The effects of smoking on tendon thickness and degeneration in young amateur sportspeople

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ABSTRACT

The aim of this study was to assess the effects of smoking on tendinopathies detected by ultrasound in active amateur sportspeople.

Fourteen male, 19 female, total 33 amateur sportspeople (mean age, 20.5±2.5) were investigated. Both patellar and Achilles tendons were imaged by high resolution ultrasound. The antero-posterior thicknesses and pathologies (if present) of the tendons were determined.

Twenty two (66.7%) of the sportsmen were nonsmokers and 11 (33.3%) were smokers. The mean number of cigarettes smoked in a day was 9.4±4.5; mean duration of cigarette smoking was 3.7±1.6 years. 17 tendinopathies (12.9% of tendons) were detected in 12 sportsmen (36.4% of sportsmen). No significant difference was found between the smoker and nonsmoker groups according to the incidence of tendinopathies *(p>0.05).* The thickness of the tendons did not differ according to smoking status for both patellar and Achilles tendons (p>0.05 for all comparisons).

In conclusion, the tendon thickness and incidence of tendinous degeneration do not differ between smoker and nonsmoker sportsmen.

Keywords: Ultrasonography, musculoskeletal ultrasound, patellar, Achilles tendon, smoking.

ÖZET

Sigaranın genç amatör sporcularda tendon kalınlığı ve dejenerasyonu üzerine etkileri

Bu çalışmanın amacı, sigara içiminin aktif amatör sporcularda ultrasonografi ile saptanan tendinopatiler üzerindeki etkilerini değerlendirmektir.

On dördü erkek, 19'u kadın, toplam 33 amatör sporcu (ortalama yaş 20.5±2.5 yıl) incelendi. Patellar ve aşil tendonları yüksek çözünürlüklü ultrason ile görüntülendi. Tendonların ön-arka kalınlıkları ölçüldü ve –varsa- tendinopati sıklıkları belirlendi.

Sporcuların 22 (%66.7)'si sigara içmeyen, 11'i (%33.3) sigara içen kişilerdi. Günlük ortalama içilen sigara sayısı 9.4±4.5; ortalama sigara içme süresi 3.7±1.6 yıl idi. Toplam olarak 12 sporcuda (%36.4), 17 adet tendinopati (tendonların %12.8'u) saptandı.

Sigara içen ve içmeyen gruplar arasında tendinopati sıklığı bakımından fark gözlenmedi (p>0.05). Tendon kalınlığı hem patellar hem de Aşil tendonları için sigara içip içmeme durumuna bağlı bir değişiklik göstermedi (p>0.05).

Sonuç olarak, tendon kalınlığı ve tendon dejenerasyon sıklığı sigara içen ve içmeyen sporcular arasında bir farklılık göstermemektedir.

Anahtar kelimeler: Ultrasonografi, kas-iskelet ultrasonu, patellar, Aşil tendonu, sigara içimi

INTRODUCTION

The etiology of tendon pathologies is multifactorial, including mechanical or anatomical factors, acute or chronic traumas, increasing age, previous injury, rheumatologic disease, hypercholesterolemia and

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hypovascularity[1-4]. Repetitive microtraumas cause degenerative changes in tendons in sportsmen. The Achilles tendon and patellar tendon are the tendons most often injured. This repetitive exposure to excessive strain damages the tendon at a microscopic level. A reparative process begins, but if exposure to excessive tendon strain persists, the tendon becomes disrupted macroscopically. The normal healing process of the tendon is then disturbed and a cycle of chronic tendinopathy and symptoms ensues[3-6].

Acute tobacco smoke inhalation reduces blood flow to various structures such as the skin, muscle, carotid artery, brain, liver, intestines and rectum[7]. Another impact of smoking is that nicotine impairs the synthesis of collagen, which is required for tendon repair. Both hypovascularity and immature collagen synthesis inhibits or delays tendon repair[1,8]. Although the harmful effects are well known, it is thought that smoking is common among sportsmen; however, due to the fact that smoking is inconsistent with the pursuit of sport, smoker sportsmen tend to keep it a secret. Consequently, it seems that the exact incidence of smoker sportsmen cannot be determined. In Turkey, a study indicated that the incidence of smokers among the students of a Sports and Physical Education High School was 53%; 20% of them smoked 17-20 cigarettes per day, 15% of them smoked 6-11 cigarettes per day, 12% of them smoked more than 20 cigarettes and 6% of them smoked 1-5 cigarettes per day[9].

