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Estimation of Serum Leptin Levels and Its Effect on Body Mass Index: A Teaching Hospital Based Study

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ABSTRACT

Background: Defects in leptin production or function are associated with obesity in animal models and humans. Though increased leptin levels have been demonstrated in obesity implicating leptin resistance in human obesity, there is a paucity of data regarding mutations in leptin and leptin receptor genes. **Subjects and Methods:** All the 90 subjects were divided into three groups according to their body mass index. **Results:** The levels of BMI, FBS, total cholesterol, triglycerides, LDL and leptin were higher in overweight obese; while the HDL levels were lower as compared to control group. Serum leptin levels in overweight, obese were significantly higher than controls. **Conclusion:** Positive correlation was observed between leptin levels and age & BMI.

Keywords: Body mass index, Leptin and Obesity

INTRODUCTION

Leptin, a 167 amino acid 16-kDa peptide product of ob gene, secreted by the adipose tissue plays a crucial role in the regulation of appetite, glucose homeostasis and body fat. Defects in leptin production or function are associated with obesity in animal models and humans. Though increased leptin levels have been demonstrated in obesity implicating leptin resistance in human obesity[1], there is a paucity of data regarding mutations in leptin and leptin receptor genes[2-3]. Leptin expression is regulated by several tightly controlled factors influencing energy metabolism and levels of leptin are related to parameters of obesity.

It is now known to be an important regulator of body weight, triggering various physiological mechanisms according to the state of the body's energy balance: serum leptin levels act as sensors of the energy balance, communicating information on energy accumulated in adipose tissue to the hypothalamus. High levels lead to reduced appetite and increased energy expenditure, while low levels, reflecting weight loss, increase appetite and reduce energy expenditure.

However, in obese individuals, these mechanisms are impaired: high leptin levels do not have these effects. This is leptin resistance. The physiological mechanisms behind leptin synthesis, secretion and receptor binding and its regulation of appetite and energy expenditure involve numerous hormones and neurotransmitters. These mechanisms are complex and are not fully understood [4, 5, 6]. In this study BMI was used as an indicator of general obesity. This present study was aim to the estimation of serum leptin levels in obese subject and to observe its correlation to demographic and biochemical parameters of obesity.

Subjects and Methods:

This present study was conducted in the P.G Department of Biochemistry, VIMSAR, Burla, in collaboration with General medicine Department during the period from October, 2018 to November, 2019. The study was approved by institutional ethical committee and informed consent was obtained from individuals. Randomly selected, 90 subjects with an age ranged from 21 to 55 years. After complete history taking, anthropometric parameters were recorded. Subjects with any evidence of disease, illness, addictions or taking any medications were excluded from the study. Fasting venous blood samples were drawn and checked for serum blood sugar, lipid profile on Roche e411 Chemistry Analyzer and Serum Leptin by Full Automated Elisa Reader. All the 90 subjects were divided into three groups according to their body mass index:

Group A: 30 subjects as control (BMI 18.5–24.9 Kg/m2)

Group B: 30 Overweight subjects (BMI 25.0–29.9 Kg/m2)

Group C: 30 Obese subjects (BMI ≥30.0 Kg/m2)

Statistics:

- All the results were expressed in mean± SD.
- The significance of difference between the two groups was done by unpaired student t-test.
- p value of <0.05 was considered statistically significant.
- Pearson correlation coefficient was used to evaluate any relationship between different variables.

Statistical analyses were done by SPSS-20 software.

Observation and Results:

A total of 90 subjects were included; 30 healthy subjects in group-A, 30 overweight subjects in group-B and 30 obese subjects in group-C. Mean values of all parameters and p-values are given in table-1, 2 &3. The results which were obtained from the controls, overweight and obese were shown in [Table1, 2 & 3]. The levels of BMI, FBS, total cholesterol, triglycerides, LDL and leptin were higher in overweight obese, while the HDL levels were lower as compared to control group. Serum leptin levels in overweight, obese were significantly higher than controls (Table I, 2, 3). Fasting blood sugar levels were significantly higher in overweight, obese as compared to controls. Serum total cholesterol, triglycerides, and LDL-cholesterol levels were significantly higher in overweight, obese as compared to controls group while HDL-cholesterol levels were not significantly different in the all the threegroup. Positive correlation was observed between leptin levels and age (r = 0.37; p=0.04) & BMI (r = 0.45; p=0.01). No correlation of leptin with fasting blood glucose levels was observed in this study. The relationship between serum leptin and lipids remains unclear. Positive correlation was seen between serum leptin and total cholesterol, LDL and triglycerides. No correlation was seen with HDL-cholesterol.

