Research Article Open Access

Vitamin D deficiency as a risk factor for idiopathic carpal tunnel syndrome: A case-control study

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Submission Date: July 9th, 2025, **Acceptance Date:** August 20th, 2025, **Publication Date:** September 12th, 2025

Please cite this article as: Ahmed S. F., Abdelsadek S. E., Maklad S. A., Osman M. A., Elshimy S. M., Mahmoud A. M., Abonar E. N. N., Elmesiry A. M., Ahmed S. A., Salama D. M., Yassien M. A. A. A., Abdelrahman E. K., Bukhari A. A. S., Abousteit A. M. Vitamin D deficiency as a risk factor for idiopathic carpal tunnel syndrome: A case-control study. *Bioactive Compounds in Health and Disease* 2025; 8(9): 365 – 374. DOI: https://doi.org/10.31989/bchd.8i9.1709

ABSTRACT

Background: Vitamin D (VD) plays essential roles in neurological health and may influence nerve function through both neuroprotective mechanisms and anti-inflammatory.

Objective: To investigate the association between VD deficiency and idiopathic CTS, this study evaluates the independent relationship of low serum levels of VD with CTS risk and symptom severity. This study uniquely investigates VD deficiency in CTS patients with normal electrodiagnostic findings, offering new insights into early-stage disease pathophysiology.

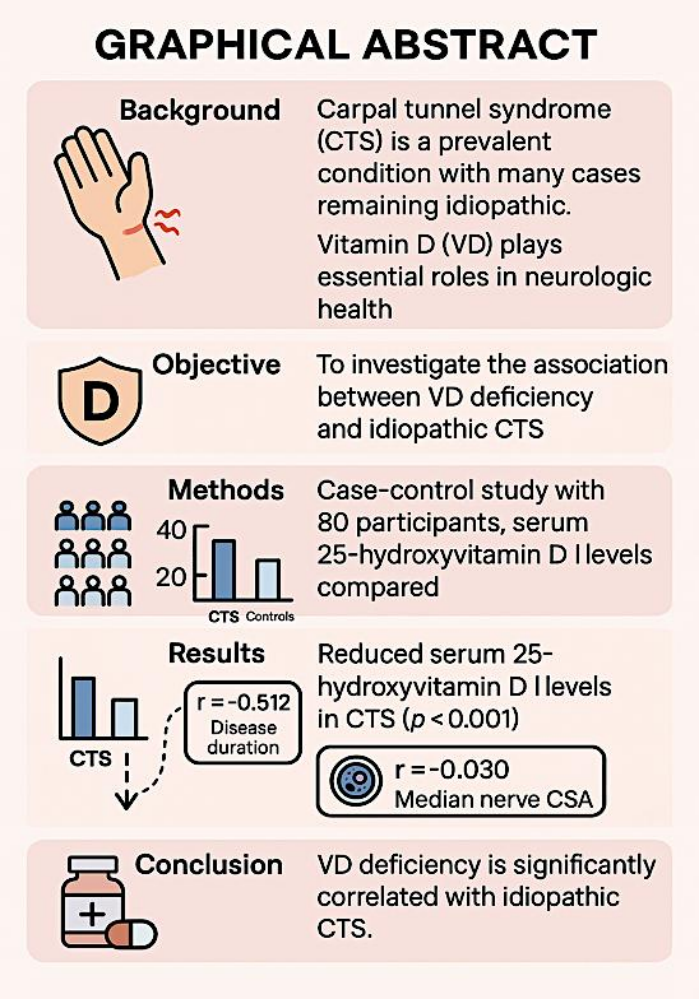
Methods: A case-control investigation was performed with 80 participants, comprised of 40 clinically suspected CTS patients with normal electrodiagnostic findings and 40 healthy controls, recruited from Al Zahraa University Hospital between November 2023 and July 2024. The levels of serum 25-hydroxyvitamin D were compared between groups.

Correlations were assessed using Pearson's correlation coefficient for VD levels relative to clinical parameters, including disease duration and median nerve cross-sectional area (CSA).

