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Effect of smoking on vital hemodynamic parameters and lipid profile of young smokers

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Abstract

Background and objectives: Tobacco use is associated with cardiovascular, respiratory and peripheral vascular diseases. The short term effects of tobacco smoking on vital hemodynamic parameters and lipid profile of young smoker with increased quantity of smoking is still debatable. The objective of this study was to evaluate the effect of smoking on vital hemodynamic parameters and lipid profile of young smokers.

Materials and methods: The current study was an observational cross sectional study conducted in a tertiary care hospital over a period of 18 months and included smokers and non-smokers. Data on vital hemodynamic parameters like blood pressure, heart rate, oxygen saturation (SPO2) and lipid profile were collected. Chi-square and analysis of variance (ANOVA) tests were used to analyze the data.

Results: A total of 80 smokers and 80 non-smokers were enrolled in the study. Blood pressure, heart rate and mean SpO2 were significantly ($p<0.001$) lower in non-smokers compared to smokers. Breath holding time (BHT) and single breath count (SBC) were higher in non-smokers. Mean values of total cholesterol (T-chol), low density lipoprotein (LDL) and triglyceride (TG) were significantly ($p<0.001$) higher in smokers than non-smokers, while high density lipoprotein (HDL) was significantly low in smokers. SBP, T -chol and TG significantly ($p<0.05$) increased as the quantity of smoking increased.

Conclusion: Smoking is associated with derangement of vital hemodynamic parameters and lipid profile across the age. Anti-smoking campaign should be organized to discourage both personal smoking and smoking in public places.

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Introduction

The tobacco epidemic is one of the biggest public health issue that the world that has ever faced. It kills about 8 million people each year around the world [1]. Over 80% of the 1.3 billion tobacco users worldwide live in low- and middle-income countries. Cigarette smoking is the most common form of tobacco use worldwide. Smoking is causally associated with lower body mass index (BMI), higher level of adiposity and is strongly associated with elevated blood pressure and is also considered a major risk factor for cardiovascular diseases [2-4]. Smoking tobacco is linked to early onset atherosclerosis, increased risk of acute

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myocardial infarction (AMI), stroke, peripheral artery disease, aortic aneurysm and sudden death [5,6]. The main objective of this study was to evaluate cumulative effect of smoking on vital hemodynamic parameter and lipid profile of young smokers.

Material and methods

The current study was an observational cross sectional study conducted at the Department of Respiratory Medicine and Medicine at Sri Aurobindo Medical College and Postgraduate Institute, Indore over a period of 18 months from January 2015 to June 2016. All protocols and procedures were approved by the institutional ethics and scientific committee. Written informed consent was obtained from all participants.

The study included non-smokers and smokers. Inclusion criteria of smokers for enrollment in the study were informed and willing young smokers with no prior history of any chronic disease, age between 20-50 years, body mass index (BMI) of 18-25 kg/m^2 and smoking history of 1-20 pack years. Non-smoker persons attending executive health checkup for yearly routine self-care were enrolled as non-smoker control group. Individuals with comorbidities like diabetes mellitus, hypertension, coronary artery disease or other systemic illness, smoker for > 20 pack year, smoker less than 20 years of age or more than 50 years of age, and alcohol dependence were excluded from study. Both smoker and non-smoker were divided into two age groups namely 20 to 35 years (Group-1) and 36 to 50 years (Group-2). Smokers were divided into 4 groups according to the number of pack years (py) they used to smoke. Groups were: group-A (1-5 spy), group-B (5-10 spy), group-C (10-15 spy) and group-D (15-20 spy).

The study tools used for collecting data were history, physical examination, body mass index, pulmonary function test, vital hemodynamic parameter measurements like - blood pressure, pulse rate, oxygen saturation (SPO2) and lipid profile. Weight (kg) of the participants was measured with a calibrated electronic scale and standing height (cm) was measured with a fixed stadiometer. Blood pressure (systolic and diastolic) was measured using a standard mercury sphygmomanometer in the right arm in a sitting position. Resting heart rate (HR) and percentage oxygen saturation (% SpO2) were measured by a pulse oximeter type SMART CARE model SC 500 B. All the individuals were first explained and demonstrated the methods to perform BHT and SBC. The breath-holding test was carried out as described previously [7]. Individuals were asked to inspire deeply and to stop breathing at the end of inspiration. The counting of the duration of the breath-holding was made by a stopwatch from the beginning of the inspiration to the appearance of reflex contractions of the diaphragm. Single breath count (SBC) was the measurement of how far an individual could count in a normal speaking voice after a maximal effort inhalation [8]. The smoking was quantified by pack-year. It is a unit for measuring the amount a person has smoked over a long period of time. It was calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person smoking. Lipid profile data were collected from the Pathology Laboratory of SAMC and PG Institute. Kit method was used for the estimation of lipids.

