

Exercise training can help recovery of impaired aortic elasticity at young smokers

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SUMMARY

Although it has been shown that increased aortic stiffness and decreased aortic strain (AS) in smoker individuals, and improved aortic elastic properties with exercise training (ET), there is no satisfied evidence revealing how the regular ET influences this harmful effect of smoking. We aimed to evaluate this effect in young adults in this study. 75 smoker and 36 non-smoker individuals who stated that hadn't done regular exercise previously were enrolled to study. Individuals who were not able to perform exercise or changed smoking habit were excluded. Body mass indexes (BMI), systolic and diastolic blood pressures (BP) were recorded. Aortic systolic (ASD) and diastolic diameters (ADD) were measured from the same view on the M-mode tracing at a level of 3 cm above the aortic valve on echocardiographic examination. Hence AS, aortic distensibility (AD) and β stiffness index (β SI) were calculated. Then all of the individuals were underwent exercise protocol. After exercise period, the parameters mentioned above measured again and aortic strain and distensibility recalculated. Before ET, there were no any significant difference in age, BMI, BP and pulse pressure, interventricular septum thickness and LV ejection fractions between two groups. However AS and AD in smokers were significantly lower and β SI was higher than those of controls. AS, AD and β SI of both groups improved with the ET.

This study revealed that smoking affects aortic strain and distensibility, indicators of atherosclerotic process, adversely even in young ages similar to shown in previously. However regular exercise can help to recovery of aortic elastic properties.

Egzersiz genç sigara içicilerde bozulmuş aortik elastisitenin düzelmesine yardım edebilir

ÖZET

Sigara içenlerde aortik strain (AS) ve distensibilite (AD) azalma ile egzersizin aortik elastik özelliklerini iyileştirdiği bilinmekle birlikte düzenli egzersizin sigaranın olumsuz etkisine nasıl biretke ettiğine dair yeterli kanıt mevcut değildir. Biz bu çalışmada genç erişkinlerde bu etkiyi ortaya koymayı amaçladık. Daha önce düzenli egzersiz yapmadığını ifade eden sigara içen ve içmeyen 36 erkek birey çalışmaya alındı. Vücut kitle indeksi (VKİ), sistolik (SKB) ve diastolik kan basınçları (DKB) kaydedildi. Ekokardiyografide M-Mod ile alınan görüntülerden aort kapağının 3 cm üzeri seviyeden aortik sistolik (ASÇ) ve diastolik (ADÇ) çapları ölçüldü. Böylece aortik elastisite parametreleri olan AS, AD ve β sertlik indeksi (β SI) değerleri hesaplandı. Dahasonrabireyler, egzersiz sürecine tabi tutuldu. Egzersiz sonrasında bahsedilen parametreler tekrar ölçüldü. Egzersiz öncesi bulgulardaya, VKİ, SKB, DKB, nabız basıncı, IVS kalınlıkları ve sol ventrikül EF açısından herhangi bir farklılık görülmedi. Bununla birlikte, AS ve AD sigara içicilerde kontrol grubuna göre belirgin olarak düşük, β SI ise yüksekti. Egzersiz dönemi sonrasında, AS ve AD değerlerinde düzelme görüldü. Bu çalışma, sigaranın aterosklerotik sürecin göstergeleri olan aortik strain ve distensibilite değerlerini genç yaşlarda bile etkilediğini ortaya koymuştur. Bununla birlikte düzenli egzersiz aortik elastik özelliklerinde iyileşmeye yardımcı olabilir.

Anahtar kelimeler: Egzersiz, Sigara, Aortik strain ve distensibilite

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Introduction

Atherosclerosis is a long lasting process that begins before teenagers (1). Many risk factors, including smoking, increase and accelerate this process even in young ages (1,2). Therefore, the primary prevention strategies are the most important part of the treatment of this process. Exercise training (ET) and life habit changes have been the first step of preventive treatment recommendations according to the results of many studies (3-6).

Aortic stiffness, measured by aortic strain (AS) and distensibility (AD), is an independent non-invasive parameter for indicating atherosclerotic process at aorta which is important for conduit function of cardiovascular system (2,7,8). Although it has been shown that aortic stiffness increased and AS decreased in smoker individuals (9), and aortic elastic properties improved with ET (10,11), there is no satisfied evidence revealing how the regular ET affects this harmful effect of smoking. In this study we aimed to evaluate this effect in young adults.

