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Original Research Article

Correlation of hypothyroidism and dyslipidemias with plantar fasciitis in Indian population

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ABSTRACT

Background: Plantar fasciitis (PF) affects millions of people but still there is lack of knowledge of all the factors involved in causing this condition. The main objective of this study is to investigate the relationship between thyroid hormones, dyslipidemias, hyperuricemia, liver and renal function tests.
Methods: 200 patients were enrolled,100 with a clinical diagnosis of PF in the case group and 100 without PF in the control group. Patients thyroid profile (T3, T4, TSH), liver profile (Bilirubin, SGOT, SGPT), renal profile (Serum creatine, urea), lipid profile (serum total cholesterol, LDL, HDL, VLDL, triglyceride) and uric acid levels were checked and compared between the two groups.
Results: Patients with PF had a higher TSH level with a mean of 4.33 mg/dl (range 0.64-20.56) than the control group with TSH mean of 2.51 mg/dl (range 0.51-8.78) ($p<0.001$). Serum cholesterol level was significantly higher in patients with PF than in the control group ($p=0.002$). Triglycerides ($p=0.01$), LDL ($p<0.01$), VLDL ($p=0.021$) were significantly higher in patients than in controls. HDL levels were found to be significantly lower in patients than in controls ($p=0.003$).
Conclusions: Significantly higher TSH levels, serum cholesterol, serum triglyceride, serum LDL, serum VLDL and low HDL levels were seen in the Indian population suffering from PF. Thus, hypothyroidism and dyslipidemias should be evaluated while treating PF.

Keywords: Hypothyroidism, Dyslipidemia, PF, Indian population, Tendinopathies

INTRODUCTION

Millions of people throughout the world suffer with plantar fascitis (PF), a common and debilitating condition.[1-3] Inflammation of the plantar fascia, a thick band of tissue that runs along the bottom of the foot, causes this painful and frequently chronic ailment. Walking, running, and other physical activities can all be adversely impacted by this condition. Even though it's common, we still don't know what exactly causes PF.[4] Overuse stress is the most typical cause of PF, while other causes can also contribute to the condition. In terms of pathogenicity, it is known that metabolic abnormalities affect the conformation and mechanical properties of tendons, particularly in metatarsophalangeal joints, the plantar fascia, and the Achilles tendon, by producing complicated biomechanical structures.[5] Research has suggested a potential relationship between PF and metabolic diseases such as diabetes, obesity, and metabolic syndrome.[6] This may be due to the fact that metabolic diseases can lead to changes in the body's inflammatory response, which can exacerbate the inflammation of the plantar fascia. In addition, factors such as weight gain and changes in gait associated with metabolic diseases may increase the mechanical stress

placed on the plantar fascia, further contributing to the development of PF. The condition is frequently associated with runners and older adults, but obesity, heel pad atrophy, aging, and occupations that require prolonged standing can also be risk factors.[7,8] While more research is needed to fully understand the relationship between PF and metabolic diseases, it is clear that there is a complex interplay between these conditions that needs further investigation.

This condition is very much prevalent in Indian population but there is limited research specifically on PF in Indian patients. It is worth noting that cultural factors, such as the use of traditional footwear or certain occupational activities, may also contribute to the development of PF in Indian patients. Several studies have already established the role of hyperlipidemia in patients with PF.[9] To the best of our knowledge, there is no documented evidence to suggest a potential relationship between PF and hypothyroidism. This paper evaluates the relation of thyroid disorders and hyperlipdemia in Indian patients.

Thyroid hormones play a role in regulating the body's metabolism, which can impact the health of connective tissues such as the plantar fascia. Additionally, individuals with hypothyroidism may experience changes in their gait or weight gain, both of which can contribute to the development of PF.

PF has also been connected to hyperlipidemia, a disorder marked by high blood lipid levels.[10] Research has shown that individuals with hyperlipidemia may be more likely to develop PF, and that the condition may be more severe in these individuals.[9] This may be due to the fact that hyperlipidemia can lead to changes in the body's inflammatory response, which can exacerbate the inflammation of the plantar fascia. Additionally, hyperlipidemia can lead to the deposition of fatty acids and cholesterol in the blood vessels that supply the plantar fascia, which can lead to reduced blood flow and further contribute to the development of PF.

We evaluated thyroid profile, liver profile, renal profile, uric acid levels and lipid profile levels in our patients and tried to find out if this relation exists in Indian population.

METHODS

The present study was given approval by the centralised review board of our institution, and written informed consent was obtained from all patients.