Results: Patients with CTS had significantly reduced serum 25-hydroxyvitamin D levels (12.57 ± 3.21 ng/mL) compared to controls (24.02 ± 3.07 ng/mL), ($p< 0.001$), indicating a high prevalence of deficiency among CTS patients. Disease duration was inversely correlated with serum 25-hydroxyvitamin D levels ($r = -0.512$, $p < 0.001$), suggesting that lower VD levels corresponded with longer symptom duration. No significant relationships were detected between VD levels and wrist median nerve CSA ($r = -0.030$, $p = 0.855$) or in the forearm ($r = 0.118$, $p = 0.470$), indicating that VD deficiency may not be directly related to structural nerve changes in CTS patients.

Conclusion: VD deficiency is significantly correlated with idiopathic CTS, particularly with disease duration. These data suggest the role of VD as a modifiable risk factor, suggesting the need for further research into VD supplementation as a therapeutic intervention for CTS management.

Keywords: Carpal tunnel syndrome, vitamin D deficiency, 25-hydroxyvitamin D, neuropathy, neuroprotective properties



INTRODUCTION

Carpal tunnel syndrome (CTS) is the predominant entrapment neuropathy of the upper extremity, characterized by compression of the median nerve within the fibro-osseous carpal tunnel at the wrist [1]. Clinically, CTS manifests with paresthesia, pain, and numbness in the median nerve distribution, often progressing to weakness or atrophy of the thenar musculature in advanced cases [2, 3]. Despite extensive research into its etiologies including systemic diseases (e.g., rheumatoid arthritis, hypothyroidism), anatomical variations, and occupational exposures, a substantial proportion of CTS cases remain idiopathic, underscoring the need to identify novel modifiable risk factors [4].

Vitamin D (VD), traditionally recognized for its role in calcium–phosphate homeostasis and skeletal health, has emerged as a neuroactive steroid with anti-inflammatory and neuroprotective properties [5]. The active form, 1,25-dihydroxyvitamin D, modulates immune responses by downregulating pro-inflammatory cytokines and upregulating neurotrophic factors, while also regulating intraneuronal calcium influx and combating oxidative stress–mediated injury [6, 7].

The mechanistic underpinnings linking VD deficiency to CTS risk likely involve a combination of increased neuroinflammation, impaired myelination, and altered calcium homeostasis within the carpal tunnel microenvironment [8]. VD's suppression of L-type calcium channel expression and enhancement of antioxidant defenses may mitigate ischemia-reperfusion injury and perineural edema under conditions of mechanical compression [9]. Additionally, VD deficiency has been associated with heightened musculoskeletal pain and psychological symptoms that may exacerbate symptom perception and functional impairment in CTS [6].

Serum 25(OH)D levels in CTS patients are consistently lower than in healthy controls, according to clinical investigations. In a Jordanian case, a control study of 48 CTS patients and matched controls, most CTS subjects exhibited VD deficiency (<20 ng/mL) versus controls; logistic regression identified deficiency as an independent CTS risk factor [6]. Similarly, the severity of electrophysiological abnormalities including distal motor delay and slower sensory conduction has been inversely correlated with 25(OH)D concentrations, even after adjusting for body mass index (BMI) and age [7, 10]. Emerging interventional trials suggest that repletion of VD in deficient CTS patients can improve nerve conduction parameters and reduce pain scores, although data on functional outcomes remain limited [11].

This study aims to determine if VD is independently associated with idiopathic CTS.

METHODS

Study Design: This study utilized a case-control design to investigate the effect of VD on individuals with clinically suspected CTS but without electrodiagnostic confirmation (normal nerve conduction studies, NCS), compared to healthy controls.

Participants: This study recruited participants between November 2023 and July 2024 from Al Zahraa University Hospital. Individuals referred from the Neurology, Rheumatology, and Rehabilitation Departments with clinical suspicion of CTS were assessed for eligibility. Of these referrals, about 40 patients presenting with clinical features suggestive of CTS and exhibiting normal NCS findings were enrolled in the study and constituted Group 1. A control group (Group 2) comprising 40 healthy participants, matched for age and gender with the patient group, was also recruited for comparison.

