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Evaluation of Apelin, Elabela, and Certain Biomarkers in Patients with Metabolic Disease Associated with Obesity

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Abstract

Background: A novel peptide known as Elabela was recently discovered; it functions similarly to apelin and acts through apelin receptors. This research is to compare the characteristics and biological roles of apelin and Elabela in patients with metabolic syndrome who are obese and those who are not.

Patients and Methods: A cross-sectional study included 90 participants, ages 20–45, whose samples were collected from Al-Yarmouk-Hospital/Al-Karkh/Baghdad, from April–July/2024. Classified into three groups: group1 was obese metabolic syndrome patients (N=30), group 2 was metabolic syndrome patients without obesity (N=30), and group 3 was control (N=30). Anthropometrics and parameters were assessed for all study groups. Fasting blood glucose levels and lipid profiles were determined using an enzymatic process with spectrophotometric methods, while insulin, apelin, and Elabela were evaluated by enzyme-linked immunosorbent assay.

Results: The result shows a significant difference in Body Mass Index, Waist to Hip Ratio, Lipid profile, FBS, apelin, insulin, and HOMA-IR, while there was no significant difference between the study groups in Elabela levels. In addition, there is a positive correlation between apelin and BMI, FBS, and insulin. apelin levels have a higher diagnostic value for obesity than metabolic syndrome, respectively.

Conclusion: This study suggests that apelin and Elabela are powerful modulators of the metabolism of adipose cell and highlights the crucial role of apelin in MetS in both non-obese and obese MetS, as well as in clinical and biochemical markers associated with obesity.

Keywords: Apelin, APJ, Elabela, Metabolic Syndrome, Obesity.

Introduction

The global health community is concerned about the rising prevalence of obesity since being overweight raises the risk of developing a number of illnesses, including cancer, diabetes, and cardiovascular diseases (1). There is significant variation in the prevalence of obesity among different populations due to the interaction between local environmental factors and genetic factors, as well as

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as well as the drivers of the global food system. According to epidemiology, middle-aged adults are primarily affected by obesity in low-income countries, but people of all ages and both genders are affected in high-income countries. However, rising obesity rates have a significant negative impact on people's health and economies globally (2).

A collection of metabolic illnesses, including abdominal obesity, hypertension, dyslipidaemia, and impaired glycemia, is collectively referred to as metabolic syndrome (MetS). It is very common everywhere in the world and has two main definitions. More than one-third of adults with MetS were reported by the National Health and Nutrition Examination Survey (NHANES), and this number increased by more than 35% between 1988–1994 and 2007–2012. All things considered, the prevalence of MetS increases in tandem with the obesity rate. In ten extensive European cohorts comprising 163,517 individuals, the age-standardized proportion of obese subjects with Metabolic Syndrome varied between 24 and 65% for females and between 43% and 78% for males (3).

Due to their widespread prevalence throughout the world and link to a higher risk of developing chronic illnesses, obesity and MetS are serious public health concerns (4). Apelin is the name of the peptide that binds to the G-protein-coupled receptor APJ. There are numerous active apelin forms, including apelin-13, apelin-36, apelin-17, and apelin-13's pyro-glutamate form. In particular, the hypothalamus and numerous peripheral tissues in the central nervous system express apelin and APJ. Studies have demonstrated the involvement of apelin in the control of angiogenesis, food intake, cardiovascular and fluid homeostasis, and cell proliferation. Apart from its widespread presence, apelin is classified as an adipokine since it is generated and released by adipocytes (5). This adipokine has been shown to play a significant role in the development of metabolism and eating behavior during storage, as well as in maintaining physiological balance and preventing obesity-related diseases, such as high blood pressure and type II diabetes. Although apelin was found in the white adipose tissue of rats, the cells secreting the substance were unknown. The identification of apelin transcripts may be the result of several cell types present in this tissue, aside from the adipocytes themselves (6).

Elabela is a peptide hormone found after apelin and acts by binding to purinergic receptors. It was first described by the riversides' group as the first ligand of APJ in zebrafish embryos and shown to have effects on endodermal differentiation and carcinogenesis. Elabela is primarily present in embryonic stem cells, vascular endothelium, kidney, prostate tissue, and placenta. Elabela is similar to apelin, both bind to the same receptor APJ and cause similar effects. Although they have similar effects, these two peptides differ from each other in that they employ distinct signaling pathways and induce diverse biological effects. Additionally, there have been reports indicating that Elabela produces its effects via a receptor other than the APJ receptor (7).

Patients and Methods

Study design and blood sample collection: The cross-sectional study was conducted from April to July 2024 at the Department of Chemistry, College of Science, Al-Nahrain University. Ninety selected participants, ages 20-45, collected from Al-Yarmouk Hospital, Al-Karkh, Baghdad, Iraq, classified into three groups: group1 patients diagnostic with obesity (≥30 kg/m2) and MetS (score ≥3) (obese metabolic syndrome patients) (N = 30), group 2 patients diagnostic only with MetS (score ≥3) without obesity (normal weight (<25 kg/m2) (non-obese metabolic syndrome patients) (N = 30), and control group (N = 30). Blood samples were taken from all individuals in the study, who were

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fasting between 7:00 and 9:30 AM. After being placed in gel tubes, the venous blood samples were centrifuged for 10 minutes at 3000 rpm in to extract serum, and they were allowed to clot for 30 minutes. Serum was examined for standard laboratory parameters. The remaining serum samples were kept at -20 °C until the day of analysis for the Apelin and Elabela measurement.