Materials and Methods

Retrospectively data about baseline and post exercise routine echocardiographic examination of 130 healthy young male individuals who reported that no having previous regular exercise habit extracted from archive records in 2008 and 2009 of our outpatient clinic. Then these parameters combined with data about exercise protocol records of their sportive club. Individuals were classified as two groups according to their smoking habits. Initially, 87 smoker and 43 non-smoker subjects were enrolled to the study. But after combination with exercise records, 12 smoker and 7 non-smoker ones had to be excluded, because of non-cardiovascular reasons, including not able to perform ET protocol (due to orthopedic challenges) or changed smoking habit. Finally, 75 smoker (age $20,64 \pm 1,06$) ($12,4 \pm 4,35$ cig./day > one year; mean $2,6 \pm 1,26$ years) and 36 non-smoker (never smoke) (age $20,83 \pm 1,35$) individuals were enrolled.

TABLE 1: Baseline characteristics

	Smokers (n=75)	Nons-smokers (n=36)	p
Age (years)	20,64 $\pm$ 1,06	20,83 $\pm$ 1,35	0,398
Pulse rate (bpm)	72,16 $\pm$ 8,54	73,22 $\pm$ 9,24	0,452
Family history of CAD (n (%))	9 (%12)	5 (%13,8)	0,088
Height (m)	1,73 $\pm$ 0,086	1,74 $\pm$ 0,078	0,537
Weight (kg)	67,51 $\pm$ 4,24	69,12 $\pm$ 4,12	0,366
BMI (kg/m²)	22,55 $\pm$ 2,61	23,11 $\pm$ 2,66	0,387
Systolic BP (mmHg)	122,24 $\pm$ 6,17	120,92 $\pm$ 5,46	0,324
Diastolic BP (mmHg)	6,67 $\pm$ 5,12	74,96 $\pm$ 4,92	0,226

CAD: Coronary artery disease; BMI: Body mass index; BP: Blood pressure.

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Data about heights, weights, body mass indexes (BMI) (kg/m²), systolic (SBP) and diastolic (DBP) blood pressures and family histories of premature vascular disease were extracted. Local ethics committee approved this observational retrospective study. Routine examinations of individuals were as following. All baseline records were made at the beginning of training. Then, echocardiographic examination performed for all individuals. While subjects were on left sided position, aortic systolic (ASD) and diastolic diameters (ADD) were measured from the same view on the M-mode tracing at 3 cm above the aortic valve in left parasternal long axis views by using 3S probe. Hence the AS, AD and β stiffness index (β SI) were calculated as described previously (7,12).

The formulas to calculate these parameters were; $AS(\%) = (ASD - ADD) / ADD \times 100$; $AD(cm^2/dyn/106) = (2 \times AS) / (SBP - DBP)$; and $\beta SI = \ln(SKB/DKB) / AS$. By the way, interventricular septum thickness, left ventricular diameters and ejection fractions (by Teichholz formula) were also measured. None of subjects had any valvular or nonvalvular cardiovascular disease, or took any medication.

Then individuals undergone ET protocol. During this period, all individuals lived in the same place day and night, and were nourished with the same meal about 4000 kcal/d. All the subjects' physical activities, including chin-up (5-9 a day), push-up (25-40 a day), sit-up (30-50 a day), physical-fitness exercises (10-25 min) and 3 km running, were recorded each day. After ET period, the parameters of echocardiographic examination mentioned above were repeated similarly; and AS, AD and β SI were recalculated.

Data were expressed as mean $\pm$ SD or percent. Student's t-test or Mann Whitney-U test were used for comparison of the groups. Statistical analyses were performed by using SPSS 11.0 for Windows. P value <0.05 was considered as significant.

