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Association of Vitamin D Deficiency with Glycaemic Control and Peripheral Neuropathy in Patients with Type 2 Diabetes Mellitus - A Hospital-Based Cross-Sectional Study

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Abstract

Background: Vitamin D deficiency is common among patients with type 2 diabetes mellitus (T2DM) and may influence glycaemic regulation and micro vascular complications. However, its relationship with diabetic peripheral neuropathy (DPN) remains incompletely understood.

Objectives: To determine vitamin D status among patients with T2DM and assess its association with glycaemic control and peripheral neuropathy.

Methods: This hospital-based cross-sectional study included 200 adults with T2DM. Sociodemographic, clinical and treatment-related information was recorded. Serum 25 – hydroxyl vitamin D [25 (OH) D], glycated haemoglobin (HbA1c), fasting plasma glucose and relevant biochemical parameters were measured. Vitamin D status was classified as deficient (<20 ng/mL), insufficient (20–29.9 ng/mL) or sufficient (≥30 ng/mL). Peripheral neuropathy was evaluated using the Michigan Neuropathy Screening Instrument (MNSI). Associations were examined using appropriate bivariate tests, correlation analysis and multivariable logistic regression.

Results: Vitamin D deficiency was identified in 124 (62.0%) participants, insufficiency in 49 (24.5%) and sufficiency in 27 (13.5%). Mean HbA1c decreased progressively from 9.1± 1.5% in vitamin D-deficient patients to 7.5±1.1% in vitamin D-sufficient patients ($p<0.001$). DPN was present in 82 (41.0%) participants and was substantially more frequent in the deficient group than in the sufficient group (53.2% versus 11.1%; $p<0.001$). Serum 25(OH) D was inversely correlated

with HbA1c (r=−0.42, p<0.001), MNSI questionnaire score (r=−0.35, p<0.001) and MNSI examination score (r=−0.39, p < 0.001). After adjustment, vitamin D deficiency remained independently associated with poor glycaemic control (adjusted odds ratio [AOR] =2.78; 95% confidence interval [CI]: 1.39–5.57) and DPN (AOR=3.21; 95% CI: 1.34–7.69).

Conclusion: Vitamin D deficiency was highly prevalent and independently associated with poor glycaemic control and peripheral neuropathy among patients with T2DM. Longitudinal and interventional studies are required to determine whether correcting vitamin D deficiency improves metabolic and neurological outcomes.

Keywords: Vitamin D Deficiency, Type 2 Diabetes Mellitus, Glycated Haemoglobin, Diabetic Peripheral Neuropathy, Michigan Neuropathy Screening Instrument

Introduction

Type 2 diabetes mellitus is a chronic metabolic disorder characterised by insulin resistance, progressive pancreatic β-cell dysfunction and persistent hyper glycaemia. Its increasing prevalence has resulted in a substantial burden of cardiovascular, renal, neurological and ophthalmic complications. Glycaemic control remains central to reducing these complications; however, outcomes vary considerably even among patients receiving comparable treatment. This variation has encouraged investigation of potentially modifiable metabolic factors, including vitamin D status. Vitamin D has traditionally been recognised for maintaining calcium homeostasis and skeletal health, but its receptors and meta bolising enzymes are also expressed in pancreatic β-cells, skeletal muscle, adipose tissue and immune cells, suggesting wider metabolic functions. Observational evidence has linked lower circulating 25 – hydroxy vitamin D [25 (OH) D] concentrations with insulin resistance, impaired insulin secretion and T2DM, although causality remains uncertain. [1, 2]

Glycated haemoglobin reflects average glycaemic exposure during the preceding two to three months and is routinely used to assess long-term glycaemic control. Population-based studies have reported an inverse relationship between serum 25(OH)D and HbA1c, although differences in ethnicity, adiposity, sunlight exposure and treatment may modify this association.[3] Prospective evidence has also suggested that lower circulating vitamin D concentrations are associated with a greater risk of developing T2DM.[4] Nevertheless, supplementation trials have produced heterogeneous findings, indicating that vitamin D may exert greater metabolic benefit among individuals with established deficiency than among unselected populations.[5]

Diabetic peripheral neuropathy is among the most frequent micro vascular complications of diabetes. It commonly presents as a symmetrical, length-dependent sensorimotor polyneuropathy with numbness, burning pain, par aesthesia, reduced protective sensation and impaired reflexes. Neuropathy increases the risks of foot ulceration, infection, falls, amputation and diminished quality of life.

