ORIGINAL ARTICLE

BMI is Associated with Serum Leptin and Lipid Profile

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ABSTRACT

Aim: To find a possible association of serum leptin and lipid profile with obesity
Place of study: The subjects were selected from Outpatient Department of Internal Medicine, Mayo Hospital and British Sliming Center.
Methods: The consent from the patients was taken before their participation in this study. The subjects were classified into two categories; obese (BMI>30) and Non-Obese (BMI<25). The body weight of each individual dressed in light clothing was measured using a carefully calibrated weighing balance. The height of each individual was measured using a vertical-measuring rod; a waist and hip circumference was also measured to calculate waist/hip ratio. Blood samples were taken early in the morning, 12 hours postprandial. About 10 ml of venous blood was drawn from the subjects. The serum samples were stored at 2-5°C for not more than 24 hours prior to lipid profile and plasma Leptin Measurements. Human leptin ELISA kit (ENZO-ALX-850-044-KI01) was used (that is designed with sandwich enzyme immunoassay method for the quantitative measurement of human leptin). Triacylglycerol concentration was measured by using GPO-PAP method, Triglyceride LABKIT was used for this purpose. Cholesterol concentration was measured by using CHOD/PAP method, Cholesterol LABKIT was used for this purpose.
Results: The mean age of the patients was 45.35 ± 5.96 years with min and max age 32-58 years respectively. Total 91 subjects were selected. 43 (47.3%) of them were males and remaining 48 (52.7 %) were females. The results shows positive significant correlation in BMI and Serum Leptin, r=0.316 (0.002), significant correlation in BMI and total Cholesterol, r=0.0.195 (0.000), positive significant correlation in BMI and HDL Cholesterol, r=0.0.108 (0.000), no significant correlation in BMI and LDL cholesterol, r=-0.001 (0.747), no significant correlation in BMI and LDL cholesterol to HDL Cholesterol ratio, r= -.041 (0.270).
Conclusion: Based on the results of this study it can be concluded that there exists some association between lipid profile and serum leptin with Body Mass Index. Further studies must be carried out to further investigate how leptin interacts with lipid profile and vice versa
Keywords: obesity, leptin, lipid profile, Body Mass index (BMI)

INTRODUCTION

Leptin has a key role in body weight regulation by serving as an adipose satiety signal to the brain thereby influencing food intake and energy expenditure in a negative feedback loop[2]. Leptin mRNA and leptin secretion in adipocytes have been shown to be modulated by thyroid hormones[3]. Leptin exerts a number of regulatory functions; most of them are poorly understood[4]. Plasma leptin displays a strong association with cardiovascular risk factors, including obesity, insulin resistance, hypertension, dyslipidaemia, hyperuricemia, inflammatory markers[5].

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Obesity is associated with a 3-or-more-fold increase in the risk of fatal and nonfatal myocardial infarction[6]. The American Heart Association has reclassified obesity as a major, modifiable risk factor for coronary heart disease[7]. The increased prevalence of premature coronary heart disease in obesity is attributed to multiple factors[8]. A principal contributor to this serious morbidity is the alterations in plasma lipid and lipoprotein levels. The dyslipidaemia of obesity is commonly manifested as high plasma triacylglycerol levels, low high-density lipoprotein cholesterol (HDLc), and normal low-density lipoprotein cholesterol (LDLc) with preponderance of small dense LDL particles[9]. All this literature reflects that there might be a plausible relationship between plasma lipid profile and leptin. Leptin is thought to provide the central nervous system with feed-back information about fat storage of the body[10]. Thus; leptin is thought to be a part of the regulation of appetite, food intake and the lipid

metabolism[11]. It was also reported that school children with higher plasma leptin levels have significantly higher triglyceride, LDL-c and Apo protein A levels than those with relatively lower leptin levels[12]. In contrast, it was also demonstrated that a relationship between leptin concentrations and lipid profile and lipoprotein levels among hyperlipidaemic adult patients was not statistically significant[13].

