

The Effect of Cigarette Smoking on Blood Pressure and Hypertension

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Abstract

Cigarette smoking and hypertension are significant cardiovascular risk factors. The effect of cigarettes on blood pressure and the development of hypertension are unclear. Studies on the subject report contradictory results. The purpose of this study was to investigate the effect of cigarette smoking on blood pressure and hypertension. Our study population consisted of 712 patients with a mean age of 51.2±15.1 years, 44% of them were male and 56% of them were female. Thirty-six percent of patients were smokers, 9% of them were ever smoked and stopped smoking or ex-smoker and 55% of them were nonsmoker. Systolic and diastolic blood pressures were lowest in the smokers, higher among ex-smokers compared to smokers and highest in the non-smokers ($P<0.001$). The mean systolic and diastolic blood pressures were 119/74 mmHg in the smokers, 134/81 mmHg in the ex-smokers and 150/88 mmHg in the non-smokers. Prevalence of hypertension did not change by smoking status. A significant variation was determined, at 15% in smokers, 33% in ex-smokers and 55% in non-smokers ($P<0.001$). In conclusion, blood pressures and prevalence of hypertension were significantly low in smokers in this study. Despite the limitless harm it has, cigarette smoking exhibits a lowering effect in blood pressure, although the mechanism involved is unclear.

Keywords: Blood pressure; cigarettes; hypertension

1. Introduction

Cigarette smoking and hypertension are the two most important long-term risk factors for atherosclerosis, coronary artery disease, acute myocardial infarction and sudden death (1,2). Cigarette smoking alone causes the deaths of

5.4 million people per year across the world (3). Although studies have investigated the effect of cigarette smoking on blood pressure and the development of hypertension, the mechanism involved is still unclear (4). The acute effect of cigarette smoking is a temporary increase in

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Received: 14-05-2013, Accepted: 28-05-2013, Published: 07-06-2013

doi:10.7575/aiac.abcm.v.1n.1p.6 URL: <http://dx.doi.org/10.7575/aiac.abcm.v.1n.1p.6>
<http://www.abcm.v.1n.1p.6>



heart rate and blood pressure with an increase in epinephrine and norepinephrine production due to activation of the sympathetic system (5). It has been suggested that long-term cigarette smoking raises blood pressure by causing an increase in inflammation, endothelial dysfunction, plaque progression and vascular damage. Some studies support this view. An increase in blood pressure and development of hypertension in cigarette smokers compared to non-smokers has been reported in some studies (6,7). Other studies, however, have reported less development of hypertension in non- or ex-smokers compared to smokers (8,9). Studies have also reported that cigarette smoking does not affect development of hypertension (10,11). Since the relationship between cigarette smoking and hypertension is unclear, current guidelines do not recommend quitting smoking in the treatment and prevention of hypertension (12-13). The relationship between cigarette smoking and hypertension during pregnancy period is similarly unclear. While some studies have emphasized that cigarette smoking increases pregnancy-induced hypertension (14), others have reported that cigarette use reduces the development of hypertension (15).

The purpose of this study was to investigate the effect of cigarette smoking on blood pressure and the development of hypertension and to contribute to this still controversial issue.

2. Methods

2.1 Patients

In a cross-sectional study, 712 consecutive patients applied to outpatient clinic of chest disease of Educational and Research Hospital of Recep Tayyip Erdoğan University, Rize, Turkey,

between 01 February and 30 April, 2013, were enrolled. Patients' age, height, weight, body mass index (BMI) and blood pressure were measured. Cigarette smoking status was determined. Drugs used were recorded for those patients with a diagnosis of hypertension. Pregnant patients, patients aged under 20 or over 80 years, patients with previous coronary artery, peripheral vascular, cerebrovascular disease, active infection, cancer, heart failure, atrial fibrillation, moderate or severe heart valve disease, malnutrition and renal or hepatic failure were excluded. The study was carried out in accordance with the principles in the Declaration of Helsinki and was approved by the local Ethics Committee.

2.2 Blood pressure

Systolic and diastolic blood pressures (BP) were measured twice at 5 min intervals from the left arm after at least 15 min of rest. BP values were obtained by the traditional auscultatory method using a sphygmomanometer (Erka, Germany) in an office. The means of the two measurements were recorded as mean systolic and diastolic blood pressure. Mean blood pressure (MAP) was calculated using the following formula: $MAP = \text{diastolic blood pressure} + \frac{1}{3} (\text{systolic blood pressure} - \text{diastolic blood pressure})$.

2.3 Blood measurements

Blood samples were drawn by venipuncture to measure routine blood chemistry parameters after fasting for at least 8 h. Biochemical parameters including glucose, blood urea nitrogen, creatinine, hepatic function tests and lipid profile were determined using standard methods. BMI was determined using the following formula: $BMI = \text{weight (kg)} / \text{height}^2$



(m).

