Association of BMI with lumbar spinal canal stenosis in patients with low back pain

Farzana Shegufta[1*], Shahjamal Khan[2], Rezwana Sobhan[3], Rabeya Khatoon[4]

ARTICLE INFO

Received: 14 Apr 2026
Accepted: 23 Apr 2026
Published Online: 5 May 2026

DOI:

Volume: 9, Number: 2, Page: 189-193

e-ISSN: 2789-5912
ISSN: 2617-0817

*Corresponding author

ABSTRACT

Background: Lumbar spine degenerative disease is a common cause of low back pain and disability worldwide. Increased body mass index (BMI) has been implicated as a potential risk factor for lumbar disc degeneration, yet the pattern of involvement across different lumbar levels in relation to BMI remains underexplored. Objective: To evaluate the distribution of lumbar disc degeneration across different spinal levels and examine its association with BMI in adult patients undergoing MRI. **Materials & Methods:** This retrospective study was conducted in the Radiology Department of BIRDEM General Hospital from January 2024 to December 2024. A total of 70 adult patients with MRI-confirmed degenerative changes in the lumbar spine were included. Exclusion criteria comprised prior spine surgery, spinal infections, malignancy, metabolic bone disease, or incomplete imaging/BMI data. Demographic data, height, weight, and clinical indications for MRI were recorded. BMI was calculated and categorized according to WHO criteria. MRI scans were reviewed by a consultant radiologist using standard lumbar spine imaging protocols to identify disc bulges, central and paracentral herniations at levels L1–L2 through L5–S1. Data were analyzed using descriptive statistics and chi-square tests to assess associations between BMI and disc pathology. **Results**: The mean age of participants was 43.15 ± 8.37 years, with 52.9% females. BMI distribution showed 50.0% normal weight, 35.7% overweight, 11.4% obese, and 2.9% underweight. Disc pathology was most prevalent at L4–L5 (71.4% disc bulge, 7.1% central and paracentral herniation) and L5–S1 (35.7% disc bulge, 25.7% central herniation, 7.1% paracentral herniation). Upper lumbar levels (L1–L2 and L2–L3) were largely normal. Higher BMI was associated with increased frequency of disc bulges and herniations, particularly at lower lumbar levels. **Conclusion**: Lumbar disc degeneration predominantly affects lower lumbar segments (L4–L5 and L5–S1), and elevated BMI is associated with increased severity and frequency of multi-level disc pathology. Weight management may play a key role in reducing the burden of degenerative lumbar spine disease.

Keywords: *Lumbar spine, disc degeneration, disc bulge, herniation, body mass index, MRI*

1. *Associate Professor, Department of Radiology and Imaging, BIRDEM General Hospital Dhaka Bangladesh (ORCID: 0000-0001-6092-6946)*
2. *Professor, Department of Endocrinology and Metabolism, Enam Medical College, Savar, Bangladesh*
3. *Associate Professor, Department of Endocrinology and Metabolism, Enam Medical College, Savar, Bangladesh*
4. *Assistant Professor, Department of Radiology and Imaging, BIRDEM General Hospital, Dhaka, Bangladesh*