Very few studies have been carried out to investigate the possible role of leptin and lipid profile in obese in non- obese population and the existing data on the relationship between lipid profile and leptin is conflicting.

METHODOLOGY

In the present study, 100 subjects were selected(50 obese and 50 non obese) from the Outpatient Department of Internal Medicine, Mayo Hospital Lahore and British Sliming Center, Lahore, Pakistan. Ethical approval was obtained from the local institution's review committee and consent was obtained from all participants. For the purposes of this study, subjects were classified into two categories; Subjects with obesity, BMI>30 and subjects without obesity, BMI<25. Subjects with diabetes mellitus, hypertension, cardiovascular disease or suffering from any malignancies, hepatitis B or any contagious disease were not included.

Analytical method: The nutritional status of all subjects was assessed by means of anthropometric measurements. The body weight of each individual dressed in light clothing was measured using a carefully calibrated weighing balance. The height of each individual was measured using a vertical-measuring rod; waist and hip circumferences were also measured to calculate waist/hip ratio. BMI was calculated as weight in kg divided by squared height (in m^2). Blood samples were taken early in the morning, 12 hours postprandial. About 10 ml of venous blood shall be drawn from the subjects. The serum samples were stored at 2-5°C for not more than 24 hours prior to serum leptin and Lipid profile (LDL, HDL, VLDL, and LDL to HDL ratio) determination.

Laboratory techniques:

1. Leptin measurement through ELISA

Human leptin ELISA kit (ENZO-ALX-850-044-KI01) was used (that is designed with sandwich enzyme immunoassay method for the quantitative measurement of human leptin).

2. Lipid profile

Triacylglycerol concentration: Triacylglycerol concentration was measured by using GPO-PAP method, Triglyceride LABKIT was used for this purpose. After the absorbance of standard and sample was measured against blank at 546nm within 60min, Triglyceride concentration was determined by putting values in the following formula: Triglyceride concentration(mg/dl)=A_{sample}/ $A_{standard}$ X Concentration of Standard (mg/dl)

Cholesterol concentration: Cholesterol concentration was measured by using CHOD/PAP method, Cholesterol LABKIT was used for this purpose. After the absorbance of standard and sample was measured against blank at 546nm within 60min, cholesterol concentration was determined by putting values in the following formula: Cholesterol concentration (mg/dl)= A_{sample} / $A_{standard}$ X Concentration of standard(mg/dl)

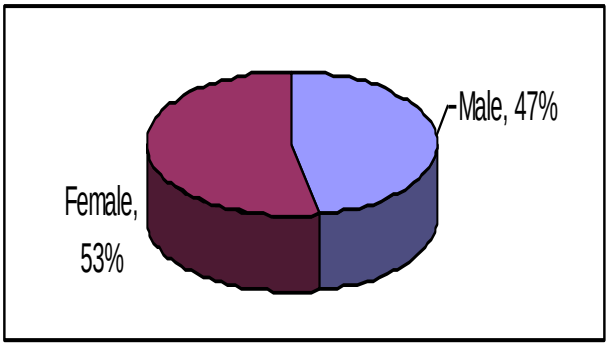
HDL-Cholesterol Concentration: HDL-Cholesterol concentration was measured by phosphotungstic-precipitation method, HDL-Cholesterol LABKIT (ref# 30188). After the absorbance of standard and sample was measured against blank at 546nm within 30min, HDL-Cholesterol concentration was determined by putting values in the following formula: HDL-cholesterol concentration (mg/dl)= ΔA X Factor

LDL-Cholesterol concentration: LDL-Cholesterol concentration was measured by Friedwald Method. To measure LDL-Cholesterol concentration; Triglyceride, cholesterol and HDL-cholesterol concentrations were measured and then putting their values in the following formula: LDL Cholesterol concentration was measured (Friedwald et al.,1972): LDL-Cholesterol (mg/dl)= Total cholesterol (mg/dl)_triglyceride/5(mg/dl)_HDL-Cholesterol(mg/dl)

