JAMC Vol. 12 No. 3, 2000

LIPID PROFILE IN SMOKING

M. Zamir Ahmad Akbari, Muhammad Sarwar Bhatti, Muhammad Shakoor
Department of Biochemistry, Allama Iqbal Medical College Lahore.

Background: Smoking is one of the major risk factors in the genesis of coronary atherosclerosis and development of coronary heart disease. Smoking which is recognized as a major risk factor for the development of ischaemic heart disease may lead to alter the normal plasma lipoprotein pattern. ***Methods:*** Fifty adult smokers were selected along with thirty normal controls. Lipid profile was studied and estimations of cholesterol, total lipids, triglycerides, HDL, LDL, VLDL and chylomicrons were made. ***Results:*** All these parameters except HDL level were significantly increased in smokers while HDL level was significantly decreased, showing greater risk of these persons to atherosclerosis and coronary heart disease (CHD). Various ratios like LDL/HDL, VLDL/HDL, TG/HDL and TC/HDL were calculated to find out the risk of atherosclerosis and CHD so that early measures could be adopted to avoid complicated disease process.

INTRODUCTION

Smoking is one of the major risk factors in the genesis of coronary atherosclerosis and development of coronary heart disease [1, 2.]

Relationship of CHD and smoking was first developed by White *et al.* [3] and later Doll *et al.*[4]. Incidence of developing CHD is directly related to the number of cigarette smoked[5] . Sudden death is 2-4 times more in heavy smokers than in non smokers Stanler[6]. It has been suggested that cigarette smoking when it is consumed more than 10/day on regular constitute a major risk factor for CHD[7].

Some studies of smoking and serum lipids however have shown that plasma HDL cholesterol level tend to be lower in smokers than in non smokers[8]. Smoking which is recognized as a major risk factor for the development of ischaemic heart disease may lead to alter the normal plasma lipoprotein pattern. Earlier Friedman[5] has showed that increased cholesterol levels and CHD are observed in smokers. Cook *et al.*[9] have also suggested that apart from other risk factors hypercholesterolaemia and cigarette smoking are the major ones for CHD.

Tobacco smoke contains many constituents, nicotine is one of the main constituents. Nicotine causes increase in triglyceride, cholesterol and VLDL levels and decrease in HDL levels, Augestin [10], later on Cluette Brown[11] also studied that long term consumption of oral nicotine increased LDL cholesterol and decreased HDL cholesterol. It has been described that nicotine increases the circulatory pool of atherogenic LDL via accelerated transfer of lipids from HDL and impaired clearance of LDL from plasma compartment therefore it increases the deposition of LDL cholesterol in the arterial wall, Honjack [12].

MATERIALS AND METHODS

Fifty non obese smokers who smoked more than ten cigarettes per day regularly were selected and thirty non smokers non obese persons were included in the study as controls. Diabetics, hypertensives and those with history of angina were not included in the study. Fasting blood sample was taken, serum was separated and was analyzed for Total lipids, cholesterol, triglycerides, HDL, LDL, VLDL and chylomicrons. Only males were included in the study, as smoker females are almost non existent in our area.

RESULTS

The results of total lipids in controls and smokers are shown in Table-1 showing mean value, standard deviation (SD) and standard error of the mean.

Table-1: TOTAL LIPIDS *(mg/dl)*

	Controls	Smokers
Mean	724	969
S.D.	39.34	40.1
S.E.M	7.2	5.67

Control vs Smokers: P < .05

Serum triglyceride values are shown in Table-2.

Table-2: TRIGLYCERIDES *(mg/dl)*

	Controls	Smokers
Mean	151	204
S.D.	28.86	29.2
S.E.M	5.25	4.13

Control vs Smokers: P < .05

Mean serum cholesterol level are shown in Table-3

Table-3: CHOLESTEROL *(mg/dl)*

	Controls	Smokers
Mean	176	234
S.D.	16.10	33.35
S.E.M	2.94	4.72

Control vs Smokers: P < .05

HDL, LDL, VLDL and chylomicrons are useful and important components of lipid transport and utilization in the body. These parameters were also analyzed. The results of serum level of chylomicrons are shown in the following table.

