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Vitamin D Deficiency And Neurofilament Light Chain (Nfl) As Predictors Of Type 2 Diabetes-Associated Distal Symmetric Polyneuropathy: A Prospective Study

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Abstract: Aim: The main aim of this study to evaluate the association of serum vitamin D levels and neurofilament light chain (NFL) concentrations with the presence and severity of Distal symmetric polyneuropathy in patients with type 2 diabetes.
Background:
Distal symmetric polyneuropathy (DSPN) is a common and debilitating complication of type 2 diabetes mellitus (T2DM). Early identification of biomarkers that predict DSPN progression is crucial. This study aimed to evaluate serum 25-hydroxyvitamin D [25(OH)D] and neurofilament light chain (NFL) as predictors of DSPN development and progression over a 3-month follow-up period.
Methods:
A prospective cohort study was conducted on 430 T2DM patients without other causes of neuropathy. Baseline assessments included clinical examination, serum 25(OH)D, NFL levels, and nerve conduction studies. DSPN was diagnosed using the Michigan Neuropathy Screening Instrument (MNSI) and electrophysiological criteria. Participants were followed for 3 months, and changes in DSPN status, NFL, and vitamin D levels were recorded.
Results:
At baseline, 38.1% of participants had DSPN. After 3 months, DSPN prevalence increased to 44.9%. Among vitamin D–deficient individuals, 12.7% developed new DSPN compared to 3.9% in those with sufficient vitamin D ($p < 0.001$). NFL levels significantly increased in participants with new-onset DSPN ($p < 0.001$). Multivariate regression showed vitamin D deficiency (OR: 2.38; 95% CI: 1.31–4.33; $p = 0.004$) and elevated NFL (>40 pg/mL) (OR: 3.14; 95% CI: 1.76–5.59; $p < 0.001$) were independent predictors of DSPN progression.
Conclusion:
Vitamin D deficiency and elevated NFL levels are significant predictors of DSPN development in T2DM patients over a short-term follow-up. Monitoring these biomarkers may aid in early detection and risk stratification of diabetic neuropathy.

Keywords:
Type 2 diabetes mellitus, distal symmetric polyneuropathy, vitamin D deficiency, neurofilament light chain, diabetic neuropathy

1. Introduction

The most common long-term complication of type II diabetes mellitus is peripheral neuropathy (DPN). In India, the prevalence of diabetic peripheral neuropathy (DPN) ranges from 18.8% to 61.9% among individuals with diabetes [1]. The prevalence increases with adjacence of age, and duration of diabetes. Aggravating factors like poor glycemic control, hypertension, and other diabetic complications like nephropathy and retinopathy can also contribute to the development and severity of DPN [2]. Many biomarkers including interleukin different subtypes, tumor necrosis factor alpha (TNF-a), neurofilament light chain (NFL), and transforming growth factor beta1 (TGF-b1) changes homeostasis of nerves and it's supporting glia cells [3].

The neurofilament light chain (NFL) is a cytoskeletal component of mature neurons that provides structural stability and determines axonal diameter. NFL is more abundant in large myelinated axons facilitating a faster conduction velocity [4]. Upon axonal injury, NFL is released from axons into the circulation. The efficacy of serum NFL as a biomarker of neuroaxonal damage emerged initially in multiple sclerosis, where it serves for prognosis and treatment monitoring [5]. Currently, there is growing scientific evidence associating serum NFL with other neurodegenerative diseases and peripheral neuropathies in humans and animals [6,7]. However, evidence of a potential association between NFL and DSPN is scarce. One recent preliminary study indicated inverse correlations between serum NFL and some nerve function measures in individuals with type 2 diabetes who had a known diabetes duration of 3 years or less. An earlier study reported higher blood NFL (also known as NEFL) mRNA levels in people with impaired glucose tolerance and/or impaired fasting glucose and peripheral neuropathy than those without [8]. However, whether serum NFL is associated with prevalent DSPN in individuals with type 1 or type 2 diabetes of a very short known duration remains unknown [9].

Emerging evidence suggests that vitamin D deficiency may play a contributory role in the development of diabetic neuropathy through its effects on nerve health, inflammation, and oxidative stress modulation [10]. Vitamin D receptors are expressed in neurons and glial cells, and its active form, calcitriol, has neuroprotective and anti-inflammatory properties [11]. Several cross-sectional and cohort studies have reported that low serum 25-hydroxyvitamin D [25(OH)D] levels are associated with increased risk and severity of DSPN in individuals with T2DM [12-15].

