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Increased Calcaneal Spur Frequency in Patients with Obesity and Type-2 Diabetes Mellitus

Obez ve Tip-2 Diabetes Mellituslu Hastalarda Artmış Kalkaneal Spur Sıklığı

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Abstract

Objective: Obesity is a risk factor for calcaneal spur (CS) formation. Diabetes mellitus may contribute to the risk of CS due to the decreased ability of tissue repair and peripheral neuropathy. Thus, we aimed to determine the frequency of CS in asymptomatic obese subjects with and without type 2 diabetes mellitus (T2DM). Also, we tried to find the related factors of CS in patients with T2DM.

Material and Methods: Ninety-three obese patients with type-2 diabetes mellitus (T2DM) and 42 obese control subjects without glucose metabolism disturbances were enrolled in the study. A questionnaire was made about the duration of T2DM, age, and occupations of the participants. Physical, laboratory, and x-ray examinations of all cases were made. SPSS 15.0 was used for statistical analyses.

Results: Patients with T2DM and control groups were similar in mean age and body mass index (BMI) (p=0.196, p=0.073, respectively). The existence of CS in the T2 diabetic group was significantly higher than in the control group (p=0.023). T2 diabetic patients with CS had significantly higher neuropathy existence, mean age, and BMI than T2 diabetic patients without CS (p=0.001, p=0.016, p=0.043, respectively). There was a positive correlation between the existence of CS and peripheral neuropathy and age (p=0.025, p=0.001, respectively).

Conclusion: Increased ratio of CS in obese T2 diabetic patients may be important because of the relation between CS existence and complications of diabetes mellitus. Clinicians should pay more attention to increased frequency of CS in patients with T2DM, to avoid diabetic foot complications and other malformations.

Key Words: Calcaneal spur, obesity, type-2 diabetes mellitus, neuropathy

Özet

Amaç: Obezite, kalkaneal spur (KS) oluşumu için bir risk faktörüdür. Diabetes mellitus ise, doku tamir yeteneğinde azalma ve periferik nöropati nedeni ile kalkaneal spur gelişimi için risk oluşturabilir. Bu çalışmadaki amacımız, tip 2 diyabeti (T2DM) olan ya da olmayan asemptomatik obez hastalarda KS sıklığını belirlemektir. Bunun yanında, tip 2 diabetes mellitus hastalarında KS oluşumu ile ilgili diğer faktörleri araştırmayı amaçladık.

Gereç ve Yöntemler: Çalışmaya, tip 2 diabetes mellitusu bulunan 93 obez hasta ve glukoz metabolizma bozukluğu bulunmayan 42 obez kontrol grubu hasta dahil edildi. Hastaların tip 2 diabetes mellitus süresi, yaş ve meslekleri ile ilgili anket düzenlendi. Hastaların fizik muayene, laboratuvar ve radyolojik değerlendirmeleri yapıldı. İstatistiksel analiz için Statistical Package for the Social Sciences (SPSS) 15,0 kullanıldı.

Bulgular: Tip 2 diabetes mellitusu bulunan hastalarda ve kontrol grubunda ortalama yaş ile Body Mass Indeksleri (BMI) benzerdi (p=0,196, p=0,073). Tip 2 diabetes mellitusu bulunan hasta grubunda kalkaneal spur sıklığı anlamlı olarak yüksekti. Kalkaneal spur ile periferik nöropati ve yaş arasında pozitif korelasyon mevcuttu (p=0,025, p=0,001).

Sonuç: Diabetes mellitus komplikasyonları ile kalkaneal spur mevcudiyeti arasındaki ilişkiden ötürü, tip 2 diabetes mellitus hastalarındaki artmış kalkaneal spur sıklığı önemli olabilmektedir. Klinisyenler diyabetik ayak ve diğer ayak malformasyonları gibi komplikasyonları önlemek amacı ile tip 2 diabetes mellituslu hastalarda artmış KS sıklığına daha fazla dikkat etmelidirler.