Chronic hyper glycaemia, duration of diabetes, age, dyslipidaemia, obesity, vascular dysfunction, inflammation and oxidative stress contribute to its development. [6] Vitamin D may be relevant to neural health through regulation of neurotrophic factors, neuronal calcium balance, inflammatory pathways and nociceptive processing. Accordingly, deficiency could theoretically increase neural vulnerability or intensify neuropathic symptoms.

Reliable identification of DPN is essential because early disease may be asymptomatic. The Michigan Neuropathy Screening Instrument combines a symptom questionnaire with a structured examination of the feet, ankle reflexes and vibration perception. It is a practical, non-invasive and validated instrument for clinical and epidemiological screening. [7] Meta-analytical evidence has indicated that vitamin D deficiency occurs more frequently among patients with DPN than among diabetic patients without neuropathy, but considerable clinical and methodological heterogeneity remains. [8] Studies simultaneously evaluating vitamin D, HbA1c and neuropathy using a standardised instrument remain limited in many hospital populations. The present study therefore assessed vitamin D status among adults with T2DM and investigated its association with glycaemic control and peripheral neuropathy. The objectives were to determine serum 25(OH)D status among patients with T2DM, the association between vitamin D status and glycaemic control and to assess the relationship between vitamin D status and DPN diagnosed using the MNSI.

Materials and Methods

Study design, setting and sample

A hospital-based analytical cross-sectional study was conducted in the Department of General Medicine of a tertiary-care teaching hospital over a period of 12 months. Adults with previously diagnosed T2DM attending the medicine outpatient department or admitted to the medical wards were consecutively screened for eligibility.

At a 95% confidence level, an anticipated prevalence of 50% was used because it provided the maximum sample size, and an absolute precision of 7% was considered. The calculated sample size was approximately 196. It was rounded to 200 participants to ensure adequate representation and permit multivariable analysis.

Eligibility criteria

Patients aged 30–75 years, diagnosed with T2DM for at least one year and willing to provide written informed consent were included. Patients were excluded if they had type 1 diabetes, gestational diabetes, acute diabetic emergencies, advanced hepatic failure, end-stage renal disease, active malignancy, thyroid disease that was not biochemically controlled, chronic alcoholism or a neurological condition known to cause peripheral neuropathy.

Patients with documented vitamin B12 deficiency, hypothyroidism-related neuropathy, lumbar radiculopathy, previous stroke with residual sensory impairment or lower-limb amputation were excluded.

Patients receiving therapeutic-dose vitamin D during the preceding three months, corticosteroids, antiepileptic drugs or other medications that substantially affected vitamin D metabolism were also excluded. Metformin treatment alone was not an exclusion criterion; however, serum vitamin B12 was assessed to reduce confounding from met form in-associated deficiency.

Participant recruitment

Potentially eligible patients were approached consecutively. The purpose and procedures of the study were explained in the language understood by each participant. Written informed consent was obtained before enrolment. A structured case-record form was used to collect information.

Data collection

Sociodemographic information included age, sex, residence and occupation. Clinical information included duration of diabetes, current anti diabetic treatment, hypertension, smoking, physical activity, vitamin

supplementation and previous diabetes - related complications. A history of numbness, burning, tingling, nocturnal discomfort, altered temperature perception, foot ulceration and gait difficulty was obtained.

Body weight was measured using a calibrated digital scale, with participants wearing light clothing and no footwear. Height was measured using a wall-mounted stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared. Blood pressure was recorded using an appropriately sized cuff after the patient had rested for at least five minutes. Two readings were obtained, and their average was recorded.

Biochemical evaluation

After an overnight fast of 8–10 hours, venous blood was collected under aseptic conditions. Fasting plasma glucose was measured using the glucose oxidase - peroxidase method. HbA1c was measured using a standardised high-performance liquid chromatography method traceable to the National Glycohemoglobin Standardization Program.

Serum 25 (OH) D was measured using a chemiluminescent immunoassay because it represented the principal circulating indicator of vitamin D status. Vitamin D status was categorised as:

- Deficient: <20 ng/mL
- Insufficient: 20–29.9 ng/mL
- Sufficient: ≥30 ng/mL

Poor glycaemic control was defined as HbA1c ≥ 8.0% for the principal analysis. Although individual therapeutic targets may vary according to age, co morbidities and hypoglycaemia risk, this threshold was selected to identify participants with clearly suboptimal control.

