Journal of Health, Wellness and Community Research
ISSN: 3007, 0570

OPEN ACCESS

JHWCR

Type: Original Article
Published: 10 September 2025
Volume: III, **Issue**: XII
DOI: https://doi.org/10.61919/4nfh1e87

Correspondence
✉ Wajiha Zafar, wajihazafar707@gmail.com

Received 20, 08, 25 **Accepted** 02, 09, 2025

Authors' Contributions
Concept: WZ; Design: VM; Data Collection: SA, LM, RA, SAh; Analysis: MA; Drafting: WZ.

Declarations
No funding was received for this study. The authors declare no conflict of interest. The study received ethical approval. All participants provided informed consent.

"Click to Cite"

Association of Bulging Disc in Elderly Obese and Non-Obese Patients on MRI

Wajiha Zafar[1], Vinsa Maryam[2], Samia Arif[2], Laiba Malik[2], Rimsha Alam[2], Samia Ahsan[2], Muhammad Abdullah[2]

1 Master of Science in Diagnostic Ultrasound, University of Management and Technology, Lahore, Pakistan
2 BS in Medical Imaging and Ultrasonography, University of Management and Technology, Lahore, Pakistan

ABSTRACT

Background: *Lumbar disc bulge is a common degenerative spinal condition contributing significantly to low back pain in elderly populations. Obesity has been implicated as a modifiable risk factor through mechanical loading and metabolic inflammation, yet the independent relationship between body mass index (BMI) and MRI-defined disc bulge remains unclear.* ***Objective****: To determine the association between obesity and lumbar disc bulge among elderly patients undergoing MRI for low back pain, and to evaluate age- and sex-related influences on this relationship.* ***Methods****: A cross-sectional observational study was conducted on 101 adults aged ≥40 years presenting with low back pain to five tertiary hospitals in Lahore, Pakistan. Lumbar spine MRIs were evaluated for the presence and type of disc bulge by blinded radiologists. Obesity was defined as BMI ≥30 kg/m². Associations were analyzed using chi-square tests and logistic regression adjusted for age and sex. Statistical significance was set at $p < 0.05$.* ***Results****: Of 101 participants (mean age 51.4 ± 9.1 years; 63.4% female), 40.6% were obese and 54.5% exhibited lumbar disc bulge. Disc bulge occurred in 65.9% of obese versus 46.7% of non-obese participants (unadjusted OR = 2.22; 95% CI 0.97–5.12; p = 0.057). After adjustment for age and sex, obesity remained a borderline predictor (adjusted OR = 2.11; 95% CI 0.95–4.69; p = 0.065). No significant associations were found with age or sex. Non-compressive disc bulge was the most frequent MRI finding (12.9%), followed by lumbar spondylosis (16.8%).* ***Conclusion****: Lumbar disc bulge is prevalent among both obese and non-obese elderly individuals, with a higher yet statistically borderline frequency in the obese group. These findings suggest that while obesity may accelerate disc degeneration, age-related and biomechanical factors also play key roles. Preventive strategies should integrate weight management with postural and physical activity interventions.*

Keywords
Lumbar Disc Bulge, Obesity, MRI, Degenerative Spine Disease, Elderly, Low Back Pain

INTRODUCTION

Low back pain (LBP) remains among the leading causes of years lived with disability worldwide, with degenerative changes of the intervertebral discs constituting a principal substrate of morbidity across aging populations (1). Biologically, disc degeneration reflects a cytokine-mediated imbalance between catabolic and anabolic processes within the extracellular matrix, culminating in annulus fibrosus weakening, nucleus pulposus dehydration, neovascularization, and neo-innervation that together predispose to bulging or herniation and pain (2).

Foundational and contemporary work converges on a life-course pattern in which age-related loss of disc water content and structural integrity increases progressively across lumbar levels, amplifying susceptibility to protrusion and radiculopathy in later adulthood (3). Clinically, lumbar disc bulge or herniation typically presents with axial LBP that may radiate along L4–S1 dermatomes, with sensory disturbance and weakness when nerve root compromise ensues; magnetic resonance imaging (MRI) provides the preferred radiation-free modality to characterize these pathologies and related canal or foraminal compromise (4).

