Original Research Article

Assessment of Dyslipidemia in Obese and Non-Obese Individuals: Cross-sectional Study

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Abstract

Background: Dyslipidemia is a disorder of lipoprotein metabolism includes both lipoprotein overproduction and deficiency. Overweight and obesity are major causes of co-morbidities including dyslipidemia, hyperglycaemia and non-alcoholic fatty liver disease. **Objective:** To evaluate and compare fasting serum lipid profile in non-obese and obese individuals. **Materials and Methods:** A Cross Sectional study was conducted in 50 Obese and 50 Non-Obese individuals in age group of 25 to 45 years. Fasting serum lipid profile was estimated. Statistical analysis was done by using unpaired t test. **Results:** Mean values of Total Cholesterol (TC), Triglycerides (TG), LDL-Cholesterol (LDL-C) and VLDL-Cholesterol (VLDL-C) were significantly higher while mean value of HDL-Cholesterol was significantly lower in obese individuals as compared to non-obese individuals. **Conclusion:** The high proportion of dyslipidemia in asymptomatic obese subjects emphasizes the need for routine health screening of obese individuals for early preventive measures.
Key Word: BMI; Obesity; Dyslipidemia

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INTRODUCTION

People are predisposed to various diseases based on their way of living and daily occupational habits. These lifestyle diseases characterize those diseases whose occurrence is primarily based on daily habits of people. The onset of these lifestyle diseases is insidious, they take years to develop and once encountered do not lend themselves easily to cure. The main factors contributing to these lifestyle diseases include bad food habits like consumption of diets rich in highly saturated fats, sugars, salt, fast food and lack of regular physical activity. An increased intake of energy dense foods that are high in fat and physical inactivity as indicated by a sedentary lifestyle both are associated with the occurrence of major lifestyle diseases like obesity and metabolic syndrome.[1] Obesity can be defined as a state of excess adipose tissue mass. Adipose tissue mass is increased by enlargement of adipose cells through lipid deposition as well as by an increase in the number of adipocytes. Adipose tissue in obese people is also characterized by increased number of infiltrating macrophages.[2] Globally prevalence of obesity has doubled between 1980 and 2008. In 2008, 10% of men and 14% of women in the world were obese (BMI ≥ 30 kg/m2) compared to 5% of men and 8% of women in 1980. According to recent WHO estimates in 2014, more than 1.9 billion adults (39%) aged 18 years and older were overweight of which over 600 million adults (13%) were obese.[3] Prevalence of obesity in India, a country whose 270 million people live below the poverty line seems to be a distant issue meant only for the rich kids of first world but India too is under the siege of junk food, alcohol and sedentary lifestyle which are leading us to silent self destruction, making one in every five Indian men and women either obese or overweight. India is reported to be just behind US and China in the global hazard list of top 10 countries with highest number of

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obese people. An exhaustive body of literature has already shown that overweight and obesity are major causes of co-morbidities including dyslipidemia, hypertension, hyperglycaemia and non-alcoholic fatty liver disease. The conglomeration of these conditions is known as metabolic syndrome. Obesity is an independent risk factor for dyslipidemia, type 2 diabetes mellitus and cardio vascular diseases which can lead to further morbidity and mortality. Obesity is considered to be the link between insulin resistance and metabolic abnormalities resulting in occurrence of dyslipidemia, diabetes and hypertension all of which are risk factors for coronary artery disease.[4-7] Dyslipidemia, a disorder of lipoprotein metabolism; includes both lipoprotein overproduction and deficiency. Dyslipidemia characterized by increased triglycerides (TG), decreased high density lipoprotein (HDL) levels and abnormal low density lipoprotein (LDL) composition with small dense particles has been reported among obese individuals.

With this perspective this comparative cross sectional study of serum lipid profile in obese and non obese individuals was undertaken with the objective to assess the lipid parameters among obese individuals and compare it with non obese individuals in the study subjects enrolled from Aurangabad city of Marathwada region in Maharashtra.

METHODS

Approval of institutional ethics committee was taken prior to commencement of this study. The study was undertaken in the Department of Physiology Government Medical College Aurangabad in collaboration with Department of Biochemistry. The present study is a Cross Sectional study conducted from December 2015 to December 2016. 50 Obese subjects having BMI $\geq$ 25 kg/m^2 between age group of 25 to 45 years and 50 non-obese controls having BMI between 18 to 22.9 kg/m^2 between age group of 25 to 45 years were selected from general population. BMI was calculated as per formula: Weight (Kg)/ Height (meter)^2 (Quetelet's Index). The procedure was explained to the obese subjects and non-obese controls in their mother tongue and informed consent was obtained.