INTRODUCTION

Low back pain (LBP) is one of the leading causes of disability worldwide and constitutes a major public health challenge across all age groups. It is frequently chronic or recurrent and is associated with substantial functional limitation, reduced quality of life, and significant socioeconomic burden [1,2]. Globally, more than 500 million individuals were affected by LBP in 2017, accounting for a considerable proportion of years lived with disability (YLDs) [3]. Among the structural etiologies of LBP, degenerative disorders of the lumbar spine—particularly lumbar spinal canal stenosis and degenerative spondylolisthesis—are increasingly recognized as important contributors to persistent pain, neurological deficits, and impaired mobility. Lumbar spinal canal stenosis is characterized by progressive narrowing of the spinal canal, leading to compression of neural elements and clinical manifestations such as low back pain, radiculopathy, neurogenic claudication, and sensory disturbances [4]. The condition is predominantly degenerative and has been strongly associated with advancing age, abnormal mechanical loading, genetic susceptibility, smoking, metabolic syndrome, and psychosocial factors [5,6]. Degenerative spondylolisthesis, most commonly occurring at the L4–L5 or L5–S1 levels, further aggravates canal narrowing through anterior vertebral slippage, facet joint hypertrophy, and ligamentous thickening, thereby compounding neural compression and symptom severity [7]. Obesity has emerged as a key modifiable risk factor in the multifactorial pathogenesis of LBP and degenerative lumbar spine disease. Numerous epidemiological studies have demonstrated that individuals with elevated body mass index (BMI) have a significantly higher prevalence and severity of LBP compared with those of normal weight [8-10]. From a biomechanical perspective, excess body weight increases axial loading on the lumbar spine, elevates intradiscal pressure, and accelerates intervertebral disc degeneration, facet joint arthropathy, and ligamentum flavum hypertrophy—hallmark pathological features of lumbar spinal canal stenosis [11-12]. Obesity-related alterations in posture and sagittal spinopelvic alignment may further contribute to segmental instability and the progression of degenerative spondylolisthesis [13]. Beyond mechanical stress, obesity contributes to spinal degeneration through complex biological mechanisms. Adipose tissue–derived cytokines and adipokines, including leptin and adiponectin, promote chronic low-grade systemic inflammation, impair disc cell metabolism, and accelerate extracellular matrix degradation within the intervertebral disc [14,15]. Additionally, metabolic dysregulation, insulin resistance, and endothelial dysfunction may compromise nutrient diffusion to the avascular disc, enhancing hypoxia-induced degeneration and apoptosis [16]. More recently, spinal epidural lipomatosis—excessive fat accumulation within the spinal canal—has been proposed as a potential mediator linking elevated BMI to symptomatic lumbar spinal stenosis through direct mechanical compression of neural structures [17,18]. Despite accumulating evidence supporting a relationship between obesity, disc degeneration, and LBP, the association between BMI and lumbar spinal canal stenosis remains controversial. While

several imaging-based and population studies have reported a positive correlation between increased BMI and degenerative spinal changes, including stenosis [7,19], others have failed to demonstrate a consistent or independent association after adjustment for age, sex, and genetic factors [20,21]. These conflicting findings highlight persistent knowledge gaps and underscore the need for further focused investigation. Given the rising global prevalence of obesity particularly in Asian populations—and the parallel increase in LBP-related disability, clarifying the relationship between BMI and lumbar spinal canal stenosis is of considerable clinical and public health importance [22,23]. A clearer understanding of this association may facilitate improved risk stratification, guide preventive strategies, and support individualized management approaches for patients presenting with low back pain.

METHODS & MATERIALS

This retrospective observational study was conducted in the Department of Radiology at BIRDEM General Hospital over a one-year period from January 2024 to December 2024. A convenience sampling method was used, and a total of 70 adult patients who met the eligibility criteria were included. Patients aged 18 years or older who were referred for lumbosacral spine MRI and demonstrated degenerative changes on imaging were enrolled. Patients with a history of spinal surgery, sacroiliac arthritis, spinal fractures, spinal infections including tuberculosis, active malignancy or metastatic disease, metabolic bone disorders, incomplete imaging studies, or missing height or weight data were excluded from the study.

All MRI examinations were performed using a 1.5-Tesla Philips Ignetia MRI scanner following a standardized lumbosacral spine protocol. The imaging protocol included T1-weighted and T2-weighted sequences obtained in both sagittal and axial planes. Images were reviewed by a consultant radiologist with extensive experience in spinal imaging. Each MRI scan was systematically evaluated for degenerative changes at lumbar levels from L1–L2 to L5–S1. Disc degeneration was assessed using established radiological criteria, including the Pfirrmann grading system, with specific attention to disc bulges, central and paracentral disc herniations, and vertebral alignment abnormalities.

Demographic and clinical data were collected from medical records and, when necessary, through direct patient interviews. Clinical indications for MRI included low back pain, sciatica, walking difficulty, neurogenic claudication, and a history of trauma. Occupational information was also recorded to explore potential associations between work-related physical demands and patterns of lumbar spinal degeneration. Height and weight measurements were obtained to calculate body mass index (BMI) using the standard formula: BMI = weight (kg) / height (m²). Patients were classified according to the World Health Organization BMI categories as underweight, normal weight, overweight, obese class I, obese class II, and obese class III.

