RESEARCH PAPER

Association between Serum 25-Hydroxyvitamin D Levels, Lumbar Disc Herniation Severity, and Neuropathic Pain Intensity in Patients with Chronic Low Back Pain

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ABSTRACT

Background and Objective: Vitamin D may influence pain modulation and intervertebral disc pathology, but its relationship with lumbar disc herniation severity and neuropathic pain remains unclear. This study aimed to determine the association of serum 25-hydroxyvitamin D [25(OH)D] levels with lumbar disc herniation severity and pain intensity in patients with chronic low back pain.

Methods: This observational cross-sectional study included 66 adults with chronic low back pain and MRI-confirmed lumbar disc herniation who were recruited consecutively at Dr. Wahidin Sudirohusodo Hospital and affiliated healthcare facilities in Makassar, Indonesia, from June to July 2026. Neuropathic pain characteristics were assessed using the PainDETECT questionnaire, while overall pain intensity was assessed using the Numeric Pain Rating Scale. Serum 25(OH)D was measured using a competitive two-step enzyme-linked immunosorbent assay. Associations were evaluated using the Mann–Whitney U test and chi-square test, as appropriate.

Results: The median serum 25(OH)D concentration was 34.05 ng/mL, with vitamin D deficiency, insufficiency, and sufficient status observed in 16.7%, 19.7%, and 63.6% of participants, respectively. Serum 25(OH)D did not differ significantly between protrusion and extrusion groups ($p = 0.210$). In contrast, participants with moderate pain had significantly higher serum 25(OH)D concentrations than those with severe pain ($p = 0.025$). Categorical vitamin D status was not significantly associated with pain intensity ($p = 0.120$).

Conclusion: Serum 25(OH)D was not associated with lumbar disc herniation severity but was associated with overall pain intensity. These findings suggest that vitamin D status may be related more closely to pain perception than to structural disc severity.

Keywords: Intervertebral Disc Degeneration; Low Back Pain; Neuropathic Pain; Vitamin D; Vitamin D Deficiency

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INTRODUCTION

Low back pain (LBP) is the leading global cause of years lived with disability, affecting approximately 619 million people in 2020 and projected to reach 843 million by 2050 [1]. Chronic LBP substantially impairs physical function, quality of life, and work participation and may involve nociceptive, neuropathic, or mixed pain mechanisms [2,3]. Lumbar disc herniation (LDH) is an important structural cause of LBP and radicular pain through mechanical nerve-root compression and local inflammatory responses [4]. However, MRI abnormalities do not necessarily correlate with pain severity, suggesting that structural changes alone cannot fully explain individual differences in clinical presentation [5]. Disc morphology can be objectively characterized on MRI as protrusion, extrusion, or sequestration according to standardized nomenclature [6].

Vitamin D may influence musculoskeletal health and pain beyond its established role in calcium and bone metabolism [7]. Through VDR-mediated

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immunomodulatory, neuroprotective, and anti-inflammatory pathways, vitamin D may influence nociceptive processing and intervertebral disc homeostasis [8,9]. Observational evidence has associated low serum 25-hydroxyvitamin D [25(OH)D] with LBP [10], although clinical evidence remains inconsistent, with supplementation trials showing no consistent reduction in chronic LBP intensity [11]. Moreover, vitamin D has been associated with general pain intensity but not consistently with neuropathic pain characteristics, suggesting that its relationship with pain may depend on the underlying clinical phenotype [12].

Despite these findings, the relationship between serum 25(OH)D, LDH severity, and neuropathic pain remains poorly defined. Previous studies have generally examined vitamin D status in relation to pain, disc disease, or treatment outcomes separately, without simultaneously integrating biochemical status, MRI-defined herniation severity, and neuropathic pain assessment [13,14]. This distinction is clinically relevant because structural disc abnormalities and neuropathic pain may represent related but distinct dimensions of LDH. Therefore, this study aimed to determine the association of serum 25(OH)D levels with MRI-defined LDH severity and pain characteristics in patients with chronic LBP, integrating painDETECT for neuropathic pain assessment and NPRS for overall pain intensity.

