



Lipid profile Markers in Obese and Normal Weigh Individuals and their Relation with Obesity Determinants

Assadi Fatemeh and Nooraei Farahnaz
Department of Biology, North Tehran Branch,
Islamic Azad University, Tehran, IRAN

(Corresponding author: Assadi Fatemeh)

(Received 07 September, 2014, Accepted 17 October, 2014)

(Published by Research Trend, Website: www.researchtrend.net)

ABSTRACT: Accumulating evidence suggests that obesity plays an important role in developing type 2 diabetes and metabolic syndrome. In this study, we aimed 1) to compare lipid profile markers between obese and normal weight subjects and 2) to assess the relationship between these variables with obesity determinants in obese subjects. For this purpose, anthropometrical markers and fasting blood were collected in thirteen obese men (Body mass index, BMI, 30-36 kg/m²) and thirteen normal weight men (BMI, 20-25 kg/m²) matched for age 35-50 years. Blood used for triglyceride (TG), total cholesterol (TC), and low-density lipoprotein (LDL) and high-density lipoprotein (HDL). Pearson's correlation coefficients were used to evaluate the correlations between variables. At baseline, TG, TC and LDL levels were significantly higher in obese group in comparison to normal weight subjects ($p < 0.05$). Except HDL, a positive association was found between other lipid profile markers (TG, TC and LDL) with obesity determinants such as abdominal obesity, body fat (%) and visceral fat ($p < 0.05$). Our findings indicate that obesity might be a contributor to the increased prevalence of cardiovascular disease or metabolic syndrome.

Keywords: Obesity, Metabolic syndrome, Lipid profile

INTRODUCTION

Over the past two decades, obesity has been considered the main focus of health science researchers as one of the most significant metabolic uncontagious disorders. The mounting increasing risk of obesity and its following hazardous consequences, along with the industrialization of societies and lifestyle shifts, has made the prevention and treatment of obesity outstand as a subtle challenge for health systems. Increased fat percentage and adipose tissue in obesity, on top of the emotional distress and the fatigue due to tolerating the extra supply of body fat, also disrupts the endocrine system and impairment of hormone or peptide mediator's levels that interpose substantially to promote these forms of illness in the obese population [1].

Thus, it is clearly vivid that adipose tissue besides being the most massive reservoir of fat in the body, it is also an endocrine organ, that secretes enormous amounts of peptide mediators in the form of adipokines or adipocytokines [2]. In this regard, it has been shown that obesity is not only influenced by impairment of some metabolic factors like fat profile markers including TG, TC, LDL, and HDL but also in many instances by the

disruption of adipose tissue endocrine functions. The prevalence of these diseases cumulatively correlates with metabolic syndrome intensifies disease status [3]. Some studies have shown that inflammatory cytokines increase production of vLDL, which in turn justifies the correlation of cytokine with plasma TG [4]. The direct and significant integrity of inflammatory cytokines such as TNF- with TG and cardiovascular diseases have also been reported by other studies [5]. Conversely, although medical references have advocated the raise of cardiovascular risk factors in the obese population compared to the normal society, nevertheless, apart from body weight, the correlation amongst cardiovascular risk factors with other Anthropometric indexes for instance abdominal perimeter and body fat percentage has been less explored. Therefore, the following study deliberates to explore the linkage amongst cardiovascular risk factors incorporating the two parameters of abdominal perimeter and body fat percentage in addition to the association of cardiovascular risk factors among obese and normal populations.

MATERIALS AND METHOD

Thirteen adult obese (BMI, 30-36 kg/m²) and the same number normal weight (BMI, 20-25 kg/m²) matched for sex (men) and age (35-50 kg) participated in the study. All subjects were otherwise in good health were taking no medications. All subjects were non-smokers. All participants had not participated in regular exercise/diet programs for the preceding 6 months. We also excluded people who had any self reported physician diagnosed chronic disease (arthritis, stroke, diabetes, hypertension, cancer, heart attack, chronic cough, or bronchitis). The study was conducted with the approval of the Ethics Committee of the Islamic Azad University, Iran. All study participants completed the consent process and provided written informed consent prior to randomization.

