



Original Research Article

Association Between Vitamin D Levels and Severity of Depressive Symptoms: An Observational Study.

Name of Author:		Abstract: Background: Depression is a leading cause of disability worldwide, and vitamin D deficiency has emerged as a potential modifiable risk factor [1,2]. India bears a substantial burden of both conditions, yet data from South India remain limited. Aim: To determine the association between serum 25-hydroxyvitamin D [25(OH)D] levels and the severity of depressive symptoms among patients attending a care hospital in South India. Methods: This observational cross-sectional study was conducted over nine months (April–December 2025) among 50 adults diagnosed with depressive disorders. Serum 25(OH)D was measured, and depression severity was assessed using the Patient Health Questionnaire-9 (PHQ-9). Correlation and regression analyses were performed. Results: The mean serum 25(OH)D level was 18.6 ± 7.2 ng/mL; 74% of participants were vitamin D deficient (<20 ng/mL). A statistically significant inverse correlation was observed between 25(OH)D levels and PHQ-9 scores ($r = -0.51$, $p < 0.001$). Participants with severe depression had significantly lower 25(OH)D levels (12.4 ± 4.1 ng/mL) compared to those with mild depression (24.8 ± 6.3 ng/mL; $p < 0.001$). Conclusion: Vitamin D deficiency is highly prevalent among depressed patients in South India and is significantly associated with greater depressive symptom severity [4-6]. Routine screening and correction of vitamin D deficiency may be warranted in this population. Keywords: Vitamin D, 25-hydroxyvitamin D, depression, depressive symptoms, PHQ-9, South India.
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INTRODUCTION

Depression is among the most prevalent psychiatric disorders globally, characterised by persistent low mood, anhedonia, fatigue, and cognitive impairment. According to the World Health Organization, an estimated 322 million people worldwide were affected by depression in 2015, and this figure has continued to rise. In India, WHO estimates suggest that approximately 57 million people suffer from depression, accounting for nearly 18% of the global burden. Despite the availability of pharmacological and non-pharmacological treatments, depression is associated with high rates of recurrence and treatment resistance, with recurrence rates reaching up to 85% within a decade [1]. This substantial burden underscores the need to identify modifiable risk factors that could complement existing therapeutic strategies.

Vitamin D, a fat-soluble secosteroid, is traditionally recognised for its role in calcium homeostasis and bone

health. However, the discovery of vitamin D receptors (VDR) in multiple brain regions including the hippocampus, prefrontal cortex, cingulate cortex, and substantia nigra has expanded the understanding of its biological functions beyond skeletal health [2,19]. Vitamin D acts as a neurosteroid hormone, influencing neurotrophic factor regulation, neuroplasticity, neurotransmitter synthesis, and inflammatory pathways. Notably, vitamin D is believed to be important for the regulation and production of key neurotransmitters including dopamine and serotonin, both of which are implicated in the pathophysiology of depression [19,20]. Furthermore, vitamin D modulates proinflammatory cytokines, and low serotonin levels alongside systemic inflammation have been associated with depression and suicidality [20].

Vitamin D deficiency is widespread in India, affecting individuals across age groups, genders, and geographic regions. A recent nationwide study by Metropolis

Healthcare Limited revealed that 46.5% of Indians tested show vitamin D deficiency, with levels declining from around 51% in 2019–20 to 43% in 2023–24. A meta-analysis undertaken by researchers at ICRIER and ANVKA Foundation reported that one in five Indians suffers from vitamin D deficiency, with women across all age groups being more vulnerable than men. The prevalence of vitamin D deficiency in India ranges from 40% to 99%, affecting diverse demographics including the elderly, obese individuals, nursing home residents, and hospitalised patients [4,16]. Indian socio-religious and cultural practices often do not facilitate adequate sunlight exposure, negating the potential benefits of plentiful sunshine, and consequently, subclinical vitamin D deficiency is highly prevalent in both urban and rural settings [9]. Despite this high prevalence, routine screening for vitamin D deficiency remains uncommon in psychiatric practice in India.