MATERIALS AND METHODS

Study Design and Setting

This study employed an observational analytic cross-sectional design. The study was conducted at Dr. Wahidin Sudirohusodo Hospital and affiliated healthcare facilities in Makassar, Indonesia, from June to July 2026, following ethical approval. The study population comprised adult patients with chronic low back pain and lumbar disc herniation who were evaluated and managed at the participating facilities.

Participants and Eligibility Criteria

Eligible participants were adults aged ≥18 years with chronic low back pain persisting continuously or recurrently for ≥12 weeks, radiologically confirmed lumbar disc herniation on lumbosacral MRI, and a PainDETECT score of ≥19, indicating a likely neuropathic pain component. Participants were required to be able to complete the study questionnaires with minimal assistance and to provide written informed consent.

Patients were excluded if they had acute infection, malignancy, or spinal fracture; had received vitamin D supplementation within the previous three months; had conditions affecting vitamin D metabolism, including chronic kidney disease, chronic liver disease, cirrhosis, chronic hepatitis, hyperparathyroidism, hypoparathyroidism, hyperthyroidism, or hypothyroidism; or were receiving medications known to interfere with vitamin D metabolism, including long-term systemic corticosteroids for ≥3 months or enzyme-inducing anticonvulsants such as phenytoin, phenobarbital, or carbamazepine. Additional exclusion criteria were neuropathic pain attributable to other causes, including diabetes mellitus; pregnancy or breastfeeding; and epidural injection or spinal surgery within the previous three months.

Sample Size and Sampling

The minimum sample size was calculated for a correlation-based analysis using a two-sided type I error of 5%, statistical power of 90%, and a minimum clinically meaningful correlation coefficient of 0.40. The calculation yielded a minimum requirement of 62 participants. Participants were recruited using consecutive sampling, whereby eligible patients presenting during the study period were enrolled sequentially until the required sample size was achieved.

Study Variables and Outcomes

The primary independent variable was serum 25(OH)D concentration, analyzed as a continuous variable. The primary clinical outcome was overall pain intensity assessed using the NPRS. PainDETECT was used as a screening tool to identify patients with a likely neuropathic pain component. Lumbar disc herniation severity based on MRI was considered the primary structural outcome, whereas NPRS was used as a secondary clinical outcome to characterize overall pain intensity. Potential confounding variables included age, sex, body mass index (BMI), physical activity, pain duration, and sun exposure assessed using the Sun Exposure Index (SEI). Lumbar disc herniation severity was additionally considered as a clinical covariate in regression models evaluating neuropathic pain.

Clinical and Radiological Assessment

Baseline demographic and clinical information was obtained through structured interviews, physical examination, and medical record review. Data included age, sex, occupation, pain onset and duration, pain location and characteristics, relevant chronic diseases, medication and vitamin D supplementation history, physical activity, and sun exposure.

Neurological and musculoskeletal examinations were performed to characterize the clinical presentation. Lumbosacral MRI findings were reviewed based on the available radiological report. Disc morphology was classified according to standardized nomenclature as bulging, protrusion, extrusion, or sequestration. Because the eligibility criteria required radiologically confirmed herniation, the primary analysis of herniation severity focused on protrusion and extrusion.

Assessment of Neuropathic Pain Screening and Pain Intensity

The PainDETECT questionnaire was used as a screening tool to identify patients with chronic low back pain with a likely neuropathic pain component. Scores range from 0 to 38, with scores ≤12 indicating a low likelihood of a neuropathic component, 13–18 indicating an uncertain or possible neuropathic component, and ≥19 indicating a high

likelihood of a neuropathic component. Only participants with scores ≥19 were included in the study.

Pain intensity was assessed using the Numeric Pain Rating Scale (NPRS), scored from 0 to 10, with 0 representing no pain and 10 representing the most severe pain imaginable. For categorical analyses, scores of 1–3, 4–6, and 7–10 were classified as mild, moderate, and severe pain, respectively. For the primary categorical analysis, participants were classified into moderate and severe pain groups according to the observed distribution.

