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Research Article

Assessment of the Relationship between Vitamin D Deficiency and Epin Calcanei

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Abstract

Aim: There is no research exploring the connection between vitamin D insufficiency and epin calcanei, despite strong evidence linking it to a number of health issues, including diabetes, infections, autoimmune disorders, cancer, cardiovascular illnesses, and widespread muscular discomfort. In this study, we examined whether vitamin D insufficiency is linked to epin calcanei.

Material and Methods: 205 patients with foot pain clinically diagnosed with epin calcanei by radiograph and 205 patients without epin calcanei clinically diagnosed by radiograph were evaluated. These patients' data were reviewed retrospectively. Radiographic evaluation was performed on all patients for epin calcanei. Vitamin D levels were evaluated by looking at the 25-hydroxycholecalciferol (25(OH)D) level.

Results: The 25(OH) vitamin D values in the group with epin calcanei showed a significant (p=0.001) difference.

Conclusion: In our research, epin calcanei and vitamin D levels were shown to be significantly correlated. There are research on vitamin D levels in a variety of fields, but none have looked at how it could relate to epin calcanei. More research is required to fully comprehend the possible contribution of vitamin D levels on the etiology of epin calcanei.

Keywords: Epin calcanei, heel pain, vitamin D

INTRODUCTION

Heel pain is a common problem. In an American study, the prevalence of heel pain was found to be 7% (1). German surgeon Plettner used the term epin calcanei anatomically for the first time in 1900. When the plantar fascia inserts, it frequently happens on the medial calcaneal tuberosity. The radiological appearance of epin calcanei has been found between 11% and 16% in studies (2). The radiological appearance of epin calcanei is mostly seen in women, elderly patients, patients with osteoarthritis, and patients with heel pain (2-4). The main complaint is pain felt in the heel which increases over time. Pain is more common when we wake up in the morning and when we start walking. It is stinging and burning, especially in the inner part of the heel. It decreases after walking for a while but increases towards the evening with the load depending on the activity. Rest relieves and reduces pain. However, when we start walking after sitting, it increases again in the first step. The diagnosis is made with clinical and radiological radiography.

There are many factors in the etiology of epin calcanei disease, the exact cause of which is unknown. The most common cause is inflammation caused by repetitive traction at the beginning of the plantar fascia and healing of this inflammation by ossification (5). This is called the longitudinal traction hypothesis. A significant part of the etiology is played by recurrent microtraumas, persistent injury to the tiny foot muscles, and the onset of the plantar aponeurosis. Intrinsic foot muscle weakness is one of the most important factors in the etiology of epin calcanei, but hereditary conditions and metabolic disorders may be just as important by accelerating the inflammatory process (6,7). Chronic injury results from a reduction in the elasticity of the insertional cartilage. Mesenchymal cells in the scar tissue fill the cracks in the damaged cartilage. With the re-formation of blood vessels, the scar gradually ossifies to form a bony spur (8). Another reason is the increasing degeneration of the elastic adipose tissue in

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the heel with aging. Therefore, it is thought to be more common in elderly patients.

Mineral and vitamin supplements are utilized for the skeletal and bone system, joints, and cartilage to develop and regenerate themselves in a healthier and faster way (9,10). Vitamin D is a class of sterols that act as hormone precursors and are classified as hormones since they may be created endogenously. It is also considered one of the fat-soluble vitamins. Its most important effect is on bone mineralization, phosphorus, and calcium metabolism (11,12). Vitamin D deficiency and insufficiency, a general health issue for a wide range of acute and chronic disorders, is a risk factor (13). Vitamin 25(OH)D deficiency leads to secondary hyperparathyroidism. Osteoclasts are responsible for bone resorption. As a result, adult osteopenia and osteoporosis may accelerate and worsen (14). Vitamin D increases osteoblastic activity and supports bone mineralization by keeping parathormone (PTH) levels at normal levels (14,15). Its main function is to affect calcium and bone metabolism in an anabolic direction. Vitamin D has been shown to have many effects in different systems such as neuroprotective, anti-inflammatory, and antiproliferative effects (16).

Recent studies have shown links between vitamin D deficiency and insufficiency and a variety of chronic illnesses, including cancer, immune system disorders, neurodegenerative diseases, psychiatric illnesses, metabolic syndrome, diabetes mellitus, insulin resistance, cardiovascular diseases, and infectious diseases (17,18). Although significant research has linked vitamin D insufficiency to a number of illnesses, including cancer, diabetes, autoimmune disorders, cardiovascular diseases, infections, and general muscular discomfort, no research has looked specifically at the connection with epin calcanei. In this study, our aim investigate whether there is any relationship between epin calcanei and vitamin D deficiency in patients presenting to the physiotherapy outpatient clinic with heel pain.

MATERIAL AND METHOD

Patient Population

With the consent of the local ethics committee (with the decision dated 29.05.2018 and 001), a total of 410 patients (205 epin calcanei +, 205 epin calcanei-) who applied to the Department of Physical Therapy and Rehabilitation between January 2021 and November 2022 with complaints of pain in the foot, clinically diagnosed as epin calcanei, 25(OH)D level was checked and lateral standing with a load direct foot radiography was taken were retrospectively analyzed. 205 individuals without epin calcanei were used as the control group. The socio-demographic characteristics of the patients were recorded.

