LIPID PROFILE IN OBESITY

Muhammad Sarwar Bhatti, M. Zamir Ahmad Akbri and Muhammad Shakoor

Department of Biochemistry Allama Iqbal Medical College, Lahore

Background: Obesity is associated with social and medical risks that especially make it a problem. The importance of obesity in the prediction of cardiovascular disease has been the subject of long standing debate. Direct correlation between plasma triglycerides and body weight have been noticed. We report the results of a study in our center. ***Methods:*** Fifty adult subjects who were obese (body mass index > 25 Kg/m) and nonsmokers were selected along with thirty non obese nonsmokers as controls. Lipid profile was studied including total lipids, total cholesterol. HDL, LDL, VLDL and chylomicrons. Various ratios like LDL/HDL, VLDL/HDL, TG/HDL and TC/HDL ratios were calculated to find the risk of atherosclerosis and coronary heart disease. ***Results:*** All the parameters except serum HDL. level showed significant increase in obese persons while HDL level was significantly decreased.

INTRODUCTION

Obesity which increases the risk of cardiovascular disease, hypertension and diabetes mellitus[1]"' is quite simply the result of caloric intake in excess of body needs. It usually begins in childhood or adolescence and the longer it is allowed to persist the less likely that it can be controlled. It is important to understand that obesity is not necessarily the result of over consumption of fat but it can result from excess calorie intake from any source whether carbohydrates or proteins. Obesity is associated with social and medical risks that especially make it a problem'[1]. Garrow[1] also devised a chart for classifying obesity based on body mass index (BMI) a concept that was first suggested over a hundred years ago by Quetlet[4]. Garrow'[1] subdivided obesity by using Quetlet's index into mild (Grade-1) BMI 25-30 Kg/m, moderate (Grade II) BMI 30-40 Kg/m and severe (Grade III) BMI > 40 Kg/m. A person is considered non obese when BMI is between 20-25 Kg/m[5].

The importance of obesity in the prediction of cardiovascular disease has been the subject of long standing debate. According to Framingham study[25] weight gain leads to rise in atherogenic trait and weight loss to decline. Trussmel[5] described that obesity increased the risk of coronary artery disease 35% more than in non-obese subjects. The mechanism by which obesity leads to premature death from cardiovascular disease probably involve risk factors such as hypertension, lipid disturbances and impaired glucose tolerance[6]. Obesity is associated with elevated blood lipids and lipoproteins[7]. A negative association between HDL and incidence of obesity have been reported in several studies[8]. Direct correlation between plasma triglycerides and body weight have been noticed[9] as high percentage of patients with myocardial infarction exhibited hypertriglyceridaemia[l0].

MATERIALS AND METHODS

Fifty adult volunteer subjects between 30-50 years of age were selected who were obese (BMI > 25 Kg/m) and were nonsmokers and were not having other ailment. Thirty healthy non obese, nonsmokers and sex matched persons were taken as controls. Diabetes, hypertension and those with history of angina were ruled out. Five ml of venous blood was collected after 12-14 hours of night fasting and was left to clot, then it was centrifuged to separate the serum which was then analyzed for total lipids, total cholesterol, HDL, LDL, VLDL and chylomicrons using reagent kits supplied by Boehringer Manheim.

RESULTS

The results of total lipids in controls and obese are shown in Table-1, showing mean value, standard deviation and standard error of the mean. Mean serum levels in controls were 724 ± 39.34 and in obese mean levels were 995124.33 mg/dl showing statistically significant increase (p<05).

Table-1: Total Lipids

	Controls	Obese
Mean	724	995
S.D.	39.34	24.33 P
S.E.M	7.2	3.44

Control* vs- *obese: P <.05

Serum triglyceride values are shown in Table 2, mean serum triglyceride levels in controls were 151± 28.86 and in obese 238 ± 34.80 mg/dl.

Table – 2: Serum Triglycerides

	Controls	Obese
Mean	151	238
S.D.	28.86	34.8
S.E.M	5.27	4.92

Controls vs Obese: P<.05

Mean serum cholesterol level in controls were 176 + 16.10 and in obese the mean values were 251 + 26.20 mg/dl.

