Research Square

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Obesity and Prolonged Symptom Duration as Independent Correlates of Calcified Lumbar Disc Herniation: A 1:3 Matched Case-Control Study

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Research Article

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Abstract

Background

Calcified lumbar disc herniation (CLDH) presents significant surgical challenges due to severe nerve adhesion. While the biological link between obesity and spinal degeneration is established, the specific risk profile for CLDH—particularly the interplay between obesity and symptom duration—remains poorly quantified. This study aims to investigate the association of obesity and prolonged symptom duration with CLDH in a Chinese tertiary-care population.

Methods

A retrospective case-control study was conducted with a 1:3 matching ratio. Forty patients with CLDH treated at our hospital were identified as the case group. Simultaneously, 120 patients with simple non-calcified lumbar disc herniation, matched for age and sex, were randomly selected as the control group. Univariate and multivariate binary logistic regression analyses were performed to identify independent correlates of CLDH.

Results

In the 160-patient cohort, obesity was present in 18/40 cases (45.0%) and 22/120 controls (18.3%). Disease duration ≥ 2 years was present in 17/40 cases (42.5%) and 26/120 controls (21.7%). Univariate analysis showed that obesity (*OR = 3.65, 95% CI: 1.68–7.92, P = 0.001*) and disease duration ≥ 2 years (*OR = 2.67, 95% CI: 1.25–5.73, P = 0.011*) were significantly associated with CLDH. After mutual adjustment in the multivariate model, both obesity (*OR = 4.05, 95% CI: 1.80–9.09, P = 0.0007*) and prolonged symptom duration (*OR = 3.04, 95% CI: 1.36–6.82, P = 0.007*) remained independently associated with CLDH.

Conclusions

In this exploratory study, obesity and a disease duration ≥ 2 years were independently associated with CLDH. Clinically, these findings suggest a critical intervention window: obese patients with lumbar disc herniation should be considered for early aggressive management (within 2 years) to prevent calcification and reduce subsequent surgical difficulty.

Background

Lumbar disc herniation is a common clinical condition in which the herniated structure compresses the nerve root, manifesting mainly as low back pain and lower limb numbness, which severely reduces the patient's quality of life[1]. When the herniated lumbar disc structure becomes calcified under the influence of certain factors, it is termed calcified lumbar disc herniation (CLDH). CLDH causes more

pronounced nerve root compression symptoms, not only bringing more severe clinical symptoms and higher treatment costs to patients, but also increasing the difficulty and risk of surgery for surgeons[2][3]. Therefore, investigating the factors associated with its occurrence has important clinical significance.

To date, few studies have investigated the risk factors for CLDH, with existing studies focusing mostly on surgical approaches and treatment outcomes. Some studies have shown an association between obesity and spinal ligament calcification[4], suggesting that obesity may be related to lumbar disc herniation with calcification. Unhealthy lifestyle habits have led to a year-on-year increase in the obese population[5], and the association between obesity and spinal system diseases has received increasing attention. Studies have confirmed that obesity accelerates the degeneration of intervertebral discs[6] by increasing the mechanical load on the lumbar spine and inducing chronic inflammatory responses. However, in the specific pathological state of CLDH, whether obesity is associated with it remains unclear, and current research is still relatively limited, without a definitive conclusion. Therefore, this study aims to analyze the relationship between CLDH, obesity, and symptom duration. We hypothesize that obesity and prolonged symptom duration act synergistically to increase the risk of calcification. By providing subtype-specific evidence, this study hopes to identify a critical clinical intervention window.

