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Association of vitamin D deficiency and supplementation with clinical outcomes in multi-tendon chronic tendinopathy: a retrospective clinical study

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Abstract

Background:

Tendinopathy is a common musculoskeletal condition associated with persistent pain and functional limitation. Although vitamin D deficiency has been widely implicated in musculoskeletal disorders, its association with clinical outcomes in tendinopathy remains incompletely understood. This study investigated the association between serum vitamin D levels, clinical severity of tendinopathy, and the effects of vitamin D supplementation.

Methods:

This retrospective observational study included 350 patients with tendinopathy treated at a

tertiary referral center between 2023 and 2025. Patients were classified into a vitamin D supplementation group (n = 221), receiving 50,000 IU weekly for 4 weeks followed by 2,000 IU daily for 8 weeks, and a nonsupplemented group (n = 129). Pain intensity (VAS), functional status (0–100 scale), disease duration, chronicity, and ultrasonographic inflammation severity were evaluated.

Results:

Patients with serum vitamin D levels below 20 ng/mL exhibited significantly higher pain scores (7.3 ± 1.2 vs. 5.7 ± 1.3, $p < 0.001$), longer disease duration (8.2 vs. 4.1 months, $p < 0.001$), and a higher prevalence of chronic tendinopathy (79.6% vs. 24.0%, $p < 0.001$). Following supplementation, serum vitamin D levels increased from 14.2 to 38.4 ng/mL ($p < 0.001$), accompanied by significant reductions in pain and improvements in functional status compared with the nonsupplemented group ($p < 0.001$). Severe vitamin D deficiency (<10 ng/mL) was independently associated with a sevenfold increased likelihood of severe and chronic tendinopathy (OR: 7.2, $p < 0.001$).

Conclusions:

Vitamin D deficiency is strongly associated with greater clinical severity and chronicity of tendinopathy. Vitamin D supplementation is associated with meaningful improvements in pain and functional outcomes, suggesting that vitamin D status represents a clinically relevant and potentially modifiable factor in tendinopathy management.

Keywords:

Tendinopathy, Vitamin D deficiency, Pain, Functional outcome, Supplementation

Background

Tendinopathies affect millions of people worldwide as a common musculoskeletal condition. They account for nearly 30% of all musculoskeletal consultations and represent a major cause of pain and disability among active adults. The extent of this problem becomes evident, because tendon-related procedures number more than 30 million annually [1]. The economic

impact of this condition results from both medical expenses and employee time lost at work. The rotator cuff together with the Achilles tendon and patellar tendon experiences the highest number of tendon injuries. Sport-related overuse injuries result in approximately half of all tendon problems [1]. The treatment of tendons faces difficulties because of their distinct biological characteristics. The restricted blood flow and intricate mechanical requirements of tendons result in inadequate recovery from injuries [2]. Surgical interventions for tendon repair do not always produce successful results. The failure rates for tendon-to-bone healing after rotator cuff surgery span between 20% and 94% [3].

Given these high recurrence and failure rates, researchers have sought biological factors that may influence tendon homeostasis, inflammation, and healing capacity. The world population exceeds one billion people who have insufficient vitamin D levels according to recent research [2,4]. Research indicates that vitamin D maintains essential functions for bone strength, neuromuscular coordination, and muscle function. In addition to its skeletal role, vitamin D has been shown to influence collagen synthesis, tenocyte proliferation, and inflammatory modulation through its receptors expressed in tendon tissue.

Research indicates that between 8.3% and 71% of patients with rotator cuff injuries have vitamin D levels below normal [5]. The continuous mechanical stress that tendons experience has led researchers to investigate how vitamin D affects their health [6]. However, most published studies have focused exclusively on the rotator cuff, while data concerning other tendon sites (such as the patellar and flexor tendons) remain limited.

Research indicates vitamin D supports tendon healing, but scientists need to fill existing knowledge gaps. The majority of research conducted about rotator cuff repair has been published. The current understanding of vitamin D effects on Achilles tendons and patellar tendons and flexor tendons remains insufficient [2]. The scientific community has not established the exact mechanism by which vitamin D deficiency leads to tendinopathy development. The optimal method for vitamin D supplementation remains unknown to medical professionals [5]. The reviews about vitamin and amino acid effects on musculoskeletal healing

identify multiple methodological problems. The high risk of bias exists in most human studies conducted about this topic [7]. The lack of clear evidence makes it difficult to verify biological effects.