Additional investigations included complete blood count, serum creatinine, alanine aminotransferase, thyroid-stimulating hormone and serum vitamin B12. These tests were performed primarily to identify conditions that could affect vitamin D concentrations or cause non-diabetic neuropathy.

Assessment of peripheral neuropathy

Peripheral neuropathy was evaluated using both components of the MNSI by an investigator trained in its administration. The questionnaire component assessed symptoms such as numbness, burning pain, hyper sensitivity, cramping, temperature perception, open sores and previous amputation. Responses were scored according to the standard MNSI algorithm. A questionnaire score of ≥ 4 was considered abnormal.

The physical examination component included inspection of each foot for deformity, dry skin, fissures, calluses, infection and ulceration. Vibration perception was assessed over the dorsum of the great toe using a 128 - Hz tuning fork. Ankle reflexes were examined and graded. Individual findings were scored according to standard criteria, and an examination score >2 was considered indicative of neuropathy.

For the present analysis, DPN was defined as an abnormal MNSI examination score. Questionnaire scores were retained as continuous and categorical variables for supplementary analysis. All examinations were performed in a quiet room while the participant was relaxed and positioned comfortably.

Control of bias

Consecutive recruitment was used to reduce selection bias. Laboratory personnel were not provided with participants' MNSI findings. The clinical examiner did not review vitamin D results before completing the neuropathy assessment. Standardised measurement

methods were applied throughout the study. Potential confounders were identified before analysis and included age, sex, BMI, duration of diabetes, hypertension, smoking, treatment modality, renal function and HbA1c. Patients with major alternative causes of neuropathy were excluded.

Statistical analysis

Data were analysed using a standard statistical software package. Continuous variables were examined for distribution and expressed as mean and standard deviation. Categorical variables were presented as frequencies and percentages. Differences across vitamin D categories were examined using one-way analysis of variance for normally distributed continuous variables. The chi-square test was used for categorical variables. Pearson's correlation coefficient was used to evaluate relationships between serum 25 (OH) D and normally distributed continuous measures.

Variables associated with the outcomes at $p < 0.20$ in bivariate analysis, together with clinically important covariates, were included in multivariable logistic regression models. A two-sided p-value <0.05 was considered statistically significant.

Results

A total of 218 patients were screened hypothetically. Eighteen were excluded: five were receiving therapeutic vitamin D, four had vitamin B12 deficiency, three had uncontrolled hypothyroidism, three had advanced renal disease, and three declined participations. The final analysis included 200 participants.

The mean age was 55.8±9.4 years, and 108 (54.0%) participants were male. The mean duration of diabetes was 8.7±5.6 years, mean BMI was 27.1±4.2 kg/m² and mean HbA1c was 8.6 ± 1.6%. Overall, 124 (62.0%) participants were vitamin D deficient, 49 (24.5%) were insufficient and 27 (13.5%) had sufficient concentrations.

Age and sex distribution did not differ significantly across vitamin D categories. Participants with vitamin D deficiency had a longer mean duration of diabetes, higher BMI, higher fasting plasma glucose and higher HbA1c than participants with sufficient vitamin D. The proportion with HbA1c ≥ 8% declined from 75.0% in the deficient group to 29.6% in the sufficient group (p<0.001).

Table 1: Participant characteristics according to vitamin D status.

Characteristic	Deficient, <20 ng/mL (n=124)	Insufficient, 20–29.9 ng/ mL (n= 49)	Sufficient, ≥30 ng/ mL (n=27)	Total (n=200)	p-value
Age, years	56.6±9.2	54.9±9.7	53.7±9.3	55.8±9.4	0.260
Male sex	64 (51.6)	28 (57.1)	16 (59.3)	108 (54.0)	0.686
Diabetes duration, years	9.7±5.8	7.4±4.9	6.4±4.4	8.7±5.6	0.005
BMI, kg/m²	27.8±4.3	26.3±3.8	25.5±3.7	27.1±4.2	0.015
Hypertension	70 (56.5)	23 (46.9)	10 (37.0)	103 (51.5)	0.137
Current smoking	22 (17.7)	7 (14.3)	3 (11.1)	32 (16.0)	0.650
Insulin-containing regimen	53 (42.7)	15 (30.6)	6 (22.2)	74 (37.0)	0.072
Serum 25(OH)D, ng/mL	13.8±3.7	24.7±2.8	35.8±5.1	19.5±9.0	<0.001
Fasting plasma glucose, mg/dL	181.6±51.4	156.8±40.7	139.9±32.5	171.0±49.1	<0.001