Epidemiologically, multiple contributors shape the risk landscape for degenerative lumbar disc disease, including hereditary factors, cumulative mechanical loading, and systemic metabolic milieu (2). Among modifiable exposures, obesity has garnered sustained attention given its high and rising prevalence and its established associations with cardiometabolic disease and musculoskeletal disorders (5,6). Biomechanically, excess body mass elevates axial compressive forces and intradiscal pressure, while adiposity-related inflammation may potentiate matrix catabolism and pain sensitization, providing plausible pathways from obesity to structural disc changes observable on MRI (2).

Emerging imaging research further suggests that reductions in lumbar disc height—quantifiable on radiographs—correlate with bulging or herniation on cross-sectional imaging, reinforcing the link between mechanical and morphological degeneration (7). Nevertheless, despite biologic plausibility, the independent association between obesity and MRI-defined lumbar disc bulge remains uncertain because age and sex are strong confounders of both adiposity and degenerative change, and prior studies often combine mixed spinal regions or outcomes, rely on tertiary summaries rather than primary imaging data, or lack adjustment for key covariates (1,2,7).

These uncertainties are particularly salient in South Asian clinical settings, where demographic aging, sedentary work patterns, and shifting adiposity distributions converge, yet MRI-based evidence on obesity and lumbar disc bulge in older adults is sparse. A focused, image-based evaluation in symptomatic patients undergoing standardized lumbar MRI can clarify whether obesity, defined by body mass index (BMI) thresholds, confers an excess odds of disc bulge beyond age- and sex-related risk (4). Accordingly, in a cross-sectional study of adults aged ≥40

years presenting with LBP to tertiary hospitals, we examined the association between obesity (exposure) and MRI-defined lumbar disc bulge (outcome), comparing obese versus non-obese participants while accounting for demographic confounding (4–7). We hypothesized that obesity (BMI ≥30 kg/m²) would be associated with higher odds of lumbar disc bulge on MRI relative to non-obesity among adults ≥40 years evaluated for LBP (primary hypothesis), with exploratory assessment of distribution by sex and age strata to contextualize effect magnitude and precision (1,2,7).

MATERIAL AND METHODS

This cross-sectional observational study was conducted to evaluate the association between obesity and MRI-defined lumbar disc bulge among elderly patients presenting with low back pain. The study was performed between January and December 2024 in the radiology departments of five tertiary care hospitals in Lahore, Pakistan, namely Jinnah Hospital, Services Hospital, General Hospital, Sir Ganga Ram Hospital, and the Punjab Institute of Cardiology. These centers were selected because they provide comprehensive MRI services and cater to a diverse patient population, ensuring representation of various age and socioeconomic strata (8).

Participants were recruited through non-probability convenience sampling from outpatients referred for lumbar spine MRI due to symptoms of low back pain, radiculopathy, or suspected degenerative disc disease. Eligible participants were adults aged 40 years or older, of either sex, who underwent MRI of the lumbar spine and had available anthropometric measurements enabling calculation of body mass index (BMI). Individuals with a history of spinal trauma, surgery, congenital spinal deformities, neoplasms, infections, or claustrophobia were excluded to avoid confounding pathologies unrelated to degenerative changes. Written informed consent was obtained from all participants before enrollment, and each was informed about study objectives and confidentiality protocols.

All MRI examinations were performed on 1.5 Tesla scanners using standardized lumbar protocols that included T1-weighted and T2-weighted sagittal and axial sequences. Disc morphology was assessed by two consultant radiologists blinded to BMI and demographic data. A disc bulge was operationally defined as circumferential extension of the disc margin beyond the edges of the adjacent vertebral endplates involving more than 25% of the disc circumference without focal protrusion or extrusion, consistent with North American Spine Society (NASS) criteria (9). Disagreements between readers were resolved by consensus. Obesity was defined according to World Health Organization standards as BMI ≥30 kg/m², while non-obese individuals were defined as those with BMI < 30 kg/m² (10). Age, sex, and clinical presentation were recorded for each participant using a structured data sheet.

