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Original Research Article

A Study on the Association between Serum Vitamin D Levels and Acute Stroke

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Abstract

Background: Vitamin D has emerged as an important neurosteroid with anti-inflammatory, vasoprotective, and neuroprotective effects. Increasing evidence suggests that vitamin D deficiency may influence both the risk and outcome of acute stroke, but data from Indian populations remain limited.

Aim: To evaluate the association between serum vitamin D levels and stroke severity, functional outcome, and vascular risk factors in patients with acute stroke.

Methods: This prospective observational study included 132 patients with acute stroke (ischemic or haemorrhagic) admitted within 72 hours of symptom onset. Serum 25-hydroxyvitamin D [25(OH) D] levels were measured within 24 hours of admission and categorized as deficient (<20 ng/mL), insufficient (20–30 ng/mL), or sufficient (>30 ng/mL). Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS), and functional outcome at discharge was evaluated using the Modified Rankin Scale (mRS). Associations between vitamin D status, stroke severity, outcomes, and vascular risk factors were analysed using ANOVA and Chi-square tests.

Results: Among the 132 patients, 57.6% were vitamin D deficient, 28.8% insufficient, and only 13.6% had sufficient levels. Mean NIHSS scores were significantly higher in vitamin D–deficient patients (16.8 ± 4.3) compared to insufficient (12.4 ± 3.6) and sufficient groups (8.9 ± 2.8) ($p < 0.001$). Poor functional outcome (mRS >2) was observed in 76.3% of deficient patients compared with 52.6% of insufficient and 27.8% of sufficient patients ($p < 0.001$). Vitamin D deficiency was also significantly associated with higher prevalence of hypertension, diabetes mellitus, and dyslipidaemia ($p < 0.05$).

Conclusion: Vitamin D deficiency is highly prevalent among patients with acute stroke and is strongly associated with greater stroke severity and poorer functional outcomes. Low serum 25(OH) D levels are also linked to a higher burden of vascular risk factors. Routine screening and correction of vitamin D deficiency may represent a simple and cost-effective strategy to improve stroke prognosis.

Keywords: Vitamin D, Acute Stroke, NIHSS, Modified Rankin Scale, Stroke Outcome, Hypovitaminosis D.

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Introduction

Stroke is a major global health problem and remains one of the leading causes of mortality and long-term disability worldwide. It is characterized by a sudden interruption of blood supply to the brain, resulting in neuronal injury and loss of neurological function. Ischemic stroke, caused by vascular occlusion, accounts for approximately 80–85% of all stroke cases, while haemorrhagic stroke occurs due to rupture of cerebral blood vessels. Despite advances in acute management such as thrombolysis and mechanical thrombectomy, the burden of stroke-related disability continues to rise, highlighting the need to identify modifiable risk factors and novel therapeutic targets. Vitamin D, traditionally known for its role in calcium homeostasis and bone metabolism, has emerged as a multifunctional neurosteroid hormone with important effects on the cardiovascular and nervous systems [1, 6, 7, and 10[. Vitamin D receptors are widely distributed in the brain, vascular endothelium, and immune cells, suggesting a broader physiological role beyond skeletal health

[11, 12]. Increasing evidence indicates that vitamin D plays a critical role in regulating inflammation, endothelial function, blood pressure, and glucose metabolism, all of which are closely involved in stroke pathophysiology [6, 7, 9, 14]. Vitamin D deficiency is highly prevalent worldwide, particularly among the elderly, individuals with limited sunlight exposure, and patients with chronic illnesses [1, 6]. Several epidemiological studies have reported a strong association between low serum 25-hydroxyvitamin D levels and an increased risk of cardiovascular diseases, including hypertension, coronary artery disease, and cerebrovascular disorders [6, 7, 10]. In the context of stroke, vitamin D deficiency has been linked not only to higher incidence but also to greater stroke severity and worse functional outcomes [3–5, 13, 15]. The potential neuroprotective properties of vitamin D are of particular interest. Experimental and clinical studies suggest that vitamin D can reduce oxidative stress, suppress pro-inflammatory cytokines, regulate calcium homeostasis, and inhibit neuronal apoptosis following ischemic injury [9, 11, 12]. These mechanisms indicate that vitamin D may influence both the development of stroke and the extent of brain damage after its occurrence.

Aim and Objectives: To evaluate the relationship between serum vitamin D levels and the incidence, severity, and outcomes of acute stroke.

Materials and Methods

Study Design and Setting: This was a hospital-based prospective observational study conducted over a period of 8 months at the Tertiary care hospitals multicentre, Andhra Pradesh, Telangana India.

The study aimed to evaluate the relationship between serum vitamin D levels and acute stroke.

Study Population and Sample Size: A total of 132 patients diagnosed with acute stroke were enrolled in the study. Patients presenting to the emergency department or admitted to the neurology and medical wards during the study period were screened for eligibility. Sample size was determined based on hospital admission rates and feasibility during the study period.