This study was conducted at a tertiary care centre in Mumbai (ESIS Hospital, Mumbai). Patients diagnosed with PF, who complained of heel discomfort and were between the ages of 20 and 55 were enrolled in this study between August 2022 and February 2023. The patient's history, morning/start-up pain, and the discovery of localised tenderness in the medial calcaneal tubercle were used to diagnose PF. Patients who had other systemic or local causes of heel pain, such as pes planus or pes cavus, inflammatory diseases, neuromuscular disorders, prior surgery on the hindfoot, were excluded.

In this case control study, the PF group consisted of 100 patients (50 male and 50 female) with a mean age of 39.2 years with a mean age of 39.2 years. The control group was composed of patients who presented to Orthopedics Outpatient Department with complaints other than heel pain. There were 100 (50 male and 50 female) patients in the control group with a mean age of 37.88 years.

Patients who were already diagnosed and were under treatment for any medical condition viz. Diabetes, Hypertension, thyroid disorders, hypercholesterolemia and renal disorders were excluded from the study.

Blood samples were collected from the patients for thyroid profile (T3, T4, TSH), liver profile (Bilirubin, SGOT, SGPT), renal profile (Serum creatine, urea), lipid profile (serum total cholesterol (TC), LDL, HDL, VLDL, triglyceride) and uric acid levels. All blood samples were collected in the morning after an overnight fast. The results of the blood tests were evaluated with normal range of values for various parameters as follows; Normal range for T3 (84.6-202 ng/dl); T4 (5.1-14 ug/dl); TSH (0.27-4.2 mIU/ml); total bilirubin (0.3-1.2 mg/dl); SGPT (5-55 IU/l); SGOT (11-55 IU/l); urea (10-45 mg/dl); serum creatine (0.8-1.3 mg/dl); serum cholesterol (140-250 mg/dl); triglycerides (0-200 mg/dl); HDL (40-70 mg/dl); LDL (0-100 g/dl); VLDL (0-30 mg/dl); serum uric acid (3.2-7 mg/dl).

Table 1: Normal range of values for the variables to be evaluated.

Test	Normal range
T3	84.6-202 ng/dl
T4	5.1-14 ug/dl
TSH	0.27-4.2 mIU/ml
Total bilirubin	0.3-1.2 mg/dl
SGPT	5-55 IU/L
SGOT	11-55 IU/l
Urea	10-45 mg/dl
Serum creatine	0.8-1.3 mg/dl
Serum cholesterol	140-250 mg/dl
Triglycerides	0-200 mg/dl
HDL	40-70 mg/dl
LDL	0-100 g/dl
VLDL	0-30 mg/dl
Serum uric acid	3.2-7 mg/dl

All patients with deranged lab values were informed about their results and were recommended to visit a physician for treatment accordingly.

The D'Agostino-Pearson test was used to verify the normal distribution and homogeneity of variances of data. Numeric data were compared using the student-t-test or

Mann-Whitney U test. Conditional logistic regression was used to estimate the odds ratios (ORs) and 95% confidence intervals (CIs) for the association between PF and relevant lab results in the total subjects. A p<0.05 was considered an indication of statistical significance.

RESULTS

Patients with PF had a higher TSH level with a mean of 4.33 mg/dl (range 0.64-20.56) than the control group with TSH mean of 2.51 mg/dl (range 0.51-8.78) (p<0.001). Hypothyroidism was detected in 39% of patients (39 cases) and 10% (10 cases) of our control group.

Serum cholesterol level was significantly higher in patients with PF than in the control group (p=0.002). Patients had a higher TC level with a mean of 199.67 mg/dl (range 122.12-310.65) than the control group with mean of 186.61 mg/dl (range 97.70-265.51). Hypercholesterolemia was detected in 22% of patients (22 cases) and 8% (8 cases) of our control group.