Procedure for estimation of fasting serum lipid profile: Blood sample was collected after an overnight fast of 12 hours. 5 ml of blood was collected in a plain bulb from anti cubital vein under all aseptic precautions. The blood sample was allowed to clot and the serum was separated by centrifugation. Serum was stored at 2^{0}C till further processing. Lipid profile was done as per standard methods.

1. Serum total cholesterol done by Cholesterol Oxidase Peroxidase method (CHOD-POD). Reference values: 140 to 250 mg/dl (Erba Cholesterol kit manual).
2. Serum triglyceride estimation done by Lipase/ Glycerokinase/Glycerophosphate.
   Reference values: 25 to 160 mg/dl (Erba Triglyceride kit manual).
3. Serum HDL estimation done by selective inhibition method. Reference values: Males: 35.3 to 79.5 mg/dl, Female: 42 to 88 mg/dl (Agappe Direct HDL-C kit manual).
4. Serum VLDL and LDL estimation done by Friedewald's formula. VLDL (mg/dl) = TG/5, LDL (mg/dl) = Total Cholesterol - (HDL + VLDL).
   All tests were performed on Biochemistry analyzer Erba XL 640.

The results were analyzed by Graph pad prism software and Microsoft Excel 2007. The results were interpreted as mean ± S.D. p value was obtained from unpaired t test and value of < 0.05 was considered as statistically significant.

OBSERVATIONS

Table 1: Lipid profile parameters in Obese and Non Obese Individuals

Parameter	Non-obese Individuals (n=50) Mean ± S.D.	Obese Individuals (n=50) Mean ± S.D.	p value
TC (mg/dl)	169.28 ± 18.66	195.66 ± 31.71	<0.0001*
TG (mg/dl)	118.86 ± 23.68	181.1 ± 52.67	<0.0001*
LDL-C (mg/dl)	101.93 ± 19.94	120.24 ± 32.43	0.0010*
VLDL-C (mg/dl)	23.48 ± 4.81	35.76 ± 9.98	<0.0001*
HDL-C (mg/dl)	44.62 ± 8.56	38.68 ± 8.80	0.0009*

* Statistically highly significant

Mean age of study subjects was 34.82 ± 5.67 years in non obese group and 35.84 ± 6.07 years in obese group. Mean BMI of study subjects was 21.22 ± 0.84 in non obese group and 29.43 ± 3.37 in obese group. Table 1 show the study results.

DISCUSSION

The hallmark pattern of dyslipidemia seen in obesity is elevated triglyceride in combination with the preponderance of small dense LDL-C particles and low HDL-C level. The elevated triglycerides may be the major cause of the lipoprotein abnormalities since it will lead to delayed clearance of the triglyceride rich lipoproteins. Mechanism contributing to altered lipid profile in obesity is multifactorial and it involves,

A. Increased free fatty acids in circulation:

There are only two sources where plasma free fatty acids may be derived from; one is lipolysis of triglyceride-rich lipoproteins within the circulation and other intracellular lipolysis in adipose tissue. It is widely recognized that plasma free fatty acids are elevated in obese people as a consequence of an increased fatty acid release from adipose tissue and a reduction in plasma free fatty acid clearance. Increased free fatty acid delivery to the liver further increases synthesis of triglycerides and secretion of VLDL rich in triglycerides into circulation resulting in clinically detected elevation of fasting triglyceride blood levels. Triglyceride rich VLDL subsequently undergoes lipolysis by lipoprotein lipase to form LDL which is having less cholesterol-ester and high triglyceride content even after the activity of CETP. However, the increased TG content within the LDL is hydrolyzed by hepatic lipase which leads to the formation of small, dense LDL particles which is more toxic and atherogenic. The development of small dense LDL in obesity is mainly due to increased TG concentrations and these are relatively slowly metabolized with a five day residence time which enhances its atherogenicity. Small dense LDL also has an increased affinity for arterial proteoglycans resulting in their enhanced subendothelial retention. Various fatty acids are cytotoxic and their cytotoxicity depends on the type. Saturated fatty acids, arachidonic acid and linoleic acid can stimulate the synthesis of pro-inflammatory cytokines like IL-1, IL-6 and TNF-α, whereas eicosapentaenoic acid has anti-inflammatory properties. Since various fatty acids are cytotoxic, an escape mechanism is present in which insulin and the acylation-stimulating protein (ASP) / C3adesArg-pathway play an important role in peripheral fatty acid trapping. [8-9]