Our hypothesis is that, if smoking is a risk factor for the development of tendinopathies, it is expected that the incidence of tendinopathies should be higher in smoker athletes. The tendon thicknesses and the incidence of tendinopathies were been compared between smoker and nonsmoker sportspeople. Ultrasonography (US) is relatively inexpensive, noninvasive, repeatable, accurate, provides no radiation, and is commonly used to evaluate the tendon's morphology and paratendinous structures [4,6,9-12]. Using color and power Doppler imaging, neovascularization, the growth of new blood vessels due to the degeneration of the tendon, can be assessed [13-15].

MATERIAL AND METHODS

The study protocol was approved by the Ethical Committee of the Faculty of Medicine at Trakya University. Informed consent was obtained in each case.

Subjects, questionnaire and physical examination:

14 male and 19 female, for a total of 33 active amateur sportspeople in the Sports Academy at Trakya University were studied. All the participants were asymptomatic and randomly selected from Sports Academy at Trakya University. The sportsmen's subdivision, age, gender, height, weight, sport age, duration (hour) and frequency (per week) of training were recorded. Histories of possible steroidal performance-enhancing drugs and smoking status were asked to each athlete. Smoking age (year) and the number of the cigarettes (per day) were recorded for smokers. Sportsmen with pain or swelling on the knee or ankle were excluded.

Ultrasonographic assessment:

All subjects were examined by the same experienced musculoskeletal radiologist using a 13.4 Mhz linear transducer (Siemens Medical Solutions Acuson X300, USA). US of the Achilles and patellar tendons were performed with the ankle and knee flexed 90˚ in order to avoid bow-stringing of the tendons. No stand off medium was required. The heels were examined in the transverse and sagital plane. A US abnormality was defined as a) a hypoechoic region evident in both the longitudinal and the transverse scans, b) a fusiform swelling without hypoechoic areas, c) peritendinous (Infrapatellar or Kager's fat pad's) soft tissue changes, or d) altered vascularity on power Doppler US.

Maximum thickness of the tendons was measured at the level of the lower pole of the patella and at the middle third of the Achilles tendon on the longitudinal axis. Presence or absence of any US abnormalities was recorded. Color and power Doppler were used to detect blood vessels entering the tendon and to detect

blood flow within the tendon. For assessment of the distribution of tendon microtears, the Achilles and patellar tendons were nominally divided into thirds according to overall tendon length.

Statistical analysis:

The Student's t test, Mann-Whitney U test and Chi-square test were used to compare the tendinopathy (+) and tendinopathy (-) cases. A p value <0.05 was considered statistically significant. Statistica 7.0 statistical software was used in the analysis.

RESULTS

Descriptive data:

Eleven football, 7 volleyball, 5 basketball, 5 athletism, 2 tennis, 1 handball, 1 judo and 1 wrestling players were included. Mean age was 20.48±2.48 years (median 20.00, minimum 17, maximum 30), mean sport age was 8.28±3.30 years (median 8.0, minimum 2, maximum 18), mean frequency of training was 2.97±1.1 times per week (median 3, minimum 2, maximum 6); mean duration of training was 94.84±38.02 minutes (median 90, minimum 30, maximum 180). There was no history of usage of a steroidal performance drug. 22 (66.7%) of the sportsmen were nonsmokers and 11 (33.3%) of them were smokers. Mean number of cigarettes smoked in a day was 9.38±4.53 (median 7, minimum 6, maximum 20); mean duration of cigarette smoking was 3.71±1.60 years (median 3, minimum 2, maximum 6).

Imaging findings and anthropometric data:

A total of 132 tendons of 33 sportsmen were visualized by ultrasound imaging.

Mean thickness of both sided patellar and Achilles tendons in female and male sportspeople are presented in table 1.

Table 1. Mean thickness (mm) of both side patellar and Achilles tendons athletes.

	Women (n=19)	**Men (n=14)**
Patellar tendon (mm):		
Right	2.84±0.38	3.41±0.85
Left	2.98±0.52	3.52±1.0
Mean	**2.91±0.45**	**3.47±0.93**
Achilles tendon (mm):		
Right	3.79±0.72	4.54±1.4
Left	3.96±0.62	4.5±0.79
Mean	**3.88±0.67**	**4.52±1.1**

17 tendons (12.9%) had sonographically detected pathologies. Among them, 14 tendinopathies in the patellar tendon and 3 tendinopathies in the Achilles tendon were detected. 13 tendinous hypoechogenicities and 2 peritendinous soft tissue abnormalities were detected for the patellar tendon. 3 of the patellar tendon lesions were in the middle third; the rest were located at the proximal third of the tendon. No increased vascularity was found. For the Achilles tendon, 2 tendinous hypoechogenicities (Figure 1), 2 peritendinous soft tissue abnormalities and 2 altered vascularities (Figure 2) were found. Both tendinous pathologies were in the middle third of the tendon.

