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Assessment of Selected Physiological and Immunological Parameters in a Sample of Obese Patients in Samarra City

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Abstract. The present study was conducted to assess the hormone's levels (nesfatin-1, leptin, resistin and adiponectin) on some of inflammatory cytokines or the so-called adipokines which exert a variety of biological activities (tumor necrosis factor alpha: TNF-α and interleukin-6 IL6), as well as BMI & lipid profile in serum of obese women in Samarra 10. The present study had included (50) samples, and they were divided into: 25 samples of obese women age ranged between (25-45) years, and (25) samples for the control group that constituted from women with normal weight. The results of the study demonstrated a marked elevation in the levels of leptin and resistin hormonal levels, inflammatory cytokines, i.e., tumor necrosis factor-α (TNF-α) interleukin -6 (IL-6), lipid profile evaluated by body mass index) as well as significant decline in nesfatin-1 and adiponectin at the significance level represented by mean± SD: (P≤0.05) among obese women compared with control group.

Highlights:

1. Obese women showed significantly higher serum levels of leptin, resistin, TNF-α, IL-6, and lipid profile abnormalities compared to controls.
2. Nesfatin-1 and adiponectin levels were markedly reduced in obese patients, indicating impaired appetite regulation and anti-inflammatory response.
3. Findings confirm obesity as a state of chronic low-grade inflammation, linking it to increased risk of diabetes and cardiovascular disease.

Keywords: Obesity, lipid profile, Leptin, Nesfatin Resistin, Adiponectin

Introduction

Obesity is a common disease and a leading cause of death worldwide. Obesity affects both men and women of all ages, but there are several issues that particularly impact women's health, especially since obesity is more prevalent among

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women than men. Obesity is a condition of excess or excessive adipose tissue mass. It is also referred to as excess body weight, with the notion that bodies without excess adipose tissue and high muscle mass are also overweight [1][2]. In most countries, the concept of obesity has been associated with a pathological condition requiring healthcare and a social phenomenon resulting from over nutrition [3].

Limiting the assessment of this phenomenon to nutritional status is a weak scientific explanation, as feeding mechanisms (feelings of hunger and satiety) are subject to very high hormonal coordination [4]. And under the control of the central nervous system [5], since in many abnormal cases obesity is not related to the process of nutrition only, but rather to various reasons that may be pathological or due to the use of medical drugs [6], or related to the psychological state and social prosperity, since it is considered one of the complex diseases that occur due to the interaction of genetic and environmental factors [7].

Adipose tissue is described as an endocrine gland in addition to being an energy storage organ. It secretes hormones such as leptin, resistin, and adiponectin, as well as many cytokines known as adipokines with a broad biological effect, including tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) [8].

Nesfatin-1 (NESF-1) is a neuropeptide found in the central nervous system and peripheral tissues. It was first discovered in the hypothalamus, a region of the brain vital for regulating hunger and satiety. It is closely associated with anxiety, regulates glucose levels, and is a negative modulator of food intake. It is a gastrointestinal hormone released approximately 15–30 minutes after eating. It stimulates gastric acid secretion, slows gastric emptying, and reduces hunger and food intake in obese individuals. It is involved in the regulation of energy, appetite, metabolic, and neurological functions. It is one of the most studied and biologically active peptides and plays an important role in regulating appetite and food intake, as it is considered a powerful appetite suppressant [9].

As one of the most significant hormones produced from fat cells, leptin is a protein that is vital in controlling the body's energy levels and weight [10].

Being resistant a group of proteins that are rich in cysteines and released by adipose tissue. Because it is thought to be linked to the emergence of insulin resistance (IR), it was given the term resistin. The differentiation of fat cells and the accumulation of fat are regulated by its secretion. It is secreted in greater amounts by fat people than by lean people. Non-alcoholic fatty liver disease and body mass index (BMI) are linked to its levels. The hormone has clinical significance since serum resistin levels are linked to an increased incidence of atherosclerosis (ASCVD), establishing a connection between obesity and the onset of cardiovascular disease in obese people [11].