Table-4: CHYLOMICRONS *(mg/dl)*

	Controls	Smokers
Mean	49	103
S.D.	14.30	26.64
S.E.M	2.61	3.77

Control vs Smokers: P < .05

HDL levels are shown in Table-5

Table-5: H.D.L *(mg/dl)*

	Controls	Smokers
Mean	39.4	32.04
S.D.	5	4.4
S.E.M	0.91	0.62

Control vs Smokers: P < .05

Serum LDL and VLDL values are shown in the tables 6 &7.

Table-6: L.D.L *(mg/dl)*

	Controls	Smokers
Mean	165	206
S.D.	32.6	31
S.E.M	5.95	4.38

Control vs Smokers: P < .05

Table-7: V.L.D.L *(mg/dl)*

	Controls	Smokers
Mean	97	113
S.D.	15.68	29
S.E.M	2.86	4.10

Control vs Smokers: P < .05

LDL/HDL ratios and VLDL/HDL ratios were also calculated as shown in Tab. 8 and 9.

Table-8: LDL/HDL Ratio

	Controls	Smokers
Mean	5.23	10.37
S.D.	0.97	1.48
S.E.M	0.19	0.21

Table-9: VLDL/HDL Ratio

	Controls	Smokers
Mean	2.48	3.53
S.D.	0.46	0.504
S.E.M	0.083	0.071

DISCUSSION

It has been reported that incidence of coronary heart disease is directly related to number of cigarettes smoked[10]. Sudden death is 2-4 times more often in heavy smokers than in non smokers[6] and smoking more than 10 cigarettes on regular basis constitutes a major risk factor for ischaemic heart disease. Those who continue to smoke have twice as many fatal and non fatal events as compared to those who do not smoke[13]. It has long been established that one of the constituents of tobacco i.e. nicotine has a considerable influence on increasing the lipid levels in blood[14]. The current study showed significantly higher levels of total lipids in smokers as compared to that of controls and the results are in accordance with study of Friedman[10]. Increased total lipids are considered to be an important contributory factor for development of atherosclerosis[15].

Increased cholesterol levels and CHD are observed in cigarette smokers[2]. In present study statistically significant increase ($P<0.05$) was observed in the serum cholesterol level in smokers as compared to that of control; these results are in agreement with those of Gorden[15] . Higher level of cholesterol are associated with CHD[16].

Similarly higher levels of triglycerides were found in smokers as compared to controls. Recent studies have suggested that triglyceride levels are the most important factor leading to CHD[17] although in fact triglyceride as a risk factor has been suggested by various research workers[18].

Chylomicron levels were slightly higher as compared to that of normal range but statistically significant levels were observed as compared to that of controls, as the serum chylomicron level is diet dependent and in this study 14 hours fasting samples were collected so much emphasis cannot be laid on this parameter due to the same reason considerable less work has been done on this parameter.

HDL level showed statistically significant decrease in smokers as compared to controls. These results are in conformity with those of Scrot (1989) who observed low levels of HDL in smokers as the result of threat of development of atherosclerosis and CHD is increased. Direct relationship of smoking towards CHD has been mentioned by MRFIT[20], who described that increase in HDL level by 1 mg/dl was associated with decrease in the risk of CHD by 3%.

LDL & VLDL levels were also significantly increased in smokers and are in agreement with results of Kesaneimi and Grundy[21]. LDL/HDL and VLDL/HDL ratios were significantly higher in smokers as compared to that of controls as evidenced by Martin[22] who suggested that with increase in these ratios risk of developing CHD also increases proportionately. In addition to these TG/HDL & TC/HDL ratios are useful as quick summary of disease risk in smokers. These can be easily counted and risk of disease evaluated. TC/HDL ratio is of

very high significance as values higher than accepted dangerous limit of >4.5 require intervention and indicate very high risk of CHD[23,24]. TC/HDL ratio estimates the net effect of two way traffic of cholesterol in and out of tissues[25]. This ratio has been suggested to be the most important predictor of premature development of CHD[26]. Persons considered at higher risk of CHD can then be immediately identified and properly advised.