2.4 Definitions

Smokers were defined as subjects who had smoked at least 100 cigarettes in their life period and who were still smoking. Ex-smokers were those who had smoked at least 100 cigarettes in their life period and stopped the smoking for at least 1 year. Non-smokers had smoked fewer than 100 cigarettes in their life period or who had never smoked at all. Diagnosis of hypertension was based on systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg. Hypertension was defined as longstanding in patients with previous diagnosis of hypertension on antihypertensive therapy. New hypertension was defined in those without any previous diagnosis of hypertension who were diagnosed with hypertension during the study period.

2.5 Statistical analysis

The SPSS statistical software (SPSS for windows, version 16; SPSS Inc., Chicago, IL, USA) was used for all statistical calculations. Continuous variables were defined as mean \pm standard deviation (SD) and categorical variables were defined as percentages. Independent-Samples T test and analysis of variance (ANOVA) were used for parametric variables at comparison of means and Chi square test in the comparison of levels. Univariate analysis and analysis of covariance were used in order to eliminate the effect of age and BMI in order to determine whether cigarette smoking status is an independent predictor factor affecting the blood pressure. $P < 0.05$ was regarded as statistically significant.

3. Results

In the present study, 712 patients were

included. The mean age was 51.2 ± 15.1 years (range 20-80); 310 (44%) participants were male and 402 (56%) were female. In terms of smoking status, 254 (36%) patients were still smoking, 65 (9%) were ex-smokers and 393 (55%) had never smoked. Smoking level in the smoking group was 28 ± 17 pack-years, and 32 ± 15 pack-years in the ex-smokers group. The amount smoked was higher among the ex-smokers, although the difference was not statistically significant ($P = 0.15$). The mean time since quitting among ex-smokers was 7.9 ± 7.5 years (range 1-30 years). The characteristics of the three groups established on the basis of smoking status are shown in Table 1. Males were in the majority in the smoker and ex-smoker groups, while women predominated significantly in the group that had never smoked ($P < 0.001$). Age was lower among the smokers compared to the ex-smoker and non-smoker groups, at 47.1 ± 12.7 , 56.1 ± 13.6 and 53.4 ± 16.9 years, respectively ($P < 0.001$). Ex-smokers and non-smokers were comparable in terms of age ($P = 0.384$). Systolic, diastolic and mean blood pressures in the smoking group were significantly lower compared to the ex- and non-smoker groups ($P < 0.001$). In addition, blood pressure levels were significantly lower in the ex-smokers compared to those who had never smoked ($P < 0.001$). The differences between the groups' blood pressures are shown in Figure 1. Non-smoking group BMI (30.0 ± 6.0 kg/m²) was higher than those in the smoking (26.4 ± 4.7 kg/m²) and ex-smoker (27.2 ± 4.4 kg/m²) groups ($P < 0.001$). The smoking and ex-smoker groups were similar in terms of BMI ($P = 0.57$). Covariate and univariate analyses were performed due to the presence of variations between the groups in terms of



age and BMI and the effect of these factors was thus eliminated. The analysis revealed that cigarette-smoking status affected blood pressure as an independent factor. The differences in systolic, diastolic and mean blood

pressures between the smokers and the ex-smokers at analysis of covariance were $P=0.007$, $P=0.03$ and $P=0.01$, respectively. Between the smoker and non-smoker groups these were $P<0.001$ for all three parameters.

Characteristics	Smokers	Ex-smokers	Non-smokers	P value
Patient number	254	65	393	-
Age	47.1±12.7	56.1±13.6	53.4±16.9	<0.001*
Sex (M/F)	163/91	53/12	94/299	<0.001**
Systolic BP (mmHg)	119±23	134±26	150±29	<0.001*
Mean BP (mmHg)	89±17	99±16	109±19	<0.001*
Diastolic BP (mmHg)	74±14	81±13	88±15	<0.001*
BMI (kg/m ²)	26.4±4.7	27.2±4.4	30.0±6.0	<0.001*
Longstanding HT-Male (yes/no)	13/149	8/45	27/66	<0.001**
Longstanding HT-Female (yes/no)	10/82	1/11	103/196	<0.001**
Total longstanding HT (yes/no)	23/231	9/56	130/263	<0.001**
New HT-Male (yes/no)	9/154	9/44	20/74	0.001**
New HT-Female (yes/no)	8/83	2/10	68/231	0.01**
Total new HT (yes/no)	17/237	11/54	88/305	<0.001**
All HT (yes/no)	40/214	20/45	218/175	<0.001**

