration. Fear-avoidance behavior was assessed using the self-reported Fear-Avoidance Beliefs Questionnaire (FABQ). The FABQ comprises two subscales: physical activity (FABQ-PA) and work (FABQ-W). Higher scores on either subscale indicate greater fear-avoidance beliefs in CLBP [19].

Dixon MRI

Lumbar spine MRI was performed using a 1.5-Tesla scanner (Siemens Magnetom Prisma, Erlangen, Germany) with a 32-channel spine coil. Participants were positioned supine with hips and knees flexed to 45 degrees using a foam wedge to standardize lumbar lordosis. Axial T2-weighted Dixon turbo spin-echo sequences were acquired through the L4 vertebral body using the following parameters: repetition time 600 ms, echo time 11 ms, field of view 260 mm, matrix 512×512 , slice thickness 4 mm, and interslice gap 1 mm. Axial T2-weighted-Dixon sequences were acquired at the L4 vertebral level, providing optimal muscle-to-fat contrast for segmentation. Slice thickness, field of view, repetition time, and echo time were standardized across all scans [20]. Two axial slices were identified per participant: the UL and LL of the L4 vertebral body (Figure 1). All imaging was performed by a single experienced radiographer blinded to the clinical status of participants.

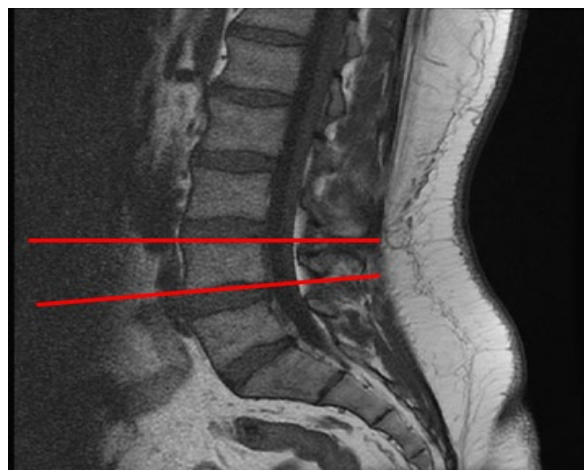


Figure 1: Lumbar L4 level. Slice positioning on a sagittal localizing scan at levels corresponding with UL and LL of the endplate L4.

Image analysis

Bilateral LM segmentation was performed on each axial slice using ImageJ version 1.54 (National Institutes of Health, Bethesda, MD, USA) by a single trained analyst blinded. The LM was identified on each slice according to established anatomical landmarks and was positioned at the deep and superficial LM compartments bilaterally. The GM and CM LM layers were then delineated separately. ROIs were placed at two distinct locations within the LM. The ROIs of the GM LM were taken immediately of the whole LM, the L4 vertebrae including fat (Figure 2), and the CM LM was taken without fat (Figure 3) [7].

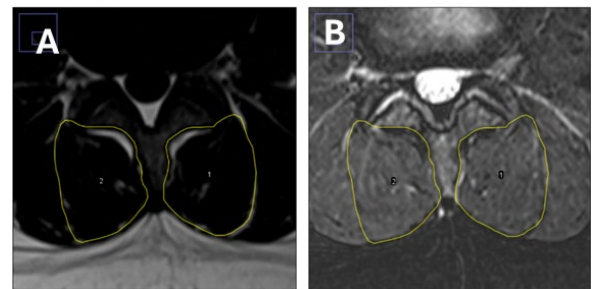


Figure 2: Gross muscle ROI. A) left GM lumbar multifidus, B) Right GM lumbar multifidus. Illustrates the DIXON image demonstrating the ROI for the GM and CM lumbar multifidus, the left imaging showing the fat image and right side showing the water images.

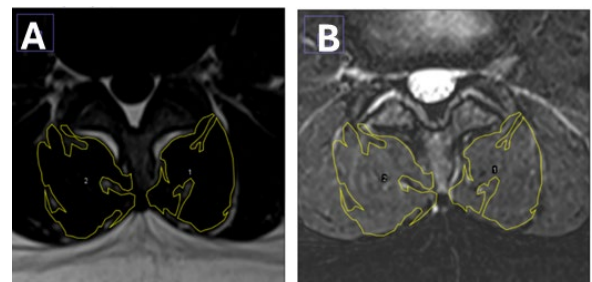


Figure 3: Contractile muscle ROI. A) Left CM lumbar multifidus, B) Right CM lumbar multifidus. Illustrates the DIXON image demonstrating the ROI for the GM and CM lumbar multifidus, the left imaging showing the fat image and right side showing the water images.

