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Original Research Article

# VITAMIN D STATUS AND MUSCULOSKELETAL SYMPTOMS IN TYPE 2 DIABETES: A CROSS-SECTIONAL CORRELATION STUDY

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**ABSTRACT**

**Background:** Vitamin D deficiency and musculoskeletal complications are common in patients with type 2 diabetes mellitus (T2DM). However, their interrelationship remains underexplored, especially in Indian populations where hypovitaminosis D is endemic. **Aim:** To assess the correlation between vitamin D status and musculoskeletal symptoms in patients with type 2 diabetes mellitus.

**Materials and Methods:** A hospital-based cross-sectional observational study was conducted among 240 patients with T2DM. Clinical data including demographics, BMI, duration of diabetes, and HbA1c were collected. Musculoskeletal symptoms were assessed using structured questionnaires and clinical examination. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using ELISA and categorized as deficient (<20 ng/mL), insufficient (20-29 ng/mL), or sufficient (≥30 ng/mL). Statistical analysis included chi-square, ANOVA, and Pearson correlation tests, with $p < 0.05$ considered significant.

**Results:** The mean age of participants was 54.7 ± 9.3 years, with 53.3% males. The prevalence of vitamin D deficiency was 60.8%, insufficiency 25.8%, and sufficiency 13.4%, with an overall mean of 20.7 ± 7.5 ng/mL. Musculoskeletal symptoms were reported in 42.5% of patients, the most common being generalized muscle pain (25.4%) and low back pain (19.6%). Patients with vitamin D deficiency showed significantly higher prevalence of symptoms (56.2%) and higher mean pain scores (6.5 ± 1.6) compared to sufficient individuals (12.5%). A significant negative correlation was observed between vitamin D levels and musculoskeletal symptom severity ($r = -0.38$, $p < 0.001$).

**Conclusion:** Vitamin D deficiency is highly prevalent in T2DM patients and is strongly associated with musculoskeletal symptoms. Routine screening and timely correction of hypovitaminosis D may help reduce musculoskeletal morbidity and improve patient outcomes.

**Keywords:** Vitamin D deficiency. Type 2 diabetes mellitus. Musculoskeletal symptoms.

## INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most prevalent chronic metabolic disorders worldwide, characterized by hyperglycemia, insulin resistance, and progressive β-cell dysfunction. With its rising prevalence across all age groups, T2DM has become a major public health problem, particularly in low- and middle-income countries where lifestyle transitions, urbanization, and changes in dietary patterns are accelerating. Beyond the well-documented complications such as retinopathy, nephropathy, neuropathy, and cardiovascular disease, there is growing recognition of the musculoskeletal system as a significant target of diabetic complications. Musculoskeletal pain, stiffness, reduced joint mobility, and muscle weakness are increasingly reported among individuals with T2DM, yet these symptoms remain under-recognized and frequently overlooked in clinical practice.[1]

Vitamin D, a fat-soluble secosteroid hormone, plays a critical role in calcium homeostasis, bone mineralization, and muscle function. Over the past two decades, interest in vitamin D has expanded beyond bone health, with evidence linking its deficiency to metabolic diseases, immune dysfunction, cardiovascular risk, and neuromuscular impairment. Hypovitaminosis D is highly prevalent worldwide, affecting nearly one billion people, particularly in South Asia and the Middle East despite abundant sunlight. In patients with T2DM, vitamin D deficiency is observed at disproportionately high rates compared to the general population, raising the possibility of a bidirectional relationship between vitamin D status and diabetic complications.[2]

The pathophysiological link between vitamin D and musculoskeletal symptoms in T2DM is multifaceted. Vitamin D receptors (VDRs) are widely expressed in skeletal muscle cells, and vitamin D signaling is essential for muscle protein synthesis, mitochondrial function, and calcium uptake. Deficiency may contribute to muscle weakness, sarcopenia, impaired balance, and chronic musculoskeletal pain. Additionally, vitamin D deficiency is associated with low-grade systemic inflammation and poor glycemic control, both of which further exacerbate musculoskeletal complications in diabetic patients. Several studies have suggested correlations between serum vitamin D levels and markers of muscle performance, grip strength, fall risk, and pain perception in T2DM populations. However, results remain heterogeneous, with variations depending on study design, population, and methodology.[3]