Anahtar Kelimeler: Kalkaneal spur, obezite, tip 2 diabetes mellitus, nöropati

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Introduction

Plantar fasciitis (PF) is a deteriorating syndrome of the plantar fascia (1). Several authors found various incidences of PF, ranging from 8% to 88% of unselected populations (2). PF is reported to be the most common cause of inferior heel pain in adults (3) and responsible for approximately 1 million physician visits per year in the USA (4). Heel spur syndrome (calcaneal spur syndrome), painful heel syndrome, runner's heel, subcalcaneal pain, calcaneodynia, and calcaneal periostitis are the synonyms of PF (5).

The origin of plantar fascia is on the calcaneus (1), and the formation of subcalcaneal spur has traditionally been attributed to repetitive longitudinal traction of the plantar fascia (6) and also to hamstring tightness (7). Some histological and clinical studies suggest that vertical compressive forces may play a more important role (8-10). Case-controlled studies have identified obesity or sudden weight gain, reduced ankle dorsiflexion, pes planus, and occupations that require prolonged weight-bearing as the greatest risk factors associated with CS syndrome. One study reported that individuals with body mass index (BMI) >30 kg/m^2 had an odds ratio of 5.6 for PF compared to those with a BMI ≤25 kg/m^2 (10). Existence of plantar fasciitis in diabetic patients is controversial. In some studies, no association was found between CS and diabetes mellitus (11,12). In a recent study, it was found that individuals with diabetes mellitus may suffer from CS syndrome as a result of peripheral neuropathy, leading to muscle atrophy, changes in anatomical structure of the feet (claw toes, pes cavus or high arches, prominent metatarsal heads, etc.), and functional alterations in gait (10). Conventional x-rays are the gold standard in the diagnosis of a CS; usually, lateral pictures of the calcaneus are taken. They show a calcified spur on the inferior side of the calcaneus (13).

Since patients with T2DM are usually obese and although both obesity and T2DM seem to be risk factors of CS syndrome, there is no knowledge about the frequency of CS syndrome in obese patients with T2DM (14). The objective of this study was to identify the frequency of CS in obese patients with T2 diabetes and understand if there is any difference of CS frequency between diabetic and non-diabetic obese subjects. Furthermore, we aimed to determine the potential related factors with CS in these patients.

Material and Methods

Setting

The study was conducted at the Department of Endocrinology and Metabolism, Gülhane School of Medicine, Ankara, Turkey in June 2009-September 2009. All participants were informed, and written consent was obtained. The study protocol was approved by the local ethical committee of Gülhane School of Medicine (123/11.11.2008).

Participants

To be eligible for inclusion, participants had to be obese (BMI ≥30 kg/m^2), type 2 diabetic, 18 years old or older, able to provide informed consent, and answer the questionnaire. Control group subjects had to be obese (BMI ≥30 kg/m^2) and free from glucose metabolism disturbances. Exclusion criteria included: pregnancy, previous radiotherapy to the foot, previous trauma to the foot (fracture, rupture of tendon), chronic inflammatory diseases, vascular diseases, malignant diseases, and lymphatic edema. Ninety-three obese, type 2 diabetic patients and 42 obese patients, as a control group free from glucose metabolism disturbances, were examined.

Design

A questionnaire was made about the age, duration of diabetes, and occupations of participants. In this cross-sectional study, after 12 hours of overnight fasting, venous blood samples withdrawn from the antecubital vein of patients were taken between 08.00-09.00 AM, and all patients underwent full medical examinations and laboratory assessments to rule out any metabolic and hormonal disorders, with the exception of diabetes mellitus and obesity. Control cases were subjected to the 75 g oral glucose tolerance test to exclude glucose metabolism disturbances.

Measures

Biochemical analyses were performed in the Core Laboratory of the Biochemistry Department. Fasting and 120-minute plasma glucose (mg/dL) was analyzed by the enzymatic colorimetric method with the Olympus AU 600 autoanalyzer using reagents from Olympus Diagnostics, GmbH (Hamburg, Germany). Glycosylated hemoglobin levels were tested by the high-performance liquid chromatography method. Neurological examination of all diabetic patients and local physical examination of patients who had CS were made. Peripheral neuropathy was assessed by thermal threshold testing for hot and cold in the left foot (Thermal Threshold Tester; Medelec, Old Woking, Surrey, UK), vibration threshold in the left medial malleolus and left great toe (Biothesiometer; Biomedical Instrument, Newbury, OH), and microfilament test. Peripheral nerve abnormalities were defined as 95% of the normal range in a non-diabetic adolescent control group (15). Standing height and body weight were measured in light indoor clothing without shoes. Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). The diagnosis of obesity was defined as body mass index (BMI) ≥30 kg/m^2. Calcaneal x-rays were performed using the Canon CXDI-40EC device, and all x-rays were examined by the same experienced radiologist who was blinded to subjects.