Assessment of Vitamin D Status

Venous blood samples of approximately 3–5 mL were collected from the antecubital vein after completion of the clinical assessment and questionnaires. Blood samples were processed to obtain serum and analyzed at the Hasanuddin University Medical Research Center (HUM-RC), Faculty of Medicine, Hasanuddin University.

Serum 25(OH)D concentrations were quantitatively determined using a competitive two-step ELISA. During the first incubation, 25(OH)D in calibrators, controls, and serum samples was released from vitamin D-binding protein and allowed to bind to immobilized anti-25(OH)D antibodies in microplate wells. After washing, a 25(OH)D-biotin–streptavidin-horseradish peroxidase conjugate was added during the second incubation, competitively binding to the available antibody sites. Following a subsequent washing step, tetramethylbenzidine substrate was added. The enzymatic reaction produced a color signal inversely proportional to the concentration of 25(OH)D in the sample. The reaction was terminated using stop solution, and absorbance was measured at 450 nm using a microplate reader. A calibration curve generated from the provided calibrators was used to determine serum 25(OH)D concentrations. For descriptive and categorical analyses, serum 25(OH)D was classified as deficient (<20 ng/mL), insufficient (20–29 ng/mL), or sufficient (≥30 ng/mL).

Assessment of Covariates

BMI was calculated as body weight in kilograms divided by height in meters squared and categorized as underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), or obesity (≥30.0 kg/m²). Physical activity was assessed using the International Physical Activity Questionnaire and categorized as low, moderate, or high. Sun exposure was assessed using the SEI and categorized according to the predefined study classification. Information regarding chronic kidney disease, chronic liver disease, and medications potentially affecting vitamin D metabolism was obtained from medical records and participant interviews.

Data Collection and Management

All demographic, clinical, radiological, questionnaire, and laboratory data were recorded in a standardized research form. Data were checked for completeness, consistency, and accuracy before statistical analysis. Participant confidentiality was maintained throughout data collection and analysis.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics. Continuous variables were assessed for distribution using the Shapiro–Wilk test. Normally distributed variables were summarized as mean ± standard deviation, whereas non-normally distributed variables were presented as median with minimum–maximum values. Categorical variables were summarized as frequencies and percentages.

Differences in serum 25(OH)D concentrations according to lumbar disc herniation severity were assessed using the Mann–Whitney U test because the primary structural analysis comprised two groups, protrusion and extrusion. The same test was used to compare serum 25(OH)D concentrations between moderate and severe pain groups. Categorical associations between vitamin D status and pain intensity were assessed using the chi-square test. Statistical significance was defined as a two-sided p-value <0.05.

Ethical Considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University (No. 862/UN4.6.4.5.31/PP36/2026). All participants received information regarding the study objectives, procedures, potential benefits and risks, and their rights as research participants before enrollment. Written informed consent was obtained from each participant before data collection and blood sampling.

RESULTS

Participant Characteristics

A total of 66 patients with chronic low back pain associated with lumbar disc herniation were included in the analysis. The median age was 52 years, with participants ranging from 19 to 71 years, and women comprised the majority of the study population. The mean BMI was within the overweight range. Most participants reported moderate physical activity and moderate sun exposure according to the SEI. The detailed demographic and clinical characteristics are presented in Table 1.

All participants had a high likelihood of a neuropathic pain component according to the PainDETECT eligibility criterion, with a median score of 22 (range, 19–27). Overall pain intensity was predominantly moderate to severe, with a median NPRS score of 6 (range, 4–8). MRI evaluation identified protrusion as the predominant herniation morphology, whereas extrusion accounted for the remainder of the structural findings.

The median serum 25(OH)D concentration was 34.05 ng/mL (range, 9.57–171.49 ng/mL). Most participants had sufficient vitamin D status, while approximately one-third had either insufficiency or deficiency. The complete

distribution of vitamin D status and other baseline variables is provided in Table 1.

