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ORIGINAL ARTICLE

THE CORRELATION BETWEEN APPETITE HORMONE AND LIPID PROFILE IN OVERWEIGHT MALES WITH HYPERTENSION

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ABSTRACT

Background: Leptin and adiponectin are hormones that regulate food intake, energy expenditure, and fat metabolism. Elevated levels of leptin and decreased levels of adiponectin have been associated with adverse cardiovascular outcomes. This research study examines the intricate relationship between hunger hormones and lipid profiles in overweight adults with hypertension.

Materials & Methods: Sample size was 120 patients, ranging in age from 30 to 60 years. Of these patients, 60 were overweight with hypertension, while the other 60 were normal weight hypertensive males. The blood levels of appetite hormones (leptin, adiponectin, and the leptin/ adiponectin ratio) were evaluated and analyzed for their correlation with lipid profiles, including total cholesterol, low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides.

Results: The research findings clarify the potential associations between hunger hormones, lipid profiles, and hypertension in overweight individuals. The patient exhibited an elevation in leptin levels and a reduction in adiponectin levels. Additionally, there was a negative association between BMI and adiponectin, and a positive correlation between BMI and leptin, as well as the leptin/adiponectin ratio and lipid profile. Furthermore, there was an inverse link between leptin and adiponectin.

Conclusions: Overweight individuals with hypertension had elevated levels of leptin, triglycerides (TG), cholesterol, very low-density lipoprotein (VLDL), and low-density lipoprotein (LDL). There was a positive correlation between leptin and TG, cholesterol, VLDL and LDL whereas there was a negative correlation between adiponectin and the same variables.

KEY WORDS: Appetite hormone; Lipid profile; Obesity; Hypertension.

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INTRODUCTION

The obesity poses a great danger for many diseases that are result of obesity, and the most important diseases are cardiovascular diseases (CVD), high blood pressure, and type 2 diabetes, as obesity has become a debilitating health epidemic all over the world. Almost 70% of primary high blood pressure cases are linked to obesity[1] due to the large number of studies concerned with obesity in females, as there is prevalence of obesity in females more than in males, for this reason the study addressed the effect of overweight in males.[2] Hypertension is a heritable condition that strongly predicts the occurrence of cardiovascular disease events and death[3], it is important to explain the mechanism of action that lead to obesity-related hypertension. Obesity does not directly cause an increase in blood pressure. However, it is necessary for the presence of leptin and adiponectin in order for obesity-related increases in blood pressure to occur. Leptin, the first identified adipokine (a cytokine derived from fat), plays a significant role in the development of various chronic diseases, including blood pressure

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issues.[4] Adiponectin, referred to as adipocyte complement-related protein of 30 kDa, is an adipokine that is synthesized and released by white and brown adipose tissue.[5]

The link between the leptin pathway and heightened obesity in males, as well as hypertension, occurs by directly stimulating the production of leptin in subcutaneous adipose cells. This, in turn, triggers sympathetic activation through the activation of leptin receptors in the hypothalamus, ultimately resulting in elevated blood pressure.[6] Leptin receptors are present in several areas of the hypothalamus in the brain, indicating that in men, this communication channel plays a crucial role in leptin-induced hypertension.[7] While the pathway in females is through the activation of leptin production from the hypothalamus gland, and this directly affects the pathway of producing the hormone aldosterone from adrenal gland and causes disturbances in the electrolyte balance, which causes high blood pressure[8], as shown in Figure 1.

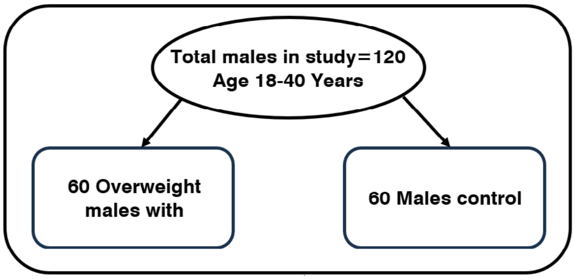
Most obese individuals have much greater leptin levels than normal individuals , and they often exhibit leptin resistance due to the disruption of several hypothalamic areas and altered melanocortin signaling caused by high - fat diets. The metabolic and energy balance disorder obesity ,hyperlipidemia, insulin resistance and hyperglycemia are associated with low leptin level.[10,11] An essential source of energy for the body, fatty acid oxidation is primarily regulated by leptin and adiponectin in the liver and muscles.[12] The study aims to assess the correlation between leptin and adiponectin with lipid profile in hypertensive males.