Table -3: Total Cholesterol

	Controls	Obese
Mean	176	251
S.D.	16 10	26 20
S.E.M	2 94	3 71

Controls vs Obese: P<.05

HDL, LDL, VLDL and chylomicrons are useful and important components of lipid transport and utilization in the body so these parameters were also analyzed in controls and obese persons. The results of serum levels of chylomicrons in controls were 49 + 14.30 and in obese 107 -H 18.77 mg/dl and have been shown in the following table.

Tahle-4: Chylomicrons

	Controls	Obese
Mean	49	107
S.D.	14 30	18 77
S.E.M	2 61	2 65

Control vs obese: P <.05

HDL levels are shown in tab V and the mean values in controls were 39.4 + 5.0 and in obese 29.5 + 4.50 mg/dl.

Table-5: HDL

	Controls	Obese
Mean	39 4	29.5
S.D.	5 0	4.5
S.E.M	0.91	0.64

Control vs obese: P <.05

Table-6: VLDL

	Controls	Obese
Mean	97	106
S.D.	15.68	30
S.E.M	2.86	4.24

Control vs obese: P <.05

LDL/HDL ratio. VLDLTIDL ratio, TG/HDL ratio and TC/HDL ratios were calculated and are shown in Tab.8 and 9.

Table-8: LDL/HDL Ratio and VLDL/HDL

	Controls	Obese
LDL/HDL Ratio	5.23 ± 097	7.18 ± 1.03
VLDL/HDL Ratio	2.48±0.46	3.70 ± 0.53

Table-9: TG/HDL Ratio and TC/HDL Ratio

	Controls	Obese
TG/HDL Ratio	3.96 ± 0.74	8.39 ± 1.2
TC/HDL Ratio	2.48 ± 0.46	8.51±1.22

DISCUSSION

Obesity is associated with elevated blood pressure, blood lipids and blood glucose levels, this also increases the risk of coronary heart disease in obese persons[11].

Amongst the lipid components considerable stress has been made on estimation of total cholesterol, HDL and triglycerides however other components like total lipids, LDL and VLDL along with chylomicrons have also been a point of interest for many workers. The effect of obesity on various lipid components was observed in this study. The results of total lipids clearly show much higher levels in obese persons as compared to controls. Gorden[12], have shown similar results in obese persons. Increased lipid level may contribute its role towards atherosclerosis indirectly having relationship with coronary heart disease. There might be various reasons for finding higher levels of total lipids and lipoproteins in obese persons. The obese subjects have the habit of overeating and less consumption of calories and also have sluggish pattern of life.

Increased levels of cholesterol have also been reported in obese persons and the present study also showed significantly increased levels of cholesterol in obese person. Framingham study[25] confirmed that weight gain is associated with higher cholesterol levels and other factors like blood pressure, glucose and uric acid. Similar results have been reported by Gorden[12].

Higher levels of triglycerides are found in obese persons as shown in Table-2, this increase is statistically significant and correlate with the findings of Varley[13], who suggested that triglyceride levels are the most important factor leading to CHD as almost 50% of patients with asymptomatic atherosclerosis were hypertriglyceridaemia. Hojnack[14] also have found strong positive correlation of triglycerides with obesity.

Chylomicron level in the blood is totally diet dependent as higher levels are seen within hours of dietary intake but after longer period chylomicron level become well below the normal limits. In the present study the samples were taken after 14 hours of fasting and were only at the upper level of normal limits but still higher than controls. Due to this not much emphasis is given to chylomicron levels.

HDL estimation showed significantly decreased levels in obese as compared to the controls. This is considered as the single major risk factor for predicting the risk of atherosclerosis and CHD. These results are in conformity with those of Rabkin[15] & Sorenson[16]. After weight reduction in obese person significant increase in HDL level is seen[17].

LDL & VLDL levels were also increased statistically in obese persons as compared to the controls, these results are amply supported by Kesaneimi & Grundy[18] and Clautte Brown[19]. The National Cholesterol

Education Programme[20] identified LDL as the major atherogenic lipoprotein. Stein[21] documented the benefit of lowering elevated plasma LDL Cholesterol and preventing coronary artery disease.

LDL/HDL and VLDL/'HDL ratios as shown inverse relationship with risk of CHD as Castelli[22] suggested that LDL/HDL ratio exceeding 4.5 indicates higher risk of developing CHD. TC/HDL ration estimates the net effect of two-way traffic of cholesterol in and out of the tissues[23]. This ratio has been suggested to be the most important predictor of premature development of CHD[24]. Framingham study[25] has mentioned TC/HDL ratio to be one of the most powerful predictor of CHD and further suggested that it should be included in any coronary risk screening profile. TC/HDL ratio of more than 4.5 generally required intervention[26,27].