Association Between Serum 25(OH)D and Lumbar Disc Herniation Severity

Serum 25(OH)D concentrations did not differ significantly according to lumbar disc herniation morphology. Participants with protrusion had a median concentration of 31.61 ng/mL, compared with 36.62 ng/mL among those with extrusion. The Mann–Whitney U test showed no statistically significant difference between the two groups ($p = 0.210$), indicating that serum 25(OH)D was not significantly associated with the radiological severity of disc herniation in this population (Table 2).

The distribution of serum 25(OH)D concentrations across the two herniation categories is illustrated in Figure 1. Although the extrusion group showed a numerically higher median concentration, the distributions overlapped substantially, consistent with the absence of a statistically significant group difference.

Association Between Serum 25(OH)D and Pain Intensity

Serum 25(OH)D concentrations differed significantly according to pain intensity category. Participants with moderate pain had higher serum 25(OH)D concentrations than those with severe pain, with the between-group difference reaching statistical significance ($p = 0.025$; Table 2). This finding indicates an inverse pattern between vitamin D status and overall pain intensity, with lower serum 25(OH)D concentrations observed among participants experiencing more severe pain. The distribution of serum 25(OH)D concentrations across pain intensity categories is shown in Figure 2.

Association Between Vitamin D Status and Pain Intensity

The distribution of pain intensity according to vitamin D status is presented in Table 3. Severe pain was proportionally more frequent among participants with vitamin D deficiency, whereas moderate pain predominated among those with insufficient or sufficient vitamin D status. However, the categorical analysis did not demonstrate a statistically significant association between vitamin D status and pain intensity (chi-square test, $p = 0.120$). Thus, although the continuous analysis demonstrated significantly lower serum 25(OH)D concentrations among participants with severe pain, categorization of vitamin D status into deficiency, insufficiency, and sufficiency did not yield a significant association with pain intensity.

DISCUSSION

This study found that serum 25(OH)D was not significantly associated with lumbar disc herniation severity, whereas higher serum 25(OH)D was associated with lower overall pain intensity. Importantly, serum 25(OH)D was not significantly associated with neuropathic pain characteristics assessed using PainDETECT. These findings suggest that vitamin D may be more closely related to the intensity of pain perception than to the radiological severity or neuropathic phenotype of lumbar disc herniation. The relatively favorable vitamin D status observed in this population compared with previous reports may reflect differences in demographic characteristics, body composition, sun exposure, nutritional status, and laboratory methods. Qazi et al. (2022) reported a substantially higher prevalence of inadequate vitamin D status among patients with lumbar disc herniation, particularly among those with higher BMI [15], whereas Ali et al. (2026) reported an even greater burden of vitamin D deficiency among patients with chronic low back pain [16]. These differences emphasize that vitamin D status in patients with spinal disorders is strongly influenced by population-specific characteristics rather than by spinal pathology alone.

The absence of an association between serum 25(OH)D and herniation severity does not necessarily contradict the proposed biological role of vitamin D in intervertebral disc pathology. Colombini et al. (2021) demonstrated that the cellular response of intervertebral disc tissue to vitamin D may vary according to VDR genetic background, suggesting that circulating vitamin D concentration alone may not adequately reflect its local biological activity [17]. More recently, Vajarintarangoon et al. (2025) reported altered VDR expression and promoter methylation in patients with lumbar disc degeneration, with greater VDR dysregulation associated with more advanced degeneration [18]. Together, these findings suggest that the vitamin D–VDR pathway may influence disc biology through tissue-specific and molecular mechanisms that cannot be captured solely by measuring serum 25(OH)D. Furthermore, lumbar disc herniation is a multifactorial process involving mechanical loading, degeneration, inflammation, genetic susceptibility, muscle condition, and metabolic factors. Faur et al. (2019) demonstrated that structural disc degeneration has only a modest relationship with paraspinal muscle characteristics, supporting the concept that no single biological marker is likely to explain the full spectrum of structural spinal changes [19].

