The Relationship between Serum Leptin and Blood Pressure in Obese and Non Obese Male Subjects

Hassan M. Eissa[1], Sahar M. Kamal[1]Tarek M. Ali[2], and Ahmed A T[2]

[1]Department of physiology, Faculty of Medicine, Cairo University, Egypt
[2]Department of physiology, Faculty of Medicine, Beni-Suef University, Egypt
*Correspondence author: e- mail: tarek70ali@gmail.com

ABSTRACT

Objectives: To clarify the relationship between serum leptin level and arterial blood pressure (ABP) and lipid profile in obese and non-obese male subjects. Methods: The present study was performed in Beni Suef University, Faculty of medicine between December 2007 and December 2008. Fasting serum leptin level, arterial blood pressure, lipid profile, waist circumference (WC) and body mass index (BMI) were measured in 10 lean normotensive (LN), 10 obese normotensive (ON), 10 lean hypertensive (LH) and 10 obese hypertensive males (OH). Results: In the present study, serum leptin, BMI, and WC were significantly elevated in obese subjects than in lean subjects whether normotensive or hypertensive (P < 0.001). In lean groups, serum leptin level was significantly higher in hypertensive than in normotensive persons (P < 0.001). In all studied subjects, mean ABP was significantly related to serum leptin. In obese groups, and in lean groups, serum leptin was positively correlated with mean ABP and all measured lipid profile parameters except high density lipoprotein (HDL) to which it was negatively correlated. Serum leptin was positively correlated with WC in obese normotensive group, (r =0.657) (p= 0.039), but it was positively correlated with BMI in obese hypertensive group (r =0.675) (p=.032). Conclusion: This study suggested that hyperleptinemia may play a role in the pathophysiology of hypertension independently of adiposity and the most correlated parameter to mean ABP among the measured parameters was serum leptin.
Keywords: Leptin, Hypertension, Obese. Lipid profile

INTRODUCTION

Obesity has become one of the most serious health problems in industrialized societies. Weight gain is associated with a high risk of developing cardiovascular and metabolic diseases such as coronary heart disease, hypertension, diabetes & dyslipidemia[1] Epidemiological studies have demonstrated a close relationship between body mass index (BMI) and hypertension[1]. The association between body weight and blood pressure has been found even in normotensive subjects with normal BMI [2]. Subsequently, clinical studies have demonstrated that weight loss induced by low calorie diet or gastric bypass reduces arterial blood pressure and corrects diabetes and other co morbidities associated with obesity [3].

Adipose tissue was considered exclusively as a body energy store

without other functions. However, the realizations that adipocytes can produce many hormones including leptin, resistin and adiponectin have led to a view that this tissue is an endocrine secretory organ [4].

Leptin, a peptide hormone discovered to be involved in the regulation of food intake and satiety, as well as in the control of fat accumulation[5]. Leptin (from the Greek *leptos*, meaning thin) is a protein hormone approximately ~16 kDa in mass and encoded by the obese (ob) gene [6]. Leptin depresses appetite and inhibits fat deposition, particularly in visceral depots and at least part of its effects is mediated by sympathetic nervous system activation[7]. Human obesity is associated with elevated plasma leptin levels and resistance to the metabolic effects of the hormone to the extent that high plasma leptin levels are ineffective in reducing fat accumulation[8]. In human, the strong interrelation between leptin, BMI and other measures of body fat have made it difficult to investigate the possible influence of that peptide on ABP [9]. BMI does not take into account the body fat distribution which appears to confer considerable risk for metabolic abnormalities and cardiovascular disease; hypertension is much more prevalent in persons with central compared with lower body obesity [10]. The serum concentrations of cholesterol and triglycerides are positively correlated with obesity. [11]. Leptin synthesis is induced by hyperglycemia, hyperlipidemia, and a replete fat mass and also leptin suppresses insulin production [12]. The aim of the present study is to clarify the relationship between serum leptin, blood pressure, body mass index (BMI) and lipid profile, and to explain the possible mechanisms for leptin to produce hypertension in obese males.

SUBJECTS & METHODS

Subjects:

The study was carried out in the Department of Medical Physiology, Faculty of Medicine, Beni-Suef University, Egypt. Cases and control subjects were chosen from those attending the Internal Medicine Outpatient's Clinic in Beni Suef University Hospital and healthy volunteers. They were only male subjects to avoid the multiple variable parameters related to females like adiposity, sex hormones and menopausal state. Participants agreed to participate by written consent. The protocol was based on inclusion and exclusion criteria and is approved by local ethical committee.

