(e) ISSN Online: 2321-9599

(p) ISSN Print: 2348-6805

ORIGINAL ARTICLE

Lipid profile in obese and non- obese individuals- A biochemical study

Johar Iqbal

Assistant Professor, Department of Biochemistry, Major S D Singh Medical College & Hospital, Farrukhabad, Uttar Pradesh, India

ABSTRACT:

Background: Research shows that obese people's lipid profiles can be improved by losing body weight (BW). The present study was conducted to assess lipid profile in obese and non- obese subjects. **Materials & Methods:** 114 subjects of both genders were recruited. Blood pressure, height, weight, and BMI were measured. They were divided into two groups by us. Subjects in group I had a normal BMI, while those in group II had a higher BMI. Triglycerides (TGL), total cholesterol, high density lipoprotein (HDL), and low-density lipoprotein (LDL) were examined as part of the lipid profile. **Results:** Group I had 30 males and 27 females and group II had 29 males and 28 females. The mean age in group I was 37.2 years and in group II was 38.4 years. BMI was 31.1 Kg/m^2 in group I and 23.4 Kg/m^2 in group II. The difference was significant ($P< 0.05$). The mean total cholesterol in group I was 190.2 mg/dl and in group II was 162.4 mg/dl, triglyceride was 167.4 mg/dl in group I and 128.6 mg/dl in group II, HDL cholesterol was 45.6 mg/dl in group I and 43.8 mg/dl in group II and LDL cholesterol was 138.5 mg/dl in group I and 116.3 mg/dl in group II. The difference was significant ($P< 0.05$). **Conclusion:** Obese and non-obese patients had significantly different levels of total cholesterol and LDL cholesterol.

Keywords: cholesterol, lipid, Obese

Corresponding author: Johar Iqbal, Assistant Professor, Department of Biochemistry, Major S D Singh Medical College & Hospital, Farrukhabad, Uttar Pradesh, India

This article may be cited as: Iqbal J. Lipid profile in obese and non- obese individuals- A biochemical study. J Adv Med Dent Scie Res 2014;2(4):281-283.

INTRODUCTION

Research shows that obese people's lipid profiles can be improved by losing body weight (BW). However, not enough research has been done on the relationship between lipid profile changes and BW in the general population, which includes both obese and non-obese people.[1] Obesity is one of the most overlooked public health issues of our time, according to the WHO, and it affects people everywhere.[2] Between 1980 and 2008, the prevalence of obesity roughly doubled globally. Overweight/obesity causes at least 2.8 million deaths worldwide each year. In India, obesity has become an epidemic, with 5% of the population suffering from morbid obesity.[3]

Excess body fat resulting from higher energy intake than energy expenditure is referred to as obesity. Adult obesity has been linked to a higher risk of metabolic syndrome.[4] Increased levels of free fatty acids due to insulin resistance, elevated LDL cholesterol, VLDL cholesterol, and triglycerides, and decreased HDL cholesterol are among the metabolic abnormalities that result from obesity.[5] The overproduction of VLDL is most likely caused by the liver's presentation of increased free fatty acids as a result of obesity, and this is likely the key to increased LDL via the sequence: VLDL→ intermediate density lipoprotein (IDL)→ LDL. It has also been demonstrated that the percentage of body fat and insulin levels are directly correlated with VLDL generation.[6]The present study was conducted to assess lipid profile in obese and non- obese subjects.

MATERIALS & METHODS

The present study comprised of 114 subjects of both genders. All agreed with their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. A comprehensive analysis was conducted. Blood pressure, height, weight, and BMI were measured. They were divided into two groups by us. Subjects in group I had a normal BMI, while those in group II had a higher BMI. Triglycerides (TGL), total cholesterol, high density lipoprotein (HDL), and low-density lipoprotein (LDL) were examined as part of the lipid profile. Results were compared and analysed statistically. P value less than 0.05 was considered significant.

RESULTS

Table I Comparison of parameters

Parameters	Group I (57)	Group II (57)	P value
M:F	30:27	29:28	0.96
Age (years)	37.2	38.4	0.64
BMI (Kg/m^2)	31.1	23.4	0.05

Table I, graph I shows that group I had 30 males and 27 females and group II had 29 males and 28 females. The mean age in group I was 37.2 years and in group II was 38.4 years. BMI was 31.1 Kg/m^2 in group I and 23.4 Kg/m^2 in group II. The difference was significant ($P< 0.05$).

