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VITAMIN D DEFICIENCY AND CLINICAL OUTCOMES IN CHRONIC LOW BACK PAIN: INSIGHTS FROM A MULTICENTRE REAL-WORLD STUDY

Orthopaedics

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ABSTRACT

Background Chronic low back pain (CLBP) is a leading cause of disability worldwide and has been increasingly associated with vitamin D deficiency. Vitamin D exerts immunomodulatory, anti-inflammatory, and neuromuscular effects that may influence nociceptive processing and musculoskeletal function. This large multicenter real-world study evaluated the prevalence of vitamin D deficiency in patients with CLBP and assessed the impact of vitamin D3 supplementation on biochemical, pain, and functional outcomes.**Methods** This retrospective multicenter study included adult patients (≥18 years) diagnosed with chronic low back pain (≥3 months duration) who had documented baseline and follow-up serum 25-hydroxyvitamin D levels and received vitamin D3 supplementation as part of routine clinical care. Data were collected through retrospective review of medical records and structured physician-reported questionnaires across participating centers in India. Pain severity was assessed using the Visual Analogue Scale (VAS; 0–10), and functional impairment was measured using the Functional Impairment Scale (FIS; 0–3). Pre–post comparisons were analyzed using paired t-tests. Effect sizes (Cohen's d) and 95% confidence intervals were calculated. **Results** A total of 13,824 patients with chronic low back pain were included in the analysis. Baseline mean serum vitamin D level was 11.41 ± 18.14 ng/mL, indicating a high prevalence of vitamin D deficiency. Following vitamin D3 supplementation, mean serum vitamin D levels increased to 28.45 ± 37.19 ng/mL (mean change +17.04 ± 21.03 ng/mL; 95% CI 16.69–17.39; $p < 0.001$). Mean VAS scores decreased from 5.50 ± 2.19 to 3.74 ± 2.20 (mean change −1.76 ± 2.40; 95% CI 1.72–1.80; $p < 0.001$), while mean FIS scores improved from 2.26 ± 0.75 to 1.63 ± 0.88 (mean change −0.63 ± 1.02; 95% CI 0.61–0.65; $p < 0.001$). Vitamin D3 supplementation was generally well tolerated, with no serious adverse events reported. **Conclusion** Vitamin D deficiency is highly prevalent among patients with CLBP. Vitamin D3 supplementation significantly improves serum vitamin D levels, reduces pain intensity, and enhances functional outcomes in real-world clinical practice.

KEYWORDS

Vitamin D3 Deficiency, Chronic Low Back Pain, Vitamin D3 Supplementation, 60,000 Iu, Neuropathic Pain, Nociceptive Pain

INTRODUCTION

Chronic low back pain (CLBP) is among one of the most prevalent musculoskeletal disorders worldwide and remains a leading cause of years lived with disability across all age groups. According to the Global Burden of Disease Study, low back pain consistently ranks as one of the top contributors to disability-adjusted life years globally, imposing substantial socioeconomic and healthcare burdens[1]. Despite a wide range of therapeutic options, including nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, physical therapy, interventional procedures, and surgery, clinical outcomes remain suboptimal in many patients, reflecting the multifactorial pathophysiology of CLBP.

Vitamin D has traditionally been recognized for its role in calcium homeostasis and skeletal integrity[2]. However, accumulating evidence highlights broader biological functions. Vitamin D receptors (VDRs) are expressed in skeletal muscle, immune cells, and neural tissues, supporting pleiotropic effects beyond bone metabolism. Vitamin D influences muscle protein synthesis, neuromuscular coordination, and postural stability, all of which are relevant to spinal biomechanics. In addition, vitamin D exerts immunomodulatory effects by regulating pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), and by modulating T-cell responses[3]. These anti-inflammatory properties influence chronic nociceptive signaling and peripheral sensitization through modulation of pro-inflammatory cytokines and neural pathways.