Methods

Study design and participants

We retrospectively identified patients diagnosed with lumbar disc herniation at our hospital between January 2024 and June 2026, confirmed by CT, with complete clinical data. Patients with concurrent spinal tumors, infections, fractures, or other diseases; a history of previous lumbar surgery; long-term use of glucocorticoids or other drugs affecting bone metabolism; metabolic bone diseases such as hyperparathyroidism; and poor-quality imaging were excluded. The study was conducted in our hospital; the report follows the STROBE guidelines and adheres to the Declaration of Helsinki. The sample size was determined by the availability of consecutive patients. No a priori power calculation was performed; thus, the study is exploratory, and the precision of effect estimates is reflected in the 95% confidence intervals.

Diagnostic criteria for CLDH

CT imaging showed a high-density shadow within the herniated disc (CT value ≥ 100 HU), with density close to the vertebral cortical bone and clear borders (as shown in Fig. 1).

Grouping

The case group consisted of patients with CLDH diagnosed by lumbar CT. The control group was randomly selected from patients with non-calcified lumbar disc herniation during the same period using

random numbers generated by Stata 17.0, matched for age and sex at a 1:3 ratio.

Data collection

Imaging data of patients diagnosed with lumbar disc herniation and CLDH were collected through the imaging system and re-reviewed by two physicians with many years of imaging diagnostic experience to confirm the diagnosis. Clinical data were collected from the electronic medical records of our hospital. The following information was extracted: demographic data (sex, age, and BMI; according to the Chinese criteria for adult obesity, BMI ≥ 28 kg/m² was defined as obese and < 28 kg/m² as non-obese[7]), and clinical characteristics, including disease duration (time from symptom onset to consultation), occupational type (sedentary vs. non-sedentary), and the presence of hypertension and diabetes mellitus.

Statistical analysis

Data analysis was performed using SPSS 27.0 statistical software (IBM, New York, USA). Continuous data were compared between groups using the independent samples t-test, and categorical data were analyzed using univariate binary logistic regression analysis, adjusting for confounding factors. Variables with a *P* value ≤ 0.05 in the univariate logistic regression analysis were included in the multivariate binary logistic regression model. A *P* value < 0.05 was considered statistically significant.

Results

Baseline characteristics

A total of 160 patients were included, comprising 40 patients in the CLDH case group and 120 patients in the non-calcified control group (1:3 matching ratio). The case group included 16 males (40.0%) and 24 females (60.0%), while the control group included 51 males (42.5%) and 69 females (57.5%). The mean age was 45.5 ± 9.6 years in the case group and 45.2 ± 8.1 years in the control group. There were no statistically significant differences in sex *($\chi^2 = 0.077$, $P = 0.782$)* or age (*$t = 0.189$, $P = 0.850$*) between the two groups, indicating that the baseline characteristics were comparable (Table 1).

Table 1
Baseline characteristics between the calcified and non-calcified groups

Baseline data	Calcified group (n = 40)	Non-calcified group (n = 120)	Statistic	*P* value
Gender			*(χ^2 = 0.077)*	*0.782*
Male	16 (40%)	51 (42.5%)		
Female	24 (60%)	69 (57.5%)		
Age (years, mean ± SD)	45.5 ± 9.6	45.2 ± 8.1	*(t = 0.189)*	*0.850*

Note: Gender data were analyzed using the chi-square test, and age data using an independent samples t-test; data are presented as mean ± standard deviation.

Univariate analysis

In the univariate logistic regression analysis, the prevalence of obesity in the case group was significantly higher than that in the control group (45.0% vs. 18.3%), demonstrating a strong association with CLDH (*OR = 3.650, 95% CI: 1.680–7.920, P = 0.001*). Furthermore, the proportion of patients with a disease duration of ≥ 2 years was significantly higher in the case group compared to the control group (42.5% vs. 21.7%), with an *OR* of 2.67 (*95% CI: 1.250–5.730, P = 0.011*). Hypertension showed a borderline positive association (27.5% vs. 14.2%; *OR = 2.30, 95% CI: 0.970–5.450, P = 0.059*). However, no significant associations were observed for diabetes mellitus (*OR = 1.570, P = 0.437*) or a sedentary lifestyle (*OR = 2.630, P = 0.350*) (Table 2).