Fat signal intensity (Sifat) and water signal intensity (SIwater) were extracted from the ROIs of the LM muscles. Total muscle fat index (MFI) was then estimated for each muscle using the formula $Sifat / (Sifat + SIwater)$. Total CSA was defined as the number of voxels within the ROI multiplied by the voxel size and was computed for both the right and left LM. Lean muscle CSA (muscle tissue free from fat index) was subsequently calculated as $total\ CSA \times (1 - total\ MFI)$. The CSA of fatty tissue was then derived by subtracting muscle CSA from total CSA [21].

Statistical analysis

All analyses were performed using IBM SPSS Statistics version 27 (IBM Corporation, Armonk, NY, USA). Descriptive statistics are reported as mean and standard deviation (SD). The Shapiro-Wilk

test was used to assess normality of distribution. Within-subject differences between GM CSA measurements were examined using paired samples t-tests. These were computed separately for each side (left and right) and each spinal region (upper L4 and lower L4). Variables compared included total muscle CSA, fat CSA, muscle CSA, and MFI. Statistical significance was set at $p < 0.05$.

RESULTS

Seven young adult women with NSCLBP participated in this study, with a mean age of 24.13 ± 1.55 and a mean body mass index (BMI) of 27.12 ± 5.96 kg/m². Participants reported a mean symptom duration of 12.13 ± 10.17 months. Fear-avoidance beliefs assessed using the Fear-Avoidance Beliefs Questionnaire (FABQ) showed a mean score of 15.57 ± 2.37 for the Physical Activity subscale (FABQ-PA) and 15.57 ± 9.07 for the Work subscale (FABQ-W). All baseline demographic and clinical characteristics are presented in Table 1.

Table 1: Demographic participants characteristics

Variable	Result
Age (year)	24.13±1.55
Body Mass Index (kg/m ²)	27.12±5.96
Symptom duration (month)	12.13±10.17
FABQ – PA	15.57±2.37
FABQ – W	15.57±9.071

Values are presented as mean±SD. NRS: Numeric Rating Scale, FABQ-PA: Fear Avoidance Belief Questionnaire – Physical Activity, FABQ-W: Fear Avoidance Belief Questionnaire – Work.

At the UL L4 level, significant differences were observed between GM and CM measurements across all variables. Total CSA differences were significant bilaterally (left mean difference= 6.05, $t(6)$ = 6.587, $p < 0.001$; right: mean difference= 6.28, $t(6)$ = 5.208, $p = 0.002$). Fat CSA also demonstrated significant differences (left mean= 2.24, $p = .001$; right: mean= 2.57, $p = 0.003$). MFI% was significantly higher in GM compared to CM measurements (left mean= 5.65; right mean= 7.62; for both $p < 0.001$). Similarly, muscle CSA showed significant differences (left mean = 3.81, $p < 0.001$; right mean= 3.71, $p = 0.002$). At the LL L4 level, the magnitude of differences was greater. Total CSA differences ranged from 9.06 to 10.66, $p < 0.001$, with the largest difference observed on the right side ($pt(6)$ = 19.164, $p < 0.001$). Fat CSA differences were also significant (means= 3.99–4.58, $p < 0.001$). MFI% demonstrated substantial differences (range= 8.28–9.65, $p < 0.001$), indicating greater fat contribution in GM measurements. Muscle CSA differences were similarly significant (means=5.07–6.07, $p < 0.002$) (Table 2).

DISCUSSION

This study examined differences between GM and CM measurements in young women with NSCLBP. While contractile measurements assess the function, speed, and stiffness of the tissue, gross muscle measurements in young women with NSCLBP show the size, volume, or mass of the muscle [18]. GM

measurements exceeded CM measurements across all variables.

