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Original Research Article

Evaluation of Association Between Vitamin D Level and Low Back Pain

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Abstract

Introduction: Low back pain (LBP) is a prevalent musculoskeletal condition affecting a substantial portion of the global population. It is a leading cause of disability worldwide and poses a considerable burden on healthcare systems and individuals alike. Despite its widespread occurrence, the etiology of low back pain remains multifactorial and complex, often involving a combination of mechanical, psychological, and environmental factors.

Aims: The aim of the study is to investigate the association between low back pain and Vitamin D deficiency and to determine if vitamin D levels correlate with pain intensity in indivisuals with low back pain.

Materials and Methods: The present study was a institution based prospective observational study, conducted at the Orthopaedic Department of I.P.G.M.E.R. & S.S.K.M. Hospital, Kolkata, a tertiary care center catering to people of West Bengal.

Result: The study found that 68 patients had low serum vitamin D levels (<20 ng/ml), with the majority (59%) falling into the mild deficiency category (10–19.9 ng/ml), 7% in the moderate deficiency category (5–9.9 ng/ml), and 2% in the severe deficiency category (<5 ng/ml). The mean duration of symptoms did not significantly differ between patients with normal vitamin D levels (21.13 ± 4.61 weeks) and those with vitamin D deficiency (20.69 ± 4.47 weeks, p = 0.655). Dietary preferences were comparable between vegetarians (57%) and non-vegetarians (43%, p = 0.742). However, the mean VAS score was significantly higher in the vitamin D deficiency group (5.60 ± 1.46) compared to the normal vitamin D group (4.88 ± 1.21, p = 0.016). Similarly, the mean ODI score was significantly higher in the deficient group (20.29 ± 13.12) than in the normal group (7.81 ± 5.62, p = 0.0002), indicating worse functional outcomes in vitamin D-deficient patients.

Conclusion: We conclude that, the study highlights that low serum vitamin D levels are prevalent among patients, with the majority exhibiting mild deficiency. While the duration of symptoms and dietary preferences showed no significant association with vitamin D levels, patients with vitamin D deficiency demonstrated significantly worse pain (as indicated by higher VAS scores) and functional outcomes (reflected by higher ODI scores) compared to those with normal vitamin D levels. These findings underscore the potential impact of vitamin D deficiency on pain and functional health, emphasizing the need for further attention to vitamin D status in clinical practice.

Keywords: Vitamin D, Low Back Pain (LBP), Lumbar Spine and Musculoskeletal Pain.

Introduction

Low back pain (LBP) is a prevalent musculoskeletal condition affecting a substantial portion of the global population. It is a leading cause of disability worldwide and poses a considerable burden on healthcare systems and individuals alike (Vos et al., 2016). Despite its widespread occurrence, the etiology of low back pain remains multifactorial and complex, often involving a combination of mechanical, psychological, and environmental factors.[1] The annual prevalence of low back pain in the worldwide is estimated at 15% to 20%,[2] According to IMCR report near about 8% peoples all state of India, and females are slightly high than male Yearly lived with Disability (YLD) low back pain. Yearly lived with Disability (YLD) low back

pain ranked 2nd place in Empowered Action Group states (Uttar Pradesh, Chhattisgarh, Madhya Pradesh, Odisha, Jharkhand, Bihar, Rajasthan, Uttarakhand) and other states (Haryana, Karnataka, Gujarat, Andhra Pradesh, West Bengal, Jammu Kashmir, Punjab, Maharashtra, Tamil Nadu, Telengana, Delhi, Union Territories, Himachal Pradesh, Goa, Kerala), 3rd in North East state (Assam, Tripura, Meghalaya, Arunachal Pradesh, Manipur, Mizoram, Nagaland, Sikkim) among 15 non-communicable diseases. It showed that higher proportion of YLD among females than among in all three states groups in 2017. [3]

Low back pain refers to spinal and paraspinal symptoms in the lumbosacral region. "Acute" typically means a duration of less than 2 to 4 weeks, subacute is up to 12 weeks, and chronic typically refers to more than 12 weeks. Various diagnostic classifications for patients with low back pain exist.[4,5] The Quebec Task Force on Spinal Disorders categorizes patients based upon history (location and duration of symptoms and working status), clinical findings and response to treatment.[5] The differential diagnosis of low back pain is broad and includes mechanical and nonmechanical causes.