MATERIALS AND METHODS

Study design: Case control study.

Sample collection: The study samples were collected from patients who attended private clinics in the Babylon province. A total of 120 males, aged between 18 and 40 years, were selected using purposive sampling technique. This sample size was determined based on a statistical power analysis, which indicated that 120 participants would be sufficient to detect meaningful differences between groups with a power of 0.80 and an alpha level of 0.05 the study involved 120 males, the age ranged between (18-40) years old which were divided into two groups.

a. 60 overweight males(body mas index between 25-30) with hypertension (more than 140/90 mmHg) without other causes for hypertension and without being on treatment.
b. 60 normal weight hypertensive males and without being on treatment.



Scheme(2-1) Design of sample groups

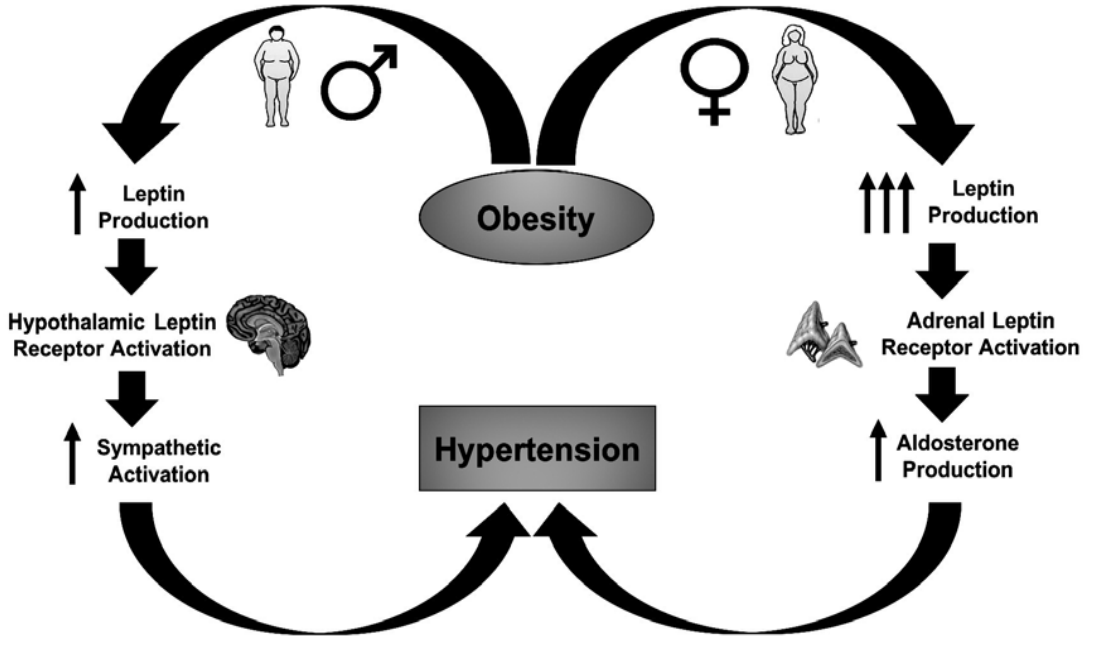


Figure 1: Leptin pathway in males and females to regulation blood pressure[9].

Sampling: Blood was collected from both, the cases and control group. Blood separated by centrifuge for 10 min at 3000 Xg to get serum for leptin, adiponectin detection and lipid profile tests.

Biochemical detection: Leptin and adiponectin were measured by enzyme-linked immunosorbent assay (ELISA) kit, while the lipid profile (triglyceride, cholesterol, HDL, LDL and VLDL) was measured by spectrophotometer in wave length 520nm.

