The Effect of Obesity on Serum Leptin and Lipid Profile

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ABSTRACT:

BACKGROUND:

Obesity has become a leading health concern; this condition is a chronic, complex, multifactorial disease in which a person's weight is ≥ 20% of the ideal weight for a given height.

OBJECTIVE:

To measure serum leptin level and lipid profiles levels in Iraqi obese individuals comparing the results with matching non obese subjects.

METHODS:

The study was carried on 30 individuals divided into two groups:

1. Obese subjects group (n=20).
2. Non-obese subjects group (n=10).

For all subjects studied measurements of fasting serum leptin and lipid profile have been done.

RESULT:

Our study showed that mean serum leptin level was significantly higher in obese than non-obese individuals ($P<0.001$). In addition serum leptin correlates positively and strongly with body mass index (BMI) ($r=0.765, P<0.01$). Serum leptin also correlates positively with both triglyceride (TC)($r=0.394$, $P<0.05$) and low density lipoprotein (LDL) ($r=0.366, P<0.05$) but correlates negatively with high density lipoprotein (HDL)($r=-0.408, P<0.05$).

CONCLUSION:

circulating leptin levels appear to be one of the best biological markers of obesity and hyperleptinemia is closely associated with several risk factors related to obesity syndrome.

KEYWORDS: obesity, leptin, lipid profile.

INTRODUCTION:

OBESITY

It is a state of excess adipose tissue mass[1]. Worldwide, there is an obesity epidemic. Greater number of men and women have become obese during the past decade. The main causes of obesity epidemics are sedentary lifestyle and consumption of energy dense diet [2,3]. At one level, the pathophysiology of obesity seems simple; a chronic excess of nutrient intake relative to the level of energy expenditure [1].

Lipids are ubiquitous in the body tissue as they have an important role in virtually all aspects of life acting as energy stores (Triglycerides), important structural component of cells (Cholesterol) and they could also have special functions (Hormones) [4].

The serum concentrations of cholesterol and triglycerides are positively correlated with obesity. one study in twins has shown that for an average increase of 7.3% in body mass index (BMI) there were increases of 2.5% in total cholesterol concentration, 3% in low density lipoprotein (LDL) cholesterol concentration, and 18.2% in triglyceride concentration. the increase in the concentration of cholesterol is attributable to LDL cholesterol because the high density lipoprotein (HDL) cholesterol is typically reduced [5].

Even simple obesity is associated with hyperlipidemia (typically mixed) although dyslipidemia is low among younger ages. the likelihood of these conditions is substantially greater among obese individuals. moreover, not only does the prevalence of these conditions increases with age, but the rate of increase is more rapid among obese individuals [6].

The regulation of body weight comprises a complex homeostatic system that results in a balance between energy intake and energy expenditure. the hypothalamus is the central coordinating area for this system [1,7].

With weight loss, appetite increases and energy expenditure falls. With overfeeding, appetite falls and energy expenditure increases. this latter regulatory mechanism frequently fails, however, permitting obesity to develop when food is

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abundant and physical activity is limited. a major regulator of these adaptive responses is the adipocyte-derived hormone Leptin,which acts through the hypothalamus to influence appetite, energy expenditure and neuroendocrine functions [1].

Leptin is a hormone made by adipocytes & mainly acts centrally to control body weight [8]. It conveys information to the brain about the size of energy stores & stimulates the hypothalamic centers responsible for regulation of energy intake & expenditure [9]. Its name is derived from the Greek root ***Leptos***, meaning thin [1].

Plasma leptin levels in humans are strongly correlated with the Body Mass Index (BMI) and total fat mass and are mostly elevated in obese subjects [10]. Leptin synthesis is induced by hyperglycemia, hyperlipidemia, and a replete fat mass and also leptin suppresses insulin production [11].

Leptin's effects on body weight are mediated through effects on hypothalamic centers that control feeding behavior and hunger, body temperature and energy expenditure [12]. In essence, leptin provides the body with an index of nutritional status [13].

AIM OF THE STUDY:

To measure serum leptin level in Iraqi obese subjects comparing the results with matching non obese subjects. In addition, the relationship between serum leptin & serum lipid profile in all study subjects was studied.

PATIENTS AND METHODS:

PATIENTS:

Our study was conducted in Medical City, Baghdad Teaching Hospital and Teaching Laboratories during the period from June 2007 to December 2007.

