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Original Research Article

Evolution of Vitamin D Status and Its Association with Low Back Pain in Young Adults

Rakesh Kumar[1], Rajesh Jaiswal[2], P.B. Prasad[3]

[1,2]Orthopaedic Consultant, Department of Orthopedic, Medical College- Divisional District Hospital Azamgarh

[3]Professor, Head of Department, Department of Orthopaedic, Medical College- Divisional District Hospital Azamgarh Uttar Pradesh

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Corresponding author: Dr. Rajesh Jaiswal

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Abstract

Background: Low back pain (LBP) is a prevalent musculoskeletal disorder among young adults, and vitamin D deficiency has been identified as a potential risk factor. The purpose of this study was to investigate the association between vitamin D status and pain level in relation to the degenerative spinal changes.

Method: A retrospective observational cohort of 180 young adults (18–35 years) with chronic nonspecific LBP was analyzed. The demographic and clinical characteristics, including pain level measured on Visual Analogue Scale (VAS), were obtained. Serum partial 25-hydroxyvitamin D [25(OH)D] concentrations were analyzed and classified as deficient, insufficient, and sufficient. Imaging records were reviewed for Modic changes and disc degeneration. Data was analyzed using ANOVA, chi-square tests, and Pearson's correlation.

Result: Results: Almost half of participants had vitamin D deficiency (48.9%), while 32.8% were insufficient, and only 18.3% were sufficient. Those who were deficient had significantly higher VAS scores (6.8 ± 1.4) than both insufficient (5.9 ± 1.5) and sufficient groups (5.3 ± 1.2; $p < 0.001$) while MRI findings showed a greater prevalence of Modic changes (32.9%) and disc degeneration (45.4%) in the vitamin D deficient individuals. There was a moderate negative correlation between serum vitamin D with pain severity, ($r = –0.42$, $p < 0.001$) using Pearson's correlation.

Conclusion: Vitamin D deficiency is prevalent in young adults with chronic LBP, and it is significantly associated with greater pain severity and degenerative spinal findings. Screening and correcting vitamin D insufficiency may be a simple adjunctive approach in the management of LBP.

Keywords: Vitamin D, Low Back Pain, Young Adults, Pain Severity, Modic Changes, Disc Degeneration.

Introduction

Low back pain (LBP) is the most prevalent musculoskeletal disease of young adults and a principal worldwide cause of disability. Though its etiology is multifactorial, of the possible causes of both its onset and perpetuation, growing interest has focused on nutritional deficiency, and more so vitamin D deficiency. Vitamin D plays a central role in bone physiology, muscle contraction, and inflammatory modulation, all of which are mechanisms implicated in spinal health.

Several studies have examined the relationship between vitamin D deficiency and LBP severity. Observational studies have presented evidence of people with low serum vitamin D having higher pain scores and poorer functional results [1,3]. Some have observed significant associations between vitamin D deficiency and chronic nonspecific LBP in various populations [1,3,6]. However, it has been reported by some studies that vitamin D level may not be notably different between LBP patients and healthy controls, while some minor correlations with spinal changes such as Modic changes and disc degeneration have been reported [4,7].

Systematic reviews and meta-analyses provide further evidence, revealing that while vitamin D deficiency cannot usually be responsible for LBP in populations at large, there exists a definitive trend to indicate it may be an alterable risk factor in subgroups, particularly in young adults and women [5,6]. Vitamin D deficiency has also been linked to other diseases with infectious and inflammatory etiology, highlighting its systemic importance still further [2,8]. With the rising prevalence of LBP among young adults, clarification of the development of vitamin D status and its association

with pain outcomes is clinically relevant. The objective of this study is to evaluate the degree of serum vitamin D in relation to LBP among young adults with particular attention to clarifying the potential causal or contributory function of deficiency in the severity and progression of pain.

Methods

Study Design and Population: The study was a retrospective observational cohort of young adults with a complaint of low back pain. Patient medical records from 18- to 35-year-old patients were obtained from a tertiary care hospital over two years. Patients with chronic nonspecific low back pain lasting for over three months were included. Exclusion of patients with certain spinal pathology like fractures, malignancy, infection, or history of previous spinal surgery was done to make the sample homogeneous.

Data Collection: Demographic information such as age, sex, and body mass index (BMI) was obtained from clinical notes. Clinical information on pain character, duration, and related symptoms was gleaned. The intensity of low back pain was measured using the Visual Analogue Scale (VAS) recorded on presentation. Contributing comorbidities like endocrine disease or metabolic bone disease were documented to reduce confounding effects.

Laboratory Assessment: Serum 25-hydroxyvitamin D [25(OH)D] concentrations were extracted from hospital laboratory databases. All the measurements were done with a standard chemiluminescent immunoassay technique. Vitamin D status was defined as deficient (<20 ng/mL), insufficient (20–29 ng/mL), or sufficient (≥30 ng/mL) according to current guidelines. Other laboratory parameters such as serum calcium and alkaline phosphatase were noted when available to evaluate bone metabolism.