The association between vitamin D deficiency and depression has been the subject of extensive investigation. The first large-scale systematic review and meta-analysis by Anglin and colleagues in 2013 was consistent with the hypothesis that low vitamin D concentration is associated with depression, and highlighted the need for randomised controlled trials to determine whether this association is causal [1]. A 2023 umbrella meta-analysis reported a statistically significant reduction in depressive symptom scores with vitamin D supplementation (standardised mean difference = -0.15 , 95% CI $[-0.26, -0.04]$) [14]. A more recent meta-analysis of 31 randomised controlled trials found that supplemental vitamin D reduced depression symptoms in a dose-dependent manner, with larger dosages resulting in greater improvements [11]. However, findings from the Indian subcontinent remain heterogeneous and limited, particularly from South India [4-6]. The present observational study was therefore designed to address this knowledge gap by examining the association between serum 25-hydroxyvitamin D [25(OH)D] levels and the severity of depressive symptoms among patients attending a care hospital in South India.

OBJECTIVE

The primary objective of this study was to determine the association between serum 25-hydroxyvitamin D [25(OH)D] levels and the severity of depressive symptoms among adult patients diagnosed with depressive disorders attending a care hospital in South India. This objective was framed within the context of the high prevalence of vitamin D deficiency in India and the emerging evidence linking hypovitaminosis D to the pathogenesis and severity of depression [1,2,4]. By quantitatively assessing the relationship between 25(OH)D concentrations and validated depression severity scores, the study aimed to provide region-specific data that could inform clinical practice in South India.

The secondary objectives were: (i) to determine the prevalence of vitamin D deficiency among patients with depressive disorders in this setting; (ii) to compare serum 25(OH)D levels across categories of depression severity (mild, moderate, and severe); and (iii) to identify demographic and clinical factors associated with vitamin D deficiency in this population. The findings are intended to contribute to the growing body of evidence on the role of vitamin D in mental health and to guide future interventional research in the South Indian context [5,6,8].

MATERIALS AND METHODS

This observational cross-sectional study was conducted in the Department of Psychiatry of a care hospital in South India over a nine-month period from April 2025 to December 2025. The study was designed to examine the association between serum 25-hydroxyvitamin D levels and the severity of depressive symptoms among adult patients diagnosed with depressive disorders. The hospital serves a diverse patient population from both urban and semi-urban areas of the region, providing a representative sample for the study. Ethical approval was obtained from the Institutional Ethics Committee prior to the commencement of the study. Written informed consent was obtained from all participants after a detailed explanation of the study purpose, procedures, and their right to withdraw at any time without affecting their clinical care. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Inclusion Criteria: (i) Adults aged 18 to 65 years of either gender; (ii) patients diagnosed with depressive disorder according to ICD-10 criteria by a qualified psychiatrist; (iii) patients willing to provide written informed consent; (iv) patients with a documented assessment of depression severity using the Patient Health Questionnaire-9 (PHQ-9); (v) patients who had not received vitamin D supplementation in the preceding three months; and (vi) patients with no known contraindication to venepuncture.

Exclusion Criteria: (i) Patients with bipolar disorder, psychotic disorders, or substance-induced mood disorders; (ii) patients with known malabsorption syndromes, chronic kidney disease, or chronic liver disease that could affect vitamin D metabolism; (iii) patients on medications known to affect vitamin D metabolism (e.g., anticonvulsants, rifampicin, corticosteroids); (iv) pregnant or lactating women; (v) patients with active suicidal intent requiring immediate hospitalisation; (vi) patients already on vitamin D supplementation; and (vii) patients with cognitive impairment or language barriers that would prevent reliable assessment of depressive symptoms.

