

Assessment of Some Adipocytokines in Obese Males

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ABSTRACT

Obesity metrics are essential for assessing health risks and formulating effective preventive and therapeutic strategies. The study aimed to investigate various obesity-related proteins, including ET-01, Nesfatin-1, Vaspin, Visfatin, Ghrelin, Apelin, Leptin, and Resistin, in obese persons and healthy controls. A total of 160 people (120 obese patients and 40 controls) participated in the study from January 2024 to June 2024. The samples underwent centrifugation (3000 g, 20 min.), after which the serum was isolated and stored at -20°C. Serum from both the patient and control groups was subjected to enzyme-linked immunosorbent assay (ELISA) assays for ET-01, Nesfatin-1, Vaspin, Visfatin, Ghrelin, Apelin, Leptin, and Resistin. The present study included 160 participants across various stages of obesity and age groups, ranging from 19 to 55 years, with a mean age of 31 years for overweight individuals, 39 years for type I obese, 46 years for type II obese, and 24 years for the control group, respectively. The biomarkers of obesity were evaluated in the study population, revealing a significant difference in Apelin levels with $P < 0.05$; the mean Apelin level was lowest in the obese type II group (77.6 pg/dl). The other obesity biomarkers showed no significant differences across the research groups, with almost all exhibiting $P > 0.05$. The current investigation demonstrated no significant correlation between obesity indicators and weight gain, despite a notable increase in serum Apelin levels in obese subjects.

Keywords: Obesity, BMI, WHR, NOW, WC.

1. INTRODUCTION

Obesity is characterized by the excessive or abnormal buildup of adipose tissue, adversely affecting health by increasing the risk of diabetes mellitus, cardiovascular disease, hypertension, and hyperlipidemia [1]. Assessing obesity is crucial for evaluating health risks and formulating appropriate preventative and treatment measures. Numerous techniques exist for assessing obesity, with the primary way being the body mass index (BMI), a calculation that determines body fat by dividing weight in kilograms by height in meters squared. A BMI of 30 or more is classified as obesity and correlates with a heightened risk of metabolic syndrome [2]. Overweight and obesity, critical public health concerns, have developed into a worldwide epidemic [3]. Obesity, characterized by an excessive accumulation of body fat, significantly increases the risk of several chronic illnesses, including metabolic syndrome, dyslipidemia, hypertension, infertility, diabetes mellitus, cancer, and cardiometabolic disorders [4]. Obesity is characterized by a body mass index (BMI) of 30 kg/m² or above in clinical and scientific settings. Alongside BMI, other anthropometric measurements, including waist circumference (WC) and waist-to-hip ratio (WHR), were employed to assess abdominal obesity. BMI has several limitations as a measure of body fat; however, it is widely utilized. De Lorenzo (2006) coined the term "Normal Weight Obesity" (NWO) condition to describe persons with a normal BMI yet exhibiting a significant body fat percentage [5]. Individuals are deemed healthy when their BMI falls within the usual range. Prior studies have demonstrated that persons with normal weight obesity (NWO) are susceptible to metabolic disorders, including metabolic syndrome, cardiometabolic diseases, hyperlipidemia, and cardiovascular risk factors [6]. Besides serving as a fat reservoir, adipose tissue possesses endocrine and paracrine functions and functions as a metabolically functioning organ. Adipose tissue-derived hormones, referred to as adipocytokines or adipokines, are crucial for the development of metabolic disorders and an inflammatory response. Adiponectin, leptin, resistin, visfatin, omentin, and cytokines, including IL-6 and TNF- α , are hormones secreted by adipose tissue. A new investigation reveals that obesity and type 2 diabetes correlate with the overexpression of adipocytokines, including apelin, vaspin, resistin, and TNF- α , which promote insulin resistance [7], [8], [9]. Moreover, myokines—signaling molecules having autocrine, paracrine, and endocrine functions—are secreted by skeletal muscle cells [10]. Moreover, myokines—signaling molecules having autocrine, paracrine, and endocrine functions—are secreted by skeletal muscle cells [11], [12], [13].