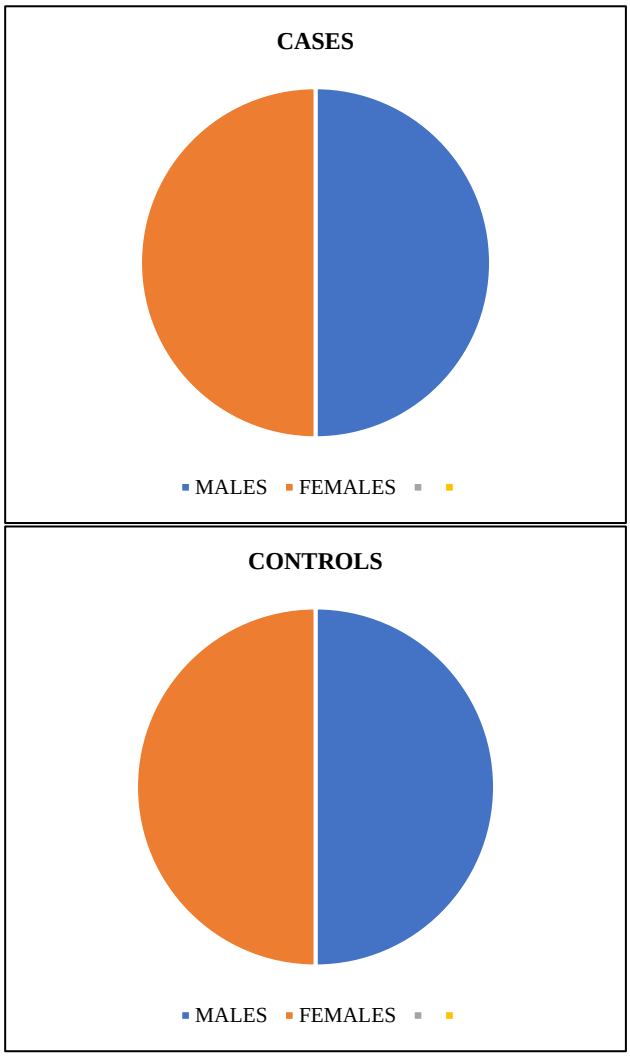

Figure 1: Gender wise distribution in cases and controls.

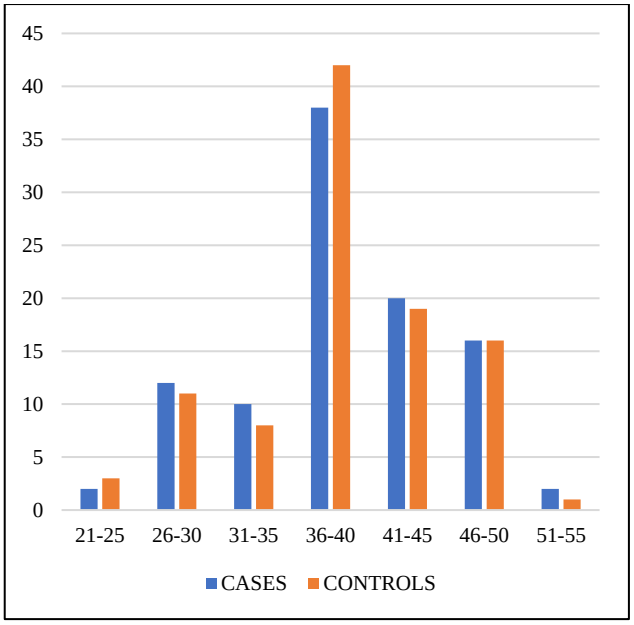

Figure 2: Age wise distribution among cases and controls.

Table 2: Result values expressed as (mean±SD) in patients and controls; p value of each variable.

Test	Patients (mean ±SD)	Controls (mean ±SD)	P value
T3	125.30± 18.63	124.98± 18.71	0.90
T4	6.68± 1.6	6.88± 1.65	0.38
TSH	4.33± 4.13	2.51± 1.55	<0.001
Total bilirubin	0.96± 0.25	0.98± 0.19	0.68
SGPT	29.78± 11.08	32.41± 11.93	0.10
SGOT	25.75± 6.42	27.55± 6.87	0.056
Urea	23.33± 8.15	24.86± 8.20	0.18
Serum creatine	0.88± 0.15	0.89± 0.12	0.66
Serum cholesterol	199.67± 51.89	186.61± 46.52	0.002
Triglycerides	178.23± 69.58	157.28± 41.58	0.010
HDL	42.99± 7.51	45.60± 4.68	0.003
LDL	109.78± 36.59	89.48± 14.35	<0.001
VLDL	30.34± 9.75	27.86± 4.28	0.021
Serum uric acid	5.35± 1.38	5.04± 0.97	0.061

Table 3: The odds ratio of developing PF.

Test	Odds ratio	95% CI
TSH	5.75	2.67, 12.39
Serum cholesterol	3.24	1.36, 7.69
Triglycerides	7.8	3.27, 18.60
HDL (low)	6.33	2.31, 17.33
LDL	5.25	2.60, 10.63
VLDL	4.27	2.17, 8.41

Triglycerides (p=0.01), LDL (p<0.01), VLDL (p=0.021) were significantly higher in patients than in controls. HDL levels were found to be significantly lower in patients than in controls (p=0.003)

The T3 level, T4 level, Total bilirubin, SGOT, SGPT, serum creatine, urea, serum uric acid levels were not significantly different between the two groups (Table 2).

