

Full length Research paper

Effect of tobacco smoking on the lipid profile of teenage male population in Lahore City

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A cross sectional comparative study was performed in Lahore city to evaluate the changes of the serum lipid profile in healthy adolescent male non smokers and smokers' subjects. A total of 100 subjects with age range 14 - 19 years were selected and grouped as non smokers (control) and smokers with 50 cases in each. Biochemical analysis of fasting serum lipid profile of each group was assessed using spectrophotometer. By applying independent sample t-test, significant differences were observed in TG, HDL-C and LDL-C and by applying chi-square (χ^2) test, significant association of TC, TG, HDL-C and LDL-C were found among smokers and controls. From the results of the present study, it may be concluded that, cigarette smoking during adolescent period induces alteration in serum lipid levels in the direction of increased risk for coronary artery disease.

Key words: Total cholesterol, triglycerides, high density lipoprotein cholesterol, low density lipoprotein cholesterol.

INTRODUCTION

Smoking is the major risk factor in the developing world but falling in developed nations. In the developing world, tobacco consumption is rising by 3.4% per year. Among young teenagers between the age of 13 to 15, about one in five smokes worldwide. Between 80,000 and 100,000 children worldwide start smoking every day - roughly half of whom live in Asia. Evidence shows that around 50% of those who start smoking in adolescent years go on to smoke in 15 to 20 years age (WHO, 2002). Smoking results in more deaths each year than AIDS, alcohol, cocaine, heroin, homicides, suicides, motor vehicle crashes and fires (Lynch and Bonnie, 1994).

Cigarette smoking (CS) is now acknowledged to be one of the leading causes of preventable morbidity and mortality and is one of the largest single preventable causes of ill health in the world (WHO, 1983). Nearly every cigarette smoker began as a teenager (DSM, 1994). The average smoker tries their first cigarette at age 12 (DiFranza, 1994) and may be a regular smoker by age 14 (Hogan, 2000). CS is a common problem and also a major public health problem associated with cardiovascular and respiratory morbidity and mortality. The prevalence of CS has reached its peak among high school students (Afrin et al., 2006). For this reason, a pediatrician, himself defined smoking as "a pediatric disease" (Kessler, 1997).

The reasons for smoking include the inadequate

understanding of the harmful effects of smoking; attractive tobacco advertising; the presence of so many other smokers; young people's rebelliousness and lack of mature judgement, inadequate legislation to control smoking, addiction, unhealthy ideas of consumption, the use of tobacco in social life and pleasure (Tsung, 1999). Nicotine and other toxic substances from tobacco smoke are absorbed through the lungs into the blood stream and are circulated throughout the body. These substances narrow or damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a non-smoker (Mitchell, 1999). In the World Health Organization's Western Pacific Region (WHO WPR), being born male is the single greatest risk marker for tobacco use (Martha and Barraclough, 2003). The male smoking prevalence (62.3%) and rate of increase are the highest in the world (Stanton, 2001).

On average, CS increases the risk of CHD by 70% compared with not smoking. The start of even modest CS during adolescence and early adulthood adversely alters the serum lipid and lipoprotein levels (Glueck et al., 1981; Freedman et al., 1986; Craig et al., 1990). Smokers had higher risk for coronary artery disease (CAD) compared to non smokers, partly attributed to some altered physiological factors including altered coagulation state damaged vascular wall and alteration in lipid and lipoprotein content (Craig et al., 1989). Atherosclerotic

Table 1. Average values of lipid profile in smokers and control group.

Parameters	Groups		p- value
	Smoker's mean \pm SD mg/dl	Control's mean \pm SD mg/dl	
TC	176.1 \pm 40.01	156.7 \pm 23.91	0.057
TG	111.5 \pm 32.54	97.74 \pm 35.68	0.047
HDLC	39.78 \pm 3.68	42.02 \pm 5.72	0.022
LDLC	104.20 \pm 31.55	93.08 \pm 22.25	0.044
VLDLC	21.42 \pm 6.16	21.22 \pm 6.94	0.879

changes found in middle age begin in childhood. The mechanisms may relate to abnormal levels of risk factors. Certain risk factors, such as serum lipid and lipoprotein levels and smoking, are thought to be related to the earliest stages of atherosclerotic CAD. In adults, high levels of low-density lipoprotein cholesterol (LDL-C) and low levels of high-density lipoprotein cholesterol (HDL-C) and its major subfractions are associated with myocardial infarction (MI) (Buring et al., 1992).