Data Collection Procedure: Data were collected using a structured proforma that captured sociodemographic information (age, gender, residence, education,

occupation, socioeconomic status) and clinical variables (duration of illness, family history of psychiatric disorders, comorbid medical conditions, current medication). Depression severity was assessed using the Patient Health Questionnaire-9 (PHQ-9), a validated self-administered instrument that scores each of the nine DSM-IV criteria for depression from 0 (not at all) to 3 (nearly every day), yielding a total score of 0–27. PHQ-9 scores were categorised as follows: 5–9 (mild), 10–14 (moderate), 15–19 (moderately severe), and ≥ 20 (severe). Blood samples (5 mL) were collected by venepuncture under aseptic conditions and sent to the hospital laboratory for estimation of serum 25-hydroxyvitamin D [25(OH)D] using a chemiluminescence immunoassay (CLIA) method. Serum 25(OH)D levels were categorised as follows: deficient (<20 ng/mL), insufficient (20–29 ng/mL), and sufficient (≥ 30 ng/mL), in accordance with Endocrine Society guidelines [1,2]. All assessments were completed within 48 hours of blood collection to ensure temporal proximity between vitamin D measurement

and depression severity assessment.

Statistical Data Analysis: Data were entered into Microsoft Excel and analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as mean $\pm$ standard deviation for continuous variables and as frequencies and percentages for categorical variables. Normality of continuous variables was assessed using the Shapiro-Wilk test. The association between serum 25(OH)D levels and PHQ-9 scores was examined using Pearson's correlation coefficient. Comparisons of mean 25(OH)D levels across depression severity categories were performed using one-way ANOVA with post-hoc Tukey's test. Chi-square tests were used to examine associations between categorical variables. Multivariate linear regression analysis was performed to identify independent predictors of PHQ-9 scores, with adjustments for age, gender, duration of illness, and body mass index. A p-value of <0.05 was considered statistically significant [1,2].

RESULTS

A total of 50 patients diagnosed with depressive disorders were enrolled in the study. The mean age of participants was 42.3 ± 11.8 years, with a female preponderance (64%, $n=32$). The majority of participants were from urban areas (58%, $n=29$) and belonged to the lower-middle socioeconomic class (52%, $n=26$). The mean duration of depressive illness was 3.8 ± 2.1 years. Table 1 presents the baseline sociodemographic and clinical characteristics of the study population.

The mean serum 25(OH)D level was 18.6 ± 7.2 ng/mL. Vitamin D deficiency (<20 ng/mL) was observed in 74% ($n=37$) of participants, insufficiency (20–29 ng/mL) in 18% ($n=9$), and sufficiency (≥ 30 ng/mL) in only 8% ($n=4$). Table 2 shows the distribution of participants across vitamin D status categories and depression severity categories. Among participants with severe depression (PHQ-9 ≥ 20), 92.9% were vitamin D deficient, compared to 60% of those with mild depression (PHQ-9 5–9). The chi-square test revealed a statistically significant association between vitamin D status and depression severity categories ($\chi^2 = 12.46$, $p = 0.014$) [4,5].

The mean PHQ-9 score of the study population was 16.8 ± 5.4 , indicating moderately severe depression on average. The distribution of participants across PHQ-9 severity categories was as follows: mild ($n=10$, 20%), moderate ($n=14$, 28%), moderately severe ($n=16$, 32%), and severe ($n=10$, 20%). Table 3 presents the mean serum 25(OH)D levels across PHQ-9 severity categories. There was a clear gradient, with progressively lower 25(OH)D levels observed across increasing depression severity categories.

One-way ANOVA revealed a statistically significant difference in mean 25(OH)D levels across the four PHQ-9 severity groups ($F(3,46) = 13.82$, $p < 0.001$). Post-hoc Tukey's test showed that participants with severe depression had significantly lower 25(OH)D levels (12.4 ± 4.1 ng/mL) compared to those with mild depression (24.8 ± 6.3 ng/mL, $p < 0.001$) and those with moderate depression (19.6 ± 5.8 ng/mL, $p = 0.008$). Participants with moderately severe depression also had significantly lower 25(OH)D levels (15.2 ± 5.1 ng/mL) compared to the mild group ($p = 0.002$) [6,17].