Emerging evidence also supports a role for vitamin D in pain modulation. Experimental and clinical data suggest that deficiency may contribute to enhanced nociceptor excitability, increased inflammatory mediator release, and altered neurotransmitter regulation[4]. Clinically, vitamin D deficiency has been associated with diffuse musculoskeletal pain, proximal muscle weakness, impaired physical performance, and increased fall risk[2]. Observational studies have reported a higher prevalence of vitamin D deficiency among individuals with chronic pain syndromes, including chronic low back pain. For example, Nimisha V. et. al. demonstrated lower serum vitamin D levels in patients with low back pain compared to controls[5]. Systematic reviews evaluating vitamin D supplementation in chronic painful conditions have reported heterogeneous but potentially beneficial effects[6].

Despite growing evidence linking vitamin D deficiency with musculoskeletal pain, large-scale real-world studies assessing the clinical effectiveness of vitamin D supplementation in patients with CLBP remain limited. Many published studies are small, geographically restricted, or lack comprehensive functional outcome measures. Furthermore, evidence derived from routine clinical practice, where supplementation is commonly integrated with standard analgesic therapy remains sparse.

Given the high global prevalence of vitamin D deficiency and its potential mechanistic relevance to pain and functional impairment, further investigation in large real-world cohorts is warranted. The present multicenter retrospective study aimed to determine the prevalence of vitamin D deficiency among patients with chronic low back pain and to evaluate the effectiveness of vitamin D3 supplementation on serum vitamin D levels, pain severity, and functional impairment in routine clinical practice.

MATERIALS AND METHODS

Study Design

This retrospective, multicenter, questionnaire-based study was conducted to evaluate the prevalence of vitamin D deficiency and the effectiveness of vitamin D3 supplementation in patients with chronic low back pain (CLBP). The study involved a systematic retrospective review of electronic medical records and structured physician-reported data collected across participating centers in India during a defined study period. The design reflects real-world clinical practice, where vitamin D supplementation was prescribed as part of routine management. The study was conducted in accordance with the principles of the International Council for Harmonisation – Good Clinical Practice (ICH-GCP). Approval was obtained from an Independent Ethics Committee (IEC) at Lifepoint Multispeciality Hospital, Pune (Protocol no: IND/CLBP/01) prior to study initiation dated [25/Apr/2024]. Data were collected for the period between May 2024 and November 2025.

Study Population

The study population consisted of adult patients aged 18 years and older diagnosed with chronic low back pain, defined as persistent or recurrent pain localized to the lower back and lasting for at least three months. Patients were eligible for inclusion if they had documented baseline serum 25-hydroxyvitamin D levels obtained as part of routine clinical evaluation and had received vitamin D3 supplementation during the course of treatment.

Patients were excluded if they had acute or subacute low back pain of less than three months' duration, incomplete medical records, contraindications to vitamin D supplementation, a history of spinal trauma or surgery, significant structural spinal deformities, metabolic bone disorders, or other medical conditions known to substantially alter vitamin D metabolism. These criteria were applied to ensure a relatively homogeneous population of patients with chronic non-structural low back pain.

Data Collection

Data were obtained through retrospective review of medical records and structured physician-reported questionnaires. Extracted variables included demographic characteristics, relevant medical history, baseline serum 25-hydroxyvitamin D levels, pain severity, functional impairment, details of vitamin D supplementation regimen, and follow-up clinical outcomes.

Pain severity was assessed using the Visual Analogue Scale (VAS), a validated 10-point scale ranging from 0 (no pain) to 10 (maximum pain intensity). Functional impairment was evaluated using the Functional Impairment Scale (FIS), scored from 0 (normal level of functioning; no disability) to 3 (severe impairment during performance of daily activities). Information regarding vitamin D3 supplementation, including dosage, duration, and adherence, was recorded. Concomitant medications prescribed for pain management or associated comorbid conditions were also documented to reflect real-world therapeutic practice.

Objectives and Outcome Measures

The primary objective of the study was to determine the prevalence of vitamin D deficiency among patients with chronic low back pain and to evaluate the effectiveness of vitamin D3 supplementation on biochemical and clinical outcomes in routine clinical practice.