Furthermore, while animal studies have demonstrated that vitamin D may exert immunomodulatory and anti-inflammatory effects beneficial for tendon repair, translation of these findings to human tissue remains inconsistent. There is still uncertainty about optimal serum thresholds, supplementation regimens, and their clinical relevance in tendinopathy.

Research conducted with animals demonstrates that vitamin D exerts immunomodulatory effects which benefit tendon repair processes. The results from animal studies do not consistently translate to human tendon–bone healing processes [3]. Research evidence demonstrates that vitamin D supports muscle tissue repair and regeneration processes. The medical community remains unsure about vitamin D's effectiveness for treating particular injuries following surgical procedures [8,9].

Accordingly, the present study was designed to address two main objectives: to evaluate the association between baseline serum vitamin D levels and clinical manifestations of tendinopathy (pain, functional limitation, and chronicity); and to assess the impact of vitamin D supplementation on pain reduction and functional recovery in affected patients.

We hypothesized that lower vitamin D levels would be associated with higher pain intensity, prolonged disease duration, and poorer functional outcomes.

Methods

Study population and sample

The study was conducted at a single tertiary referral hospital as a retrospective observational cohort study based on medical record review from January 2023 through June 2025. This retrospective observational cohort study was based exclusively on review of existing medical

records, and no additional clinical examinations, laboratory tests, or imaging procedures were performed specifically for research purposes. The research included 350 patients with tendinopathy who were between 21 and 55 years with an average age of 38.4 years. These 350 patients represent all consecutive patients diagnosed with tendinopathy during the study period who met the inclusion criteria and had complete baseline clinical, laboratory, and imaging data available in the medical records. The study included patients who received tendinopathy diagnosis through medical history and physical examination and ultrasound confirmation and had available vitamin D test results. Tendinopathy diagnosis was based on the presence of activity-related pain, localized tendon tenderness, functional limitation on physical examination, and ultrasonographic findings, including tendon thickening, hypoechogenicity, loss of fibrillar pattern, or neovascularization. Rotator cuff, Achilles, patellar, and lateral epicondyle tendinopathies were diagnosed using site-specific clinical tests and corresponding ultrasonographic confirmation. The term “multi-tendon” refers to the inclusion of different tendon locations across the study population rather than simultaneous involvement of multiple tendons in the same individual [10]. The study excluded patients who developed tendinopathy because of systemic inflammatory diseases or recent physical injuries. Patients who had received corticosteroid injections, structured physiotherapy programs, or other invasive treatments for tendinopathy within the preceding 3 months were also excluded to minimize treatment-related confounding.

Patients with metabolic disorders (such as diabetes mellitus or thyroid dysfunction), chronic kidney or liver disease, or who had used corticosteroids or vitamin D supplements in the previous 6 months were also excluded to minimize confounding effects.

The study defined vitamin D deficiency as levels below 20 ng/mL and severe deficiency as levels below 10 ng/mL, while normal levels exceeded 30 ng/mL. The study used a visual analog scale (VAS) from 0 to 10 to measure pain intensity, and functional status was assessed using a standardized 0–100 composite functional score derived from routine clinical evaluation

forms used in the department, with higher scores indicating better functional performance. Both scales have previously demonstrated high reliability and validity in musculoskeletal research.

Study procedures

The researchers obtained data through medical records review to collect information about patient demographics, clinical evaluations, and laboratory test results. The hospital laboratory measured serum 25-hydroxyvitamin D levels using standardized automated chemiluminescence immunoassays in ng/mL, with all samples analyzed in the same laboratory to minimize interassay variability. Baseline serum vitamin D measurements and ultrasonographic assessments were performed at the time of initial clinical evaluation prior to any supplementation [11].

The radiologists who performed ultrasound examinations used established imaging criteria to evaluate inflammation severity which they classified as mild–moderate or severe. Ultrasonographic evaluation was performed only at the clinically symptomatic tendon site corresponding to the patient's presenting complaint. No routine screening of asymptomatic tendons or multiple anatomical regions was performed. Due to limited sample sizes within individual tendon subgroups, analyses were performed by pooling different tendinopathy sites. Tendinopathy type was included as a covariate in multivariate analyses to account for potential site-related differences. Severe inflammation was defined as marked tendon thickening, diffuse hypoechogenicity, partial tears, or extensive neovascularization, whereas mild–moderate inflammation included focal hypoechogenicity or limited tendon thickening without structural disruption. All examinations were performed by experienced musculoskeletal radiologists following standardized institutional protocols [12]. The patients received 12 weeks of follow-up care during which their clinical parameters received reassessment at the conclusion of the period. During follow-up, all patients received standard conservative management, including activity modification and oral nonsteroidal anti-inflammatory drugs as

needed. No structured physiotherapy protocols or invasive procedures were initiated during the follow-up period, and treatment strategies did not differ between groups except for vitamin D supplementation. Completion of the 12-week follow-up was required for inclusion in the final analysis. Patients with missing or incomplete follow-up data were excluded; no eligible patients were excluded due to missing follow-up during the study period. The research team validated the reliability of VAS and functional score assessment tools through previous research studies.