For most patients with acute low back pain in primary care, the etiology is thought to be a mechanical cause involving the spine and surrounding structures. Unfortunately, in most cases, a precise pathoanatomic cause cannot be reliably confirmed by physical examination or diagnostic testing. This is due to weak associations among symptoms, examination findings, and anatomic changes. In contrast to the nonspecific etiology of most mechanical causes, nonmechanical causes (such as cancer or infection) can be diagnosed with greater certainty. However, they represent a small fraction of acute low back pain in primary care.

Thus, for patients with acute low back pain, an exact etiology is identifiable in only about 15%.[6] In recent years, there has been growing interest in the potential role of nutritional factors, specifically vitamin D, in the development and management of low back pain. Vitamin D is a fat-soluble vitamin with well-established roles in bone health, calcium metabolism, and immune function.[7] Additionally, emerging evidence suggests that vitamin D may play a crucial role in musculoskeletal health beyond its traditional functions.

Vitamin D, one of the vitamins of fat-soluble vitamins, is a group of sterols which are hormones and hormone precursors because it can also be synthesized endogenously. The most important effects are on calcium metabolism, phosphorus metabolism, and bone mineralization.[8,9] In recent years, the deficiency and insufficiency of Vitamin D have been determined to be associated with many chronic diseases.[10,11]

Materials and Methods

Study Design: This was an institution based prospective observational study

Study Area: The proposed study was institution based, conducted at the Orthopaedic Department of I.P.G.M.E.R.& S.S.K.M. Hospital, Kolkata, a tertiary care center catering to people of West Bengal.

Study Population: The patients visited the orthopaedics-OPD and emergency with Low Back Pain were screened and recruited based on fulfilment of inclusion and exclusion criteria.

Study Period: From November 2022 to May 2024 (18 months).

Sample Size: 100.

Inclusion Criteria

- Patient with low back pain within age group 20-60 years.
- Patients with both the gender.

Exclusion Criteria

- Patient with renal disease, spinal listhesis, past spinal surgery and any spinal pathology.
- Trauma, infection, and endocrinologic, neurological, and rheumatologic patients diagnosed with diseases and tumors.
- Patients with fracture of vertebra in X-ray was excluded from the screening.
- Patients are not willing to participate in the study.

Statistical Analysis: Data were entered into Excel and analyzed using SPSS and GraphPad Prism. Numerical variables were summarized using means and standard deviations, while categorical variables were described with counts and percentages. Two-sample t-tests were used to compare independent groups, while paired t-tests accounted for correlations in paired data.

Chi-square tests (including Fisher's exact test for small sample sizes) were used for categorical data comparisons. P-values ≤ 0.05 were considered statistically significant.

Result

Table 1: Prevalence of Vitamin D Deficiency and Severity of vitamin D Deficiency

Vitamin D level		**Frequency**	**Percentage**
	Normal (≥20 ng/ml)	32	32
	Deficient (<20 ng/ml)	68	68
	Total	100	100
Vitamin D level	Normal (≥20 ng/ml)	32	32%
	Deficient (<20 ng/ml)	68	68%
	mild deficient (10-19.9 ng/ml)	59	59%
	moderate deficient (5-9.9 ng/ml)	7	7%
	severe deficient (<5 ng/ml)	2	2%
	Grand Total	100	100%

Table 2: Duration of Symptoms

Duration of Symptoms	**Vitamin D Level**				
	Normal (≥20 ng/ml) (n= 32)		**Deficient (<20 ng/ml) (n=68)**		**p value**
	Mean	**±SD**	**Mean**	**±SD**	
Duration of Symptoms (weeks)	21.13	±4.61	20.69	±4.47	0.655

Table 3: Dietary Habits

	Vitamin D Level					
Dietary Habits	**Normal (≥20 ng/ml) (n= 32)**		**Deficient (<20 ng/ml) (n=68)**		**Chi square**	**p value**
	Frequency	**Percentage**	**Frequency**	**Percentage**		
Vegetarian	19	59.4	38	55.9	0.108	0.742
Non veg	13	40.6	30	44.1		
Total	32	100	68	100		

Table 4: VAS Score

VAS	**Vitamin D Level**			
	Normal (≥20 ng/ml) (n= 32)		**Deficient (<20 ng/ml) (n=68)**	
	Frequency	**Percentage**	**Frequency**	**Percentage**
0 (no pain)	0	0	0	0
1-3 (mild pain)	4	12.5	3	4.4
4-6 (moderate pain)	24	75	29	42.6
7-10 (severe pain)	4	12.5	36	52.9
Total	32	100	68	100
Mean ±SD	4.88 ±1.21		5.60 ±1.46	
Statistical Inference	p value: 0.016			

