 Vitamin D has a significant role to play in bone metabolism and neuromuscular function. Several researchers have indicated that Vitamin D deficiency may be possibly related to chronic musculoskeletal pain including chronic low back pain (CLBP).

Objectives: The present study was conducted to determine the prevalence of hypovitaminosis D and its contribution to chronic lower back pain.

Study Design: Controlled study

Setting: Outpatient pain clinic of tertiary care hospital.

Methods: Data presented in this manuscript are from patients who were screened for inclusion in an open label, single arm clinical trial aimed to assess the effectiveness of vitamin D supplementation in patients with CLBP. Consecutive patients visiting the outpatient pain clinic of a tertiary care hospital with a diagnosis of CLBP with or without leg pain were recruited. A visual analogue scale (VAS) was used to measure low back pain intensity, and the Modified Oswestry disability questionnaire (MODQ) was used to measure functional ability. Plasma 25-OHD levels of all patients were measured and the prevalence of hypovitaminosis D was calculated. The multivariate logistic regression model was used to investigate the association between vitamin D deficiency and patient characteristics.

Results: A total of 328 patients were included in the study. Mean age of the study population was 43.8 years. Two hundred eighty-two (86%) (men 153/172 [89%], women 129/156 [83%]) of patients had below normal plasma vitamin D levels. Among these, 217 (66%) (men 126 [73%), women 91 [58%]) were found to be deficient and 65 (20%) (men 27 [16%], women 38 [24%]) were had insufficient levels. Multivariate regression analysis found that men were significantly more prone to have deficiency as compared to women (OR = 1.78 (1.10 – 2.88), \( P = 0.02 \)). We also found a significantly positive relationship between vitamin D deficiency and increased functional disability (OR = 1.53 (1.24 – 1.87), \( P = 0.01 \)). However, we did not find any relationship with pain severity, presence of other co-morbidities and educational level.

Limitations: Not possible to access a good quality data on sun exposure and vitamin D dietary intake in study population. No bone scans were performed.

Conclusion: The result of this study provides a message about the high prevalence of hypovitaminosis D in the Indian CLBP population. Clinical guidelines for managing CLBP should include assessment of vitamin D status, together with advice on appropriate vitamin D supplementation in those found to be deficient.

Clinical trial registration: CTRI/2014/03/004459

Key words: Chronic low back pain, disability, hypovitaminosis D, India, pain severity, prevalence, vitamin D, visual analogue scale
Chronic low back pain (CLBP) is a clinical syndrome characterized with pain localized in the area below the costal margins and above the inferior gluteal folds and may be accompanied by leg pain or motor, sensory, and reflex deficits in nerve root distribution for ≥ 12 weeks (1). CLBP is often progressive and the exact etiology is difficult to determine. Most patients are often diagnosed with nonspecific low back pain.

Vitamin D has a significant role to play in bone metabolism and neuromuscular function (2). Its deficiency leads to rickets in children and osteomalacia in adults. Vitamin D deficiency is also being linked to nonspecific symptoms such as fatigue and chronic pain (2). The supplementation with calcium and vitamin D has demonstrated vitamin D’s effectiveness in lowering fracture risk in osteoporotic patients (3). Recent research has shown a much broader role of vitamin D and increasing numbers of investigations are being performed to show the effects of hypovitaminosis D and/or vitamin D supplementation on different health aspects. Recent data show vitamin D plays role in inflammatory- and lifestyle-related chronic disorders like diabetes, cardiovascular diseases, auto-immune disorders, depression, and some cancers (4-7). It is also been proposed to exhibit anti-inflammatory properties (8). Several researchers have indicated that vitamin D deficiency may be possibly related to chronic musculoskeletal pain (9).

Vitamin D deficiency has been reported in populations with several different types of chronic musculoskeletal pain such as osteoarthritis, rheumatoid arthritis, osteoporosis, soft tissue rheumatism, low back pain, and arthralgia (4-7). Physiologically, vitamin D plays a crucial role in maintenance of extracellular calcium levels, a prerequisite for adequate musculoskeletal function (4). Furthermore, Autier and Gandini (7) have shown mortality reduction with vitamin D supplementation.