The lack of a direct relationship between herniation morphology and pain is also consistent with previous evidence showing that MRI findings do not always correspond to symptom severity. Divi et al. (2021) emphasized that many MRI abnormalities may be present independently of clinical symptoms [20], while Motiei-Langroudi et al. (2023) showed that the clinical relevance of disc herniation depends not only on its morphological type but also on fragment size, location, degree of degeneration, and relationship with the affected nerve root [21]. This distinction is particularly relevant to the present findings because protrusion and extrusion describe the morphology of displaced disc material but do not directly quantify nerve-root irritation or inflammatory activity. Radicular pain may arise through a combination of mechanical compression and inflammatory mechanisms, including edema, ischemia, demyelination, and inflammatory sensitization. Consequently, patients with apparently comparable disc morphology may experience

substantially different pain phenotypes depending on the anatomical relationship with the nerve root and the degree of peripheral and central sensitization.

In contrast, the inverse association between serum 25(OH)D and overall pain intensity is consistent with evidence suggesting a potential role for vitamin D in pain modulation. Karamanlıoğlu et al. (2024) similarly reported lower general pain intensity among patients with higher vitamin D status, although vitamin D was not significantly associated with neuropathic pain assessed using DN4 [12]. This pattern closely resembles the present findings and may indicate that vitamin D is more relevant to the general pain experience than to the specific neuropathic component. Nevertheless, the literature remains heterogeneous. Ali et al. (2026) and Qazi et al. (2022) did not demonstrate a significant correlation between vitamin D concentrations and pain intensity in their respective populations. Interventional studies have also produced inconsistent results [15,16]; Ghai et al. (2017) reported improvement in pain following vitamin D supplementation [22], whereas Krasowska et al. (2019) observed changes in inflammatory markers in patients undergoing spinal surgery and rehabilitation, making the independent contribution of vitamin D difficult to establish [23]. Similarly, the systematic review by Abdelbaky et al. (2024) concluded that the overall effect of vitamin D supplementation on low back pain was inconsistent and generally limited [24]. Therefore, the present finding should be interpreted as an association between vitamin D status and pain intensity rather than evidence of a direct analgesic effect.

Clinically, these findings indicate that serum 25(OH)D should not be considered a surrogate marker for either lumbar disc herniation severity or neuropathic pain severity. MRI morphology remains important for defining structural pathology, but its interpretation should be integrated with neurological examination, radicular distribution, pain intensity, and validated neuropathic pain assessment. The observed association between vitamin D and overall pain intensity nevertheless supports further investigation of vitamin D as a potential biological modifier of pain rather than as a determinant of disc morphology. The discrepancy between circulating vitamin D status and structural disease severity also raises the possibility that local VDR signaling, inflammatory pathways, and individual susceptibility may be more important than serum concentration alone. Future research integrating serum 25(OH)D with inflammatory biomarkers, VDR-related molecular markers, detailed MRI characteristics, and longitudinal clinical outcomes may therefore provide a more complete understanding of the biological mechanisms linking vitamin D status with chronic low back pain and lumbar disc herniation.

LIMITATIONS

This study has several limitations that should be considered when interpreting the findings. First, the cross-sectional design does not establish temporal sequence or causality between serum 25(OH)D, pain intensity, and herniation severity. The inverse association between vitamin D status and NPRS therefore cannot be interpreted as evidence that lower vitamin D causes greater pain, as the direction of the association may also be reversed. Second, the radiological spectrum was relatively narrow because participants had only protrusion or extrusion at L4–L5 and/or L5–S1, limiting assessment across the full spectrum of herniation morphology and potentially restricting the detection of dose–response relationships.

Third, although the sample size was adequate for the primary analyses, the relatively modest number of participants limits statistical power for detecting small effects and for multivariable or subgroup analyses. Larger studies would provide more precise estimates and allow evaluation across herniation grades, spinal levels, sex, and vitamin D status. Finally, serum 25(OH)D and sun exposure were assessed at a single time point. Although SEI was incorporated into the analysis, longitudinal and seasonal variations in vitamin D status and sun exposure could not be fully captured. Residual confounding from dietary vitamin D intake, clothing practices, sunscreen use, seasonal variation, and other lifestyle factors may therefore remain.