Mean age, BMI (kg/m²), sport age, frequency and duration of training and *p* values of all subjects, with and without tendinopathy, are listed in table 2. There was a significant correlation between the thickness of the tendon and BMI (*p=0.003).*

Figure 1: Sagittal sonography of the Achilles tendon with hypoechoic region.

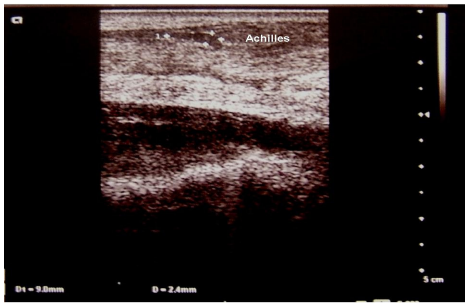

Figure 2: Doppler sonography of the Achilles tendon shows vascularity in peritendinous soft tissues.

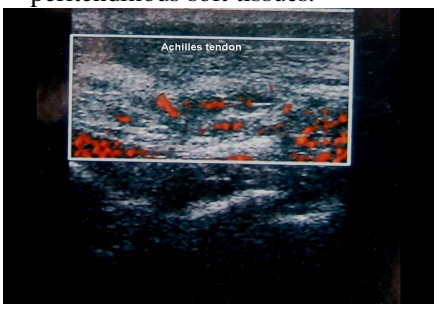

Table 2. Mean age, BMI (kg/m²), sport age (year), frequency and duration of training and *p* values of all subjects, with and without tendinopathy.

	Total	***Tendinopathy (-) (n=21)***	***Tendinopathy (+) (n=12)***	**p**
Age	20.48±2.48	19.95±1.47	21.42±3.52	*0.193* †
BMI (kg/m²)	21.3±1.00	20.19±1.77	22.41±2.22	*0.003* †
Sport age (year)	8.28±3.30	7.5±3.27	9.58±3.01	*0.084* †
Frequency of training (per week)	2.97±1.1	2.91±1.38	3.0±0.92	*0.431* ‡
Duration of training (minutes)	94.84±38.02	93.0±32.13	98.18±48.54	*0.948* ‡

† Student's t test; ‡ Mann-Whitney Test

The number of tendinopathies in the nonsmoker group (n=22) was 8 (36.4%); in the smoker group (n=11) it was 4 (36.4%). A total of 17 tendinopathies (12.9% of tendons) were detected in 12 sportsmen (36.4% of sportsmen) with US. No significant difference was found between the smoker and nonsmoker groups according to the incidence of tendinopathies *(p=0.653)*. The thicknesses of the tendons were not statistically different in smoker and nonsmoker sportsmen (Table 3).

Table 3. Mean thickness of the tendons in nonsmoker and smoker groups.

Tendon thickness (mm)	**Nonsmoker (n=22)**	**Smoker (n=11)**	***p***
Right patellar	3.15±0.70	2.95±0.64	*0.443*
Left patellar	3.2±0.94	3.2±0.43	*0.925*
Right Achilles	4.08±1.30	4.18±1.3	*0.802*
Left Achilles	4.27±0.82	4.05±0.55	*0.407*

Eight (57.1%) of the men (n=14) had tendinopathy and six (42.9%) did not. In the female group (n=19), four sportsmen (21.1%) had tendinopathy and 15 (78.9%) of them had no tendinopathy. There was no significant difference between genders *(p=0.078)*.