2. PATIENT INFORMATION

The total number of 160 subjects (120 obese cases and 40 control) were involved in the current study during the period of January 2024 to June 2024. All cases with known history of obesity with body mass index (BMI > 27). Patient data were obtained from each individual after obtaining written consent form. Blood specimens (5 ml) were collected from each participant. The samples underwent centrifugation (3000 g, 20 min.), and the serum was isolated and stored at -20°C. Serum from both the patient and control was subjected to ELISA testing (ET-01, Nesfatin-1, Vaspin, Visfatin, Ghrelin, Apelin, Leptin and Resistin) following the manufacturer instruction (SUNLUNG /Biotech, Ltd; China).

3. STATISTICAL ANALYSIS

Statistical analysis was conducted using GraphPad Prism (version 10.1, USA). Comparisons across the groups were performed with One-Way ANOVA and/or Chi-square testing if required. A P value beyond 0.05 is deemed non-significant, but a P value below 0.05 indicates substantial variance among groups.

4. INTERVENTION

The present study included 160 participants across all stages of obesity and age groups, ranging from 19 to 55 years, with mean ages of 31 years for overweight individuals, 39 years for type I obese, 46 years for type II obese, and 24 years for the control group, as seen in Table 1.

Regarding the BMI distribution among the study population, as illustrated in Table 2, revealed that the mean BMI of the obese type II group was the most elevated (38.07), followed by the obese type I group (32) and the overweight subjects (31.5).

Our findings indicated that the mean hip/waist ratio for the obese type II group was 0.93, then followed by 0.92 for the obese type I group, while the control group exhibited the lowest ratio at 0.83. A notable difference was detected ($P < 0.05$) during the One-Way ANOVA analysis, as shown in Table 3.

The biomarkers of obesity were evaluated within the study population, as shown in Table 4, revealing a significant difference in Aplin levels with $P < 0.05$; the mean Aplin value was lowest in the obese type II group (77.6 pg/dl). The other obesity biomarkers exhibited no significant differences across the research groups, with almost all having $P > 0.05$.

Table 1. The mean age of the study groups

Age	Overweight	Obese type I	Obese type II	Control
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD
	31.58 $\pm$ 4.25	39.47 $\pm$ 8.61	46.14 $\pm$ 14.08	24.82 $\pm$ 2.99
Total	40	40	40	40
	160			

Table 2. BMI distribution of the study groups

BMI	Overweight	Obese type I	Obese type II	Control	P value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
	27.03±1.23	32.0±1.26	38.07±1.73	21.81±1.52	<0.0001
Total	40	40	40	40	
	160				

Table 3: Hip/waist ratio of the study groups

Hip/waist ratio	Mean	$\pm$ SD	P value
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Overweight	0.87	0.05	<0.0001
Obese type I	0.92	0.07	
Obese type II	0.93	0.09	
Control	0.83	0.05	

Table 4: Biomarkers levels among the study groups.

Immunity parameters	Overweight		Obese type I		Obese type II		Control		P value
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	
Endothelin (ET)-1 (ng/ml)	15.89	2.45	15.19	2.63	15.27	1.57	14.80	1.52	0.2777
Nesfatin-1	49.60	7.92	47.47	8.08	48.68	1.67	47.53	8.76	0.3405
Vaspin (ng/ml)	2.58	0.41	2.48	0.34	2.27	0.39	2.57	0.37	0.2437
Visfatin (ng/ml)	0.75	0.20	0.75	0.15	0.64	0.10	0.67	0.15	0.0929
Ghrelin (pg/ml)	155.50	31.24	157.30	48.89	158.20	15.17	141.90	32.64	0.3809
Apelin (pg/ml)	105.40	17.15	104.80	22.10	77.65	6.88	105.00	20.44	0.0018
Leptin (pg/ml)	288.00	53.21	273.20	39.54	282.10	32.18	267.70	43.33	0.3289
Resistin (ng/ml)	0.24	0.07	0.21	0.05	0.22	0.04	0.21	0.05	0.6888
Total (n)	40		40		40		40		