CONCLUSIONS

In patients with chronic low back pain and lumbar disc herniation, serum 25(OH)D was not significantly associated with radiological herniation severity, indicating that circulating vitamin D status alone does not reflect the extent of structural disc displacement. In contrast, higher serum 25(OH)D was associated with lower overall pain intensity, suggesting a potential relationship between vitamin D status and the clinical experience of pain. However, vitamin D status was not associated with neuropathic pain characteristics as assessed by PainDETECT. Overall, these findings indicate that the relationship between vitamin D, lumbar disc pathology, and pain is multifactorial, with serum 25(OH)D potentially reflecting the intensity of pain rather than the structural severity or neuropathic phenotype of disc herniation.

DECLARATIONS

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Conflict of Interest

The authors declare that they have no competing interests.

Author Contributions

UER: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing – original draft, Writing – review and editing.

NH: Conceptualization, Methodology, Supervision, Validation, Writing – review and editing, Project administration.

ADW: Methodology, Investigation, Validation, Writing – review and editing.

FH: Methodology, Validation, Writing – review and editing.

MYA: Investigation, Data curation, Writing – review and editing.

AM: Supervision, Validation, Writing – review and editing.

All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request, subject to applicable ethical and institutional restrictions.

Consent for Publication

Not applicable. No individually identifiable information or images are presented in this manuscript.

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TABLES AND FIGURES

Table 1. Baseline Demographic and Clinical Characteristics of the Participants (n = 66)

Variable	Value
Age, years	52.00 (19–71)
Sex, n (%)	
Male	25 (37.9)
Female	41 (62.1)
BMI, kg/m²	25.21 ± 5.18
Physical activity, n (%)	
Low	11 (16.7)
Moderate	53 (80.3)
High	2 (3.0)
Sun exposure (SEI), n (%)	
Low	13 (19.7)
Moderate	50 (75.8)
High	3 (4.5)
PainDETECT score	22 (19–27)
NPRS score	6 (4–8)
NPRS category, n (%)	
Moderate	42 (63.6)
Severe	24 (36.4)
Herniation morphology, n (%)	
Protrusion	48 (72.7)
Extrusion	18 (27.3)
Serum 25(OH)D, ng/mL	34.05 (9.57–171.49)
Vitamin D status, n (%)	
Deficiency	11 (16.7)
Insufficiency	13 (19.7)

Sufficient	42 (63.6)

Table 2. Serum 25(OH)D Levels According to Lumbar Disc Herniation Severity and Pain Intensity

Variable	Category	Serum 25(OH)D, median (minimum–maximum), ng/mL	p-value
Lumbar disc herniation severity	Protrusion	31.61 (9.57–171.48)	0.210
	Extrusion	36.62 (21.68–116.70)	
Pain intensity	Moderate	41.91 (10.0–171.4)	0.025
	Severe	30.24 (9.5–77.4)	

Mann–Whitney U test.

Table 3. Distribution of Vitamin D Status According to Pain Intensity (NPRS)

Vitamin D status		Moderate, n (%)	Severe, n (%)	Total, n (%)	p-value
Deficiency	n	4	7	11	0.120
	%	36.4	63.6	100.0	
Insufficiency	n	9	4	13	
	%	69.2	30.8	100.0	
Sufficiency	n	29	13	42	
	%	69.0	31.0	100.0	
Total	n	42	24	66	
	%	63.6	36.4	100.0	

Chi-square test.

