Eur J Gen Med 2014; 11(2): 90-93
DOI : 10.15197/sabad.1.11. 45

Original Article

Smoking and Lipid Profile in Pilonidal Sinus Patients

Mehmet Ince[1], Halil Atilgan[2], Fatih Ozcelik[3], Erol Arslan[4]

ABSTRACT

The purpose of our study was to determine the risk factors such as lipid profile and smoking on the pathogenesis of sacrococcygeal pilonidal sinus disease (SPD). A total of 26 with SPD and 42 control patients were enrolled for this study. We retrieved data of patients comprising patient age, gender, body mass index (BMI), smoking and lipid profile. Total cholesterol (TC), triglycerides (TG), low density lipoprotein- cholesterol (LDL-C) and high density lipoprotein- cholesterol (HDL-C) were evaluated for lipid profile. Mean age and BMI of SPD and control group were 21.7±1.3 and 34.5±5.7 years (p<0.001); 25.7±1.5 and 26.0±2, 5 kg/m2 (p=0.6), respectively. TC was 163.8±29.0 and 193.6±31.0 mg / dl, LDL-C was 103.6±28,7 and 128.5±27.5 mg /dl, and HDL-C was 41.0±9.0 and 48.0±10.5 mg/dl; (p<0.001), (p<0.001) and (p=0.006), respectively, for SPD group and control group. However, TG was 94.3±47.9 and 83.9±46.3 mg/dl (p=0.3). Smoking was 22/26 (84.6%) and 16/42 (38.1%), (p<0.001), in the SPD group and the control group, respectively. TC, LDL-C and HDL-C values were found lower in the SPD group than the control group, but cigarette smoking is more. However, studies are needed to determine the effect of smoking about HDL value in the SPD group.

Key words: *Smoking, lipid profile, sinus pilonidalis*

Pilonidal Sinüslü Hastalarda Sigara Kullanımı ve Lipid Profili

ÖZET

Çalışmamızda pilonidal sinus hastalığında (PSH) risk faktörü olarak sigara kullanımı ve lipid profilinin patogenezdeki rolünü araştırmayı amaçladık. Çalışmamıza PSH olan 26 ve 42 kontrol olmak üzere toplam 68 olgu alınmıştır. Tüm olguların vücut kitle indeksleri (VKİ), kan lipit seviyeleri ve sigara kullanımları araştırıldı. PSH grubu ve kontrol grubunun sırasıyla yaş ortalaması 21,7±1,3 yıl, 34,5±5,7 yıl (p<0.001), VKİ ortalaması 25,7±1,5 kg/m2, 26,0±2,5 kg/m2 (p=0,6) idi. PSH ve kontrol gruplarında sırasıyla Total-kolesterol (TK) 163,8±29,0 mg/dl, 193,6±31,0 mg/dl (p<0.001), Düşük dansiteli lipoprotein-Kolesterol (DDL-K) 103,6±28,7 mg/dl, 128,5±27,5 mg/dl (p<0.001) ve Yüksek dansiteli lipoprotein-Kolesterol (YDL-K) 41,0±9,0 mg/dl, 48,0±10,5 mg/dl p=0.006) olup, değerler arasında anlamlı fark vardı, fakat ve Trigliserid (TG) 94,3±47,9 mg/dl, 83,9±46,3 mg/dl olup, değerler arasında fark saptamadık (p=0.3). Sigara kullanımı PSH olan grupta 22/26 (%84,6), kontrol grubunda 16/42 (%38,1) olguda saptandı (p<0.001). PSH olan olgularda TK, DDL-K ve YDL-K değerlerini daha düşük, fakat sigara kullanımının daha fazla olduğunu saptadık. Pilonidal sinüs grubunda sigara kullanımının artmış olması YDL-K değerinin düşüklüğü için bir etkendir, fakat PSH olan olgularda total kolesterol değerini düşüren bir etken olup olmadığını belirlemek için daha fazla olgunun katıldığı çalışmalara ihtiyaç vardır.

Anahtar kelimeler: *Sigara, lipid profili, pilonidal sinüs*

[1]*Konya Military Hospital, Department of General Surgery, Konya, Turkey,* [2]*Beytepe Military Hospital, Department of General Surgery, Ankara, Turkey,* [3]*Gulhane Military Medical Academy, Department of Biochemistry, Ankara, Turkey,* [4]*Gulhane Military Medical Academy, Department of Internal Medicine, Ankara, Turkey.*

Correspondence: *Mehmet Ince,*
Konya Military Hospital, Department of General Surgery. Kazim Karabekir Cd. 42020 Meram, Konya, Turkey
Tlf and Fax: *90 332 321 7358 / 90 332 321 54 08* ***Mobile:*** *90 505 818 7038*
E-mail: *drmince@yahoo.com*

Received: 09.04.2013, Accepted: 06.06.2013

INTRODUCTION

Sacrococcygeal pilonidal disease (SPD) is a well recognized source of a common surgical problem affecting primarily white men between puberty and their early thirties and a linkage between the disease and various occupations has been demonstrated (1). Incidence of SPD was found 6.1% (2) and % 8.8 in the Turkish soldiers (3). For a long time, the cause of SPD has been a matter of debate. Until the second half of the 20th century, basing their theories on the study of the human embryo, many authors proposed a congenital origin of the lesion (4, 5). But, nowadays; it widely accepted that SPD is an acquired disorder (6, 7).