Keeping in view the paucity of data on tobacco-related issues in majority of developing countries (Davies, 2003) and the expected adverse effects of smoking on coronary function profile, this study was carried out to assess the impact of active tobacco smoking on lipids profile function tests to compare the effect of smokers and non-smokers of teenage male population in Lahore city.

MATERIALS AND METHODS

Study design

The study, conducted in Lahore, was an analytical observational cross-sectional study comprising of healthy regular habitual teenage male smokers and healthy non-smokers selected through convenient non probability sampling. As part of an ongoing genetic longitudinal study of developmental changes in cardiovascular risk factors during adolescence, families from nearby community were selected. Descriptions of current study were sent by campaign. None of the teenage boys were active cigar or pipe smokers at entrance to the study. A total of 137 teenage boys were invited to participate but 37 of them disqualified the criterion of this study. Out of a total of 100 participants of mean age 17, fifty (50) were grouped into randomized healthy tobacco smokers and fifty (50) were grouped into control that is, healthy non-smokers with no major chronic disease.

Ethical considerations

It was declared by a consent proforma that participation of all recruited boys was voluntary.

Biochemical analysis

Collection of blood samples for biochemical assays was done after fasting for at least 12 h. 5 ml blood from the antecubital vein from each subject was collected aseptically without prolonged venous stasis, in disposable sterile 10 ml syringes and was allowed to clot and stored. Samples were processed within 1 h for quantitative

lipoprotein cholesterol measurements using the vertical spin ultracentrifugation technique. Serum was obtained by centrifugation for 4 min at 3000 rpm and was then transferred into properly labeled sterile vials and stored at -20°C till the performance of lipid profile. Serum TC, serum TG and HDL-C tests was evaluated by standard enzymatic kits of Human Ltd whereas LDL-C and VLDL-C was calculated according to Friedewald et al. (1972).

Statistical analysis

All the data was statistically evaluated according to Steel and Torrie (1982) by using SPSS 16.0 (Statistical Package for Social Sciences). Quantitative variables were represented as mean \pm SD whereas frequencies and percentages were applied for qualitative variables. Independent sample t test to observe group mean difference and the Chi-square (χ^2) test to observe association of variables between groups were applied. P-value of less than 0.05 or 0.01 was considered statistically significant.

RESULTS

TC

The group means for Total Cholesterol (TC) are shown in Table 1. In groups, TC was higher in smokers than controls but non significant differences were observed between the smokers and the controls ($p = 0.007 > 0.05$, Table 1). To know the comparison of both the groups TC was categorized into desirable, borderline high and high according to the ranges (Table 2) so, 35 (70%), 11 (22%) and 4 (8%) out of total 50 smokers and 46 (92%), 4 (8%) and 0 out of total 50 controls had desirable (≤ 200 mg/dl), borderline high (201 - 239 mg/dl) and high (≥ 240 mg/dl) TC levels, respectively (Figure 1). Analysis using Chi-square (χ^2) test showed significant association in TC of each group ($\chi^2 = 8.760$, $p = 0.013$).