A total number of 30 patients included in the study were divided according to their BMI into two groups:

1. Obese individuals group BMI≥25 kg/m^2 (n=20).
2. Non obese individuals group BMI<25kg/m^2 (n=10).

Body Mass Index (BMI) was the only anthropometric parameter specified, weight& height were measured by the same scale for all subjects according to the following equation:

BMI = weight (kg)/ square height (m^2) [1]

METHODS:

The following biochemical tests were done for all study subjects:

1. Measurement of Leptin in serum using the Leptin (sandwich) Enzyme immunoassay kit. This assay is intended for *in vitro* diagnostic use only. It is a solid phase enzyme-linked immunosorbent assay (ELISA) based on the sandwich principle.
2. Lipid profile assessment using Kits from SPINREACT-CЄ to measure Triglycerides (TG), Total Cholesterol (TC), Low Density Lipoprotein (LDL) & High Density Lipoprotein (HDL) levels in serum.

Statistical analysis

All data were arranged and tabulated in number and percentage. To compare the significance of the difference in the mean values of any two groups chosen, student t-test was applied; P<0.05 was considered statistically significant. Pearson correlation coefficient (R) test is used to describe the association between the different studied parameters; P<0.05 was considered statistically significant.

RESULT:

Statistical analysis of Study Subjects:

There was no significant difference in mean age& TG levels between obese and non-obese subjects, P>0.05.

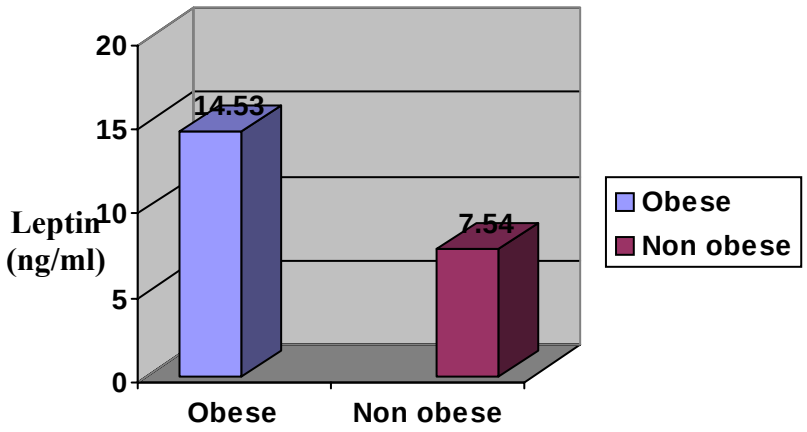
Mean serum leptin level was significantly higher in obese subjects than non-obese subjects, P<0.001. Fig(1). BMI was also significantly higher in obese subjects than non-obese subjects, P<0.001.

Serum levels of HDL were lower in obese subjects compared to non-obese subjects, P<0.05. LDL and total cholesterol levels were higher in obese subjects compared to non-obese subjects, P values being 0.006 and 0.007 respectively. Table(1).

Table (1): Statistical Data of Study Subjects

Characteristics	Obese	Non-obese	P value
Number –(%)	20 (66.7%)	10 (33.3%)	_
Age (years) ± SD	52.(10±7.21)	52.(10±6.81)	NS
TG (mg/dl) ± SD	126(±29.62)	139(±38.25)	NS
Cholesterol(mg/dl) ± SD	208.(75±23.16)	188(±15.31)	0.007
HDL (mg/dl) ± SD	42.(85±8.22)	48.(50±6.48)	<0.05
LDL (mg/dl) ± SD	141.(20±22.69)	121.(90±12.44)	0.006
BMI (kg/m^2) ± SD	34.(94±4.24)	23.(95±1.009)	<0.001
Leptin (ng/ml) ± SD	14.(53±1.89)	7.(54±1.08)	<0.001

P value < 0.05 is significant



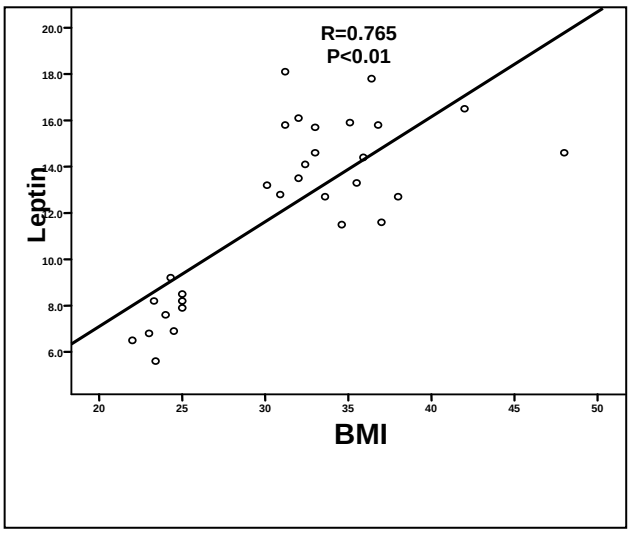
Figure(1): Serum Leptin level in study subjects

