Original Article

Study of Lipid Profile in Obesity

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Abstract

Background: *Now a day's obesity is a burden Worldwide. The Prevalence of obesity is increasing in both the developed and developing Countries.* ***Objectives:*** *To investigate lipid profile in obesity.* ***Methods:*** *This study was carried out in the Department of Physiology, Mymensing Medical College, Mymensing, Bangladesh during the period of January 2009 to December 2009. Lipid profile was measured in 150 cases of over weight and obese.* ***Results:*** *Serum total cholesterol, triacylglecerol, LDLcholesterol were high in obese group but HDL cholesterol was high in non-obese group.* ***Conclusion:*** *Obesity affects blood lipid & lipoprotein metabolism.*

Key words: *Lipid profile, HDL Cholesterol, LDL Cholesterol,Triacylglecerol, Obesity.*

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Introduction

The prevalence of obesity is increasing in both the developed and developing countries, with 10-50% of the Indian population are being reported as obese.[1] Obese children and adults, particularly those with a central or abdominal distribution of fat, have elevated concentrations of serum triacylglycerol-a surrogate measure of VLDL cholesterol-andlow concentrations of HDL cholesterol.[2] The major lipoprotein classes, however, are composed of subclasses that vary in size, composition, and atherogenicity[3] and it is possible that these subclasses are differentially associated with obesity. For example, several obesity indexes have been found to be more strongly associated with concentrations of the larger VLDL subclasses than with the smaller VLDL subclasses[4] concentrations of small HDL subclasses,[5] and a smaller mean size of LDL particles [6] The results of some studies indicate that these obesity-related differences in lipoprotein subclasses may be important in atherosclerosis.[7] Overweight and obesity have significant health and economic consequences.[2] In adults, they are associated with an increased risk of developing various non-communicable diseases (NCDs), including hypertension, coronary heart disease, diabetes, stroke and some forms of cance.[2] It is evident that there has already been a profound shift in the major causes of death and diseases in developing countries.[4] In the poorest countries, even though under nutrition and infectious diseases dominate the current disease burden, the prevalence of major risk factors for chronic diseases are increasing. For example in Bangladesh, morbidity patterns are predominantly characterized by chronic energy deficiency (CED) and infectious diseases, but diet-related NCDs also constitute a major part of the adult morbidity both in urban and rural areas.[6-8] This pattern is similar to many other mid-and low-income countries.[3-9] Over the last two decades, chronic diseases such as cardiovascular disease (CVD), metabolic disorders and malignant neoplasm have accounted for a significant fraction of death tolls worldwide[10] It is well known that cigarette smoking, obesity, lipid disorder,

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elevated blood pressure and diabetes mellitus are primary risk factors for CVD.[11-17] In addition, it has also been recognized that many CVD risk factors are associated with each other,[18-20] significantly influence serum levels of total cholesterol (TC), LDL-cholesterol (LDL-C) and HDL-cholesterol (HDL-C).[21-26] In conjunction with reductions in saturated fat and cholesterol, the inclusion of the above therapeutic diet options (including weight loss) is expected to decrease LDL-C by 20% to 30%.5 In addition to the therapeutic diet options of the therapeutic lifestyle change (TLC) diet, there is evidence that other dietary modifications, such as including soyprotein[24] and nuts, can lower LDL-C significantly. Low HDL-C is an independent risk factor for coronary artery disease.[5] There are 2 ways by which diet may affect HDL: those caused by changes in the fatty acid composition of the diet and those that affect plasma TG levels. Because dietary fatty acids have major effects on LDL-C and HDL-C, it is necessary to evaluate these effects together to assess the potential impact of HDL change on coronary disease risk. Thus, the ratio of LDL-C or total cholesterol to HDL-C is one bench mark for estimating the risk of CHD. Increased weight is a determinant of low HDL-C levels. Weight loss has favorable effects on HDL-C[15,16] during weight loss, before weight maintenance is attained, HDL-C may decrease.[15] Physical activity beneficially influences most of the atherosclerotic risk factors. The impact of regular exercise on plasma lipids and lipoproteins has been clearly defined with regard to the interactions among lipids, lipoproteins, apolipoproteins (apo), lipoprotein enzymes, and the influence of various factors such as aging, body fat distribution, dietary composition, and cigarette smoking status.[17,18] The present study was an attempt to assess the suitability of these new guidelines for classifying Indians into overweight and obese based on their atherogenic indices of lipid metabolism.