REFERENCES

1. Kannel, W.B. Metabolic risk factor for coronan heart disease in women. AM. Heart. J. 1987 114: 4 i j-19
2. Garrison. R J. Castelli W P. Obesity and Lipoprotein Cholesterol in the Farm Ingham off *spring* study Metabolism 1986:29; 1053.
3. Garrow J.S.S (1981). Treat obesity seriously. Edinburgh: Churchill Livingstone. Quetlel LA. Physique, Social. Brussels Marquardt 1969
4. Truswell A S. (1985) Obesity, diagnosis and risk in British.Med.J.1985: 291:655-57.
5. Gam, S.M & Hawthorne V.M. Fatness and mortality in the west of Scotland. Am. J Clin Nutr 1983:38 313-19.
6. Lorett P & Askevold E.M. The Oslo Study, Cardiovascular disease in middle aged and young Oslo men Acta Med. Scand. Suppl 1977: 588
7. Garden. T. Castelli. W. P Diabetes, blood lipids and the role of obesity in coronary heart disease r.sk for women The Framingham Study. Ann Intern. Med.
8. 1977:83' 393-397.
9. Sonja L.C & William E The effect of age, body weight and family relationship on plasma lipoproteins Circulation. 1982:65:1290-97.
10. Morrison J.A & Laskarzevyiski. Intra-familial Associations of Lipids and Lipoproteins in kindred with hyper triglycerdemic probands Circulation 1982 66:67
11. Robertson T. Kato H. & Garden T Epidemiological studies of coronary hear! disease & strokes in Japanese men living in Hawaii and California. Am..1. Cardiol. 1977: 39:239-43.
12. Garden, T. Castelli, W.P. Kannel W B. High density lipoproteins as a protective factor against coronary heart disease AM. J. Med 1977: 62: 707.
13. Varley H. Govenlock H. A & Bell M (1984) Practical Clinical Biochemistry Vol. l. William Heinemann Medical Books Ltd. London, p 677
14. Hojnack. J., Mulligan, J. & Cluette-Brown, J. Oral nicotine impairs clearance of plasma Low Density Lipoproteins. Proc Soc. Exp. Biol. Med. 1986: 182:444-18.
15. Rabkin S W & Mathewson F A. Relationship of body weight to development of ischaemic heart disease in a cohort of young North American Men. Manitoba Study. Amer.LCardiol.1977: 39:452.
16. Sorenson T. & Sonne H.S.M. Mortality in extremely young overweight men. J.Chronic.Dis.1977: 30: 359-67.
17. Friedman. C.L & Falko, J.M.: Serum Lipoprotein responses during active & Stable weight reduction in reproductive obese females. 1982
18. Kesaneimi. 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.
19. Cluette-Brown, J. & Hogan. S. Oral nicotine induces an atherogenic lipoprotein profile. Proc. Soc Exp. Bio Med 1986: 182:409-13.
20. National Cholesterol Education Programme Expert Panel. Detection, evaluation and treatment of high blood cholesterol in adults. Arch. Intern. Med. 1988: 148:36-39.
21. Stein E.A. Management of hypercholesterolemia. Approach to diet and drug therapy. Am. J. Med. 1989:87:20-27.
22. Castelli, W.P. Rober, D & McNamara, A.B.: Summary estimates of cholesterol used to predict coronary heart disease. Circulation 1983. 687m 730-34.
23. Guyton. C. Female Physiology before pregnancy 8th Eds. W.B. Saunder's Company Philadelphia 1991. pp 899.
24. Razay G., Heaton K.W and Bolton C H. Coronary Heart Disease risk factors in relation to menopause. Quarterly J. Med. 1992: 889-96.
25. Framingham Study. William P. Castelli The triglyceride issue: A view from Framingham. Am. Heart J August, 1986: 432-437.
26. Kannel, W.B. & Castelli W.P. Cholesterol in the prediction of atherosclerotic disease. Framingham Study. Ann. Intern. Med. 1979:90: 85.
27. Castelli WP. Cardiovascular disease in women. AN. J Obstet. Gynaecol. 1988: 1046-1060.