The means and standard deviations for the linear groups were calculated and compared using Chi square test. The means across more than two groups were compared using the Analysis of Variance (ANOVA) and p value <0.05 is statistically significant.

Result

A total of 80 smokers and 80 non-smokers were enrolled in the study. Table-1 shows the general characteristics of smokers and non smokers. Age, height, weight and BMI of smokers were not significantly different from that of and non-smokers. Among the smokers, there were 42 (52.5%) and 38 (47.5%) individuals belonged to 20-35 years (Group-1) and 36-50 years (Group-2) age groups respectively while it was 43 (53.75%) and 37 (46.25%) individuals among the non-smokers. Detail age-group specific general characteristics of the enrolled study population are shown in Table-2. The mean weight of smokers aged 36-50 years was significantly ($p<0.05$) more compared to other groups while there was no differences in other variables. Quantity of smoking was significantly ($p=0.0004$) more among the individuals aged 36-50 years compared to that of 20-35 years.

Table-1: *General characteristics of the total study population (N=160)*

Variables	Smoker (n=80) Mean ± SD	Non-smoker(n=80) Mean ± SD	p value
Age (years)	35.85 ± 4.45	36.14 ± 4.27	0.67
Height (cm)	172.17 ± 6.76	171.34 ± 6.04	0.41
Weight (kg)	67.52 ± 7.82	65.43 ± 6.24	0.06
BMI (kg/m^2)	22.75 ± 2.01	22.30 ± 1.81	0.14
SPY	8.69 ± 5.17	-	-

Note: BMI: body mass index; SPY: Smoking pack years

Table-2: *Age group specific general characteristics of the study population (N=160)*

Variables	Group-1 (Age:20-35 yrs)		Group-2 (Age: 36-50 yrs)	
	Smoker (n=42) Mean ± SD	Non-smoker (n=43) Mean ± SD	Smoker (n=38) Mean ± SD	Non-smoker (n=37) Mean ± SD
Age (years)	27.92±4.33	29.53±3.48	43.78±4.57	42.75±5.05
Height (cm)	171±7.17	170.93±6.38	173.34±6.34	171.75±5.71
Weight (kg)	64.52±7.75	64±6.80	70.52±7.89	66.86±5.67
BMI (kg/m^2)	22.10±2.29	21.86±1.67	23.39±1.73	22.73±1.94
SPY*	6.52±5.41	-	10.86±4.93	-

*Note: *p= 0.0004, smoker Gr-1 vs. smoker Gr-2.*

Table-3 shows the difference in the hemodynamic and lipid parameters of smokers and non-smokers. Blood pressure, heart rate and mean SpO2 in non-smokers were significantly (p<0.001) lower than that of smokers. Also, the breath holding time and single breath count were higher in non-smokers. Mean values of T-chol, LDL and TG were significantly (p<0.001) higher in smokers than non-smokers, while HDL was significantly low in smokers compared to non-smokers (40.28±6.79 vs. 45.17±6.84 mg/dl).

