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Does a calcaneal spur develop as a result of a systemic inflammation or a regional inflammation? A pilot study

Hacı Ahmet AYDEMIR *

Kayakyolu Family Health Center, Erzurum, Türkiye

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Abstract

Calcaneal spur (CS), which is one of the most common causes of heel pain and can cause life-limiting discomfort, is quite common throughout the whole population. CS is fibro-cartilaginous protrusions of various sizes in the calcaneal bone. There are many causes in the etiology of CS. The investigated causes generally focused on physical factors, and studies on metabolic parameters are rare. This study investigated the blood parameters of patients with plantar calcaneal spur.

Keywords: calcaneal spur, platelet count, inflammatory marker, family medicine, metabolic factor

1. Introduction

Calcaneal spur (1) is a problem that can be seen at any age and affects approximately 15-20% of the population (2). CS can affect both physically active and sedentary people (3). It is generally characterized by pain in the heel region. CS is divided into plantar and dorsal CS (4). Although the localization of pain varies, heel pain is the classic symptom in both types. Individuals with CS most commonly present with heel pain that worsens with the first few steps in the morning or with inactivity (5). For plantar CS, this heel pain is observed in an area localized in the anteromedial area at the origin of the plantar fascia over the medial tubercle of the calcaneal bone (5). The pain is typically felt when starting to walk after rest (2). The diagnosis of CS is based on medical history and physical examination. On physical examination, pain is observed with palpation at the medial tubercle of the calcaneal bone. Visualization of the spur with lateral radiography is helpful for the diagnosis (6). There are different causes in the etiology of CS. Inflammation due to chronic stretching of the plantar fascia (7), vertical compression caused by increased body weight and prolonged standing (8, 9), increased thickness of the heel pad and plantar fascia with increasing age and weight, and consequently decreased elasticity of the fascia have been shown to be effective factors in spur formation. (9). There are studies showing that both dorsal and plantar CS increase with increasing age (4) and that gender has an effect on CS (1, 10). In addition, CS formation is observed in joint diseases such as gout, spondyloarthropathies, Reiter's disease, rheumatoid arthritis, ankylosing spondylitis, osteoarthritis, and plantar fasciitis (11, 12). Apart from these diseases that primarily involve the joints, metabolic parameters have also been shown to have an effect on CS. High LDL (low-density lipoprotein) and triglycerides (TG), low HDL (high-density lipoprotein), low vitamin D level, high parathyroid hormone (PTH) level and high blood glucose levels have been shown to be effective in the formation of CS (13-16).

Various treatment modalities, including conservative, minimally invasive, and surgical methods, can be employed to manage plantar fasciitis. Some conservative treatments include cold application, stretching exercises, non-steroidal anti-inflammatory drugs, and shoe modification (17, 18). Conservative treatments are successful in the majority of patients (19). In cases where conservative treatments are ineffective, minimally invasive interventions such as corticosteroid injections, platelet-rich plasma (PRP) injections, or procedures like radiofrequency needle ablation (RFNA) and extracorporeal shock wave therapy (ESWT) can be considered. Surgical intervention is recommended for resistant cases(20-22). This study aimed to compare the demographic data and metabolic parameters of a group of patients with radiological plantar CS and a group of adults without heel pain and a diagnosis of CS.

2. Materials and methods

2.1. Design

The study was planned as a cross-sectional pilot study.

2.2. Participants:

Patients over the age of 18 who applied to Çat Township Hospital Family Medicine Outpatient Clinic between 31 December 2020 and 1 June 2021 were retrospectively reviewed within the scope of our study. The total number of applications in this period was 1418 patients. Ten of these patients who presented with heel pain and were radiologically diagnosed with plantar calcaneal spur were included in our study. Patients with clinical heel pain but no radiological diagnosis were

*Correspondence: ahmetaydemir29@gmail.com

excluded from this study group. A randomly selected group of 10 patients with similar age and gender distribution to the study group, who did not complain of heel pain and did not have a diagnosis of a calcaneal spur, were included as the control group. The presence of rheumatological joint disease in both groups of patients was accepted as an exclusion criterion.

2.3. Variables:

Routine blood counts, biochemical test parameters and demographic data of both study groups were obtained and compared. Complete blood counts were analyzed by Sysmex XN-530 (Sysmex® Japan), and biochemical test parameters were analyzed by Cobas-c 501 (Roche® Diagnostics Turkey).

Neutrophil, lymphocyte, monocyte, platelet, mean corpuscular volume (MCV) and hemoglobin values were obtained in complete blood count. Biochemical parameters included total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglyceride, albumin, C-reactive protein (CRP), magnesium, calcium and potassium. As inflammatory markers, neutrophil/lymphocyte ratio, platelet/lymphocyte ratio, lymphocyte/monocyte ratio, CRP/albumin ratio, monocyte/HDL ratio, neutrophil/HDL ratio, lymphocyte/HDL ratio and triglyceride/HDL ratio were calculated and included in our study.

