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Original Article

Physiology Section

Effect of Intensity of Cigarette Smoking on Haematological and Lipid Parameters

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ABSTRACT

Background: Smoking is the single largest preventable cause of disease and premature death and major tobacco related diseases like cancer costs India more than Rs.308.33 billion per year. Cigarette smoking increases the total peripheral blood leukocyte count but its effect on other haematological parameters is largely unexplored.

Objective: To study the effect of smoking and its intensity on hematological and lipid parameters.

Materials and Methods: This cross sectional study included 40 cigarette smokers and 40 non-smokers in the age range of 25 and 40 years at SRM Medical College, Kattangulathur, Tamilnadu, India. In this study the volunteers were otherwise healthy and the smokers were enquired about their intensity of smoking using smoking index. Common hematological and lipid parameters were measured in all subjects. Comparison of smoking status and its intensity among the study subjects were analysed using t-test and ANOVA.

Results: There is a significant increase in levels of haemoglobin, hematocrit, total leukocyte count, total cholesterol, triglycerides, low density lipoprotein (LDL), very low density lipoprotein (VLDL) and reduced levels of high density lipoprotein (HDL) among the smokers. Heavy smokers showed significant dyslipidemia, increase in red blood cell count, total leukocyte count and neutrophil count.

Conclusion: Increase in hemoglobin, hematocrit, total leucocyte count and dyslipidemia were found significant among smokers and in heavy smokers there is dyslipidemia with increased RBC count, total leukocyte count with specific increase in neutrophils. As per the existing literature, these changes may lead to future fatal cardiac diseases among the smokers.

Keywords: Cholesterol, Hematocrit, Leucocyte count, Smokers

INTRODUCTION

In India, 337 million people above 10yrs of age consume tobacco. The World Health Organization predicts that tobacco deaths in India may exceed 1.5 million annually by 2020 [1]. Cigarette smoking is an important and independent risk factor for atherosclerosis, coronary artery disease, peripheral vascular disorders, etc and several studies provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile [2,3]. However, inspite of all that information, there is still much controversy about which component in the lipid profile are mainly altered in response to cigarette smoking, and whether those lipid profile components influence other parts directly or indirectly and vice versa. Differing results were obtained by various investigators, for example, Siekmeier et al., [4] concludes that HDL-C levels are same for smokers and non-smokers whereas Ito et al., [5] obtained low levels of HDL-C in cigarette smokers.

Although some studies [6,7] have reported that leukocyte count increases with the number of cigarettes smoked daily and decreases after cessation of smoking, data on smoking characteristics, such as duration of smoking, intensity of smoking, smoked pack-year and their association with leukocytes count is scanty. Therefore in this study we aimed to analyse the effect of tobacco smoking and its intensity over the haematological and lipid parameters.

Parameters	Smokers	Non-smokers	t-value	p-value
	Mean SD	Mean SD		
Hb (g/dL)	14.25±1.4	13.81±0.21	1.94	0.05
RBC (millions/ cu.mm)	4.81±0.04	4.94±0.49	1.67	0.09
PCV (%)	43.99±0.56	44.8±1.72	2.83	0.005
Platelet (lakhs/ cu.mm)	3.57±0.051	3.59±0.62	0.203	0.83
Total WBC (cells/cumm)	8278±1168.63	6075±1749	6.62	0.000
Monocyte (%)	3.87±0.13	3.9±0.66	0.25	0.802
Eosinophil (%)	5.91±0.79	5.36±2.26	1.44	0.15
Lymphocyte (%)	29.58±3.83	28.5±4.7	1.125	0.263
Neutrophil (%)	61.42±5.68	58.95±6.54	1.802	0.07

[Table/Fig-1]: Comparison of haematological parameters between smokers and non- smokers

MATERIALS AND METHODS

This cross-sectional study included 40 healthy male subjects of age 25 -40 years and 40 age matched male smokers attending the Master Health check up Scheme, SRM Medical College and Hospital, who had the history of smoking one or more cigarette per day, regularly for at least past one year. Institutional Ethical Committee permission and approval obtained. Informed consent was obtained from all the subjects.

Subjects with diabetes mellitus, ischemic heart disease or peripheral vascular disease, chronic renal disease, hypertension, any infectious or debilitating illness, taking antibiotics, steroids, thiazide diuretics, aspirin, non steroidal anti-inflammatory drugs, immunomodulatory drugs and drugs that influence lipid level were excluded from the study. The subjects who drink alcohol, passive smokers, ex smokers and those who underwent recent hospitalisation , surgery and radiotherapy were also excluded. Since smoking is extremely rare among women in this area due to cultural reasons, women were not included.