Risk factors for vitamin D deficiency include inadequate solar exposure or dietary intake, dark skin color, advanced age, obesity, and certain medications (10). A high prevalence of hypovitaminosis D has been found in various communities like adolescent girls (11), the overweight population and African-Americans of all ages (12), failed back surgery patients (13), nonspecific musculoskeletal pain (14), etc. Thus, vitamin D deficiency is expected to be a major problem globally.

The prevalence of vitamin D deficiency is 50 – 90% on the Indian subcontinent and is attributed to low dietary intake along with skin color and changing lifestyle (15). In a study conducted in the Indian adult population, the prevalence of vitamin D deficiency is estimated to be between 62-75% in the urban population and 44 – 70% in the rural population (16). The patients with persistent, musculoskeletal pain, especially CLBP patients, are at high risk for the consequences of unrecognized and untreated vitamin D deficiency (17). There have been no prospective studies conducted on focused populations groups like patients with CLBP in India. Thus, the present study was conducted to determine the prevalence of hypovitaminosis D and its contribution to CLBP and functional disability in patients.

Methods

Study Design and Population

Data presented in this manuscript are from patients who were screened for inclusion in open label, single arm clinical trial aimed at assessing the effectiveness of vitamin D supplementation in patients with CLBP. The trial was a single center prospective study. Consecutive patients visiting the outpatient pain clinic of a tertiary care hospital setting with a diagnosis of CLBP with or without leg pain were recruited. STROBE (Strengthening the Reporting of Observational studies in Epidemiology) guidelines were followed while reporting the study. The diagnosis of CLBP with or without radicular pain was established based on signs and symptoms and investigated using magnetic resonance imaging (MRI). The trial was approved by the PGIMER institutional review board (Chandigarh, India), and registered with the Clinical Trial Registry of India (CTRI). All patients provided written, informed consent.

Participants were recruited from patients referred to the pain clinic of PGIMER from January 2013 to July 2014. The study site is located in northern India (Chandigarh). North India has a humid subtropical hot summer climate that is mild with dry winters, hot humid summers, and moderate seasonality with annual sunshine of about 2,762 hours, mean annual temperature of 73 degrees Fahrenheit, and average daylight hours/day across the year ranging from 10 to 14 hours (18). Each year, the clinic provides a comprehensive diagnostic evaluation for approximately 1000 new patients with various pain conditions.

Inclusion Criteria

Patients of either gender, aged 18 – 75 years with CLBP for ≥ 3 months, with or without leg pain radiation, were eligible for inclusion. The patients were required to have English, Hindi, or Punjabi language competency
in order to complete the baseline “pain related questionnaire.” The questionnaire included a visual analogue scale (VAS) to measure low back pain intensity, Modified Oswestry disability questionnaire (MODQ) to measure functional disability, work status, and the use of medication.

**Exclusion Criteria**

Patients were excluded if they had evidence of other causes for neuropathy and painful conditions like diabetes mellitus; rheumatoid arthritis; symptomatic osteoarthritis of the hip, knee, and ankle; epilepsy; psychiatric diseases and substance abuse; metabolic bone disease (hypo- or hyperparathyroidism); chronic renal disease, medical or surgical disorders affecting vitamin D metabolism (gastric surgery, chronic liver disease, renal failure, intestinal malabsorption, systemic infection, cancers etc.). Patients consuming drugs altering bone metabolism like corticosteroid or bisphosphonates and pregnant and lactating mothers and women intending pregnancy were also excluded. Patients taking vitamin-D supplements during past 3 months were also excluded from the present study.

**Procedure**

All new patients referred to the pain clinic during the data collection period were screened. Patients meeting the inclusion criteria were invited to participate. All eligible patients who provided written informed consent were enrolled. At enrollment, the baseline evaluation was performed by a pain physician/study investigator. All the information was recorded in a structured case record form. Baseline evaluation included socio-demographic characteristics including age, gender, education, and smoking and alcoholic status as assessed through direct patient interview. Height and weight were measured to calculate body mass index (BMI), and categorized as < 18.4, 18.5 to 24.99, 25 to 29.99, and ≥ 30 kg/m2, which are the cut-off points for underweight, normal, overweight, and obesity (19).