Inclusion Criteria

Patients were included in the study if they met the following criteria:

- Age ≥18 years
- Diagnosis of acute stroke (ischemic or haemorrhagic) confirmed by CT or MRI brain
- Presentation within 72 hours of symptom onset
- Willingness to participate and provide informed consent (by patient or legal attendant)

Exclusion Criteria

- Patients were excluded if they had:
- History of chronic kidney disease, chronic liver disease, or malabsorption syndromes
- Known parathyroid disorders or metabolic bone disease
- Prior vitamin D supplementation in the last 3 months
- Active malignancy or chronic inflammatory diseases
- Severe infection or sepsis at admission
- Recurrent stroke or prior history of cerebrovascular accident
- Pregnant or lactating women

Data Collection: Demographic details, clinical history, and vascular risk factors such as hypertension, diabetes mellitus, smoking, and dyslipidaemia were recorded. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) at admission. Functional outcome was evaluated using the Modified Rankin Scale (mRS).

Laboratory Analysis: Venous blood samples were collected within 24 hours of admission. Serum 25-hydroxyvitamin D [25(OH) D] levels were measured using a chemiluminescence immunoassay. Vitamin D status was classified as:

- **Deficient**: <20 ng/mL
- **Insufficient**: 20–30 ng/mL
- **Sufficient**: >30 ng/mL

Statistical Analysis: Data were analysed using standard statistical software. Continuous variables were expressed as mean ± standard deviation and categorical variables as percentages. The association between vitamin D levels and stroke severity and outcomes was analysed using appropriate statistical tests. Descriptive statistics (frequency and percentage) were used. One-way Analysis of Variance (ANOVA) was used to compare mean NIHSS scores across the three vitamin D groups. Chi-square (χ^2) test was used to compare categorical outcomes (good vs poor outcome) across vitamin D groups. Chi-square (χ^2) test was applied to compare proportions between the two groups. A p-value <0.05 was considered statistically significant.

Results

Table 1: Demographic Characteristics of Study Population (n = 132)

Variable	Number (n)	Percentage (%)
Age (years)		
<50	**28**	**21.2**
50-65	**57**	**43.2**
>65	**47**	**35.6**
Gender		
Male	**78**	**59.1**
Female	**54**	**40.9**

The age and gender distribution of the 132 patients included in the study. The majority of patients were in the 50–65 years age group (43.2%), followed by those older than 65 years (35.6%). Males constituted 59.1% of the study population.

Table 2: Distribution of Stroke Types

Stroke Type	Number	Percentage
Ischemic Stroke	**104**	**78.8**
Haemorrhagic Stroke	**28**	**21.2**
Total	**132**	**100**

Ischemic stroke was the predominant type (78.8%).

Table 3: Vitamin D Status in Acute Stroke Patients

Vitamin D Level	25(OH)D (ng/mL)	Number	Percentage
Deficient	**<20**	**76**	**57.6**
Insufficient	**20-30**	**38**	**28.8**
Sufficient	**>30**	**18**	**13.6**

More than half of the patients (57.6%) were vitamin D deficient (<20 ng/mL), while only 13.6% had sufficient levels. This highlights the high prevalence of hypovitaminosis D among acute stroke patients.

Table 4: Association between Vitamin D Levels and Stroke Severity (NIHSS)

Vitamin D Status	Number	Mean NIHSS ± SD
Deficient (<20)	**76**	**16.8 ± 4.3**
Insufficient (20–30)	**38**	**12.4 ± 3.6**
Sufficient (>30)	**18**	**8.9 ± 2.8**

Patients with vitamin D deficiency had the highest mean NIHSS score (16.8 ± 4.3), indicating more severe neurological impairment, while those with sufficient vitamin D had the lowest scores (8.9 ± 2.8).

Table 5: Vitamin D Status and Functional Outcome (Modified Rankin Scale at Discharge)

Vitamin D Status	Good Outcome (mRS ≤2	Poor Outcome (mRS >2	Total
Deficient	**18 (23.7%)**	**58 (76.3%)**	**76**
Insufficient	**18 (47.4%)**	**20 (52.6%)**	**38**
Sufficient	**13 (72.2%)**	**5 (27.8%)**	**18**

P-value <0.001

A significantly higher proportion of vitamin D–deficient patients (76.3%) had poor outcomes (mRS >2), whereas most patients with sufficient vitamin D (72.2%) had good outcomes. The association was highly statistically significant ($p < 0.001$), indicating that vitamin D status strongly influences early recovery after stroke.