In the Indian subcontinent, where vitamin D deficiency is endemic due to cultural, dietary, and lifestyle factors, patients with T2DM may be particularly vulnerable to musculoskeletal complications associated with low vitamin D status. Limited outdoor exposure, traditional clothing, vegetarian diets, and dark skin pigmentation all contribute to reduced cutaneous vitamin D synthesis. Moreover, comorbidities such as obesity, chronic kidney disease, and sedentary lifestyle-commonly seen in T2DM-further reduce vitamin D bioavailability and activity.[4]

Despite this evidence, few studies have systematically investigated the correlation between vitamin D levels and musculoskeletal symptoms in T2DM patients in tertiary care settings. Understanding this relationship is clinically significant as it may guide preventive and therapeutic strategies, including vitamin D supplementation, lifestyle interventions, and targeted musculoskeletal rehabilitation. Early detection and correction of vitamin D deficiency could potentially improve quality of life, reduce morbidity, and optimize metabolic and musculoskeletal outcomes in diabetic individuals.[5]

**Aim**

To assess the correlation between vitamin D status and musculoskeletal symptoms in patients with type 2 diabetes mellitus.

**Objectives**

1. To evaluate serum vitamin D levels in patients with type 2 diabetes mellitus.
2. To determine the prevalence and pattern of musculoskeletal symptoms among these patients.
3. To analyze the correlation between vitamin D status and musculoskeletal manifestations in type 2 diabetes.

## MATERIALS AND METHODS

**Source of Data**

The data were obtained from patients with type 2 diabetes mellitus attending the outpatient and inpatient departments of General Medicine and Endocrinology at a tertiary care teaching hospital.

**Study Design**

A hospital-based cross-sectional observational study design was adopted.

**Study Location**

The study was conducted at the Department of General Medicine, at tertiary care hospital catering to urban and rural populations.

**Study Duration**

The study was carried out over a period of 18 months, from January 2023 to June 2024.

**Sample Size**

A total of 240 patients with type 2 diabetes mellitus were enrolled in the study.

**Inclusion Criteria**

- Patients diagnosed with type 2 diabetes mellitus according to ADA criteria.
- Age between 30 and 70 years.
- Patients willing to provide informed consent.

**Exclusion Criteria**

- Patients with type 1 diabetes mellitus or gestational diabetes.
- Patients with chronic liver disease, chronic kidney disease (stage IV-V), or malabsorption syndromes.
- Patients on vitamin D supplementation or calcium therapy in the past 3 months.
- Patients with known musculoskeletal disorders unrelated to diabetes (e.g., rheumatoid arthritis, osteoarthritis).
- Patients on medications affecting bone metabolism (e.g., glucocorticoids, anticonvulsants).

**Procedure and Methodology**

Eligible patients were enrolled after obtaining informed written consent. Detailed demographic and clinical data were collected, including age, sex, duration of diabetes, comorbidities, lifestyle factors (sunlight exposure, diet, physical activity), and history of musculoskeletal symptoms such as pain, stiffness, weakness, and restricted mobility. A structured questionnaire and standardized clinical examination were used to document musculoskeletal complaints.

Musculoskeletal symptoms were assessed using validated tools such as the Visual Analogue Scale (VAS) for pain and functional mobility tests (e.g., timed up-and-go test, grip strength measurement). Clinical examination focused on joints, muscles, and range of motion.

Venous blood samples were collected in the morning after an overnight fast for biochemical analysis.

**Sample Processing**

Blood samples were centrifuged, and serum was separated and stored at -20°C until analysis. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using a quantitative ELISA method. Levels were categorized as:

- Deficient: <20 ng/mL
- Insufficient: 20-29 ng/mL
- Sufficient: ≥30 ng/mL

Additional biochemical tests included fasting plasma glucose, HbA1c, calcium, phosphate, and alkaline phosphatase, measured using standard laboratory protocols.

**Statistical Methods**

Data were entered in Microsoft Excel and analyzed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Correlation between vitamin D levels and musculoskeletal symptoms was analyzed using Pearson's or Spearman's correlation coefficient as appropriate. Chi-square test was used to compare categorical variables. Independent t-test or ANOVA was applied for group comparisons. A p-value <0.05 was considered statistically significant.

**Data Collection**

All data were collected prospectively through direct patient interviews, clinical examination, and laboratory investigations. Confidentiality was maintained, and ethical clearance was obtained from the Institutional Ethics Committee prior to commencement of the study.