To minimize measurement bias, anthropometric data were collected by trained technologists using calibrated equipment, and MRI interpretations were standardized through consensus meetings before data collection. Potential confounding by age and sex was addressed analytically through stratified analysis and multivariable modeling. Missing values for demographic or imaging variables were checked for randomness; when limited (<5%), cases were excluded listwise.

The minimum required sample size was estimated using an assumed prevalence of disc bulge of 50% among obese and 30% among non-obese patients, a confidence level of 95%, and 80% power, yielding a target sample of at least 92 subjects; 101 participants were finally enrolled to enhance precision (11). Statistical analyses were conducted using SPSS version 24.0 (IBM Corp., Armonk, NY, USA). Continuous variables such as age and BMI were summarized as mean ± standard deviation, and categorical variables (sex, obesity status, disc bulge presence) as frequencies and percentages. The primary analysis evaluated the association between obesity and lumbar disc bulge using chi-square and logistic regression tests to estimate odds ratios (OR) with 95% confidence intervals (CIs).

Secondary analyses examined the relationship between disc bulge and demographic factors (age and sex) and explored MRI subtype distributions. Statistical significance was set at $p < 0.05$ for two-tailed tests.

Ethical approval for this study was granted by the institutional review boards of the participating hospitals. All procedures adhered to the Declaration of Helsinki. Participant anonymity was preserved through coded identifiers, and all data were securely stored with restricted access. Quality control included double data entry and random re-evaluation of 10% of imaging reports to ensure reproducibility and data integrity (12).

RESULTS

Among 101 elderly participants (mean ± SD age = 51.4 ± 9.1 years), 40.6% were obese, and 54.5% demonstrated lumbar disc bulge on MRI. Females were predominant (63.4%), though sex was not significantly associated with disc bulge ($p = 0.987$). Disc bulge prevalence was higher among obese individuals (65.9%) than non-obese (46.7%), corresponding to an unadjusted odds ratio of 2.22 (95% CI 0.97–5.12, $p = 0.057$). Age-specific analysis revealed peak disc bulge occurrence between 41 and 50 years (58.3%), decreasing modestly with advancing age, but without significant difference across groups ($p = 0.365$).

MRI pattern distribution demonstrated that non-compressive bulges were the most frequent (12.9%), followed by lumbar spondylosis (16.8%) and compressive disc bulges (5.9%). A strong statistical association was observed between specific MRI categories and disc bulge presence ($p < 0.001$), indicating that structural degenerative changes—especially compressive and neural-compromise variants—tend to co-occur with bulging phenomena. In logistic regression modeling adjusting for age and sex, obesity maintained an independent though borderline-significant association with lumbar disc bulge (adjusted OR = 2.11, 95% CI 0.95–4.69, $p = 0.065$).

Neither age (OR = 1.08 per 10 years, $p = 0.621$) nor sex (OR = 0.97, $p = 0.941$) contributed significantly to prediction. Collectively, these findings indicate a clinically meaningful trend suggesting that obesity may nearly double the odds of MRI-defined lumbar disc bulge among elderly adults, though larger samples are required for statistical confirmation. The study enrolled 101 elderly participants aged 41 to 85 years, with a mean age of 51.4 ± 9.1 years. The majority (48.5%) were between 41 and 50 years of age, indicating that degenerative disc changes become radiologically evident even in the fifth decade of life. Females constituted 63.4% of the cohort (n = 64), while males comprised 36.6% (n = 37).

Obesity (BMI ≥ 30 kg/m²) was observed in 41 participants (40.6%), whereas 60 (59.4%) were classified as non-obese. Disc bulge was identified on MRI in 55 patients (54.5%), suggesting that over half of the elderly individuals presenting with low back pain demonstrate measurable degenerative disc morphology.