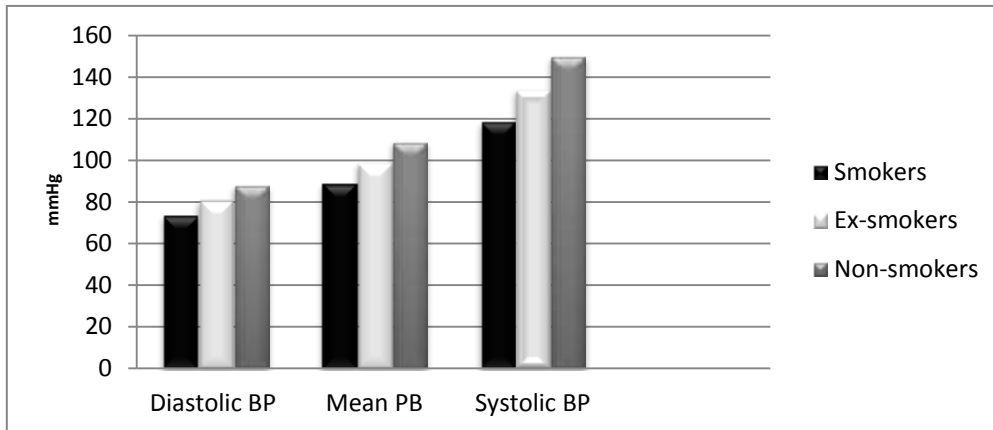
Table 1: Comparison of characteristics of groups established on the basis of smoking status M, male; F, female; BP, blood pressure; BMI, body mass index; HT, hypertension. *ANOVA performed; **Chi-square test performed

The level of patients previously diagnosed with hypertension and receiving antihypertensive therapy was 9% in the smokers, 14% among ex-smokers and 33% in those who had never smoked. When the groups were compared using the chi-square test, hypertension levels were lower among smokers compared to ex- and non-smokers ($P<0.001$). In addition, hypertension levels were lower among ex-

smokers compared to non-smokers ($P<0.001$). Levels among patients with no previous diagnosis of hypertension and who were diagnosed at examination at the clinic were 6% among smokers, 17% in ex-smokers and 22% among non-smokers. The lowest level was among smokers and the highest among non-smokers, a significant difference being observed with the chi-square test ($P<0.001$). The

Figure 1: Comparison of blood pressures in three studied groups

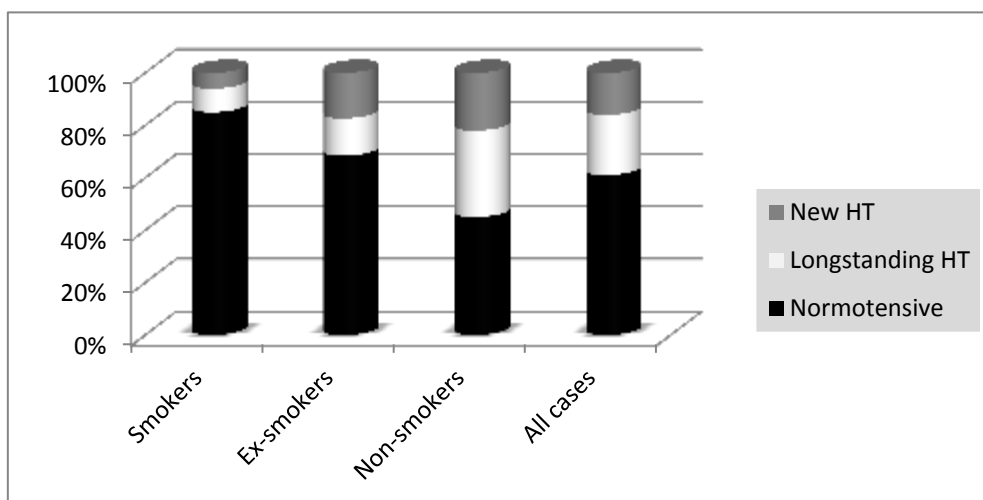




prevalence of hypertension on the basis of cigarette smoking status is shown in Table 1 and Figure 2. In terms of all cases, longstanding and new, the prevalence of hypertension was 16%

among smokers, 31% among ex-smokers and 55% among non-smokers. Hypertension was determined in 278 (39%) of the total 712

Figure 2: Hypertension levels in groups established on the basis of smoking status



patients enrolled. Men and women exhibited similar characteristics in terms of previously and newly diagnosed hypertension levels.

Diastolic, mean and systolic blood pressures in the smoking group were 74, 89 and 119 mmHg, respectively. Diastolic, mean and systolic blood pressures in the ex-smoker group were 81, 99 and 134 mmHg, respectively. Diastolic, mean and systolic blood pressures in the non-smoker group were 88, 109 and 150 mmHg, respectively.