Table 2: Comparisons of gross and contractile lumbar multifidus measurements

Variable	Gross Muscle	Contractile Muscle	p-value
UL L4			
Total CSA (Left)	15.16±3.42	9.11±1.69	<0.001
Total CSA (Right)	14.97±3.62	8.69±1.06	<0.002
Fat CSA (Left)	4.35±1.84	2.12±1.03	<0.001
Fat CSA (Right)	4.51±1.84	1.94±0.94	<0.003
MFI % (Left)	28.61±10.03	22.97±11.24	<0.001
MFI % (Right)	29.79±8.96	22.17±10.33	<0.001
Muscle CSA (Left)	10.81±2.49	7.00±1.46	<0.001
Muscle CSA (Right)	10.46±2.46	6.75±1.16	<0.002
LL L4			
Total CSA (Left)	22.84±5.48	13.78±3.47	<0.001
Total CSA (Right)	24.82±5.98	14.16±5.34	<0.001
Fat CSA (Left)	6.88±2.48	2.89±1.17	<0.001
Fat CSA (Right)	7.56±1.7	2.97±1.28	<0.001
MFI % (Left)	30.09±9.52	21.82±10.33	<0.001
MFI % (Right)	31.45±8.94	21.80±9.98	<0.002
Muscle CSA (Left)	10.89±3.38	10.89±3.38	<0.002
Muscle CSA (Right)	17.26±5.7	11.19±4.8	<0.002

Values are presented as mean±SD. UL L4: Upper Level of Lumbar 4, CSA: Cross sectional area, MFI%: Muscle fat index percentage, LL L4: Lower Level of Lumbar 4.

The differences were statistically significant throughout. This confirms that conventional CSA assessments can overestimate true muscle integrity and morphology. Non-contractile components such as intramuscular fat seem to inflate GM measurements. Muscle quality, not size alone, is a more clinically relevant indicator of lumbar muscle health in NSCLBP [13,16]. A consistent discrepancy was observed between GM and CM measurements across fat CSA, muscle CSA, and MFI%. The gap reflects the substantial contribution of intramuscular fat to gross measurements. This contribution may mask reductions in functional CM tissue. Individuals with NSCLBP often show increased fatty infiltration of the LM despite relatively preserved gross muscle size [16,22]. Relying on traditional CSA ROI therefore may overestimate the true muscle condition. Clinicians using gross CSA diagnosis alone may overlook underlying degeneration that directly impairs muscle function [13]. MFI% was significantly greater in GM measurements compared to CM assessments. This difference reflects the extent to which fat infiltration alters apparent muscle composition. MFI refers to the buildup of fat deposits inside and between muscle fibers [23]. Neuromuscular inhibition and fatty infiltration of the dysfunctional multifidus contribute to persistent pain, spinal instability, and disability that fail to resolve with conventional therapy [24]. Fat infiltration is not merely a structural change. It is closely linked to lumbar dysfunction and the persistence of pain symptoms [25]. Multifidus dysfunction is a recognized contributor to NSCLBP due to its role in segmental spinal stabilization. Targeted methods are needed for the diagnosis and treatment of multifidus dysfunction. Treatment frequently goes beyond conventional pain management and depends on restorative neurostimulation or particular motor control exercises [25]. Reduced CM capacity may compromise neuromuscular control and predispose individuals to recurrent pain episodes [25,26]. Distinguishing CM from non-contractile tissue is