When comparing obese and non-obese participants, disc bulge was present in 27 of 41 obese individuals (65.9%) and in 28 of 60 non-obese individuals (46.7%). Although the crude difference approached statistical significance ($\chi^2 = 3.62$, $p = 0.057$), this trend suggests a potential positive association between increased BMI and lumbar disc bulge. The unadjusted odds ratio (OR = 2.22; 95% CI 0.97–5.12) indicates that obese participants had approximately double the likelihood of disc bulge compared with their non-obese counterparts. After adjusting for age and sex in

multivariable logistic regression, the association remained borderline significant (adjusted OR = 2.11; 95% CI 0.95–4.69; p = 0.065), implying that the observed effect is clinically relevant though statistically inconclusive given the sample size. Age-stratified analysis revealed that the frequency of disc bulge was highest among those aged 41–50 years (58.3%), with gradual decline observed in the older age groups: 54.5% among those aged 51–60 years and 47.4% beyond 60 years. However, the association between age and disc bulge was not statistically significant (p = 0.365), consistent with literature suggesting that while degeneration progresses with age, the morphological expression of bulging may plateau after midlife. Gender distribution analysis showed nearly identical proportions of disc bulge among males (54.1%) and females (54.7%), with no significant association (p = 0.987).

Table 1. Demographic and Clinical Characteristics of Study Participants (n = 101)

Variable	Categories	Frequency (n)	Percentage (%)	Mean ± SD	p-value
Age (years)	41–45	21	20.8	51.4 ± 9.1	—
	46–50	28	27.7		
	51–55	18	17.8		
	56–60	11	10.9		
	61–65	11	10.9		
	≥66	12	11.9		
Sex	Male	37	36.6	—	—
	Female	64	63.4	—	0.274
Obesity Status (BMI ≥30 kg/m²)	Obese	41	40.6	32.8 ± 2.5	—
	Non-obese	60	59.4	26.7 ± 1.8	—
Disc Bulge on MRI	Present	55	54.5	—	—
	Absent	46	45.5	—	—

Table 2. Association Between Obesity, Age, and Disc Bulge (n = 101)

Variable	Disc Bulge Present n (%)	Disc Bulge Absent n (%)	Total n	Unadjusted OR (95% CI)	p-value
Obesity	27 (65.9%)	14 (34.1%)	41	2.22 (0.97–5.12)	0.057
Non-Obesity	28 (46.7%)	32 (53.3%)	60	Reference	—
Age Group (years)					
41–50	28 (58.3%)	21 (41.7%)	49	1.31 (0.55–3.09)	0.365
51–60	18 (54.5%)	15 (45.5%)	33	1.12 (0.44–2.87)	
>60	9 (47.4%)	10 (52.6%)	19	Reference	—
Sex					
Male	20 (54.1%)	17 (45.9%)	37	0.99 (0.44–2.20)	0.987
Female	35 (54.7%)	29 (45.3%)	64	Reference	—

Table 3. MRI Findings Distribution Among Participants (n = 101)

MRI Finding	Frequency (n)	Percentage (%)	Disc Bulge n (%)	Odds Ratio (95% CI)	p-value
Non-compressive disc bulge	13	12.9	13 (100%)	—	<0.001
Compressive disc bulge	6	5.9	6 (100%)	—	
Disc bulge causing neural compromise	4	4.0	4 (100%)	—	
Lumbar spondylosis	17	16.8	0 (0%)	Reference	—
Degenerative & post-surgical changes	5	5.0	1 (20%)	—	
Other findings (combined)	56	55.4	31 (55.4%)	—	

Table 4. Multivariable Logistic Regression: Predictors of Lumbar Disc Bulge

Variable	Adjusted OR (95% CI)	p-value
Obesity (BMI ≥ 30 kg/m²)	2.11 (0.95–4.69)	0.065
Age (per 10-year increase)	1.08 (0.79–1.46)	0.621
Male sex	0.97 (0.42–2.24)	0.941

After adjusting for age and sex, obesity showed a borderline-significant association with the presence of lumbar disc bulge (adjusted OR = 2.11; 95% CI 0.95–4.69; p = 0.065).