Information regarding habitual dietary VD intake (including ergocalciferol vs. cholecalciferol sources), sun exposure, and medications or disorders affecting VD metabolism was not recorded in this study. However, all participants were recruited from the same geographic

region and cultural background to minimize potential variation in these factors between groups.

Inclusion Criteria: In this study, participants aged 20–50 with clinically diagnosed CTS according to the American Academy of Neurology and normal NCS data were included.

Exclusion Criteria: Participants were excluded from the study if they presented with pre-existing conditions such as diabetes mellitus, hypothyroidism, or rheumatoid arthritis. Individuals with a documented history of wrist trauma or fracture, intra-articular lesions including ganglia, or the presence of a bifid median nerve were also ineligible. Furthermore, participants who had received corticosteroid injections for CTS within six months or had CPS surgery were excluded. Finally, individuals with clinically confirmed CTS confirmed by nerve conduction studies were not included in this specific investigation.

Methodology: All participants enrolled in this study underwent a comprehensive evaluation protocol. This included the collection of a detailed medical history, followed by a thorough general physical examination encompassing musculoskeletal and neurological assessments. Specific provocative maneuvers for CTS, such as Phalen's test and the carpal compression test, were performed. Additionally, discriminatory tests, including Spurling's test, were performed to assess alternative diagnoses, particularly cervical radiculopathy. Laboratory testing included total blood count, C-reactive protein, liver, kidney, lipid, glycated hemoglobin, thyroid, and VD levels. Furthermore, electrodiagnostic studies, specifically NCS, and ultrasound assessment of the carpal tunnel were utilized to provide a comprehensive diagnostic evaluation.

Chemiluminescence immunoassay was used to measure serum 25(OH)D levels with the SKT-041 Human 25-Hydroxy VD CLIA Kit (Epitope Diagnostics, Inc., San Diego, CA, 92121, USA). The manufacturer's instructions were followed, with intra- and inter-assay coefficients of variation below <10% and <12%, respectively. Results were interpreted using established criteria: values above 30 ng/mL were ideal, 21–29 ng/mL were insufficient, and 20 ng/ml or fewer were deficient.

Electrodiagnostic Approach: All participants underwent electrodiagnostic studies on a Neuro-pack NCS machine (Nihon Kohden, Japan) using the American Association of Neuromuscular and Electrodiagnostic Medicine's standard criteria for suspected CTS, as outlined by Zivkovic et al. (2020) [12]. Performed with comparison to the contralateral side, the electrodiagnostic protocol included motor, sensory, and median nerve F-wave responses in the afflicted limb. Additionally, ulnar nerve MCS, SCS, and F-wave responses were examined. The palmar-to-wrist sensory nerve action potential amplitude ratio was calculated using median-ulnar comparative sensory investigations to the ring finger and median sensory palmar stimulation.

Ultrasonographic Assessments: Ultrasonographic evaluations were conducted on all subjects with a commercially available linear array transducer (7–11 MHz; Xario200, Toshiba, Japan). Participants were positioned with their forearms lying on a table, wrists supinated, and fingers extended. Transverse sonographic pictures were acquired at the proximal carpal tunnel inlet, delineated as the area between the pisiform bone and the scaphoid tubercle. The median nerve was recognized as a rounded hypoechoic entity exhibiting internal hyperechoic fascicular patterns, situated under the flexor retinaculum and above the FDS, FDP, and FPL tendons. Precautions were implemented to ensure the transducer remained perpendicular to the nerve in order to reduce anisotropic artifacts, while exerting minimal pressure to prevent compression of the carpal tunnel structures.

The ultrasound protocol included measurement of the MN-CSA at the distal wrist crease, followed by

proximal scanning toward the forearm. A second MN-CSA measurement was obtained 12 cm proximal to the wrist crease, and the wrist-to-forearm ratio was subsequently calculated. Additional assessments included evaluation of median nerve flattening, calculated by dividing the maximal transverse width by the maximal anteroposterior height, and measurement of flexor retinaculum bowing by determining the perpendicular distance from a line drawn between key bony landmarks to the apex of the bowing.

Ethics Approval: The study was approved in October 2022 (Approval number 2119) by Ethics Committee of Al Azhar University's Faculty of Medicine. Each study participant gave informed consent. All patient data was confidential and used only for the study. (Supplementary File 1)

Consent to Participate : All study participants gave informed consent.