5. DISCUSSION

Obesity has risen in prevalence globally, and the WHO has classified it as a global pandemic. Obesity is a multifaceted, diverse, chronic, and progressive condition that significantly impacts health, quality of life, and death. Lifestyle and behavioral therapies are essential elements of obesity management [14]. The current results on BMI corresponded with another publication indicating that obese patients had a body mass index (BMI) of 30 kg/m² or above, along with hip/waist ratios of ≥ 0.85 for females and ≥ 0.90 for males [15]. Obesity is a worldwide health issue that impacts millions and elevates the risk of several chronic illnesses. To appropriately evaluate obesity and its metabolic consequences, it is essential to examine many metrics, including WHR and BMI. While BMI is a commonly utilized metric for evaluating weight status and recognizing potential health risks linked to excess body weight, it fails to differentiate between various forms of obesity or distinguish between muscle mass and fat mass in individuals, thereby constraining its specificity in certain instances [2]. The present study evaluated many characteristics associated with obesity in guys that may affect the escalation of BMI or obesity in these people. Certain metrics, such as Apelin, were considerably elevated in obese individuals (P value = 0.0018) compared to healthy and overweight controls, confirming the protein's significance in enhancing the likelihood of weight gain in predisposed participants. Apelin is a regulatory peptide and a ligand for the G-protein-coupled receptor known as APJ. It is widely distributed throughout the body, including peripheral tissues and the central nervous system. The apelin-APJ system has attracted significant research attention over the past decade owing to its potential role in several physiological processes, such as homeostasis, fluid control, cellular proliferation, and energy metabolism. Apelin is produced and secreted by adipocytes, so it is classified as an adipokine. Multiple studies have demonstrated that increased plasma apelin correlates with metabolic diseases. Evidence substantiates the regulatory role of the apelin-APJ system in glucose and lipid metabolism [16]. The current data revealed no significant variation in Vaspin levels across the investigated groups (P > 0.05), suggesting a potential underlying physiological condition in the subjects that may govern this protein's levels. Additional data indicate an increase in Vaspin levels among obese patients with a heightened BMI. In people, serum vaspin levels range from 0.2 to 2.5 ng/mL. In humans, elevated blood vaspin levels are associated with body mass index (BMI), whereas reduced serum vaspin concentrations constitute a risk factor for the progression of type 2 diabetes mellitus (T2DM) [17]. Obesity

significantly affects normal lung function. Obesity alters the mechanics of the lungs and thoracic wall due to adipose tissue, hence diminishing lung compliance. Numerous studies have indicated that excess adiposity correlates with heightened generation of inflammatory cells and induces airway hyperresponsiveness [18]. Conversely, Endothelin (ET)-1 in our data showed no significant variation among the examined groups; its rise was not markedly observed in any of the groups, since all subjects were healthy people, albeit obese, with no history of cardiac or tissue injury. Endothelin-1 (ET-1) is the most potent vasoconstrictor peptide released by the endothelium, recognized for its crucial role in modulating vascular tone and its involvement in the etiology of atherosclerotic vascular disease. Enhanced activation of the ET-1 system has been linked to the initiation and progression of several obesity-related cardiovascular conditions, including hypertension, type 2 diabetes, coronary artery disease, and chronic heart failure [19]. Concerning Nesfatin-1, our findings revealed no significant disparity in nesfatin levels among the tested groups, maybe attributable to individual variability among the participants in our study. Nesfatin-1 is a hormone released by the hypothalamus nuclei that regulate hunger. The correlation between obesity and nesfatin-1 was examined due to nesfatin-1's influence on consumption of food and energy expenditure. A link was observed between the polymorphism of the nucleobindin-2 gene and obesity. This might be a risk factor for the development of obesity [20], [21]. The current findings indicate no significant variations in Ghrelin levels across the tested groups. None of the research participants had dietary restrictions that might substantially influence the levels of this peptide in response to dietary modification. Ghrelin is an intestinal peptide that Ghrelin occurs in two forms: Acylated ghrelin (AG), which displays orexigenic, obesogenic, and diabetogenic characteristics, and Unacylated ghrelin (UAG), which has been demonstrated to mitigate these effects. Systemic ghrelin concentrations in blood plasma increase before ingestion, and its secretion is accelerated and enhanced under caloric restriction [22].

6. CONCLUSION

In conclusion, the findings presented in this article indicated no significant correlation between obesity indicators and weight gain, despite a notable increase in serum Apelin levels in obese subjects.

7. RECOMMENDATIONS

The study recommends to consume low fat diet in addition to reduction of sugar intake as well as including routine light exercise in order to maintain healthy life-style.

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Conflict of interest: no conflict of interest.

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