Male gender, obesity, occupations or sports requiring sitting, a deep natal cleft, excessive body hair, stiff or coarse hair, poor body hygiene and excessive sweating are described as the primary risk factors for SPD (8). Rate of SPD increase after puberty but it is seldom after 40 years-old (9-11). This can be explaining that increased sex hormones after puberty lead to secretion of pilosebaceous glands. However, there is a noted lack of prospective studies in the literature that analyze and confirm these risk factors except obesity. Sondenaa et al found that significant association as %37 between obesity and SPD (12). There are some studies generally focused on the role of obesity evaluating risk factors for SPD (13, 14). However, there is a noted lack of studies in the literatures that analyze and confirm risk factors which are smoking and lipid profile.

The purpose of our study was to determine the clinical value and quantitative effect of significant these risk factors on the pathogenesis of SPD. The results will help to form evidence-based counsel for the prevention of the disease and could be showed SPD is not only a surgical and local but also a metabolic disease.

MATERIALS AND METHODS

A total of 26 patients with SPD and 42 control patients were enrolled for this study that was conducted at the General Surgery Department, Konya Military Hospital, Konya and the Beytepe Military Hospital, Ankara, Turkey between June 2012 and December 2012. We retrieved data of patients' characteristics comprising patient age, gender, body mass index (BMI), smoking and lipid profile. Patients who had SPD and patients who had other disease or applying for check-up were assigned as Group A and group B, respectively. The study was designed as a case-control and retrospective trial. The study protocol was approved by the ethics committee. The patients older than 40 year-old and used anti-lipid drugs were excluded from the study. Total cholesterol (TC), triglycerides (TG), low density lipoprotein-cholesterol (LDL-C) and high density lipoprotein-cholesterol (HDL-C) were evaluated for lipid profile. The number of cigarettes consumed per day was considered to be as smoking.

Statistical analysis

The statistical analysis was carried out by using Statistical Package for Social Sciences (SPSS), version 15.0. Data were presented as mean ± standard deviation and percentage. Independent samples t-test was used to compare continuous variables. Chi-square and Fisher's exact test was used to compare for categorical ones. Significance was defined as $p<0.05$.

RESULTS

In this study, data of 26 patients with SPD and 42 control patients were evaluated from chart. All patients and controls were white and male. Mean age and BMI of groups were 21.7±1.3 and 34.5±5.7 years ($p<0.001$); 25.7±1.5 and 26.0±2, 5 kg/m2 ($p=0.6$), respectively. Smoking was 22/26 (84.6%) and 16/42 (38.1%), ($p<0.001$) in the SPD group and the control group, respectively. TC was 163.8±29.0 and 193.6±31.0 mg/dl, LDL-C was 103.6±28,7 and 128.5±27.5 mg/dl, and HDL-C was 41.0±9.0 and 48.0±10.5 mg/dl; ($p<0.001$), ($p<0.001$) and ($p=0.006$), respectively, for SPD group and control group. However, TG was 94.3±47.9 and 83.9±46.3 mg/dl ($p=0.3$).

DISCUSSION

Risk factors such as positive family history and age are not modifiable for SPD. However, TC, LDL-C and HDL-C values were found lower in the SPD group than the control group, but cigarette smoking is more.

SPD is a simple disease but it leads to high rates of postoperative complication and recurrence. Incidence of SPD is reportedly 26 per 100 000 population and it affects predominantly young adults of working age (12, 15). Therefore SPD causes to loss manpower especially in young adults. If predisposal factors and high risk

group of patients are known, preventive precautions could be applied before SPD is occurred.

Although many years have passed since SPD was first discovered by Hodge (16) in 1880, the etiology of PS has remained a problematic aspect of medicine (17). It is more common in men and in hirsute people (18). Hair is the main initiative agent in all theories that attempt to explain the pathogenesis of SPD, which may be the reason for the male predomination of the disease. Karydakis (19) and Bascom (20) offer the best explanations of pathogenesis of SPD that is an acquired disease.

Obesity has been reported as an important risk factor and to be related to higher rates of postoperative complication and recurrence in SPD (21, 22). Cubukcu et al (12) did not detect a statistical significance between BMI and SPD. In our study, there was no significantly difference between groups in BMI. Clothier et al (23) showed that the onset of SPD coincides with puberty and it is rare after age 40. Also, Can at al (24) found that SPD is more common in male subjects between the ages of 20 and 25 years. Among the patients in the present study, mean age of patients was between these ranges, while mean age of control was not.