Material and Methods

This was a cross sectional case control type of study, carried out in the department of Physiology, Mymensingh Medical College, Mymensingh, Bangladesh during the period of January 2009 to December 2009. A total number of 100 subjects male and female of obese aged 30-60 years were included in this study. Fifty individual of same age group were considered as control. Informed consent was obtained from each volunteer. Each subject answered a detailed questionnaire that includes questions on dietary habit, socio-economic condition family history and other relevant information concerned. Height was measured to the nearest 5 mm in stadiometer. Weight to the nearest 0.1 kg with the subject with light clothing without shoes. BMI was calculated as weight in kg divided by height in meter.[2]

Five ml of venous blood was collected after overnight fasting directly from antecubital vein by disposable syringe with gentle pull and transfer to a dry test tube labeled with the name of subjects, time and date of collection of blood. No anticoagulant was used. The test tube was then placed in a slanting position at room temperature until clot formation. At room temperature after clot had formed, the test tubes were centrifuged at 2000 to 3000 rpm for ten minutes. The serum obtained by centrifuging was collected in another test tube labeled with the date and time of collection. Experiment was carried out immediately after taking sample. In case of any delay, samples were stored in a refrigerator at 40 C for up to 24 hours.

Following biochemical analysis of serum was carried out for estimation of:

1. Total cholesterol by CHOD-PAP method
2. Triacylglycerol(TAG) by GPO-PAP method
3. High density lipoprotein cholesterol (HDL-C) by precipitation method.
4. Low density lipoprotein cholesterol (LDL-C) by using Friedwald's formula
5. Serum glucose by GOD-PAP method.

Results

All the results are expressed in mg/dl and the following observation were made from the results. The mean (±SD) of serum total cholesterol of control group and study group were 172. 68±29.9 mg/dl and 231. 00±46.57 mg/dl respectively. This result was highly significant P<0.001. The mean (±SD) Triacylglycerol values of control and those of study group were 149. 18 ± 38. 6 mg/dl and 206.09 ± 88. 58mg/dl respectively produced a highly significant (P<0.001) increased level. The mean (±SD) of serum HDL- cholesterol values of control group and those of study group were 61. 84±11. 17 mg/dl and 44. 39±12. 94 mg/dl respectively. The values were higher in control group. The result was highly significant (P<0.001). The mean (±SD) of serum LDL- cholesterol values of control group and those of study group were 80. 34 ±24. 50 mg/dl and146. 84±42. 50 mg/dl respectively. The values were higher in study group. The result was highly significant (P<0.001).

Table I: Comparative study of different biochemical parameters between study and control group

	Maximum		Maximum		Mean ± (Stand. Deviation)	
	Control ± group	Control ± group	Study group	Control ± group	Study group	Control ± group
FBG (mg/dl)	50	72	195	122	98.72 ± (27.66)	92.50 ± (10.65)
S.Chole sterol (mg/dl)	91	125	386	287	231.00 ± (46.57)	172.68 ± (29.9)
S.TAG (mg/dl	42	100	737	369	206.09 ± (88.58)	149.18 ± (38.60)
S.HDL (mg/dl	20	25	100	100	44.39 ± (12.84)	61.84 ± (11.77)
S.LDL (mg/dl	42	33	258	147	146.84 ± (42.50)	80.34 ± (24.50)

Abnormal HDL-cholesterol level (n=2) in study group
And (n=2) in control group

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Table II: National Cholesterol Education Program Adult Treatment Panel III

Measure fasting lipoproteins (mg/dL):	
< 200 (<5.17)	Desirable
200-239 (5.17-6.18)	Borderline high
240 (6.20)	High
LDL-cholesterol	
< 100 (<2.58)	Optimal
100-129 (2.58-3.33)	near optimal/above optimal
130-159 (3.36-4.11)	Borderline high
160-189 (4.13-4.88)	High
190 (4.91)	Very high
HDL-cholesterol	
< 40 (< 1.03)	Low
60 (1.55)	High
TG	TG
< 150 (< 1.695)	Desirable
150-199 (1.695-2.249)	Borderline high
200-499 (2.26-5.639)	High
500 (5.65)	Very high

Discussion

Assessment of socioeconomic status of the families was based on questions on family income and expenditure in the month preceding the interview. A comparative study of serum total cholesterol, triacylglycerol, HDL-C, fasting blood glucose, BMI, Waist circumference, Waist to hip ratio, blood pressure, pulse of control group and study group were done.