Given the neuroprotective potential of vitamin D and the objective quantifiability of NFL as a marker of axonal injury, this study aims to investigate the association of vitamin D deficiency and serum NFL levels with the presence and severity of distal symmetric polyneuropathy in type 2 diabetic patients. Identifying these biomarkers may offer a non-invasive strategy for early diagnosis, risk stratification, and possibly therapeutic monitoring of DSPN.

Method:

Study Design and Setting

This study was a prospective, observational cohort study conducted at the Saraswati Medical college and Hospital from October 2022 and December 2024. The protocol was approved by institutional ethical committee of Saraswati medical college, Unnao, UP. The objective was to evaluate whether serum vitamin D levels and neurofilament light chain (NFL) concentrations can predict the development or progression of distal symmetric polyneuropathy (DSPN) in patients with type 2 diabetes mellitus (T2DM) over a 3-month follow-up period.

Study Population:

The sample size calculated based on the prevalence of Distal Symmetric Polyneuropathy (DSPN) in diabetic patients from previous studies[1], DSPN prevalence (P) = 50%; Desired precision (d) = 5% and Confidence level = 95% (Z = 1.96)

Sample size formula (n) = $\frac{Z^2.\ P(1-p)}{d2}$

The calculated sample size is 385 and 10 percentage drop out considered during the study, enrolled total 430 to

remove bias of study.

The study included adults aged 35–75 years, diagnosed with type 2 diabetes mellitus (T2DM) for at least 5 years, HbA1c ≥ 6.5%, Willingness to provide informed consent and comply with follow-up visits. However, the study excluded patients already with history of other neuropathies (e.g., B12 deficiency, alcoholism, uremia, hypothyroidism, HIV), recent use of vitamin D supplements (within past 3 months), chronic kidney disease (stage 4 or above), active infections, autoimmune disorders, or malignancies and patients on neurotoxic drugs

Data Collection:

At baseline: Clinical assessment, serum 25(OH)D, NFL, HbA1c, nerve conduction study, neuropathy symptom scores

Follow-up (Month 3): NFL and 25(OH)D, nerve conduction study, neuropathy scoring

1. Clinical and Neuropathy assessment

Neuropathy was assessed using Michigan neuropathy screening instrument (MNSI)

Neuropathy Disability Score (NDS).

Nerve conduction studies (NCS) of unilateral lower limb nerves (peroneal and tibial motor nerves and the sural sensory nerve) were conducted while maintaining the room temperature 21-24ºC. Patients were classified into three subgroups no DSPN, early/subclinical DSPN, and established DSPN.

2. Laboratory investigations

i) The serum 25-hydroxyvitamin D [25(OH)D] was measured using chemiluminescent immunoassay (DxC 700 AU, Backman Coulter). The patients were divided into three subgroups deficient: < 30 nmol/L, insufficient: 30–50 nmol/L, and sufficient: > 50 nmol/L.

ii) The neurofilament Light Chain (NFL) was measured using Access 2 immunoassay Analysers (Backman Coulter). Normal range were considered for neurofilament light chain (NfL pg/mL) below 45 years old – 10 pg/ml and above 45 years old 15 pg/ml.

Outcome Measures:

a) Primary Outcome:

The incidence or progression of DSPN over 3 months, based on clinical and electrophysiological criteria

b) Secondary Outcomes:

The correlation of baseline and follow-up vitamin D and NFL levels with DSPN severity scores

Statistical Analysis

The data were analysed using SPSS 20 version. The descriptive statistics for continuous data were reported as Mean ± SD and frequency (%) for categorical data. The comparison was done using Paired t-test for normally distributed data between-group comparisons. The correlation analysis was done Pearson correlation between NFL, vitamin D, and neuropathy scores. The regression analysis was done using multivariate logistic regression to determine predictors of DSPN progression. The significance level was set at $p < 0.05$ (two-sided).

Results:

Baseline Characteristics

A total of 430 patients with type 2 diabetes mellitus (T2DM) were enrolled. The mean age was 58.4 ± 9.6 years, with 52% males and 48% females. The mean diabetes duration was 9.1 ± 3.8 years. At baseline, 52.3% of participants had vitamin D deficiency (<30 nmol/L), while 47.7% were classified as vitamin D sufficient (≥30 nmol/L).