Statistical Analysis: Analyses used SPSS 26.0. Continuous variables were defined using mean ± SD and range for regular distribution and median with IQR for non-normal conditions. Categorical variables were frequency and percentage grouped. Normality was determined by Kolmogorov-Smirnov and Shapiro-Wilk. In order to compare groups, we utilized independent samples t-tests for continuous variables and chi-square or Fisher's exact tests for categorical variables (with expected cell counts <5) Pearson computed continuous variable correlations. A scatter plot revealed the intensity and direction of linear correlations.

RESULTS

The study compared the baseline characteristics, disease duration, and serum 25-hydroxyvitamin D levels between group 1 (n=40) and group 2 (n=40). No significant variations in age, sex, or BMI were observed across groups ($p>0.05$). Serum 25-hydroxyvitamin D levels were significantly lower in the sick group compared to the control group ($p<0.001$) (Table 1).

Table 1. Demographics, disease duration, and serum 25-hydroxy VD levels between both groups. Independent t-test for Age, BMI, and Serum Vit.D; Chi-square test for sex distribution; Descriptive stats for disease duration (patients only)

Parameter	Patients Group (n=40)	Control Group (n=40)	Test Value	p-value	Significance
Age (years)	33.25 ± 9.63 (Range: 20–50)	34.03 ± 9.25 (Range: 20–50)	–0.367	0.715	NS
Sex (Female: Male)	34 (85%): 6 (15%)	32 (80%): 8 (20%)	0.346	0.556	NS
BMI (kg/m²)	28.58 ± 4.52 (Range: 18–37)	27.25 ± 5.20 (Range: 19–35)	1.216	0.228	NS
Disease Duration (months)	7.25 ± 3.22 (Range: 1–11)	–	–	–	–
Serum 25-hydroxy VD (ng/mL)	12.57 ± 3.21 (Range: 7–18)	24.02 ± 3.07 (Range: 20–28)	5.682	0.001	HS

Table 2. Nerve conduction of median nerve distribution among study group

Nerve conduction of median nerve	Patients Group (n=40)
Sensory distal peak latency (Msec)	
Mean±SD	3.09±0.28
Range	2.5-3.5
Difference between median and ulnar digit 4 sensory latencies (msec)	
Mean±SD	0.39±0.43
Range	0.0–1.2
Distal motor latency (msec)	
Mean±SD	3.29±0.44
Range	2.3-4

Nerve conduction of median nerve	Patients Group (n=40)
Distal motor amplitude (MV)	
Mean±SD	10.16±1.61
Range	7.4-14.7
Nerve conduction velocity (m/sec)	
Mean±SD	59.23±6.80
Range	48-69.7
F wave	
Normal	40 (100.0%)
Abnormal	0 (0.0%)

In addition, all parameters of the nerve conduction study in the patient group were within normal ranges (Table 2).

Our analysis shows that the patient group had significantly greater median nerve cross-sectional area (CSA) at the wrist, wrist-forearm ratio, and mid-carpal tunnel flattening ratio compared to the control group ($p<0.05$) (Table 3).

Table 3. Comparison between patient group and control group according to ultrasound parameters of median nerve. t-Independent Sample t-test for Mean±SD. NS: Non-significant; S: Significant; HS: Highly significant

Ultrasound of median nerve	Patients (Group1)	Control (Group2)	Test value	p-value	Sig.
Median nerve CSA at wrist (at carpal tunnel) (MM2)					
Mean±SD	10.88±0.94	8.02±0.61	16.137	0.001	HS
Range	9-12.9	7-8.9			
Wrist forearm ratio					
Mean±SD	1.91±0.29	1.20±0.12	9.124	0.001	HS
Range	1.4-2.58	1-1.4			
Flattening ratio at mid carpal tunnel					
Mean±SD	3.21±0.41	1.85±0.13	2.219	0.027	S
Range	2.5-3.9	1.5-2			
Bowing of flexor retinaculum					
Mean±SD	4.13±1.00	3.82±0.17	1.834	0.072	NS
Range	2.7-6.5	3-4			

The study demonstrates a significant inverse correlation between serum VD levels and key ultrasound markers of CTS severity, with the wrist forearm ratio ($r = -0.628$, $p = 0.001$) showing a highly significant association and the flattening ratio at the mid carpal tunnel ($r = -0.476$, $p = 0.037$) also reaching significance (Table 4) (Figure 1).