Parameters	Smokers	Non-smokers	t-value	p-value
	Mean SD	Mean SD		
Total Cholesterol(mg%)	197.76±19.37	155.38±24.09	8.67	<0.001
Triglycerides(mg%)	166.84±27.00	121.2±32.7	6.806	<0.001
HDL-Cholesterol(mg%)	35.91±4.51	46.9±6.71	8.59	<0.001
LDL-Cholesterol(mg%)	127.13±20.68	84.08±24.42	8.5	<0.001
VLDL-Cholesterol(mg%)	34.25±4.42	24.24±6.54	8.01	<0.001

[Table/Fig-2]: Comparison of lipid profile between smokers and non- smokers

Blood count was estimated using cell counter. Serum was separated by centrifugation at 3600 rpm for six minutes and estimation of Total cholesterol, Triglycerides(TG) and HDL-Cholesterol were done in fully automated autoanalyser. The levels of LDL cholesterol and VLDL cholesterol were calculated by using Friedewald's formula.

LDL (mg%) = Total Cholesterol – (HDL cholesterol + TG/5)

VLDL Cholesterol (mg%) = TG/5.

All the results of laboratory investigations were loaded in computerized SPSS 12.0 programmer and statistical significance were analysed by unpaired student's t-test and ANOVA. Results were expressed as mean ± SD (Standard Deviation). The p-value of <0.05 has been considered as significant.

Parameters	Mild Smokers (Pack years 10-14)	Moderate Smokers (Pack years 15-19)	Heavy Smokers (Pack years 20 & above)	f-value	p-value
	Mean SD	Mean SD	Mean SD		
HB(g/dL)	13.69±1.39	13.68±1.43	14.06±1.38	0.3452	0.71
RBC (millions/cu.mm)	4.77±0.56	4.85±0.52	4.81±0.49	11.54	0.0001
PCV (%)	43.7±1.72	44.63±1.72	43.62±1.59	1.367	0.267
Platelet (lakhs/cu.mm)	3.51±0.62	3.6±0.61	3.6±0.57	0.1	0.9
Total WBC (cells/cumm)	7248±1084.36	8038±959.84	9548±997.54	19.24	0.000001
Monocyte (%)	3.74±0.89	4±0.7	3.88±0.92	0.277	0.76
Eosinophil (%)	5±1.41	6.22±2.4	6.5±2.57	1.78	0.18
Lymphocyte (%)	34±4.7	27.66±12.7	27.08±4.35	3.06	0.06
Neutrophil (%)	55.81±6.54	61.27±10.28	67.18±6.26	5.52	0.007

[Table/Fig-3]: Comparison of haematological parameters between smokers

Parameters	Mild Smokers (Pack years 10-14)	Moderate Smokers (Pack years 15-19)	Severe Smokers (Pack years 20 & above)	f-value	p-value
	Mean SD	Mean SD	Mean SD		
Total Cholesterol (mg%)	176.38±18.54	202.78±20.56	214.13±26.98	10.05	0.0003
Triglycerides (mg%)	137.78±25.41	171.57±32.42	191.16±35.45	10.26	0.0003
HDL- Cholesterol (mg%)	40.63±3.06	35.46±3.5	31.64±2.56	32.2	0.00
LDL- Cholesterol (mg%)	84.08±24.42	104.01±18.12	133.5±21.76	10.72	0.0002
VLDL- Cholesterol (mg%)	24.24±6.54	34.35±6.53	38.62±7.34	6.54	0.003

[Table/Fig-4]: Comparison of lipid profile between smokers

A detailed history regarding current smoking status, number of cigarettes smoked per day, years of smoking, and years since quitting was obtained by using a pre-tested questionnaire. Non-smokers were the respondents who affirmed that they have not smoked yet. The pack-year is a unit for measuring the amount a person has smoked over a long period of time and was calculated by using the following formula.

Pack-years = (number of cigarettes smoked per day × number of years smoked)/20.

In our study smokers were classified into mild, moderate and heavy based on the number of pack years as 10-14, 15-19, and 20 and above, respectively [8]. Quantification of pack-years smoked is important in clinical care, where degree of tobacco exposure is closely correlated to risk of disease.