Table-1: Comparison of demographic and biochemical data in groups (A&B):

Parameters	Group A Mean±S.D.	Group B Mean±S.D.	P-Value
Age in years	32.5±7.23	36.21±7.9	0.01
Height(m)	58.6±7.9	73.2±8.4	0.001
Weight (Kg)	1.63±0.04	1.64±0.05	0.12*
BMI(Kg/m2)	22.4±2.3	27.8±1.24	0.01
FBS (mg/dl)	94.6 ± 23.7	112.8 ± 21.2	0.001
TC (mg/dl)	148.3 ± 19.8	172.6 ± 30.2	0.001
TG (mg/dl)	70.2 ± 18.9	115.2 ± 42.2	0.001
LDL (mg/dl)	72.2 ± 8.4	105.2 ± 25.2	0.001
HDL (mg/dl)	41.5 ± 4.8	40.1 ± 6.5	0.13*
Leptin (ng/ml)	12.3±4.02	24.8±7.6	0.001

(Statistically Significant at p value <0.001; *NS: Statistically not significant)

Table-2: Demographic and biochemical data in groups (A&C):

Parameters	Group A	Group C	P-Value

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	Mean±S.D.	Mean±S.D.	
Age in years	32.5±7.23	36.92±8.02	0.01
Height(m)	58.6±7.9	89.0±11.8	0.001
Weight (Kg)	1.63±0.04	1.65±0.07	0.12*
BMI(Kg/m2)	22.4±2.3	35.1±7.6	0.01
FBS (mg/dl)	94.6 ± 23.7	119.4 ± 22.2	0.001
TC (mg/dl)	148.3 ± 19.8	184.6 ± 32.6	0.001
TG (mg/dl)	70.2 ± 18.9	122.6 ± 42.4	0.001
LDL (mg/dl)	72.2 ± 8.4	112.2 ± 25.4	0.001
HDL (mg/dl)	41.5 ± 4.8	39.2 ± 6.2	0.16*
Leptin (ng/ml)	12.3±4.02	31.1±12.3	0.001

(Statistically Significant at p value <0.001; *NS: Statistically not significant)

Table-3: Demographic and biochemical data in groups (B&C):

Parameters	Group B Mean±S.D.	Group C Mean±S.D.	P-Value
Age in years	36.21±7.9	36.92±8.02	0.24*
Height(m)	73.2±8.4	89.0±11.8	0.001
Weight (Kg)	1.64±0.05	1.65±0.07	0.12*
BMI(Kg/m2)	27.8±1.24	35.1±7.6	0.01
FBS (mg/dl)	112.8 ± 21.2	119.4 ± 22.2	0.001
TC (mg/dl)	172.6 ± 30.2	184.6 ± 32.6	0.001
TG (mg/dl)	115.2 ± 42.2	122.6 ± 42.4	0.001
LDL (mg/dl)	105.2 ± 25.2	112.2 ± 25.4	0.001
HDL (mg/dl)	40.1 ± 6.5	39.2 ± 6.2	0.14*
Leptin (ng/ml)	24.8±7.6	31.1±12.3	0.001

(Statistically Significant at p value <0.001; *NS: Statistically not significant)