Table 4. Correlation Between serum VD and ultrasound markers of CTS severity in patient group

Ultrasound Parameter	r-value	p-value	Significance
Wrist forearm ratio	-0.628	0.001	HS
Flattening ratio at mid carpal tunnel	-0.476	0.037	S
Median nerve CSA at wrist	-0.030	0.855	NS
CSA 12 cm proximally in forearm	0.118	0.470	NS
Bowing of flexor retinaculum	-0.108	0.507	NS

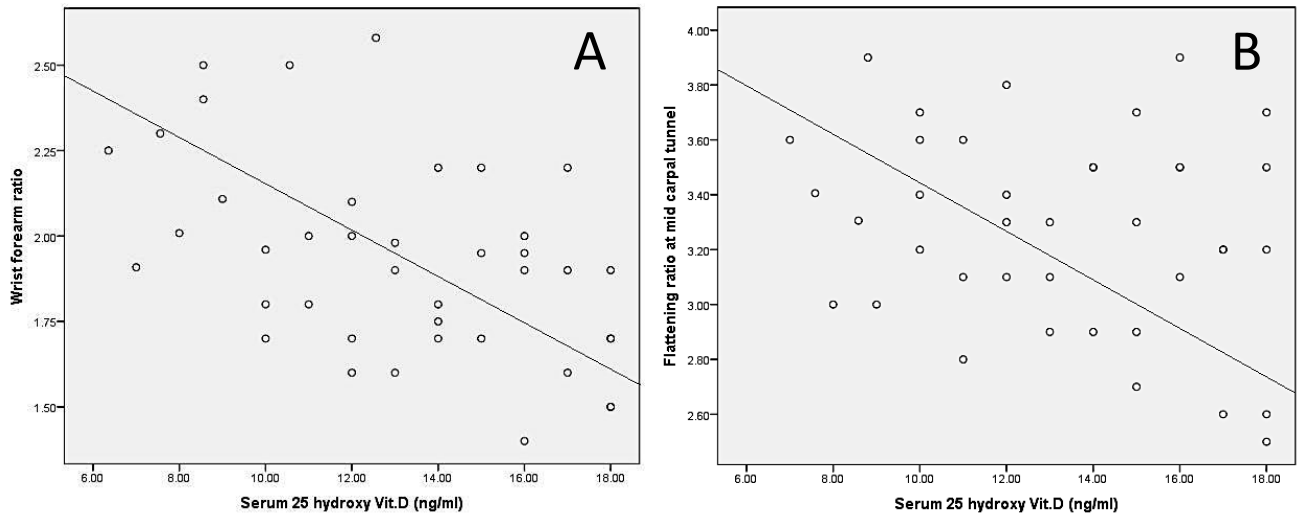


Figure 1. (A) Scatter plot between serum 25 hydroxy VD (ng/mL) and wrist forearm ratio. (B) Scatter plot between serum 25 hydroxy VD (ng/mL) and fattening ratio at mid carpal tunnel.

Receiver operating characteristics (ROC) curve was performed for Serum 25 hydroxy VD (ng/mL) and demonstrated an area under the curve of 1.000 (0.955-1.000) with P value 0.001. The best cut-off value for differentiation between the patient group and control group was ≤18 with a sensitivity of 100% and a specificity of 100%.

