

# Morphological Changes in Paraspinal Muscles on MR Imaging in Chronic Low Back Pain Patients with Unilateral Lumbar Disc Herniation: A Cross-sectional Study

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## ABSTRACT

**Introduction:** Low Back Pain (LBP) caused by Lumbar Disc Herniation (LDH) is a major cause of chronic disability worldwide. The management of LDH depends on clinical assessment and Magnetic Resonance Imaging (MRI) findings. Atrophy or fatty replacement of paraspinal muscles has a negative impact on the outcomes of both conservative and surgical treatment for LBP due to LDH.

**Aim:** To analyse the morphological changes in paraspinal muscles using MRI in patients with chronic LBP and unilateral LDH.

**Materials and Methods:** A cross-sectional study was conducted at Vardhman Mahavir Medical College and Safdarjung Hospital in New Delhi, India, from December 2020 to May 2022. A total of 59 patients, aged 21-65 years, with chronic LBP and unilateral LDH on MRI, were included in the study. The morphological changes in the Multifidus Muscle (MM) and Erector Spinae Muscle (EM) were analysed. The Cross-Sectional Area (CSA) of MM and EM, as well as the Signal Intensity (SI) of MM on the

herniated side of the lumbar disc, were recorded and compared to the contralateral side at the same level. Continuous variables were expressed as mean±SD, median, and interquartile range, while categorical variables were presented as numbers and percentages. The Mann-Whitney test was used to compare continuous variables, and a p-value of <0.05 was considered statistically significant.

**Results:** The mean age of the study participants was 39.17±12.82 years, with 59.3% male and 40.7% female participants. The CSA of MM on the side of disc herniation was 5.84±1.53 cm<sup>2</sup>, compared to 6.52±1.69 cm<sup>2</sup> on the unaffected side (p=0.02). The CSA of EM on the side of unilateral disc herniation was 13.08±3.04 cm<sup>2</sup>, compared to 14.29±3.22 cm<sup>2</sup> on the unaffected side (p=0.01). The SI of MM on the affected side was 175.84±100.99, compared to 147.70±83.50 on the unaffected side (p=0.02).

**Conclusion:** Chronic LBP due to unilateral LDH resulted in a reduction in the Cross-Sectional Area (CSA) and fatty infiltration of the ipsilateral paraspinal muscles.

**Keywords:** Atrophy of multifidus muscle, Erector spinae muscle atrophy, Fatty atrophy of multifidus muscle, Magnetic resonance

## INTRODUCTION

The complaint of LBP in patients is among the top five causes of hospital visits worldwide, affecting 80% of the population at least once in their lifetime. It affects people of all age groups, imposing limitations on regular daily activities and creating a significant economic burden [1-4]. LBP is classified as either acute (lasting less than three months) or chronic (lasting more than three months) [5,6]. Altered biomechanics affecting ligaments, zygapophyseal joints, intervertebral discs, paravertebral musculature, and spinal nerve roots of the lumbar spine leads to LBP. The most common non-idiopathic cause of LBP is LDH [4,5,7]. Increased static and kinetic stress, superimposed with inadequate reinforcement by the Posterior Longitudinal Ligament (PLL) in the lumbar spine, explains why LDH is more common than cervical and thoracic disc herniations [5,7]. The various pathophysiological factors responsible for pain in LDH include disc desiccation, neural compression, local inflammation, ligament stretching, and paraspinal muscle sprain [5-7].

LDH causing LBP is diagnosed based on clinical and radiological findings. Clinical examination plays a crucial role in differentiating between spinal versus non-spinal causes, discogenic versus non-discogenic aetiology, and determining the level and extent of pathology [7]. Magnetic Resonance Imaging (MRI) is a highly sensitive and specific diagnostic modality for confirming LDH

as the cause of LBP. It also helps rule out other potential causes of LBP, such as congenital vertebral anomalies, infective spondylodiscitis, inflammatory spondyloarthropathies, trauma, and neoplastic conditions. In patients with LDH, MRI not only confirms the diagnosis but also facilitates evaluation of disc morphology, the status of the posterior ligamentous complex, the degree of spinal stenosis (central canal, lateral recess, and neural foramina), and any morphological changes in the paraspinal muscles [8].