Table 2
Univariate logistic regression analysis of clinical characteristics

Clinical characteristics	Calcified group (n = 40)	Non-calcified group (n = 120)	*OR* value	95%CI	*P* value
Obesity	18 (45.0%)	22 (18.3%)	*3.650*	*1.680–7.920*	*0.001*
Disease duration ≥ 2 years	17 (42.5%)	26 (21.7%)	*2.670*	*1.250–5.730*	*0.011*
Hypertension	11 (27.5%)	17 (14.2%)	*2.300*	*0.970–5.450*	*0.059*
Diabetes mellitus	5 (12.5%)	10 (8.3%)	*1.570*	*0.500–4.910*	*0.473*
Sedentary behavior	27 (67.5%)	53 (44.2%)	*2.630*	*0.980–7.060*	*0.650*

Note: All variables were analyzed by univariate binary logistic regression; P < 0.05 was considered statistically significant.

Multivariate analysis

Variables with a *P*-value ≤ 0.05 in the univariate analysis (obesity and disease duration ≥ 2 years) were included in the multivariate binary logistic regression model. The results confirmed that both obesity (*OR = 4.05, 95% CI: 1.80–9.09, P = 0.0007*) and a disease duration ≥ 2 years (*OR = 3.040, 95% CI: 1.36–6.82, P = 0.007*) remained independently and significantly associated with calcified lumbar disc herniation (Table 3).

Table 3
Multivariate logistic regression analysis for CLDH

Variable	**Regression coefficient (B)**	**Standard error (SE)**	***OR* value**	**95% *CI***	***P* value**
Obesity	1.398	0.413	*4.050*	1.800–9.090	*0.0007*
Disease duration ≥ 2 years	1.112	0.412	*3.040*	1.360–6.820	*0.007*
Note: Multivariate binary logistic regression was performed for variables with P < 0.05 in univariate binary logistic regression; P < 0.05 indicated a statistically significant difference.					

Sensitivity analysis

To test the robustness of the primary model, a sensitivity analysis was performed by additionally adjusting for hypertension and sedentary behavior. The associations remained stable, with obesity (*OR = 3.920, 95% CI: 1.690–9.100*) and prolonged symptom duration (*OR = 2.890, 95% CI: 1.250–6.680*) retaining statistical significance.

Discussion

CLDH may cause more severe nerve compression[8]. In addition, repeated stimulation of the surrounding tissues by calcification can induce chronic inflammation, thereby increasing the risk of adhesion between the nerve root and surrounding tissues. The occurrence of CLDH is related to multiple factors; long-term lumbar disc degeneration leads to a reduction in disc components such as collagen, which in turn causes a decrease in water content and calcium salt deposition, triggering calcification[9]. Studies have shown that the nucleus pulposus of the lumbar disc is mainly composed of collagen and proteoglycans[10]; similarly, studies have confirmed that ligaments (a connective tissue) mainly contain collagen and proteoglycans[11]. This indicates that the composition of the lumbar disc nucleus pulposus is similar to that of the spinal ligaments, and previous studies have shown an association between obesity and spinal ligament calcification[4][12]. This study hypothesized that both obesity and prolonged symptom duration play critical roles in CLDH, and aimed to identify a potential clinical intervention window.