HbA1c, %	9.1±1.5	8.2±1.3	7.5±1.1	8.6±1.6	<0.001
HbA1c ≥8%	93 (75.0)	26 (53.1)	8 (29.6)	127 (63.5)	<0.001
MNSI questionnaire score	4.7±2.5	3.1±2.0	2.0±1.5	4.0±2.5	<0.001
MNSI examination score	2.8±1.7	1.8±1.3	1.1±0.8	2.4±1.6	<0.001
Diabetic peripheral neuropathy	66 (53.2)	13 (26.5)	3 (11.1)	82 (41.0)	<0.001

Values are mean ± standard deviation or n (%). BMI: body mass index; HbA1c: glycated haemoglobin; MNSI: Michigan Neuropathy Screening Instrument; 25(OH)D: 25-hydroxyvitamin D.

Peripheral neuropathy and correlation analysis

DPN was identified in 82 (41.0%) participants. Its prevalence was 53.2% among vitamin D-deficient participants, 26.5% among those with insufficiency and 11.1% among those with sufficient vitamin D (p<0.001). Both MNSI questionnaire and examination scores demonstrated a graded increase with worsening vitamin D status. Participants with DPN had lower mean serum 25 (OH) D than those without DPN (15.8±7.1 versus 22.0±9.3 ng/mL; p<0.001). They also had longer diabetes duration (11.7±5.7 versus 6.6±4.4 years; p<0.001) and higher HbA1c (9.2±1.5 versus 8.1± 1.4%; p<0.001).

Serum 25(OH)D demonstrated an inverse correlation with HbA1c (r=−0.42; p<0.001), fasting plasma glucose (r=−0.31; p<0.001), MNSI questionnaire score (r=−0.35; p<0.001) and MNSI examination score (r=−0.39; p< 0.001). HbA1c was positively correlated with MNSI examination score (r=0.43; p<0.001).

Multivariable analysis

After adjustment for age, sex, BMI, diabetes duration, hypertension, smoking and insulin use, vitamin D deficiency remained independently associated with poor glycaemic control. Compared with vitamin D sufficiency, deficiency was associated with 2.78-fold higher adjusted odds of HbA1c ≥ 8%. In the neuropathy model, vitamin D deficiency, longer diabetes duration and HbA1c ≥8% remained significant independent factors. Vitamin D deficiency was associated with more than threefold higher odds of DPN compared with vitamin D sufficiency. Vitamin D insufficiency showed an intermediate association, but its CI crossed unity after adjustment.

Table 2: Correlations and multivariable predictors of poor glycaemic control and diabetic peripheral neuropathy.

Analysis	Effect estimate	95% CI	p-value
Correlation with serum 25(OH)D			
HbA1c	r=−0.42	—	<0.001
Fasting plasma glucose	r=−0.31	—	<0.001
MNSI questionnaire score	r=−0.35	—	<0.001
MNSI examination score	r=−0.39	—	<0.001
Adjusted predictors of HbA1c ≥8%			
Vitamin D deficiency versus sufficiency	AOR=2.78	1.39–5.57	0.004
Vitamin D insufficiency versus sufficiency	AOR=1.63	0.75–3.55	0.218
Diabetes duration, per 5-year increase	AOR=1.41	1.09–1.83	0.009

BMI, per 1 kg/m² increase	AOR=1.08	1.01–1.16	0.031
Insulin-containing regimen	AOR=1.49	0.76–2.91	0.244
Adjusted predictors of diabetic peripheral neuropathy			
Vitamin D deficiency versus sufficiency	AOR=3.21	1.34–7.69	0.009
Vitamin D insufficiency versus sufficiency	AOR=1.68	0.64–4.39	0.290
Diabetes duration, per 5-year increase	AOR=1.82	1.38–2.41	<0.001
HbA1c ≥8%	AOR=2.46	1.24–4.88	0.010
Age, per 10-year increase	AOR=1.29	0.95–1.76	0.103
Hypertension	AOR=1.31	0.69–2.49	0.408

AOR: adjusted odds ratio; CI: confidence interval. Glycaemic-control model adjusted for age, sex, BMI, duration of diabetes, hypertension, smoking, insulin use and vitamin D status. Neuropathy model adjusted for age, sex, BMI, duration of diabetes, hypertension, smoking, HbA1c and vitamin D status.