No additional imaging or invasive procedures were performed as part of this study. The primary endpoints were pain reduction and functional improvement at 12 weeks, while secondary endpoints included disease duration, chronicity risk, and ultrasonographic inflammation severity. Chronic tendinopathy was defined as symptom duration of 6 months or longer. Chronicity status was determined based on symptom duration prior to study inclusion and before initiation of the 12-week follow-up period. Patients were categorized as having high chronicity risk if symptoms persisted for ≥6 months and low chronicity risk if symptom duration was <6 months.

The research team did not perform any additional follow-up assessments after the 12-week period for this specific analysis.

The researchers divided participants into two groups which received either high-dose vitamin D supplements group (n=221) or nonsupplemented group (n=129).

Because this study was retrospective, the grouping was based on medical records showing whether patients had been prescribed vitamin D supplementation as part of their standard clinical management.

The study design as a retrospective analysis prevented randomization, so researchers used clinical file entries to determine group assignments [13]. The intervention group received 50,000 IU vitamin D supplements weekly for 4 weeks, followed by 2,000 IU daily supplements for 8 weeks to reach vitamin D levels above 30 ng/mL. Vitamin D supplementation was

prescribed according to routine clinical practice for patients with documented vitamin D deficiency and was not determined by study-specific allocation.

This dosage regimen followed the Endocrine Society's clinical practice guideline for vitamin D deficiency treatment. The formulation used was cholecalciferol (vitamin D3) in oral capsule form [13].

The researchers tracked patient compliance through medical records by monitoring their adherence to prescribed doses at more than 80% of the total amount. Adherence was assessed through prescription refill records and follow-up clinical notes documenting patient-reported compliance. Patients with incomplete adherence data were classified as nonadherent. The nonsupplemented group received standard medical care without any supplementation, while the study lacked blinding procedures because of its design constraints. The study did not require any surgical or invasive procedures.

Statistical analysis

Sample size estimation was performed using G*Power version 3.1.9.4 (Heinrich Heine University, Düsseldorf, Germany). Based on the primary outcome of pain intensity (VAS), an expected medium effect size (Cohen's d = 0.4), alpha level of 0.05, and statistical power of 0.80, the minimum required sample size was calculated as 260 participants. Considering potential missing data, a total sample size of 350 patients was deemed sufficient. The researchers conducted all statistical calculations using IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). The study presented descriptive data through mean values with standard deviations (SD) for numerical variables, including age, BMI, vitamin D levels, VAS scores, and functional scores and through percentage values for sex distribution, tendinopathy types, vitamin D categories, inflammation severity, and chronicity risk.

The research evaluated vitamin D levels against clinical results through independent *t* tests for VAS scores and functional scores and Mann–Whitney *U* tests for disease duration and chi-

square tests for risk of chronicity and inflammation severity [9]. The researchers used one-way ANOVA to evaluate BMI differences between groups. The researchers performed multivariate logistic regression to find tendinopathy risk factors which included vitamin D deficiency, age, sex, and BMI while reporting odds ratios (OR) and 95% confidence intervals (CI). To reduce confounding by indication, multivariate regression models were adjusted for baseline age, sex, BMI, baseline vitamin D level, pain intensity, functional score, and disease duration [10].

All variables with a p value < 0.10 in univariate analysis were entered into the multivariate model. Model fit was assessed using the Hosmer–Lemeshow test and model performance was quantified by the pseudo R^2 value.

The researchers used *t* tests and chi-square tests to evaluate which factors affected treatment outcomes based on baseline and post-treatment vitamin D levels and disease duration and treatment adherence. The researchers presented all p values with two decimal places except when values were less than 0.01, because they used three decimal places (e.g., P = 0.001) and values below 0.001 received the notation P < 0.001.

A significance level of p < 0.05 was considered statistically significant for all tests.