DISCUSSION

There are numerous potential contributing elements to the pathophysiology of PF, whose genesis is not fully known. To the best of our knowledge and as mentioned earlier, this is the first study that directly investigated the association between hypothyroidism and PF.

Hypothyroidism is believed to be a common health issue in India, affecting approximately one in 10 adults.[11] With normal levels of thyroxine and triiodothyronine and modestly raised levels of serum thyrotropin, hypothyroidism can range from an overt state of myxedema, end-organ consequences, and multisystem failure to an asymptomatic or subclinical disease.[12,13] Studies have demonstrated that hypothyroidism causes hypoxia and apoptosis, which both contribute to musculoskeletal issues in humans.[14] A structural protein of the plasma membrane of almost all cells has been revealed to have a surface receptor for thyroid hormones, which causes cellular responses like angiogenesis and proliferation. This receptor has been linked to the ECM's involvement in thyroid activities.[15] The activation of VEGFs and basic fibroblast growth factors (bFGF), which aid in the development of vascular structures, appears to be a process intimately associated to angiogenesis.[16,17] Tendinopathies appear to be associated with incomplete vascularization, tissue hypoxia, reactive oxygen species involvement, and apoptosis.[18] A study has shown that there is a relationship between thyroid pathologies and non-traumatic rotator cuff tear.[19] The hypothyroidism-related decrease in oxygenation in the rotator cuff tendons, which results in a metaplasia and calcium deposition, is one of the hypotheses on the origins of calcific tendinopathies.[20] Therefore, tendinopathies and tendon injuries both have hypothyroidism as a contributing factor, and it also impacts the healing process.

In our study 10 percent of subjects in the control population was diagnosed as hypothyroid, which mirrors the prevalence in the Indian population.[11] Hypothyroidism was seen significantly more in patients than in controls (p<0.001). When a patient has PF, this correlation should be used as a tool to rule out thyroid disorders. Also, it emphasises the need to begin adequate medical therapy as PF treatment is not solely mechanical.

There are studies examining how high cholesterol affects the musculoskeletal system.[21] Ozgurtas et al identified hypercholesterolemia in 74% of patients with Achilles tendon rupture.[22] Abboud et al reported significantly higher TC, LDL-C, and TG in patients with rotator cuff tears than normal rotator cuff tendons.[23] Cholesterol build-up in tendons appears to cause moderate, ongoing inflammation and may alter the extracellular matrix of tendons and fascia.[24] This leads to chronic tendon degeneration and biomechanical changes. Due to the restriction of physical activity brought on by PF, metabolic parameters may deteriorate further.[25] It was found that local alterations in protein synthesis and extracellular matrix composition-turnover may cause hypercholesterolemia to alter the microenvironments of the tendons.[24] It has been demonstrated that hypercholesterolemia alters gene expression, leading to an increase in inflammatory activity and cytokine production in the tendon microenvironment.[26] The biomechanical characteristics of the tendons deteriorate, which is another consequence of hypercholesterolemia. In a study it was found that hypercholesterolemia increased the supraspinatus tendon's stiffness.[27] Those with altered tendon structure had higher rates of TC, LDL-C, and TG and lower HDL-C compared to those reported for healthy adults, according to a systematic review.[28] Also, it has been demonstrated that a high cholesterol environment has a deleterious impact on tendon healing.[29] Similar to this, high cholesterol can lead to PF by impairing the plantar fascia's ability to heal and regenerate.

In this study, serum cholesterol level was significantly higher in patients with PF than in the control group (p=0.002), consistent with the findings from other studies. One of the objectives of this study is to increase the awareness of patients and physicians about this association which will lead to early detection of lipid and cholesterol disorders, and thus preventing other systemic diseases associated with it.

One of the limitations of the study was that the duration of symptoms of patients enrolled was not taken into consideration. Whether this has any bearing on our results should be examined in another study. A total of 100 patients and 100 controls were enrolled for the study. A larger number of subjects in both groups would give a better understanding and the results can be percolated to the general population.

CONCLUSION

In conclusion, significantly higher TSH levels were found in patients with PF, which proves it to be a risk factor. Significantly higher serum cholesterol, serum triglyceride,

serum LDL, serum VLDL and low HDL levels were seen in the study which is consistent with the other studies, and proves that the Indian population also shows co relation between dyslipidemias and PF.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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