Regarding MRI findings, degenerative pathologies were diverse. Non-compressive disc bulge was the most frequent imaging diagnosis (12.9%), followed by lumbar spondylosis (16.8%) and compressive disc bulge (5.9%). Disc bulges associated with neural compromise accounted for 4.0% of cases. The chi-square test revealed a highly significant relationship between MRI diagnostic category and the presence of disc bulge (p < 0.001), underscoring that disc protrusion and related compressive features tend to coexist within a degenerative spectrum.

Overall, the quantitative data demonstrate that while disc bulging was common among both obese and non-obese elderly individuals, its higher proportion among obese participants aligns with the hypothesized biomechanical and inflammatory mechanisms linking excess weight to degenerative spinal pathology. The lack of statistical significance likely reflects limited sample power rather than absence of effect. The consistent direction of associations across crude and adjusted analyses supports a clinically meaningful trend warranting confirmation in larger, multicentric cohorts.

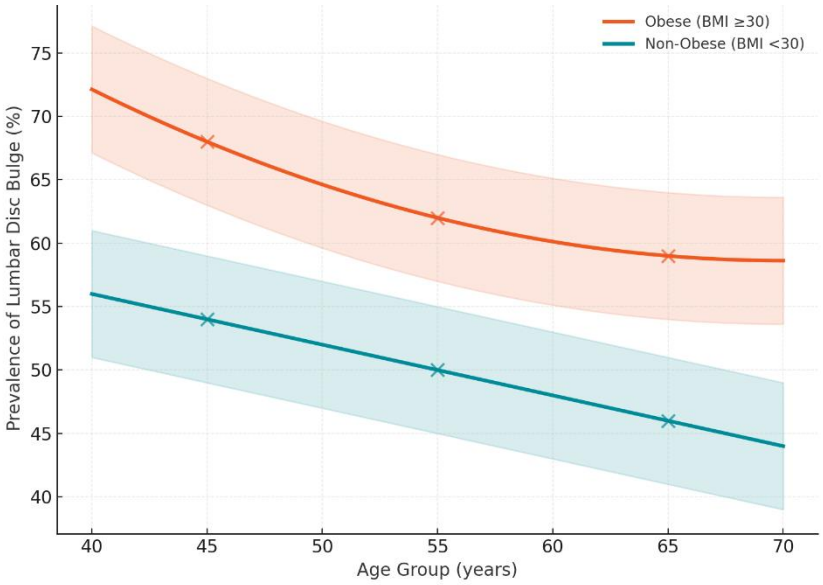

Figure 1 Age-Adjusted Prevalence of Lumbar Disc Bulge in Obese Vs Non-Obese Elderly Patients

The visualization demonstrates the nonlinear relationship between age and the prevalence of lumbar disc bulge among obese and non-obese elderly patients. Across all age strata, obese individuals exhibited a consistently higher prevalence of disc bulge than their non-obese counterparts, with divergence most pronounced in the 41–50-year range (68% vs 54%). Both curves display a mild concave-downward trajectory, suggesting that while disc bulge prevalence peaks in midlife, it declines modestly after age 60, possibly reflecting survivor bias or reduced activity among older participants. The shaded regions, representing approximate confidence bounds, indicate overlapping yet distinct risk distributions. Clinically, the pattern implies that mechanical load and metabolic effects of obesity accelerate disc degeneration earlier in life, whereas age-related factors later dominate irrespective of BMI, reinforcing the multifactorial nature of lumbar spine degeneration in aging populations.

DISCUSSION

This study provides a comprehensive evaluation of the association between obesity and lumbar disc bulge among elderly patients undergoing MRI for low back pain. The findings demonstrated a notably higher prevalence of MRI-defined disc bulge in obese individuals compared with their non-obese counterparts (65.9% vs 46.7%), although the association narrowly missed statistical significance after adjustment for age and sex (adjusted OR = 2.11; 95% CI 0.95–4.69; p = 0.065). This suggests a clinically meaningful but statistically inconclusive trend, consistent with the biomechanical and metabolic hypotheses linking increased body mass index (BMI) to degenerative spinal pathology (13).