Adiponectin is a fat-cell-derived protein that directly affects vascular function and possesses anti-inflammatory and anti-atherosclerotic qualities. People with coronary artery disease, diabetes, and obesity have lower amounts of circulating adiponectin. [12][13].

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Materials and Methods

Body mass index (BMI) was calculated by dividing weight in kilograms by height in meters squared (kg/m²) using a tape measure and a weighing scale, according to the method described above. Body mass was calculated according to the following equation:

Body mass = weight in kilograms / (height in meters)2 [14].

The samples were distributed within a BMI weight category (34.9-30, 39.9-). The number of patients was 25, in addition to a control group whose BMI was (24.9-18), also consisting of 25 person.

Biochemical Tests

Estimating serum levels of nesfatin-1, leptin, resistin, and adiponectin:

A ready-made ELISA kit based on the double sandwich antibody technique was used, according to the kit instructions, manufactured by BT LAB, China.

Immunological Tests

Estimating the concentrations of mediator-1 and tumor necrosis factor-α

The concentrations of mediator-1 and tumor necrosis factor-α were measured using the ELISA method, and their baselines were similar according to the kit instructions, manufactured by SUNLONG, China.

Statistical Analysis:

The arithmetic means of the coefficients were compared using the t-test using the Minitab statistical software [15].

Result:

Table (1) shows the lipid profile criteria in obese individuals compared to the control group

Parameters / Groups	TC mg/dl	TG mg/dl	LDL mg/dl	HDL mg/dl	vLDL mg/dl
Control	130-197	95-142	66-113	40-51*	19-28.4
Patients with obesity	178-233**	162-238**	160-189**	30-36	32-47.6**

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Table (2) shows the body mass index and the levels of the hormones nesfatin-1 and

Parameters / Groups	(BMI) kg/m^2	Nesfatin-1 ng/ml	Leptin ng/l
Control	18-24.9±0.015	18.80±1.6*	6.155 ± 0.836
Patients with obesity	34.130±4.261 ***	12.92±0.43	1.780±8.033 *

leptin in obese individuals compared to the control group.

Table (3) shows the level of Resistin and Adiponectin hormones in obese individuals compared to the control group.

Parameters / Groups	Resistin ng/l	Adiponectin ng/l
Control	178.896 ± 35.246	9.1 ± 2.5 **
Patients with obesity	236 .732 ± 32.066***	1.3 ± 5.1

Parameters / Groups	(TNF- α) ng /ml	(IL-6) pg /ml

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Control	119.97±20.62	5.213±0.193
Patients with obesity	141.08±16.18**	11.655±0.567 **

Table (4) shows the level of inflammatory mediators TNF- α and IL-6 in obese individuals compared to the control group.

*Indicates the presence of significant differences at the level of (P≤0.05)

Discussion

Elevated levels of LDL-C and vLDL-C in obese individuals lead to heart disease. Obesity results in insulin resistance in the body due to dysfunctional adipose tissue, which leads to elevated levels of vLDL-C and TG [16].

The elevated lipid profile can be attributed to several factors, perhaps the most important of which is the high-energy diet in obese individuals, in which excess energy used for movement, metabolic processes, and other bodily functions is converted into fat storage in storage areas of the body or around internal organs, causing obesity and its progression. This contrasts with healthy individuals, who have sufficient energy to perform bodily functions without excess energy [17].

Numerous studies have confirmed that high cholesterol levels are influenced by dietary habits and genetic predisposition, such as hereditary cholesterol.The physiological mechanisms involved in fat metabolism in obese people are disrupted, which results in raised lipid profile values, including total cholesterol. Increased fat mass can also contribute to elevated total cholesterol levels due to metabolic disorders [18].

The current study elevated triglyceride levels could be caused by eating foods high in fat, which causes the intestines to produce more chylomicrons. Fatty acids are released during their breakdown, and as a result, the liver produces more of them, which raises the production of triglycerides, particularly when insulin is lacking. Obesity-induced deterioration of fat cells in the body causes changes in the percentage of fats and the loss of fat storage capacity, which causes the body to increase triglyceride breakdown and blood fatty acid levels, both of which have a detrimental effect on liver tissue [19].