A. Anthropometry and biochemistry

Both populations underwent anthropometric measurements. All of measurements were conducted by the same researcher. Standing height was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. Body weight was measured in duplicate in the morning following a 12-h fast. Obesity was measured by body mass index (BMI). Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared (m²). Waist circumference (WC) was measured at the superior border of the iliac crest and was taken to the nearest 0.1 cm after a normal expiration. Waist-hip ratio (WHR) was calculated. Each of these measurements was conducted three times and the average was reported.

Fasting blood samples were taken for measure the lipid profile markers. Triglyceride, total cholesterol, HDL and LDL-cholesterol was measured directly with enzymatic methods (Randox direct kits) using Kobas Mira auto-analyzer made in Germany. All participants refrained from any severe physical activity 48 h before measurements.

B. Statistical Analysis

Data were analyzed by computer using the Statistical Package for Social Sciences (SPSS) for Windows, version 15.0. We verified normal distribution of variables with a Kolmogorov-Smirnov test, and the parametric variables with skewed distribution were expressed as mean \pm SD. Independent student t test was used for between groups comparison at baseline. The correlations between variables were determined using the bivariate correlation test (Pearson's correlation coefficients). A p value of less than 0.05 was considered as statistically significant.

RESULTS

In this study, at first, we compared cardiovascular risk factors between obese and normal weight healthy untrained men. Anthropometric and metabolic characteristics of the study participants in the normal and obese groups are shown in Table 1.

Data of independent t test showed that significant difference in TG, TC and LDL between two groups. Serum triglyceride levels were significantly higher in obese group in comparison to normal weight subjects ($p = 0.039$). Total cholesterol ($p = 0.036$) and LDL ($p = 0.000$) levels in obese subjects showed were significantly higher than those with normal weight. There was no statistically significant difference in HDL-cholesterol between two groups ($p = 0.728$). The association between lipid profile markers and obesity determinants are showed in table 2. Data of correlation analysis showed that abdominal obesity is positively correlated with TG, TC and LDL-cholesterol ($p < 0.05$). Body fat percentage was also positively correlated with TG, TC and LDL-cholesterol ($p < 0.05$). A significant positive correlation was also between TG, TC and LDL with other obesity determinants such as BMI, body weight and hip circumference ($p < 0.05$).

DISCUSSION

In the present study, serum levels of each lipid profile indicators, such as TG, TC and LDL-C in adult obese men were significantly higher than those of normal weight men. However, these findings somehow support the idea of increased cardiovascular risk factors among the obese population and emphasizes on the theme that obesity is the foreground of cardiovascular disorders. Nonetheless, the findings of the present study recites of no significant differences in HDL as a supplementary indicator of lipid profile in obese men compared to men of normal weight.

The belief that the role of adipose tissue is being the sole resort of body fat reservoir in the form of Triglycerides or fatty acids has been replaced in recent years by the new theory; that, this tissue also has a crucial role in the metabolism of fat and carbohydrates, plus, the secretion of many other numerous hormones such as angiotensin, adiponectin, and Leptin [6]. The prevalence of obesity (increased adipose tissue) and the risk factors correlated with obesity correspond closely to the prevalence of cardiovascular diseases and type 2 diabetes [7, 8]. Impairment of blood flow, and other vascular ailment risk factors, in coexistence with obesity are most predictive exclamations of metabolic syndrome disorders [9].

Table 1: Anthropometrical and metabolic markers of two groups.

	Obese group = 1 Normal group = 2	Mean	Std. Deviation
Age (year)	1	38.08	1.98
	2	37.77	1.24
Height (cm)	1	173.15	4.49
	2	170.92	1.8
Weight (kg)	1	93.85	7.7
	2	67.23	1.96
Abdominal (cm)	1	104.08	4.6
	2	87.00	2.7
Hip (cm)	1	106.38	4.81
	2	94.54	2.99
WHO	1	.9786	.02
	2	.9213	.04
BMI (kg/m2)	1	31.27	1.89
	2	23.01	.29
Body fat (%)	1	31.85	1.34
	2	21.80	.99
Visceral fat	1	13.4	2.06
	2	7.5	1.05
Total cholesterol (mg/dl)	1	199	39
	2	165	38
Triglyceride (mg/dl)	1	189	53
	2	143	56
Low density lipoprotein (mg/dl)	1	132	24
	2	92	13.62
High density lipoprotein (mg/dl)	1	47.3	4.4
	2	47.8	3.3

Table 2: The association in lipid profile markers (cardiovascular risk factor) with obesity determinants of all participants.