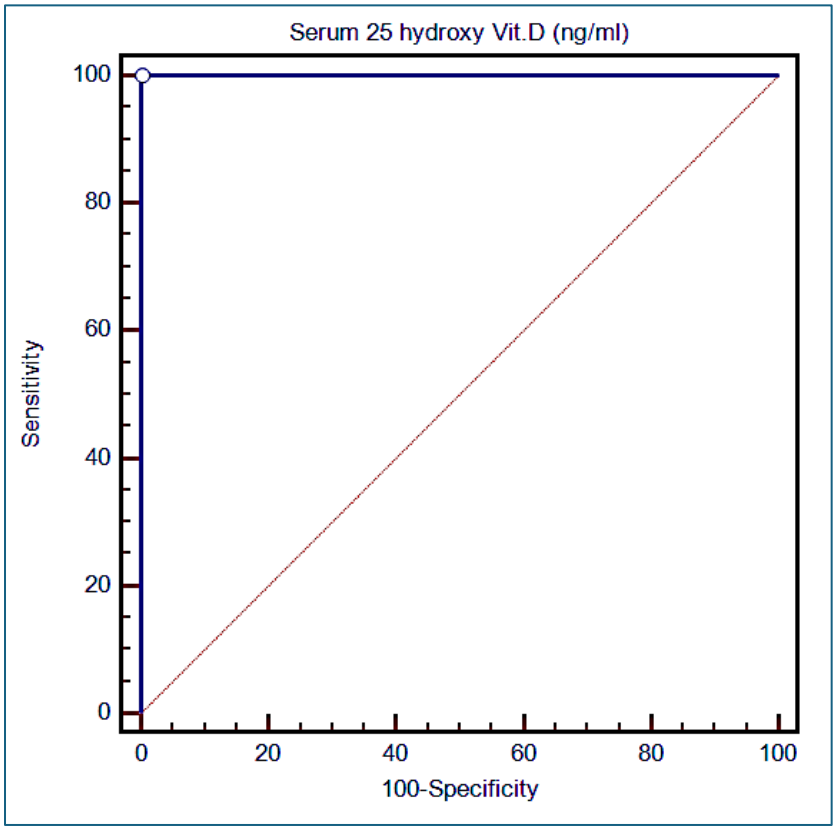


Figure 2. Receiver-operating characteristic (ROC) curve for discrimination between patients and control, using the Serum 25 hydroxy VD (ng/mL).

DISCUSSION

Multiple observational studies have demonstrated that hypovitaminosis D is highly prevalent among patients with CTS, with deficiency rates exceeding 90% in clinically diagnosed cohorts compared to roughly 74% in healthy controls, and mean serum 25hydroxyvitamin D [25(OH)D] levels significantly lower in CTS patients than in matched controls [11, 13].

Systematic reviews and case–control analyses have identified low serum 25(OH)D as an independent risk factor for both the occurrence and severity of CTS [14, 15]. Interventional data, although limited, suggest that VD supplementation may ameliorate pain and improve electrophysiological parameters in CTS patients [16]. Mechanistically, VDR in Schwann cells and dorsal root ganglion neurons promote myelination, stimulate nerve growth factor expression, and attenuate proinflammatory signaling, providing a biologically plausible link between deficiency and median nerve entrapment [17, 18]. Collectively, these findings underscore the importance of evaluating VD status as a modifiable risk factor in CTS pathogenesis and support further research into its utility for screening, prevention, and adjunctive therapy.

Our investigation revealed a substantial difference between patients and controls. In a study of 50 female CTS patients, mean blood 25(OH)D levels were considerably lower compared to age-matched controls [13]. In a study of 108 CTS patients and 52 healthy controls, 25(OH)D deficiency (<20 ng/mL) was found in 96.1% of EP+ and 94.7% of EP– patients, compared to 73.8% of controls [11].

Another study reported mean 25(OH)D levels of 19.18 ± 11.39 ng/mL in CTS patients and 21.39 ± 15.93 ng/mL in controls, with a negative correlation between VD status and disease duration [19]. This pronounced deficiency mirrors case–control observations from varied populations: AbdulRazzak and Kofahi (2020) found that over 95% of Jordanian CTS patients were VD deficient versus 23% of controls [6], while Zakaria et al. (2024) reported mean levels of 15 ± 0.3 ng/mL in CTS patients versus 21 ± 0.01 ng/mL in controls ($p = 0.002$) [20].

A recent cross-sectional analysis from Egypt corroborated a direct association between lower VD status and CTS occurrence, highlighting VD's potential neuroprotective role in maintaining nerve integrity [20]. In parallel, studies characterizing BMI and VD thresholds have classified deficiency at <20 ng/mL levels at which CTS risk appears elevated and defined insufficiency between 20 – 30 ng/mL [6].

