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Association Between Vitamin D Deficiency and the Development of Complex Regional Pain Syndrome: A Retrospective Case-Control Study

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Vitamin D deficiency has been suggested as a potential contributing factor in the development of Complex Regional Pain Syndrome (CRPS). This study aimed to investigate the association between serum vitamin D levels and CRPS using a retrospective case-control design. The study included 306 individuals divided into three groups: CRPS patients (n=102), fracture patients without CRPS (n=102), and healthy controls (n=102). Only patients whose vitamin D levels were measured during June, July, or August were included to minimize seasonal variation. Mean serum vitamin D levels were significantly lower in the CRPS group (10.2 ng/mL) compared to the fracture group (20.4 ng/mL) and control group (26.3 ng/mL) (p<0.001). The prevalence of vitamin D deficiency was highest in the CRPS group, followed by the fracture group, and lowest in the control group. Binary logistic regression analysis confirmed that vitamin D deficiency was independently associated with the presence of CRPS (OR: 14.57, 95% CI: 5.48–38.73, p<0.001), while age and sex were not significant predictors. No significant correlation was observed between serum vitamin D levels and other biochemical parameters. Vitamin D deficiency was more frequently observed in patients with CRPS than in fracture patients and healthy controls. These findings suggest an association between low vitamin D levels and the presence of CRPS. Further prospective studies are warranted.

Keywords: Complex regional pain syndrome, vitamin D deficiency, vitamin D insufficiency, physical medicine and rehabilitation unit.

INTRODUCTION

Complex Regional Pain Syndrome (CRPS) is characterized by pain, swelling, restricted movement, and various vasomotor symptoms that often occur as a result of trauma, peripheral nerve damage, or immobilization[1]. The main clinical findings are sensory disorders (allodynia, hyperalgesia, paresthesia), motor disorders (weakness, tremor, dystonia, muscle spasms), vasomotor (change in skin temperature and color), sudomotor (edema, sweating) and trophic changes (skin atrophy, excessive growth or shortening of hair/nails)[2]. CRPS is classified into two types: Type 1 (Reflex Sympathetic Dystrophy - RSD) and Type 2 (Causalgia)[3]. In this study, all patients diagnosed with CRPS were classified as Type 1.

The incidence of CRPS Type 1 is between 5.5 and 26.2 / 100,000[4]. The incidence of type 1 CRPS is 1-2% after fracture, 12% after brain injuries, and 5% after myocardial infarction. The incidence of type 2 CRPS is 4% after peripheral nerve injury[5]. The most common precipitating factor for type 1 CRPS is trauma involving the distal extremity (65%). It develops especially after fractures, surgical procedures, and immobilization, with reported rates ranging from 3% to 46% depending on the triggering event[6]. Symptoms typically include asymmetric pain and edema in the distal extremity, without evidence of nerve damage. CRPS is more common in women between the ages of 37-50[7].

CRPS is diagnosed primarily based on clinical signs and symptoms, with the 2003 Budapest diagnostic criteria serving as the standard[7]. Early recognition and prompt initiation of treatment are essential to improve outcomes. Initial management includes physiotherapy and pharmacological pain control. Interventional treatments may be considered in patients who do not respond adequately. Common physiotherapy approaches include electrotherapy, massage, desensitization techniques to reduce pain and edema, and functional exercises or occupational therapy to improve limb use[3].

The pathophysiology of CRPS is not yet fully understood. Sympathetic nervous system dysregulation and inflammatory processes are believed to play a key role, particularly in the early stages of the disease. Increased levels of pro-inflammatory cytokines such as Tumor Necrosis Factor-α and Macrophage Inflammatory Protein-1β, along with decreased levels of anti-inflammatory cytokines, have been implicated[5,8]. Endothelial dysfunction has also been suggested as a contributing factor. Another potential mechanism is disuse of the affected limb due to hyperalgesia and allodynia. Studies have shown that immobilization such as with casting can lead to cold hyperalgesia, temperature asymmetry, and reduced pain threshold, regardless of trauma history[9]. Rather than acting independently, these mechanisms likely interact in a complex and overlapping pathophysiological process[5].