	TG	TC	LDL	HDL
Weight	= 0.01, r = 0.48	= 0.02, r = 0.46	= 0.002, r = 0.57	= 0.99, r = 0.001
Waist circumference	= 0.002, r = 0.58	= 0.01, r = 0.48	= 0.000, r = 0.64	= 0.98, r = 0.005
Hip circumference	= 0.09, r = 0.33	= 0.03, r = 0.42	= 0.04, r = 0.41	= 0.93, r = 0.02
BMI	= 0.01, r = 0.50	= 0.02, r = 0.44	= 0.001, r = 0.61	= 0.98, r = 0.01
Body fat (%)	= 0.02, r = 0.45	= 0.03, r = 0.43	= 0.000, r = 0.67	= 0.99, r = 0.01
Visceral fat	= 0.02, r = 0.46	= 0.02, r = 0.46	= 0.002, r = 0.59	= 0.60, r = 0.11

From pathophysiological point of view, the quality of fat tissue is far more important than quantity of fat tissue. Crucially, however, the main function of adipose tissue or visceral fat is based on the quantity and size

[6]. Adipocytes and adipose tissue have a wide range of cytokine secretions which impinge on glucose metabolism, lipid metabolism, inflammation, blood clotting, blood pressure, and eating behaviors [10-12].

On the other hand, impaired secretion of adipocytokines from adipose tissue somehow impacts levels of lipid profile indicators or cardiovascular risk factors [13, 14]. In other words, obesity is associated with systemic inflammation in both humans and animals, and increased inflammation is associated with cardiovascular and metabolic disorders [15, 16].

Alternatively, it is known that, promoting weight loss in obese subjects marks amelioration of cardiovascular risk factors and amendment of serum C-reactive protein, and lipid levels in the blood [17]. Obese patients continuously demonstrate increased levels of fasting glucose, lipids, and insulin resistance [18-20]. Thus, however, some research have indicated no association between obesity and metabolic risk factors, such as TC and LDL [21]. Yet, other research have discovered, higher risk levels of metabolic factors subsequent to Leptin and CRP, as inspirational peptides in lipid and carbohydrate metabolism in obese patients compared to normal weight subjects [22]. In this context, the findings of another study, introduced the measurement of waist circumference as one of the referring markers of obesity, one of the most important factors of metabolic syndrome diagnosis [22].

In the present study, in addition to higher levels of cardiovascular risk factors such as TG, TC, and LDL in obese men than in normal weighted men; consequently, a significant correlation between these risk factors and indexes of obesity, such as waist circumference, visceral fat, and body fat percentage can also be perceived. In other words, the correlation between cardiovascular risk factors with obesity referring indexes such as abdomen perimeter, visceral fat, and body fat percentage were established. In other terms, the correlation between cardiovascular risk factors with obesity referring indexes in both groups of obese and normal weight subjects were direct and significant, in turn, supporting the cardiovascular risk factor theme, they collectively, have a trivial role in obesity and increased percentage of fat circulating the blood. In contrast to these findings, no significant association between HDL and obesity referring indexes were understood in both groups. As a general conclusion, the findings of this study, advocates that cardiovascular risk factors are in higher levels in obese subjects compared to normal weight men. However, the lack of significant difference of HDL levels between obese and normal weight subjects in this study is probably due to scarcity of inquiring samples.