Statistical analyses: All continous variables were summarised as mean and SD. Processed data of this study then analyzed by using SPSS program V23, the data was normally distributed, parametric test applied to compare difference between patient and control values by using independent sample ***t***-test. The Pearson's correlation analysis was done to find correlation between parameters of this study, $P < 0.05$ indicated statistical significance.

RESULTS

Baseline characteristic of overweight males with hypertension and control groups: The comparison between the patient and control groups is shown in Table 1. There were no significant variations in age between the two groups (P-value =0.22). Additionally, highly significant in BMI between groups, the control group had a lower BMI than the patients (P- value <0.00).

Hormonal variables: The result of comparison between the two groups is listed in Table 2, leptin level 5.22 and leptin / adiponectin ratio 0.07 in patients group were higher than those in control group 3.88, 0.03 respectively , while the opposite of this was observed when measured the level of adiponectin 81.5 in patients and 108.7 in control.

Lipid profile tests: Observed the result of lipid profile in Table 3, showed the levels of TG, CHO,LDL and VLDL were higher in patient when compared with control.

Correlation analysis: The correlation between variables in the overweight males with hypertension is summered in Table 4, BMI was correlated negatively with adiponectin and positively with leptin, leptin /adiponectin ratio and lipid profile, leptin and adiponectin had an inverse correlation.

Table 1: baseline characteristic of patient and control groups

Variable	Study groups	No.	Means ±SD	P- value
Age (years)	Patient	60	32.17 ±2.2	0.22
	Control	60	31.6 ±2.6	
BMI Kg/m²	Patient	60	28.09 ±1.4	<0.00
	Control	60	23.5 ±1.12	

Significant P-value ≤0.05, non-significant P-value >0.05,
BMI (body mass index) ,SD(Standard deviation).

Table 2: Hormonal variables between patient and control groups

Variable	Study groups	No.	Means ±SD	P- value
Leptin (μg/L)	Patient	60	5.22 ±1.1	<0.001
	Control	60	3.88 **±1.1**	
Adiponectin (μg/L)	Patient	60	81.53 ±11.5	<0.001
	Control	60	108.7 ±11.6	
Leptin/Adiponectin Ratio	Patient	60	0.07 ±0.024	<0.001
	Control	60	0.03 ±0.009	

Significant P-value ≤0.05, non-significant P-value >0.05,
SD(Standard deviation).

Table 3 : Lipid profile tests between patient and control groups

Variable	Study groups	No.	Means ±SD	P- value
TG (mg/dl)	Patient	60	291.48 ±45.3	<0.001
	Control	60	130.03 ±14.8	
CHO (mg/dl)	Patient	60	215.5 ±14.3	<0.001
	Control	60	166.8 ±14.4	
HDL (mg/dl)	Patient	60	16.7 ±2.0	<0.001
	Control	60	30.0 ±1.01	
LDL (mg/dl)	Patient	60	140.4 ±8.1	<0.001
	Control	60	106.12 ±7.7	
VLDL (mg/dl)	Patient	60	58.3 ±9.8	<0.001
	Control	60	26.6 ±1.05	

Significant P-value 0.05 was, P-value >0.05 was not significant, SD(Standard deviation), TG(triglyceride), CHO(cholesterol), HDL(high density lipoprotein), LDL(low density lipoprotein), VLDL(very low density lipoprotein).

Table 4 : Correlation between variables within patient

		Leptin	ADP	Lep/ADP Ratio	CHO	TG	LDL	HDL	VLDL
BMI	r	0.97	-0.91	0.95	0.97	0.86	0.75	0.5	0.86
	Sig.	.000	<0.01	<0.01	<0.01	<0.01	<0.01	>0.05	<0.01
Leptin	r	1	-0.92	0.95	0.97	0.85	0.76	0.06	0.85
	Sig.		<0.01	<0.01	<0.01	<0.01	<0.01	>0.05	<0.01
ADP	r	-0.92	1	-0.96	-0.91	-0.80	-0.73	0.36	-0.80
	sig	<0.01		<0.01	<0.001	<0.001	<0.001	>0.05	<0.001
Lep/ADP Ratio	r	0.95	-0.96	1	0.95	0.81	0.79	0.16	0.80
	Sig.	<0.01	<0.01		<0.01	<0.01	<0.01	>0.05	<0.01

Correlation is significant at the 0.01 level,

TG(triglyceride), VLDL(very low density lipoprotein), Lep/ADP Ratio (leptin /adiponectin Ratio), r(Pearson Correlation).