2.4. Statistical Analysis:

The data obtained were statistically analyzed with SPSS V23 (IBM, USA) software. Categorical data were given as frequency and percentage, and numerical data were given as mean and standard deviation. Fisher's Exact test was used to analyze categorical data. Mann-Whitney U test was used to analyze two independent data that did not show normal distribution. $p<0.05$ was considered statistically significant.

3. Results

A total of 20 individuals, ten patients with plantar CS and ten controls, were included in the study. Demographic characteristics of the individuals in the groups are presented in Table 1. Accordingly, 70% of the plantar CS patients were female, while 50% of the control group was female, but no statistically significant difference was found ($p>0.05$). A known history of chronic disease was present in 30% of the plantar CS patients and 20% in the control group, and similarly, no difference was found between the groups ($p>0.05$). The mean age of the plantar CS patients was 44.5 ± 13.66 years, while the mean age of the control group was 39 ± 8.07 years, and no statistical difference was found ($p>0.05$) (Table 2).

The mean laboratory test results and statistical comparisons of the study groups are presented in Table 2. According to the results, there was no statistical difference between the groups in all laboratory analyses, but only the platelet value was found to be significantly higher in the plantar CS group ($p=0.028$). The ratios of the two laboratory values analyzed as inflammatory markers were investigated and presented in Table 2, but no significant difference was found between the two groups in these ratios ($p>0.05$).

Table 1. Comparisons of the groups' demographic variables

		Calcaneus Spur (n, %)	Control Group (n, %)	P*
Sex	Male	3 (30%)	5 (50%)	0.650
	Female	7 (70%)	5 (50%)	
Marital Status	Married	6 (60%)	8 (80%)	0.189
	Single	2 (20%)	2 (20%)	
	Divorced	2 (20%)	0 (0%)	
Chronic Disease	No	7 (70%)	8 (80%)	0.615
	Yes	3 (30%)	2 (20%)	
Total		10 (100%)	10 (100%)	

* Fisher's Exact test

Table 2. Laboratory test results of the plantar calcaneus spur and control groups, and their comparisons statistically

	Calcaneus Spur	Control Group	P*
Age (years)	44.5 ± 13.66	39 ± 8.07	0.211
Total cholesterol (mg/dL)	186.9 ± 34.14	176.5 ± 36.49	0.384
LDL (mg/dL)	119.8 ± 53.88	98.5 ± 34.17	0.325
HDL (mg/dL)	45 ± 14.95	49 ± 13.39	0.472
Triglyceride (mg/dL)	188.4 ± 98.51	148.6 ± 90.66	0.241
Neutrophil (cells/µL)	4285 ± 1718.42	4467 ± 1007.03	0.677
Lymphocyte (cells/µL)	2735 ± 1027.78	2251 ± 821.6	0.290
Monocyte (cells/µL)	639 ± 251.11	662 ± 339.14	0.910
Platelet (cells/µL)	333700 ± 83388.05	273700 ± 35565.43	**0.028**
MCV (fL)	86.3 ± 4.79	83.6 ± 4.45	0.183
Hemoglobin (g/dL)	14.1 ± 1.28	13.83 ± 2.71	0.545
Albumin (g/dL)	4.32 ± 0.34	4.42 ± 0.35	0.423
CRP (mg/L)	7.76 ± 12.7	2.89 ± 5.06	0.819
Magnesium (mg/dL)	1.89 ± 0.19	1.96 ± 0.26	0.619
Calcium (mg/dL)	9.46 ± 0.39	9.15 ± 0.43	0.102
Potassium (mmol/L)	4.19 ± 0.36	4.36 ± 0.18	0.159
Neutrophil/Lymphocyte ratio	1.78 ± 1.06	2.2 ± 0.86	0.174
Platelet/Lymphocyte ratio	139.54 ± 64.41	135.96 ± 46.46	0.597
Lymphocyte/Monocyte ratio	4.88 ± 2.15	3.81 ± 1.45	0.257
CRP/Albumin ratio	1.78 ± 2.83	0.71 ± 1.34	0.705
Monocyte/HDL ratio	15.5 ± 7.43	15.79 ± 12.41	0.496
Neutrophil/HDL ratio	107.93 ± 52.2	98.05 ± 34.06	0.406
Lymphocyte/HDL ratio	68 ± 39.51	50.76 ± 26.43	0.226
Triglyceride/HDL ratio	5 ± 3.55	3.4 ± 2.32	0.384

* Mann–Whitney U test (LDL: Low-density lipoprotein, HDL: High-density lipoprotein, MCV: Mean Corpuscular volume, CRP: C-reactive protein)

4. Discussion

Many hypotheses have been put forward in the formation of CS. However, according to our literature review, this study seems to be the first study investigating inflammatory markers and systemic inflammation in CS. Some of them include vertical compression, stretching of the plantar fascia, and thickening of the plantar fascia (11). These hypotheses focused on mechanical effects on the plantar fascia. However, there are

studies investigating the presence of diabetes mellitus, abnormal blood lipid profile, vitamin D level, PTH level and metabolic parameters for the formation of CS (13-16). This suggests that metabolic processes may be effective in the formation of CS. In this study, we investigated these metabolic processes and examined systemic inflammatory markers that had not been analyzed in similar studies.