Management of LBP involves conservative treatment and, when necessary, surgical intervention. Physical therapy is an integral component of both conservative management and post-surgical rehabilitation to improve overall patient outcomes [9]. The focus of physical therapy is to correct vertebral alignment, improve zygapophyseal joint mobility, and strengthen the paraspinal muscles. The outcome of physical therapy is significantly influenced by the condition of the paraspinal muscles (erector spinae and multifidus). Atrophy or fatty replacement of the paraspinal muscles adversely affects the outcomes of both conservative and surgically managed LDH [10]. MRI is crucial for analysing these morphological changes in the paraspinal muscles [11,12].

Previous studies have emphasised the morphological changes in the paraspinal muscles due to chronic disc herniation

or associated disuse atrophy [10-12]. Understanding the pathomechanism of paraspinous muscle atrophy due to disc herniation is clinically relevant to comprehend the importance of rehabilitation programs and the outcomes of conservative or surgical management.

The aim of this study was to objectively analyse the morphological changes in the multifidus and erector spinae muscles on MRI in patients with chronic LBP due to unilateral LDH.

## MATERIALS AND METHODS

This cross-sectional study was conducted at Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, India, from December 2020 to May 2022, following Institutional Ethical Committee clearance (IEC/VMMC/SJH/Thesis/2020-11/CC-206). Informed consent was obtained.

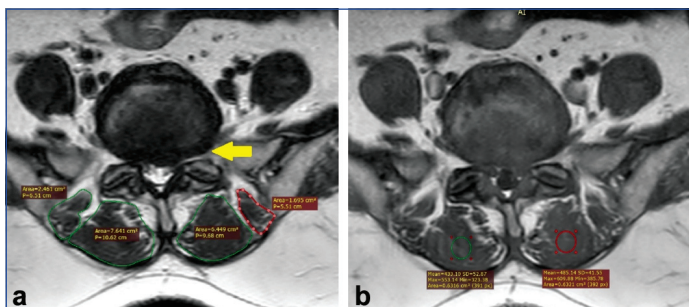
**Sample size calculation:** At a 95% confidence level and 90% power, considering the mean Cross-Sectional Area (CSA) of the multifidus as  $5.58 \pm 1.96 \text{ cm}^2$  on the painful side and  $7.22 \pm 1.97 \text{ cm}^2$  on the non-painful side by Yaltirik K et al., [13], the sample size was calculated as 59.

**Inclusion criteria:** Those patients, aged 21 to 65 years, who came to the chosen study centre with complaints of chronic LBP, reason due to unilateral LDH, as diagnosed on MRI and consented for participation were included in the study.

**Exclusion criteria:** Patients with systemic illnesses (diabetes mellitus, hypothyroidism), prior spinal surgery, chronic anaemia, and non-ambulatory status were excluded. Infective, inflammatory, congenital, traumatic, and neoplastic causes of LBP on MRI were also excluded. A total of 59 patients were included in the study.

## Procedure

**MRI acquisition and image analysis:** The patients underwent MRI of the lumbosacral spine using a 3-Tesla MRI scanner, General Electric (GE) Discovery MR 750W. T2-weighted and T1-weighted sagittal images were obtained from the right to left neural foramen level. Axial T1-weighted and axial T2-weighted images were obtained at the L1-2 to L5-S1 disc levels from the superior to inferior borders of the neural foramen. On the axial T2-weighted images at the level of the unilateral herniated discs, the Cross Sectional Area (CSA) of the multifidus and erector spinae muscles were measured by constructing a closed polygon around the outer margins of individual muscles on both sides [Table/Fig-1a]. Similarly, on axial T1-weighted images at the level of unilateral disc herniation, the Signal Intensity (SI) of the multifidus muscle was measured by placing an elliptical Region of Interest (ROI) on bilateral multifidus muscles, equidistant from the spinous process [Table/Fig-1b].