Graph I Comparison of parameters

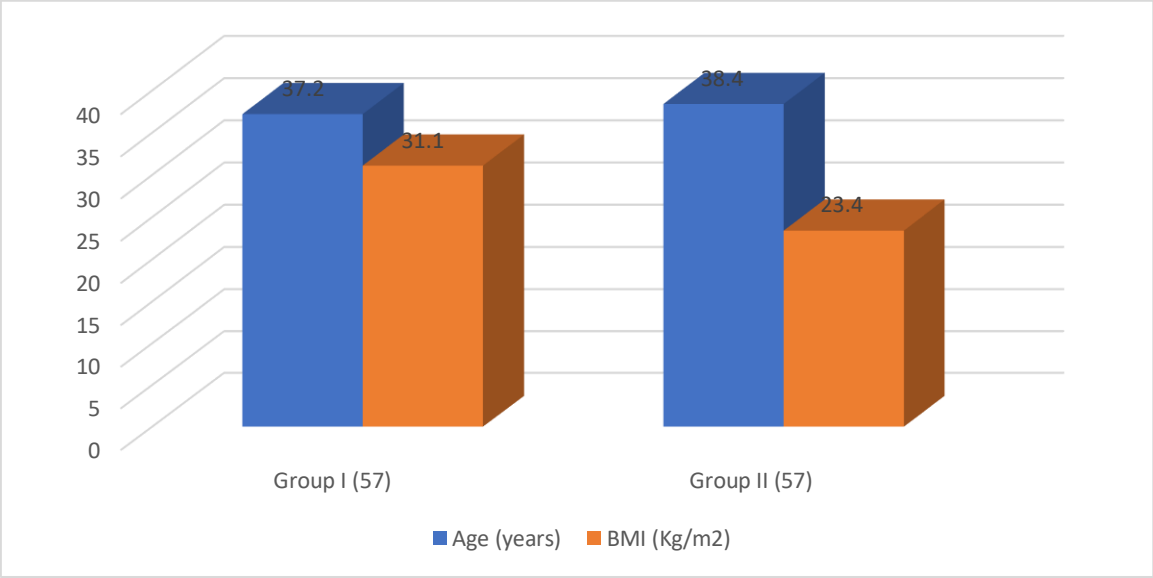

Table II Comparison of lipid profile

Parameters	Group I	Group II	P value
Total cholesterol (mg/dl)	190.2	162.4	0.02
Triglyceride (mg/dl)	167.4	128.2	0.05
HDL cholesterol (mg/dl)	45.6	43.8	0.86
LDL cholesterol (mg/dl)	138.5	116.3	0.04

Table II shows that mean total cholesterol in group I was 190.2 mg/dl and in group II was 162.4 mg/dl, triglyceride was 167.4 mg/dl in group I and 128.6 mg/dl in group II, HDL cholesterol was 45.6 mg/dl in group I and 43.8 mg/dl in group II and LDL cholesterol was 138.5 mg/dl in group I and 116.3 mg/dl in group II. The difference was significant (P< 0.05).

DISCUSSION

Obesity raises the risk of diabetes and cardiovascular disease, particularly when excess fat builds up in the central and intra-abdominal stores.8. Atherogenic dyslipidemia, which is defined by elevated plasma triglycerides, large very low-density lipoprotein (VLDL) particles, small dense low-density lipoprotein (LDL) particles, and low concentrations of high-density lipoprotein (HDL) cholesterol, is at least partially responsible for the elevated cardiometabolic risk associated with obesity.[7] Changes in the function of specific lipids as a result of peroxidation, an unbalanced fatty acid composition, or their changed flux from peripheral atherosclerosis and diabetes are also taken into consideration. Diabetes and obesity are not just health problems but also economic ones. A poor diet and a lack of physical activity are two main factors contributing to the shift in social andeconomic conditions.[8]As nations' economic levels rise to upper middle-income levels, the prevalence of elevated BMI rises as well. According to studies, male infertility has increased due to obesity in the reproductive age group. This may be linked to lower pregnancy rates and higher pregnancy loss in couples using artificial reproductive treatment.[9] The present study was conducted to assess lipid profile in obese and non-obese subjects.

We found that group I had 30 males and 27 females and group II had 29 males and 28 females. The mean age in group I was 37.2 years and in group II was 38.4 years. BMI was 31.1 Kg/m^2 in group I and 23.4 Kg/m^2 in group II. Mukhdhopadhey SK[10]studied the effect of cholesterol lowering agents on lipid profile in obese patients. Thirty obese patients received treatment with Lovastatin along with dietary measures, compared with age and sex matched controls- before and after 6 weeks of therapy. There was significant reduction in total cholesterol as well as LDL- cholesterol; HDL- cholesterol was also increased significantly. But triglycerides and VLDL-cholesterol showed small but significant increase. Cholesterol lowering agents like Lovastatin was quite effective when used long-term in dyslipidaemia in obesity towards reduction of risk factors for cardiovascular diseases, strokes, etc. Hypertriglyceridaemia should also be treated adequately.