DISCUSSION

This study included adult overweight males (BMI more than 25 and less than 30) with hypertension without other causes for higher blood pressure and without hypertension treatment.

The first part of this study measured leptin, adiponectin levels, leptin/ adiponectin ratio and lipid profile in serum, when compared the result between patient and control, it was observed level of leptin and lipid profile were higher in patients as compared to controls, but lower level of adiponectin was found in patients. This result agreed with (Kwaifa et al., 2020) and (Y. Li et al., 2023)[4,13] while some studies (S. Yosaee, 2019;), (S. Ahl, 2015) showed elevated adiponectin concentration.[14,15] Adipocytes were formerly regarded as endocrine cells responsible for producing and releasing adipokines, which have an endocrine/paracrine function that is generally proportionate to the amount of body fat. Adipocytes secrete a greater amount of leptin per unit of adipose tissue mass.[16] The subcutaneous adipose tissue is a more effective organ in manufacturing leptin compared to visceral adipose tissue. It is not obesity itself that raises blood pressure, but rather the presence of leptin is necessary for the rise in blood pressure associated with obesity.[17]

The administration of leptin throughout the body also affects the breakdown of fats in different tissues. Leptin stimulates the process of lipolysis in white fat cells, perhaps via the activation of hormone-sensitive lipase (HSL) and nitric oxide (NO) synthase.[18,19]

The second phase of this research was to assess the association between variables among patients. It was shown that leptin strongly correlated with BMI, agreed with the finding of previous study (R. Lengton, 2024), (P.-C. Chen, 2009) which confirmed that higher BMI is strongly associated with higher leptin concentrations and reduced the adiponectin concentration[20,21], in contrast with study of (J Przybyciński, 2020).[22] Additionally, alteration of leptin or adiponectin concentration direct modulated to the lipid profile, with the exception of HDL and adiponectin levels, showed a negative correlation. Adiponectin stimulates the release of apolipoprotein A-I (apo-AI)[23,24], the primary apolipoprotein found in HDL. It also increases the expression of ATP-binding cassette transporter A1 (A-BCA1), which stimulates the formation of HDL by facilitating the transfer of cholesterol out of liver cells via reverse cholesterol transport.[25] The modulation of lipoprotein lipase (LPL) activity by circulating adiponectin is inversely co-related with total cholesterol (TG), very low-density lipoprotein (VLDL), and LDL. Additionally, leptin was directly correlated with TG, cholesterol, VLDL, and LDL, which led to enhanced TG catabolism[26], however, if they move cholesterol from the locations where it is produced in the liver or from where it is absorbed in the walls of the alimentary canal to the blood vessels and circulatory system, the rates of the values of VLDL and LDL, which are thought to be the carriers of bad cholesterol, increased[27], This is made worse by the accumulation of this cholesterol on the arterial walls and may raise blood pressure.[28]

CONCLUSION

Overweight patients with hypertension had high level of leptin, TG, cholesterol, VLDL and LDL, the two adipokines were associated with lipid metabolism parameters, Leptin positively correlates with TG, cholesterol, VLDL, and LDL, while adiponectin negatively correlates with the same variables, Obesity-related metabolic problems may be brought on by a decrease in ADP levels and/or a rise in LEP levels. Consequently, it is imperative to examine the correlation between ADP and LEP and various obesity phenotypes.

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CONFLICT OF INTEREST
Authors declare no conflict of interest.
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AUTHORS' CONTRIBUTION

The following authors have made substantial contributions to the manuscript as under:

Conception or Design:	NJTAM, SSJ
Acquisition, Analysis or Interpretation of Data:	NJTAM, SSJ, DAK, HAM, MAA, LA
Manuscript Writing & Approval:	NJTAM, SSJ, DAK, HAM, MAA, LA

All the authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.