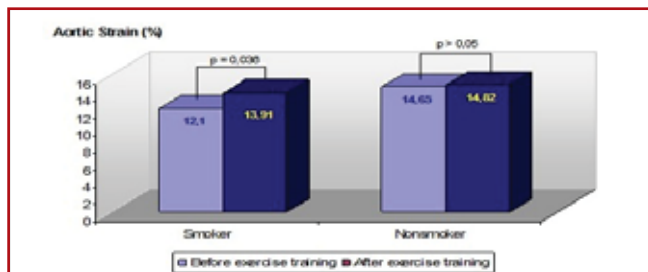


Figure 1: Improving of the AS with regular ET in smoker and nonsmoker subjects.

Results

Study population consisted of 75 smoker and 36 non-smoker subjects. Before ET, there were no any significant differences in ages, BMI's, SBP/DBP and pulse pressures, IVSd thicknesses and LV ejection fractions between two groups as shown in the table 1. However AS and AD in smokers were significantly lower and β SI was higher than those of controls (Table 2). The total exercise was 67 $\pm$ 14 minutes daily; 3-5 times a week during the average of 5,3 months. This ET mostly included isometric exercise and this comes to mean that moderate (20-50%) or high (>50%) maximal voluntary contraction and mostly moderate (40-70%) maximal O₂ uptake in according to previously defined sports classification (13).

After the ET period, AS and AD were also slightly lower and β SI was slightly higher in smokers (Table 2). However, this significance of difference was subsided. By the way, the blood pressures and the BMI's were also slightly (but not significant) lower in both groups. On the other hand, AS, AD and β SI of

both groups improved with the ET. However, the improvements of AS and AD were statistically significant in smoker subjects (14,96%, p=0,036; and 17,99%, p=0,024 respectively), while were not significant in non-smoker group (1,16%, p>0,05; and 1,86%, p>0,05). (Figure 1 and 2). The decrease of β SI was also similar (in smokers 14,91%, p=0,021; and in nonsmokers 2,42%, p>0,05).

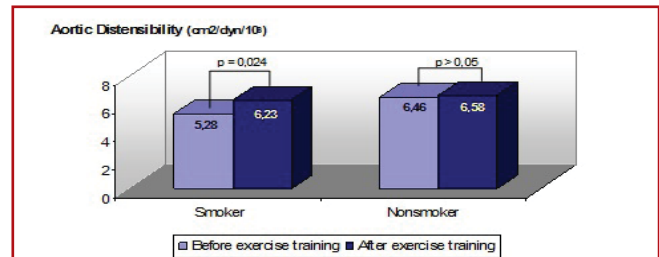


Figure 2: Improving of the AD with regular ET in smoker and nonsmoker subjects.

Discussion

The aorta has ability to absorb the pulse generated by the left ventricular ejection which is essential for integrity of cardiovascular system. Aortic stiffness has been studied to show whether the great vessels are involved and to obtain information about the pathophysiology of the great arteries in many diseases(14-16). Accordingly, it was shown that aortic stiffness increased in patients with hypertension, diabetes and in postmenopausal women.

Smoking is one of cardiovascular risk factors especially at youngs. Smokers are at risk of atherosclerotic plaque rupture at earlier ages more than nonsmokers (1). The results those we found that the AS and AD in smokers were lower than that in non-smokers are similar to that of previous studies. Sassalos et al.(9) suggested that smoking had both acute and chronic detrimental effects on ascending AD. Indeed, AS and AD measurements were lower than our results in these studies (14); but these differences are probably due to different population ages. Because, it is demonstrated that the AS and AD change with age (17); low in infants, improve gradually to peak during the ages of 10 to 15, and decrease with age thereafter.

Regular physical activity has beneficial effects on lipid levels, blood pressure, insulin sensitivity, obesity, haemostasis and endothelial functions. Increased level of physical activity leads to improve these factors simultaneously. Cardiac output, the product of stroke volume and heart rate, increase during exercise. ET results in cardiac chamber enlargement and the accompanying ability to generate a large stroke volume. The aorta also exposes to a significant hemodynamic load during exercise. The effect of prolonged physical ET on the mechanical properties of aorta has been well established in also some animal models (18). It was reported that these effects especially related to the amounts and qualities of arteriel connective tissue fibrous proteins. The elasticity parameter, contents of elastin and collagen were higher and the content of calcium in elastin was lower in training rats than sedantary ones. Nytric oxyde synthase (eNOS) phosphorylation. Especially long lasting training has beneficial effects on endothelium dependent dilation and NO synthase gene expression (10). Besides, a recent study revealed that physical activity increases the production and circulating numbers of endothelial progenitor cells via a partially NO-