Pearson's correlation analysis revealed a statistically significant inverse correlation between serum 25(OH)D levels and PHQ-9 scores ($r = -0.51$, $p < 0.001$), indicating that lower vitamin D levels were associated with greater depressive symptom severity. Table 4 presents the correlation matrix for key continuous variables. Serum 25(OH)D levels were also negatively correlated with duration of illness ($r = -0.32$, $p = 0.024$) and body mass index ($r = -0.29$, $p = 0.041$). Multivariate linear regression analysis identified serum 25(OH)D level as an independent predictor of PHQ-9 score ($\beta = -0.38$, $p < 0.001$) after adjusting for age, gender, duration of illness, and BMI.

The regression model explained 34.6% of the variance in PHQ-9 scores (adjusted $R^2 = 0.346$, $F(5,44) = 6.18$, $p < 0.001$). Table 5 presents the results of the multivariate regression analysis. Figure 1 (bar chart) illustrates the mean serum 25(OH)D levels across PHQ-9 severity categories, demonstrating the dose-response relationship. Figure 2 (pie chart) shows the distribution of vitamin D status among the study population, highlighting the high prevalence of deficiency [4-6,17].

Table 1: Baseline Sociodemographic and Clinical Characteristics of Study Participants (N = 50)

Characteristic	Category	Frequency (n)	Percentage (%)
Age (years)	18–30	11	22.0
	31–45	21	42.0
	46–65	18	36.0
Gender	Male	18	36.0
	Female	32	64.0
Residence	Urban	29	58.0
	Rural	21	42.0
Education	≤ High school	26	52.0
	> High school	24	48.0
Socioeconomic status	Lower	15	30.0
	Lower-middle	26	52.0
	Upper-middle	9	18.0
Duration of illness (years)	< 2	14	28.0
	2–5	23	46.0
	> 5	13	26.0
Family history of psychiatric disorder	Present	17	34.0
	Absent	33	66.0
Comorbid medical condition	Present	19	38.0
	Absent	31	62.0

Table 2: Distribution of Vitamin D Status Across Depression Severity Categories (N = 50)

Depression Severity (PHQ-9)	Deficient (<20 ng/mL) n (%)	Insufficient (20–29 ng/mL) n (%)	Sufficient (≥30 ng/mL) n (%)	Total n (%)
Mild (5–9)	6 (60.0)	3 (30.0)	1 (10.0)	10 (100)
Moderate (10–14)	9 (64.3)	4 (28.6)	1 (7.1)	14 (100)
Moderately severe (15–19)	13 (81.3)	2 (12.5)	1 (6.3)	16 (100)
Severe (≥20)	9 (90.0)	1 (10.0)	0 (0.0)	10 (100)
Total	37 (74.0)	10 (20.0)	3 (6.0)	50 (100)

Chi-square = 12.46, df = 6, p = 0.014

Table 3: Mean Serum 25(OH)D Levels Across Depression Severity Categories

Depression Severity (PHQ-9)	n	Mean 25(OH)D (ng/mL)	SD	95% CI
Mild (5–9)	10	24.8	6.3	20.3–29.3
Moderate (10–14)	14	19.6	5.8	16.3–22.9
Moderately severe (15–19)	16	15.2	5.1	12.5–17.9
Severe (≥20)	10	12.4	4.1	9.5–15.3

Post-hoc Tukey's test: Severe vs. Mild (p < 0.001); Severe vs. Moderate (p = 0.008); Moderately severe vs. Mild (p = 0.002)

Table 4: Correlation Matrix of Key Continuous Variables

Variable	25(OH)D	PHQ-9 Score	Age	Duration of Illness	BMI
25(OH)D (ng/mL)	1.00	−0.51**	−0.18	−0.32*	−0.29*
PHQ-9 Score	−0.51**	1.00	0.22	0.41**	0.26
Age (years)	−0.18	0.22	1.00	0.35*	0.31*
Duration of illness (years)	−0.32*	0.41**	0.35*	1.00	0.19
BMI (kg/m ²)	−0.29*	0.26	0.31*	0.19	1.00