DISCUSSION

Tendinous injuries are more frequently seen in athletes than the normal population [5,6,10]. In a large cohort of asymptomatic elite basketball, football and cricket players, Cook et al[5]. reported that asymptomatic US hypoechoic regions were present in 22% of tendons imaged and in 14% of those who had never symptoms. Overuse and repetitive microtrauma causes microtears and degenerative changes in the tendons of sportsmen. A reparative process begins, but if exposure to excessive tendon strain persists, the number of microtears increases, resulting in tendon hypertrophy. This would also explain why an athlete's tendon is often thicker than a non-athlete's tendon. More than half of complete ruptures experienced by athletes occur without any prior warning signal or symptoms; however, many tendon problems are partly due to a predisposing element, namely degenerative changes before the major trauma[13]. These microdefects and the tendon's rupture tend to be more common in the middle third of the Achilles tendon and proximal and distal enthesial attachments of the patellar tendon. These areas are a relative vascular watershed[1,3,13,16]. The blood supply of these watershed areas of the tendon is primarily via the paratenon vessels. Injury to this vascular supply as the result of repetitive paratendinitis or the failure of vessels to adequately penetrate the centre of a hypertrophied tendon may result in poor tendon healing in athletes. Therefore, in athletes, the tendon's degeneration may be accelerated as the result of both increased microtear production and a decreased rate of microdefect repair[17]. The role of vascular insufficiency in the tendon has a less well-defined contribution. The significant correlation of these factors with tendinopathy suggests the importance of their effect on microvascularity in the development of tendinopathies[1]. Obesity, hypertension, and steroids are predisposing factors for tendinopathies because of their end-organ effect of a diminution of local microvascularity[18]. Smoking is another cause of microvascular disease on all tissues [7, 19-23]. It is expected that smoking also disturbs the perfusion of the tendons. However, the effect of smoking on tendons has not yet been well established. One theory behind the high incidence of rotator cuff tears in the shoulder is that the tendon contains a zone relatively avascular in the area proximal to its insertion to greater tuberosity. A loose relation was reported between shoulder pain and tobacco use, although the reason for this is not known. It is likely due to the fact that smoking causes microvascular disease and results in a worsening of the relative disvascularity in the critical zone of the rotator cuff[1]. Mallon et al.[8] stated that nonsmokers undergoing rotator cuff repair have a greater improvement of pain and better results postoperatively than smokers. A cadaveric study showed that the presence of a macroscopic rotator cuff tear was nearly twice (22/32) more in a smoker than a non-smoker (10/32), as judged by lung pathology. In addition, there is a strong trend toward the presence of higher grades of tendon degeneration among smokers as compared with non-smokers[1].

Recently, impaired wound healing associated with smoking has been shown in different studies. The complication rates and delayed wound healing were increased in smoker patients who underwent myocutaneous flap reconstruction[22]. Nicotine inhibits the revascularization of bone grafts in spinal fusions. It would seem that cigarette smoking could impair healing because it negatively impacts vascularity, as the nicotine from cigarette smoking has been implicated as a major vasoconstrictor. Another effect of smoking on wound healing is that tobacco impairs synthesis of collagen. The amount of collagen deposition and repair at the wound was negatively correlated with the consumption of tobacco. Smokers had 1.8 times less mature collagen in their surgical wounds[9]. Abnormally thickened tendons have an abnormal collagen fiber structure, an arrangement with lower collagen concentrations but an increased extracellular matrix called myxoid degeneration[3]. Consequently, it is expected that not only

repetitive microtraumas and reparation cycles, but also vascular insufficiency and collagen synthesis failure, affects the thickness of tendons or facilitates the degeneration of tendons in smoker sportsmen. In our study, degeneration was observed mainly in the watershed areas of both tendons, which supports the notion of vascular insufficiency. We did not encounter another study focusing on the incidence of tendinopathy among amateur sportsmen in the literature. As we expected, the incidence of tendinopathies of our sportsmen group was less than in the literature in which professional elite sportsmen were investigated (Cook et al.[5] found 22%, Gibbon et al.[17] found 18% and Fredberg et al.[12] found 18%). Tendon thickness and the incidence of tendinopathies did not differ between the smoker and non smoker sportmen groups. The fact that our sportsmen's sport age was less and the frequency and duration of training was not intensive enough should be considered in interpreting these findings.

There are several limitations of this study. Firstly the subject numbers is low. We could not obtain reliable information about the nicotine concentration that the subjects consume. We did not evaluate the reliability and method error of the ultrasound measures.

Steroid usage on the tendon is another risk factor for tendinopathies[2,18]. As the number of our athletes who has steroidal performance drugs in their history was low, we cannot draw a conclusion about the effects of steroids.

Unfortunately, smoking is a reality among sportsmen, despite its harmful effects. Smoking acts as an accelerator factor for tendon degeneration on account of its negative effects on microvascular perfusion and collagen synthesis on soft tissues. This study reveals that the tendon's thickness and incidence of tendinous degeneration does not differ between smoker and nonsmoker sportsmen. However authors believes that future research may well prove this to be correct. Further studies need to be conducted among elite sportsmen (indeed, smoking is unacceptable for sportsmen) or middle-aged people, among whom tendon degeneration is probably more obvious.

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