Sex difference was not found to be a risk factor for plantar CS in our study. In a study conducted by Başdelioğlu et al. on 200 patients with plantar CS, female gender was found to be a risk factor. In the same study, the presence of chronic disease and age group were not found to be risk factors. In this respect, the results are similar to our study (1). There are also studies in which age is considered a risk factor in the formation of CS (4, 11). There are studies related to blood lipid profile. Triglyceride and LDL elevation were shown as risk factors in the formation of CS in Tekcan's study (23). In our study, no difference was observed in terms of total cholesterol, triglycerides, HDL and LDL. In addition, the triglyceride/HDL ratio, which has not been investigated in the literature in the formation of CS, was analyzed and no statistical difference was observed.

Recently, different parameters used as inflammatory markers have been used in different diseases and have been shown to be effective in showing inflammation (24-27). There is no study in the literature investigating these markers in individuals diagnosed with CS. In our study, these inflammatory markers were also investigated. No statistical difference was observed in inflammatory markers such as CRP (C-reactive protein), neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR), lymphocyte/monocyte ratio (LMR), CRP/albumin ratio (CAR), monocyte/HDL ratio, neutrophil/HDL ratio and lymphocyte/HDL ratio. However, platelet/lymphocyte ratio (PLR), lymphocyte/monocyte ratio (LMR), CRP/albumin ratio (CAR), neutrophil/HDL ratio, and lymphocyte/HDL ratio were found to be higher in the CS group. Neutrophil/lymphocyte ratio (NLR) and monocyte/HDL ratio were found to be higher in the control group. In our study, platelet count was statistically significantly higher in the group with CS than in the control group.

The small number of patients in our study is considered the most important limiting factor. Our study is a cross-sectional pilot study, and its results are not definite enough to cover the general population. Studies with larger patient groups are needed. In addition, it is possible that the cell counts in the complete blood count may change with the drugs used. The history of drug use in the patients included in the study and the control group was not questioned.

Inflammatory markers analyzed between the group with CS and the control group were not statistically significant. However, the results of our study do not mean that inflammation does not play a role in the occurrence of CS due to the small sample size. Platelet count was found to be statistically significantly higher in the group with CS. More comprehensive studies are needed since our study was a cross-sectional pilot study.

Ethical Statement

Ethics committee approval was obtained for our study with the approval of the Atatürk University Faculty of Medicine Clinical Research Ethics Committee dated 07.09.2023 and numbered B.30.2.ATA.0.00/709.

Conflict of interest

There is no conflict of interest.

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None to declare.

Authors' contributions

Concept: H.A.A., Design: H.A.A., Data Collection or Processing: H.A.A., Analysis or Interpretation: H.A.A., Literature Search: H.A.A., Writing: H.A.A.

References

1. Başdelioğlu K. Radiologic and demographic characteristics of patients with plantar calcaneal spur. J Foot Ankle Surg. 2021;60(1):51-4.
2. M McPoil TG, Martin RL, Cornwall MW, Wukich DK, Irrgang JJ, Godges JJ. Heel pain--plantar fasciitis: clinical practice guildelines linked to the international classification of function, disability, and health from the orthopaedic section of the American Physical Therapy Association. J Orthop Sports Phys Ther. 2008;38(4):A1-A18.
3. Schwartz EN, Su J. Plantar fasciitis: a concise review. The Perm J. 2014;18(1):e105.
4. Beytemür O, Öncü M. The age dependent change in the incidence of calcaneal spur. Acta Orthop Traumatol Turc. 2018;52(5):367-71.
5. Hamstra-Wright KL, Huxel Bliven KC, Bay RC, Aydemir B. Risk factors for plantar fasciitis in physically active individuals: a systematic review and meta-analysis. Sports Health. 2021;13(3):296-303.
6. McMillan AM, Landorf KB, Barrett JT, Menz HB, Bird AR. Diagnostic imaging for chronic plantar heel pain: a systematic review and meta-analysis. J Foot Ankle Res. 2009;2:1-11.
7. Bergmann J. History and mechanical control of heel spur pain. Clin Podiatr Med Surg. 1990;7(2):243-59.
8. Menz HB, Zammit GV, Landorf KB, Munteanu SE. Plantar calcaneal spurs in older people: longitudinal traction or vertical compression? J Foot Ankle Res. 2008;1:1-7.
9. Ozdemir H, Urguden M, Ozgorgen M, Semih G. The relationship between the thickness and elasticity of the heel pad and heel pain. Acta Orthop Traumatol Turc. 2002;36(5):423-8.
10. Toumi H, Davies R, Mazor M, Coursier R, Best TM, Jennane R, et al. Changes in prevalence of calcaneal spurs in men & women: a random population from a trauma clinic. BMC Musculoskelet Disord. 2014;15:1-6.
11. Velagala VR, Velagala NR, Kumar T, Singh A, Mehendale AM, Velagala N. Calcaneal Spurs: A Potentially Debilitating Disorder.

