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Research Article

Evaluation of the Relationship Between Vitamin D Deficiency and Epicondylitis

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Abstract

Aim: Though there is strong evidence connecting vitamin D deficiency to several health problems, such as diabetes mellitus, infections, autoimmune and endocrine diseases, cancer, and increased cardiovascular mortality and morbidity, studies examining the connection between vitamin D deficiency and epicondylitis are rare. The purpose of this study was to see if low vitamin D levels and epicondylitis were linked.
Material and Method: A total of 410 patients presented with the complaint of elbow pain and whose 25-hydroxycholecalciferol (25(OH) D) levels were evaluated. The files of 205 patients diagnosed with medial or lateral epicondylitis by physical examination and 205 controls not considered to have medial or lateral epicondylitis were reviewed retrospectively. Vitamin D levels were measured using 25(OH)D levels.
Results: Vitamin D levels were considerably lower (p<.001) in the epicondylitis group.
Conclusion: It is unclear precisely what causes epicondylitis, but the fact that our study's participants had much lower vitamin D levels raises the possibility that low vitamin D is one of the causes. More research is needed to understand how vitamin D levels may contribute to the etiology of epicondylitis in general.

Keywords: Elbow tendinopathy, Vitamin D deficiency, pain

INTRODUCTION

Elbow pain is most commonly caused by epicondylitis (1). Epicondylitis comes in two varieties: lateral epicondylitis, also referred to as tennis elbow, and medial epicondylitis, also known as golfer's elbow. It is most common between the ages of 40 and 60. It rarely occurs before the age of 30. It is more prevalent in women than in men. Medial epicondylitis is less common and has a milder course than lateral epicondylitis. In the general population, medial epicondylitis is more common in males (0.3-0.6%) and females (0.3-1.1%), but lateral epicondylitis is more common in males (1.0-1.3%) and females (1.1-4.0%). The annual incidence is estimated to be 0.3–1.1 and 0.1 per 100 individuals for lateral and medial epicondylitis, respectively (2). Epicondylitis is rarely bilateral and is more common in the dominant extremity (3).

Tennis elbow is thought to be caused by lesions at the lateral epicondyle of the humerus of the extensor carpi radialis brevis tendon, whereas golfer's elbow is thought to be caused by musculotendinous lesions at the medial epicondyle of the humerus of the common flexor tendon. Although it has the suffix -itis in its name, local inflammation is not frequently observed (1). Age is an essential factor in the formation of epicondylitis, and changes in the collagen content in the enthesis region decrease the number of cells and increase adipose tissue with aging, creating a predisposition. A history of overuse and repetitive and demanding activities increases the risk of developing epicondylitis (4).

A physical examination and history are used to diagnose epicondylitis. Radiologic and electrodiagnostic tests may be performed to exclude other possible diagnoses. Epicondylitis usually has an insidious and spontaneous

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onset. Pain is localized to the medial and lateral epicondyles but may radiate upward or downward. On physical examination, there is tenderness with pressure on the epicondyles. There is usually no swelling, and the elbow range of motion is normal (4). In lateral epicondylitis, an increase in pain is observed with resistant wrist extension or forearm supination. In medial epicondylitis, an increase in pain is observed with resistant wrist flexion or forearm pronation.

Calciferol, another name for vitamin D, is a fat-soluble vitamin. It comes in D2 and D3 variants. While vitamin D2 (ergocalciferol) is gained through diet, vitamin D3 (cholecalciferol) can also be generated in the skin by ultraviolet-B (UV-B) radiation from 7-dehydrocholesterol (5). Vitamins D2 and D3 are hydroxylated in the liver to form 25-hydroxycholecalciferol (25(OH)D). 25(OH)D is the result of the hydroxylation of vitamins D2 and D3 in the liver. The active form of vitamin D, 1.25-dihydroxycholecalciferol (1.25(OH)2D), is ultimately produced by the kidneys by hydroxylation (6). The level of 25(OH)D, which is the total of the amounts of 25(OH)D2 and 25(OH)D3, often indicates the amount of vitamin D present (7). Vitamin D is necessary for the health of the musculoskeletal system as well as the metabolism of calcium and phosphorus. Research has also linked vitamin D deficiency and insufficiency to a host of health issues, such as diabetes mellitus, autoimmune diseases, infections, cancer, endocrine diseases, and elevated cardiovascular mortality and morbidity (8-11). Additionally, studies have demonstrated vitamin D's neuroprotective, anti-inflammatory, and anti-proliferative properties (12). In this study, we looked into the potential link between vitamin D deficiency and epicondylitis.