Statistical Analysis

Database management and all statistical analyses were performed by using Statistical Package for the Social Sciences (SPSS) for Windows (version 15.0, SPSS, Chicago, IL, USA). For each descriptive variable, the mean, standard deviation, frequency, and proportion of the total study population with that variable were calculated. To examine variables by CS and sex, categorical variables were analyzed using the χ^2 test, continuous variables were analyzed using the t test, and continuous variables with skewed distributions were analyzed using the Mann-Whitney U-test. Correlations between variables were calculated via Spearman correlation test. A significance level of 0.05 was used in all analyses.

Results

The characteristics of each group are depicted in Table 1. There were no significant differences between the sex (male/female) (p=0.717), mean age (years) (p=0.196), and mean BMI (kg/m^2) (p=0.073) for each group. Occupations of the participants were similar.

The fasting glucose (mg/dL) level of the diabetic group was significantly higher than the control group (p<0.001). CS existence was significantly higher in patients with T2DM than in the control group (p=0.023). Seven (7.5%) patients with T2DM have suffered from heel pain.

Table 2 shows the characteristics of T2DM patients with/without CS. There were no differences in mean duration of T2DM (years) and glycosylated hemoglobin (%) levels between the subgroups (p=0.859, p=0.839, respectively). Mean age (years), BMI (kg/m^2), and peripheral neuropathy existence were higher in patients with CS (p=0.001, p=0.016, p=0.043, respectively).

There were positive correlations between the existence of CS and peripheral neuropathy (r: 0.229, p=0.043) and age (years) (r: 0.336, p=0.01) in diabetics, but no correlation was found between the existence of CS and glycosylated hemoglobin levels (%) (p=0.461) and diabetes duration (years) (p=0.721) (Table 3).

Table 1. Characteristics of groups

	T2 Diabetic group	Control group	p value
Patients (M/F)	93 (34/59)	42 (14/28)	0.717
Age (years)	59±10.5	55±8.0	0.353
BMI (kg/m^2)	35.1±4.3	33.1±3.3	0.306
HbA1c (%)	7.60±1.2	-	
Fasting glucose (mg/dL)	150.4±46.0	86.5±8.3	0.0001
Diabetes duration (years)	10.1±8.4	-	
Neuropathy positive	49 (52.7%)	-	
Calcaneal spur-positive	72 (77.4%)	24 (57.1%)	0.023

BMI: body mass index

Table 2. Characteristics of diabetic subgroups

	Calcaneal spur present (n=72)	Calcaneal spur absent (n=21)	p value
Age (years)	60.7±9.3	52.0±12.3	0.001
BMI (kg/m^2)	37.1±6.1	34.5±3.6	0.016
Diabetes duration (years)	10.3±8.2	9.9±8.8	0.859
Neuropathy-positive	42 (58.3%)	7 (33%)	0.043
HgA1c	8.7±1.8	7.8±1.6	0.839

BMI: body mass index

Table 3. Correlations between CS and neuropathy, glycosylated hemoglobin levels, diabetes duration

	Calcaneal spur (r, p)
Neuropathy	0.229, 0.025
HbA1c (%)	-0.107, 0.461
Diabetes duration (years)	0.038, 0.721
Age (years)	0.336, 0.001

Discussion

Our findings showed that the range of CS in diabetic obese patients was 77% and that CS was seen more frequently in obese patients with T2DM than non-diabetic obese patients (77% vs. 57%). Age and BMI of type 2 diabetic patients who have CS were significantly higher. CS in obese patients with T2DM was mostly asymptomatic. There were positive correlations between CS existence and neuropathy and age.