As north India enjoys well-defined 4 seasons, dates for measurement of plasma vitamin D were recorded to assess the seasonal variation in levels. The seasons were categorized as winter (December to February), spring (March to May), summer (June to August), and fall (September to November).

**Assessment of Pain and Functional Disability**

For the present study, pain assessment was done for regular/often occurrence of pain during the past month. Pain location and severity were recorded. Location was recorded using McGill Pain Map (20,21) showing the front and back of a human figure and divided as back pain and/or leg pain.

Pain severity was measured as the average pain in the past month based on a 0 – 100 (with 0 as no pain and 100 representing the worst pain) VAS. Pain severity was further categorized as: mild (0 – 49), moderate (50 – 69), and severe (70 – 100) (22).

Functional disability was assessed using the modified Oswestry Low Back Pain Disability Questionnaire (MODQ). The disability was indicated as a percentage of the summation of all the MODQ score, i.e., total score/50x100. The functional disability was categorized as minimal disability (0 – 20%), moderate (20 – 40%), severe (40 – 60%), crippled (60 – 80%), and bed bound (80 – 100%) (23).

**Measurement of Plasma Vitamin D Levels (25-Hydroxyvitamin D [25-OHD])**

After an overnight fasting, a blood sample was taken. Plasma 25-OHD levels of all patients were measured by electrochemiluminescence immunoassay (ECLIA) on an automated analyzer (ELECSYS-2010), using kits supplied by Roche Diagnostics (Germany). This technique provides a broad measuring range and high precision at the low end of detection to aid in the assessment of deficient patients. All the blood samples were collected between 9:00AM and 10:00 AM to prevent any circadian variation.

**Definition of vitamin D levels**

According to the level of 25-OHD, vitamin D deficiency was defined as a 25-OHD level of ≤ 20 ng/mL and vitamin D insufficiency as 21 to 29 ng/mL, and normal level as above 30 ng/mL (24). Further, severity of vitamin D deficiency was grouped as follows: ≤ 4 ng/mL profound deficiency; 5 – 8 ng/mL severe deficiency; 9 – 12 ng/mL moderately severe deficiency; 13 – 16 ng/mL moderate deficiency; and 17 – 20 ng/mL marginal deficiency (25-28).

**Statistical Analysis**

Descriptive data for all variables included in the study were reported as mean and standard deviation (SD), numbers and percentage (%), and median and interquartile range (IQR). Categorical variables were tested using a chi-square test and continuous variables using an independent t-test. A multivariate logistic regression model was used to investigate the association.
between vitamin D deficiency and patient characteristics. Univariate analysis of variance (ANOVA) followed by post hoc Bonferroni test was performed to assess the difference in mean 25-OHD values according to the season. All statistical tests were performed by using SPSS 15.00 version (SPSS, Inc., Chicago, IL). A P value of < 0.05 was accepted as significant.

**RESULTS**

**Patients Characteristics and Prevalence of Vitamin D**

A total of 362 patients were screened during the study period. Among them, 34 patients were excluded due to incomplete data and non-eligibility. A total of 328 (91%) patients were included in the study (Table 1). Mean (SD) age of the study population was 43.8 (13.9) years (range, 36 – 52). Among the study cohort, 172 (52%) were men and 156 (48%) women. Two-third of the patients presented with radicular leg pain which was equally distributed among both genders. All the patients belonged to the same ethnicity. Baseline characteristics were found to be comparable among men and women except that women were found to have a higher level of disability as compared to men (P = 0.01) and a higher frequency of smokers and alcohol users were found to be men (P = 0.01). Mean plasma vitamin D level at presentation was 18.4 (11.7) ng/mL. The mean (SD) pain duration was 48.3 (54) months and the mean (SD) pain intensity measured on VAS was 77 (23) (Table 1).

**Vitamin D Levels at Presentation**

Among the study cohort only 46 (14%) patients had normal vitamin D levels. Two hundred eighty-two (86%) (men 153/172 [89%], women 129/156 [83%]) patients had below normal plasma vitamin D levels. Among these, 217 (66%) (men 126 [73%], women 91 [58%]) were found to be deficient and 65 (20%) (men 27 [16%], women 38 [24%]) had insufficient levels. Among 217 patients who were found to be deficient, 58 (18%) patients had marginal deficiency, 44 (13%) had moderate, 58 (18%) had moderately severe, 48 (15%) had severe, and 9 (3%) had profound deficiency. A significantly higher number of men 126 (73%) were found to be deficient as compared to women 91 (58%) (P = 0.006) (Table 2).