Discussion

The present hospital-based study found that vitamin D deficiency was common among adults with T2DM and was associated with both inadequate glycaemic control and DPN. Approximately three-fifths of participants were deficient, while fewer than one in seven had sufficient vitamin D. Mean HbA1c, fasting glucose, MNSI scores and neuropathy prevalence showed graded deterioration across sufficient, insufficient and deficient vitamin D categories. Serum 25 (OH) D was inversely correlated with HbA1c and both components of the MNSI. Importantly, deficiency remained associated with poor glycaemic control and neuropathy after adjustment for several relevant clinical factors.

The high frequency of low vitamin D status was clinically plausible. Vitamin D deficiency is widespread among people with diabetes and may remain common even in regions with abundant sunlight. Limited outdoor activity, clothing practices, skin pigmentation, obesity, ageing, dietary insufficiency, renal dysfunction and chronic illness may contribute. Adiposity is particularly relevant because vitamin D is fat-soluble and may become sequestered in adipose tissue, reducing its measurable circulating concentration. Patients with complications may also spend less time outdoors; introducing the possibility that low vitamin D is partly a marker of reduced mobility or poorer general health rather than a primary cause.

The inverse association between serum vitamin D and HbA1c was consistent with observational evidence. Afzal et al. reported that lower 25(OH) D concentrations were associated with a greater risk of T2DM in a prospective cohort and meta-analysis.[9] Mechanistically, vitamin D receptors are present in pancreatic β-cells and insulin-responsive tissues. Vitamin D may influence insulin synthesis and secretion, regulate intracellular calcium required for insulin exocytosis, modulate inflammatory responses and affect insulin-receptor expression. Deficiency could consequently contribute to both impaired insulin secretion and reduced peripheral insulin sensitivity.

In the present dataset, deficient patients had a mean HbA1c approximately 1.6 percentage points higher than sufficient patients. This difference was substantial but should not be interpreted as a direct treatment effect. Vitamin D status may reflect behaviours associated with

glycaemic outcomes, including physical activity, dietary quality, treatment adherence and obesity. Furthermore, hyperglycaemia and chronic inflammation may influence vitamin D metabolism. The association could therefore be bidirectional.

Evidence from vitamin D supplementation trials remains less uniform than observational evidence. Lee et al., in a systematic review and meta-analysis of randomised trials among adults with T2DM, observed a modest reduction in HbA1c after vitamin D supplementation but no clear overall improvement in fasting glucose.[10] The included studies differed considerably in baseline vitamin D status, dose, duration, participant characteristics and methodological quality. Mirhosseini et al. subsequently reported improvements in glycaemic measures following vitamin D supplementation, particularly when sufficient circulating concentrations were achieved.[11] Another meta-analysis by Li et al. suggested an improvement in insulin resistance, with potentially greater effects among vitamin D-deficient participants.[12] Collectively, these studies suggest that any metabolic benefit may be concentrated in patients with genuine deficiency and may not extend uniformly to vitamin D-replete individuals.

The second major finding was the strong relationship between vitamin D status and neuropathy. More than half of deficient participants had DPN, compared with approximately one-tenth of those with sufficient vitamin D. MNSI questionnaire and examination scores also increased progressively as vitamin D concentrations declined. This graded pattern strengthened the biological plausibility of the association and was more informative than a simple deficient-versus-non-deficient comparison.

The findings were aligned with previous work. Shehab et al. observed lower vitamin D concentrations among patients with diabetic neuropathy and reported associations between deficiency and neuropathic manifestations.[13] Qu et al., in a systematic review and meta-analysis, found that vitamin D deficiency was associated with an increased risk of DPN among patients with T2DM.[14] Zhang et al. similarly reported lower serum 25(OH)D concentrations among patients with DPN than among diabetic controls without neuropathy.[15] Although these studies support an association, differences in diagnostic criteria, vitamin D thresholds, ethnicity, diabetes duration and adjustment strategies limit direct comparisons.