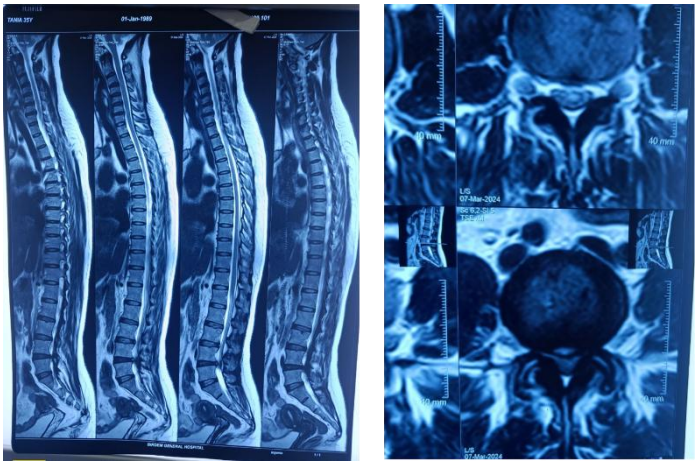

Figure 1 Disc protrusion with spinal canal stenosis (8mm) of a obese female patient.

Sagittal and axial T2W sequences of MRI of lumbar spine of 35 years old female obese patient showing posterior disc protrusion, flaval hypertrophy causing spinal canal stenosis, bilateral lateral recess and neural foraminal narrowing with compression of exiting L4 and traversing L5 nerve roots on both sides at L4-L5 level (*Figure 1*).

Statistical Analysis

Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS), version 25. Descriptive statistics were used to summarize patient characteristics and MRI findings, with continuous variables expressed as means and standard deviations and categorical variables presented as frequencies and percentages. Associations between BMI categories and disc pathology at individual lumbar levels were analyzed using the chi-square test, and a p-value of <0.05 was considered statistically significant. An exploratory logistic regression analysis was also performed to assess the predictive value of BMI for multilevel disc degeneration, although its interpretation was limited by the sample size and data distribution. Written informed consent was obtained from all participants or their legal guardians, and the study posed no direct risk to patient safety as it involved retrospective image review without invasive procedures or radiation exposure.

RESULTS

A total of 70 patients were included in the study. The mean age was 43.15 ± 8.37 years, with the majority of participants falling in the 40–44 years (22.9%) and 30–34 years (21.4%) age groups. Regarding gender distribution, 52.9% were female and 47.1% were male, showing a slightly higher proportion of females. In terms of BMI, 50.0% of participants had normal weight, 35.7% were overweight, 11.4% were obese, and 2.9% were underweight, with a mean BMI of 24.39 ± 4.12 kg/m². Overall, the study population was middle-aged, slightly female-predominant, and predominantly of normal to overweight BMI (*Table I*).

Table I
Distribution of Subjects according to Age, Gender, and BMI (*n* = 70).

Demographic characteristics	Number	Percentage
Age group (years)		
30–34	15	21.4
35–39	13	18.6
40–44	16	22.9
45–49	10	14.3
50–54	7	10.0
55–60	9	12.8
Mean ± SD	43.15 ± 8.37	30-60
Gender		
Male	33	47.1
Female	37	52.9
BMI (kg/m²)		
<18.5 (Underweight)	2	2.9
18.5–24.9 (Normal)	35	50.0
25.0–29.9 (Overweight)	25	35.7
≥30 (Obese)	8	11.4
Mean ± SD	24.39 ± 4.12	

Among the 70 patients, the majority of discs at the upper lumbar levels (L1–L2 and L2–L3) were normal, with 94.3% and 85.7% unaffected, respectively. Disc bulges were more common at these levels but remained low (4.3% at L1–L2 and 12.9% at L2–L3), while central and paracentral herniations were rare. At the mid-lumbar level (L3–L4), normal discs accounted for 64.3%, with a higher proportion of disc bulges (30.0%) and minor occurrences of central and paracentral herniations (2.9% each). The lower lumbar levels (L4–L5 and L5–S1) showed the greatest pathology. At L4–L5, 71.4% had disc bulges, and central and paracentral herniations were observed in 7.1% each, leaving only 14.3% normal. At L5–S1, 35.7% showed disc bulges, 25.7% had central herniation, 7.1% paracentral herniation, and 31.4% remained normal. Overall, disc pathology increased at lower lumbar levels, with L4–L5 and L5–S1 being the most affected levels (*Table II*).

Table II
Distribution of Disc Pathology by Lumbar Level (*n* = 70).