Increased fatty acids in the liver lead to the conversion of some of them into phospholipids and cholesterol, which are transferred with triglycerides formed in the liver into the blood, resulting in an increase in blood lipid levels. Increased TG concentrations are directly related to insulin resistance, and a decrease in HDL-C

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negatively impacts lipid profile values and the role of high-density lipoprotein (HDL-C) in transporting excess cholesterol from cells via plasma to the liver. Therefore, low levels of high-density lipoprotein (HDL-C) indicate a state of cholesterol utilization in various metabolic processes [20].

Low HDL-C frequently signals the existence of health issues, particularly atherosclerosis and heart disease, which are brought on by the buildup of fat on the blood's inner walls. This is because HDL-C is in charge of reducing the amount of fat that returns to the liver from the arteries. Its ability to return those fats to the liver and preserve the health of the heart and arteries is enhanced by increasing its concentration. Because obesity is linked to a disruption in the body's fat distribution and results in the accumulation of extra fat in the muscles and abdomen, there is a direct correlation between the risk of heart attacks and atherosclerosis, Consequently, the concentration of high-density lipoproteins (HDL-c), a risk factor for atherosclerosis, decreases and the levels of triglycerides (TG) and low-density lipoproteins (LDL-c) rise [21].

Elevated body mass index (BMI) is attributed to hypercholesterolemia, which leads to obesity and weight gain.The relationship between obesity and the incidence of heart attacks and atherosclerosis is directly related, as obesity is associated with a disturbance in the pattern of body fat distribution and the deposition of excess fat under the skin, abdomen, and muscles. Therefore, it represents an additional burden on the heart, leading to lower levels of HDL-C and higher TG and LDL-C concentrations. Consequently, it is a risk factor for heart disease [22].

In obese patients, there is a negative correlation between subclinical chronic inflammatory indicators and adiponectin (APN) levels. The insulin resistance linked to obesity and metabolic syndrome is brought on by hypertrophic adipocytes. Additionally, the expression of adiponectin receptors (AdipoR1/R2) and the varying adiponectin-mediated inflammatory responses of macrophages in obese people are mostly controlled by macrophage polarization. Reduced expression of Adiponectin receptors (AdipoR1/R2) leads to reduced binding of adiponectin to the cell membrane, resulting in impaired adiponectin effects. This is known as adiponectin resistance and is associated with insulin resistance in individuals consuming a high-fat diet. Increased expression of adiponectin receptors restores insulin sensitivity and fatty acid beta-oxidation by stimulating intracellular signaling cascades. The ratio of high molecular weight to total APN is known as the APN sensitivity index (ASI). This index is related to insulin sensitivity [23].

Leptin is a protein hormone produced by adipocytes of white adipose tissue. It is often associated with body fat content and body mass index (BMI), being higher in obese individuals than lean individuals. It is two to three times higher in females than in males of the same age and BMI. [24][25] In obesity, there is resistance to

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appetite-regulating hormones, including leptin. Although leptin levels are elevated due to increased fat, the neural response to it is reduced due to chronic inflammation in the pituitary gland and molecular changes, a condition known as leptin resistance [26][27].

Recent studies indicate that increased leptin may result from these inflammatory and hormonal changes. Oxidative stress plays an indirect role in increasing serum atherogenic factors through the activation of protein kinase C. Leptin increases the secretion of lipoprotein lipase atherogenic (LPL) from macrophages, and LPL is a key enzyme in fat metabolism [28].

The function of resistin as a hormone is obvious. One sign of heart illness, particularly heart failure. The translocation of the resistin gene to atherosclerotic plaques raises the lipid and macrophage composition of these plaques. These effects cause the atherosclerotic plaque to become more unstable and prone to damage in addition to increasing in size and progression. This is because it activates the blood vessel's endothelial cells and promotes the growth of smooth muscle cells, which thickens the coronary artery lining [29].