In order to ensure accurate and reproducible blood results, overnight 12-14 hrs fasting blood samples were collected from these subjects. The subjects underwent the following tests: Blood pressure examination to rule out hypertension, estimation of the serum lipid profile, evaluation of the fasting and post prandial blood glucose levels to rule out diabetes, red blood cell count, total leukocyte count, differential leukocyte count, platelet count, packed cell volume, haemoglobin, serum urea and creatinine levels.

RESULTS

[Table/Fig-1] compares the hematological parameters between smokers and non- smokers which shows a statistically significant increase in total leucocyte count, hematocrit and haemoglobin in smokers. The rest of the parameters were not statistically significant. [Table/Fig-2] compares the lipid profile between smokers and non- smokers and shows a statistically significant increase in total cholesterol, triglycerides, LDL, VLDL and significant decrease in HDL in smokers. [Table/Fig-3] compares haematological parameters between mild, moderate and heavy smokers which shows that the number of RBCs, total leucocytes and in particular the neutrophils were significantly increased in heavy smokers. The comparison of lipid profile among smokers is illustrated in [Table/Fig-4]. The abnormalities of lipid profile were even more significant when smoking intensity increases.

DISCUSSION

In the present study the mean total WBC count of smokers was significantly higher ($p<0.05$) than non-smokers [Table/Fig-1] and WBC count increases with intensity of smoking as shown in [Table/Fig-3]. This finding is consistent with Zalokar JB et al., [9].

Corre et al., [10] reported that the mean leukocyte count was 1,000/cu mm greater in cigarette smokers than in non-smokers. Increased leukocyte count among smokers might be due to nicotine-induced release of catecholamines. Inflammation on respiratory tree due to the irritant effect of cigarette smoke might be a contributory factor for the higher WBC count among smokers.

In the current study, it has been observed that of all the different leukocyte subsets, the neutrophil count was increased in smokers when compared to non-smokers. The eosinophil, basophil, lymphocyte and monocyte were within the normal range. Similar increase in neutrophil count was noticed in a study conducted by Schwartz J and Weiss ST [11] on smokers. Jensen et al., [12] found an increase in neutrophils, lymphocytes and basophils in smokers and a study by Kumar et al., [13] showed an increase in neutrophil and the eosinophil count.

In our study the hematocrit and Hb level were significantly higher in smokers and among the smokers the RBC count was significantly increased as the intensity of smoking increases. Whitehead et al., [14] in their study observed that hemoglobin concentration and hematocrit was significantly increased in those smoking more than 10 cigarettes per day.

The excessive carbon monoxide (CO) exposure may produce polycythemia in humans as well in animals. The half life of the CO in the body is 3-5hrs. In a person who smokes frequently and continuously, the carboxy Hb levels increases and produces a progressive hypoxemia and as the CO binds with Hb, functional anaemia is produced. This causes the impaired oxygenation of tissues and change in heamatological parameters [15].

Current study documented that there is a significant increase in levels of total cholesterol, triglyceride, LDL-C, VLDL-C and significant reduction in level of HDL-C in smokers when compared to non smokers. These results were analogous with the observations of Devaranavadgi BB et al., [16].

We have observed that there is a significant worsening of the altered lipid profile with increase in intensity and duration of smoking. The possible mechanisms for dyslipidemia among smokers were following.

(i) Nicotine stimulates the release of adrenaline from the adrenal cortex leading to increased serum concentration of free fatty acids (FFA) which further stimulates hepatic synthesis and secretion of cholesterol as well as hepatic secretion of very low density lipoprotein (VLDL) and increased TGL.

(ii) HDL concentration was inversely related to VLDL concentration in serum [17].

(iii) Smoking increases insulin resistance and causes hyperinsulinemia. LDL, VLDL and TGL are elevated in hyperinsulinemic conditions due to decreased activity of lipoprotein lipase and hepatic lipase has been activated, which converts VLDL to LDL [18]. Smoking impairs human serum paraoxygenase (PON1) activity and thereby compromises anti-oxidant defense mechanism [19].

CONCLUSION

In this study the smokers had dyslipidemia and a significant increase in hemoglobin, hematocrit and total leucocyte count. As the intensity of smoking increases as in heavy smokers, there is increased RBC count; total leukocyte count with specific increase in neutrophils and the altered lipid profile is also worsened. This dyslipidemic levels among the smokers may expose the vascular endothelium to potentially atherogenic lipoproteins which predisposes the greater risk of developing atherosclerotic plaques and coronary heart disease among the smokers. Thus earliest intervention of cessation of smoking may reverse these pathways of causing future serious health hazards.

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