**Variables associated with Vitamin D Deficiency**

The majority of the study population belonged to the age group 39 – 58 years and the prevalence of vitamin D deficiency was found to be maximum in this age bracket (P = 0.001). The prevalence of vitamin D deficiency was found to be maximum in women (P = 0.001). Mean plasma vitamin D level at presentation was 18.4 (11.7) ng/mL. The mean (SD) pain duration was 48.3 (54) months and the mean (SD) pain intensity measured on VAS was 77 (23) (Table 1).

**Table 1. Demographic and clinical characteristics of patients with chronic low back pain (n = 328).**

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (%)</td>
<td>172 (52)</td>
<td>156 (48)</td>
<td>328 (100)</td>
<td></td>
</tr>
<tr>
<td>Age (Yr)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>43.4 (13.9)</td>
<td>44.2 (13.9)</td>
<td>43.8 (13.9)</td>
<td>0.603a</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>44 (35 – 51)</td>
<td>43.5 (38 – 53)</td>
<td>44 (36 – 52)</td>
<td></td>
</tr>
<tr>
<td>BMI (Kg/m2)</td>
<td>25.2 (6.8)</td>
<td>24.8 (8)</td>
<td>25.2 (7.4)</td>
<td>0.627a</td>
</tr>
<tr>
<td>VAS (0 – 100),</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>78 (22)</td>
<td>76 (24)</td>
<td>77 (23)</td>
<td>0.320a</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>80 (60 – 100)</td>
<td>80 (60 – 100)</td>
<td>80 (60 – 100)</td>
<td></td>
</tr>
<tr>
<td>Pain Duration (month)</td>
<td>44.6 (50.6)</td>
<td>52.3 (57.46)</td>
<td>48.3 (54)</td>
<td>0.199a</td>
</tr>
<tr>
<td>Median (range)</td>
<td>36 (12 – 48)</td>
<td>36 (12 – 72)</td>
<td>36 (12 – 60)</td>
<td></td>
</tr>
<tr>
<td>Presence of leg pain, n (%)</td>
<td>117 (68)</td>
<td>111 (71)</td>
<td>228 (70)</td>
<td>0.551b</td>
</tr>
<tr>
<td>Vitamin D level (ng/ml)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>17.3 (9.9)</td>
<td>19.6 (13.3)</td>
<td>18.4 (11.7)</td>
<td>0.072a</td>
</tr>
<tr>
<td>MODQ</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>40.7 (17.5)</td>
<td>46.2 (19.9)</td>
<td>43.3 (18.8)</td>
<td>&lt; 0.01a</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>42 (32 – 51)</td>
<td>49 (35 – 60)</td>
<td>44 (33 – 57)</td>
<td></td>
</tr>
<tr>
<td>Smokers n (%)</td>
<td>49 (29)</td>
<td>4 (3)</td>
<td>53 (16)</td>
<td>&lt; 0.01b</td>
</tr>
<tr>
<td>Alcohol users n (%)</td>
<td>10 (5.81)</td>
<td>0</td>
<td>10 (3.04)</td>
<td>&lt; 0.01b</td>
</tr>
</tbody>
</table>

a- Assessed using independent t-test; b- Assessed using chi-square test. BMI-Body mass index, VAS-Visual analogue scale, MODQ-Modified Oswestry disability questionnaire, IQR- Inter quartile range
Prevalence of Hypovitaminosis D in Chronic Low Back Pain

We also found that the deficiency was more prevalent in the overweight population (68%) and in men (73%) (Table 3).

Further on multivariate regression analysis we found men to be significantly more prone to have deficiency as compared to women (OR = 1.78 (1.10 – 2.88), P = 0.02). We also found a significant relationship between vitamin D deficiency and increased functional disability (OR = 1.53 (1.24 – 1.87), P = 0.01). However, we did not find any relationship with pain severity, presence of other co-morbidities, and educational level (Table 4).