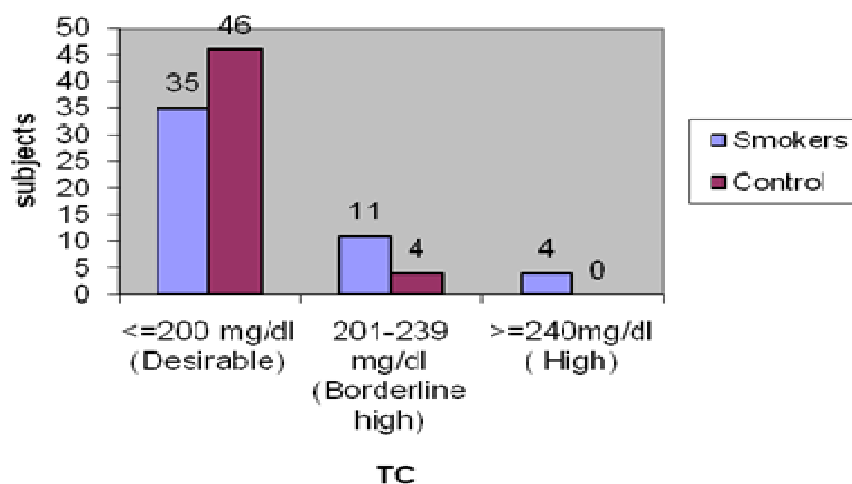
TG

The group means for Triglyceride (TG) are shown in Table 1. In groups, TG was higher in smokers than controls and significant differences were observed between the smokers and the controls ($p = 0.047 < 0.05$, Table 1). To know the comparison of both the groups TG was categorized into desirable, borderline high and high according to the ranges (Table 2) so, 41 (82%), 7 (14%)

Table 2. Comparison of parameters between smokers and control.

Parameters	Groups (*n = 100)	
	Smokers (*n = 50)	Controls (*n = 50)
≤200 mg/dl _ desirable	35	46
TC 201-239 mg/dl _ borderline high	11	4
≥240 mg/dl _ high	4	0
≤150 mg/dl _ desirable	41	48
TG 151-199 mg/dl _ borderline high	7	1
≥200 mg/dl _ high	2	1
≥45 mg/dl _ desirable	5	15
HDLC 35-44 mg/dl_ standard risk level	41	33
≤34 mg/dl _ risk indicator	4	2
≤130 mg/dl _ desirable	40	47
LDLC 131-160 mg/dl_ borderline high	5	3
≥161 mg/dl _ high	5	0
≤40 mg/dl _ desirable	48	48
VLDLC ≥41 mg/dl _ high	2	2

*n = No. of subjects.

**Figure 1.** Comparison of TC level of smokers and control.

and 2 (4%) out of total 50 smokers and 48 (96%), 1 (2%) and 1 (2%) out of total 50 controls had desirable (≤150 mg/dl), borderline high (151 - 199 mg/dl) and high (≥200 mg/dl) TG levels, respectively (Figure 2). Analysis using Chi-square (χ^2) test showed significant association in TG of each group ($\chi^2 = 5.384$, $p = 0.05$).

HDL-C

The group means for High Density Lipoprotein Cholesterol (HDL-C) are shown in Table 1. In groups,

HDL-C was lower in smokers than controls and significant differences were observed between the smokers and the controls ($p = 0.022 < 0.05$, Table 1). To know the comparison of both the groups HDL-C was categorized into desirable, standard risk level and high according to the ranges (Table 2) so, 5 (10%), 41 (82%) and 4 (8%) out of total 50 smokers and 15 (30%), 33 (66%) and 2 (4%) out of total 50 controls had desirable (≤45 mg/dl), standard risk level (35 - 44 mg/dl) and high (≥34 mg/dl) HDL-C levels, respectively (Figure 3). Analysis using Chi-square (χ^2) test showed significant association in HDL-C of each group ($\chi^2 = 6.532$, $p = 0.038$).

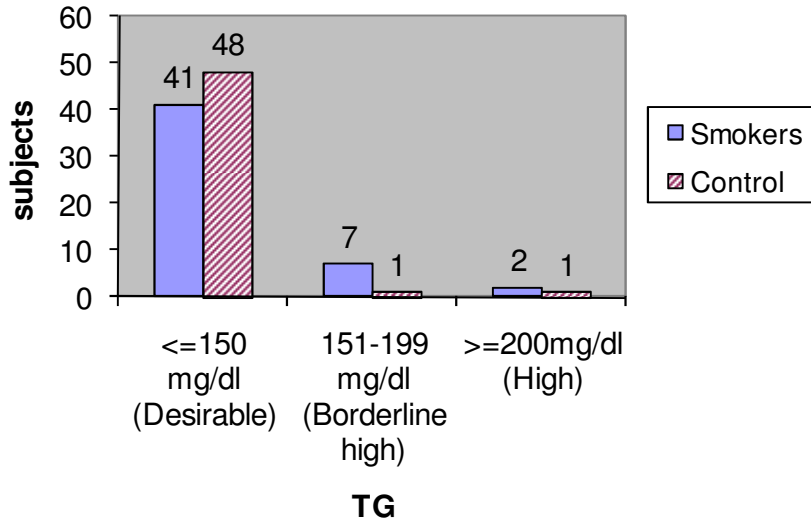


Figure 2. Comparison of TG level of smokers and control.