As the study used retrospective data, patient consent was obtained at the time of initial hospital admission for the use of anonymized clinical records in research. This consent procedure is routinely applied at hospital admission and does not represent study-specific prospective enrollment.

The research team maintained patient privacy through data encryption and coding methods which also protected personal information. The research followed Helsinki Declaration principles and Good Clinical Practice standards.

Results

Study population characteristics

The research included 350 patients who received tendinopathy confirmation. The study participants averaged 38.4 years, while their ages spanned between 21 and 55 years. The study included 53.1% women who participated more than men. The majority of participants were overweight, because their average BMI reached 26.2 kg/m². The researchers discovered unacceptably low vitamin D levels throughout the entire study population. The participants' average vitamin D level measured at 18.2 ng/mL which is significantly lower than the recommended threshold for health optimization. The study results showed that less than 10% of patients maintained vitamin D levels above 30 ng/mL, while the rest displayed different levels of deficiency. The severe deficiency category which defined vitamin D levels below 10 ng/mL affected more than 20% of all participants. The study found rotator cuff tendinopathy as the leading condition, while Achilles, patellar, and lateral epicondylitis occurred at similar rates. The patients presented with severe pain when they first arrived which they rated at 6.8 on the visual analog scale (Table 1).

Table 1. Demographic and clinical characteristics of participants

Parameter	Value	n (%)
Age (years)		
• Mean ± SD	38.4 ± 8.2	
• Median (IQR)	38 (32–44)	
• Min–Max	21–55	
Sex		
• Female		186 (53.1)
• Male		164 (46.9)
BMI (kg/m²)		
• Mean ± SD	26.2 ± 4.1	
• Median (IQR)	26.1 (23.4–28.9)	
Baseline Vitamin D (ng/mL)		
• Mean ± SD	18.2 ± 9.8	
• Median (IQR)	16.4 (11.2–24.8)	

Vitamin D Classification	
• Severe deficiency (<10 ng/mL)	74 (21.1)
• Deficient (10–20 ng/mL)	147 (42.0)
• Insufficient (20–30 ng/mL)	91 (26.0)
• Normal (>30 ng/mL)	38 (10.9)
Type of Tendinopathy	
• Rotator cuff	98 (28.0)
• Achilles	91 (26.0)
• Patellar	84 (24.0)
• Lateral epicondylitis	77 (22.0)
Pain intensity (VAS 0–10)	
• Mean ± SD	6.8 ± 1.4

Association between vitamin D deficiency and clinical severity

The analysis of patients according to their vitamin D levels revealed substantial differences between their clinical results. Patients who had vitamin D levels below 20 ng/mL reported higher pain intensity than those with better vitamin D levels according to their pain scale ratings which averaged 7.3 vs. 5.7. The length of time patients experienced their symptoms showed significant differences between these two groups. Median disease duration prior to study inclusion was significantly longer in patients with insufficient vitamin D levels (8 months) compared to those with sufficient levels (4 months). The assessment results for functional ability followed the same pattern as other measurements. The deficient group achieved 42.3 points on functional assessments but patients with adequate vitamin D levels reached 61.4 points. The development of chronic tendinopathy presented the most critical concern. The risk assessment for chronic tendinopathy revealed that 80% of patients with vitamin D deficiency would develop chronic tendinopathy but this risk decreased to 24% for patients with sufficient vitamin D levels. The ultrasound results supported clinical findings by showing that deficient

patients had severe inflammation in 65% of cases but patients with sufficient vitamin D levels had inflammation in less than 20% of cases (Table 2).

Table 2. Relationship between vitamin D level and tendinopathy parameters

Parameter	Vitamin D <20 ng/mL (n = 221)	Vitamin D ≥20 ng/mL (n = 129)	*p* value
Pain intensity (VAS)	7.3 ± 1.2	5.7 ± 1.3	<0.001*
Disease duration (months)	8.2 (5.4–12.1)	4.1 (2.8–6.3)	<0.001*
Functional score (0–100)	42.3 ± 11.2	61.4 ± 13.8	<0.001*
Risk of chronicity, n (%)			<0.001*
• High	176 (79.6)	31 (24.0)	
• Low	45 (20.4)	98 (76.0)	
Inflammation on ultrasonography			<0.001*
• Severe	142 (64.3)	23 (17.8)	
• Mild–moderate	79 (35.7)	106 (82.2)	

Statistical significance set at p < 0.05. VAS: Visual Analog Scale; BMI: Body Mass Index; IQR: Interquartile Range; SD: Standard Deviation; USG: Ultrasonography.