New hypertension was detected in 17 (6%) of

the 254 smokers, longstanding hypertension in 23 (9%) and no hypertension in 214 (85%). New hypertension was present in 11 (17%) of the ex-smokers, longstanding hypertension in 11 (17%) and no hypertension (76%) in 45. New hypertension was present in 88 (22%) of the 393 non-smokers, longstanding hypertension in 130 (33%) and no HT in 175 (45%). New hypertension was present in 116 (16%) of all 712 cases, longstanding hypertension in 162 (23%) and no hypertension in 434 (61%).

4. Discussion



Although many studies have been performed, the effect of cigarette smoking on blood pressure and development of hypertension is still unclear (4). Mutually incompatible results have been obtained from wide-ranging, prospective studies (6-10). Chronic cigarette smoking was shown to lead to endothelial dysfunction in a study by Li et al. (16), to atherosclerotic plaque formation in a study by Sharrett et al. (17), and to arterial rigidity in a study by Kim et al. (18). It has been suggested that chronic cigarette smoking increases blood pressure and the incidence of hypertension through these mechanisms. In a study involving 12,417 in which only males participated, Halimi et al. showed that smoking increased the risk of systolic hypertension in cases aged 60 and over that continued to smoke (6). They also identified an increased prevalence of hypertension attributed to weight gain in ex-smokers compared to non-smokers. The fact that it involved only males was a major weak point of that study (6). In a study by Bowman et al., 28,236 women were monitored for a mean 9.8 years, and the incidence of hypertension rose, particularly in subjects smoking more than 15 cigarettes per day. The limitation of this important study was that it was performed with women only and therefore its results would not reflect the entire society (7). Yin et al. reported high systolic and diastolic blood pressure and prevalence of hypertension in smokers compared to non-smokers in a study of 1780 patients (19). There are also a limited number of studies reporting that cigarette smoking has no effect on blood pressure. Primates et al. observed no difference in systolic and diastolic blood pressures between smokers, ex-smokers and non-smokers (20).

In this study, systolic and diastolic blood pressures were lower and the prevalence of hypertension significantly less in smokers compared to ex- and non-smokers. The levels were significantly lower in both patients with a previous diagnosis of hypertension and those diagnosed during the study. Similar results were obtained when men and women were analyzed separately. Blood pressures were lower and the prevalence of hypertension significantly lower among ex- compared to non-smokers. Lee et al. monitored 8170 individuals for 4 years in a prospective study and reported a rise in both blood pressure and incidence of hypertension in ex-smokers compared to subjects who still smoked (8). The results of that important study were similar to ours. John et al. reported a higher incidence of hypertension in obese patients among ex-smokers compared to smokers (21). However, no difference was determined among subjects of normal weight. The results of that study to a great extent confirm those of our own.

In a recent study of 679 individuals in Ethiopia, Awoke et al. reported a prevalence of hypertension of 28.8% in non-smokers, 25% in ex-smokers and 15.8% in smokers (22). These data are compatible with our study findings. Prevalence of hypertension in our study was 55% in non-smokers, 33% in ex-smokers and 15% in smokers. The prevalence of hypertension in non-smokers was significantly high. We mainly attribute this to our study population being more obese. The mean age in the study by Awoke et al. was 51.5 and mean BMI was 23.35, compared to a mean age of 51.2 and mean BMI of 28.5 in our study (22). In a recent study involving 10,738 participants providing significant data for Turkey,



Süleymanlar et al. reported a prevalence of hypertension of 32.7% (23). The mean age in their study was 40.5 ± 16.3 , with 55.7% of the population being female (23). Prevalence of hypertension in our study was 39%, while 56% of participants were women. Our population resembled that of Süleymanlar et al. from that perspective (23). However, the age of our participants was higher, at 51.2 ± 15.1 . We attribute the higher prevalence of hypertension in our study to significantly more advanced age. The effect of cigarette smoking on hypertension in pregnancy has also been investigated. Yang et al. performed a retrospective study of nearly 3 million women with single pregnancies and reported that cigarette smoking significantly reduced pregnancy-induced hypertension (15). In another recent study, Rauchfuss et al. reported that cigarette smoking had no effect on the development of pregnancy-induced hypertension (14).

The main limitation of this study is the sample size and selection of the participants from a single outpatient clinic rather than a general population. We used specific patient inclusion criteria to avoid potential confounders.

In conclusion, despite many studies performed, the effect of cigarette smoking on blood pressure and the development of hypertension is uncertain. Our study showed that cigarettes reduce the prevalence of hypertension and lower blood pressure. However, the mechanism underlying this is unclear. The effect of cigarettes on preventing weight gain may be a significant factor. Further research is needed on the subject.

Conflicts of interest

The authors declare that they have no conflict of interest.

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