The study was done on 40 subjects. They were divided into 2 main groups:

Normotensive group (A) which was subdivided into:

Lean normotensive (LN) subgroup (no. =10) with BMI < 25 Kg/m²

Obese normotensive (ON) subgroup (no. =10) with BMI > 30 Kg/m²

Hypertensive group (B) which was subdivided into:

Lean hypertensive (LH) subgroup (no. =10) with BMI < 25 Kg/m²

Obese hypertensive (OH) subgroup (no. =10) with BMI > 30 Kg/m² .

Exclusion criteria

Persons with secondary hypertension and elevated blood sugar levels, patients with other endocrinal

disorder, infection and inflammation, patients with liver or renal disease and smoker subjects were excluded from the study.

Methods:

Samples collection:

A venous blood sample of 6 ml was withdrawn from each subject while fasting for at least 12 hours. Lipid profile, including serum cholesterol, triglycerides (TGs), low density lipoprotein (LDL), high density lipoprotein (HDL) and very low density lipoprotein (VLDL) were measured after 12 hours overnight fasting by enzymatic colorimetric test. Leptin hormone concentrations were analyzed using Human leptin enzymatic light immunosorbant assay (ELISA) kit from Linco Research (6 Park Drive. St. Charles, Missouri 63304 USA). The analysis of leptin was carried out by using DSX Automated ELISA System from (DYNEX Technologies .Inc.). Lipid parameters were measured by kits supplied by Point Scientific, Inc., Michigan, (U.S.A)

Anthropometric measurements:

Body Mass Index (BMI) was calculated as the weight (kg) divided by the square of the height in meters (m)[13]. Waist circumference (cm) was taken with a tape measure as the point midway between the costal margin and iliac crest in the mid-axillary line, with the subject standing after full expiration [14].

Blood pressure measurements: Blood pressure was measured by using the auscultatory & palpatory methods using a stethoscope and a sphygmomanometer.

Statistical analysis:

Analysis of data was done by IBM computer using Statistical Program for Social Science (SPSS) version 13. Unpaired student t- test was used to compare quantitative variables between two independent groups in parametric data. The normality of distribution of all measured parameters was checked by Kolmogorov – Smirnov test. Analysis of Variance (ANOVA) test was used to collectively indicate the presence of any significant difference between several groups. The test is based on comparing the variance between the groups to the variance within the groups. It is measured as the ratio between these 2 variances [15] followed by Post - hoc LSD analysis to compare various groups with each other. Results were expressed as mean ± standard deviation (SD). Linear regression analysis was used to find out the effect of different parameters on serum leptin. $P<0.05$ was considered statistically significant. Pearson correlation coefficient (r) test was used to describe the association between the different studied parameters; $P <0.05$ was considered statistically significant.

RESULTS

All subjects completed the study protocol. As shown in table 1, the 4 subgroups were compared for age, BMI, WC, blood pressure, lipid profile and serum leptin. BMI, WC and serum leptin level, were significantly elevated in obese subjects than in lean subjects whether normotensive or hypertensive ($P< 0.001$). Serum leptin and mean ABP were significantly higher in hypertensive than in normotensive

whether obese or lean subjects (P<0.001). Cholesterol, TG, LDL, and VLDL were significantly higher in the obese subjects, than in the lean only in the normotensive not in the hypertensive subjects. Also these parameters were significantly elevated in hypertensive groups than in normotensive groups whether obese or lean. Among the normotensive subjects, these parameters were higher in the obese group than in the lean group. HDL was significantly higher in the lean normotensive subjects compared to obese normotensive and the lean hypertensive subjects. (Table 1.)

Study of all groups showed that serum leptin was positively correlated with BMI, WC, blood pressure, lipid profile parameters except HDL (Table 2)

In the obese groups (ON & OH together), and lean groups (LN & LH together), serum leptin was positively correlated with mean ABP and all lipid profile parameters except HDL to which it was negatively correlated. (Table 3)

Table 4 shows that serum leptin was not significantly correlated with mean ABP either in normotensive (LN & ON together) or in hypertensive subjects (LH & OH together). (figure 1), while it was positively correlated with BMI, WC (figure 2) and LDL in both normotensive groups and hypertensive groups It was positively correlated with VLDL in normotensive groups and with Cholesterol (figure 3) and TG (figure 4) in hypertensive groups, while it was negatively correlated with HDL in both groups.