Table-3: *Hemodynamic parameters and lipid profile of smokers and non smokers*

Variables	Smokers (n=80) (Mean±SD)	Non-smokers (n=80) (Mean±SD)	p value
SBP (mm Hg)	126.27±5.21	120.07±4.35	
DBP (mm Hg)	81.17±6.57	75.62±4.74	
HR (per minute)	78.75±4.05	74.07±3.48	
SPO2 (%)	97.93±0.92	98.36±0.63	
BHT (seconds)	45.8±8.20	53.46±8.62	
SBC	59.96±15.01	74.52±14.02	< 0.001
T-chol (mg/dl)	179.41±28.41	146.75±24.47	
HDL (mg/dl)	40.28±6.79	45.17±6.84	
LDL (mg/dl)	135.3±21.85	117.65±18.40	
TG (mg/dl)	158.52±33.02	135.52±30.79	

Note: SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate;
SpO2: Oxygen saturation; BHT: breath holding time; SBC: single breath count;
T-chol: total cholesterol; HDL: high density lipid; LDL: low density lipid; TG: triglyceride

Table-4 shows the age-group specific hemodynamic and lipid profiles of smokers and non-smokers study participants. SBP was significantly higher in smokers of both age groups compared to non-smokers. Smokers of age group 36-50 years had also significantly higher SBP compared to smokers of 20-35 years age group indicating that both smoking and increasing age was a risk factor for increased systolic blood pressure. Diastolic blood pressure was almost same in smokers of both age groups. Baseline heart rate was same in smokers while more in case of non-smokers of age group-2 when compared with non-smokers of group-1. Mean SpO2 had least value in smokers of age group-2 indicating decrease in mean SpO2 value with increase in duration and intensity of smoking. There was statistically significant difference (p= 0.03) in breath holding time of smokers in two age groups. BHT was almost same for non-smokers of both groups while it was least for smokers in age group-2 which could be due to increased age as well as increased number of pack years of smoking. Smokers of both age groups had significantly low SBC compared to non-smokers of both groups. The mean values of T-chol, LDL and TG were significantly high in smokers than age matched controls of non-smoker group. The mean values of T-chol, LDL and TG were higher in smokers of age group 36-50 years. HDL was significantly higher in non-smokers compared to smokers of both age groups.

The Table-5 shows the comparison of hemodynamic and lipid profile according to the quantity of smoking in terms of number of pack year. ANOVA test revealed that as the number of pack years increases, the mean value of SBP, T -chol and TG

Table-4: *Age group specific hemodynamic and lipid profiles of smokers and non smokers (N=160)*

Variables	Group-1 (Age:20-35 yrs)		Group-2 (Age: 36-50 yrs)	
	Smoker (n=42)	Non-smoker (N=43)	Smoker (N=38)	Non-smoker (N=37)
SBP(mm Hg)	124.80±5.25	120.55±3.86	127.89±4.65	119.51±4.79
DBP(mm Hg)	81.28±6.82	74.27±3.38	81.05±6.28	77.18±5.54
HR(/min)	78.76±4.43	72.88±3.14	78.73±3.58	75.45±3.34
BHT (seconds)	47.66±8.89	53.23±8.95	43.73±6.79	53.72±8.22
SBC	63±16.47	78.74±11.52	56.60±12.36	69.62±15.04
SpO2 (%)	98±0.95	98.39±0.61	97.86±0.89	98.32±0.65
Total-chol (mg/dl)	173.07±28.41	142.06±17.51	187.44±26.44	152.18±29.71
HDL (mg/dl)	39.97±5.70	45.83±5.43	40.63±7.80	44.40±8.10
LDL (mg/dl)	132.95±21.11	122.86±14.65	134.18±22.62	111.59±20.36
TG (mg/dl)	158.90±34.21	131.25±19.37	157.23±31.62	140.48±39.59

Table-5: *Hemodynamic and lipid profiles of smokers according to the quantity of smoking (n=80)*

Variables	Group A (n=32)	Group B (n=16)	Group C (n=22)	Group D (n=10)	p value
SBP (mm Hg)	122.81±5.54	128.75±2.10	128±4.04	129.6±3.2	0.000
DBP(mm Hg	81.06±7.12	83.62±6.25	80.09±5.50	80±6.38	0.381
HR(/min)	77.96±4.57	79.12±2.61	78.68±4.38	80.8±2.22	0.283
SpO2(%)	98.09±0.97	97.62±0.92	97.95±0.70	97.9±1.04	0.444
T-Chol (mg/dl)	163.03±24.97	195.93±23.07	183.13±26.96	201.1±14.80	0.000
HDL (mg/dl)	42.40±5.98	42.62±8.76	37.18±4.52	36.6±5.37	0.005
LDL (mg/dl)	128.59±20.69	133.5±24.18	139±21.95	137.4±17.24	0.355
TG (mg/dl)	146.25±38.81	156.18±32.49	170.90±18.11	171±22.51	0.027

Note: Group-A (1-5 spy), Group-B (5-10 spy), Group-C (10-15 spy) and Group-D (15-20 spy).

significantly ($p<0.05$) increased while the mean value of HDL decreased. There was no significant ($p=0.355$) difference in LDL with increase in pack years of smoking.