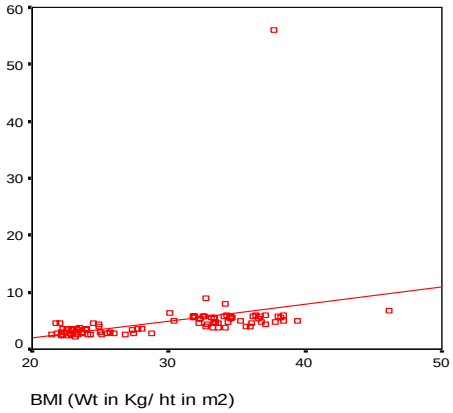
VLDL-Cholesterol concentration: VLDL concentration was measured by dividing triglyceride to 5: VLDL cholesterol (mg/dl): Triglyceride concentration (mg/dl)

Statistical analysis: Results were presented as the mean +/- standard deviation. Data was analyzed using the Statistical Package for the Social Sciences (SPSS) version 16 (SPSS, Evanston, IL, USA) for Windows. Biochemical parameters not normally distributed were analyzed after being logarithmically transformed. Students' unpaired t-test and one-way analysis of variance (ANOVA) were used to compare the results of the different groups. Simple and partial correlation coefficients between the variables were determined and multiple regression analysis was performed to determine the association between the variables of interest. Data was expressed as mean (SD) or median (range); statistical significance was set at $p < 0.05$.

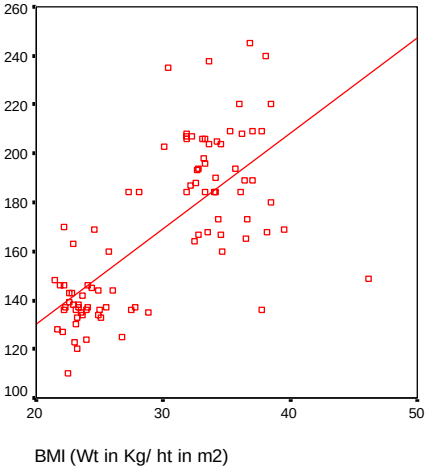
RESULTS



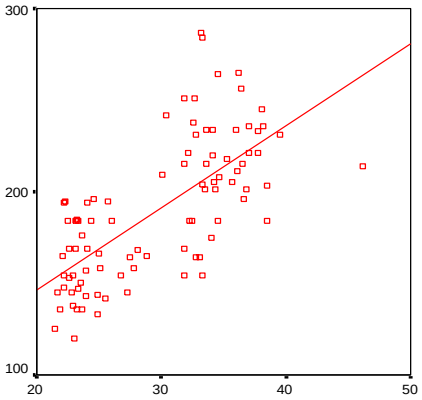
Graph 1: Male: Female Ratio in the subjects.



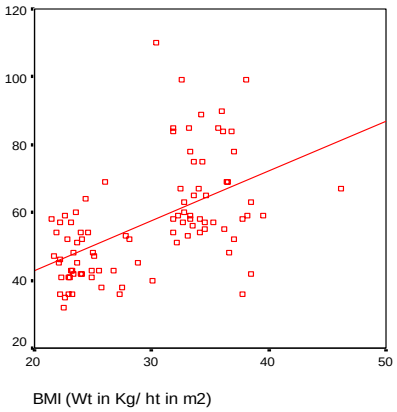
Graph 2: Comparison between Triacylglycerol (TAG) and Body Mass Index (BMI). The graph shows positive significant correlation in BMI and Serum Leptin, r=0.316 (0.002).

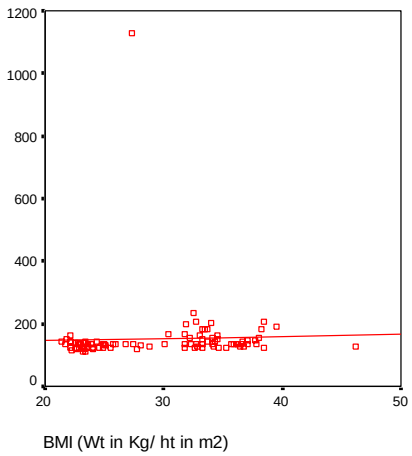


Graph 3: Comparison between Triacylglycerol (TAG) and Body Mass Index (BMI). The graph shows positive significant correlation in BMI and Serum Leptin, r=0.316 (0.002).