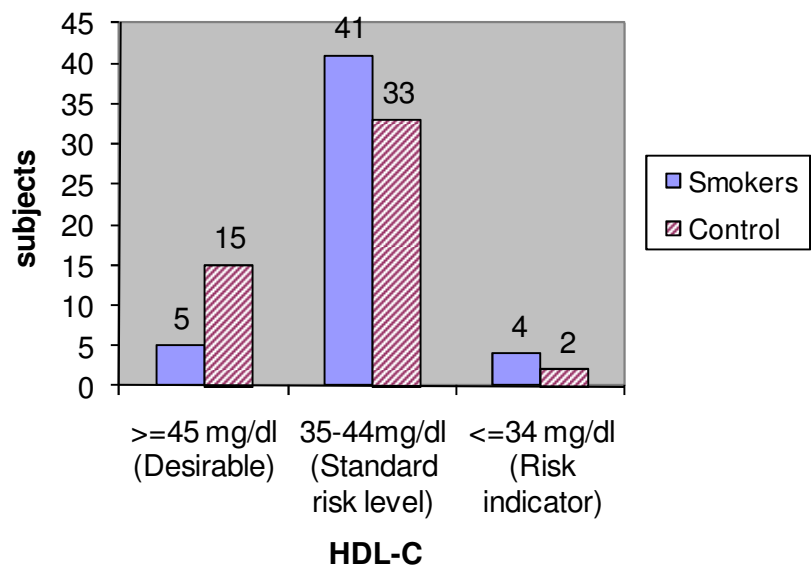


Figure 3. Comparison of HDL-C level of smokers and control.

LDL-C

The group means for low density lipoprotein cholesterol (LDL-C) are shown in Table 1. In groups, LDL-C was higher in smokers than controls and significant differences were observed between the smokers and the controls ($p = 0.044 < 0.05$, Table 1). To know the comparison of both groups, LDL -C was categorized into desirable, borderline high and high according to the ranges (Table 2) so that, 40 (80%), 5 (10%) and 5 (10%) out of total 50 smokers and 47 (94%), 3 (6%) and 0 out of total 50 controls had desirable (≤ 130 mg/dl), standard risk level (131 - 160 mg/dl) and high (≥ 161 mg/dl) LDL -C

levels, respectively (Figure 4). Analysis using Chi-square (χ^2) test showed significant association in LDL -C of each group ($\chi^2 = 6.063$, $p = 0.048$).

VLDL-C

The group means for very low density lipoprotein cholesterol (VLDL-C) are shown in Table 1. In groups, VLDL-C was higher in smokers than controls but non significant differences were observed between the smokers and the controls ($p = 0.879 > 0.05$, Table 1). To know the comparison of both the groups VLDL -C was

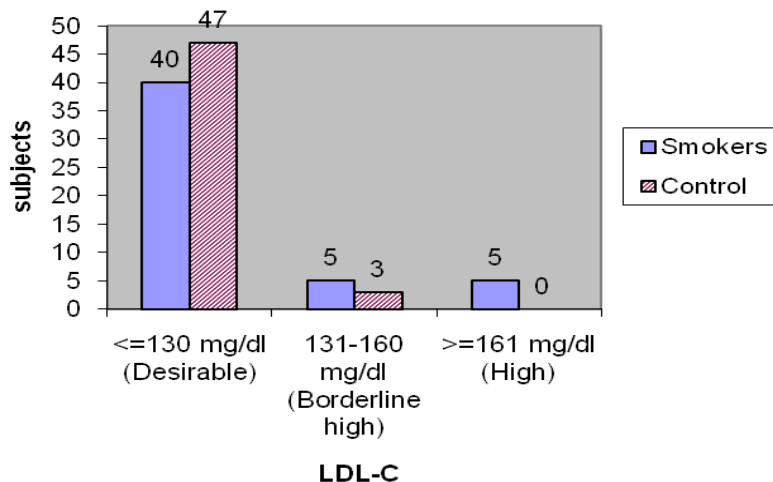


Figure 4. Comparison of LDL-C level of smokers and control.