Lumbar Level	Normal n (%)	Disc Bulge n (%)	Central Herniation n (%)	Paracentral Herniation n (%)
L1–L2	66 (94.3)	3 (4.3)	1 (1.4)	0 (0.0)
L2–L3	60 (85.7)	9 (12.9)	1 (1.4)	0 (0.0)
L3–L4	45 (64.3)	21 (30.0)	2 (2.9)	2 (2.9)
L4–L5	10 (14.3)	50 (71.4)	5 (7.1)	5 (7.1)
L5–S1	22 (31.4)	25 (35.7)	18 (25.7)	5 (7.1)

Among the underweight group (N = 2), all cases were observed at the L5–S1 level (100%), with no disc pathology detected at the upper lumbar levels. In the normal weight group (N = 35), the highest frequency of disc bulge/herniation was noted at L4–L5 (20 cases, 57.1%), followed by L3–L4 (9 cases, 25.7%) and L2–L3 (5 cases, 14.3%). Minimal involvement was seen at L1–L2 (1 case, 2.9%), while no cases were recorded at L5–S1 in this group. For overweight patients (N = 25), disc pathology was most commonly found at L4–L5 (10 cases, 40.0%), followed by L5–S1 (7 cases, 28.0%) and L3–L4 (6 cases, 24.0%). Lower frequencies were observed at L1–L2 (1 case, 4.0%) and L2–L3 (1 case, 4.0%). In the obese class I group (N = 8), the majority of cases involved the L4–L5 level (4 cases, 50.0%), followed by L5–S1 (3 cases, 37.5%) and L3–L4 (1 case, 12.5%). No disc bulge or herniation was identified at L1–L2 or L2–L3 levels in this group. Overall, across all BMI categories, lower lumbar levels (L4–L5 and L5–S1) showed the highest burden of disc bulge/herniation, with an increasing shift toward L5–S1 involvement as BMI increased (*Table III*).

Table III
Distribution of Disc bulge/Herniation by BMI Category (*n* = 70).

Lumbar Level	Underweight N=2	Normal Weight N=35	Overweight N=25	Obese I N=8
L1–L2	0	1	1	0
L2–L3	0	5	1	0
L3–L4	0	9	6	1
L4–L5	0	20	10	4
L5–S1	2	0	7	3

DISCUSSION

In this study, a total of 70 patients were included, with a mean age of 43.15 ± 8.37 years. The majority of participants were aged 40–44 years (22.9%) and 30–34 years (21.4%), and females slightly predominated (52.9%) over males (47.1%). The BMI distribution showed that 50% of participants had normal weight, 35.7% were overweight, 11.4% were obese, and 2.9% were underweight, with a mean BMI of 24.39 ± 4.12 kg/m². This demographic profile is consistent with previous studies indicating that middle-aged, overweight, and obese individuals are more prone to lumbar spine degenerative changes [3]. Aziz et al. [3] reported a high prevalence of degenerative lumbar spine changes among overweight

and obese individuals, with L4–L5 and L5–S1 being the most commonly affected levels. Interestingly, their study also highlighted a significant association between higher BMI and disc pathology at the upper lumbar levels (L1–L2 and L2–L3), supporting emerging evidence that overweight individuals may experience early degenerative changes in these segments [24]. Similarly, our analysis revealed significant associations at L1–L2 ($\chi^2 = 13.30$, $p = 0.0207$) and L2–L3 ($\chi^2 = 16.85$, $p = 0.0048$), suggesting that both axial loading and pro-inflammatory adipokines may contribute to early degeneration. In our population, the majority of discs at upper lumbar levels remained normal (L1–L2: 94.3%, L2–L3: 85.7%), with disc bulges and herniations being relatively rare. Mid-lumbar (L3–L4) levels exhibited moderate pathology, whereas lower lumbar levels (L4–L5 and L5–S1) showed the highest prevalence of disc degeneration. At L4–L5, 71.4% of discs exhibited bulges, and central and paracentral herniations were each observed in 7.1% of cases. At L5–S1, disc bulges were present in 35.7%, central herniation in 25.7%, and paracentral herniation in 7.1%, with only 31.4% remaining normal. These findings corroborate global data showing the predominance of degenerative changes at lower lumbar levels due to cumulative mechanical stress and age-related disc degeneration [25,26]. The distribution of disc pathology according to BMI demonstrated that lower lumbar levels (L4–L5 and L5–S1) had the highest burden across all BMI categories. Notably, among underweight patients (N=2), pathology was confined to L5–S1, while in normal-weight participants (N=35), the highest frequency was at L4–L5 (57.1%), followed by L3–L4 (25.7%) and L2–L3 (14.3%). Overweight patients (N=25) showed maximum involvement at L4–L5 (40%), with L5–S1 and L3–L4 also affected. Obese class I patients (N=8) had the majority of pathology at L4–L5 (50%) and L5–S1 (37.5%). These results are consistent with prior studies demonstrating a positive association between BMI and multi-level lumbar disc degeneration [27,28]. Interestingly, while lower lumbar levels showed the highest frequency of disc pathology, we did not observe a statistically significant association with BMI at L4–L5 or L5–S1. This may be due to the high prevalence of degenerative changes at these levels among symptomatic individuals, limiting the ability to detect differences across BMI groups [3,29]. Furthermore, genetic predisposition may play a more prominent role in disc degeneration among younger populations, which could explain variability in upper versus lower lumbar involvement [30]. Overall, our findings support the established link between elevated BMI and lumbar spine degeneration, particularly at lower lumbar levels, while highlighting that upper lumbar segments may also be affected in overweight individuals. This reinforces the need for early preventive measures, weight management, and ergonomic interventions to mitigate the risk of multi-level lumbar disc pathology.