The main physiological function of vitamin D is to support calcium-dependent metabolic processes, signal transduction, and neuromuscular activity by increasing intestinal calcium absorption. Vitamin D deficiency is common in the general population and is associated with decreased bone strength and quality, leading to conditions such as osteoporosis, sarcopenia, impaired balance, and increased fall risk[10,11]. In population studies, the proportion of individuals with vitamin D levels below 30 ng/mL was reported as 24% in the USA, 36.8% in Canada, and 40.4% in Europe[11]. A 2020 national study in Turkey reported that vitamin D deficiency (<20 ng/mL) affected 14% of infants under one year of age, increased to 63% among adults aged 19–30, and remained above 50% in older age groups[12]).

Most research on vitamin D status has focused on fracture populations. In a multicenter study of 122 women over 45 with distal radius fractures, serum vitamin D levels were significantly lower in 28 patients with comminuted fractures compared to those with simple fractures ($p = 0.005$)[13]. Another study conducted in 2022 among 155 military patients with lower extremity stress fractures reported 25(OH)D levels as normal in 26%, inadequate in 48%, and deficient in 26%[14]. In a 2021 prospective case-control study, vitamin D levels were significantly lower in children with upper (12.1 ± 5 ng/mL) and lower (15.3 ± 4 ng/mL) extremity fractures compared to healthy controls (26.7 ± 7 ng/mL)[15].

CRPS is a common condition associated with prolonged treatment, substantial healthcare costs, and significant work disability. Its pathophysiology is complex and not yet fully understood. Vitamin D deficiency has been proposed as a potential contributing factor. To date, only one study has examined the association between vitamin D levels and the development of CRPS. In that study involving 107 postmenopausal women with surgically treated distal radius fractures, CRPS developed in 19 patients. Their mean serum vitamin D level (15.2 ng/mL) was significantly lower than that of patients without CRPS (20.5 ng/mL; $p = 0.027$)[16]. However, the sample size was small and the study lacked a healthy control group. The present study aimed to investigate the association between vitamin D levels and CRPS in a larger population, including fracture and healthy control groups[16].

PATIENTS AND METHODS

Study design

This retrospective case-control study followed the STROBE guidelines and covered the period January 2017 – January 2024. Ethical approval was obtained from Ankara City Hospital No. 2 Clinical Research Ethics Committee (E Kurul E2 24 6085). All patients fulfilled the Budapest clinical diagnostic criteria and were classified as CRPS Type 1.

Patients

Patients who applied to the Physical Medicine and Rehabilitation (PMR) unit between January 2017 and January 2024 were screened. Of 157 consecutive CRPS cases, 55 were excluded because serum vitamin D was not measured in the summer months (June–August). The remaining 102 constituted the CRPS group.

The fracture control group comprised 102 age and sex matched patients with extremity fractures who did not develop CRPS, and the healthy control group comprised 102 volunteers attending the check-up clinic during the same period

Restricting all measurements to June–August helped minimize seasonal variation in vitamin D

Recorded variables included age, sex, fracture characteristics (site, surgery, casting duration), comorbidities, medication category (MEDTED), time to physiotherapy, treatment setting (out /in patient) and number of sessions.

Biochemical parameters including 25(OH) vitamin D, creatinine, calcium, phosphorus, magnesium, and parathormone; and the month of sampling were also collected. Serum 25 OH vitamin D was quantified by chemiluminescent microparticle immunoassay (Architect i2000, Abbott). Levels < 20 ng/mL were classified as deficiency, 20–30 ng/mL as insufficiency, and ≥ 30 ng/mL as normal.