Cureus. 2022;14(8).

12. Kirkpatrick J, Yassaie O, Mirjalili SA. The plantar calcaneal spur: a review of anatomy, histology, etiology and key associations. J Anat. 2017;230(6):743-51.

13. Adanaş C, Özkan S, Alp HH. The levels of 25-hydroxy vitamin D, parathyroid hormone, calcitonin and lipid profiles in patients with calcaneal spur. Turk J Phys Med Rehabil. 2022;68(1):55.

14. Gariani K, Waibel FWA, Viehöfer AF, Uçkay I. Plantar Fasciitis in Diabetic Foot Patients: Risk Factors, Pathophysiology, Diagnosis, and Management. Diabetes Metab Syndr Obes. 2020;13:1271-1279.

15. Hosseinian SHY, Bagheri F, Sistanian F, Mousavian A, Ebrahimzadeh MH, Kachooei AR, et al. Association of Plantar Fasciitis with Lipid Profile and Glucose Level: A Case-Control Study, 09 February 2022, PREPRINT (Version 1) available at Research Square [https://doi.org/10.21203/rs.3.rs-1328369/v1].

16. Priesand SJ, Schmidt BM, Ang L, Wrobel JS, Munson M, Ye W, et al. Plantar fasciitis in patients with type 1 and type 2 diabetes: A contemporary cohort study. J Diabetes Complications. 2019;33(10):107399.

17. Sanmak ÖDY, Külcü DG, Mesci N, Altunok EÇ. Comparison of effects of low-level laser therapy and extracorporeal shock wave therapy in plantar fasciitis treatment: A randomized, prospective, single-blind clinical study. Turk J Phys Med Rehabil. 2019;65(2):184.

18. Toomey EP. Plantar heel pain. Foot Ankle Clin. 2009;14(2):229-45.

19. Yalcin E, Keskin Akca A, Selcuk B, Kurtaran A, Akyuz M. Effects of extracorporal shock wave therapy on symptomatic heel spurs: a correlation between clinical outcome and radiologic changes. Rheumatol Int. 2012;32:343-7.

20. Çatal B, Bilge A, Ulusoy RG. Endoscopic Plantar Fascia Release versus Cryosurgery for the Treatment of Chronic Plantar Fasciitis: A Prospective Randomized Study. J Am Podiatr Med Assoc. 2020;110(5).

21. Nayar SK, Alcock H, Vemulapalli K. Surgical treatment options for plantar fasciitis and their effectiveness: a systematic review and network meta-analysis. Arch Orthop Trauma Surg. 2023;143(8):4641-51.

22. Yinilmez Sanmak ÖD, Geler Külcü D, Mesci N, Altunok E. Comparison of effects of low-level laser therapy and extracorporeal shock wave therapy in plantar fasciitis treatment: A randomized, prospective, single-blind clinical study. Turk J Phys Med Rehabil. 2019;65(2):184-90.

23. Tekcan A. Kalkaneal Spur hastalığı ile metabolik bozukluklar arasındaki ilişki. Haydarpaşa Numune Eğitim ve Araştırma Hastanesi Tıp Dergisi.141.

24. Gasparyan AY, Ayvazyan L, Mukanova U, Yessirkepov M, Kitas GD. The platelet-to-lymphocyte ratio as an inflammatory marker in rheumatic diseases. Ann Lab Med. 2019;39(4):345.

25. Kim H-A, Jung J-Y, Suh C-H. Usefulness of neutrophil-to-lymphocyte ratio as a biomarker for diagnosing infections in patients with systemic lupus erythematosus. Clin Rheumatol. 2017;36:2479-85.

26. Nazik H, Nazik S, Çoban FG. Nötrofil lenfosit ve platelet lenfosit oranlarının aktif Behçet hastalarındaki önemi. Bozok Medical Journal.33.

27. Şirin FB, Şirin MC. Kan dolaşımı enfeksiyonlarının erken tanısında inflamatuvar belirteçlerin değerlendirilmesi. SDÜ Tıp Fakültesi Dergisi. 2021;28(2):291-9.