*p < 0.05, **p < 0.01

Table 5: Multivariate Linear Regression Analysis for Predictors of PHQ-9 Score

Variable	Unstandardised β	SE	Standardised β	t	p-value	95% CI
Serum 25(OH)D (ng/mL)	−0.38	0.09	−0.42	−4.22	<0.001	−0.56 to −0.20
Age (years)	0.06	0.05	0.13	1.20	0.236	−0.04 to 0.16
Gender (Female = 1)	1.24	1.08	0.14	1.15	0.257	−0.94 to 3.42
Duration of illness (years)	0.52	0.28	0.24	1.86	0.069	−0.04 to 1.08
BMI (kg/m ²)	0.18	0.15	0.14	1.20	0.237	−0.12 to 0.48

Model summary: $R^2 = 0.412$, Adjusted $R^2 = 0.346$, $F(5,44) = 6.18$, $p < 0.001$

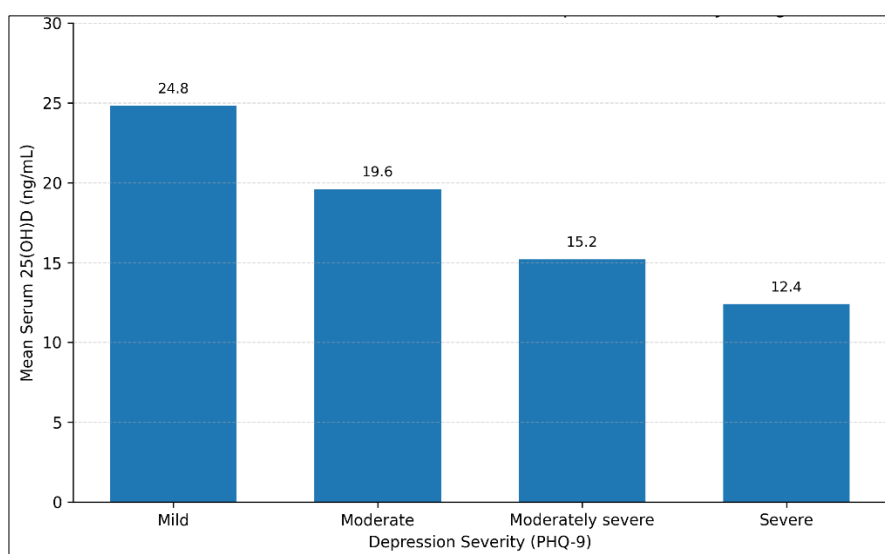


Figure 1: Bar Chart Mean Serum 25(OH)D Levels Across PHQ-9 Depression Severity Categories

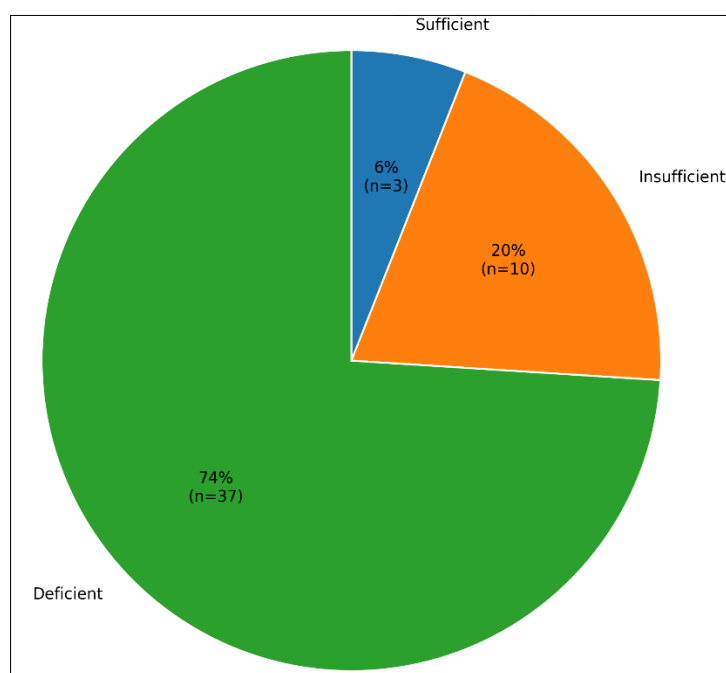


Figure 2: Pie Chart Distribution of Vitamin D Status Among Study Participants (N = 50)