The primary outcome measures included the prevalence of vitamin D deficiency, defined as serum 25-hydroxyvitamin D levels below 20 ng/mL, the change in serum vitamin D levels following supplementation, the change in pain severity as measured by VAS, and the change in functional impairment as measured by FIS. Secondary analyses explored associations between baseline clinical characteristics and response to supplementation, including potential relationships between demographic factors and magnitude of improvement.

Statistical Analysis

Continuous variables were summarized as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Pre–post comparisons of serum vitamin D levels, VAS scores, and FIS scores were performed using paired t-tests to evaluate within-patient changes over time. Effect sizes were calculated using Cohen's d for paired samples to quantify the magnitude of change, and 95% confidence intervals were computed for mean differences to assess the precision of estimates.

Analyses for each outcome were conducted using all available paired baseline and follow-up data for that specific parameter (available-case analysis), reflecting real-world data capture where completeness may vary across endpoints.

RESULTS

Study Population and Baseline Characteristics

A total of 13,824 patients with chronic low back pain (CLBP) were included in the analysis. The mean age of the study population was 50.35 ± 10.98 years (range: 18–99 years). Most patients were male (8,657; 62.6%), while 5,165 (37.4%) were female and 2 patients (0.01%) were categorized as other gender.

The majority of patients belonged to the 40–59 years age group (9,167; 66.3%), followed by ≥60 years (2,418; 17.5%) and 18–39 years (2,239; 16.2%). Neuropathic pain was the predominant pain phenotype, reported in 12,129 patients (87.7%), whereas 1,682 patients (12.2%) had nociceptive pain.

Vitamin D deficiency was highly prevalent, affecting 13,704 patients (99.1%) at baseline. The mean baseline serum vitamin D level was 11.41 ± 18.14 ng/mL. Most patients received vitamin D3 supplementation in the form of 60,000 IU weekly (13,424; 97.1%), while smaller proportions received 2,000 IU daily (1.8%), 6,000 IU daily (0.8%), or 4,000 IU daily (0.2%).

At baseline, the mean Visual Analogue Scale (VAS) score was 5.50 ± 2.19, indicating moderate pain severity. The mean Functional Impairment Scale (FIS) score was 2.26 ± 0.75, reflecting moderate functional limitation.

Table 1. Baseline Demographic and Clinical Characteristics

Baseline Characteristic	Value
Total patients	13,824
Age (years), mean ± SD	50.35 ± 10.98
Male, n (%)	8,657 (62.6)
Female, n (%)	5,165 (37.4)
Other, n (%)	2 (0.01)
Age Ranges	
Age 18–39 years, n (%)	2,239 (16.2)
Age 40–59 years, n (%)	9,167 (66.3)
Age ≥60 years, n (%)	2,418 (17.5)
Pain Characteristics, n (%)	
Neuropathic pain, n (%)	12,129 (87.7)
Nociceptive pain, n (%)	1,682 (12.2)
Vitamin D deficiency, n (%)	13,704 (99.1)
Baseline serum vitamin D (ng/mL), mean ± SD	11.41 ± 18.14
Baseline VAS score, mean ± SD	5.50 ± 2.19
Baseline FIS score, mean ± SD	2.26 ± 0.75
Vitamin D Dose and Frequency	
60,000 IU weekly, n (%)	13,424 (97.1)
2,000 IU daily, n (%)	244 (1.8)
6,000 IU daily, n (%)	113 (0.8)
4,000 IU daily, n (%)	25 (0.2)

Effectiveness Outcomes

Following vitamin D3 supplementation, significant improvements were observed in serum vitamin D levels, pain severity, and functional impairment.

Mean serum vitamin D levels increased from 11.41 ± 18.14 ng/mL at baseline to 28.45 ± 37.19 ng/mL at follow-up. The mean increase was 17.04 ± 21.03 ng/mL. Paired t-test analysis demonstrated a statistically significant improvement in vitamin D levels ($t = -95.29$, $df = 13,823$, $p < 0.001$), with a mean paired difference of -17.04 ng/mL (95% CI: -17.39 to -16.69).