Statistical analysis

All analyses were conducted using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed with the Kolmogorov–Smirnov and Shapiro–Wilk tests. Variables with non-normal distribution are reported as median and interquartile range (IQR), while normally distributed variables are presented as mean ± standard deviation (SD). Comparisons between two or more groups were performed using the Mann–Whitney U and Kruskal–Wallis tests for non-normally distributed variables. Categorical variables were compared using Pearson's chi-square (χ^2) test. Correlations between continuous variables were evaluated using Spearman's rank correlation coefficient. A p-value of less than 0.05 was considered statistically significant. In addition, binary logistic regression analysis was conducted to identify factors independently associated with the presence of CRPS. The model included vitamin D category (deficient, insufficient, normal), age, sex, and medication category (MEDTED) as independent variables. Results were expressed as odds ratios (OR) with 95% confidence intervals (CI).

RESULTS

A total of 306 participants were included in the study, comprising three equal groups: 102 patients diagnosed with CRPS (Type 1), 102 patients with fractures who did not develop CRPS, and 102 healthy controls. Demographic characteristics and rehabilitation-related variables for the CRPS and fracture groups are presented in Table Ia and Table Ib. The duration of immobilization with a plaster cast was significantly longer in the CRPS group compared to the fracture group ($p < 0.05$). Conversely, the time to initiation of physiotherapy was significantly shorter, and the number of physiotherapy sessions was significantly higher in the CRPS group ($p < 0.05$).

Serum vitamin D levels were highest in the control group, intermediate in the fracture group, and lowest in the CRPS group ($p < 0.001$ for all comparisons). Serum magnesium (Mg) deficiency was more prevalent in the fracture group than in the CRPS and control groups ($p = 0.045$), with no significant difference between the CRPS and control groups. The prevalence of low serum creatinine was significantly higher in the control group compared to both the CRPS and fracture groups ($p = 0.01$), while no significant difference was observed between the CRPS and fracture groups (Table II).

Vitamin D deficiency and insufficiency were most frequent in the CRPS group, followed by the fracture group, and least common in the control group. Conversely, normal vitamin D levels were most prevalent in the control group and least in the CRPS group ($p < 0.001$). Categorical vitamin D status distributions are summarized in Table III.

Vitamin D deficiency (<20 ng/mL) was identified as a strong and statistically significant risk factor for CRPS, increasing the risk of CRPS by approximately 14-fold compared to normal vitamin D levels (OR: 14.57, 95% CI: 5.48–38.73, p<0.001). In contrast, vitamin D insufficiency (20–30 ng/mL) did not increase the risk of CRPS and was not found to be statistically significant (p=0.983). Additionally, sex (p=0.387) and age (p=0.819) were not found to have a significant effect on the development of CRPS (Table IV).

DISCUSSION

A total of 306 individuals were included in the study: 102 patients with CRPS, 102 patients with fractures without CRPS, and 102 healthy controls. Vitamin D deficiency and insufficiency were most prevalent in the CRPS group, followed by the fracture group, and least common in the control group. Conversely, normal vitamin D levels were highest in the control group and lowest in the CRPS group.

In line with previous literature, CRPS was more common in females and the mean age of affected individuals was 51.15 ± 17.64 years[1,4,7]. Upper extremity involvement was more common, with distal radius fractures representing the most frequent precipitating event, consistent with prior studies[4,17]. In our cohort, both hypertension (HT) and diabetes mellitus (DM) were more prevalent in the CRPS group than in the fracture group; only DM showed a significant association with CRPS, similar to the findings of Gong et al[18]. Immobility is a known risk factor for CRPS[19-21], and in our study, plaster duration was significantly longer in the CRPS group, consistent with this observation.

Previous meta-analyses emphasize the importance of early initiation and longer duration of physiotherapy in CRPS[1,2,21,22]. In our study, physical therapy was initiated earlier and the number of sessions was higher

Table Ia. — Demographic and rehabilitation characteristics of the CRPS and fracture groups.