We found that mean total cholesterol in group I was 190.2 mg/dl and in group II was 162.4 mg/dl, triglyceride was 167.4 mg/dl in group I and 128.6 mg/dl in group II, HDL cholesterol was 45.6 mg/dl in group I and 43.8 mg/dl in group II and LDL cholesterol was 138.5 mg/dl in group I and 116.3 mg/dl in group II. In participants without known diabetes (DM2), Gianni et al[11] evaluated the

association between visceral adiposity and lipid profile and fasting (FPG) and post-load glucose (2hPG). Three groups of 3030 subjects were created: nonobese subjects with an increased waist circumference, nonobese subjects without an increased waist circumference, and obese subjects (OB; n = 490). They conducted a linear regression analysis between the three groups' lipid fractions, fasting, and 2 hours postpartum, whether or not DM2 was diagnosed at that point. High triglycerides and low HDL-C were significantly correlated with fasting and 2hPG in all three groups, including non-HDL cholesterol. In contrast, total cholesterol (TC) was only significantly correlated with fasting glucose in OB and NOB/W+ subjects. Although a stronger association was seen in participants without DM2, the analysis with and without DM2 showed no difference in statistical significance. Additionally, a noteworthy trend in the prevalence of fasting hyperglycemia in obese and NOB/W+ individuals was noted for every quartile of TC.

Katsuki Aet al[12] investigated the association between visceral adiposity or triglyceride (TG) metabolism and insulin resistance in metabolically obese, normal weight (MONW) Japanese individuals with normal glucose tolerance. They evaluated body fat areas, lipid profiles, and the glucose infusion rate (GIR) during a euglycemic-hyperinsulinemic clamp study in 20 MONW subjects (BMI <25 kg/m^{2}and visceral fat areas 100 cm^2) with normal glucose tolerance. Body fat areas were measured by computed tomography scans. Control data were obtained from 20 normal subjects (BMI <25 kg/m^2 and visceral fat areas <100 cm^2). MONW subjects showed a significant increase in fasting serum levels of TG ($P < 0.01$) and a decrease in GIR ($P < 0.01$) compared with normal subjects. There were significant correlations between visceral fat areas ($r = -0.563, P < 0.01$) or serum levels of TG ($r = -0.474, P < 0.05$) and GIR in MONW subjects. Multiple regression analyses showed that visceral fat areas ($F = 7.702, P < 0.02$) and serum levels of TG ($F = 7.114, P < 0.05$) were significantly associated with GIR in all (MONW and normal) subjects.

The shortcoming of the study is small sample size.

CONCLUSION

Authors found that obese and non-obese patients had significantly different levels of total cholesterol and LDL cholesterol.

REFERENCES

1. Anjana RM, Pradeepa R, Das AK, Deepa M, Bhansali A, Joshi SR, Joshi PP, Dhandhania VK, Rao PV, Sudha V, et al. Physical activity and inactivity patterns in India – results from the ICMR-INDIAB study (Phase-1). Int J BehavNutr Phys Act 2014;11:26.
2. Yach D, Stuckler D, Brownell KD. Epidemiologic and economic consequences of the global epidemics of obesity and diabetes. Nat Med 2006 Jan;12(1):62-66.
3. Palmer NO, Bakos HW, Fullston T, Lane M. Impact of obesity on male fertility, sperm function and molecular composition. Spermatogenesis 2012 Oct 1;2(4):253-263.
4. Bakos HW, Henshaw RC, Mitchell M, Lane M. Paternal body mass index is associated with decreased blastocyst development and reduced live birth rates following assisted reproductive technology. Fertil Steril 2011 Apr;95(5):1700-1704.
5. Taskinen MR. Type 2 diabetes as a lipid disorder. Curr Mol Med 2005; 297–308.
6. Wenk MR. The emerging field of lipidomics. Nat Rev Drug Discov 2005;4: 594–610.
7. Despres JP, Moorjani S, Lupien PJ, Tremblay A, Nadeau A, et al. Genetic aspects of susceptibility to obesity and related dyslipidemias. Mol Cell Biochem 1992;113: 151–169.
8. Kalra S, Unnikrishnan AG. Obesity in India: the weight of the nation. J Med NutrNutraceut2012;1:37-41.
9. Bhatti MS, Akbri MZ, Shakoor M. Lipid profile in obesity. J Ayub Med Coll Abbottabad. 2001 Jan-Mar;13(1):31-3.
10. Giannini S, Bardini G, Dicembrini I, Monami M, Rotella CM, Mannucci E. Lipid levels in obese and nonobese subjects as predictors of fasting and postload glucose metabolism. Journal of clinical lipidology. 2012 Mar 1;6(2):132-8.
11. Mukhdhopadhey SK. Study of lipid profile in obese individuals and the effect of cholesterol lowering agents in them. Al Ameen J Med Sci 2012;5(2):147-151.
12. Katsuki A, Sumida Y, Urakawa H, Gabazza EC, Murashima S, Maruyama N, Morioka K, Nakatani K, Yano Y, Adachi Y. Increased visceral fat and serum levels of triglyceride are associated with insulin resistance in Japanese metabolically obese, normal weight subjects with normal glucose tolerance. Diabetes care. 2003 Aug 1;26(8):2341-4.