2. Prevalence and Progression of DSPN

At baseline, 38.1% (n = 164) of participants had clinical or subclinical distal symmetric polyneuropathy (DSPN) based on MNSI scores and nerve conduction studies.

At 3-month follow-up the DSPN prevalence increased to 44.9% (n = 193).
Among those vitamin D deficient at baseline, 28 new cases (12.7%) developed DSPN.
Among vitamin D sufficient participants, only 8 new cases (3.9%) developed DSPN ($p < 0.001$).
Based on baseline serum levels, 56 (49.1%) patients were vitamin D deficient, 38 (33.3%) had insufficient levels, and 20 (17.5%) had sufficient levels. DSPN progression at 3 months was observed in 62.5% of patients in the vitamin D deficient group compared to 23.7% and 10.0% in the insufficient and sufficient groups, respectively ($p < 0.001$).
Patients who showed DSPN progression had significantly higher baseline serum NFL levels (mean 36.8 ± 6.5 pg/mL) compared to those without progression (24.3 ± 5.2 pg/mL; $p < 0.001$). NFL levels increased over the 3-month period in the progression group but remained stable in the non-progression group.
Serum vitamin D levels showed a significant inverse correlation with neuropathy severity scores ($r = –0.47$, $p < 0.01$). NFL levels were positively correlated with both neuropathy scores ($r = 0.52$, $p < 0.001$) and vibration perception thresholds ($r = 0.39$, $p = 0.002$).
On multivariate logistic regression analysis, vitamin D deficiency (OR 2.9; 95% CI: 1.6–5.4) and elevated NFL levels (>30 pg/mL) (OR 4.2; 95% CI: 2.1–8.1) were independently associated with DSPN progression. The combined model had an AUC of 0.82 (95% CI: 0.74–0.89) for predicting neuropathy progression.
3. Changes in Biomarkers over time:
Vitamin D–deficient group showed a non-significant mean increase in serum 25(OH)D from 25.1 ± 4.3 to 26.4 ± 4.6 nmol/L ($p = 0.08$). There was no significant seasonal variation was observed.
NFL Levels
Participants who developed new DSPN showed a significant increase in NFL levels from 33.2 ± 9.1 to 41.6 ± 10.4 pg/mL ($p < 0.001$). NFL levels remained stable in participants without DSPN progression ($p = 0.24$).
4. Correlation Analysis
Baseline 25(OH)D levels were inversely correlated with MNSI scores at follow-up ($r = –0.42$, $p < 0.001$). NFL levels were positively correlated with both baseline and follow-up DSPN severity scores ($r = 0.51$, $p < 0.001$).

Discussion

In this prospective cohort study, we evaluated the predictive value of serum 25-hydroxyvitamin D [25(OH)D] and neurofilament light chain (NFL) levels in the development and progression of distal symmetric polyneuropathy (DSPN) among patients with type 2 diabetes mellitus (T2DM). Our results demonstrate both vitamin D deficiency and elevated NFL levels at baseline were significantly associated with increased risk of incident or worsening DSPN, showing their potential as early biomarkers for diabetic neuropathy.
Our study findings showed that DSPN prevalence increased from 38.1% at baseline to 44.9% after 3 months, indicating that short-term progression of neuropathy is not uncommon among high-risk T2DM populations. Notably, participants with baseline vitamin D deficiency had a threefold higher incidence of new DSPN cases compared to those with sufficient vitamin D levels (12.7% vs. 3.9%, $p < 0.001$) (figure-1). This supports previous cross-sectional studies that link hypovitaminosis D with the presence of diabetic neuropathy, possibly through mechanisms involving chronic inflammation, oxidative stress, impaired neurotrophic signaling, and altered calcium homeostasis [9,12].
This study demonstrates that both vitamin D deficiency and elevated neurofilament light chain (NFL) levels are associated with worsening neuropathic symptoms and signs over a 3-month period, as measured by the

Michigan Neuropathy Screening Instrument (MNSI) [14].

Participants with vitamin D deficiency (< 30 nmol/L) had consistently higher MNSI questionnaire and examination scores at baseline and after 3 months compared to those with sufficient vitamin D levels (table-2). More notably, their scores increased more sharply over the 3-month period, indicating a faster progression of neuropathic symptoms. This finding aligns with growing evidence that vitamin D plays a neuroprotective role by modulating inflammation, oxidative stress, and nerve regeneration. Deficiency in vitamin D may thus exacerbate the metabolic and vascular damage seen in diabetic peripheral nerves.