therefore clinically meaningful in muscle evaluation. Differences between GM and CM measurements were consistently greater at the lower L4 region than the upper. This suggests region-specific degeneration at the lower lumbar segment. The lower spine is biomechanically fragile because it bears the greatest weight and manages the highest amount of movement, especially the L4-L5 segments. Fat deposits and connective tissue (referred to as fatty infiltration) replace healthy, contractile muscle fibers at L4 over time or as a result of injury [27]. Evidence from MRI-based studies consistently identifies lower lumbar segments, particularly L4-L5 and L5-S1, as the most sensitive sites for detecting degenerative and atrophic changes in the multifidus [6,14]. Increased mechanical loading and functional demand at these levels are likely to account for this pattern. Structural and compositional changes in paraspinal muscles are also more pronounced at mid-to-lower lumbar levels such as L3-L5 [11,16]. Spatial analyses of multifidus fat infiltration further show that degeneration varies across regions within the muscle, reinforcing the importance of level-specific assessment [28]. Despite the relatively young age of participants, significant alterations in muscle composition were observed. The build-up of fat deposits in muscular tissue deteriorates the functional strength and quality of the muscle [29]. Morphological changes may therefore occur in the early window of young age and in the progression of NSCLBP. Fat infiltration can develop in young individuals with persistent LBP, independently of age-related degeneration [14,17]. Such changes may precede visible muscle atrophy and contribute to ongoing dysfunction if undetected [16]. Precise assessment of muscle composition is therefore necessary for timely intervention. Accurately determining the proportion of lean muscle tissue to fat, water, and bone is necessary for a precise assessment of muscle composition. Conventional metrics like weight or BMI often fail to identify early muscle loss or harmful fat accumulation within the muscles, making prompt intervention essential [30]. These findings reveal a limitation in current assessment practice. Appraising paraspinal muscle health through gross CSA alone is like judging a load-bearing wall from its painted surface. The expression serves as a comparison, emphasizing that gauging GM's size alone and the CSA of paraspinal muscles is an unreliable predictor of spinal health or the underlying cause of back pain since it ignores important parameters like tissue quality and function [31]. Internal degradation is still undetectable. GM assessments frequently overstate muscle characteristics, indicating that the genuine state of muscle health is not reflected effectively by conventional imaging techniques. Muscle mass does not equate to muscle quality. Internal deterioration is frequently overlooked by gross measurements (such as limb circumference or basic muscle volume) [32]. GM measurements evaluate a muscle's total mass, volume, or size (e.g., using bulk DXA scans, tape measures, or skinfolds). Because they are unable to discern between pure muscle tissue, fluid retention,

or fat deposits, they often exaggerate the characteristics of muscles. As a result, they don't accurately represent functional muscle health [33]. Paraspinal muscles are central to spinal stability [7]. Failure to detect reductions in CM tissue may lead to suboptimal rehabilitation. Region-of-interest segmentation, as applied in this study, may provide accurate and clinically measurable muscle morphology [6,14].

Study Limitations

Several limitations require consideration, however. The small sample size ($n = 7$) limits generalisability. The strong statistical significance across all comparisons nonetheless indicates consistent within-subject effects. Future studies with larger samples and longitudinal designs are needed to clarify the progression of muscle changes and their clinical implications.

Conclusion

This study indicated that GM measurements significantly overestimate CM properties due to the inclusion of non-contractile elements such as fat. The findings emphasize the importance of distinguishing between muscle size and muscle quality, particularly at clinically relevant levels such as L4. These results contribute to a more complex understanding of lumbar muscle morphology in NSCLBP and support the need for more precise imaging-based assessment approaches in both research and clinical practice.

ACKNOWLEDGMENTS

The data was presented in ICoSHR Conference, 2026. The authors thank the students of the Sports Education Study Program who participated in this study and the faculty and institution for providing administrative and technical support during the research process.

Conflict of interests

The authors declared no conflict of interest.

Funding source

The authors did not receive any source of funds.

Data sharing statement

The data that supports the findings of this study are available from the corresponding author upon a reasonable request.

REFERENCES

1. Zhou T, Salman D, McGregor AH. Recent clinical practice guidelines for the management of low back pain: a global comparison. *BMC Musculoskelet Disord.* 2024;25(1):344. doi: 10.1186/s12891-024-07468-0.
2. Ghimire N, Lakhe P, Kumar A, Ghimire N, Kutum C, Choudhary D, et al. The impact of paraspinal muscle morphology on functional outcome in patients with degenerative lumbar spine disease undergoing surgery - A