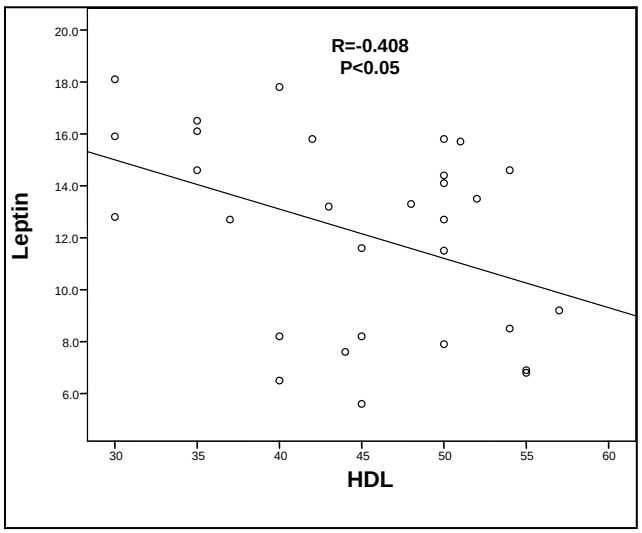
Correlations:

Our study shows that serum leptin correlates positively and strongly with BMI (R=0.765,P<0.01). Fig(2-A). Serum leptin also correlates positively with both TC (R=0.394, P<0.05) and LDL (R=0.366,P<0.05) but correlates negatively with HDL (R= -0.408,P<0.05). Fig(2-B).

Serum LDL correlates positively with both TC(R=0.823,P<0.01) and BMI (R=0.370,P<0.05). This study also shows that serum HDL correlates negatively with BMI (R=-0.377,P<0.05).

(A)





(B)

Figure(2): Correlation between serum leptin level and (A) BMI, (B) HDL in study subjects

DISCUSSION:

The current working hypothesis is that factors produced and released by white adipose tissue including adipokines such as leptin, cytokines such as IL-1 & TNF-α, and others are responsible for a chronic subclinical pro-inflammatory state in obese subjects [14].

Leptin is expressed predominantly by adipocyte, which fits the idea that body weight is sensed as a total mass of fat in the body[15]. Serum leptin concentration is increased in obese subjects and is closely related to fat mass and BMI and declines with weight loss [16]. Leptin plays a central role in the long-term maintenance of weight homeostasis by acting on the hypothalamus to decrease food intake and increase energy expenditure [1].

Serum leptin was positively& strongly correlated with BMI which is an important index of obesity. Similar correlation was reported by previous studies [12, 18, and 19].

Minocci A. *et al* concluded that fat distribution contributes to the variability in serum leptin in

obese patients. In particular, subcutaneous abdominal fat is a determinant of leptin concentration [20].

The inability of such elevated leptin levels in most obese individuals to alter the obese state of subjects may be related to "leptin resistance "[21], an inability of leptin to enter the cerebral spinal fluid to reach the hypothalamus regions that regulate appetite, or it may simply reflect the large amount of fat tissue in the body [22].

Obesity is associated with several deleterious changes in lipid metabolism, including high serum concentrations of total cholesterol, LDL, VLDL and TG, and reduction in serum HDL concentration [24]. Previous studies showed that in all age groups, HDL levels were significantly lower in patients who had a high BMI [25]. Hypertriglyceridemia is often associated with reduced levels of HDL suggesting a possible metabolic interaction between these two lipid fractions [26]. The key to this relation may be that the increase in fat deposition in obese individuals is associated with insulin resistance [27], which will lead to increase synthesis of TG-rich lipoproteins in the liver. The increase of TG in lipid particles changes their metabolism. TG-rich HDL particles are hydrolyzed more rapidly causing HDL level to fall [28].

The significant association between obesity, BMI and lipid profile in our results is consistent with what was reported by other authors [29-31].

The significant correlations between leptin levels and plasma lipids and between obesity and leptin levels suggest that changes in these parameters play a significant role in determining serum leptin concentrations in obese subjects [32].

CONCLUSION:

For our confined number of subjects we conclude that there is an elevated serum leptin level in obese subjects when compared with non-obese ones. In addition, elevated serum leptin level is associated with abnormal lipid profile.

Thus circulating leptin levels appear to be one of the best biological markers of obesity& hyperleptinemia is closely associated with several risk factors related to obesity syndrome.

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