Serum leptin was positively correlated with WC in the obese normotensive group and with BMI in the obese hypertensive group. However, it was negatively correlated with HDL in obese hypertensive group. (Table 5). Using the linear regression analysis, in all studied subjects (LN, LH, ON, OH), revealed that mean ABP was significantly related to serum leptin (Table 6).

Discussion: In the present study there is a significant positive correlation between plasma leptin and body mass index (r = 0.652, p < 0.001) and waist circumference (r = 0.606, p < 0.001) were found in all studied groups. The increase in leptin level in obese persons is explained by dependency of leptin level on the increase in body adiposity. There is a positive correlation between leptin level and measures of increase adiposity (16). Serum leptin correlation with the percentage of body fat, suggests that most obese persons are insensitive to endogenous leptin production (17). Leptin acts in the hypothalamus to decrease food intake and increase thermogenesis, as well as increase sympathetic nerve activity (SNA) to nonthermogenic organs (18). Increasing evidence suggests that these actions can be dissociated in obesity, with resistance to the anorexic and thermogenic effects of leptin (mediated through the arcuate nucleus) but preservation of cardiorenal sympathoactivation (mediated through ventral and dorsal medial hypothalamic nuclei. This phenomenon might explain in part how hyperleptinemia could be accompanied by obesity (partial loss of appetite and metabolic actions of

leptin) but still contribute to sympathetic over activity and hypertension because of preservation of the sympathetic actions of leptin to some organs involved in blood pressure regulation (18). Serum leptin in our study shows a highly significant positive correlation with the mean ABP (r = 0.612, p<0.001) found in all studied groups .

The present study shows higher leptin levels in the obese hypertensive group than in the lean hypertensive group (36.57±8.47 versus 13.29±2.84 ng/ml) respectively. These results were in agreement with those reported by **Uckaya** et al.[19], who found a significantly higher leptin level in essential hypertensive patients than in controls as well as a significant correlation between leptin level and blood pressure. However, these results could not be confirmed by **Kokot** et al. [20], who found no difference in plasma leptin levels between hypertensive patients and normotensive controls.

In another study, **Hirose** et al.[21] reported a correlation between leptin levels and mean blood pressure after adjustment for age and body mass index. Although, **Suter** et al. [22], found no association between leptin level and blood pressure in hypertensive men; however they reported a positive relationship on a separate analysis for one women subgroup, as well as normotensive men. In a sample of Japanese men, **Masuo** et al. [23], revealed a BMI dependant positive relationship between leptin and blood pressure.

According to **Hu** et al. [24], study in a rural Chinese population sample, the association between blood pressure and leptin was heavily influenced by body fat mass and distribution. Leptin may contribute to end-organ damage in hypertensive individuals such as left ventricular hypertrophy, retinopathy and nephropathy, independent of regulating blood pressure [25]. **Aizawa-Abe** et al.[26], found that chronic infusion of leptin and transgenic overexpression of leptin has been shown to increase arterial blood pressure and heart rate. **Beltowski** et al. [27], have speculated that if patients with hyperleptinemia are resistant to the facilitative effects of leptin on sodium excretion, but are not resistant to the stimulatory effects of leptin on sympathetic and/or reticular activating system (RAS) activity, this would explain why hypertension occurs so often with obesity. Finally, **Esler** [28] and **Sharma** et al. [29], reported that in obesity, there is stimulation of sympathetic outflow to the kidneys, evident in increased rate of spillover of noradrenaline into the renal veins and to skeletal muscles vasculature. Also, the high renal sympathetic tone contributes to hypertension development by stimulating renin secretion and through promoting renal tubular reabsorption of sodium. In our study there is a significant positive correlation between leptin and plasma cholesterol (r = 0.635, p < 0.001), triglycerides (r = 0.581, p < 0.001) and LDL (r = 0.705, p < 0.001),and VLDL (r = 0.497, p < 0.05), while there is a significant negative correlation between leptin & HDL (r = - 0.652, p < 0.05) in all studied groups. 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 [30]. **Palou** et al, [31] showed that in all age groups, HDL levels were significantly lower in patients who had a high BMI. Hypertriglyceridemia is often associated with reduced levels of HDL suggesting a possible metabolic interaction between these two lipid fractions [32]. The key to this relation may be that the increase in fat deposition in obese individuals is associated with insulin resistance [33], 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 [34]. The significant association between obesity, BMI and lipid profile in our results is consistent with what was reported by **Turki** et al [35] who found in their study, that serum leptin correlates positively and strongly with BMI (r=0.765,P<0.01), and also correlates positively with both total cholesterol (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). 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 [36]. One of the major mechanisms leading to the development of obesity-induced hypertension appears to be leptin-mediated sympathoactivation. Leptin adversely shifts the renal pressure natriuresis curve, leading to relative sodium retention. Although obesity is generally associated with resistance to the anorexic and weight-reducing actions of leptin there is preservation of its sympathoexcitatory and pressor actions. This selective leptin resistance of obesity, coupled with hyperleptinemia, may play a critical role in the cardiovascular complications of obesity. Increased information about leptin and its mechanisms of actions should help the development of safe and effective pharmacological treatments of obesity and obesity-related hypertension [37]