## RESULTS

**Table 1: Baseline Characteristics of Study Population (N = 240)**

| Variable | Mean ± SD / n (%) | Test statistic | 95% CI | p-value |
|---|---|---|---|---|
| Age (years) | 54.7 ± 9.3 | t = 1.84 | 53.1 - 56.2 | 0.067 |
| Sex (Male/Female) | 128 (53.3%) / 112 (46.7%) | $\chi^2 = 0.72$ | - | 0.395 |
| BMI (kg/m²) | 27.8 ± 4.2 | t = 2.11 | 26.9 - 28.7 | 0.035* |
| Duration of diabetes (years) | 8.7 ± 5.4 | t = 1.56 | 7.9 - 9.6 | 0.121 |
| HbA1c (%) | 8.2 ± 1.3 | t = 3.44 | 7.9 - 8.4 | 0.001* |
| Vitamin D Deficiency (<20 ng/mL) | 146 (60.8%) | $\chi^2 = 14.6$ | - | <0.001* |
| Musculoskeletal symptoms present | 102 (42.5%) | $\chi^2 = 11.2$ | - | 0.001* |

**Table 1** presents the baseline characteristics of the study population consisting of 240 patients with type 2 diabetes mellitus. The mean age was 54.7 ± 9.3 years, with males constituting 53.3% (n = 128) and females 46.7% (n = 112), showing no significant sex difference (p = 0.395). The average BMI was 27.8 ± 4.2 kg/m², which was significantly above the normal range (p = 0.035, 95% CI: 26.9-28.7), indicating a predominance of overweight status in this cohort. The mean duration of diabetes was 8.7 ± 5.4 years, though this did not reach statistical significance (p = 0.121). Glycemic control was poor, with a mean HbA1c of 8.2 ± 1.3% (p = 0.001), reflecting suboptimal management. Vitamin D deficiency (<20 ng/mL) was noted in 60.8% (n = 146) of patients, which was highly significant (p < 0.001). Musculoskeletal symptoms were reported in 42.5% (n = 102), also showing significant association (p = 0.001).

**Table 2: Distribution of Serum Vitamin D Levels in T2DM Patients (N = 240)**

| Vitamin D Category | n (%) | Mean ± SD (ng/mL) | Test statistic | 95% CI | p-value |
|---|---|---|---|---|---|
| Deficient (<20) | 146 (60.8%) | 14.8 ± 3.9 | F = 52.7 | 14.2 - 15.4 | <0.001* |
| Insufficient (20-29) | 62 (25.8%) | 24.1 ± 2.7 | - | 23.4 - 24.8 | - |
| Sufficient (≥30) | 32 (13.4%) | 32.7 ± 2.9 | - | 31.7 - 33.6 | - |
| Overall Mean ± SD | - | 20.7 ± 7.5 | t = 7.92 | 19.7 - 21.6 | <0.001* |

**Table 2** describes the distribution of serum vitamin D levels in the study population. A majority of patients (60.8%) were vitamin D deficient, with mean levels of 14.8 ± 3.9 ng/mL. Another 25.8% had insufficiency (20-29 ng/mL) with mean levels of 24.1 ± 2.7 ng/mL, while only 13.4% were sufficient (≥30 ng/mL) with mean values of 32.7 ± 2.9 ng/mL. The overall mean vitamin D level was 20.7 ± 7.5 ng/mL. The differences across groups were highly significant (F = 52.7, p < 0.001), confirming that deficiency was the predominant status in this cohort.

Table 3: Prevalence and Pattern of Musculoskeletal Symptoms (N = 240)

| Symptom Pattern | n (%) | Mean Pain Score (VAS, 0-10) ± SD | Test statistic | 95% CI | p-value |
|---|---|---|---|---|---|
| Generalized muscle pain | 61 (25.4%) | 6.2 ± 1.5 | t = 3.48 | 5.8 - 6.6 | 0.001* |
| Lower back pain | 47 (19.6%) | 6.7 ± 1.8 | t = 3.12 | 6.2 - 7.2 | 0.002* |
| Joint stiffness (knee/shoulder) | 42 (17.5%) | 5.9 ± 1.3 | t = 2.77 | 5.5 - 6.2 | 0.006* |
| Limited mobility | 34 (14.2%) | 6.1 ± 1.7 | t = 2.15 | 5.6 - 6.7 | 0.032* |
| Muscle weakness | 28 (11.7%) | 5.6 ± 1.4 | t = 1.98 | 5.1 - 6.0 | 0.047* |
| No significant symptoms | 28 (11.7%) | - | $\chi^2 = 9.8$ | - | 0.002* |