Serum total cholesterol

In this study there was significant increase in mean serum total cholesterol in study group in comparison to the control group. Significant increase in serum total cholesterol in obesity was also observed by in a smaller sample, higher body weight is associated with higher levels of total serum cholesterol in both men[19] and women[20] at levels of BMI> 25. Several large longitudinal studies also provide evidence that overweight, obesity and weight gain are associated with increased cholesterol levels,[20,21] in women, the incidence of hypercholesterolemia also increases with increasing BMI.[13] In addition, the pattern of fat distribution appears to affect cholesterol levels independently of total weight. Total cholesterol levels are usually higher in persons with predominant abdominal obesity.[22]

Serum triglycerides

In this study there was significant increase in mean serum triacylglycerol in study group in comparison to the control group. The strong association of triglyceride levels with BMI has been shown in both cross-sectional and longitudinal studies, for both sexes and all age groups.[22-24] In three adult age groups, namely 20 to 44 years, 45 to 59 years, and 60 to 74 years, higher levels of BMI, ranging from 21 or less to more than 30, have been associated with increasing triglyceride levels; the difference in triglycerides ranged from 61 to 65 mg/dL (0.68 to 0.74 mmol/L) in women 30 and 62 to 118 mg/dL (0.70 to 1.33 mmol/L) in men.[25]

High-density lipoprotein cholesterol

There was significant increase of mean serum HDL-cholesterol in control group in comparison to study group. HDL-cholesterol levels at all ages and weights are lower in men than in women. Although low HDL-cholesterol in this study was defined as < 35 mg/dL (0.91 mmol/L) in men and < 45 mg/dL (1.16 mmol/L) in women the panel accepts the definition of low HDL-cholesterol as < 35 mg/dL for men and women used by the National Cholesterol Education Program's Second Report of the Expert Panel on the Detection, Evaluation and treatmentof High Blood Cholesterol inadults (Adult Treatment Panel II Report).[27] Cross-sectional studies have reported that HDL-cholesterol levels are lower in men and women with higher BMI.[26-27] Longitudinal studies have found that changes in BMI are associated with changes in HDL-cholesterol. A BMI change of 1 unit is associated with an HDL-cholesterol change of 1.1 mg/dL for young adult men and an HDL- cholesterol change of 0.69 mg/dL for young adult women.[23] Blood lipid levels are often abnormal in obese persons. High-density lipoprotein (HDL) cholesterol, a higher level of which has been clearly implicated in decreased risk for coronary heart disease, is lower in obese persons [23,24]. It is not eworthy that in some other studies, low HDL-C levels were associated with obesity.[25]

Low-density lipoprotein cholesterol

In this study there was significant increase in mean serum LDL-cholesterol in study group in comparison to the control group. The link between total serum cholesterol and CHD is largely due to low-density lipoprotein (LDL). A high-risk LDL-cholesterol is defined as a serum concentration of <160 mg/dL. This lipoprotein is the predominant atherogenic lipoprotein and is therefore the primary target of cholesterol lowering therapy. Cross-sectional data suggest that LDL-cholesterol levels arehigher by 10 to 20 mg/dL in relation to a 10 unit difference in BMI, from levels of 20 to 30 kg/m2.[27,28]

According to extensive epidemiological data, a 10 mg/dL rise in LDL-cholesterol corresponds to approximately a 10 percent increase in CHD risk over a period of 5 to 10 years.[29] Few large-scale epidemiological data are available on small, dense LDL particles.[24-28] Clinical studies have shown that small, dense LDL particles are particularly atherogenic and tend to be present in greater proportion in hypertriglyceridemia patients with insulin resistance syndrome associated with abdominal obesity.[26-30] Elevated low-density lipoprotein cholesterol (LDL-C) is a major cause of coronary heart disease (CHD) associated with abdominal obesity.[26-30] Elevated low-density lipoprotein cholesterol (LDL-C) is a major cause of coronary heart disease (CHD).

Conclusion

Obesity impairs the lipid and lipoproteins metabolism. The observed mean total cholesterol, TAG, LDL-C were increased and HDL-C was decreased in obese persons. Large sample size, long duration and sophisticated technology are needed to understand the patho- physiology of underlying cause. Healthy food, life style modification and regular exercise should be taken to prevent obesity and obesity related complications.

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