B. Impaired lipolysis:

Lipoprotein lipase (LPL) is the primary enzyme for triglyceride lipolysis in the circulation and is strongly expressed in tissues that require large amounts of free fatty acids like the heart, skeletal muscle and adipose tissue. Lipolysis is impaired in obesity by reduced mRNA expression of Lipoprotein lipase (LPL) in adipose tissue, reduction in Lipoprotein lipase (LPL) activity in skeletal muscle and competition between very lowdensity lipoprotein (VLDL) and chylomicrons for lipolysis. Lipoprotein lipase (LPL) activity is stimulated by insulin and inhibited by Apo C-III. Impaired lipolysis together with increased number of remnants of chylomicrons and VLDL strongly affect HDL metabolism. The increased number of triglyceride rich lipoproteins results in increased cholesterol ester transport protein (CETP) activity, which exchanges cholesterol esters from HDL for triglyceride from VLDL and LDL. Further lipolysis of this triglyceride rich HDL occurs by hepatic lipase resulting in small HDL which is thermodynamically unstable and results in reduced affinity for Apo A-I which leads to dissociation of apoprotein A-I from HDL. This further leads to reduction in circulating HDL particles with impairment of reversed cholesterol transport. [8-9]

C. Adipose tissue dysfunction:

Excessive adipocyte hypertrophy also causes dysfunction of organelles especially of the mitochondria and endoplasmic reticulum, hormone dysregulation and immunopathic responses. This dysfunction of adipose tissue and adipocyte is defined as adiposopathy or ‘‘sick fat’’. Adiposopathy is also associated with increased production of reactive oxygen species, tumor necrosis factor alpha (TNF-α), interleukin-6 (IL-6), C-reactive protein (CRP) (pro inflammatory responses) and decreased adiponectin (anti-inflammatory responses). It is also associated with oxidative stress. An increase in body fat increases the generation, accumulation and activity of adipose tissue associated macrophages. Adipose tissue macrophages may be responsible for almost all adipose tissue TNF-α expression and significant amounts of other inflammatory factors. Adipocytes and adipose tissue also secrete the most well-described immunologic bio-active proteins adipokines which include classic cytokines, complement factors, enzymes, growth factors, hormones and matrix proteins. An increase in body fat often increases the secretion of pro inflammatory adipokines by both adipocytes and adipose tissue-associated macrophages. The net effect of increased pro inflammatory adiposopathic responses and decreased anti inflammatory responses is the onset or worsening of metabolic disease including dyslipidemia.[8-9] Such pathogenic structural, endocrine and pro inflammatory consequences of adiposopathy further promote or worsen metabolic diseases in which dyslipidemia is central to the adverse clinical consequences of adipocyte and adipose tissue dysfunction. This adiposopathic dyslipidemic pattern is distinctly characteristic of the abnormal lipid levels found with pathogenic adipose tissue and adipocyte dysfunction and differs with other dyslipidemias related to isolated severe hypercholesterolemia which is often the result of genetic disturbances along with hormonal

dysfunction. Similar study findings were shown by: David C Nieman et al (2002) [10], Nagila A et al (2008) [11], Kawaljit Kaur et al (2014) [12], Priyanka N Pawaskar et al (2014) [13], Neha Saboo et al (2014) [14], Mohammad N Khan et al (2016) [15], Gulab Kanwar and Rahul Kabraa (2016) [16], Dharmishtha Chawada et al (2016) [17]. Some of the studies showed different findings: Rubina Mushtaq et al (2014) [18], Bridget O et al (2015) [19]. In the present study possible mechanism for alteration of lipid profile in obese individuals is hypertriglyceridemia due to increased plasma free fatty acids and adipose tissue dysfunction both of which collectively cause further lipid abnormalities. Further study with consideration of dietary factors, physical activity, socio-economic status and measurement of Apo B or non-HDL-C concentrations will help to explain in more detail the cause of alteration of lipid profile in obesity and will reflect the associated risk factors and specific abnormalities in lipid derangement.

CONCLUSIONS

Present study shows that levels of atherogenic lipids like total cholesterol (TC), low density lipoprotein cholesterol (LDL-C were raised and level of anti-atherogenic lipid like high density lipoprotein cholesterol (HDL-C) was lowered in individuals of obese group compared to individuals in non-obese group. This indicates that risk of cardiovascular diseases is increased in obesity due to alteration in lipid levels. The prevalence of dyslipidemia in asymptomatic obese subjects emphasizes the need for routine health screening for early preventive measures. Since both dyslipidemia and obesity are the risk factors for coronary artery diseases (CAD), deranged lipid profile may be the marker for the future development of coronary artery diseases (CAD). Early and immediate interventional measures like increase in physical activity, healthy dietary habits and regular surveillance are required in them to prevent development of irreversible complications. Hence, screening for dyslipidemia along with routine health examination is recommended in every obese which will enhance obesity related evaluation of cardiovascular risk factors and thus will help in prevention of future health hazards. It will also help in instituting corrective measures to reduce disease burden on population.

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