- prospective observational study. *Brain Spine*. 2024;4:104141. doi: 10.1016/j.bas.2024.104141.
3. Ibrahim DRY, Darwesh AA, Mohamed OY, Mabrouk O. Structural and functional changes of multifidus muscle in patients with lumbar radiculopathy: A narrative review. *Egypt Rev Med Health Sci*. 2026;8(1):103-108. doi: 10.21608/ermhs.2026.472022.1134.
 4. Güler MA, Demirdel E, Gezer İA, Nayman A, Tezcan EA. Effect of core stabilization exercises on lumbar multifidus morphology and functional outcomes in chronic non-specific low back pain: a randomized controlled trial. *BMC Musculoskelet Disord*. 2025;27(1):83. doi: 10.1186/s12891-025-09433-x.
 5. Fallahasady E, Rahmanloo N, Seidi F, Rajabi R, Bayattork M. The relationship between core muscle endurance and functional movement screen scores in females with lumbar hyperlordosis: a cross-sectional study. *BMC Sport Sci Med Rehabil*. 2022;14(1):182. doi: 10.1186/s13102-022-00567-2.
 6. Li X, Dong F, Chen X, Luo X, Wang W. Radiological frontiers in understanding paraspinal muscle pathophysiology in chronic low back pain. *Front Med*. 2025;12:1653876. doi: 10.3389/fmed.2025.1653876.
 7. Hodges PW, Bailey JF, Fortin M, Battié MC. Paraspinal muscle imaging measurements for common spinal disorders: review and consensus-based recommendations from the ISSLS degenerative spinal phenotypes group. *Eur Spine J*. 2021;30(12):3428-3441. doi: 10.1007/s00586-021-06990-2.
 8. Wang K, Wang X, Wang Y. Factors, mechanisms and improvement methods of muscle strength loss. *Front Cell Dev Biol*. 2024;12:1509519. doi: 10.3389/fcell.2024.1509519.
 9. Wang K, Deng Z, Chen X, Shao J, Qiu L, Jiang C, et al. The role of multifidus in the biomechanics of lumbar spine: Musculoskeletal modeling study. *Bioengineering (Basel)*. 2023;10(1). doi: 10.3390/bioengineering10010067.
 10. Pinto SM, Cheung JPY, Samartzis D, Karppinen J, Zheng YP, Pang MYC, et al. Are morphometric and biomechanical characteristics of lumbar multifidus related to pain intensity or disability in people with chronic low back pain after considering psychological factors or insomnia? *Front Psychiatry*. 2022;13:2022. doi: 10.3389/fpsy.2022.809891.
 11. Chen X, Cui P, Li Y, Wang Y, Lu S. Links among MRI features in paraspinal muscles, inflammatory processes, and related back pain in patients with lumbar disc herniation. *J Spine*. 2024;7(1):e1310. doi: 10.1002/jsp2.1310.
 12. Kang JI, Kim SY, Kim JH, Bang H, Lee IS. The location of multifidus atrophy in patients with a single level, unilateral lumbar radiculopathy. *Ann Rehabil Med*. 2013;37(4):498-504. doi: 10.5535/arm.2013.37.4.498.
 13. Liu S, Reitmaier S, Mödl L, Yang D, Zhang T, Becker L, et al. Quality of lumbar paraspinal muscles in patients with chronic low back pain and its relationship to pain duration, pain intensity, and quality of life. *Eur Radiol*. 2025;35(6):3652-3660. doi: 10.1007/s00330-024-11236-y.
 14. Varma N, Bopaiah H, Sakalecha AK, H LR, D NS. MRI analysis of paraspinal muscle morphology in patients with chronic low back pain: A case-control study. *Cureus*. 2025;17(10):e94072. doi: 10.7759/cureus.94072.
 15. Masi S, Rye M, Roussac A, Naghdi N, Rosenstein B, Bailey JF, et al. Comparison of paraspinal muscle composition measurements using IDEAL fat-water and T2-weighted MR images. *BMC Med Imag*. 2023;23(1):48. doi: 10.1186/s12880-023-00992-w.
 16. Kalli I, Niglas M, Naeini MK, Freidin M, Thomas L, Menni C, et al. Paraspinal muscle quality in chronic low back pain: a systematic review and meta-analysis of muscle atrophy and fat infiltration. *Eur Spine J*. 2025;1-19. doi: 10.1007/s00586-025-09454-z.
 17. Yang Y, Lin WS, Wen HQ, Luo XW, Zhou X, Zou FY, et al. Quantitative evaluation of risk factors for low back pain in young patients using synthetic magnetic resonance imaging and proton density fat fraction analyses. *Ther Adv Chronic Dis*. 2024;15:20406223241293260. doi: 10.1177/20406223241293260.