Treatment response and vitamin D supplementation

The intervention phase produced significant findings about vitamin D supplementation as a therapeutic approach. The intervention group reached normal vitamin D levels after 12 weeks of high-dose vitamin D supplementation, which increased their levels from 14.2 ng/mL to 38.4 ng/mL. No significant change in serum vitamin D levels was observed in the nonsupplemented group. The treatment brought about significant medical advancements. The treatment group achieved at least 50% pain score reduction in four out of five patients, but the nonsupplemented group achieved this level of pain reduction in only one out of five

patients. The treatment group achieved complete recovery at a rate of 60.6%, which was four times higher than the 14.7% recovery rate in the nonsupplemented group. The treatment group showed better functional results, because more than 80% of patients achieved at least 30 points of improvement in their functional assessments, while controls reached this benchmark at less than 25% (Table 3).

Table 3. Improvement following vitamin D treatment (12-week follow-up)

Parameters	High-dose vitamin D group (n = 221)	Nonsupplemented group (n = 129)	*p* value
Baseline vitamin D (ng/mL)	14.2 ± 4.8	28.4 ± 5.1	—
12th week vitamin D (ng/mL)	38.4 ± 6.2	27.9 ± 5.3	<0.001*
VAS score reduction ≥50%	177 (80.1%)	26 (20.2%)	<0.001*
— No improvement	44 (19.9%)	103 (79.8%)	—
Complete recovery	134 (60.6%)	19 (14.7%)	<0.001*
— No complete recovery	87 (39.4%)	110 (85.3%)	—
Functional improvement (≥30-point increase)	181 (81.9%)	32 (24.8%)	<0.001*
—No functional improvement	40 (18.1%)	97 (75.2%)	—

Note: Data are presented as *mean* ± *standard deviation* or *n (%)* as appropriate. *VAS* = Visual Analog Scale. Statistical significance was set at $p < 0.05$.

Predictors of severe and chronic tendinopathy

Multivariate logistic regression analysis identified severe vitamin D deficiency as the strongest independent factor associated with severe and chronic tendinopathy. Patients with serum vitamin D levels below 10 ng/mL were seven times more likely to develop severe and chronic tendinopathy compared to those with sufficient vitamin D levels (OR: 7.2, $p < 0.001$). Similarly,

patients with vitamin D levels between 10 and 20 ng/mL exhibited a four-fold increased likelihood of severe and chronic tendinopathy (OR: 4.8, p < 0.001). After adjustment for potential confounders, including age, sex, and BMI, none of these demographic or anthropometric variables showed a statistically significant association with tendinopathy severity. The predictive model demonstrated good discriminative capacity, correctly classifying more than 75% of cases (Fig. 1).