dependent antiapoptotic effect, inhibits neointima formation, and enhances angiogenesis that can potentially underlie exercise-related beneficial effects on cardiovascular disease (23). Dysregulation of vasoregulatory molecules, such as soluble epoxide hydrolase, calcitonin receptor-like peptide, and complement factor H are considered to be an important aspect of the complex cardiovascular response to chronic smoke exposure (24). Other mechanisms through which smoking may decrease AD include sympathetic activation and inflammation (25). It has been suggested that rapid loss of the ability of endothelium to generate NO, which enhances oxidative stress via the reactive oxygen species react with NO to form peroxynitrite and mitochondrial DNA damage, can help for explaining acute endothelial dysfunction (26). We speculate that the increasing of the NO production can improve

especially impaired endothelium dependent dilation. Previous studies showed that both active and passive chronic smokers had decreased endothelium-dependent dilation, which was potentially reversible after smoking cessation (27). In our study, impaired dilation of aorta in smoker individuals significantly improved with regular ET. Although AD, AS and β SI changed with the ET even in non-smoker group the ET had significant beneficial effect in a diseased condition. That the number of control group was smaller can be accepted as a limitation. We had only limited individuals in the same population. An important limitation of our study was also unmeasured lipid profiles. It is well known that HDL-cholesterol is significantly lower and LDL-cholesterol is mildly higher in smokers (28); and the ET increases HDL-cholesterol, decreases LDL and total cholesterol levels.

TABLE 2: Changing of characteristics with exercise training

	Before exercise		p	After exercise		
	Smokers (n=75)	Nonsmokers (n=36)		Smokers (n=75)	Nonsmokers (n=36)	p
BMI (kg/m ²)	22,55±2,61	23,11±2,66	0,387	21,17±2,48	21,82±2,66	0,328
Systolic BP (mmHg)	122,24±6,17	120,92±5,46	0,324	120,16±6,01	120,53±5,56	0,533
Diastolic BP (mmHg)	76,67±5,12	74,96±4,92	0,226	74,83±5,12	73,72±4,96	0,387
Pulse Pressure (mmHg)	46,43±3,25	46,17±3,33	0,454	45,64±3,12	46,67±3,38	0,217
IVSd thickness (mm)	9,25±1,24	9,15±1,12	0,267	9,52±1,16	9,63±1,14	0,294
LV Ejection fraction (%)	67,52±4,36	68,03±4,23	0,346	70,03±4,66	70,82±4,02	0,334
ASD (mm)	27,22±1,79	27,72±2,37	0,390	27,82±1,79	27,93±2,24	0,518
ADD (mm)	24,30±1,70	24,17±2,57	0,452	24,42±1,67	24,27±2,51	0,401
Aortic strain (%)	12,10±3,41	14,65±4,23	0,006	13,91±3,26	14,82±4,17	0,047
Aortic distensibility (cm ² /dyn/10 ³)	5,28±1,70	6,46 ±2,16	0,0032	6,23±1,58	6,58±2,33	0,043
β Stiffness index	3,89±1,24	3,26±1,12	0,012	3,31±1,32	3,23±1,17	0,062

BMI: Body mass index; **BP:** Blood pressure; **IVSd:** Interventricular septum diastolic thickness; **LV:** Left ventricular; **ASD:** Aortic systolic diameter; **ADD:** Aortic diastolic diameter.

However we think that the improving of the AS and AD in five months can not be attributed to the changing of lipid parameters even if it may contribute a little. Another limitation was to not excluded the non-smoker subjects who had probably been exposed to smoke passively during previous life. But we may speculate that if this was possible, study results may have been more significant. But we excluded the subjects if he had smoke in any time in his previous life or if he reported his smoking habit during exercise. The other limitation was that study population consisted only of young male subjects; so claiming that our results can be valid for other population ages is disputable.

Conclusion

This study revealed that smoking affects aortic strain and distensibility adversely even in young ages similar to shown previously. However regular exercise partly neutralized this harmful effect of smoking. Investigations explaining how the exercise can do these effects are still needed.

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