Measurement of Vitamin D Level

Vitamin D levels measured in venous blood obtained from all patients were evaluated. A total of 25 (OH) vitamin D was quantitatively determined in human serum and plasma (EDTA, lithium heparin, and sodium heparin) using Atellica IM Analyser. 25(OH)D levels below 30 ng/ml were considered as vitamin D deficiency.

Statistical Analysis

The data obtained using the IBM SPSS 26 program were analyzed. Firstly, analyses were performed to determine the normality distribution. Accordingly, Kolmogorov-Smirnov test results were found to be $p<0.05$. Then, skewness kurtosis values were analyzed by considering the literature (19). The data were distributed normally according to these values. Therefore, independent groups T-Test, one of the parametric tests, was used in the analyses. The highest, lowest, median, mean, and standard deviation values were used in the statistics of descriptive data. Analyzing qualitative independent data involved using the chi-square test.

RESULTS

Epin calcanei was present in 205 of the patients included in the study. Of these, 30 were men and 175 were women. The mean age was 48.8±11.5 years. Vitamin D level was 17.5±10.2 (4-62) ng/ml. 23 patients had normal results. 182 patients had vitamin D deficiency. 205 of them did not have epin calcanei. Of these, 65 were men and 140 were women. The mean age was 49.3±16.5 years. Vit- D level was 21.3±11.9 (6.1-72.3) ng/ml. The results of 38 patients were normal. Vitamin D deficiency was found in 167 patients. Socio-demographic characteristics and 25(OH) D levels of all patients are presented in Table 1.

The age of the patients in the epine calcanei (+) group did not differ significantly (p=0.758) from the epine calcanei (-) group. Gender distribution in the epine calcanei (+) and epine calcanei (-) groups differed significantly (p=0.000). Vitamin D levels of patients with epin calcanei (X=17.54, SD=10.2) were lower than vitamin D levels of individuals without epin calcanei (X=21.34, SD=11.90). The significance of the difference was evaluated using the independent groups t-test. According to this, vitamin D level was significantly lower in individuals with epin calcanei [t (408)=-3.47, p=0.001] (Table 2).

Table 1. Descriptive data on gender, age, 25-OH-D* vitamin level and 25-OH-D* vitamin level 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
Epin calcanei (+)	205	175	30	48.80	48.00	11.56	23	84	17.50	15.40	10.20	182	23
Epin calcanei (-)	205	140	65	49.30	50.00	16.50	17	88	21.30	18.90	11.90	167	38

*25-OH-D: 25- hydroxy-cholecalciferol

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

	Group	N	Mean	SD	t	p
Vit-D level	Epin calcanei (+)	205	17.5430	10.27722	-3.465	.001
	Epin calcanei (-)	205	21.3495	11.90550		

*25-OH-D: 25- hydroxy-cholecalciferol

DISCUSSION

In the present study, we looked for a relationship between vitamin D insufficiency and epin calcanei. In addition, we found that vitamin D deficiency was found at a very high rate of 85% (n=349). Studies have reported that vitamin D deficiency has a high prevalence in developing countries including Turkey (20,21). In our investigation, the ages did not significantly differ. However, research has revealed that vitamin D insufficiency is more prevalent among women (20). The genders differed significantly in our study. This condition is comparable to other studies.

Epin calcanei (calcaneal spur) is a bony protrusion larger than 2 mm, which is seen at the attachment site of the plantar musculofacial structure to the calcaneus starting from the inner projection of the calcaneal tuberosity. It has been shown in the study of Aydogdu et al. (22) that patients with epin calcanei are over middle age and overweight, and women are more affected. Overload causes an increase in regional pressure and tension in the plantar fascia. Chang et al. (23) showed that the thickness of the plantar fascia 1 cm distal to the starting point is directly related to body weight and emphasized that overloading of the plantar fascia is effective in this mechanism. In studies, it has been reported that female gender, age, high body mass index, and plantar fascia thickness, as well as functional disorders such as intrinsic muscles of the foot, soleus, and gastrocnemius weakness, are associated with epin calcanei and plantar fasciitis (20,23).

The etiology of epin calcanei has many causes and these causes are not fully known. There are many mechanical hypotheses related to the formation of epin calcanei. These are vertical compression and longitudinal traction hypotheses. The most common cause is inflammation due to repetitive traction at the beginning of the plantar fascia and healing of this inflammation by ossification (5). This is called the longitudinal traction hypothesis. Repetitive microtraumas and chronic damage to the small muscles of the foot and the beginning of the plantar aponeurosis caused by repetitive injuries play an important role in the pathogenesis. Intrinsic foot muscle weakness is one of the most important factors in the etiology of epin calcanei, but hereditary conditions and metabolic disorders may be just as important by accelerating the inflammatory process (6,7). Chronic damage occurs with a decrease in the elasticity of the insertional cartilage. Mesenchymal cells in the scar tissue fill the cracks in the damaged cartilage. With the re-formation of blood vessels, the scar gradually ossifies to form a bony spur (8). Another cause is the increasing degeneration of the elastic adipose tissue in the heel with aging.