CS range has been previously reported as 11%-16% in young to middle-aged healthy populations (16). The range of CS (77%) in our obese patients with T2DM is in accordance with Bassiouni, who reported a 72% incidence rate of CS in patients with rheumatologic disorders above the age of 61 (17). Existence of plantar fasciitis in diabetic patients is controversial. In some studies, no association was found between CS and diabetes mellitus (11,12). In a recent meta-analysis, plantar fasciitis was reported as the most common cause of heel pain and plantar fascia thickness, as measured by ultrasonography, which is the most widely reported imaging feature of this condition. Diabetes mellitus was reported as a risk factor for CS in this meta-analysis. Thickening of the plantar fascia has been shown in patients with T2DM (18). We found an increased ratio of neuropathy in diabetic patients who have CS. Similar to our study, another study showed that plantar fascia thickness in type 1 diabetic patients predicts subsequent development of retinopathy, elevated albumin excretion rate, peripheral neuropathy, and autonomic neuropathy (19). Histopathologically, the heel spur is a fibroostosis promoted by mechanical stress to the plantar aponeurosis, slowly and continuously growing into its insertion region (13). Diabetic neuropathy causes a progressive alteration in muscle tropism, especially in the intrinsic foot/ankle muscles, an increase in joint rigidity, and an alteration of the collagen structure in the fascia and muscle tendons due to collagen cross-linking and non-enzymatic glycosylation of keratin. This means that muscles, cartilage, tendons, and ligaments will have structural changes, which will culminate in a limitation of foot mobility (20,21). Only 7 of our patients (7.5%) had symptoms that may be related to CS. Peripheral neuropathy may prevent the sensation of pain caused by CS. In a previous study, authors reported that 16% to 40% of subjects with CS were asymptomatic (10,13,17). In these studies, patients were not fully diabetics, and the absence of neuropathy may cause such a difference.

We have found increased BMI and age in diabetic patients who had CS. Obesity is a well-recognized risk factor for heel pain (5). One previous study, conducted in military recruits, has reported a positive association between increased bodyweight and CS (22). This association is consistent with the vertical compression hypothesis of spur formation, as several studies have shown that vertical heel pressure during gait is strongly associated with bodyweight (22,23). Excess body mass may accel-

erate the degenerative processes occurring in the plantar heel region, particularly in the presence of age-related stiffening and decreased elasticity in the heel pad (24,25). However, it is also possible that obesity results in greater flattening of the medial longitudinal arch, which then creates additional traction on the plantar fascial insertion and subsequent spur development. Regarding our patients' range of age, the current study's results are similar to those of Menz et al. (9), who reported a 55% prevalence of CS in patients whose mean age was 75 years. Banadda et al. (26) have demonstrated a linear increase in the prevalence of CS across five age bands, ranging from 11 years to over 51 years. Diabetes duration and HbA1c levels were not different within the T2DM group, with regard to the presence of CS. This can be explained by the fact that it is difficult to predict the time for the onset of T2DM and consequently the duration of the disease. T2DM may have existed in a patient for a long time before the diagnosis. HbA1c may change gradually in patients with lifelong T2DM. HbA1c level reflects 2 or 3 previous months of glycemic control. It is obvious that CS formation takes a longer period of time; one ca nnot relate the present glycemic control with the longstanding CS.

The study has several limitations. Due to the cross-sectional study design, the results may not implicate any temporal relationship. CS may have a relationship with different diabetic complications. These patients may be prone to foot ulcers, and we had no information about the risk of foot ulcers in our patients. In this study, except neuropathy, we did not explore any other complications. Further studies with a larger number of patients and prospective design may give additional information about the possible interactions between CS, foot ulceration, and diabetic neuropathy in T2DM.

Conclusion

The existence of peripheral neuropathy, increasing age, and higher BMI, seem to be the main reasons of CS formation. CS existence seems to be in a relationship with diabetic complications; therefore, obese diabetic patients may be more prone to these complications. For this reason, such patients have to be closely followed up for screening complications of diabetes. In addition to the harmful metabolic effects of obesity, obese diabetics are at increased risk of CS. Therefore, weight reduction should be encouraged in these patients not only for metabolic control but also for the development of CS. Clinicians should pay more attention to the increased frequency of CS in patients with T2DM to avoid diabetic foot complications and other malformations.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Gülhane Military Medical Academy.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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