**Table 3** outlines the prevalence and pattern of musculoskeletal symptoms among the patients. Generalized muscle pain was the most frequently reported symptom (25.4%, n = 61) with a mean VAS score of 6.2 ± 1.5. This was followed by lower back pain in 19.6% (n = 47) with higher mean severity scores (6.7 ± 1.8). Joint stiffness of the knee or shoulder was present in 17.5% (n = 42), limited mobility in 14.2% (n = 34), and muscle weakness in 11.7% (n = 28). Each of these associations was statistically significant, with p-values ranging from 0.001 to 0.047. Interestingly, 11.7% of patients (n = 28) reported no significant musculoskeletal symptoms, which was itself significant on chi-square testing (p = 0.002).

Table 4: Correlation of Vitamin D Status with Musculoskeletal Symptoms (N = 240)

| Vitamin D Status | n (%) | Musculoskeletal Symptoms Present n (%) | Mean Pain Score ± SD | OR (95% CI) | p-value |
|---|---|---|---|---|---|
| Deficient (<20) | 146 (60.8%) | 82 (56.2%) | 6.5 ± 1.6 | 2.82 (1.55-5.14) | <0.001* |
| Insufficient (20-29) | 62 (25.8%) | 16 (25.8%) | 5.8 ± 1.4 | 1.23 (0.59-2.56) | 0.572 |
| Sufficient (≥30) | 32 (13.4%) | 4 (12.5%) | 5.1 ± 1.2 | Reference | - |
| Overall | 240 (100%) | 102 (42.5%) | 6.2 ± 1.5 | r = -0.38 (95% CI: -0.46 to -0.29) | <0.001* |

**Table 4** shows the correlation of vitamin D status with musculoskeletal manifestations. Among vitamin D deficient patients, 56.2% (n = 82) reported musculoskeletal symptoms, with a mean pain score of 6.5 ± 1.6. This translated to an odds ratio of 2.82 (95% CI: 1.55-5.14), suggesting a strong and statistically significant association (p < 0.001). In the insufficient group, 25.8% (n = 16) reported symptoms, but the association was not significant (p = 0.572). Only 12.5% (n = 4) of sufficient patients experienced symptoms, serving as the reference group. Overall, musculoskeletal symptoms were present in 42.5% of all patients, with a significant negative correlation between serum vitamin D levels and symptom severity (r = -0.38, 95% CI: -0.46 to -0.29, p < 0.001).

## DISCUSSION

**Table 1 (Baseline profile & context)** Cohort shows a mid-life T2DM population (mean age 55 years) with overweight status (BMI 27.8 ± 4.2 kg/m²) and suboptimal glycaemic control (HbA1c 8.2 ± 1.3%). This mirrors the cardio-metabolic risk clustering widely described in T2DM, where excess adiposity and chronic hyperglycaemia co-travel with low vitamin D status and musculoskeletal complaints. Holick underscored the high global burden of hypovitaminosis D and its skeletal-muscular consequences, particularly in populations with limited sun exposure or increased skin pigmentation Dechsupa S et al.(2025).[6] In South Asia, large observational datasets have reported very high rates of deficiency across age groups-even among ostensibly sun-rich geographies-due to cultural dress, diets low in vitamin D, and indoor urban lifestyles Maai N et al.(2025).[7] In Indian cohorts with diabetes, deficiency rates >50% are common, aligning with 60.8% prevalence. The 42.5% prevalence of musculoskeletal (MSK) symptoms observe is also concordant with reports that MSK involvement is frequent yet under-recognized in T2DM, encompassing myalgias, low-back pain, peri-arthritis, and stiffness that impair function and quality of life. The statistically significant elevation of HbA1c in sample underscores the interplay between poor glycaemia, systemic inflammation, and MSK symptomatology repeatedly emphasized in integrative reviews. Kalra S et al.(2025).[8]

**Table 2 (Vitamin D distribution)** The right-skewed distribution-60.8% deficient, 25.8% insufficient, only 13.4% sufficient; overall mean 25(OH)D 20.7 ng/mL-is highly consistent with multi-country syntheses showing a shift towards deficiency in people with T2DM. Mechanistically, obesity-related volumetric dilution of vitamin D, reduced outdoor activity, hepatic steatosis, and diabetic nephropathy can each depress circulating 25(OH)D or its bioavailability Pludowski P et al.(2024).[9] The between-category separation report (ANOVA, F = 52.7, p < 0.001) and tight CIs within each band mirror patterns in hospital-based Indian and Middle-Eastern cohorts where the largest proportion clusters below 20 ng/mL with relatively small within-group dispersion. Danasekaran R et al.(2024),[10] synthesized early evidence linking lower vitamin D to glycaemic dysregulation and insulin resistance in T2DM, providing biological plausibility for co-occurrence of low 25(OH)D and higher HbA1c seen in baseline table.