We also tried to assess the impact of seasonal variation (depending upon sunlight) on vitamin D deficiency. The mean levels during all the seasons, i.e., spring 16.3 (11.9), summer 20.9 (11.8), fall 18.5 (8.1), and winter 19.6 (12.5) were comparable (P > 0.05), (Fig. 1). However, we found a significantly lower prevalence of vitamin D deficiency in summers 57% (54/94) and winters 51% (27/53) as compared to spring 76% (108/142) and fall 72% (28/39) (P < 0.05).

**DISCUSSION**

This is the first study to investigate the prevalence of hypovitaminosis D in patients with CLBP with or without radicular pain residing in north India presenting in a tertiary care referral pain clinic. No sufficient studies have been conducted until now to analyze hy-

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**Table 2. Prevalence of vitamin D deficiency according to various categories of deficiency.**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number of patients</th>
<th>25-OHD (ng/mL)</th>
<th>Total (328) N (%)</th>
<th>Men (172) N (%)</th>
<th>Women (156) N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>&gt; 30</td>
<td>46 (14)</td>
<td>19 (11)</td>
<td>27 (17)</td>
</tr>
<tr>
<td>Below Normal</td>
<td></td>
<td>≤ 30</td>
<td>282 (86)</td>
<td>153 (89)</td>
<td>129 (83)</td>
</tr>
<tr>
<td>Insufficient</td>
<td></td>
<td>21 – 30</td>
<td>65 (20)</td>
<td>27 (16)</td>
<td>38 (24)</td>
</tr>
<tr>
<td>Deficient</td>
<td></td>
<td>&lt; 20</td>
<td>217 (66)</td>
<td>126 (73)</td>
<td>91 (58)</td>
</tr>
<tr>
<td>Marginal Deficiency</td>
<td></td>
<td>&gt; 16 – 20</td>
<td>58 (18)</td>
<td>36 (21)</td>
<td>22 (14)</td>
</tr>
<tr>
<td>Moderate Deficiency</td>
<td></td>
<td>&gt; 12-16</td>
<td>44 (13)</td>
<td>32 (19)</td>
<td>12 (8)</td>
</tr>
<tr>
<td>Moderately Severe Deficiency</td>
<td></td>
<td>&gt; 8 – 12</td>
<td>58 (18)</td>
<td>35 (20)</td>
<td>23 (15)</td>
</tr>
<tr>
<td>Severe Deficiency</td>
<td></td>
<td>&gt; 4 – 8</td>
<td>48 (15)</td>
<td>21 (12)</td>
<td>27 (17)</td>
</tr>
<tr>
<td>Profound Deficiency</td>
<td></td>
<td>≤ 4</td>
<td>9 (3)</td>
<td>2 (01)</td>
<td>7 (05)</td>
</tr>
</tbody>
</table>

*a* Assessed using chi-square test
povitaminosis D as a contributory factor for CLBP.

The result of this screening data revealed a high prevalence of patients (86%) having below normal plasma vitamin D levels. Among these, 217 (66%) (men 126 [73], women 91 [58]) were deficient and 65 (20%) (men 27 [16] women 38 [24]) had insufficient levels.

Vitamin D is a hormone that helps to maintain bone strength by regulating the minerals like calcium and phosphorus. It is an essential nutrient for bone metabolism and neuromuscular function (26). Without the presence of activated vitamin D, normal bone metabolism is altered so that only 10% of calcium and 60% of phosphorus is absorbed (27) resulting in the skeleton becoming the body’s source of calcium. Bony osteoclasts dissolve bone and raise plasma calcium. This process might exacerbate osteoporosis and osteopenia and also might lead to osteomalacia (26). Vitamin D supplementation is being considered as an adjuvant therapy for musculoskeletal pain (28). Vitamin D probably also has immunomodulatory effect (29). Its supplementation could also reduce the synthesis of inflammatory cytokines and increase the synthesis of anti-inflammatory cytokines. Vitamin D deficiency can affect patients of all ages and might be an underlying factor in undiagnosed musculoskeletal pain in adults and is a potentially treatable cause (30).