Discussion

Cigarette smoking produces a chronic inflammatory state that contributes to the atherogenic disease processes and elevates the levels of biomarkers of inflammation [9,10,]. In our study, there was no significant difference in the mean anthropometric parameters like age, height, weight, body mass index the smokers and non-smokers. Cigarette smokers in our study usually smoked non-filter cigarettes which are cheap and easily available. In our study, the blood pressure and heart rate was higher in smokers than in non-smokers. The rise in blood pressure could be due to an increase in cardiac output and total peripheral vascular resistance [10]. Cigarette smoking has an acute hypertensive effect mediated by the stimulation of the sympathetic nervous system [11]. Saladini et al. investigated the effect of smoking on peripheral and central blood pressure in a group of young stage I hypertensive individuals [12]. Central systolic blood pressure and pulse pressure were higher in smokers than in non-smokers, thus implying a predominant effect of smoking on central blood pressure. Also, other studies reported significantly higher blood pressure and heart rates in smokers compared to non-smokers [12-14]. However, Saafan A Al-Safi reported that smoking had statistically non-significant effects on heart rate in females while heart rate values were significantly higher in male smokers than in non-smokers [14].

In our study, we have found that as the number of pack years of smoking increases, systolic blood pressure increases, while there were very minimal changes in diastolic blood pressure and even DBP is lesser for heavy smokers group like group C and D. We found increased heart rate and decreased SpO2 with increase in number of pack years. We have found that severity of smoking decreases the baseline SpO2 value in smokers despite of the group to which they belong. On the contrary, Chandra et al reported no significant difference in pulse oximetric (SpO2) values in subjects with a smoking history of <10 pack years compared to subjects with a smoking history of >10 pack years ($p>0.05$) [15].

It has been suggested that smoking, even of short duration and moderate consumption of cigarettes, is associated with adverse lipoprotein profiles [16]. In our study, mean values of T-chol, LDL and TGs were significantly ($p<0.05$) higher in smokers than non-smokers, while HDL was lower in smokers indicating derangement of lipid profiles in smokers. Duration and intensity of smoking are correlated with lipid profile. In our study we found that as the smoking pack years increases, mean value of T-chol, LDL and TG increases while that of HDL decreases. The difference was found to be statistically significant. Almost similar results were observed by other authors for cholesterol and triglycerides in smokers. Meenakshisundaram et al. [17] in their study on 274 asymptomatic male smokers showed that number of smoking pack years was directly proportional to the derangements in lipid profile variables. Previous studies by Neki [18] and Venkatesan et al [19] have demonstrated a rise in T-chol, TG, LDL and Apo-B, and a fall in HDL and Apo-A in smokers; and this association was dose dependent. Serum HDL concentration has an inverse relationship with smoking. In our study, serum HDL gradually decreased as the duration and intensity of smoking increased from group A to Group D, thus increasing atherogenic risk. Maximum prevalence of dyslipidemia was found in higher age smokers (age group-2). Though the mean values were within normal range for both smokers and non-smokers but they were close to upper reference range in smokers. It was also affected by number of cigarettes smoked. Amongst the two groups of smokers based on age, the smokers of higher age group had higher values of T-chol, TG and LDL, while lower values of HDL.

Thus, the present study shows that smoking has an adverse effect on lipid profile and vital hemodynamic parameters of young smokers. Smoking induces hypertension and reduces lung oxygenation capacity. Therefore, young individuals should be strongly advised to stop smoking and policy makers should take necessary measures to prohibit smoking.

Authors' contributions

BKJ: study conception, design, literature search, manuscript preparation, editing and review; AS: study conception, design, literature search, data collection, data analysis, manuscript preparation, editing and review; UMC and JN: study conception, design, manuscript preparation, editing and review.

Conflict of Interest: Nothing to declare.

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