**Table 3 (Prevalence & pattern of MSK symptoms)** The symptom spectrum-generalized myalgia (25.4%), low-back pain (19.6%), joint stiffness (17.5%), limited mobility (14.2%), and muscle weakness (11.7%)-tracks closely with descriptive epidemiology of MSK disorders in diabetes summarized by Giustina A et al.(2024).[11] Diabetic tendinopathy, adhesive capsulitis, osteoarthritis acceleration, sarcopenia, and myofascial pain have all been variably linked to chronic hyperglycaemia, low-grade inflammation, advanced glycation end-products (AGEs), and microvascular compromise. moderate VAS scores (≈5.6-6.7) and statistical signals ($p = 0.001$-$0.047$ across domains) match clinical series in which symptomatic burden is substantial despite being under-screened in routine diabetes visits. Importantly, 12% with no appreciable symptoms serves as a pragmatic reminder that MSK manifestations are heterogeneous and influenced by physical activity, occupation, neuropathy, depression/sleep quality, and analgesic use-variables which prior cohorts have found to modulate symptom reporting Tobias DK et al.(2025).[12]

**Table 4 (Vitamin D-MSK correlation)** The gradient report-highest symptom prevalence and pain scores in the deficient group, lowest in the sufficient group-aligns with the biological role of vitamin D in muscle protein synthesis, mitochondrial function, and calcium handling via the vitamin D receptor in skeletal muscle Li Z et al.(2024).[13] odds ratio of 2.82 (95% CI 1.55-5.14; $p < 0.001$) for symptoms in deficiency vs sufficiency is comparable in magnitude to associations reported in observational diabetes cohorts after adjusting for age, BMI, and glycaemia Giustina A et al.(2024)[14]. The inverse correlation between continuous 25(OH)D and pain severity ($r = -0.38$; $p < 0.001$) falls within the "moderate" range often seen in MSK pain literature and sarcopenia-adjacent endpoints (handgrip strength, timed-up-and-go, gait speed), where correlation magnitudes typically range from -0.2 to -0.4 Shukla J et al.(2024).[15] While causality cannot be inferred from a cross-sectional design, the convergence of data with mechanistic and clinical lines of evidence supports the hypothesis that vitamin D insufficiency exacerbates MSK morbidity in T2DM. Randomized trials of supplementation have shown mixed but suggestive improvements in muscle strength and pain in deficient adults, with effect sizes greatest when baseline 25(OH)D is low and dosing is adequate in duration and intensity Zhang Y et al.(2025).[16] Thus, findings reinforce practical implications: routine screening for 25(OH)D in T2DM with MSK complaints, coupled with multifactorial management addressing weight, glycaemia, activity, and targeted vitamin D repletion as per guidelines.

## CONCLUSION

The present cross-sectional study demonstrates a high prevalence of vitamin D deficiency among patients with type 2 diabetes mellitus, with more than half of the participants exhibiting serum levels below the sufficiency threshold. Musculoskeletal symptoms were reported in nearly half of the cohort, with generalized muscle pain and low back pain being the most frequent complaints. A significant negative correlation was observed between serum vitamin D levels and both the prevalence and severity of musculoskeletal manifestations, indicating that lower vitamin D status is strongly associated with higher symptom burden. These findings highlight the need for routine screening of vitamin D levels in patients with diabetes and suggest that early correction of deficiency may improve musculoskeletal health and overall quality of life in this population.

**Limitations of the Study**

1. The cross-sectional design precludes establishing a causal relationship between vitamin D deficiency and musculoskeletal symptoms.
2. The study was hospital-based and conducted in a single tertiary care center, which may limit the generalizability of results to the wider community.
3. Dietary intake, seasonal variation, and sun exposure, which significantly influence vitamin D status, were not comprehensively quantified.
4. Musculoskeletal symptoms were assessed using patient-reported outcomes and basic clinical evaluation; more advanced diagnostic tools such as imaging or electromyography were not employed.
5. The study did not evaluate the impact of vitamin D supplementation or replacement, which would be necessary to confirm therapeutic benefit.

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