In this study, both obesity and a disease duration ≥ 2 years were confirmed as independent correlates of CLDH. Our results are consistent with some previous studies, but also differ in some respects. A study pointed out that elevated BMI is a risk factor for CLDH[13], which may be related to obesity increasing the mechanical stress on the lumbar spine, promoting inflammatory responses, and accelerating lumbar disc degeneration, thereby leading to lumbar disc herniation and calcification[14]. Obesity may accelerate damage to the annulus fibrosus by increasing the mechanical stress on the lumbar spine[15], while promoting the production of inflammatory cytokines such as IL-1β, IL-6, and IL-8 by adipocytes, thereby accelerating disc degeneration[16]. Obesity also leads to atherosclerosis and vascular lumen narrowing, thereby reducing the blood supply to the disc, accelerating disc degeneration, and ultimately promoting calcification formation[17]. Our data demonstrated that patients with a disease duration ≥ 2 years had a significantly higher risk of CLDH. This may be because calcification is a gradual accumulation process; the longer the disc undergoes aging and degeneration, the greater the opportunity for calcium salt deposition[18]. These findings suggest a clear clinical window: for obese patients with lumbar disc herniation, conservative treatment exceeding 2 years significantly increases the risk of calcification and subsequent surgical difficulty. In addition, the proportions of patients with comorbid diabetes mellitus and hypertension were slightly higher, although the differences did not reach statistical significance. However, microvascular lesions caused by diabetes mellitus and hypertension may affect the blood supply to the disc, thereby indirectly promoting the calcification process[19][20]. In this study, the hypertensive and diabetic patients may have had mainly mild or well-controlled disease, so their damaging effects on the disc were not fully reflected. This study showed no significant association between sedentary behavior and CLDH. However, it is known that a sedentary lifestyle leads to disc degeneration[21].The difference in the proportion of sedentary individuals between the two groups was not significant. This may be because the loading effect of a sedentary lifestyle on the lumbar spine interacts strongly with obesity, thereby masking the independent effect of sedentary behavior.

This study has several limitations. First, the single-center retrospective design may introduce selection bias. Second, potential spine-specific confounders, such as smoking and cumulative occupational lumbar load, were not available in the electronic medical records. Third, while the current sample size (160 patients) was adequate for the two-variable multivariate model, the 95% confidence interval for obesity remained relatively wide. Therefore, the results should be interpreted as exploratory, and large-scale multicenter prospective studies are needed for verification.

Conclusions

In this exploratory study, obesity and a disease duration ≥ 2 years were independently associated with calcified lumbar disc herniation. Clinically, particular attention should be paid to obese patients with lumbar disc herniation. Implementing aggressive interventions (such as weight management and early treatment) within 2 years of symptom onset may help reduce the risk of calcification and improve patient outcomes.

Abbreviations

CLDH
calcified lumbar disc herniation
BMI
body mass index
CT
computed tomography
HU
Hounsfield units
OR
odds ratio
CI
confidence interval
IL-1β
interleukin-1β
IL-6
interleukin-6
IL-8
interleukin-8.

Declarations

Clinical trial number: Not applicable.

Ethics approval and consent to participate:

This study was approved by the Ethics Committee of the Affiliated Hospital of Xuzhou Medical University (ethics approval number: XYFY2024-KL634-01, approval date: 2025-01-09). It retrospectively reviewed de-identified medical records of patients from January 2024 to June 2026. All procedures performed in this study were in accordance with the Declaration of Helsinki and the STROBE guidelines. The requirement for written informed consent was waived by the Ethics Committee owing to the retrospective nature of the study.

Consent for publication:

Not applicable.

Competing interests:

The authors declare that they have no competing interests.

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Author Contribution

T.Y.conceived and designed the study, collected and analysed the clinical data, and drafted the manuscript. S.Y.Z. and L.M.Z. contributed to data collection and clinical data acquisition. S.L. performed the statistical analysis and helped interpret the results. W.S. supervised the study and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

Acknowledgements:

Not applicable.

Data Availability

The de-identified datasets analyzed in this study are not publicly available owing to patient confidentiality restrictions. Data may be obtained from the corresponding author on reasonable request after ethics approval.