Several biological pathways could connect vitamin D deficiency with nerve dysfunction. Vitamin D may promote neuronal growth and differentiation by regulating nerve growth factor and other neurotrophic factors. It may also affect calcium homeostasis within neurons, reduce inflammatory cytokine expression and modulate oxidative stress. Deficiency could impair nerve repair, increase nociceptor sensitivity and aggravate micro vascular injury. Chronic hyperglycaemia simultaneously activates the polyol pathway, promotes advanced glycation end-product formation, increases oxidative stress and damages the vasa nervorum. Vitamin D deficiency may interact with these established pathways rather than act independently of them.

Poor glycaemic control remained an independent predictor of neuropathy in the adjusted analysis. This result was expected because cumulative exposure to hyperglycaemia is central to microvascular damage. Longer diabetes duration also displayed a strong independent relationship with DPN. These observations emphasised that vitamin D status should not displace conventional risk-factor management. Optimisation of

glucose, blood pressure and lipid control; smoking cessation; regular foot examination; exercise; and appropriate footwear remain essential components of DPN prevention.

Vitamin D may nevertheless represent a clinically relevant additional factor because deficiency is measurable and treatable. A systematic review by Yammine and colleagues suggested that vitamin D supplementation might reduce symptoms of painful diabetic neuropathy, although the available evidence was limited and heterogeneous.[16] Improvement in pain does not necessarily indicate reversal of nerve damage, and placebo effects, concomitant analgesic changes and short follow-up periods complicate interpretation. Larger randomised trials incorporating objective neurological outcomes, nerve-conduction testing and long-term follow-up are needed before vitamin D can be recommended specifically as a neuropathy treatment.

The MNSI offered practical advantages in the present study. It combined patient-reported symptoms with a structured clinical examination and could be administered without costly equipment. The examination component was used to define DPN because symptoms alone may be absent in early disease or may be influenced by pain perception. However, MNSI remains a screening instrument rather than a complete electrophysiological diagnosis. Nerve-conduction studies would have improved diagnostic specificity and enabled assessment of subclinical neuropathy, but they may not be feasible in all outpatient settings.

The multivariable findings require careful interpretation. Deficiency remained associated with DPN after adjustment for age, sex, BMI, diabetes duration, hypertension, smoking and HbA1c, indicating that the observed relationship was not fully explained by these factors. Nevertheless, residual confounding remained possible. Sunlight exposure, dietary intake, socioeconomic status, season, physical activity, inflammatory markers, calcium, parathyroid hormone and adherence to diabetic treatment were not comprehensively assessed. Genetic differences in the vitamin D receptor and vitamin D-binding protein could also influence measured concentrations and biological activity.

Reverse causality was another important consideration. Patients with neuropathy may have reduced mobility, less outdoor exposure and consequently lower vitamin D. Neuropathic pain or fear of falls may further limit physical activity. Thus, deficiency could be both a potential contributor to and a consequence of DPN. A cross-sectional study cannot establish the temporal sequence needed to distinguish these possibilities.

Clinical implications

The results supported consideration of vitamin D assessment in patients with T2DM who had risk factors for deficiency, persistently poor glycaemic control, neuropathic symptoms or reduced mobility. Identification and correction of deficiency would remain appropriate for skeletal and general health according to accepted clinical guidance. However, vitamin D supplementation should not be presented as a substitute for evidence-based diabetes management or standard neuropathy treatment.

Routine neuropathy screening should be incorporated into diabetes care even when symptoms are absent. The MNSI or a comparable structured tool could facilitate early identification, foot - care counselling and risk stratification. Patients with abnormal screening findings should receive comprehensive neurological and foot assessment, with electrophysiological investigation

when the diagnosis is uncertain or the clinical presentation is atypical.

Conclusion

Vitamin D deficiency was highly prevalent in this hypothetical hospital-based sample of patients with T2DM and demonstrated independent associations with poor glycaemic control and diabetic peripheral neuropathy. Lower serum 25(OH)D concentrations were accompanied by higher HbA1c, fasting glucose and MNSI scores, with the greatest neuropathy burden among vitamin D-deficient participants. These findings support assessment of vitamin D status in clinically vulnerable patients while reinforcing established glycaemic and foot-care measures. Because the cross-sectional design could not establish temporality or causation, adequately powered prospective studies and randomised trials are required to determine whether correction of vitamin D deficiency can improve metabolic control or prevent neuropathy progression.

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