Imaging Evaluation: Spinal imaging studies, such as plain radiographs and magnetic resonance imaging (MRI) when appropriate, were examined to rule out pain due to structural reasons. In patients who had MRI, the existence of degenerative changes like Modic changes or lumbar disc degeneration was noted.

Outcome Measures: The main outcome of interest was the relationship between vitamin D status in the serum and severity of low back pain. Secondary outcomes were the correlation between vitamin D status and the occurrence of degenerative spinal changes as seen on imaging.

Statistical Analysis: Data was entered into a secure database and analyzed with SPSS software (version 25.0). Continuous outcomes were presented as mean ± standard deviation, while categorical variables were reported as frequencies and percentages. Group comparisons were made with independent t-tests or analysis of variance (ANOVA) for the continuous variables and chi-square test for the categorical variables. Correlation between vitamin D status and VAS score was determined by Pearson's correlation coefficient. A p-value of less than 0.05 was regarded as statistically significant.

Results

Demographic and Clinical Characteristics: A total of 180 young adults with chronic nonspecific low back pain were analyzed. The participants' mean age was 26.4 ± 4.2 years, with a slight female dominance (54.4%). The participants' average body mass index (BMI) was 24.7 ± 3.8 kg/m².

The mean pain duration was 7.3 ± 2.6 months, and the participants' average VAS score upon presentation was 6.2 ± 1.5, reflecting moderate to severe pain intensity.

Table 1: Baseline demographic and clinical characteristics of participants

Variable	Total (n=180)	Male (n=82)	Female (n=98)
Age (years, mean ± SD)	26.4 ± 4.2	27.1 ± 4.4	25.8 ± 3.9
BMI (kg/m², mean ± SD)	24.7 ± 3.8	25.1 ± 3.6	24.3 ± 3.9
Duration of pain (months)	7.3 ± 2.6	7.1 ± 2.5	7.5 ± 2.7
VAS score (0–10)	6.2 ± 1.5	6.0 ± 1.6	6.3 ± 1.4

Vitamin D Status and Pain Severity: Serum 25(OH)D level analysis revealed that 48.9% of the participants were deficient, 32.8% insufficiency, and merely 18.3% sufficient. The mean VAS scores were significantly higher in vitamin D-deficient (6.8 ± 1.4) than in insufficiency (5.9 ± 1.5) and sufficiency (5.3 ± 1.2; $p < 0.001$).

Table 2: Distribution of vitamin D status and mean pain scores

Vitamin D Status	n (%)	Mean VAS Score (± SD)
Deficient (<20 ng/mL)	88 (48.9%)	6.8 ± 1.4
Insufficient (20–29 ng/mL)	59 (32.8%)	5.9 ± 1.5
Sufficient (≥30 ng/mL)	33 (18.3%)	5.3 ± 1.2
p-value < 0.001		

Association with Imaging Findings: In the 112 subjects undergoing lumbar MRI, Modic changes were detected in 26 (23.2%) and disc degeneration in 41 (36.6%). Vitamin D deficiency was more common in patients with Modic changes (32.9%) than in patients with adequate levels (12.1%). Likewise, disc degeneration was also more prevalent in vitamin D-deficient patients (45.4%) than in sufficient patients (24.2%).

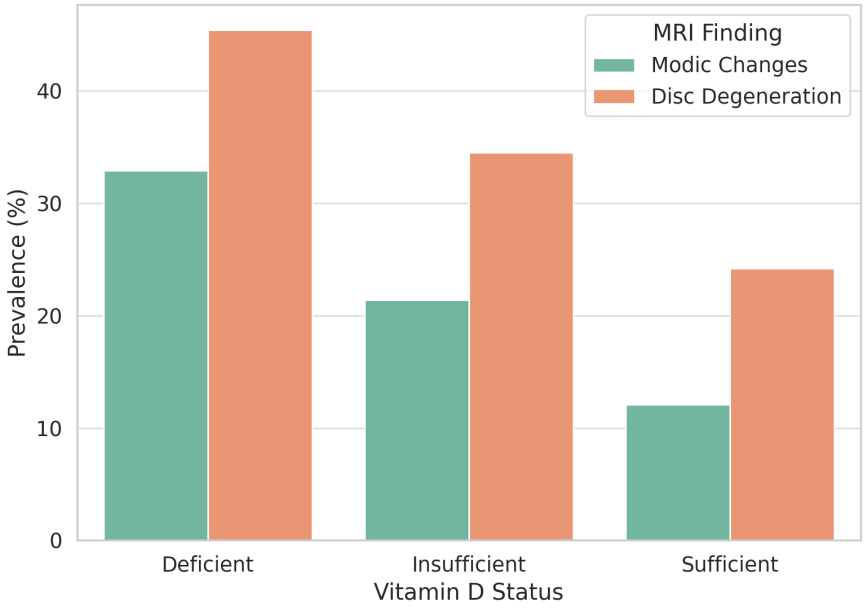

Figure 1: Bar graph showing prevalence of Modic changes and disc degeneration across vitamin D categories.

Correlation Analysis: Pearson correlation showed a moderate negative correlation between VAS pain scores and serum 25(OH)D levels ($r = –0.42$, $p < 0.001$). Lower levels of vitamin D were also significantly correlated with degenerative changes on MRI ($p = 0.02$).