REFERENCES

- Hart CL, Hole DJ, Lawlor DA, Smith GD. (2007). Obesity and use of acute hospital services in participants of the Renfrew/Paisley study. *J Public Health (Oxf)*. **29**: 53-6.
- Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med*. **346**: 393-403.
- Bays H, Abate N, Chandalia M. (2005). Adiposopathy: sick fat causes high blood sugar, high blood pressure and dyslipidemia. *Future Cardiol*. **1**(1): 39-59.
- Mari A, Schmitz O, Gastaldelli A. (2002). Meal and oral glucose tests for assessment of beta-cell function: modeling analysis in normal subjects. *Am J Physiol Endocrinol Metab*. **283**: 1159-1166.
- Ludvik B, Nolan JJ, Baloga J. (1995). Effect of obesity on insulin resistance in normal subjects and patients with NIDDM. *Diabetes*. **44**: 1121-1125.
- Hajer GR, van Haeften TW, Visseren FL. (2008). Adipose tissue dysfunction in obesity, diabetes, and vascular diseases. *Eur Heart J*. **29**(24): 2959-71.
- Ford ES. (2005). Prevalence of the metabolic syndrome defined by the International Diabetes Federation among adults in the U.S. *Diabetes Care*. **28**: 2745-2749.
- Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai P Jr, Razak F, Sharma AM, Anand SS, INTERHEART Study Investigators. (2005). Obese and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet*. **366**: 1640-1649.
- Executive summary of the third report of The National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *JAMA* 2001; **285**: 2486-97.
- Yamauchi T, Kamon J, Waki H, Terauchi Y, Kubota N, Hara K, Mori Y, Ide T, Murakami K, Tsuboyama-Kasaoka N, Ezaki O, Akanuma Y, Gavrilova O, Vinson C, Reitman ML, Kagechika H, Shudo K, Yoda M, Nakano Y, Tobe K, Nagai R, Kimura S, Tomita M, Froguel P, Kadowaki T. (2001). The fat-derived hormone adiponectin reverses insulin resistance associated with both lipodystrophy and obesity. *Nat Med*. **7**: 941-946.

- Ran J, Hirano T, Fukui T, Saito K, Kageyama H, Okada K, Adachi M. (2006). Angiotensin II infusion decreases plasma adiponectin level via its type 1 receptor in rats: an implication for hypertension-related insulin resistance. *Metabolism*. **55**: 478-88.
- Chu NF, Spiegelman D, Hotamisligil GS, Rifai N, Stampfer M, Rimm EB. (2001). Plasma insulin, leptin, and soluble TNF receptors levels in relation to obesity-related atherogenic and thrombogenic cardiovascular disease risk factors among men. *Atherosclerosis*. **157**: 495–503.
- Oda E. (2013). High-sensitivity C-reactive protein and white blood cell count equally predict development of the metabolic syndrome in a Japanese health screening population. *Acta Diabetol*. **50**(4): 633-8.
- Sanip ZI, Ariffin FD, Al-Tahami BA, Sulaiman WA, Rasool AH.(2013). Obesity indices and metabolic markers are related to hs-CRP and adiponectin levels in overweight and obese females. *Obes Res Clin Pract.*, **7**(4): 315-20.
- Singer K1, Eng DS, Lumeng CN, Gebremariam A, Lee JM. (2014). The relationship between body fat mass percentiles and inflammation in children. Obesity (Silver Spring). [Epub ahead of print]
- Hribal ML, Fiorentino TV, Sesti G1. (2014). Role of C Reactive Protein (CRP) in Leptin Resistance. *Curr Pharm Des*. **20**(4): 609-15.
- Dow CA, Thomson CA, Flatt SW, Sherwood NE, Pakiz B, Rock CL. (2013). Predictors of improvement in cardiometabolic risk factors with weight loss in women. *J Am Heart Assoc*. **2**(6): 000152.
- Gregg EW, Cheng YJ, Cadwell BL, Imperatore G, Williams DE, Flegal KM, Narayan KM, Williamson DF. (2005). Secular trends in cardiovascular disease risk factors according to body mass index in US adults. *JAMA*. **293**: 1868-74.
- Lumeng CN, Saltiel AR. (2011). Inflammatory links between obesity and metabolic disease. *J Clin Invest*. **121**: 2111–2117.
- Taylor R. (2008). Pathogenesis of type 2 diabetes: tracing the reverse route from cure to cause. *Diabetologia*. **51**: 1781–1789.
- Bal Y, Adas M, Helvaci A. (2010). Evaluation of the relationship between insulin resistance and plasma tumor necrosis factor-alpha, interleukin-6 and C-reactive protein levels in obese women. *Bratisl Lek Listy*. **111**(4): 200-4.
- Li CW, Jiang DL, Qiao J, Sun JM, Huang JP, Chen HP, Zhu H. (2010). Changes of leptin resistance, blood lipids and inflammatory response before and after the exercise therapy in children with obesity *Zhongguo Dang Dai Er Ke Za Zhi*. **12**(1): 40-2.