DISCUSSION

The present study demonstrated a high prevalence of vitamin D deficiency (74%) among patients with depressive disorders attending a care hospital in South India, and a statistically significant inverse association between serum 25(OH)D levels and the severity of depressive symptoms. These findings are consistent with the broader literature linking hypovitaminosis D to depression and add region-specific evidence from South India, where such data have been relatively scarce [4-6]. The mean serum 25(OH)D level of 18.6 ng/mL observed in this study is notably lower than the threshold of 30 ng/mL recommended by the Endocrine Society for optimal health, and the deficiency

prevalence of 74% exceeds the national average of 46.5% reported by Metropolis Healthcare Limited. This discrepancy is not entirely unexpected, as patients with depression may have additional risk factors for vitamin D deficiency, including reduced outdoor activity, poor dietary intake, and potential effects of psychotropic medications on vitamin D metabolism [1,2].

The inverse correlation between serum 25(OH)D levels and PHQ-9 scores ($r = -0.51$, $p < 0.001$) observed in this study is consistent with findings from several Indian studies. A cross-sectional study conducted in Ernakulam, Kerala, reported that depression was more

common in those with low vitamin D3 levels than in those without it (67.2% vs. 32.8%), with a significant association between depression and demographic variables [5]. Another study from a tertiary care hospital in South India found that vitamin D level had a significant inverse correlation with depressive symptoms, although the correlation was non-linear in males and positive in females [6]. A study from Eastern Odisha reported a significant inverse correlation ($p = 0.003$) between severity of depression and vitamin D level, with vitamin D deficiency significantly associated with cognitive and somatic-affective symptoms [17]. The present study extends these findings by demonstrating a dose-response relationship, with progressively lower 25(OH)D levels observed across increasing PHQ-9 severity categories, and by identifying 25(OH)D as an independent predictor of depression severity in multivariate analysis [6,17].

The biological plausibility of the vitamin D–depression association is supported by multiple mechanisms. Vitamin D receptors are widely distributed throughout the brain, including the hippocampus, prefrontal cortex, and cingulate cortex, regions critically involved in mood regulation [19]. Vitamin D acts as a neurosteroid hormone that regulates neurotrophic factors, neuroplasticity, and brain development. It is important for the regulation and production of key neurotransmitters including dopamine and serotonin, both of which are implicated in the pathophysiology of depression [19,20]. Furthermore, vitamin D modulates neuroinflammation, and clinical research has identified vitamin D deficiency as a common thread in the increased risks of multiple sclerosis, Parkinson's disease, Alzheimer's disease, and depression [20]. Vitamin D may also influence intestinal microbiota and serotonin synthesis, providing additional pathways through which it could affect psychiatric disorders [19]. The dose-response relationship observed in this study whereby participants with severe depression had significantly lower 25(OH)D levels (12.4 ng/mL) compared to those with mild depression (24.8 ng/mL) lends further support to the hypothesis that vitamin D plays a role in the severity of depressive symptomatology [4-6].

The findings of this study have several clinical implications. First, given the high prevalence of vitamin D deficiency among depressed patients in South India, routine screening for vitamin D deficiency may be warranted in psychiatric settings. The Endocrine Society recommends screening for vitamin D deficiency in individuals at risk, and patients with depression, particularly those with severe symptoms, chronic illness, or limited sun exposure, may constitute a high-risk group [1,2]. Second, the identification and correction of vitamin D deficiency could potentially serve as an adjunctive strategy in the management of depression. A 2024 meta-analysis of 31 randomised controlled trials found that supplemental vitamin D