Table 6: Association between Vitamin D Deficiency and Vascular Risk Factors

Risk Factor	Deficient (n=76)	Non-deficient (n=56)	p-value
Hypertension	**58 (76.3%)**	**31 (55.4%)**	**0.01**
Diabetes Mellitus	**42 (55.3%)**	**20 (35.7%)**	**0.03**
Dyslipidaemia	**39 (51.3%)**	**19 (33.9%)**	**0.04**
Smoking	**28 (36.8%)**	**14 (25.0%)**	**0.12**

Hypertension, diabetes, and dyslipidaemia were significantly more common in vitamin D–deficient patients ($p < 0.05$), whereas smoking did not show a statistically significant association.

Discussion

This hospital-based prospective observational study examined the relationship between serum vitamin

D levels and the clinical severity and short-term functional outcomes of acute stroke in 132 patients. The findings demonstrate a high prevalence of vitamin D deficiency among acute stroke patients and reveal a strong association between low vitamin D levels, increased stroke severity, and poorer functional outcomes. These results reinforce the growing body of evidence that vitamin D plays a significant role in cerebrovascular disease and neurological recovery [3–5, 13, 15].

In the present study, 57.6% of patients were vitamin D deficient, while only 13.6% had sufficient levels. This prevalence is similar to earlier studies from India and other countries, which report widespread vitamin D deficiency in both the general population and patients with cardiovascular and cerebrovascular disorders [1, 6, 7]. Factors such as limited sunlight exposure, poor dietary intake, aging, sedentary lifestyle, and chronic illnesses contribute to low vitamin D levels [1, 6]. Stroke patients, who are often elderly and have multiple comorbidities, are particularly vulnerable to vitamin D deficiency.

A key finding of this study is the significant inverse relationship between serum vitamin D levels and stroke severity as measured by the NIHSS. Patients with vitamin D deficiency had much higher NIHSS scores at admission than those with sufficient levels, indicating more severe neurological impairment. This observation is consistent with previous studies. Daubail et al. reported that low vitamin D levels were associated with larger infarct volumes and more severe neurological deficits in ischemic stroke patients (3). Similarly, Turetsky et al. found that vitamin D-deficient patients had worse functional and neurological status at stroke onset [4]. Tu et al. also showed that low vitamin D predicted poorer short-term outcomes in acute ischemic stroke [15].

The biological mechanisms underlying this association are multifactorial. Vitamin D has anti-inflammatory and immunomodulatory properties that reduce the release of pro-inflammatory cytokines such as interleukin-6 and tumour necrosis factor-α, which contribute to secondary neuronal injury after stroke (9, 14]. Vitamin D also reduces oxidative stress and stabilizes the blood–brain barrier, thereby limiting cerebral oedema and neuronal damage [11, 12]. In addition, vitamin D regulates intracellular calcium levels, preventing excitotoxicity neuronal death that occurs during ischemia [11, 12]. Functional outcome at discharge, measured by the Modified Rankin Scale, was also significantly influenced by vitamin D status. In this study, more than 76% of vitamin D-deficient patients had poor functional outcomes, whereas over 70% of patients with sufficient vitamin D achieved good outcomes. Similar associations have been reported by Daubail et al. and Tu et al., who showed that vitamin D predicts functional recovery and prognosis after stroke [3, 15]. Vitamin D promotes neuroplasticity and neuronal repair by stimulating neurotrophic factors such as nerve growth factor and glial cell-derived neurotrophic factor [11,12], and also improves muscle strength and balance, which are critical for rehabilitation.

The relationship between vitamin D deficiency and vascular risk factors observed in this study further supports its role in stroke pathogenesis. Patients with vitamin D deficiency had significantly higher rates of hypertension, diabetes mellitus, and dyslipidaemia. Vitamin D suppresses renin production, improves insulin sensitivity, and reduces vascular inflammation, thereby protecting against these risk factors [6, 7, 10, 14]. Zhou et al. and Annweiler et al. also demonstrated that low vitamin D status is associated with an increased risk of ischemic stroke [5, 13]. Several studies suggest that vitamin D deficiency is more strongly associated with ischemic stroke than haemorrhagic stroke due to its effects on atherosclerosis, endothelial dysfunction, platelet activation, and thrombogenesis [5, 7, 10, 14].

Conclusion

Vitamin D deficiency is highly prevalent among patients with acute stroke and is strongly associated with increased stroke severity, poorer functional outcomes, and a higher burden of vascular risk factors. Lower serum 25(OH) D levels were linked to higher NIHSS scores and worse Modified Rankin Scale outcomes, indicating more severe neurological impairment and reduced recovery. These findings suggest that vitamin D may play a significant role in both the pathogenesis and prognosis of acute stroke.

Routine screening for vitamin D deficiency in patients at risk of stroke and in those presenting with acute stroke may be a simple, cost-effective strategy to improve clinical outcomes. Early identification and correction of vitamin D deficiency through supplementation and lifestyle measures could potentially reduce stroke severity and enhance neurological recovery. Large-scale randomized controlled trials are needed to confirm whether vitamin D supplementation can improve stroke prevention and rehabilitation outcomes.

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