An insufficient consumption of foods containing vitamin D, insufficient sun exposure, or issues with absorption are the causes of vitamin D insufficiency. It has been observed that vitamin D insufficiency should be included in the differential diagnosis of muscle and bone pain, and that correction of deficiency is an essential component in the treatment of these individuals (24). There is controversy about the limited value of vitamin D in studies. Plasma 25-OH-D3 is the best clinical indicator of vitamin D because it includes all dietary and cutaneous synthesized vitamin D (25). It is recommended to assess the 25(OH)D level, which accounts for both endogenous synthesis and vitamin D consumption and has a half-life of 2-3 weeks in an individual. 1,25(OH)2D active biological form is not suitable for ideal measurement. Considering that its half-life is as brief as 4-6 hours and that its circulation levels are 1000 times lower than those of 25(OH)D. There have been studies to identify vitamin D insufficiency and deficiency, as well as the typical range of 25(OH)D values. According to these studies, if the 25(OH)D level is below 20 ng/mL, it is defined as vitamin D insufficiency, and if it is between 21-29 ng/mL, it is defined as vitamin D deficiency. Between 30-60 ng/mL' is defined as normal. Over 150 ng/mL is considered as vitamin D intoxication (26,27). Low vitamin 25(OH)D levels cause secondary hyperparathyroidism. Thus, they may accelerate and worsen osteopenia and osteoporosis in adults because they cause bone resorption via osteoclasts (14). Vitamin D increases osteoblastic activity and supports bone mineralization by keeping PTH levels in the normal range and consequently significantly reduces the likelihood of falls and fractures (14,15). Studies are showing that the probability of fracture is lower in patients with vitamin 25(OH)D levels >30 ng/mL (26). In our study, we accepted the limit value of vitamin D as 30 ng/ml. There is a positive relationship between vitamin 25(OH)D and proximal muscle strength, physical activity, and lower extremity functions. Postural and dynamic balance and muscle strength can be increased by vitamin D supplementation (28,29). The Framingham Study suggested that low vitamin D levels may be associated with the development of cartilage loss and progression of knee osteoarthritis (30). Similarly, moderate evidence has shown that low vitamin D levels are associated with increased progression of radiographic osteoarthritis. In our study, serum vitamin D levels were decreased in epin calcanei formation, suggesting that this may be a predisposing factor.

Vitamin D has a variety of vascular effects. Thrombosis, smooth muscle cell proliferation, and inflammation modulation are some of them. PTH is a hormone that increases vascular remodeling and myocyte hypertrophy. Studies are showing that adequate vitamin D reduces inflammation at the cellular level and has a protective effect on cell functions (31). Low vitamin D may impair bone mineralization and cause pain in muscles and joints associated with diffuse or isolated bone pain. It has been suggested that vitamin D has a cartilage-supporting effect and that osteoarthritis may develop or progress as a result of cartilage thinning due to the loss of this support in vitamin D deficiency (32). Vitamin D has anti-inflammatory effects via macrophages, tumor necrosis factor, and interleukin (31). Muscle tone and muscle strength decrease in low vitamin D levels. As a result of the interaction of vitamin D with its receptors in muscle, protein synthesis increases, and muscle strength and mass increase (33,34). Vitamin D deficiency leads to increased inflammation, thinning of cartilage, and especially weakness of muscles. Thus, we have seen in our study that microtraumas in the bone and impaired mineralization of the newly formed bone due to vitamin D deficiency an etiological factors in the formation of epin calcanei. Since there are no publications on epin calcanei in the literature, we wanted to show its relationship with epin calcanei in our study. This study will allow us to look at patients diagnosed with epin calcanei from a different perspective or to develop a new perspective to prevent epin calcanei when heel pain is present before it occurs. With the discovery of vitamin D receptors in several tissues, interest in vitamin D has recently grown. Several research have been carried out to determine how low vitamin D levels relate to different disorders. In our study, low vitamin D levels were seen in both groups, although the epin calcanei group had lower vitamin D levels than the control group. This suggests that it is important in the pathogenesis of epin calcanei. This conclusion needs to be supported by more research.

Our research has several limitations. The data in the study were collected retrospectively from file records and were not evaluated seasonally. The duration of sun exposure of the patients was also not taken into consideration. Vitamin D insufficiency may be a factor in a number of musculoskeletal diseases.

CONCLUSION

As a result, our study's case group had statistically lower levels of vitamin 25(OH)D than the control group. Low levels of vitamin 25(OH)D, in our opinion, are predisposing factors in the formation of epin calcanei. We believe that vitamin D supplementation may be able to stop the development of epin calcanei in individuals with low vitamin D levels at various points in time. Supporting our data with studies with large patient groups may allow a better understanding of etiology.

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