Table 5: ODI Score

Vitamin D status				
ODI Score (%)	**Deficient**		**Normal**	
	Frequency	**Percentage**	**Frequency**	**Percentage**
0-20 (minimum disability)	3	4.41%	4	12.5
21-40 (moderate diabilty)	25	36.76%	25	78.13%
41-60 (severe disabilty)	36	52.94%	3	9.38%
61-80 (cripple)	4	5.88%	0	0%
81-100 (bed bound)	0	0%	0	0%
Total	68	100%	32	100%
Mean ±SD	20.29 ± 13.12		7.81 ± 5.62	
Statistical Inference	P value= 0.0002			

In the present study we found that 68 patients had a low serum vitamin D levels (<20ng/ml).The majority of the population (59%) falls into the mild deficiency category, with a range of (10-19.9 ng/ml) and the moderate deficiency category (5-9.9 ng/ml) accounts for 7% and severe deficiency category (<5 ng/ml) is accounting for only 2% of the population. In the present study we found that 68 patients had a low serum vitamin D levels (<20ng/ml).The majority of the population (59%) falls into the mild deficiency category, with a range of (10-19.9 ng/ml) and the moderate deficiency

category (5-9.9 ng/ml) accounts for 7% and severe deficiency category (<5 ng/ml) is accounting for only 2% of the population.

Mean duration of symptoms among patients with normal vitamin D and vitamin D deficiency was 21.13 ±4.61 weeks and 20.69 ±4.47 weeks respectively with no statistically significant difference (p value = 0.655). In dietary preferences, vegetarianswere 57 (57%) and 43 (43%) were nonvegs. Both the groups were comparable in terms of dietary habits (p value =0.742). In comparison of mean VAS score the groups, patients with normal vitamin D (4.88±1.21) was statistically significantly lower than patients with vitamin D deficiency (5.60±1.46) (p value =< 0.016). The results show that the mean ODI Score for the Deficient group is significantly higher than for the Normal group (20.29 vs. 7.81). The standard deviation is also higher for the Deficient group (13.12 vs. 5.62). The p-value (0.0002) is extremely small, indicating that there is a statistically significant difference between the means of the two groups.

Discussion

The present study was carried out at the Orthopaedic department of I.P.G.M.E.R. & S.S.K.M. Hospital, Kolkata. A total of 100 cases of low back pain within age group 20-60 yrs were included in the present study.

In the present study out of total 100 cases of lower back pain 68 patients had a low serum Vitamin D level (<20ng/ml). Hence the prevalence of vitamin D deficiency in the present study was 68%.

Alfaraj et al found that 83% of patients with CLBP had vitamin D deficiency, whereas this percentage was 81,7% in the study conducted by Lotfi et al, 74,3% in the study by Hwan- Kim et al, and 22,5% in eSilva et al's study.[11]

Vitamin D deficiency was detected in 84 (85.7%) out of 98 patients, while Vitamin D level was found to be normal in 14 (14.3%) of the patients as reported by Gokcek and Kaydu.[12]

Out of 1,152 patients, 599 (52%) patients had deficient vitamin D levels and 347 (30.1%) had insufficient levels. Normal levels were observed in 204 (17.7%); two patients had toxic levels of vitamin D, i.e., above 100 ng/mL as reported by Kumar M et al.[13] Y. Çalık et al in their study revealed that the categorization of patients according to vitamin D levels showed that 22,8% (n:33) had vitamin D deficiency, 42,8% (n:62) had vitamin D insufficiency, and 34,5% (n:50) had normal levels of vitamin D.[14] Kanaujia et al in their study reported that with respect to vitamin D levels, 302 (80.32%) patients were Vitamin D deficient, and in 74 (19.68%) patients, Vitamin D levels were normal.[15]

The mean age of the normal vitamin D and vitamin D deficient groups (35.38 ±9.14 years vs. 37.28 ±9.84 years), sex, BMI (24.47 ±4.38 kg/m2 vs. 26.28 ±3.56kg/m2), education level, marital status, and working conditions were compared and any statistically significant differences were not found. Similar findings were also reported by Gokcek and Kaydu, Kanaujia et al and Parikh K et al.[16,17,18]

In comparison of vitamin D levels of the groups, patients with normal vitamin D (38.08±6.40 ng/mL) was statistically significantly higher than patients with vitamin D deficiency (13.33±2.92 ng/mL) (p value =< 0.0001).