Some studies shown that plasma HDL-C level tend to be lower in smokers than in non smokers (25). HDL-C level showed statistically significant different in SPD patients as compared to controls in this study. Martin et al (26) found that LDL-C/HDL rates were higher in smokers as compared to that of controls. However, there is no difference between groups for this rate (2.5 vs 2.6). Additionally, there was no significantly difference between groups in TG. Our study is important. Because, this is the first study evaluating the lipid profile in patients with SPD. Smokers were high in the study data is noteworthy, also. Smoking causes some diseases such as cardiovascular problems and cancer.

In conclusion, more cigarettes used is a risk factor which reduces the value of HDL-C in patients with SPD. We think that it would be appropriate to question the use of smoke in patients with SPD. But, more studies are needed to determine the effect of smoking about TC value in the SPD group.

REFERENCES

1. *Chintapatla S, Safarani N, Kumar S, Haboubi N. Sacrococcygeal pilonidal sinus: historical review, pathological insight and surgical options. Tech Coloproctol 2003; 7: 3-8.*
2. *Sekmen U, Kara VM, Altıntoprak F, Şenol Z. Pilonidal sinus in the army: Its incidence and risk factors. National Surg J 2010; 26: 95-8. (Turkish)*
3. *Akinci OF, Bozer M, Uzunköy A, Düzgün SA, Coşkun A. Incidence and aetiological factors in pilonidal sinus among Turkish soldiers. Eur J Surg 1999; 165: 339-42.*
4. *da Silva JH. Pilonidal cyst: cause and treatment. Dis Colon Rectum 2000: 43:1146-56.*
5. *Gage M. Pilonidal sinus: an explanation of its embryologic development. Arch Surg 1935; 31:175-89.*
6. *Davage ON. The origin of sacrococcygeal pilonidal sinuses: based on an analysis of four hundred sixty-three cases. Am J Pathol 1954; 30:1191-205.*
7. *Bascom J. Pilonidal sinus: origin from follicles of hairs and results of follicle removal as treatment. Surgery 1980: 87:567-72.*
8. *Hull TL, Wu J. Pilonidal disease. Surg Clin North Am 2002; 82:1169-85.*
9. *Clothier PR. Haywood IR. The natural history of the postnatal pilonidal sinus. Ann R Coll Surg Engl 1984; 66: 201-3.*
10. *Klass AA. The so-called pilonidal sinus. Can Med Assoc J 1956; 75: 737-42.*
11. *Price ML. Griffiths WAD. Normal body hair- a review, Clin Exp Dermatol 1985; 10: 87-97.*
12. *Sondenaa K, Andersan E, Nesvik I, Soreide JA. Patients characteristic and symptoms in chronic pilonidal sinus disease. Int J Colorectal Dis 1995; 10: 39-42.*
13. *Cubukçu A, Carkman S, Gönüllü NN, Alponat A, Kayabaşi B, Eyüboğlu E. Lack of evidence that obesity is a cause of pilonidal sinus disease. Eur J Surg 2001;167:297-8.*
14. *Cubukçu A, Gönüllü NN, Paksoy M, Alponat A, Kuru M, Ozbay O. The role of obesity on the recurrence of pilonidal sinus disease in patients,who were treated by excision and Limberg flap transposition. Int J Colorectal Dis 2000; 15:173-5.*
15. *Buie LA, Curtiss RK. Pilonidal disease. Surg Clin North Am 1952; 32: 1247-59.*
16. *Hodge RM. Pilonidal sinus. Poston Med Surg J 1880; 103:485-6.*
17. *Doll D, Friederichs J, Dettmann H, et al. Time and rate of sinus formation in pilonidal sinus disease. Int J Colorectal Dis 2008; 23: 359-64.*
18. *Louis AB. 1890-1975: Jeep disease (pilonidal disease of mechanized warfare). Dis Colon Rectum 1982; 25:384-90.*
19. *Karydakis GE. New approach to the problem of pilonidal sinus. Lancet 1973; 2:1414-5.*
20. *Bascom J. Pilonidal sinus: origin from follicles of hairs and results of follicle removal as treatment. Surgery 1980:87:567-572.*

21. Arda IS, Guney LH, Sevmis S, Hicsonmez A. High body mass index as a possible risk factor for pilonidal sinus disease in adolescents. World J Surg 2005; 29:469-71.

22. Sakr M, El-Hammadi H, Moussa M, Arafa S, Rasheed M. The effect of obesity on the results of Karydakis technique for the management of chronic pilonidal sinus. Int J Colorectal Dis 2003; 18:36-9.

23. Clothier PR, Haywood IR. The natural history of the postanal pilonidal sinus. Ann R Coll Surg Engl 1984; 66: 201-3.

24. Can MF, Sevinc MM, Yilmaz M. Comparison of Karydakis flap reconstruction versus primary midline closure in sacrococcygeal pilonidal disease: results of 200 military service members. Surg Today 2009; 39:580-6.

25. Bain, C. & Jesse M.J. Cigarette consumption and deaths from coronary artery disease. Lancet 1978; 1:1087-8.

26. Martin M.J, Hulley S.B. Serum Cholesterol, B.P & mortality. Lancet; 1986; 2:933-36.