Theoretically, there are 2 possible links between hypovitaminosis D and low back pain. First, in patients with low back pain, the diffuse pain in bones and muscles, weakness, and paraesthesia may be caused by hypovitaminosis D. Second, hypovitaminosis D could play a role in the development of modic changes via the increased susceptibility to inflammation in the vertebral end plates. In a recently published Danish study of 152 patients with nonspecific low back pain, a significant correlation was found between vitamin D deficiency and modic changes (31).

Recent studies have highlighted a high prevalence of vitamin D deficiency in specific populations with chronic pain similar to our study like rheumatology patients (86%) (29), women with CLBP during child bearing period (81.7%) (32), failed back surgery cases (all 6 cases) (11), non-specific musculoskeletal pain (93%) (25), patients with lumbar spinal stenosis (97.1%) and Saudi Arabian patients with CLBP (83%) (33). Our results also show that the deficiency is equally prevalent in patients with mild, moderate,
and severe CLBP, implicating no correlation between pain intensity and vitamin D deficiency as reported in earlier published studies also (31). However, this is in contrast to the study conducted in patients with lumbar spinal stenosis where a significant correlation was found between vitamin D deficiency and pain intensity (24).

Vitamin D supplementation, either oral or injectable, is required by vitamin D deficient patients. In our institute, the standard treatment guideline for replenishing vitamin D in deficient patients (serum vitamin D level 5 – 30 ng/mL) is to prescribe active vitamin D3 in a dose of 60,000 IU/week in the form of oral sachets for a period of 8 weeks. Patients having serum vitamin D level below 5 ng/mL are prescribed 60,000 IU orally for 5 days and then 60,000 IU/week for the period of 8 weeks. If serum vitamin D levels remain below the normal range (30 ng/mL) after 8 weeks of therapy, then the above mentioned treatment regimen is repeated. If the repeat measured level is in the normal range after 8 weeks of therapy (30 to 60 ng/mL) then patients are prescribed 60,000 IU every month until the serum vitamin D level is 60 ng/mL or more. This is slightly different from, but in line with, recommendations of the Endocrine Society Task force (50,000 U per week for 8 weeks in adults) (34).

Vitamin D supplementation improves muscle strength and balance (28). Physical activity is expected to be closely related to disability which was investigated as MODQ scores. We found significantly higher functional disability in vitamin D deficient patients as compared to others which is in agreement with previously reported studies (24).

Studies have also shown the climatic and cultural impact on vitamin D deficiency as the prevalence has been found to be high in Pakistani (35), south Arabian (32), and Norwegian populations (36) and low in Danish population (31). This might possibly be due to variable sunlight exposure and skin color differences. Persons with darker skin require increased ultraviolet B exposure for the equal production of vitamin D as compared to persons with lighter skin. Though India enjoys ample sunlight throughout the year, we have found seasonal variations in vitamin deficiency. The prevalence of vitamin D deficiency was found to be significantly low in summer (57%) and winter (51%) as compared to spring (76%) and fall (72%). This low prevalence in winter is probably due to the fact that though sunlight is less in winter, people prefer to stay out and get good sunlight exposure and because of high consumption of vitamin D rich foods in winter in India. These results are similar to that reported by Kim et al (24) where sunlight exposure score was found to be significantly lower in patients with vitamin D deficiency.

The prevalence of low back pain is highest in the age group of 40 – 50 years as compared to other age groups and women are more prone to low back pain than men. In accordance, we also found that younger patients (age range 18 – 58, 67 – 69%) had more vitamin D deficiency than older patients. We also noted the comparable prevalence of below normal levels (≤ 30 ng/mL) in both genders (89% vs. 83% in men and women, respectively). However, the prevalence of vitamin D deficiency (≤ 20 ng/mL) was significantly more (P ≤ 0.006) in men (73%) as compared to women (58%). This is in contrast to earlier published studies where a high prevalence of vitamin D deficiency was found in women (81%) (32) and postmenopausal women (79%) (30). This difference might be explained by the difference in vitamin D deficiency cut-off levels (Lotfi et al [32] have considered ≤ 40 ng/mL), difference in age group (mean age in Rkain et al [30] study is 56.5 vs. 44.2 in our study), and cultural variation and lifestyle changes. The higher prevalence of vitamin D deficiency in men may also be due to more men being smokers (29%) in comparison to women (3%). Smoking is directly associated with the increased likelihood of vitamin D deficiency (37).