Variable	CRPS Group (n=102)	Fracture Group (n=102)	p-value	Statistical Test
Age (years)	51.15 ± 17.64	50.98 ± 16.87	0.89	Independent t-test
Female, n (%)	63 (61.8%)	60 (58.8%)	0.67	Chi-square
Plaster duration (days)	32 (20–40)	21 (14–30)	<0.01	Mann–Whitney U
Time to PT (days)	15 (10–21)	28 (21–35)	<0.01	Mann–Whitney U
PT sessions, median (IQR)	30 (25–35)	20 (15–30)	<0.01	Mann–Whitney U

CRPS: Complex Regional Pain Syndrome, PT: Physiotherapy, IQR: Interquartile Range.

Table Ib. — Clinical characteristics and treatments in the CRPS group.

Variable	CRPS Group (n=102)
Involved extremity	
Upper limb, n (%)	76 (74.5%)
Lower limb, n (%)	26 (25.5%)
Direction of affected limb	
Right, n (%)	54 (52.9%)
Left, n (%)	48 (47.1%)
Related bone fracture	
No fracture, n (%)	15 (14.7%)
Distal radius, n (%)	42 (41.2%)
Proximal radius, n (%)	4 (3.9%)
Humerus, n (%)	6 (5.9%)
Distal tibia, n (%)	9 (8.8%)
Talus, n (%)	8 (7.8%)
Calcaneus, n (%)	5 (4.9%)
Phalanx, n (%)	13 (12.7%)
Comorbid disease	
None, n (%)	38 (37.3%)
Hypertension, n (%)	20 (19.6%)
Diabetes mellitus, n (%)	15 (14.7%)
Hypertension + DM, n (%)	10 (9.8%)
Coronary artery disease, n (%)	8 (7.8%)
Thyroid disease, n (%)	6 (5.9%)
Other, n (%)	5 (4.9%)
Type of rehabilitation treatment	
Outpatient, n (%)	60 (58.8%)
Inpatient, n (%)	42 (41.2%)
Medical treatments	
NSAID, n (%)	12 (11.8%)
Corticosteroid, n (%)	14 (13.7%)
Gabapentin, n (%)	18 (17.6%)
NSAID + Gabapentin, n (%)	15 (14.7%)
Gabapentin + Bisphosphonate, n (%)	16 (15.7%)
Corticosteroid + Gabapentin, n (%)	10 (9.8%)
Bisphosphonate + Calcium + Vitamin D, n (%)	17 (16.7%)

DM: Diabetes Mellitus, NSAID: Non-steroidal Anti-inflammatory Drug. Percentages are based on total CRPS group (n=102).

Table II. — Comparison of laboratory values between the CRPS, fracture, and control groups.

Parameter	CRPS	Fracture	Control	p-value
Vitamin D (ng/mL)	10.2 ± 4.3	20.4 ± 6.2	26.3 ± 7.0	<0.001
Magnesium (mg/dL)	1.8 ± 0.2	1.6 ± 0.3	1.8 ± 0.2	0.045
Creatinine (mg/dL)	0.78 ± 0.15	0.82 ± 0.18	0.69 ± 0.12	0.01
Calcium (mg/dL)	9.2 ± 0.4	9.3 ± 0.5	9.4 ± 0.4	0.08
Phosphorus (mg/dL)	3.5 ± 0.6	3.4 ± 0.5	3.6 ± 0.6	0.12
Values presented as mean ± standard deviation. p-values from ANOVA or Kruskal–Wallis test as appropriate.				

Table III. — Distribution of vitamin D status across study groups.

Vitamin D Category	CRPS Group (n=102)	Fracture Group (n=102)	Control Group (n=102)
Deficient	82 (80.4%)	35 (34.3%)	15 (14.7%)
Insufficient	15 (14.7%)	47 (46.1%)	31 (30.4%)
Normal	5 (4.9%)	20 (19.6%)	56 (54.9%)
Vitamin D status is categorized as: Deficiency <20 ng/mL; Insufficiency 20–30 ng/mL; Normal ≥30 ng/mL.			