Conclusions: From the present results, it was obvious that leptin levels were increased with the increase of the mean ABP in both obese and lean hypertensive patients. Serum leptin, being very high in the obese hypertensive group compared to lean hypertensive group; this may open a new era of the possible effect of hyperleptinemia in the obese hypertensive patients. This is through its relation to the metabolic syndrome being a part of its component or by its influence on the body fat mass, reticular activating system (RAS) and sympathetic nervous system which affect blood pressure in obese patients. There is elevated serum leptin level in obese subjects 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 and hyperleptinemia is closely associated with several risk factors related to obesity syndrome.

Table (1): Base line age, body mass index (BMI), waist circumference (WC) mean arterial blood pressure (MBP), lipid profile & serum leptin data for lean normotensive, obese normotensive, lean hypertensive, and obese hypertensive subjects

Parameters	Lean Normotensive LN)(	Obese Normotensive (ON)	Lean Hypertensive (LH)	Obese Hypertensiv (OH)
AGE (year)	28.20 ± 1.98	39 ± 9.21^{a}	49.80 ± 8.18^{b}	53.30 ± 6.29^{c}
BMI (kg/m^2)	23.88 ± 0.80	39.40 ± 6.19^{a}	23.49 ± 0.92	38.58 ± 6.47^{d}
WC (cm)	90.50 ± 3.29	121.10 ± 13.20^{a}	95.30 ± 4.32	119.60 ± 13.90^{d}
MBP (mmHg)	88.47 ± 5.28	90.13 ± 5.18	123.64 ± 6.40^{b}	126.12 ± 7.60^{c}
CHOL (mg/dl)	185 ± 11	203.50 ± 7.90^{a}	238.40 ± 18.60^{b}	246 ± 16.60^{c}
TG (mg/dl)	107.30 ± 11.90	169.60 ± 16.40^{a}	210.40 ± 12.90^{b}	206.10 ± 21.40^{c}
HDL (mg/dl)	52.90 ± 6.35	41 ± 4.05^{a}	39.30 ± 3.80^{b}	36.80 ± 4.71
LDL (mg/dl)	102.30 ± 10	122.10 ± 4.70^{a}	146.80 ± 9.40^{b}	156.70 ± 7.90^{c}
VLDL (mg/dl)	29.80 ± 6.44	40.40 ± 9.57^{a}	52.30 ± 12.10^{b}	52.50 ± 13.10^{c}
LEPTIN (ng/ml)	5.94 ± 1.37	14.91 ± 3.03^{a}	13.29 ± 2.84^{b}	$36.57 \pm 8.47^{c\ d}$

[a]**P** Shows significance between normotensive groups (Lean & obese).

[b]**P**: Shows significance between lean groups (normotensive & hypertensive subjects).

[c]**P**. Shows significance between obese groups (normotensive & hypertensive subjects).

[d]**P**: Shows significance between hypertensive groups (obese & lean subjects).

Table (2): Correlation between serum leptin & all parameters in all groups (no.=40).

