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Serum Lipid Profile and Electrocardiographic changes in Young Smokers

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ABSTRACT

Smoking represents an important and rapidly growing global cause of cardiovascular mortality and morbidity.Cigarrette smoking is one of the major modifiable risk factors of cardiovascular disease, unless smokers are able to quit, approximately 40% of them will die prematurely.The need of the hour is timely intervention through smoking cessation.Our study was carried out in young smokers to demonstrate the effects of smoking on lipid profile and electrocardiographic changes. We aimed to study the effect of smoking on lipid profile and electrocardiographic changes in young smokers.The study design was a cross sectional study comprising 75 male smokers and 75 healthy controls.Smoking history and physical examination was done .Fasting sample was analysed for lipid profile and electrocardiograph of all subjects were recorded.The statistical anaylsis between mean values were evaluated by student't' test. Statistical significance was assessed by chi-square test, $p<0.001$ was considered to be significant.The mean pulse rate, the mean systolic blood pressure, abnormal lipid profile and prolonged Qt interval were significantly higher in smokers ($p<0.001$).We demonstrated significant lipid abnormalities and electrocardiographic changes in young smokers who form an important risk group and target population in whom smoking cessation counseling should be carried out to decrease long term cardiovascular risk.

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1. INTRODUCTION

Smoking represents an important and rapidly growing global cause of cardiovascular mortality and morbidity[1, 2].Worldwide, 1.2 billion people smoked in 2000, a number that is projected to increase to 1.6 billion by 2030.[3]

The earliest epidemiological study to demonstrate the association between smoking and ischemic heart disease(IHD) were the Framingham study and Sir Richard Doll's classic study of British doctors [4, 5].

Cigarrette smoking is one of the major modifiable risk factors of cardiovascular disease, in the form of atherosclerosis and coronary artery disease(CAD), unless smokers are able to quit, approximately 40% of them will die prematurely.It accelerates the atherosclerotic process by its proatherosclerotic and prothromboticactions.Acute events such as ventricular fibrillation, and sudden cardiac death are also increased by smoking, particularly in the presence of pre existing coronary heart disease(CHD) by augmenting the autonomic system [3].

The overall prevalence of current smoking was 15.5%(29.3% among males &0.6% among females) in Karnataka a survey conducted by ICMR with financial support by WHO in 2000[4]

Thus the need of the hour is timely intervention through smoking cessation.Our study was carried out in young smokers to demonstrate the effects of smoking on lipid profile and electrocardiographic changes, and the potential benefits of primordial prevention in such populations. To study the effects of smoking on lipid profile and electrocardiographic changes in young smokers.

2. RESEARCH METHOD

The study design was a cross sectional study comprising of 150 male subjects, 75 smokers and 75 age and sex matched healthy controls.

Inclusion criteria:

Smokers in the age group 25-40 years who smoked for atleast one year.Exclusion criteria:Patients with

1) Diabetes mellitus
2) Hypertension
3) Renal disease
4) Hepatic disease
5) Obesity
6) History of alcohol abuse
7) Drugs which can alter lipid profile and cause ECG abnormalities

A detailed history with emphasis on duration and number of cigarette/bidis smoked was taken. Smokers were divided in to two categories

1) Depending on the number of years of smoking:
   - 1-5 years
   - 5-10 years
   - >10 years
2) Depending on the number of cigarettes/bidis smoked
   - Light smokers-1-20 cigarette/bidi per day
   - Heavy smokers >20/day

A detailed general physical examination with vital signs and cardiovascular system examination was carried out.

Biochemical analysis

Blood sample was taken in fasting state for analysis of total cholesterol, LDL, HDL, triglycerides, V LDL, which was estimated by enzymatic colorimetric method. Electrocardiograph was taken for all subjects and analysed for any cardiac abnormality in terms of rate, axisdeviation, conductiondefect, arrhythmia, chamber hypertrophy or changes suggestive of ischemia.

3. RESULTS AND ANALYSIS

The study design was a cross sectional study, we studied 150 male subjects, of which75 were smokers and 75 age and sex matched healthy non smokers.The mean age was 32.08 among non smokers and 31.93 in smokers. There was no difference noticed among the groups with reference to diet and physical activity.

Distribution according to number of years of smoking and number of cigarette per dayOf the 75 smokers, 34 subjects smoked upto 20 cigarettes/day for 5-10 years(43.3%), 16 smoked for 1-5 years(21.3%) and 8 smoked for more than 10 years(10%).17 subjects smoked more than 20 cigarettes for more than 10 years(22.6%)

Statistical Analysis

The differences between mean values were evaluated by student ‘t’ test. The statistical significance was assessed by using chi-square test, $p<0.001$ was considered to be significant.

The mean pulse rate was higher in smokers 81.16 vs 74.81, the mean systolic blood pressure was significantly higher in smokers 124.19 vs 115.97 in non smokers($p<0.001$) Table 1.

The lipid profile among smokers were comparatively higher($p<0.001$) than in non smokers, with a lower HDL level among smokers.Also as the duration of smoking and number of cigarettes increased so did the lipid abnormalities (Figure 1).