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Figures

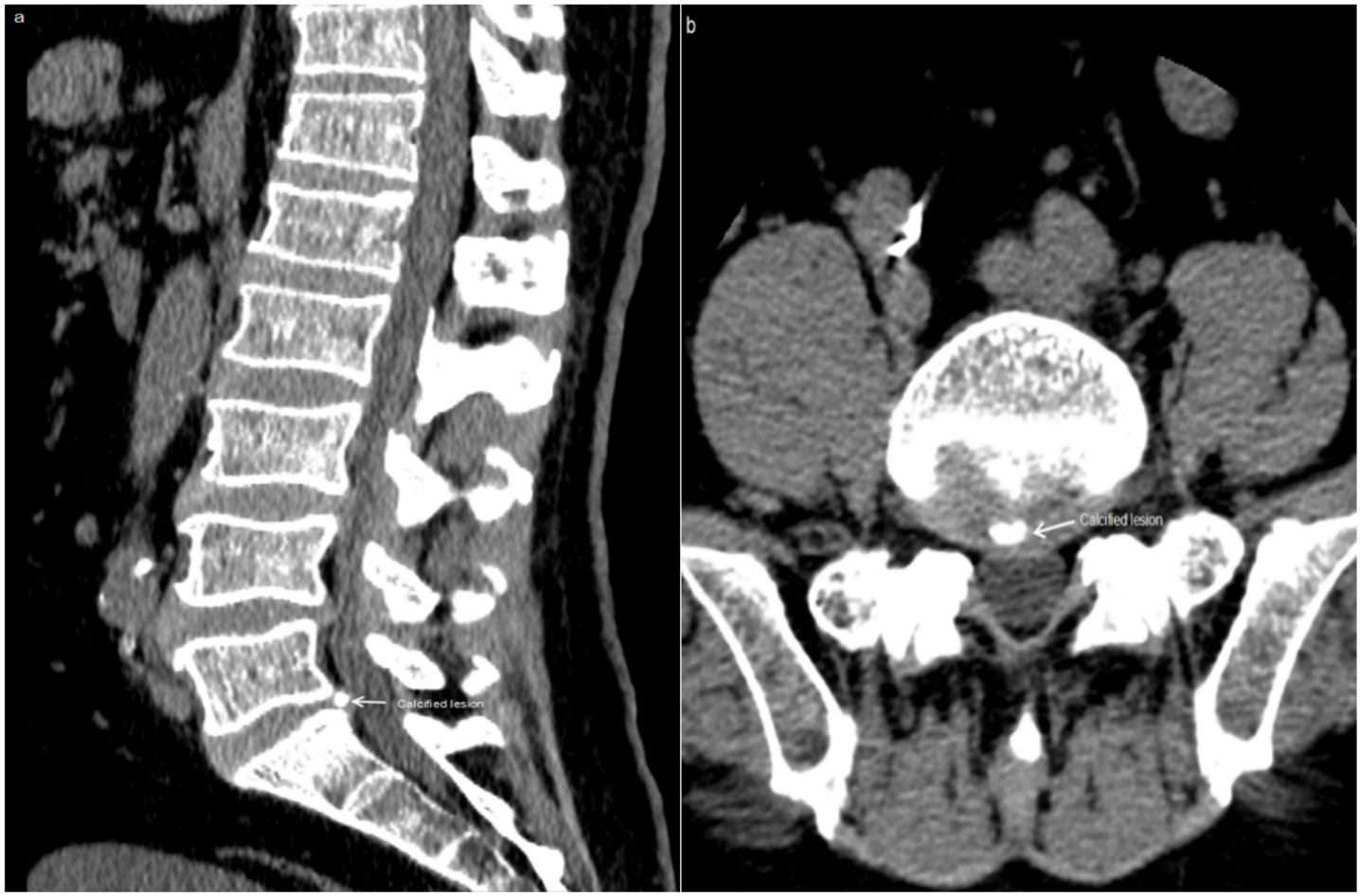


Figure 1

(a) Sagittal CT of L5–S1; (b) axial CT of the same level. Arrows indicate the calcified herniated disc (high-density shadow, CT ≥ 100 HU).