Parameters	R	P
AGE (year)	0.547	0.080
BMI (kg/m^2)	0.652	<0.001
WC (cm)	0.606	<0.001
MSBP (mmHg)	0.612	<0.001
CHOL (mg/dl)	0.635	<0.001
TG (mg/dl)	0.581	<0.001
HDL (mg/dl)	-0.623	<0.007
LDL (mg/dl)	0.705	<0.001
VLDL (mg/dl)	0.497	0.002

Table (3): Correlation between serum leptin & all parameters in Lean groups (LN+LH) (no.=20) & Obese groups (ON+OH) (no.=20).

	lean groups		Obese groups	
Parameters	r	P	r	P
AGE (year)	0.719	<0.001	0.502	0.224
BMI (kg/m²)	0.032-	0.893	0.231	0.327
WC (cm)	0.670	0.101	0.100	0.676
MSBP (mmHg)	0.706	<0.001	0.867	<0.001
CHOL (mg/dl)	0.823	<0.001	0.761	<0.001
TG (mg/dl)	0.838	<0.001	0.648	0.002
HDL (mg/dl)	0.745-	<0.001	-0.575	0.008
LDL (mg/dl)	0.878	<0.001	0.805	<0.001
VLDL (mg/dl)	0.700	<0.001	0.530	0.016

Table (4): Correlation between serum leptin & all parameters in normotensive groups (LN+ON) (no.=20) & Hypertensive groups (LH+OH) (no.=20).

Parameters	Normotensive groups		Hypertensive groups	
	r	P	r	P
AGE (year)	0.662	<0.001	0.016	0.945
BMI (kg/m²)	0.900	<0.001	0.920	<0.001
WC (cm)	0.907	<0.001	0.768	<0.001
MSBP (mmHg)	0.246	0.296	0.200	0.398
CHOL (mg/dl)	0.228	0.334	0.679	<0.001
TG (mg/dl)	-.0.082	0.731	0.823	<0.001
HDL (mg/dl)	0.655-	0.002	0.500-	0.025
LDL (mg/dl)	0.675	0.031	0.509	0.022
VLDL (mg/dl)	0.596	0.006	0.095	0.689

Table (5): Correlation between serum leptin levels with all parameters in different subgroups**.**

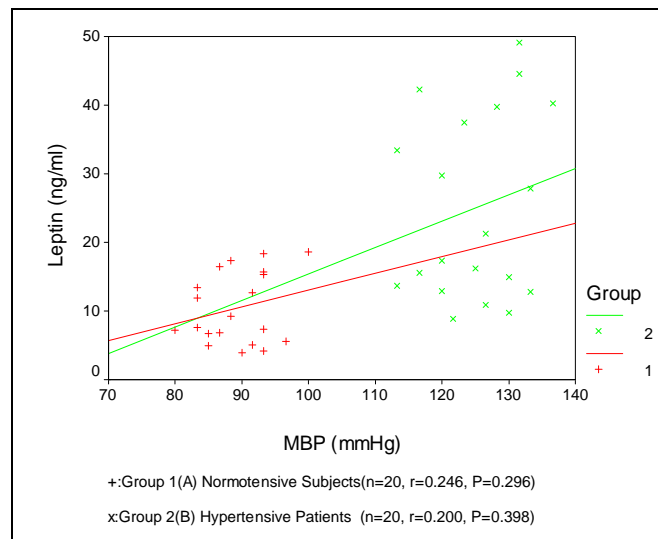| Parameters	Control "Normotensive" group				"Hypertensive" group			
	LN		ON		LH		OH	
	r	P	r	P	r	P	r	P
AGE (year)	0.436-	0.208	0.309	0.385	0.189-	0.601	0.703-	0.123
BMI (kg/m²)	0.196-	0.587	0.595	0.069	0.593	0.071	0.675	0.032*
WC (cm)	0.254	0.0479	0.657	*0.039	0.554	0.097	0.230	0.522
MSBP (mmHg)	0.457-	0.184	0.560	0.092	0.276-	0.441	0.195	0.589
CHOL (mg/dl)	0.103	0.779	0.191	0.597	0.309	0.384	0.006-	0.986
TG (mg/dl)	0.325-	0.360	0.123	0.736	0.054	0.882	0.073	0.841
HDL (mg/dl)	0.049	0.893	0.160	0.659	0.369-	0.294	0.637-	*0.048
LDL (mg/dl)	0.071	0.845	0.462-	0.178	0.621	0.055	0.033-	0.927
VLDL (mg/dl)	0.019	0.959	0.320	0.367	0.112	0.757	0.242	0.501

Table (6): Linear regression analysis showing the effects of different parameters on mean ABP.