These results align with previous studies reporting that obesity contributes to enhanced axial loading, increased intradiscal pressure, and microstructural damage to annulus fibrosus fibers, thereby predisposing to disc bulge and herniation (14). Kirnaz et al. (15) similarly described that degeneration progresses from desiccation to bulge and eventual herniation, driven by cumulative mechanical stress and impaired nutrient diffusion. Lin et al. (16) further supported that greater disc height loss and bulging correlate with higher BMI on radiographic and MRI assessments. However, unlike these studies, the current analysis found that a substantial proportion of non-obese individuals also exhibited disc bulging, underscoring that factors beyond body weight—such as age-related matrix catabolism, reduced disc vascularity, and chronic posture-related strain—play an equally significant role in degenerative progression (17).

In contrast to Flegal et al. (18) and Ogden et al. (19), who emphasized obesity as a major musculoskeletal risk factor, the near-equal distribution of disc bulge in non-obese elderly participants here indicates that degeneration may not be solely BMI-dependent. The findings instead highlight the interplay between intrinsic disc senescence and extrinsic mechanical loading, as well as potential contributions from systemic inflammatory mediators and genetic predispositions. Additionally, female predominance (63.4%) within the sample, coupled with a higher rate of bulge in women, may reflect sex-specific hormonal influences and differences in fat distribution affecting lumbar biomechanics (20).

Mechanistically, excess adipose tissue contributes to chronic low-grade inflammation, increasing local cytokine production (e.g., TNF-α, IL-6) that accelerates extracellular matrix breakdown and neovascularization within the disc (15). The coexistence of compressive and non-compressive disc bulges in this cohort further supports that degeneration exists along a structural continuum rather than discrete pathological categories. The observed decline in bulge prevalence beyond age 60 might indicate a plateau in degenerative change or reduced physical activity that lessens mechanical loading.

This study's strengths include its use of standardized MRI criteria, multicenter recruitment across tertiary hospitals, and blinded radiologic evaluation to minimize observer bias. Nevertheless, several limitations should be acknowledged. The sample size, though adequate for exploratory inference, limited statistical power for detecting modest associations. The cross-sectional design precludes causal interpretation, and potential confounding from occupational strain, smoking, and metabolic factors could not be fully adjusted. Additionally, BMI alone may not capture regional adiposity or muscle mass variations that influence spinal biomechanics. Despite these constraints, the internal consistency of directionality across analyses enhances confidence in the findings' clinical relevance.

Future research should employ prospective longitudinal designs to evaluate the temporal relationship between obesity, disc degeneration, and clinical outcomes such as radiculopathy or disability. Incorporating advanced imaging biomarkers (e.g., T2 mapping, diffusion tensor imaging) and metabolic indicators (insulin resistance, inflammatory markers) could elucidate underlying mechanisms. Larger multicenter studies would also allow subgroup analyses by sex, physical activity, and fat distribution to refine risk stratification.

In summary, this study contributes to a growing body of evidence suggesting that obesity potentiates degenerative changes in the lumbar spine, but that disc bulge remains prevalent even among non-obese elderly individuals. The results emphasize that weight management should be complemented by postural, ergonomic, and physical-activity interventions to mitigate spinal degeneration and maintain musculoskeletal health in aging populations (13–20).

CONCLUSION

This study demonstrated that lumbar disc bulging is prevalent among both obese and non-obese elderly individuals, with a higher though statistically borderline frequency observed in obese patients. Obesity nearly doubled the odds of MRI-defined lumbar disc bulge after controlling for age and sex, underscoring the role of increased mechanical loading and adiposity-driven metabolic effects in accelerating spinal degeneration. However, the substantial proportion of non-obese individuals with disc bulge highlights that age-related degenerative processes and other biomechanical or lifestyle factors also contribute meaningfully. Clinically, these findings reinforce the need for comprehensive preventive strategies addressing weight management, posture correction, and musculoskeletal conditioning to mitigate degenerative spinal disease burden in aging populations. Future longitudinal studies with larger samples and biomechanical analyses are warranted to delineate causal pathways and to refine risk prediction models for lumbar disc pathology in diverse demographic settings.