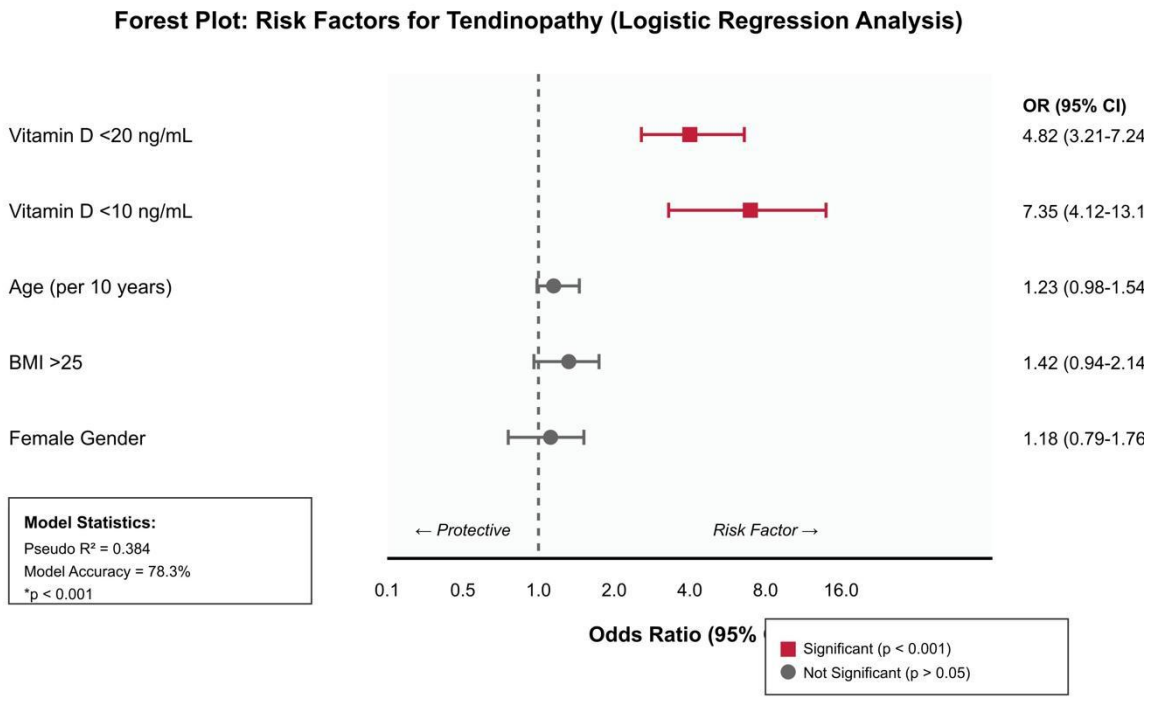

Figure 1. Forest plot of factors associated with severe and chronic tendinopathy from multivariate logistic regression analysis (n = 350). Red squares indicate statistically significant risk factors (p<0.001); gray circles indicate nonsignificant factors (p>0.05). The vertical dashed line represents the null effect (OR=1.0). Horizontal lines represent 95% confidence intervals. OR: Odds Ratio; CI: Confidence Interval; BMI: Body Mass Index

Factors influencing treatment success

The patients who received vitamin D supplements showed different levels of response to treatment, while specific elements determined their treatment outcomes. The treatment success depended on patients' initial vitamin D blood levels, because responders began with lower levels than nonresponders. The treatment success rate depended on post-treatment vitamin D levels, because responders achieved 40 ng/mL but nonresponders only reached 32 ng/mL. The success of treatment depended heavily on when patients received their treatment. The treatment success rate reached more than 80% when patients received their first dose within 6 months after symptom appearance, but the success rate decreased to 27% when symptoms lasted longer. The success of treatment depended heavily on how well patients followed their prescribed vitamin D supplement regimen. The 95% of patients who achieved success in treatment followed their supplement plan correctly by taking more than 80% of their prescribed doses, but nonresponders showed poor adherence at a rate of almost 50% (Table 4).

Table 4. Factors affecting treatment response

Factor	**Responders (n = 177)**	**Nonresponders (n = 44)**	***p* value**
Baseline Vitamin D (ng/mL)			<0.001*
• **Mean ± SD**	12.8 ± 3.9	19.4 ± 4.2	
Post-treatment Vitamin D (ng/mL)			<0.001*
• **Mean ± SD**	39.8 ± 5.4	32.1 ± 6.8	
Disease duration (months)			<0.001*
• **<6 months**	142 (80.2%)	12 (27.3%)	
• **≥6 months**	35 (19.8%)	32 (72.7%)	
Treatment adherence (%)			<0.001*

• **>80%**	168 (94.9%)	24 (54.5%)
• **≤80%**	9 (5.1%)	20 (45.5%)

Statistical significance set at p < 0.05. SD: Standard Deviation.

No serious adverse events related to vitamin D supplementation were observed during the 12-week follow-up period.

Discussion

This study investigated how subclinical vitamin D deficiency affects clinical outcomes in patients with tendinopathy and evaluated the impact of vitamin D supplementation. The research established that patients with insufficient vitamin D levels experience increased pain symptoms, decreased functional ability, and longer disease duration. The research findings demonstrate vitamin D's importance for tendon health and its potential to help treat tendinopathy patients.

This large-scale retrospective study provides new clinical evidence supporting the role of vitamin D as a modifiable systemic factor influencing both the development and prognosis of tendinopathy. Our findings expand current understanding by simultaneously evaluating baseline deficiency, supplementation response, and multi-tendon involvement within the same patient cohort.

The research revealed that patients with vitamin D levels below 20 ng/mL experienced higher pain scores (VAS: 7.3 vs. 5.7, $p<0.001$) and longer disease duration (8.2 vs. 4.1 months, $p<0.001$) and lower functional scores (42.3 vs. 61.4, $p<0.001$) and higher chronicity risk (79.6% vs. 24.0%, $p<0.001$) and more severe inflammation (64.3% vs. 17.8%, $p<0.001$). The research indicates that vitamin D deficiency could lead to worse tendinopathy results.

These results align with previous findings suggesting that low vitamin D levels impair tendon matrix turnover, reduce collagen synthesis, and exacerbate local inflammatory activity [6,14].