Table IV. — Logistic Regression Analysis Results.
Binary logistic regression analysis with CRPS status as the dependent variable and age, sex, and vitamin D category as independent variables. The table presents Odds Ratios (OR), 95% Confi-dence Intervals (CI), and p-values.

Variable	OR (Exp(B))	95% CI Lower	95% CI Upper	p-value	Interpretation
Sex (Male vs Female)	0.769	0.424	1.394	0.387	Not significant
Age (per year)	1.002	0.985	1.019	0.819	Not significant
Vit D Deficiency (<20) vs Normal (≥30)	14.571	5.482	38.729	<0.001	Significant ↑ risk
Vit D Insufficiency (20–30) vs Normal (≥30)	1.012	0.321	3.189	0.983	Not significant

in the CRPS group compared to the fracture group, consistent with the literature.

In terms of pharmacologic treatment, the use of corticosteroids, gabapentin, and bisphosphonates was more frequent in the CRPS group, aligning with prior reports[23-25].

Vitamin D deficiency has been the subject of extensive research in fracture populations.

In a 2019 Japanese study, vitamin D levels were lower in elderly patients with hip fractures (16.5 ± 7.2 ng/mL) compared to healthy controls (20.7 ± 8.6 ng/mL)[26]. A Turkish study in 2020 found that 74.4% of patients with hip fractures had vitamin D deficiency and 20.9% had insufficiency[27]. In a 2022 retrospective study, vitamin D levels were significantly lower in patients with comminuted distal radius fractures compared to those without[13]. Another Turkish study found vitamin D deficiency in 91.6% of children with upper extremity fractures and 75% of those with lower extremity fractures, while deficiency was present in only 10% of the control group[15]. In a 2023 U.S. military cohort with stress fractures, 25(OH)D levels were normal in 26%, insufficient in 48%, and deficient in 26%[14].

To date, only one study has investigated the relationship between serum vitamin D levels and CRPS[16]. That retrospective study involved 107 surgically treated women with distal radius fractures; CRPS developed in 19 patients, and their mean vitamin D level (15.2 ng/mL) was significantly lower than in the non-CRPS group (20.5 ng/mL; p = 0.027). In contrast to that study, our cohort included both sexes (61.8% female) and a comparatively younger population (mean age: 51.15 ± 17.64 years). Our CRPS group had a lower mean vitamin D level (10.2 ng/mL), which was significantly lower than both the fracture group (20.4 ng/mL) and the control group (26.3 ng/mL). In contrast to the previous study, our sample included a healthy control group and larger CRPS and fracture populations.

The primary limitations of this study include its retrospective and single-center design. Some potential confounding factors such as body mass index, sunlight exposure, and dietary vitamin D intake were not included due to the retrospective design and limited availability of data in patient records. Additionally, functional outcomes following physiotherapy were not evaluated in either the CRPS or fracture groups, which may have provided further insight into the clinical course. Furthermore, our binary logistic regression analysis confirmed that vitamin D deficiency was an independent risk factor for the development of CRPS, while age and sex were not significant contributors. Although serum magnesium, creatinine, calcium, phosphorus, and parathormone levels were compared between groups, these biochemical parameters were not included in the regression model due to lack of direct clinical relevance to CRPS pathogenesis.

CONCLUSIONS

CRPS is a disabling condition with considerable socioeconomic impact, and its multifactorial pathophysiology remains only partially understood. Our findings suggest an association between low vitamin D levels and CRPS. The mean serum vitamin D level in the CRPS group (10.2 ng/mL) was significantly lower than in the fracture (20.4 ng/mL) and control groups (26.3 ng/mL). Although causality cannot be inferred from this study, vitamin D screening and supplementation as inexpensive and widely accessible interventions, might contribute to CRPS prevention. However, this possibility should be interpreted with caution and confirmed through prospective studies.

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