Pain severity improved substantially following supplementation. Mean VAS scores decreased from 5.50 ± 2.19 at baseline to 3.74 ± 2.20 at follow-up, corresponding to a mean reduction of 1.76 ± 2.40 points. This reduction was statistically significant ($t = 86.44$, $df = 13,823$, $p < 0.001$), with a mean paired difference of 1.76 points (95% CI: 1.72 to 1.80). Similarly, functional impairment improved following supplementation. Mean FIS scores decreased from 2.26 ± 0.75 at baseline to 1.63 ± 0.88 at follow-up, corresponding to a mean reduction of 0.63 ± 1.02 points. This improvement was statistically significant ($t = 72.25$, $df = 13,823$, $p < 0.001$), with a mean paired difference of 0.63 points (95% CI: 0.61 to 0.65).

Table 2 Clinical Outcomes Following Vitamin D3 Supplementation (N = 13,824)

Outcome	Baseline Mean ± SD	Follow-up Mean ± SD	Mean Change ± SD	95% CI of Mean Difference	t-value (df)	p-value
Serum Vitamin D (ng/mL)	11.41 ± 18.14	28.45 ± 37.19	+17.04 ± 21.03	16.69 to 17.39	95.29	<0.001
VAS Score	5.50 ± 2.19	3.74 ± 2.20	−1.76 ± 2.40	1.72 to 1.80	86.44	<0.001
FIS Score	2.26 ± 0.75	1.63 ± 0.88	−0.63 ± 1.02	0.61 to 0.65	72.25	<0.001

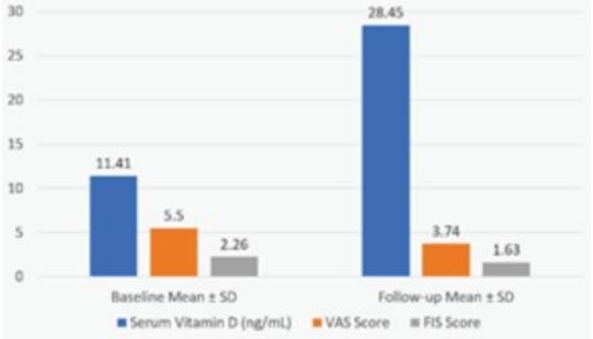

Figure 1: Mean Change in Serum Vitamin D levels, VAS Score and FIS Score from Baseline to Follow-up.

At follow-up, the most frequently reported VAS scores were 2 (20.4%), 3 (20.4%), and 1 (14.3%), indicating a substantial shift toward lower pain intensity categories. Overall, more than half of patients reported VAS scores ≤3 at follow-up.

Functional status also improved considerably. The proportion of patients reporting minimal or no functional impairment (FIS scores 0–1) increased from 14.2% at baseline to 49.2% at follow-up. Conversely, the proportion of patients with severe functional impairment (FIS score 3) decreased from 42.7% at baseline to 19.9% at follow-up.

Analysis of change scores demonstrated that 7,695 patients (55.7%) experienced improvement in functional impairment (FIS reduction ≥1 point), while 9,356 patients (67.7%) reported improvement in pain severity (VAS reduction ≥1 point), supporting the effectiveness of vitamin D3 supplementation in routine clinical practice.

Exploratory Analysis of Pain Reduction Across Clinical Subgroups

An exploratory subgroup analysis was performed to evaluate differences in pain reduction across selected demographic and clinical characteristics. Mean VAS reduction did not differ significantly between male and female patients (1.74 ± 2.41 vs 1.79 ± 2.37; p = 0.267). Patients with nociceptive pain demonstrated significantly greater pain reduction compared with those with neuropathic pain (2.99 ± 2.44 vs 1.59 ± 2.34; p < 0.001). VAS reduction also varied significantly across age groups (p < 0.001 by ANOVA), with mean reductions of 1.18 ± 2.45 in patients aged 18–39 years, 1.85 ± 2.43 in those aged 40–59 years, and 1.96 ± 2.10 in patients aged ≥60 years, indicating greater improvement among older patients.