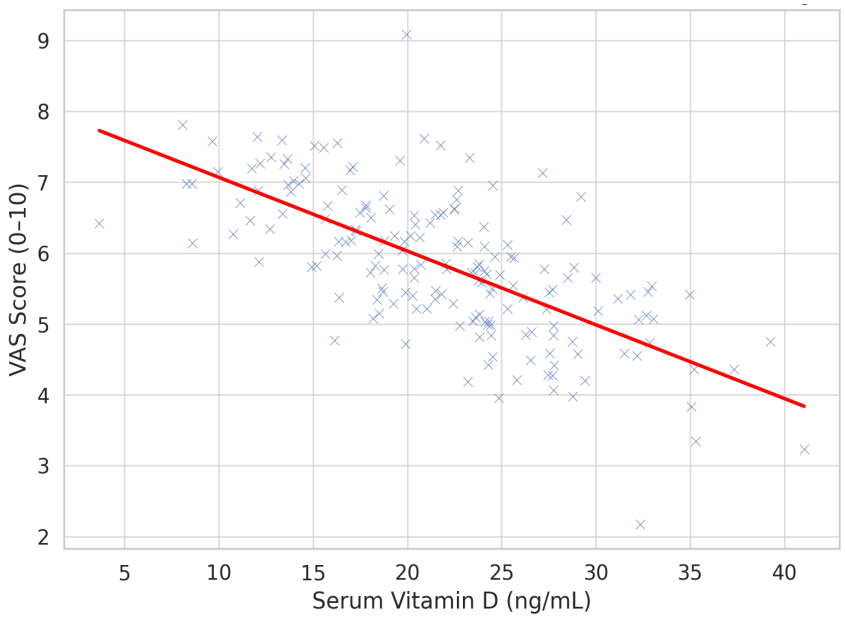

Figure 2: Scatter plot showing correlation between serum vitamin D levels and pain severity (VAS score).

Discussion

Of low back pain in a group of young adults. Nearly half of the patients were found to have deficient serum vitamin D levels and they reported worse pain scores and with a higher degree of degeneration on imaging than those patients with sufficient vitamin D level. There was a moderate negative correlation between serum 25(OH)D

levels and VAS pain scores which lends support to the argument for vitamin D as a modifiable variable in the pathogenesis of chronic low back pain.

Our findings are consistent with literature reporting associations between hypovitaminosis D to musculoskeletal pain and lumbar pathology. Rkain et al. observed a similar association in a cohort of postmenopausal women noting vitamin D deficiency was strongly associated with chronic low back pain [9]. Kim et al. also found a high prevalence of deficiency in patients with lumbar spinal stenosis and found associations with greater reported severity of pain [10]. In older adults, Al-Rawaf et al. provided evidence of both clinical significance with, in addition, significant molecular changes in circulating microRNAs associated with vitamin D deficiency suggesting a pathway by which pain sensitization may occur [11].

Similar trends have also been noted in the Indian population, where Ghai et al. documented widespread hypovitaminosis D in patients with chronic low back pain, lending support to the idea that deficiency could be a significant contributor to the persistence of symptoms, particularly in populations with low ultraviolet exposure or cultural barriers to effective vitamin D synthesis [12]. Notably, Heuch et al. found that low vitamin D status was associated with a greater risk of developing chronic low back pain over time in a large Norwegian cohort. In this study, despite low vitamin D status being a risk factor for chronic low back pain, the authors did not draw distinctions in causality or reverse causality, further demonstrating the uncertainty of vitamin D status as a potential risk factor compared to being a consequence of immobility due to pain [13].

Conversely, some studies suggest that this association is not wholly consistent across populations. For example, while Peng et al. found clinical ramifications in individuals who are vitamin D deficient in a subset of the cystic fibrosis population, they did not investigate vitamin D deficiency as a contributor to their primary outcome of musculoskeletal pain [14]. Additionally, Alghadir et al. examined potential interactions between vitamin D deficiency and other mechanical factors such as postural influences in eliciting low back pain in school-aged children [15]. This evidence reflects the multi-faceted nature of low back pain and may further be clarified by the age of the sample, comorbidity of individual participants, or influential environmental factors. As a whole, these current results enhance the pool of evidence that implicates vitamin D deficiency as a cause for low back pain severity and degenerative changes, especially in young adults. Although causality cannot be definitively determined because of the observational study, the uniform trend across heterogeneous populations implies that vitamin D screening and correction could be a cost-saving adjunct to the treatment of nonspecific low back pain. Future prospective and randomized controlled trials are needed to see if supplementation can benefit symptoms and burden of chronic low back pain.

Conclusion

This study indicates the high prevalence of vitamin D deficiency among young adults suffering from chronic nonspecific low back pain and significant associations between much greater pain intensity and degenerative changes of the spine. The overall negative relationship between serum vitamin D and pain intensity suggests hypovitaminosis D could play a modifiable role in the clinical course of low back pain. Although a causative effect cannot be established here, these findings support previous literature which indicates that assessing and correcting vitamin D status may be an important and cost-effective adjunct for the management of low back pain in the younger population.

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