Similarly, individuals with elevated NFL levels (≥40 pg/mL), a marker of axon-specific neuronal injury, also showed higher MNSI scores at both time points and greater score progression than those with lower NFL levels. This suggests that rising NFL levels may serve as an early biomarker for active neurodegeneration in patients at risk of or experiencing DSPN. The strong association between high NFL levels and worsening MNSI scores reinforces its potential utility in tracking nerve damage progression non-invasively [17,18].

Importantly, the parallel trends observed in both vitamin D–deficient and NFL-elevated groups highlight the complementary role of these two markers. While vitamin D deficiency may act as a modifiable risk factor, NFL levels may serve as a real-time indicator of neural injury, providing a valuable framework for early detection, monitoring, and intervention in DSPN [19,20].

Complementing the MNSI findings, the nerve conduction study (NCS) results provide objective electrophysiological evidence of neuropathy progression over the 3-month follow-up period. Specifically, both the tibial motor and sural sensory nerves demonstrated signs of deteriorating nerve function, consistent with the clinical progression observed in patients with distal symmetric polyneuropathy (DSPN) (table-1) [21].

In the tibial nerve, there was a notable reduction in amplitude (from 6.3 ± 2.2 mV to 5.4 ± 2.0 mV; $p = 0.011$), and a significant slowing of conduction velocity (from 41.6 ± 3.8 m/s to 39.2 ± 4.5 m/s; $p = 0.032$), both of which suggest axon loss and demyelination. Although the increase in distal latency (from 4.7 ± 0.9 ms to 5.1 ± 1.1 ms) did not reach conventional statistical significance ($p = 0.051$), the trend points to a gradual delay in impulse conduction, further supporting early or subclinical worsening (table-1) [22,23].

Similarly, changes in the sural nerve further reinforce sensory nerve involvement. There was a significant increase in latency ($p = 0.041$), a marked reduction in amplitude ($p = 0.013$), and a significant decrease in conduction velocity ($p = 0.001$), all of which are typical of progressive sensory axonal neuropathy, a hallmark of DSPN. These findings are particularly relevant given that the sural nerve is often affected early in diabetic neuropathy and correlates well with symptom burden.

Taken together with the higher and progressively worsening MNSI scores in patients with vitamin D deficiency and elevated NFL levels, the NCS data provides robust physiological confirmation that these patients are at increased risk of nerve function decline. The alignment between subjective symptoms (MNSI), biochemical markers (NFL, vitamin D), and objective nerve conduction abnormalities offers strong evidence for a multifactorial model of DSPN progression—where nutritional status, axon-specific injury markers, and neurophysiological decline interact over time.

These results emphasize the importance of early detection and comprehensive monitoring in diabetic patients, including routine assessment of vitamin D levels, NFL concentrations, MNSI scoring, and NCS. They also highlight potential windows for intervention, where vitamin D supplementation or neuroprotective strategies might delay or prevent irreversible nerve damage.

Importantly, NFL levels were significantly elevated in patients who developed new or worsening DSPN. NFL, a structural protein of large-caliber myelinated axons, has been recognized as a sensitive marker of axonal injury and neurodegeneration in multiple central and peripheral nervous system disorders [23]. Our study adds to the

growing body of evidence supporting NFL as a noninvasive and dynamic biomarker for peripheral nerve damage in diabetic populations. The observed correlation between NFL and clinical/electrophysiological markers of DSPN reinforces its predictive value [24].

Multivariate analysis revealed that vitamin D deficiency and NFL elevation independently predicted DSPN progression, even after adjusting for diabetes duration, glycemic control, and age. This suggests a potential complementary role for these biomarkers in stratifying neuropathy risk beyond traditional metabolic parameters.

Strengths and Limitations

A major strength of this study is its prospective design, use of objective nerve conduction studies, and dual biomarker assessment. The inclusion of a relatively large cohort and 3-month follow-up allowed for early detection of subclinical neuropathy progression.

However, some limitations must be acknowledged. First, the short follow-up duration may not capture the full trajectory of neuropathy progression, which typically develops over years. Second, seasonal variations in vitamin D synthesis and potential dietary confounders were not fully controlled. Third, NFL levels may be influenced by comorbid neurodegenerative conditions, though strict exclusion criteria were applied.