Table 3. Exploratory Analysis of Mean VAS Reduction Across Clinical Subgroups

Subgroup	Category	Mean VAS Reduction ± SD	Statistical Test Used	Test Statistic	p-value
Gender	Male (n = 8,659)	1.74 ± 2.41	Independent Samples t-test	t = 1.11	0.267
	Female (n = 5,165)	1.79 ± 2.37			
Pain Type	Neuropathic (n = 12,142)	1.59 ± 2.34	Independent Samples t-test	t = -22.18	<0.001
	Nociceptive (n = 1,682)	2.99 ± 2.44			
Age Group (years)	18–39 (n = 2,239)	1.18 ± 2.45	One-Way ANOVA	F = 80.41	<0.001
	40–59 (n = 9,167)	1.85 ± 2.43			
	≥60 (n = 2,418)	1.96 ± 2.10			

Compliance

After exclusion of implausible values (>24 months), the mean duration of supplementation was 6.32 months (median 6 months; range 0–24 months), indicating sustained adherence in the majority of patients.

Safety and Tolerability

Vitamin D3 supplementation was generally well tolerated. A total of 13,383 adverse events were reported across the cohort; all were mild and transient. No serious adverse events or clinically significant safety concerns were documented. There were no reported cases of hypercalcemia or treatment discontinuation due to intolerance.

DISCUSSION

This large multicenter real-world study demonstrates a high prevalence of vitamin D deficiency among patients with chronic low back pain (CLBP), with a mean baseline serum 25-hydroxyvitamin D level of 11.49 ng/mL, well below commonly accepted sufficiency thresholds. These findings are consistent with prior observational studies reporting a high burden of vitamin D deficiency among individuals with chronic musculoskeletal pain syndromes[5,6]. In a case–control study, Nimisha V. et al. demonstrated significantly lower serum vitamin D levels in patients with low back pain compared with healthy controls[5]. The present study extends these observations in a substantially larger and more heterogeneous real-world cohort.

Following supplementation, serum vitamin D levels increased significantly, confirming biochemical responsiveness to therapy. However, the mean follow-up level remained below optimal sufficiency thresholds, suggesting that longer duration of therapy or structured maintenance regimens may be required for full correction in routine clinical practice. Similar observations have been described in clinical practice–based populations with severe baseline deficiency[7].

Importantly, biochemical improvement was accompanied by substantial reductions in pain severity and improvements in functional impairment. Vitamin D receptors are expressed in skeletal muscle, immune cells, and neural tissues, and experimental studies support a role for vitamin D in modulation of inflammatory cytokines and nociceptive signalling pathways[3]. These mechanisms provide biological plausibility for the symptomatic improvement observed.

In addition, vitamin D plays a fundamental role in calcium homeostasis by enhancing intestinal calcium absorption and maintaining skeletal integrity. Deficiency can result in impaired bone mineralization, muscle weakness, and increased susceptibility to musculoskeletal pain. Restoration of vitamin D levels may therefore contribute to improvement in pain and functional outcomes not only through anti-inflammatory and neuromodulatory mechanisms but also via correction of underlying calcium imbalance. In routine clinical practice, vitamin D is often co-administered with calcium supplementation, which may have an additive effect on musculoskeletal health and symptom relief[2].

Published interventional studies evaluating vitamin D supplementation in chronic pain have shown heterogeneous results. While some randomized controlled trials have reported modest or inconsistent benefits, meta-analyses suggest that individuals with documented deficiency may derive greater symptomatic improvement compared to vitamin D replete populations[4,6]. The present large real-world cohort suggests that clinically meaningful improvement may be achievable in routine practice, particularly among patients with nociceptive pain phenotypes.