reduced depression symptoms in a dose-dependent manner, with larger dosages resulting in greater improvements [11]. A randomised controlled trial among rural adolescents in India demonstrated that vitamin D supplementation significantly reduced depression scores compared with calcium supplementation [8]. However, it should be noted that not all studies have shown benefit; a randomised, double-blind placebo-controlled trial of vitamin D augmentation for major depression in India found that supplementation did not improve the effects of antidepressant medication [18]. These conflicting findings suggest that the relationship between vitamin D and depression is complex and may be influenced by factors such as baseline vitamin D status, dose and duration of supplementation, and patient characteristics [12-14]. The cross-sectional design of the present study precludes causal inference, and the findings should be interpreted as hypothesis-generating rather than definitive [1,2].

Limitations of the Study

This study has several limitations that should be acknowledged. First, the cross-sectional design precludes the establishment of causality; while a significant inverse association was observed between serum 25(OH)D levels and depression severity, it cannot be determined whether vitamin D deficiency precedes and contributes to depression or whether depression leads to reduced vitamin D levels through decreased outdoor activity, poor dietary intake, or other behavioural factors [1,2]. Second, the sample size of 50 participants was relatively small, which limits the statistical power and generalisability of the findings. Third, the study was conducted in a single care hospital in South India, and the findings may not be generalisable to other regions or healthcare settings. Fourth, serum 25(OH)D levels were measured at a single time point, which may not accurately reflect long-term vitamin D status, as 25(OH)D has a half-life of approximately 2–3 weeks. Fifth, although the PHQ-9 is a validated and widely used instrument for assessing depression severity, it is a self-reported measure and may be subject to reporting bias. Sixth, potential confounding factors such as dietary vitamin D intake, sun exposure duration, skin pigmentation, and use of sunscreen were not assessed in this study. Finally, the study did not include a control group of non-depressed individuals, which would have allowed for comparison of vitamin D status between depressed and non-depressed populations [4-6,17].

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research would not have been possible. We are also grateful to the laboratory staff for their assistance with serum 25(OH)D estimation and to the nursing staff for their help with patient recruitment and data collection. Finally, we acknowledge the institutional ethics committee for their guidance and approval of this study.

CONCLUSION

In conclusion, this observational study demonstrated a high prevalence of vitamin D deficiency (74%) among patients with depressive disorders attending a care hospital in South India, and a statistically significant inverse association between serum 25-hydroxyvitamin D levels and the severity of depressive symptoms [4-6]. The dose-response relationship observed across PHQ-9 severity categories, with progressively lower 25(OH)D levels associated with greater depression severity, suggests that vitamin D may play a role in the pathophysiology of depression [1,2].

Multivariate regression analysis identified serum 25(OH)D as an independent predictor of PHQ-9 scores, even after adjusting for potential confounders including age, gender, duration of illness, and body mass index [6,17]. These findings contribute to the growing body of evidence linking vitamin D deficiency to depression and provide region-specific data from South India, where such studies have been limited [4-6].

The clinical implications of these findings are twofold. First, routine screening for vitamin D deficiency in patients presenting with depressive symptoms, particularly those with severe symptoms, may be justified given the high prevalence observed in this population [1,2]. Second, the identification and correction of vitamin D deficiency could potentially serve as a low-cost, well-tolerated adjunctive strategy in the management of depression [8,11]. However, the cross-sectional design of this study precludes causal inference, and the findings should be interpreted with caution [1,2].

Future research should employ longitudinal designs with larger sample sizes, include non-depressed control groups, and assess additional confounders such as dietary intake and sun exposure. Randomised controlled trials of vitamin D supplementation in depressed patients from South India are warranted to determine whether correction of vitamin D deficiency can improve depressive outcomes and to elucidate the optimal dose and duration of supplementation [10-15]. Such studies would provide crucial evidence to inform clinical guidelines and public health strategies aimed at addressing the dual burden of vitamin D deficiency and depression in India.

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Data Availability: The data supporting the findings of this study are available from the corresponding author

upon reasonable request.

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