REFERENCES

1. Thelle, D.S. & Sharper, A.G.. Blood lipids in middle-aged Britishmen. Br. Heart.J. 1983: 49:205-13.
2. White, P.D.. Coronary disease & coronary thrombosis in youth. J.Med. Soc. J. 1935:32: 596-605
3. Framingham Study: Paul Sorlie & Tavia Gorden Body build & mortality. JAMA, 1980: May 9, Vol. 243.
4. Doll, R, & Hill, A.B.. Mortality in relation to smoking. Br. Med. J. 1964: ii: 399-440.
5. Friedman, G.D. & Dale, L. G.. Mortality in middle-aged smokers & non-smolers. N. Engl. J. Med. 1979: 300-213.
6. Stamler, J.Wentworth D. Is the relationship between serum cholesterol & risk of premature death from coronary heart disease continuous & graded. JAMA 1986: 256:2823-28.
7. Garrison, R.J. Kannel, W.B.. Cigarette smoking & HDL-cholesterol. Atherosclerosis: 1979: 30,17.
8. Bain, C. & Jesse M.J.. Cigarette consumption and deaths from coronary artery disease. Lancet, 1978:1:1087-8.
9. Cook, D.G. & Sharper, A. G. Giving up smoking and the risk of heart attacks. Aeport from British Regional Study. Lancet: 1986:12.
10. Cluette-Brown, J. & Hugan. S. Oral nicotine induces an atherogenic lipoprotein profile. Proc. Soc. Exp. Bio. Med 1986:182:409-13.
11. Hojnack, J.Mulligan, J.& Cluette-Brown,J. Oral nicotine impairs clearance of plasma Low Density Lipoproteins. Proc. Soc. Exp.Biol.Med. 1986:182:414-18.
12. Ramsdale, D.R.& Bened D. Smoking & coronary artery disease assessed by routine coronary arteriography. Brit. Med. J. 1985: 290; 197-200.
13. Augustin, J. & Beedgen, B. The influence of smoking on plasma lipoproteins. Intern Med. 1982: 9: 104-8.
14. Gorden, T. Castelli, W.P. Diabetes, blood lipids and the role of obesity in coronary heart disease risk for women. The Framingham Study. Ann. Intern. Med. 1977: 83:393-397.
15. Dawber, T. R. Kannel, W.B. & Gorden, T. Some factors associated with the development of coronary heart disease. Am. J.Publ. Health: 1959: 49: 1349-56.
16. Goldbourt U, Holtlman E.. Total & high density lipoprotein cholesterol in the serum & risk of mortality. BMJ; 1985: Vol. 290:1239-12-42.
17. Varley, H. Gowenlock, H.A. and Bell,M. (Editors) 1984) Practical clinical biochemistry. Vol.1, 5th. William heinemann Medical Books Ltd. London. P677.
18. Carlson, L.A., Bottiger L.E.. Ischaemic heart disease in relation to fasting values of plasma triglycerides and cholesterol. Stockholm Prospective Study. Lancet. 1972:865.
20 Multiple Risk Faltor Intervention Trial Research group (MRFIT):. JAMA; 1982: 248: 1465-77.
21. Martin M.J.Hulley S.B. Serum Cholesterol, B.P & mortality. Lancet; 1986.:2:933-36.
22. Kesaniemi, Y.A. and Grundy. Significance of low density lipoprotein production in the regulation of plasma cholesterol level in men. J.clin.Invest. 1982: Vol. 70:13-22.
23. Kannel W.B. Metabolic risk factor for coronary heart disease in women. Am. Heart. J. 1987:114:413-19.
24. Castelli WP. Cardiovascular disease in women. AM.J. Obstet. Gynaecol. 1988: 1046-1060.
25. Guyton. C. Female Physiology before pregnancey and female hormones, in text book of Medical Physiology 8th Ed. W.B. Saunders company Philadelphia 1991 pp 899.
26. Razay G, Heaton KW, Boltan CH, coronary heart disease risk factors in relation to menpower. Quately J.med. 1992; 889-96