The study by Ammerman et al. discovered that 65.7% of young women had low vitamin D levels and 54.6% had muscle/tendon injury-related deficiency [14]. The research did not investigate how vitamin D deficiency affects inflammation or functional decline. Tarantino et al. discovered that vitamin D deficiency causes tendinopathy and disrupts collagen production [6]. The study by Khatri et al. demonstrated that vitamin D deficiency raises the risk of distal biceps tendinopathy in men by 2.81 times and in women by 1.69 times [10]. Bouchard et al. conducted a review which demonstrated that vitamin D deficiency leads to rotator cuff healing functional deficits but the evidence for other tendons remains scarce [2]. The research supports previous findings about vitamin D deficiency's negative effects on inflammation and functional results by presenting detailed data from multiple tendon types. Subgroup analyses according to individual tendon types (e.g., rotator cuff, Achilles, patellar, and lateral epicondyle) were not performed due to limited statistical power within each subgroup. Therefore, the present results should be interpreted as a global multi-tendon association rather than tendon-specific effects.

This multidimensional approach strengthens the external validity of the findings, as it reflects real-world variation in tendinopathy etiology and anatomical distribution.

This study demonstrated that taking high doses of vitamin D (n=221) resulted in significant vitamin D level increases from 14.2 ng/mL to 38.4 ng/mL during 12 weeks ($p<0.001$), while patients achieved better pain relief (≥50% VAS decrease: 80.1% vs. 20.2%, $p<0.001$) and complete recovery (60.6% vs. 14.7%, $p<0.001$) and functional improvement (≥30-point increase: 81.9% vs. 24.8%, $p<0.001$) than the nonsupplemented group (n=129).

This suggests that adequate restoration of serum vitamin D levels contributes not only to analgesic improvement but also to functional recovery, possibly via modulation of neuromuscular performance and tendon remodeling capacity.

Our findings indicate that vitamin D shows potential as an additional treatment for tendinopathy patients. Bouchard et al. demonstrated that vitamin D assists tendon recovery through its ability to control inflammation and extracellular matrix production, but their research focused on

rotator cuff repair only [2]. Qiu et al. discovered that tendinopathy patients who received nutritional supplements containing vitamin D experienced decreased pain levels (95% CI -1.37 to -0.10; $p<0.05$), although their functional results remained unclear [15]. Vitali et al. demonstrated that patients who received nutraceuticals combined with ESWT achieved better VAS and UCLA score results ($p=0.0002$) for various tendinopathies [16]. Kim et al. proved that vitamin D hydrogel application protected tendons from damage in laboratory tests and promoted tissue recovery in animal studies [17]. The research supports previous studies which demonstrate vitamin D effectiveness for pain management and functional improvement in different tendon conditions.

Collectively, these results highlight the biological plausibility of vitamin D as an adjuvant therapy for tendinopathy. Vitamin D may exert its beneficial effects through downregulation of pro-inflammatory cytokines (TNF-α and IL-6) and enhancement of type I collagen synthesis, leading to improved tendon regeneration and reduced pain perception.

The study demonstrated that patients with vitamin D levels below 10 ng/mL had a sevenfold higher likelihood of developing severe and chronic tendinopathy (OR: 7.2, $p<0.001$), while patients with levels between 10 and 20 ng/mL had a four-fold increased likelihood of severe and chronic tendinopathy (OR: 4.8, $p<0.001$) than patients with levels above 30 ng/mL. The study results showed no statistically significant association between age, sex, and BMI and the severity or chronicity of tendinopathy ($p > 0.05$). The research demonstrates that insufficient vitamin D levels create a major risk for developing tendinopathy.

This finding supports the hypothesis that systemic biochemical factors, rather than demographic or anthropometric variables, may play a dominant role in tendon vulnerability.

Yaka et al. discovered that patients with lateral epicondylitis had lower vitamin D levels than healthy participants (16.47±8.22 ng/mL vs. 23.64±8.4 ng/mL, $p<0.001$) and 77.5% of participants had vitamin D deficiency below 20 ng/mL [9]. Baart et al. conducted a study which failed to detect any relationship between vitamin D levels and lower limb tendinopathy in

runners, although mechanical factors seemed to play a more significant role [18]. Malliaras et al. identified load and biomechanical factors as essential risk elements for tendinopathy but they did not evaluate systemic factors, including vitamin D [19]. Kabore et al. identified multiple systemic factors which contribute to chronic tendinopathy development but they did not investigate vitamin D levels [20]. The research evidence supports vitamin D's potential as a tendon health promoter which should be considered for tendinopathy risk evaluation.