MATERIAL AND METHOD

Research and Publication Ethics

The levels of 25(OH)D in a total of 410 patients with elbow pain, including 205 patients with medial or lateral epicondylitis by physical examination and 205 controls who were not considered to have medial or lateral epicondylitis, were retrospectively analyzed between January 2021 and November 2022 with the approval of Erzurum City Hospital Ethics Committee (decision no. 18-173 dated November 24, 2022). The exclusion criteria were trauma history, neoplasia, enfectious and rheumatological diseases.

Measurement of the Level of vitamin D

Every patient's venous blood was tested for vitamin D, and the results were assessed. Using the Atellica IM Analyzer, a total of 25(OH)D in human serum and plasma was quantitatively measured. Under 30 ng/ml of 25(OH)D was regarded as a vitamin D deficiency.

Statistical Analysis

IBM SPSS 26 was utilized for the analysis of the data. Analyses were first conducted to ascertain the distribution of normalcy. As a result, it was discovered that the Kolmogorov-Smirnov test findings were $p<.05$. These numbers explain the normal distribution of the data. Consequently, one of the parametric tests, the independent group T-test, was applied in the analysis. The statistical analysis of the descriptive data employed the values of the highest, lowest, median, mean, and standard deviation. The chi-square test was employed to analyze independent qualitative data.

RESULTS

A total of 205 patients had lateral or medial epicondylitis. There were 143 female patients and 62 male patients. The mean vitamin D level was 16.5±9.9 (2.92-69.70), and the mean age was 46.1±10 years. The results for 20 patients were normal. The vitamin D levels of 185 patients were low. Two hundred-five did not have lateral epicondylitis or medial epicondylitis. There were 140 females and 65 males among them. 49.3±16.5 years was the mean age, and 21.3±11.9 (6.1-72.3) was the mean level of vitamin D. 38 patients had normal results. It was found that 167 people had a deficit of vitamin D. Table 1 displays each patient's sociodemographic traits and 25(OH)D levels.

Table 1. Descriptive data on gender, age, 25(OH)D* vitamin levels and 25(OH)D* vitamin grades of the groups

Group	N	Gender		Age					25(OH)D* vitamin level			25(OH)D* vitamin grade	
		Women	Men	Mean	Median	SD	Min	Max	Mean	Median	SD	<30	>30
Epicondylitis (+)	205	143	62	46.11	45.00	10.08	16	76	16.50	13.70	9.99	185	20
Epicondylitis (-)	205	140	65	49.30	50.00	16.50	17	88	21.30	17.90	11.90	167	38

*25(OH)D: 25-hydroxycholecalciferol

Compared to the epicondylitis (-) group, the epicondylitis (+) group's patient age was considerably ($p=.019$) lower. There was no significant difference observed in the gender distribution between the epicondylitis (+) and epicondylitis (-) groups ($p=.831$). It was found that the vitamin D levels in patients with epicondylitis (X=16.54, SD=9.99) were lower than in individuals without epicondylitis (X=21.34, SD=11.90). An independent sample t-test was applied to determine whether the difference was significant. Consequently, patients with epicondylitis had considerably decreased vitamin D levels [$t(408)=-4.421$, $p<.001$] (Table 2).

Table 2. 25(OH)D* vitamin levels of the groups

	Group	N	Mean	SD	t	p
25(OH)D* vitamin level	Epicondylitis (+)	205	16.5498	9.99491	-4.421	<.001
	Epicondylitis (-)	205	21.3495	11.90550		

*25(OH)D: 25-hydroxycholecalciferol

DISCUSSION

Our investigation explored the possible connection between low serum vitamin D levels and epicondylitis. Furthermore, it was discovered that up to 85.7% of people (n=347) had vitamin D deficiency. According to studies, Türkiye and other developing nations have significant rates of vitamin D deficiency (13,14). There was no noticeable variation in gender distribution across the groups in our investigation. The group with epicondylitis had a substantial gender difference that favored the females. As in our study, epicondylitis has been reported in the literature as more common in females (2). Moreover, research indicates that vitamin D deficiency is more common in women than in men (13). To ensure that the study's findings are unaffected, it is crucial that there be no discernible variation in the groups' gender distribution.