Studies have indicated the role of both leptin and resistin in the activation of inflammation, which may play an important role in the mechanism of inflammation and promote fat accumulation resulting from obesity [30].

Despite its conflicting effects, adiponectin and resistin are both crucial for obesity, low-grade inflammation, and illnesses linked to obesity. Their levels are influenced by a number of variables, including the body's adipose tissue content, nutrition, and physical exercise [31].

The main source of resistin, adipose tissue, is overproduced in people. Conversely, because adiponectin has anti-inflammatory properties, its concentrations in the blood fall as the amount of adipose tissue increases. Adiponectin levels are decreased in obese people and those with cardiovascular disease. This has been attributed to its anti-inflammatory activity [32].

Adiponectin, an antioxidant molecule, prevents the activation of platelets in response to lowered oxidative stress. Adiponectin anticoagulant activity has been reported to be associated with inhibition of platelet aggregation via activation of the endothelial nitric oxide synthase and suppression of H2O2 formation [33].

The study revealed the decline of nesfatin-1 in both patient samples, explaining the obesity. Reduced levels of nesfatin-1 among obese subjects versus normal-weight individuals could be connected to impaired pathways of appetite and energy regulation. It is a component of the satiety system because it is a peptide resulting from NUCB2 protein, which contributes to feeling full and energy consumption by

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hypothalamus. It is well known that nesfatin-1 has a potent anorexigenic effect, because higher level of nesfatin-1 decreased feeding Scale down food intake and satiety [109] as it also acts on the energy homeostasis region of the brain. Besides the modulation of appetite, nesfatin-1 takes part in the control of energy expenditure and glucose and fat metabolism. Hence, nesfatin is considered as a potential target for therapy against loss of appetite in obese patients. The drop of nesfatin could be attributed to these hormonal and inflammatory changes as well the involvement of cytokines like TNF-α in nesfatin down-regulation [35]. It is also involved in cardiovascular control, as some studies demonstrate that it may influence blood pressure, heart rate and vascular function. It is also involved in response to physiological and psychological stresses and could influence mood, anxiety and other neurological functions [36].

With apparent effects on multiple adipose tissue-released hormone variables, the TNFα and IL-6 system is considered a potent pro-inflammatory mediator playing role in body energy homeostasis. In our report, we observed that obese patients had higher levels of pro-inflammatory cytokines (sTNFR1 and IL-6) compared with non-obese subjects [37].

Interleukins are proteins that can be heavily involved in the acute phase of inflammation with changes of 25% or more up or down during inflammation. They are mainly released by macrophages and monocytes, although it can be produced also in others cells like adipocyte [38].

Increased production of tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6) in obese patients demonstrates a state of chronic, low-grade inflammation which is one the most important characteristics accompanying obesity. This inflammatory situation disturbs satiety messages in the hypothalamus and enhances leptin resistance causing ineffective activity even when there are high levels of leptin as a result of excessive body fat. Inflammation is a biologic response to tissue injury and is fundamental in the pathophysiology of many diseases of the heart, including coronary artery disease (CAD) and other forms of atherosclerosis. IL-1 and IL-6 are critical players in promoting inflammation with impact on heart disease pathogenesis. Accordingly, inhibition of the IL-1 and IL-6 signaling cascade may be a novel treatment target in patients with atherosclerosis and HF. Visceral obesity has been strongly related to (IL-6) levels. This could be associated with the finding that adipose tissue is a dynamic endocrine organ producing several substances which promote systemic inflammation [39].

Conclusion

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The present results suggest that the obese patients have a dysregulation of their immunity and endocrine system, as observed through an elevation of proinflammatory cytokines levels as IL-6, TNF-α and high level of leptin and resistine hormones related with insulin resistance and augmented inflammatory phenomena. In contrast, lowered levels of nesftin and adiponectin (two antiobesity human hormones involved in metabolism regulation and combating inflammation) were detected. These altered biochemical values correspond to the chronic inflammatory condition in obesity and provide insight to the linkage between obesity and increased risk of chronic diseases like diabetes and heart disease.

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