	Model	Sum of Squares	df	Mean Square	F	Sig.
1	Regression	12348.252	10	1234.825	20.492	.000[a]
	Residual	1747.544	29	60.260		
	Total	14095.796	39			

a. Predictors: (Constant), LEPTIN, VLDL, HEIGHT, AGE, WEIGHT, HDL, TG, WC, LDL, BMI

b. Dependent Variable: MBP

	Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	S.E.	Beta		
1	(Constant)	-390.522	186.116		-2.323	.027
	AGE	.505	.192	.319	2.632	.013
	BMI	4.964	1.976	2.321	2.512	.018
	WC	.416	.182	.372	2.283	.030
	TG	3.567E-02	.063	.084	0.562	.578
	HDL	.191	.291	0.78	.654	.518
	LDL	.235	.138	.283	1.703	.099
	VLDL	.167	.136	.123	1.231	.228
	LEPTIN	.708	.200	.463	3.531	.001

+:Group 1(A) Normotensive Subjects(n=20, r=0.246, P=0.296)

x:Group 2(B) Hypertensive Patients (n=20, r=0.200, P=0.398)

Fig.(1): Correlation between serum leptin and mean Arterial Blood Pressure (mean ABP) in A & B groups.
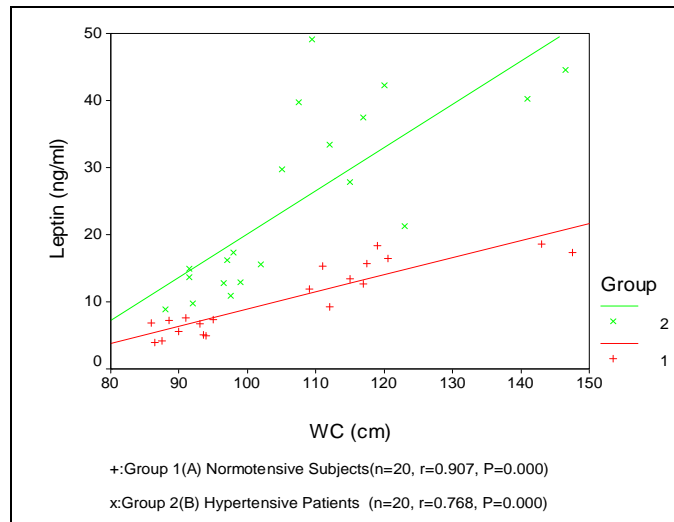
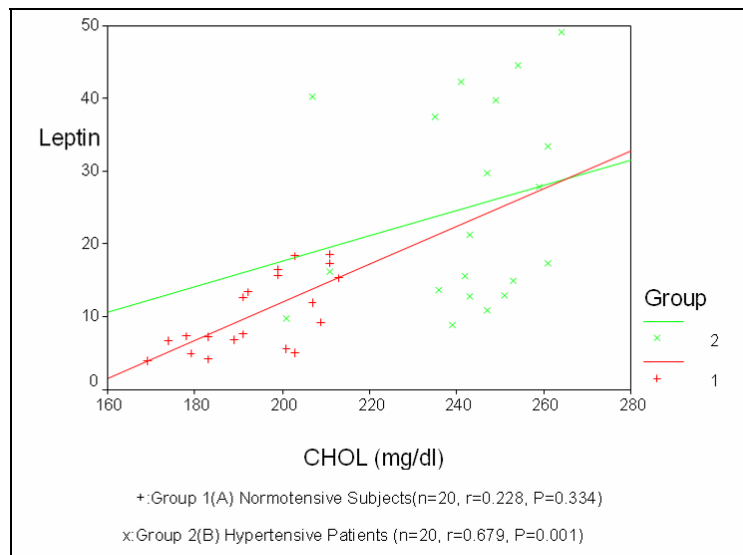**Fig.(2): Correlation between serum leptin and W.C. in A & B groups**