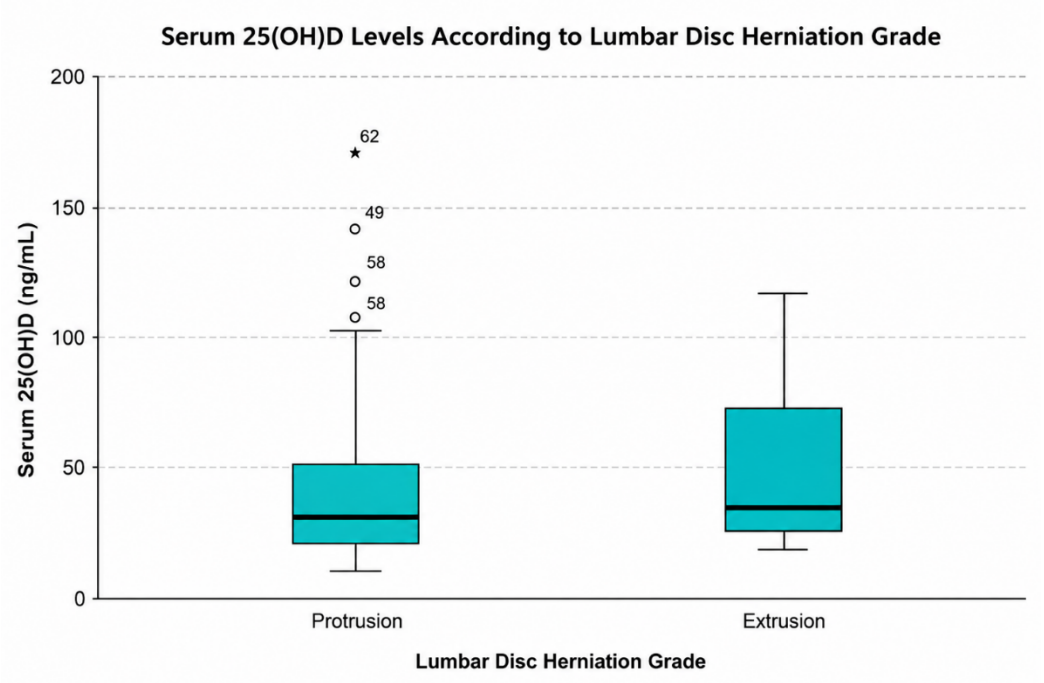

Figure 1. Boxplot of Vitamin D Levels According to Lumbar Disc Herniation Grade

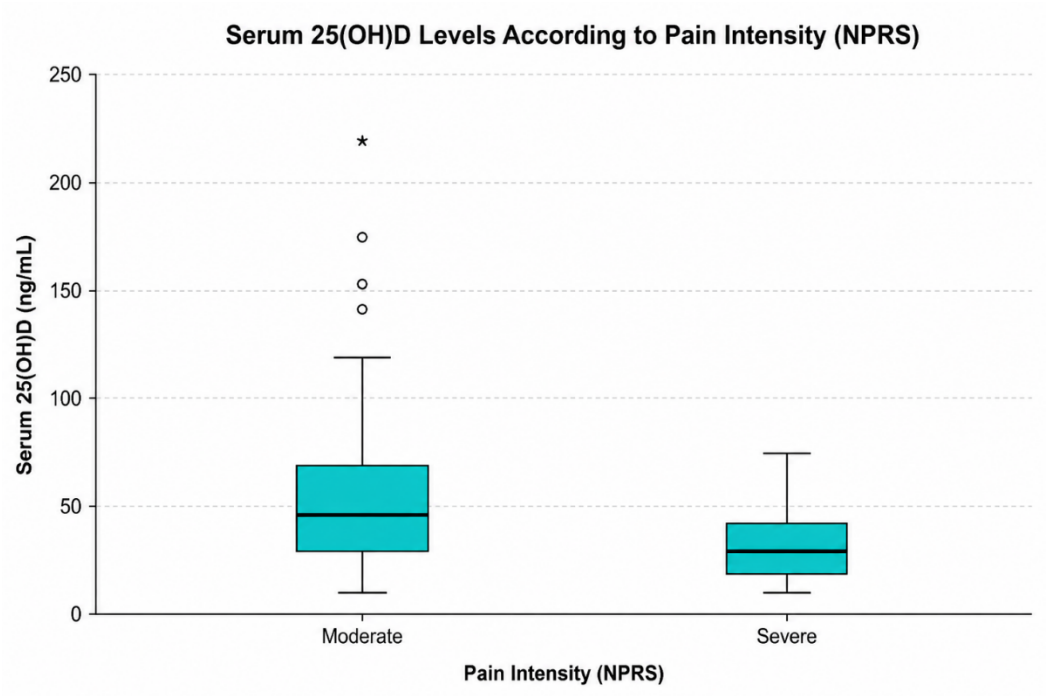

Figure 2. Boxplot of Vitamin D Levels According to NPRS Category