Clinical Implications and Future Directions

Our findings suggest that early measurement of serum 25(OH)D and NFL could guide risk stratification and early interventions in diabetic patients. Future studies with longer follow-up periods, intervention arms (e.g., vitamin D supplementation), and serial NFL measurements could better clarify the causal and therapeutic implications. Moreover, the integration of these biomarkers into predictive algorithms alongside clinical scores could enhance early diagnosis of DSPN in routine practice.

Conclusion

In summary, this study demonstrates that vitamin D deficiency and elevated NFL levels are independently associated with short-term progression of DSPN in T2DM patients. These biomarkers may serve as valuable tools for early identification and monitoring of patients at risk for diabetic neuropathy.

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Table:1 Comparison of lower limb NCS parameters at baseline and after 3 months

Nerve	Parameter	Baseline (Mean ± SD)	3-Month (Mean ± SD)	p-value
Tibial Nerve	Distal Latency (ms)	4.7 ± 0.9	5.1 ± 1.1	0.051
	Amplitude (mV)	6.3 ± 2.2	5.4 ± 2.0	0.011
	Conduction Velocity (m/s)	41.6 ± 3.8	39.2 ± 4.5	0.032
Sural Nerve	Latency (ms)	3.2 ± 0.7	3.5 ± 0.8	0.041
	Amplitude (μV)	9.8 ± 3.1	7.6 ± 2.9	0.013
	Conduction Velocity (m/s)	43.2 ± 4.0	40.1 ± 4.8	0.001

Table:2 MNSI scores at baseline and 3 months according to Vitamin D status and NFL levels in patients with DSPN

Group	Time Point	MNSI Questionnaire Score (Mean ± SD)	MNSI Examination Score (Mean ± SD)
Vitamin D Sufficient (>30 ng/mL)	Baseline	4.8 ± 1.7	2.3 ± 1.0
	3 Months	5.1 ± 1.8	2.5 ± 1.1
Vitamin D Deficient (<20 ng/mL)	Baseline	6.2 ± 1.9	3.0 ± 1.1
	3 Months	7.4 ± 2.0	3.5 ± 1.2
NFL < 40 pg/mL	Baseline	5.0 ± 1.8	2.4 ± 1.1
	3 Months	5.4 ± 1.9	2.6 ± 1.1
NFL ≥ 40 pg/mL	Baseline	6.1 ± 1.8	3.1 ± 1.0
	3 Months	7.3 ± 2.1	3.6 ± 1.2

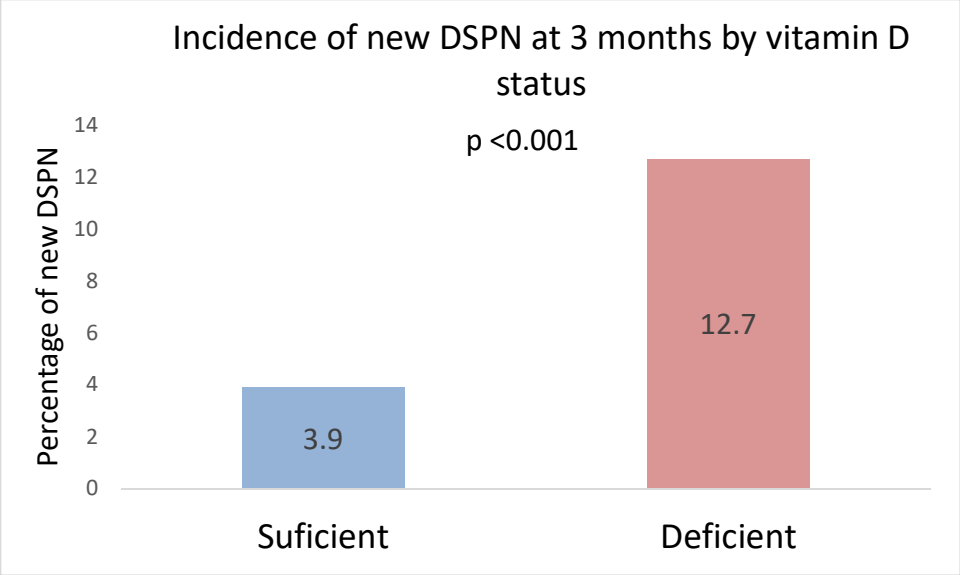

Figure:1 Incidence of new DSPN at 3 months by vitamin D status