This differs from studies like Anusitviwat et al. (2021), which reported increased CSA in CTS patients but did not examine VD status [16]. It may be that VD deficiency preferentially influences perineural edema and retinacular tightness captured by wrist forearm and flattening ratios rather than gross nerve enlargement.

A few pilot interventional studies suggest that correcting VD deficiency can improve CTS symptoms and nerve function. Savadjani et al. (2023) reported symptomatic relief and improved electromyographic parameters following VD supplementation in deficient CTS patients [14], and another study highlighted a cohort in which adjunctive VD reduced pain and tingling three months posttherapy [15]. Although these trials are small, they lend clinical plausibility to our finding that deficiency correlates with anatomical markers of severity.

Our study reinforces the recommendation that serum 25(OH)D be routinely assessed in patients with suspected CTS even when NCS are normal since deficiency appears to track with early ultrasound detectable changes. Given the low cost and safety of VD repletion, future large-scale, randomized controlled trials should test whether supplementation can prevent progression from subclinical nerve compression (evident on ultrasound) to overt electrophysiological and clinical CTS. Mechanistic studies exploring how VD modulates perineural inflammation and retinacular biomechanics

would further refine our understanding of its role in CTS pathogenesis.

This study did not collect detailed data on dietary VD sources, sun exposure habits, or medications and conditions that may influence VD metabolism. Although the matched design and recruitment from the same population likely minimized group-level differences in these variables, residual confounding cannot be excluded. Future research should include these parameters to allow a more comprehensive assessment of modifiable factors influencing VD status in CTS patients.

CONCLUSION

Patients with idiopathic CTS and normal NCS exhibited significantly lower serum 25-hydroxyvitamin D levels that were inversely correlated with ultrasound markers of median nerve compression. VD deficiency thus represents a modifiable risk factor in CTS; routine 25(OH)D screening and targeted supplementation warrant evaluation in future randomized trials.

List of Abbreviations: CTS, Carpal Tunnel Syndrome; VD, Vitamin D; 25(OH)D, 25-hydroxyvitamin D; NCS, Nerve Conduction Study; CSA, Cross-Sectional Area; BMI, Body Mass Index; MCS, Motor Conduction Study; SCS, Sensory Conduction Study; FDS, Flexor Digitorum Superficialis; FDP, Flexor Digitorum Profundus; FPL, Flexor Pollicis Longus; VDR, Vitamin D Receptor; ROC, Receiver Operating Characteristic; AUC, Area Under the Curve; SD, Standard Deviation; IQR, Interquartile Range; SPSS, Statistical Package for the Social Sciences; USA, United States of America; CLIA, Chemiluminescence Immunoassay; NS, Non-Significant; S, Significant; HS, Highly Significant.

Competing Interests: All authors declared noncompeting interests.

Author's Contributions: Sahar Fares Ahmed and Seham Elsaid Abdelsadek contributed to the conceptualization of the study. The methodology was developed by Sahar Fares Ahmed, Shaimaa A. Maklad, and Marwa Abdellah Osman. Sahar Fares Ahmed, Saad Mohamed Elshimy, and Abdullah Metwally Mahmoud were responsible for data curation, while Sahar Fares Ahmed, Amal M. Elmesiry, and Sally Abdelaziz Ahmed conducted the formal analysis. The investigation was carried out by Sahar Fares Ahmed, Doaa Mohamed Salama, and Marwa Abdelaziz Abosaree Yassien. Sahar Fares Ahmed, Ashfaq Ahmad Shah Bukhari, and Eman Kamel Abdelrahman prepared the original draft, and the review and editing were performed by Sahar Fares Ahmed and Alaa Mohamed Abousteit. All authors have read and approved the final manuscript.

Acknowledgement/Funding: We thank Mahmoud M. Ali, ITMO University, Saint Petersburg, Russia, for his assistance during the drafting of this manuscript. We also thank ANCOVA for Clinical Research Solutions, Mansoura City, Egypt, for their assistance in drafting this manuscript. No funding was received.

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