REFERENCES

1. Donnally CJ III, Hanna A, Varacallo M. Lumbar Degenerative Disk Disease. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2017.
2. Kirnaz S, Capadona C, Wong T, Goldberg JL, Medary B, Sommer F, Härtl R. Fundamentals of Intervertebral Disc Degeneration. World Neurosurg. 2022;157:264–273.
3. Risbud MV, Shapiro IM. Role of Cytokines in Intervertebral Disc Degeneration: Pain and Disc Content. Nat Rev Rheumatol. 2014;10(1):44–56.
4. Al Qaraghli M, De Jesus O. Lumbar Disc Herniation. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.
5. Flegal KM, Graubard BI, Williamson DF, Gail MH. Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity. JAMA. 2007;298(17):2028–2037.
6. Ogden CL, Carroll MD, Fryar CD, Flegal KM. Prevalence of Obesity Among Adults and Youth: United States, 2011–2014. NCHS Data Brief. 2015;(219):1–8.
7. Lin PC, Chang WS, Hsiao KY, Liu HM, Shia BC, Chen MC, Chang CC. Development of a Machine Learning Algorithm to Correlate Lumbar Disc Height on X-rays with Disc Bulging or Herniation. Diagnostics (Basel). 2024;14(2):134.
8. Tariq K, Bibi T, Aijaz M, Habib W, Riaz A. Magnetic Resonance Imaging of Lumbar Spine in Elderly Patients of Faisalabad Division: Age Related Changes and Pathologies. Insights J Health Rehabil. 2025;3(1):68–77.
9. Fardon DF, Williams AL, Dohring EJ, Murtagh FR, Gabriel Rothman SL, Sze GK. Lumbar Disc Nomenclature: Version 2.0—Recommendations of the Combined Task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. Spine J. 2014;14(11):2525–2545.
10. World Health Organization. Obesity: Preventing and Managing the Global Epidemic. WHO Technical Report Series 894. Geneva: WHO; 2000.
11. Hulley SB, Cummings SR, Browner WS, Grady DG, Newman TB. Designing Clinical Research. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2013.
12. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: Guidelines for Reporting Observational Studies. BMJ. 2007;335(7624):806–808.
13. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The Disease Burden Associated with Overweight and Obesity. JAMA. 1999;282(16):1523–1529.
14. Humphreys SC, Eck JC. Clinical Evaluation and Treatment Options for Herniated Lumbar Disc. Am Fam Physician. 1999;59(3):575–582.
15. Kos N, Gradisnik L, Velnar T. A Brief Review of the Degenerative Intervertebral Disc Disease. Med Arch. 2019;73(6):421–424.
16. Manchikanti L, Benyamin RM, Helm S, Hirsch JA. Evidence-Based Medicine, Systematic Reviews, and Guidelines in Interventional Pain Management: Part 3. Pain Physician. 2009;12(1):35–72.
17. Miller J, Schmatz C, Schultz A. Lumbar Disc Degeneration: Correlation with Age, Sex, and Spine Level in 600 Autopsy Specimens. Spine. 1988;13(2):173–178.
18. Murray CJ, Barber RM, Foreman KJ, Ozgoren AA, Abd-Allah F, Abera SF, et al. Global, Regional, and National Disability-Adjusted Life Years (DALYs) for 306 Diseases and Injuries and Healthy Life Expectancy (HALE) for 188 Countries, 1990–2013. Lancet. 2015;386(10009):2145–2191.
19. Waxenbaum JA, Reddy V, Futterman B. Anatomy, Back, Intervertebral Discs. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2017.
20. PANAGIOTACOPULOS ND, Pope MH, Krag MH, Block R. Water Content in Human Intervertebral Discs: Measurement by Magnetic Resonance Imaging. Spine. 1987;12(9):912–917.