Insufficient sun exposure, poor dietary intake, or absorption issues can all lead to vitamin D deficiency. It is important to test the level of 25(OH)D, which comprises both endogenous and dietary vitamin D and has a half-life of two to three weeks for vitamin D in the individual. The active biological form of 1.25(OH)2D is unsuitable for measurement. Because its circulating levels are 1000 times lower than 25(OH)D, and its half-life is 4-6 hours. Studies have been conducted to determine the normal range for 25(OH)D levels as well as to define vitamin D insufficiency and deficiency. These studies have led to the definition of vitamin D deficiency, which is now known as vitamin D insufficiency if the 25(OH)D level is between 21 and 29 ng/mL, normal if it is above 30 ng/mL (desired range: 30 to 60 ng/mL), and vitamin D intoxication if it is above 150 ng/mL (15,16).

The etiopathology of epicondylitis is not known precisely. Histologic investigations have not shown the presence of inflammatory cells in the tissue, such as neutrophils, lymphocytes, or macrophages. Histopathologic findings such as microfractures, periostitis, fibrinoid degeneration, immature collagen tissue, hyaline degeneration, radio-humeral joint disease, radial nerve entrapment, and annular ligament lesions have been reported in a few chronic cases. Ischemic stress may be significant in the etiology because the tenoperiosteal junction and the surrounding tendon are relatively avascular. These findings have been considered to be lateral elbow tendinosis by researchers. The term tendinosis describes a degenerative process characterized by vascular hyperplasia of fibroblasts and the dense presence of immature collagen in the area. The terms tendinosis or tendinopathy indicate the absence of chemical inflammation, but the pathology is painful. In most cases, the cause of pain is musculotendinous lesions that develop at or around the insertion site of the common extensor tendon to the lateral epicondyle (4).

Although water makes up 70% of typical tendon tissue, type I collagen accounts for most of the dry weight (17,18). The primary component of the extracellular matrix is type I collagen; this protein's quality and quantity significantly impact the mechanical and structural characteristics of tendon tissue (17). Tenocytes regulate type I collagen to preserve the integrity of the extracellular matrix. In contrast, tenoblasts produce extracellular matrix components, particularly type I collagen (17,19). The impact of vitamin D on tenocytes was examined by Min et al. (20). They demonstrated that in tenocytes treated with dexamethasone, the expression of tenomodulin, tenascin, type I, and type III collagen genes dropped. However, when vitamin D was administered, the expression of tenomodulin and type I collagen was restored to normal, and the quantity of reactive oxygen species decreased. In conclusion, they reported that vitamin D benefits tendons, bones, and muscles. According to Chen et al., inhibiting tumor necrosis factor-α (TNF-α) may protect against tendon degeneration (21). Vitamin D has been shown to reduce inflammation at the cellular level with its effects on macrophages, TNF-α, and interleukin and to have a protective effect on cell functions (8). Because of its interaction with muscle receptors, vitamin D has been shown to improve muscle growth and strength through protein synthesis. However, a loss in muscle tone and strength has been linked to vitamin D deficiency (22). In addition, another study reported low vitamin D levels in patients with lateral epicondylitis, similar to our study (23).

There are several restrictions on our investigation. The study's data were retrospectively gathered from file records. Our findings could have been impacted by the fact that each patient's vitamin D level was not assessed at the same time or season. A low vitamin D level may also be linked to several musculoskeletal issues. In patients with epicondylitis, the complaint change after vitamin D replacement could not be determined. Therefore, prospective studies are needed to determine the improvement in complaints after vitamin D replacement in these patients.

CONCLUSION

Patients with epicondylitis had significantly low vitamin D levels, suggesting a link between low vitamin D levels and epicondylitis. We believe this association may also apply to tendons in other regions of the musculoskeletal

system, not just the epicondylitis that was the subject of our investigation. The fact that some people with epicondylitis have normal vitamin D levels indicates that various elements, including anatomical, biomechanical, and occupational components, are involved in the genesis of the disease and that vitamin D deficiency is neither the sole nor the most significant cause. More research will shed light on the connection between epicondylitis and vitamin D.

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