

DR. MUMTAZ ALI SHAIKH

FCPS Medicine; D.Card
Associate Professor of Medicine
Liaquat University of Medical and Health Sciences
Jamshoro/Hyderabad Sind Pakistan

DR. DARGAHI SHAIKH

Senior Anaesthetist
SMBBU, Larkana

DR. DUR-E-YAKTA

Assistant Prof Ophthalmology
GMMMC SUKKER SMBBU, Larkana

ABSTRACT... Objective: This study is conducted to observe the serum lipoprotein alterations in chronic heavy smokers in LUMHS Sindh. **Study Design:** Analytical study **Material and Methods:** It was conducted on 60 non obese adult smokers, both sexes, who smoked more than twenty cigarettes or beeries a day regularly for more than five years. All the participants were current smokers. Sampling technique was simple random technique. **Setting:** The study was conducted in outdoor department of LUMHS Jamshoro/Hyderabad. **Period:** Jan 2010 to Jan 2011. Eighteen hours fasting blood sample was sent for lipid studies. Lipids studied were serum cholesterol, serum triglycerides, high density lipoproteins and low density lipoproteins. For control values ATP 3 guidelines were used. **Statistics:** Mean of statistical values was calculated with standard deviation and variance in standard deviation. Results obtained were analyzed by SPSS 11. **Results:** Age of the patients was 25-70 years. Mean age was 47.81 ± 12.96 years. Median age was 50 years. Male/ female ratio was 45/15. Patients were from both rural and urban areas. Mean duration of smoking was 25.5 ± 9.15 years. The study carried out on 60 chronic heavy smokers, showed deranged lipid levels as: Total cholesterol mean $237.57 \text{ mg/dl} \pm 37.89$; TG mean $203.76 \text{ mg/dl} \pm 47.08$; LDL-C mean $158.62 \text{ mg/dl} \pm 17.25$; HDL-C mean $29.67 \text{ mg/dl} \pm 3.12$. Results are shown in Table No I. **Conclusions:** Our study concluded at LUMHS showed that by continuous heavy smoking the serum lipid levels get deranged.

Key words: Smoke, Lipids, Sindh.

INTRODUCTION

Smoking is a recognized risk factor that leads to alteration of the serum lipoprotein pattern. The most probable way by which cigarette smoking increases risk of myocardial infarction is by altering the levels of lipoproteins. Tobacco smokers have arterial changes associated with preclinical atherosclerosis. Long-term exposure to smoke is estimated by measuring blood levels of cotinine, a byproduct of nicotine. Further research has shown that maximum intima/media thickness in both the carotid and aorta increased, as exposure to cigarette smoking increased, and endothelial function as measured by brachial artery flow-mediated dilation was decreased and in addition, (apoprotein) apoB and apoB/apoA-I ratio increased with increases in cotinine level¹. Cigarette smoking is quite common in our country. Smoking is an increasing public health problem especially in Pakistan. It is a risk factor for accelerated atherosclerotic vascular diseases².

One of studies done at Islamabad students it was found that 12.6% were regularly smoking cigarettes³. Another

study at Karachi showed that overall, 23% of students (31% male and 6% female) were classified as regular smokers⁴. Study at Sind showed, that high number (10%) of adult women were smokers⁵. Tobacco smoke contains many constituents; nicotine is one of the main constituents. Nicotine causes increase in triglyceride TG, cholesterol and very low density lipoproteins VLDL levels and decrease in High density lipoproteins HDL levels⁶. Considering the smoking as major risk factor for heart diseases which happens due to alteration in serum lipoproteins; this study is carried out to observe the alterations in plasma fasting total lipids, triglycerides, total cholesterol, HDL-C and low density lipoprotein LDL-C in chronic heavy smokers at LUMHS Sindh.