CONCLUSION

Disc disease progressively develops from the upper to lower lumbar levels, with L4–L5 and L5–S1 being the most damaged, according to this research of 70 adult patients with lumbar spine degenerative alterations. The influence of overweight and obesity on lumbar spine degeneration was highlighted by the considerable percentage of patients with higher BMI who had disc bulges and herniations, especially at L4–L5 and L5–S1. Although early degenerative alterations were seen in overweight people, upper lumbar levels (L1–L2 and L2–L3) were maintained. Overall, the results point to a link between higher BMI and a higher likelihood and severity of multi-level lumbar disc degeneration. These findings highlight the significance of preventative measures and weight control in lowering the incidence of degenerative lumbar spine illness.

REFERENCES

1. Knezevic NN, Candido KD, Vlaeyen JWS, Van Zundert J, Cohen SP. Low back pain. Lancet. 2021;398(10294):78–92.
2. GBD 2021 Low Back Pain Collaborators. Global, regional, and national burden of low back pain, 1990–2020, its attributable risk factors, and projections to 2050: a systematic analysis. Lancet Rheumatol. 2023;5(6):e316–29.
3. Aziz R, Khan MI, Afzal F, Siddique F, Nisar M. Clinico-radiologic patterns and BMI association in degenerative disease of the lumbosacral spine: a retrospective MRI study from a tertiary care hospital in Pakistan. Prof Med J. 2025;32(12):1713–8.
4. Katz JN, Harris MB. Lumbar spinal stenosis. N Engl J Med. 2008;358(8):818–25.
5. Samartzis D, Karppinen J, Chan D, Luk KDK, Cheung KMC. The association of lumbar intervertebral disc degeneration on magnetic resonance imaging with body mass index in overweight and obese adults: a population-based study. Arthritis Rheum. 2012;64(5):1488–96.
6. Sekiguchi M, Yonemoto K, Kakuma T, et al. Relationship between lumbar spinal stenosis and psychosocial factors: a multicenter cross-sectional study (DISTO project). Eur Spine J. 2015;24(10):2288–94.
7. Kalichman L, Kim DH, Li L, Guermazi A, Berkin V, Hunter DJ. Spondylolysis and spondylolisthesis: prevalence and association with low back pain in the adult community-based population. Spine. 2009;34(2):199–205.
8. Webb R, Brammah T, Lunt M, Urwin M, Allison T, Symmons D. Prevalence and predictors of intense, chronic, and disabling neck and back pain in the UK general population. Spine. 2003;28(11):1195–202.
9. Leboeuf-Yde C, Kyvik KO, Bruun NH. Low back pain and lifestyle. Part II—Obesity: information from a population-based sample of 29,424 twin subjects. Spine. 1999;24(8):779–83.
10. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between obesity and low back pain: a meta-analysis. Am J Epidemiol. 2010;171(2):135–54.
11. Adams MA, Freeman BJ, Morrison HP, Nelson IW, Dolan P. Mechanical initiation of intervertebral disc degeneration. Spine. 2000;25(13):1625–36.
12. Bakker EWP, Verhagen AP, van Trijffel E, Lucas C, Koes BW. Spinal mechanical load as a risk factor for low back pain: a systematic review of prospective cohort studies. Spine. 2009;34(8):E281–93.
13. Schuller S, Charles YP, Steib JP. Sagittal spinopelvic alignment and body mass index in patients with degenerative spondylolisthesis. Eur Spine J. 2011;20(5):713–9.
14. Ruiz-Fernández C, Francisco V, Pino J, et al. Molecular relationships among obesity, inflammation and intervertebral disc degeneration: are adipokines the common link? Int J Mol Sci. 2019;20(8):2030.