Graph 4: Comparison between Total Cholesterol and Body Mass Index (BMI). The graph shows positive significant correlation in BMI and total Cholesterol, r=0.0.195 (0.000)





Graph 5: Comparison between Body Mass Index (BMI) and High Density Lipoproteins (HDL). The graph shows positive significant correlation in BMI and HDL Cholesterol, r=0.0.108 (0.000)

Table 1: Statistical analysis of lipid profile

	serum leptin (ng/ml)	TG (mg/dl)	T-Chol (mg/dl)	HDL-C (mg/dl)	LDL-C(mg/dl)	LDL-C/HDL-C ratio
BMI (Wt in Kg/ ht in m2)						
Correlation	.316(**)	.703(**)	.691(**)	.531(**)	.034	-.117
p-value	.002	.000	.000	.000	.747	.270
serum leptin (ng/ml)						
Correlation		.266(*)	.195	.108	-.001	-.041
p-value		.011	.064	.310	.993	.700
TG (mg/dl)`						
Correlation			.619(**)	.631(**)	.098	-.061
p-value			.000	.000	.353	.568
T-Chol (mg/dl)						
Correlation				.531(**)	-.080	-.207(*)
p-value				.000	.453	.049
HDL-C (mg/dl)						
Correlation					-.068	-.301(**)
p-value					.524	.004
LDL-C (mg/dl)						
Correlation						.965(**)
p-value						.000

Table 2: Lipid profile in obese and non-obese patients

	Obesity	Mean	Std. Deviation	p-value
waist/hip ratio	Obese	1.076	0.059	0
	Non-obese	0.88	0.053	
serum leptin (ng/ml)	Obese	6.36	7.38	0.004
	Non-obese	3.13	0.617	
TAG (mg/dl)	Obese	193.04	23.225	0
	Non-obese	140.65	15.127	
T-Cholesterol (mg/dl)	Obese	216.08	31.249	0
	Non-obese	160.72	20.76	
HDLc (mg/dl)	Obese	66.45	16.099	0
	Non-obese	46.72	8.46	
LDLc (mg/dl)	Obese	151.35	27.18	0.893
	Non-obese	154.37	152.3	
LDLc:HDLc ratio	Obese	2.39	0.64	0.076
	Non-obese	3.54	4.3	
	Non-obese	1.97	0.264	

DISCUSSION

Although much has been learnt regarding the leptin hormone, its physiology but its precise role in the endocrine system remains to be defined. One of the difficulties inherent to these studies lies in the fact that leptin physiology seems to be rather different in humans and rodents. Not only is the circadian rhythm of its plasma levels different but also its regulation and the relationship with other physiological parameters differ. The aim of the present study was to demonstrate the association of serum leptin with serum lipid with obesity. The findings of the current studies show that there is significant positive correlation between BMI and Total Cholesterol, HDLc and TAG. However, no correlation has been found between LDLc and HDLc to LDLc ratio. The association of serum leptin concentration with serum lipids has been inconsistent and in a study conducted by Hallikainen[14], it was found that high serum leptin concentration was associated with high cholesterol synthesis and low cholesterol absorption but not with serum lipids. Leptin has been associated with atherosclerosis and has been shown to interfere with lipoprotein profiles[15].

CONCLUSION

In the present study, significantly high levels of serum leptin have been observed in obese patients. As the sample size is small in this study, a comprehensive study must be conducted to fully understand the association of BMI to lipid profile and leptin levels. Different factors influences the levels of HDL, LDL

and TAG and it is not practical to conclude that lipid profile determines the obesity of the individual, a detailed study, taking into account all the factors associated with lipid profile should be designed and conducted to elucidate the possible association of lipid profile and obesity

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