METHODOLOGY

This prospective, descriptive and analytical study was conducted on 60 non obese adult smokers, both sexes, who smoked more than twenty cigarettes or beeries a day regularly for more than five years. All the participants were current smokers. Sampling technique was random technique. The study was conducted in outdoor

department of LUMHS Jamshoro/Hyderabad from Jan 2010 to Jan 2011. Diabetics, hypertensive, primary dyslipidaemics, alcoholics, hepatic or renal disease, obesity, patients taking any medicines like B-blockers, diuretics or lipid lowering agents and patients with any other serious illness were excluded from the study. Eighteen hours fasting blood sample was sent for lipid studies. Lipids studied were Serum cholesterol, serum triglycerides, high density lipoproteins and low density lipoproteins. Obesity was excluded by calculating Body Mass Index. Normal values of fasting lipid profile were taken as;

Total cholesterol desirable	<200mg/dl.
Triglycerides	<150 mg/dl.
High density lipoprotein cholesterol	> 40 mg/dl
Low density lipoprotein cholesterol (ATP 3 guidelines).	<130 mg/dl,

STATISTICS

Mean of statistical values was calculated with standard deviation and variance in standard deviation. Results obtained were analyzed by SPSS 11.

RESULTS

Age of the patients was 25-70 years. Mean age was 47.81 ± 12.96 years. Median age was 50 years. Male/female ratio was 45/15. Patients were from both rural and urban areas. Mean duration of smoking was 25.5 ± 9.15 years. The study carried out on 60 chronic smokers, showed deranged lipid levels as: Total cholesterol mean $237.57 \text{ mg/dl} \pm 37.89$; TG mean $203.76 \text{ mg/dl} \pm 47.08$; LDL-C mean $158.62 \text{ mg/dl} \pm 17.25$; HDL-C mean $29.67 \text{ mg/dl} \pm 3.12$. Results are shown in Table-II.

DISCUSSION

This study revealed that all chronic heavy smokers in our set up had lipid levels deranged. Their mean levels were total cholesterol mean $237.57 \pm 37.89 \text{ mg/dl}$; TG mean $203.76 \pm 47.08 \text{ mg/dl}$; LDL-C mean $158.62 \pm 17.25 \text{ mg/dl}$; HDL-C mean $29.67 \pm 3.12 \text{ mg/dl}$ (Table I). These results were significant and comparable to results of other studies. One of the studies carried out has proved that in smokers the levels of total cholesterol, LDL-C were significantly elevated when compared with the controls⁷.

Table-I. Lipid profile in smokers: >20 cigarettes/day for >5 years (N= 60)

Lipid Profile	Mean mg/dl	± SD
Total cholesterol	237.57	37.89
TG	203.76	47.08
LDL-C	158.62	17.25
HDL-C	29.67	3.12

*TG : Triglycerides,
LDL-C : Low density lipoprotein cholesterol,
HDL-C : High density lipoprotein cholesterol.
SD: Standard Deviation*

Another study has shown that smokers have significantly higher ESR values, serum total cholesterol, LDL-C and mean serum TG levels. Mean serum HDL-C was significantly lower in smokers as compared to non smokers⁸. Another study has shown that smokers tend to have significantly high incidence of multi-vessel disease⁹. It has been observed that obesity and smoking are dangerous diseases which are becoming epidemic problems around the whole world including Pakistan and are quite evident risk factors for heart and vascular diseases. It is further associated with lipoprotein derangement and diabetes mellitus¹⁰. Other study has shown that smoking produces adverse effects on lipid profile, therefore increasing the cardiovascular disease risk¹¹. Tobacco smoke contains more than 4000 chemicals including 43 that are known to cause cancer¹². Another study has shown that smokers, total cholesterol, TG, and LDL-C are increased and HDL-C is markedly decreased showing greater risk of these persons to atherosclerosis and coronary heart disease¹³. According to the Framingham study ex-smokers who quit smoking for more than one year have serum cholesterol levels similar to non-smokers. This means that smokers who can stay quit for one year or more may normalize their lipid profiles. Smoking cessation improved HDL-C, total HDL-C, and large HDL-C particles, especially in women¹⁴. The findings indicate that the start of even modest cigarette smoking may have potentially long-term atherogenic effects. Prevention of smoking in early life should therefore be an important aspect of cardiovascular disease intervention¹⁵.

CONCLUSIONS

It has been concluded from this study that by continuous heavy smoking the serum lipid levels get deranged.

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Correspondence Address:

Dr. Mumtaz Ali Shaikh
205 A, Al-Raheem Heights Unit No.6
Latifabad-Hyderabad
ali_mumtazali@yahoo.com

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