15. Chen HH, Hsu HT, Liao MH, Teng MS. Effects of sex and obesity on LEP variant and leptin level associations in intervertebral disc degeneration. Int J Mol Sci. 2022;23(20):12275.
16. Illien-Jünger S, Lu Y, Qureshi SA, et al. Chronic ingestion of advanced glycation end products induces degenerative spinal changes and hypertrophy in aging pre-diabetic mice. PLoS One. 2015;10(2):e0116625.
17. D'Agostino V, Petrera MR, Tedesco G, Pipola V, Ponti F, Spinnato P. Could spinal epidural lipomatosis be the hallmark of metabolic syndrome on the spine? A literature review with emphasis on etiology. Diagnostics. 2023;13(2):322.
18. Rigsby RK, Barnes S, Sabaté J, Oyoyo U, Chowdhury S, Peters EM. Correlation of spinal epidural fat volume with body mass index: a longitudinal study. Clin Imaging. 2023;98:61–6.
19. Liuke M, Solovieva S, Lamminen A, Luoma K, Leino-Arjas P, Luukkonen R, Riihimäki H. Disc degeneration of the lumbar spine in relation to overweight. Int J Obes. 2005;29(8):903–8.
20. de Schepper EIT, Damen J, van Meurs JB, et al. The association between lumbar disc degeneration and low back pain: the influence of age, gender, and individual radiographic features. Spine. 2010;35(5):531–6.
21. Videman T, Battié MC, Gibbons LE, et al. Associations between back pain history and lumbar MRI findings. Spine. 2009;34(6):582–8.
22. Pan WH, Yeh WT, Weng LC. Epidemiology of metabolic syndrome in Asia. Asia Pac J Clin Nutr. 2008;17(Suppl 1):37–42.
23. GBD 2021 Adult BMI Collaborators. Global, regional, and national prevalence of adult overweight and obesity, 1990–2021, with forecasts to 2050: a forecasting

study for the Global Burden of Disease Study 2021. Lancet. 2025;405(10481):813–38.
24. Heuch I, Heuch I, Hagen K, Zwart J. Overweight and obesity as risk factors for chronic low back pain: A new follow-up in the HUNT Study. BMC Public Health. 2024; 24(1):2618.
25. Hartvigsen J, Hancock MJ, Kongsted A, Louw Q, Ferreira ML, Genevay S, et al. What low back pain is and why we need to pay attention. Lancet. 2018; 391(10137):2356-67
26. Wu D, Hoy D, March L, Smith E, Cross M, Fransen M, et al. FRI0552 Global, regional, and national burden of low back pain, 1990–2019: A systematic analysis for the Global Burden of Disease Study 2019. Ann Rheum Dis. 2020; 79(Suppl 1):877-8.
27. Ajiboye LO, Gbadegesin SA, Oboirien M, Alimi M, Oyeniyi EO, Sadiq NA. Body mass index and severity of lumbar degenerative disc disease in adult patients using Oswestry Disability Index: any correlation? East Afr Orthop J. 2019; 13(1):34-41
28. Samartzis D, Karppinen J, Mok F, Fong DY, Luk KD, Cheung KM. A population-based study of juvenile disc degeneration and its association with overweight and obesity, low back pain, and diminished functional status. J Bone Joint Surg Am. 2011; 93(7):662-70
29. Ding Y, Ruan S, Liu L, Zhang X, Chen R, Chen Q, Xu S. A new four-area method for evaluating biochemical changes in lumbar facet joint degeneration on T2* mapping. BMC Musculoskeletal Disorders. 2025 May 15;26(1):480.
30. Deguchi T, Hashizume H, Terao C, Yamada H, Nakamura M, Matsumoto M, et al. A longitudinal population-based study identifies THBS2 as a susceptibility gene for intervertebral disc degeneration. Eur Spine J. 2024; 33(9):3334-42