 18. Lee H, Lee S, Kim C, Jeon K. A Comparison of contractile properties and acute muscle fatigue response in adult females with non-specific chronic low back pain. *Bioengineering (Basel)*. 2024;11(12). doi: 10.3390/bioengineering11121202.
 19. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain*. 1993;52(2):157-168. doi: 10.1016/0304-3959(93)90127-b.
 20. Adnan R, Danneels L, Willems T, Van Oosterwijck S, Dhondt E, Goubert D, et al. A novel subgrouping approach to low back pain: Exploring differences in lumbar muscle morphology and function—A cross-sectional study. *J Back Musculoskelet Rehabil*. 2026;10538127261416001. doi: 10.1177/10538127261416001.
 21. Goubert D, De Pauw R, Meeus M, Willems T, Cagnie B, Schoupe S, et al. Lumbar muscle structure and function in chronic versus recurrent low back pain: a cross-sectional study. *Spine J*. 2017;17(9):1285-1296. doi: 10.1016/j.spinee.2017.04.025.
 22. Ballatori AM, Shahrestani S, Nyayapati P, Agarwal V, Krug R, Han M, et al. Influence of patient-specific factors when comparing multifidus fat infiltration between chronic low back pain patients and asymptomatic controls. *JOR Spine*. 2022;5(4):e1217. doi: 10.1002/jsp2.1217.
 23. Avesani CM, de Abreu AM, Ribeiro HS, Brismar TB, Stenvinkel P, Sabatino A, et al. Muscle fat infiltration in chronic kidney disease: a marker related to muscle quality, muscle strength and sarcopenia. *J Nephrol*. 2023;36(3):895-910. doi: 10.1007/s40620-022-01553-0.
 24. Lorio MP, Lewandowski KU, Lavelle W, Essig DA, Yue J, Ratliff J, et al. ISASS Recommendations and coverage criteria for restorative neurostimulation for multifidus dysfunction, lumbar region: Coverage indications, limitations, and/or medical necessity—An ISASS 2025 Guideline Update. *Int J Spine Surg*. 2025;19(6):760. doi: 10.14444/8833.
 25. Abd-Elsayed A, Kurt E, Kollenburg L, Hasoon J, Wahezi SE, Storlie NR. Lumbar multifidus dysfunction and chronic low back pain: overview, therapies, and an update on the evidence. *Pain Pract*. 2025;25(5):e70044. doi: 10.1111/papr.70044.
 26. Moens M, Genot LV, Van Gestel F, Piliotis JG, Billot M, Roulaud M, et al. Multifidus fat infiltration in patients with persistent spinal pain syndrome type II treated with spinal cord stimulation: a preliminary report. *Journal of clinical medicine*. 2025;14(9):3124. doi: 10.3390/jcm14093124.
 27. Mardulyn T, Delafontaine A, Jissendi P, Fabeck L. Lumbar muscle fatty infiltration and atrophy in patients with low back pain and degenerative spinal pathologies: A CT imaging study. *J Clin Med*. 2025;14(6). doi: 10.3390/jcm14062125.
 28. Khattab K, Dzieszinski LK, Zhou J, Scheffler A, Fields AJ, O'Neill CW, et al. Chronicity and physical activity levels independently associate with spatial patterns of multifidus fat infiltration in chronic low back pain patients. *Eur Spine J*. 2025;1-8. doi: 10.1007/s00586-025-09660-9.
 29. Sheenam N, Gonnade NM, Gaur R, Abins TK, Ghosh A, Hussain R. The hidden architecture of back pain: ultrasound-based lumbar multifidus pennation angle analysis - a cross-sectional pilot study. *BMC Musculoskelet Disord*. 2026;27(1). doi: 10.1186/s12891-026-09604-4.
 30. Holmes CJ, Racette SB. The utility of body composition assessment in nutrition and clinical practice: An overview of current methodology. *Nutrients*. 2021;13(8). doi: 10.3390/nu13082493.
 31. Suo M, Zhang J, Sun T, Wang J, Liu X, Huang H, et al. The association between morphological characteristics of paraspinal muscle and spinal disorders. *Ann Med*. 2023;55(2):2258922. doi: 10.1080/07853890.2023.2258922.
 32. Wang L, Lu S, Feng J, Ma K, Duan F, Wang R, et al. Muscle imaging assessments in sarcopenia: A statement from China National Center for Orthopedics (NCO) and the East Meets West Action Group of the European Calcified Tissue Society (ECTS). *Calcif Tissue Int*. 2025;116(1):144. doi: 10.1007/s00223-025-01447-w.
 33. Friedl KE, Silva AM, Bergouignan A, Yamada Y, Potter AW. Skeletal muscle mass is not compositionally uniform: the role of fluid compartments in health and performance monitoring. *Eur J Clin Nutr*. 2026. doi: 10.1038/s41430-026-01770-x.