Our results also showed that vitamin D deficiency was more prevalent in overweight patients than underweight, normal, and obese patients which are similar to the earlier published study (38).

Milk and other dairy products are the natural sources of calcium, phosphorus, and vitamin D (39). Sunlight exposure of the skin is one of the most important source of vitamin D. Persons with darker skin require increased ultraviolet B exposure for the equal production of vitamin D as compared to persons with lighter skin. The current sun exposure guidelines do not express the importance of clothing and race in limiting vitamin D production and other factors such as age, season, sunscreen used, obesity, smoking status, and air pollution severity (40).

The probable explanations for the high prevalence of vitamin D deficiency in patients with CLBP are inadequate sunlight exposure and sun avoidance due to difficult mobility and lifestyle modifications with more indoor activities and white collar jobs. Another probable reason could be concomitant use of medications in patients with radicular pain which might enhance the vitamin D catabolism such as anticonvulsants (41). How-
ever, one-third of patients with deficiency may have no identifiable risk factors. Although lower back pain with radicular pain in the legs may have a neurologic link in CLBP, the contribution of vitamin D deficiency on pain cannot be completely ruled out. Therefore, it can also be stated that lower vitamin D levels in patients with CLBP leads to increased pain perception from neurologic and musculoskeletal components.

The strengths of the present study are consecutive patients’ recruitment, standardized institutional methods were used for collecting self-reported data, blood was taken on the same day of filling in the self-report questionnaires, and use of a sensitive and reliable method of vitamin D level measurement i.e., 25-OH-D. In human plasma, vitamin D3 (cholecalciferol) and vitamin D2 (ergocalciferol) are bound to the vitamin D binding protein (VDBP) and transported to the liver, where both are hydroxylated to 25-OH-vitamin D (25-OH-D). This primary circulating form of vitamin D has a half-life of 2 – 3 weeks in plasma, is biologically inert but immunoreactive, and is approximately 1000-fold greater than the active form of vitamin D i.e., 1,25-dihydroxy vitamin D [1,25-(OH)2D3]. Therefore, it is considered that 25-OH-D, is the most accurate and reliable marker of vitamin D status in the human body and is the major storage form of vitamin D (42).

The present study is conducted in a public funded tertiary care hospital serving patients from at least 8 different states (Jammu & Kashmir, Haryana, Rajasthan, Punjab, Delhi, Orissa, Uttaranchal, and Uttar Pradesh). The point to be noted here is that the population included in this study represents a Northern Indian population which receives ample sunlight and covers a good part of country. Thus, the results may be generalizable across the country and to the populations residing in similar climatic conditions.

The limitations of this study include patients are representative of CLBP patients in the referral pain clinic and may not be generalized to the whole CLBP population. It was not possible to assess good quality data on sun exposure and vitamin D dietary intake diet in our study population. Moreover, bone scans were not done. So, the correlation of bone density with vitamin D levels could not be ascertained. We measured only plasma 25-OH-D levels, and bone activity markers were not studied. Therefore, it is not possible to draw conclusions about increased bone turnover in the investigated patients. Since it is a cross-sectional study, a correlation between hypovitaminosis D and CLBP, but not causation, can be derived. However, this is real world population based data reflecting the reality of clinical practice.

This study has provided vitamin D deficiency prevalence based on one time screening data only. A prospective controlled trial is required to further investigate the impact of vitamin D supplementation in deficient patients with CLBP on functional status and pain intensity. One such study to evaluate the effect of vitamin D supplementation on improvement of pain symptoms in CLBP patients is in progress by us and a randomized trial is planned by us in this regard.

**Conclusion**

The result of this study provides a message about the high prevalence of hypovitaminosis D in Indian CLBP population. Current clinical guidelines for managing CLBP should include assessment of vitamin D status, together with advice on appropriate vitamin D supplementation in those found to be deficient.

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Prevalence of Hypovitaminosis D in Chronic Low Back Pain


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