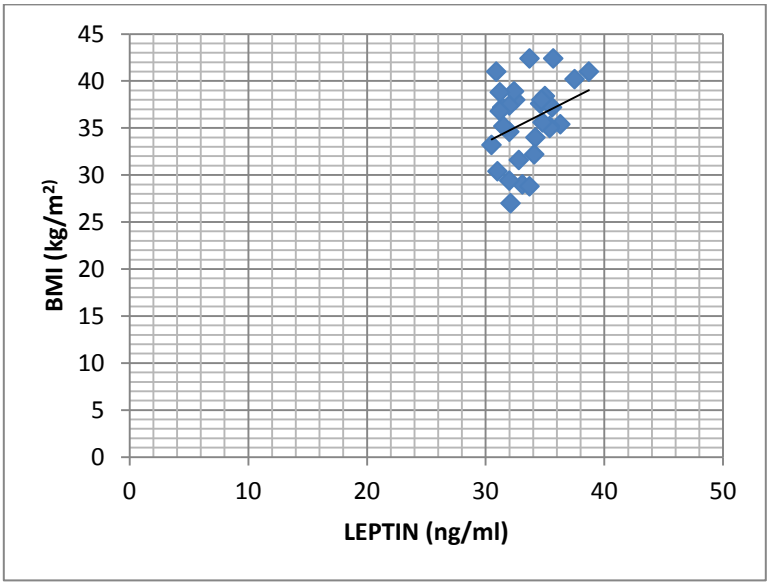

Fig.1: Shows the correlation of Leptin with BMI.

Discussion:

This present study described significantly high serum leptin levels among obese and overweight subjects as compared to the non-obese control group. Leptin belongs to a family of hormones that regulate body weight. It is released from the fat cells, and its level in blood is maintained in proportion to body fat. It acts as an auto-regulator of energy intake by acting on the hypothalamus to inhibit the biosynthesis of neuropeptide Y causing decrease in appetite. The serum Leptin level increased as BMI increases irrespective of gender; however, men showed a greater reactive response of increasing leptin secretion as their fat mass increased, while women having already higher basal levels showed a much smaller reactive response. The initial increase was marked in overweight group as compared to the normal group. Serum Leptin did not increase to the same extent between overweight and obese groups. This indicates that most of the time serum Leptin increases during transition from normal to overweight and there after slowing down while approaching to obesity. This finding is also supported by a previous study of Fried et al[7], that on average leptin release per gram of adipose tissue is 2–3 times greater in obese than in lean subjects, because fat cells are usually enlarged 2–4 times in the obese. When expressed per fat cell, Leptin secretion can be

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up to 7 times higher in obese than in lean subjects. At certain level this increase of serum leptin becomes constant, that is why the increase of serum Leptin for overweight to obese was not as high as for normal to overweight. This is explained by the fact that significant transport saturation for Leptin occurs between 10–15 ηg/ml and 20–25 ηg/ml respectively, almost full saturation exists.[8]

In this study a significant positive correlation of serum Leptin and BMI was found. As BMI increased serum Leptin also increased in all subjects among BMI groups. Despite good correlation of leptin and BMI, we observed significant variability in leptin levels in subjects with similar BMI. This may be related to differences in body composition and fat distribution. Maffie, et al. described significant heterogeneity in leptin concentration in subjects with similar BMI.[9] It is possible that subjects with appropriate leptin levels are able to keep their weight stable. Another possibility could be the differential sensitivity of individuals to leptin. Leptin resistance in some animal models of obesity is due to mutation in the receptor gene[10] or leptin resistance induced by a high fat diet.[11] Further studies are needed to explore whether genetic or acquired leptin resistance exists in man.

No correlation of leptin with fasting blood glucose levels was observed in this study, similar to diabetic patients in a previous study.[12] Leptin levels were not related to diabetic severity in African Americans and Japanese subjects.[13,14] Neither was any correlation seen in prepubertal, pubertal nor in young adult, obese and non-obese diabetics.[15]

The relationship between serum leptin and lipids remains unclear. Some studies found no significant relationship between leptin and lipids or lipoproteins[16], whereas in others lipids showed significant correlation with leptin levels.[17] In our study, a positive correlation was seen between serum leptin and total cholesterol, LDL and triglycerides. No correlation was seen with HDL-cholesterol. These observations indicate a role of leptin resistance in the pathogenesis of dyslipidemia. This needs confirmation in large cohort studies. Leptin levels are shown to predict the development of the metabolic syndrome independent of baseline obesity.[18] None of the subjects in this study had evidence of metabolic syndrome.

Conclusion:

In conclusion, our study shows that serum leptin levels correlate with clinical and biochemical parameters of overweight & obesity. Increase in serum Leptin concentration was observed with an increase in BMI. Significant difference between Leptin concentrations was found in normal, overweight, and obese subjects. This indicates the important role of Leptin in human metabolism and obesity.

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