**[Table/Fig-1]:** A 54-year-old lady presented with Low Back Pain (LBP) radiating to left lower limb for four months. a. T2W axial MRI of the Lumbosacral spine at L5-S1 level demonstrate left paracentral and foraminal disc herniation causing compression upon the left traversing S1 and exiting L5 nerve root compression (arrow). The CSA of left Multifidus Muscle (MM) measures  $6.45 \text{ cm}^2$ , while the right MM measures  $7.61 \text{ cm}^2$  and the CSA of left erector spinae measures  $1.70 \text{ cm}^2$ , while the right erector spinae measure  $2.45 \text{ cm}^2$ ; indicating atrophy of left multifidus and erector spinae. b. T1W axial MRI at the same level demonstrated increased SI in left MM (mean: 485) as compared to the right multifidus (mean 433), indicating fatty infiltration of left MM.

## STATISTICAL ANALYSIS

The data obtained was entered into an MS excel spreadsheet, and statistical analysis was performed using Statistical Package for Social Sciences (SPSS) version 21.0. Quantitative variables like age of patients, CSA, and SI of muscles were expressed as mean  $\pm$  SD, median, and interquartile range. Categorical variables such as the gender of patients and level of LDH were expressed as numbers and percentages (%). Continuous variables were compared using the Mann-Whitney test, and a p-value of  $<0.05$  was considered statistically significant.

## RESULTS

The age range of the study participants was 21-65 years, with a mean age of  $39.17 \pm 12.82$  (mean  $\pm$  SD). The gender distribution was 35 (59.3%) male and 24 (40.7%) female [Table/Fig-2]. Out of the 59 patients, 32 (54%) had LDH on the right-side, while 27 (46%) had LDH on the left-side. The predominant level of unilateral disc herniation was at the L4-5 level, followed by the L3-4 and L5-S1 levels. Eight patients had multi-level unilateral disc herniation [Table/Fig-3].

Demographic details of patient	No.	%	
<b>Age group</b>			
21-30 years	18	30.5	Mean age: $39.17 \pm 12.82$ years Age range: 21-65 years
31-40 years	16	27.1	
41-50 years	11	18.6	
51-60 years	10	16.9	
>60 years	4	6.8	
<b>Gender</b>			
Male	35	59.3	Male: Female 1.45:1
Female	24	40.7	

**[Table/Fig-2]:** Age and gender distribution of study subjects (n=59).

Level of involvement	No.	%
L1-L2	1	1.7
L2-L3	3	5.1
L3-L4	14	23.7
L4-L5	22	37.3
L5-S1	11	18.6
L3-L4, L4-L5	5	8.5
L4-L5, L5-S1	2	3.4
L2-L3, L3-L4, L4-L5	1	1.7

**[Table/Fig-3]:** Level of disc involvement in study subjects (n=59).

There was a statistically significant difference ( $p=0.02$ ) observed in the Cross-Sectional Area (CSA) measured in the multifidus muscle on the side of disc herniation ( $5.84 \pm 1.53 \text{ cm}^2$ ) compared to the unaffected side ( $6.52 \pm 1.69 \text{ cm}^2$ ). Similarly, the CSA of the erector spinae muscle on the side of unilateral disc herniation was  $13.08 \pm 3.04 \text{ cm}^2$ , compared to the unaffected side ( $14.29 \pm 3.22 \text{ cm}^2$ ) with a p-value of 0.01 [Table/Fig-4].

Muscle	Parameters (CSA)	Affected side ( $\text{cm}^2$ )	Unaffected side ( $\text{cm}^2$ )	p-value
Multifidus	Mean $\pm$ SD	$5.84 \pm 1.53$	$6.52 \pm 1.69$	<b>0.02</b>
	Median (IQR)	5.83 (5.03-6.95)	6.57 (5.60-7.62)	
	Range	1.97-8.57	2.20-10.07	
Erector Spinae	Mean $\pm$ SD	$13.08 \pm 3.04$	$14.29 \pm 3.22$	<b>0.01</b>
	Median (IQR)	12.63 (10.76-4.5)	8.18-24.87	
	Range	8.38-22.67	(12.08-15.72)	

**[Table/Fig-4]:** Comparison of Cross-sectional Area (CSA) of multifidus and Erector Spinae Muscles (EM) between affected and unaffected side by Mann-Whitney U test.