Exploratory analysis demonstrated greater pain reduction among patients with nociceptive pain compared with neuropathic pain, as well as progressively greater improvement in older age groups. These findings align with the hypothesis that vitamin D's anti-inflammatory and musculoskeletal effects may exert stronger influence in nociceptive or inflammatory pain states than in primarily neuropathic pain conditions. However, because these analyses were exploratory and not pre-specified, they should be considered hypothesis-

generating.

Reduced vitamin D levels have been implicated in enhanced pain sensitivity and modulation of neuroinflammatory pathways in chronic pain states, vitamin D plays a profound role in astrocyte detoxification pathways, and thereby provides a neuroprotective action. Vitamin D exerts neuroprotective effects through its action on glial cells, including astrocytes and microglia, with downstream effects on inflammatory mediators such as tumor necrosis factor-α and interleukin-1β, which are known contributors to pain sensitization. Improvement in vitamin D status may therefore contribute to regulation of neuronal excitability and central sensitization mechanisms. These pathways are particularly relevant in the context of neuropathic pain, which constituted a substantial proportion of the present cohort. Although the magnitude of improvement was greater in nociceptive pain, the potential role of vitamin D in modulating neuropathic pain pathways warrants further investigation[8].

Functional improvement paralleled pain reduction, indicating that symptomatic benefit translated into measurable enhancement of daily activity. Restoration of function is a primary therapeutic goal in CLBP management, and concordant improvement in both VAS and FIS supports internal consistency of the findings.

The large sample size and multicenter design enhance external validity and generalizability. Unlike tightly controlled trials with restrictive eligibility criteria, this real-world study reflects heterogeneous patient populations, varied comorbidities, and concurrent medication use typical of routine clinical practice. Real-world evidence complements randomized trials by evaluating effectiveness in broader populations[9].

A key methodological consideration is the use of available-case analysis for outcome evaluation. In real-world datasets, variability in documentation across endpoints is expected, reflecting routine clinical practice rather than protocol-driven data collection. By analyzing all available paired data for each outcome, the present study maximizes data utilization and enhances generalizability, although it may introduce minor variability in denominators across endpoint.

Nevertheless, several limitations warrant consideration. The retrospective design limits causal inference, and the absence of a placebo-controlled comparator precludes definitive attribution of symptom improvement solely to vitamin D supplementation. Concomitant therapies, including analgesics and muscle relaxants, may have contributed to observed clinical benefit. Follow-up duration varied across patients, and long-term sustainability of improvement was not assessed. Residual confounding and documentation bias inherent to real-world datasets cannot be excluded. The use of available-case analysis resulted in varying denominators across endpoints, which may introduce potential selection bias if missing is not completely random. However, the large sample size and consistency of findings across outcomes mitigate this concern.

Despite these limitations, the magnitude of symptomatic and functional improvement observed in a large, diverse cohort provides clinically relevant insight into the potential role of vitamin D supplementation as part of multimodal management in CLBP. Prospective randomized studies with stratification by pain phenotype and standardized dosing protocols are warranted to confirm these findings and clarify optimal therapeutic strategies.

CONCLUSION

In this large real-world cohort of patients with chronic low back pain, vitamin D deficiency was found to be highly prevalent, highlighting the potential contribution of suboptimal vitamin D status to the overall burden of musculoskeletal symptoms in routine clinical practice. Vitamin D3 supplementation was associated with meaningful improvements in serum vitamin D levels, accompanied by reductions in pain intensity and improvements in functional status during follow-up. These findings suggest that identification and correction of vitamin D deficiency may represent an important component of the comprehensive management of chronic low back pain, particularly in populations at high risk of deficiency. While chronic low back pain is multifactorial and requires a multimodal treatment approach, optimization of vitamin D status may complement existing therapeutic strategies and contribute to improved patient outcomes. Further prospective studies are warranted to better define the long-term clinical impact of vitamin D supplementation and to identify patient subgroups most likely to benefit from targeted intervention.

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