Pain intensity in the present study was assessed through VAS score. In comparison of mean VAS score the groups, patients with normal vitamin D (4.88±1.21) was statistically significantly lower than patients with vitamin D deficiency (5.60 ±1.46) (p value =< 0.016). We found a significant negative correlation between pain intensity and vitamin D level (r value= -0.446).

In our study, a significant relationship was found between vitamin D levels and pain intensity, as measured by the Visual Analog Scale (VAS) score. However, YalkınÇalık et al.[14] did not observe a significant correlation between vitamin D levels and VAS scores in their study. Despite this, they emphasized the clinical importance of assessing vitamin D levels in musculoskeletal pain patients, suggesting that deficiency may impact functional capacity. Differences in findings may stem from various factors such as study populations, methodologies, and statistical analyses.

The discrepancy in findings may be attributed to several factors, including differences in study populations, methodologies, sample sizes, and statistical analyses. Additionally, variations in geographic location, patient demographics, and healthcare practices could also contribute to differences in study outcomes.

The underlying causes of non-specific LBP are poorly understood in all age groups, and studies on risk factors associated with low back pain have reported controversial results.[19] Recently, vitamin D deficiency has been proposed to be a predisposing factor for low back pain, particularly in adults. This has attracted huge attention because vitamin D supplementation would potentially be an easy preventive intervention. Vitamin D, one of the vitamins of fat-soluble vitamins, is a group of sterols which are hormones and hormone precursors because it can also be synthesized endogenously. The most important effects are on calcium metabolism, phosphorus metabolism, and bone mineralization. In recent years, the deficiency and insufficiency of Vitamin D have been determined to be associated with many chronic diseases. In our country also, the deficiency or

insufficiency of Vitamin D has appeared more and more in the recent period, with increasing opportunities and availability of healthcare. In some sources, the deficiency of Vitamin D is now considered as a global epidemic.[20]

Correlation of deficient vitamin D levels with chronic low back pain has been studied, but definitive evidence is still lacking.[21]

There is no definitive mechanism to explain how vitamin D levels can influence LBP, there are several plausible mechanisms through which vitamin D deficiency may increase the risk of LBP. First, vitamin D deficiency may have pro inflammatory effect, which may contribute to LBP. This is supported by several studies which demonstrated that vitamin D supplementation reduces inflammatory markers. Second, vitamin D levels may modulate sensory neuron excitability and influence muscle strength, which could both be related to LBP. Finally, low vitamin D levels decrease the uptake of Ca and reduce bone mineralisation (osteomalacia or osteoporosis), which may potentially lead to back pain.[22] Several epidemiological studies have attempted to establish the association between vitamin D and LBP and reported conflicting results.

Based on the above discussion we can suggest that Vitamin D is one of the fat solvent hormones, the most critical work of which is the hemostasis of calcium. Vitamin D deficiency presents in different ways, ranging from nonspecific musculoskeletal pain to definitive clinical presentation of osteomalacia such as pain, tenderness, weakness of muscles, and even difficulty in walking. The decreased vitamin D levels influence chronic low back pain in our body; decreased levels may also lead to increased pain sensitivity and diminished neurological and muscular functional activity. Deficient levels of vitamin D raise the chances of inflammatory activity at the vertebral endplates, causing the diminished pain threshold and thus resulting in generalized pain in the muscle and bone leading to weakness.[23]

Our study also indicates a connection between Vitamin D deficiency and the severity of the pain in patients with LBP. The widespread screening of Vitamin D levels in individuals with LBP should be taken into consideration.

Conclusion

We conclude that, the present study highlights the prevalence and impact of vitamin D deficiency among patients, with the majority falling into the mild deficiency category (10–19.9 ng/ml). While there was no statistically significant difference in the mean duration of symptoms between patients with normal and deficient vitamin D levels, significant differences were observed in clinical outcomes. Patients with vitamin D deficiency exhibited higher mean VAS scores, indicating greater pain severity, and significantly higher mean ODI scores, reflecting a greater degree of functional impairment, compared to those with normal vitamin D levels. These findings emphasize the importance of evaluating and addressing vitamin D deficiency as part of the clinical assessment and management of patients, particularly in improving pain outcomes and functional status. Further studies are warranted to explore potential interventions and their long-term benefits.

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