The Signal Intensity (SI) of the affected multifidus muscle was compared to the unaffected side on the axial T1-weighted image to observe fatty infiltration. A statistically significant difference was observed in the SI of the multifidus muscle on the affected side [Table/Fig-5].

Signal Intensity (SI)	Affected side	Unaffected side	p-value	Test
Mean±SD	175.84±100.99	147.70±83.50	0.02	Mann-Whitney U test
Median (IQR)	146.46 (114.02-202.55)	129.63 (91.88-171.18)		
Range	64.97-569.31	30.67-465.83		

**[Table/Fig-5]:** Comparison of Signal Intensity (SI) between affected and unaffected side of Multifidus Muscle (MM).

However, when comparing the CSA of the affected multifidus muscle and erector spinae muscle at the single level of unilateral LDH to multiple levels of unilateral disc herniation, the difference was found to be statistically insignificant [Table/Fig-6].

Muscle	Parameters (CSA)	Single level disc (cm <sup>2</sup> )	Multiple level disc (cm <sup>2</sup> )	p-value
Multifidus	Mean±SD	5.74±1.52	6.50±1.55	0.25
	Median (IQR)	5.81 (4.83-6.91)	6.68 (5.16-7.93)	
	Range	1.97-8.58	4.06-8.57	
Erector Spinae	Mean±SD	12.89±3.05	14.24±2.89	0.16
	Median (IQR)	12.44 (20.68-14.43)	14.04 (12.07-15.21)	
	Range	8.38-20.67	10.46-20.18	

**[Table/Fig-6]:** Comparison of Cross-Sectional Area (CSA) of multifidus and Erector Spinae Muscle (EM) between single and multiple level discs. Mann-Whitney U Test was used

## DISCUSSION

LBP is one of the leading global causes of years lived with disability. In recent years, the prevalence of LBP has increased, especially in younger individuals, with LDH being one of the common causes [14-17]. The therapeutic goals are to provide a pain-free life to the patient and restore physical and functional capacity, which is essential for leading a healthy routine life [10,18]. The paraspinous muscles (multifidus and erector spinae) are predominantly responsible for providing stability and strength to the lumbar spine. Degeneration of the paraspinous muscles can result in spinal instability and predispose to further injury [19-22]. The multifidus muscle is located in the deeper planes of the paraspinal compartment. It attaches laterally to the lumbar mammillary processes on their posterior aspects (or sacrum in the lower lumbar spine) and inserts medially on the spinous processes of the vertebrae above. The multifidus muscle has a short lever arm and unisegmental innervation.

Contraction of the unilateral multifidus muscle leads to axial rotation of the spine to the contralateral side without trunk flexion [23]. The Erector Spinae Muscle (EM) is superficial to the Multifidus Muscle (MM) and consists of two parts: the longissimus and iliocostalis muscles. The longissimus muscle originates from the lumbar intermuscular aponeurosis, the medial part of the sacro-pelvic surface of the ilium, and the posterior sacroiliac ligament. It inserts into the transverse and accessory processes of L1-L5 vertebrae. The iliocostalis part of the EM is located laterally to the longissimus. It originates from the thoracolumbar fascia, the medial end of the iliac crest, and the lateral crest of the sacrum, and inserts at the tips of the transverse processes of L1-L4 vertebrae and the angle of the 5<sup>th</sup>-12<sup>th</sup> ribs. The EM has a longer lever arm than the MM, so it provides most of the momentum [24]. The EM is more crucial in producing lateral flexion of the trunk during standing and walking. It generates torque in all lumbar spine movements, including lateral bending, flexion-extension, and axial twisting motions [24-26].

Both the MM and EM are innervated by the dorsal ramus of the spinal nerve at the same level. Therefore, long-term disc herniation causes atrophy of both the MM and EM at the same level. Yaltirik K et al., observed a reduction in the CSA of the MM and EM in patients with LBP with radiculopathy and single-level LDH compared to patients with LBP without radiculopathy and LDH in their retrospective study [13]. In the systematic review by Fortin M and Macedo LG, they also found that the paraspinous muscles were significantly reduced in size on the symptomatic side of patients with chronic unilateral LBP. The pooled standardised mean difference in the CSA of the MM between the symptomatic and asymptomatic sides was -0.43 (95% confidence interval -0.68, -0.18), which was statistically significant [27].