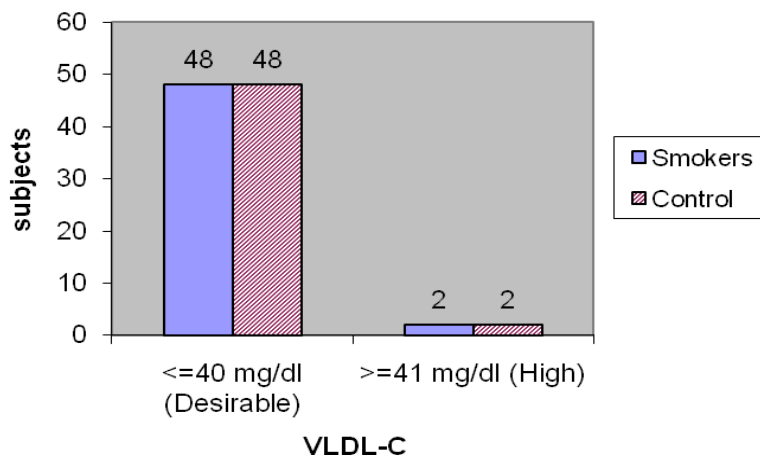


Figure 5. Comparison of VLDL-C level of smokers and control.

categorized into desirable and high according to the ranges (Table 2) so, 48 (96%) and 2 (4%) out of total 50 smokers and 48 (96%) and 2 (4%) out of total 50 controls had desirable (≤ 40 mg/dl) and high (≥ 41 mg/dl) VLDL -C levels, respectively (Figure 5). Analysis using Chi-square (χ^2) test showed no association in VLDL -C of each group ($\chi^2 = 0.000$, $p = 1$).

DISCUSSION

Cigarette smoking is one of the leading causes of preventable morbidity and mortality that usually starts in adolescence and continues into adult life (WHO, 1983; Lillian and Muula, 2004). In the developing world, tobacco consumption is rising especially among young teenagers (WHO, 2002). The tobacco smoking is also responsible

for premature development of CAD (Bhatt, 2003) and abnormal levels of risk factors such as serum lipid and lipoprotein levels, hypertension, and smoking are related to the earliest stages of atherosclerotic CAD (Buring et al., 1992).

No significant differences were observed between the groups for TC level (Table 1 and Figure 1) that was in agreement with the work of Craig et al. (1990) and Buring et al. (1992) in which they observed no statistical significance in level of TC among smokers and non-smokers because teenagers has the high immunity and are therefore likely to be a less sensitive indicator of the possible lipid-related excess coronary artery disease risk in smokers.

No significant differences were observed between the groups VLDL-C (Table 1 and Figure 5). Naslund et al. (1996) obtained similar results in a study that analyzed

only lower HDL-C levels and an unparallel effect on the VLDL-C levels of healthy smokers and non-smokers. During puberty and early adolescence, levels of HDL-C decrease in children that are more pronounced in boys than in girls (Berenson et al., 1981). The influence of sex hormones and their changes during puberty are obviously important, with HDL-C levels falling in boys in association with increases in testosterone levels (Kirkland et al., 1987). Passive cigarette smoking may further diminish HDL-C and its subfractions that may be associated with premature atherosclerotic changes. In a recent study, mean HDL-C levels were lower in dyslipidemic boys (Neufeld et al., 1997). Active smoking may worsen the risk profile for early atherosclerosis among such high-risk children.

The results of the present study indicate that adolescents with > 2 years smoking history and with approx. > 10 cig/day tobacco smoking had significant differences for TG, HDL-C and LDL-C were observed (Table 1 and Figures 2, 3, 4) and analysis using χ^2 tests also reported a significant association of serum lipids and lipoproteins and an insignificant association of VLDL-C between smokers and non smokers. These results are in concordance with the work of Imamura et al. (2000), who reported that smoking alters the lipids profile and these changes are related to the duration and amount of smoking.

It was also concluded from the present study that tobacco smoking is associated with dyslipidaemia (Increase LDL-C and decrease HDL-C levels), which is atherogenic in nature. As tobacco smoking interacts with other risk factors, the tobacco smokers get additional benefit if these factors are diagnosed and managed adequately. These risk profiles may be helpful in developing preventive cardiovascular strategies for children.

Conclusion

From the results of the present study, it may be concluded that, cigarette smoking during adolescent period induces dyslipidaemic state in the direction of increased risk for coronary artery disease. So it is strongly recommended to avoid smoking for the benefit of cardiac health.

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