Table 1.Corelation between Pulse rate and BP

	GROUP	N	MEAN	Std DEVIATION	t
SBP	NON SMOKERS	75	115.97	8.249	5.883 P<0.001vhs
	SMOKERS	75	124.19	8.982	
DBP	NON SMOKERS	75	73.2267	5.88992	0.18800 P=0.851 vhs
	SMOKERS	75	73.4133	6.25385	
PULSE RATE	NON SMOKERS	75	77.8133	6.81353	2.95700 p=0.004 hs
	SMOKERS	75	81.1600	7.04626	

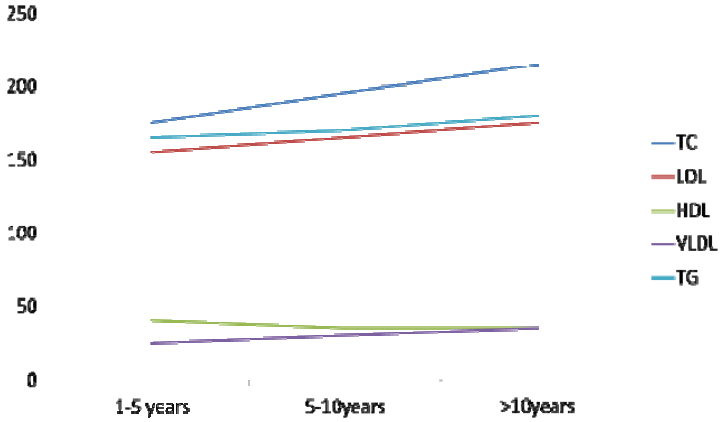

Figure 1. Depicts as the duration of smoking increases so does lipid abnormalities).

The Qt interval was prolonged among smokers 391.85±35 Vs 369.03±30.68(p<0.001), the PR interval was reduced among smokers but not statistically significant neither was there any statistical difference in P wave, QRScomplex, T and ST segment changes (Figure 2).

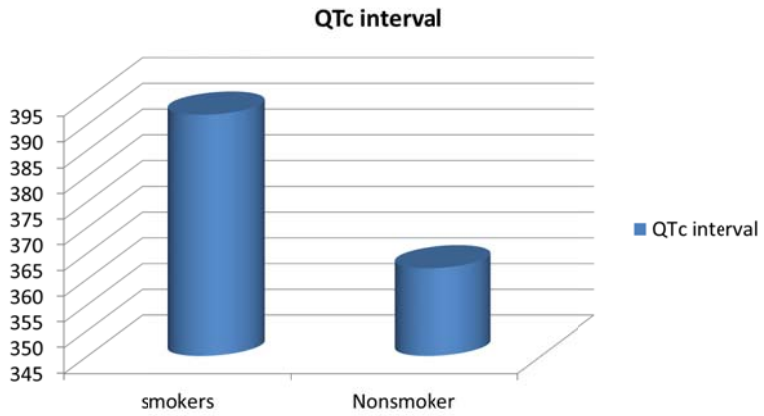

Figure 2. Qt interval was prolonged in smokers Vs nonsmokers

4. DISCUSSION

Ciagarette smoking is reported to be associated with an average 70% increase in the risk of death from coronary artery diseas e[6]. It has been observed that smoking affects lipid profile and increases the risk of cardiovascular death.Thus measurement of lipid profile in clinical laboratory has become increasingly important because of predictive association with coronary heart disease [7].

In a study conducted by Craig et al it was observed that young smokers are relatively at a higher risk of developing cardiovascular disease (CVD) compared to older adults[7], our study comprised of subjects were in the age group 25-30, they form the productive work force of the society and hence were studied to reflect the impact it might have with respect to cardiovascular disease and economic burden in future.

In our study smokers had a significantly higher pulse rate and systolic blood pressure compared to non smokers.Nicotine and one or more toxic agents in cigarette smoke are implicated in the pathogenesis. Although tachycardia during smoking contributes to increase in blood pressure, other mechanisms by which cigarette smoking may raise blood pressure include α1 adrenoreceptor mediated vasoconstriction, vasopressin release and possible direct effects on endothelial function[8].

Abnormal lipid levels and smoking are the two most important risk factors for having a heart attack[4]. High cholesterol levels are estimated to cause 56% of ischemic heart disease and 18% of strokes, amounting to 4.4 million deaths annually[9]. In our study we found that smokers had significantly higher level of lipid abnormalities and a lower HDL. Direct relationship of smoking towards CHD has been mentioned in MRFIT trial, it was described that increase in HDL 1mg/dl was associated with decrease in the risk of CHD[10].The incidence of developing CVD is directly related to the number of cigarettes smoked[11].In our study we were able to show a direct correlation between duration and number of cigarettes smoked with the magnitude of dyslipidemia. These results were in conformity with a study conducted by Ramachandran et al[12].

Smoking is also known to induce autonomic changes and may precipitate arrhythmias and sudden cardiac death. In our study the QTc was found to be prolonged among smokers, this may increase the threshold for arrhythmias, and QTc prolongation may double the risk for sudden cardiac death as studied by Okin PM et al[13].

Thus this study demonstrated significant lipid abnormalities and electrocardiographic changes in young smokers who form an important risk group and a target population in whom smoking cessation counseling should be carried out to decrease long term cardiovascular risk.

5. CONCLUSION

In a country like ours where tobacco represents an important cash crop and a source of employment, two things that are often in short supply, the smoking rates among men are staggeringly high. If the current smoking pattern continues by 2030 the global burden of disease attributable to tobacco will reach 10 million deaths annually, thus prevention through health education is imperative to bring down mortality and morbidity due to cardiovascular disease contributed to by smoking.

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