The MM has unisegmental innervation, while the EM has multisegmental innervation [28,29]. This may explain the atrophy of the MM due to chronic dorsal ramus compression by a herniated disc at a single level. However, it does not explain the atrophy of the EM. Lee HI et al., determined that the CSA of the EM, rather than the MM, was a prognostic factor in chronic LBP [30]. The extensor momentum of the trunk is primarily generated by the EM, as it has a longer lever arm compared to the MM. Hence, they postulated that failure to adequately react to sudden loading of the lumbar spine may increase the risk of tissue damage in the EM, resulting in atrophy in patients with chronic LBP. Several other studies have provided supportive evidence that atrophy predominantly occurs in the MM in patients with chronic LBP due to disc herniation [31-33]. Long-term compression of the dorsal ramus by a unilateral herniated disc and biomechanical failure in response to lumbar loading could be possible reasons for the atrophy of both the EM and MM in chronic LBP patients in this study.

Contradicting the results of the present of unilateral atrophy of the MM and EM in ipsilateral disc herniation, various studies have observed bilateral atrophy of the MM in patients with chronic unilateral LBP and concluded that MM atrophy is related to the spinal level of pain, but not the side of pain [34-36]. In an experimental porcine model, unilateral electrical stimulation of the lumbar disc led to electromyographic responses not only in the ipsilateral MM but also to a lesser extent in the contralateral MM. This reflex activity of the MM in response to electrical stimulation of the lumbar disc was reduced with saline or lidocaine injected into the facet joint. It was considered that chronic alteration of afferent input in this segmental reflex activity led to bilateral inhibition and, eventually, bilateral MM atrophy in chronic disc disease [35,36]. However, since the authors compared the CSA of paraspinous muscles on both sides in patients with unilateral disc herniation, rather than comparing them with age-matched healthy controls, they may have overlooked lesser degrees of atrophy on the contralateral side. The lack of long-term follow-up in the present study could be another reason why only unilateral atrophy of the multifidus was observed in these patients with unilateral disc herniation.

In this study, among patients with unilateral LDH, the authors not only observed a reduction in the CSA of the ipsilateral MM and EM, but also noticed fatty infiltration of the ipsilateral MM in the form of increased muscle SI on T1-weighted images. LBP leads to altered biomechanics and eventually altered neuromuscular functioning of the paraspinous muscles, predominantly the MM. This, in turn, leads to changes in muscle histology in the form of fatty infiltration, which is more common in adults than in adolescents [37-40]. The reduction in CSA of the paraspinous muscles is said to occur before fatty infiltration [38,40]. An increase in muscle fat content reduces the muscles' contractility



and makes them prone to segmental instability [37,38]. Specific muscle training to enhance the functional activity of stabiliser paraspinal muscles can prevent severe fatty infiltration of the muscle [41-43].

### Limitation(s)

In the present study, the authors did not determine the correlation between the severity of pain/disability and the degree of reduction in CSA and fatty infiltration of the paraspinal muscles. They only evaluated the overall CSA of the MM and EM, not the functional fat-free CSA. Also, follow-up MRI was not performed, after targeted physical therapy to determine the outcome of the paraspinal muscles.

### CONCLUSION(S)

Chronic LBP caused by unilateral disc herniation leads to a reduction in CSA and fatty infiltration of the ipsilateral paraspinal muscles, which can result in segmental spine instability. Therefore, specific targeted muscle training should be instituted to prevent atrophy and fatty replacement of the paraspinal muscles, in order to achieve better outcomes in the conservative and surgical management of LBP.

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**PLAGIARISM CHECKING METHODS:** [\(Lain H et al.\)](#)

- Plagiarism X-checker: Jul 11, 2023
- Manual Googling: Sep 15, 2023
- iThenticate Software: Oct 19, 2023 (13%)

**ETYMOLOGY:** Author Origin**EMENDATIONS:** 6**AUTHOR DECLARATION:**

- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Jul 06, 2023**Date of Peer Review: **Sep 07, 2023**Date of Acceptance: **Oct 23, 2023**Date of Publishing: **Dec 01, 2023**