Fig.(3): Correlation between serum leptin and total cholesterol in A & B groups
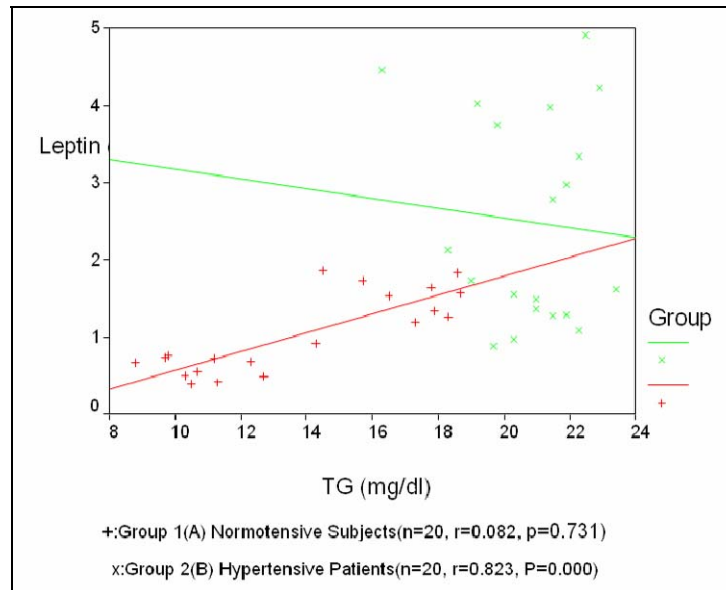
Fig.(4): Correlation between serum leptin and TG$_s$ in A & B groups

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العلاقة بين مستوى هرمون الليبتن في الدم و ضغط الدم في الذكور البدينة والغير بدينة

حسن محمد عيسي (١) *ـ سحر محمد كمال* (١) *ـ طارق محمد علي* (٢) *ـ احمد عبد التواب* (٢)

(1) كلية الطب – جامعة القاهرة   ـ (٢) كلية الطب – جامعة بنى سويف

الهدف من البحث: تم تصميم هذه الدراسة بهدف توضيح العلاقة بين مستوى الليبتين في الدم وارتفاع ضغط الدم و مستوى الدهون فى الدم في الذكور البدناء و غير البدناء وكشف الآليات الممكنة لهرمون الليبتين فى رفع ضغط الدم في الذكور البدناء.

طرق البحث: وقد أجريت هذه الدراسة في جامعة بني سويف، كلية الطب بين ديسمبر ٢٠٠٧ وديسمبر ٢٠٠٨. تم قياس مستوى هرمون الليبتين الصيامي فى مصل الدم ، مستوى الدهون فى الدم ومحيط الخصر ومؤشر كتلة الجسم في ١٠ من الذكور النحفاء ذوى ضغط الدم الطبيعى ، و ١٠ من الذكور البدناء ذوى ضغط الدم الطبيعى ، و ١٠ من الذكور النحفاء ذوى ضغط الدم المرتفع و ١٠ من الذكور البدناء ذوى ضغط الدم المرتفع.

النتائج : وأظهرت هذه الدراسة ارتفاعا ذا دلالة احصائية فى مؤشر كتلة الجسم، ومحيط الخصر ومستوى الليبتين في الدم، في مجموعات الذكور البدناء سواء مضبوطى او مرتفعى ضغط الدم مقارنة مع المجموعات النحفاء سواء مضبوطى او مرتفعى ضغط الدم والتي كانت قابلة للمقارنة. وكان مستوى الليبتين في المصل في مجموعات النحفاء مرتفعى ضغط الدم أعلى بكثير مما كان عليه في الاشخاص النحفاء مضبوطى ضغط الدم واظهر تحليل الانحدار الخطي، في جميع مجموعات الدراسة ضغط الدم متعلق بشكل كبير مع مستوى الليبتين في المصل. السمنة في مجموعات ، وفى مجموعتى النحفاء ومجموعتى البدناء قد ارتبط مستوى الليبتين في المصل ايجابيا مع ضغط الدم وجميع قياسات الدهون إلا البروتين الدهني مرتفع الكثافة الذي يرتبط سلبا معه. وفي الذكور البدناء مضبوطى ضغط الدم ، كان هناك ارتباط كبير بين مستوى الليبتين و محيط الخصر . بينما في مجموعة الذكور البدناء ذوى ضغط الدم المرتفع ، فقد ارتبط مستوى الليبتين بشكل إيجابي مع مؤشر كتلة الجسم

الخلاصة : من النتائج الحالية، اقترحت هذه الدراسة ان ارتفاع مستوى الليبتين قد يلعب دورا في الفيزيولوجيا المرضية لارتفاع ضغط الدم بشكل مستقل عن السمنة و ان المعلمة الأكثر ارتباطا لمتوسط ضغط الدم من بين المعلمات كان مستوى الليبتين فى المصل.