Thus, combining vitamin D assessment with conventional biomechanical and imaging parameters may improve early risk stratification and targeted prevention strategies in at-risk populations such as athletes or workers with repetitive strain.

The research findings demonstrated that participants who responded to vitamin D supplements started with lower vitamin D concentrations (12.8 ± 3.9 vs. 19.4 ± 4.2 ng/mL, $p<0.001$) and achieved higher post-treatment levels (39.8 ± 5.4 vs. 32.1 ± 6.8 ng/mL, $p<0.001$), and they had shorter disease duration (<6 months: 80.2% vs. 27.3%, $p<0.001$) and better treatment adherence (>80% dose: 94.9% vs. 54.5%, $p<0.001$).

These observations imply that early supplementation initiation and consistent adherence optimize therapeutic efficacy. This is consistent with the time-dependent remodeling phase of tendon healing, which is sensitive to metabolic and nutritional support.

The research indicates that patients who start treatment early and follow their treatment plan better will achieve better results from vitamin D therapy for tendinopathy. The study by Ali et al. demonstrated that patients who received vitamin D supplementation together with physiotherapy achieved better pain reduction than patients who received physiotherapy alone (slope = -1.126, $p=0.035$) [21]. The research by Chevalley et al. demonstrated that patients with low vitamin D levels at the beginning of treatment will achieve the most benefits from supplementation [22]. The research did not investigate disease duration or treatment compliance as this study did. The research by Mak demonstrated that SLE patients with low vitamin D levels experience deteriorating musculoskeletal symptoms, which indicates that

vitamin D plays a wider role in health. The research by Ali and Uddin established a link between vitamin D deficiency and musculoskeletal problems, but they did not evaluate the elements that influence possible results.

Collectively, these findings emphasize that vitamin D deficiency may represent both a risk marker and a modifiable therapeutic target in musculoskeletal rehabilitation.

The study contains two main limitations, because it uses a retrospective approach and does not track patients' treatment responses.

In addition, the absence of randomization and the reliance on medical records may have introduced selection and reporting bias. Serum vitamin D levels were measured only once, preventing assessment of seasonal variability or long-term maintenance. Seasonal variation, sunlight exposure, dietary calcium intake, and physical activity level were not systematically recorded and may have influenced baseline vitamin D levels. Therefore, residual confounding related to environmental and lifestyle factors cannot be excluded. The study also did not evaluate potential confounders, such as calcium intake, sun exposure, or physical activity level.

The research results demonstrate that early treatment initiation combined with proper medication compliance leads to the best outcomes, which could reduce its applicability to other cases. The study's main advantage comes from its large, diverse patient population and its strong statistical methods.

Future prospective and randomized studies should aim to confirm these associations and to identify optimal supplementation thresholds for tendon-specific recovery.

Conclusion

The findings of this study suggest that vitamin D deficiency is associated with more severe tendinopathy symptoms, while vitamin D supplementation is associated with better pain

management and functional improvement. Vitamin D deficiency should, therefore, be considered a clinically relevant and potentially modifiable factor in tendinopathy management.

The research findings demonstrate that healthcare providers should perform vitamin D tests and provide treatment to patients with tendinopathy as it may contribute to improved treatment outcomes.

Routine screening for vitamin D levels, particularly in patients with recurrent or chronic tendon pain, may contribute to earlier intervention, enhanced recovery, and improved quality of life.

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Ethics approval and consent to participate:

The study received ethical approval from the Harran University Clinical Research Ethics Committee on 28.04.2025 through approval number HRÜ/25.08.08. All participants in the study provided their consent through written documentation. The research team maintained patient privacy through data encryption and coding methods that also protected personal information. The research followed Helsinki Declaration principles and Good Clinical Practice standards.

Consent for publication:

Not applicable.

Clinical trial registration:

Clinical trial number: not applicable. This study was a retrospective observational study and was, therefore, not registered as a clinical trial.

Author contributions

All authors contributed substantially to the conception, design, data collection, and

interpretation of the study results.

Conceptualization: AL; data curation: NKK; formal analysis: MMA; funding acquisition: none; investigation: VK; methodology: MMA; project administration: FE; resources: MC; software: SEK; supervision: AL; validation: MC; visualization: NKK; writing—original draft: VK; writing—review and editing: SEK

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