Inclusion criteria: Healthy men and women without metabolic syndrome, and obese or non-obese patient with metabolic syndrome. All parse participants aged (45-20).

Exclusion criteria: Diabetes mellitus patients with thyroid disorders (hyperthyroidism or hypothyroidism), and gastrointestinal diseases.

Biochemical assays: Measurement of glucose and Lipid profile (Cholesterol, triglycerides, HDL, LDL, VLDL) using a spectrophotometer instrument (Human kit, Germany).

Insulin measurement and HOMA-IR calculation: Insulin levels were determined using VIDAS KUBE, an immunoassay autoanalyzer, according to FISH methods (Durham, North Carolina 27712, USA). Using the following formula, the homeostatic model assessment of insulin resistance (HOMA-IR) was determined:

[HOMA-IR = (mg/dL of glucose x mU/L of insulin)/405].

Apelin and elabela quantification: A Human Apelin and Elabela ELISA kit, using the sandwich method (My BioSource, USA), was used to measure serum apelin and Elabela levels. This kit is a sandwich kit used for the quantitative determination of parameters in serum.

Anthropometric and metabolic syndrome assessment: Body mass index (BMI) was classified into three categories: normal weight (less than 25 kg/m^2), overweight (between 25 and 30 kg/m^2), and obesity (more than 30 kg/m^2). For metabolic patients who are obese, the following criteria must be met: more than 150 mg/dL of triglycerides, more than 110 mg/dL of fasting glucose, and more than 102 cm of abdominal obesity in women and 88 cm in men, respectively; HDL cholesterol ranges between 40 and 50 mg/dL for both sexes. additionally evaluated the elements that comprise the metabolic syndrome (MetS) and computed the MetS score. Individuals were categorized as presenting with MetS if their score was ≥3. The MetS score ranges from 0 to 6 (3). The waist-to-hip ratio (WHR) was calculated by dividing the waist circumference (measured in cm) by the hip circumference (measured in cm).

Statistical analysis

A program, SPSS version. 29, was used for analysis. The variables were reported as means ± standard deviations, with one-way ANOVA and correlation coefficients. To identify the risk factors for MetS, multiple logistic regression analyses were carried out. Apelin and Elabela's ideal threshold values for MetS diagnosis prediction were calculated using a receiver operating characteristic (ROC) curve. Statistical significance was defined as a P value of ≤ 0.05.

Results

Glucose and lipid profile levels assessment: A total of 90 individuals, 30 obese with metabolic syndrome patients, 30 non-obese with metabolic syndrome patients, and 30 healthy controls, were included in this study. The mean BMI values of obese Mets patients and non-obese Mets patients compared to control groups were 36.24±2.22, 23.47±1.7, vs. 22.25±1.9 kg/m^2, respectively. The obesity with MetS was significantly greater than non-obesity patients with MetS, and healthy groups among participants ($p<0.05$), as appears in Table 1.

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Table 1. Characteristics of patients from the study groups with and without obesity, MetS and control.

Groups Parameters	Obese Patient with MetS group No. (30)	Non-Obese Patient with MetS group No. (30)	Control group No. (30)	p-value
BMI (kg/m^2)	36.24±2.22	23.47±1.7	22.25±1.9	≤ 0.05
W/H ratio	0.93±0.09	0.90±0.1	0.82±0.05	≤ 0.05

The mean values of fasting blood glucose and lipid profile (triglycerides, cholesterol, HDL, VLDL, and LDL) for each of the research groups were included in Table 2. The mean standards in this table revealed that there were important variances in triglycerides, HDL, and LDL among the obese and non-obese patients with MetS groups, and the control group ($p \leq 0.05$), while there were no important variances in fasting blood glucose and cholesterol among all study groups ($p \geq 0.05$).

Table 2. The FBS and lipid profile of the study groups with and without obesity, MetS, and control groups.

Groups Parameters	Obese Patient with MetS group No. (30)	Non-Obese Patient with MetS group No. (30)	Control group No. (30)	p-value
FBG (mg/dL)	97.20±1.33	94.75±1.31	83.42± 0.95	NS
TC (mg/dL)	189.45±30.45	180.57±27.41	172.32±25.30	NS
TG (mg/dL)	269.99±63.11	249.12±56.14	132.6±50.4	≤ 0.05
HDL-C (mg/dL)	40.11±0.61	45.32±0.59	60.4±1.5	≤ 0.05
LDL-C (mg/dL)	186.8±28.66	90.6±1.22	85.4±0.99	≤ 0.05

Apelin, elabela, insulin, HOMA IR levels assessment: Table 3 presents the values of Apelin (pg/ml), Elabela (ng/mL), Insulin (μIU/mL), and HOMA IR for every group under study in the current research. The table demonstrated a statistically important variance among the obese MetS patients, non-obese MetS patients, and control group with ($p \leq 0.05$).

Table 3. Biochemical parameters of the study groups.

Groups Parameters	Obese Patient with MetS group No. (30)	Non-Obese Patient with MetS group No. (30)	Control group No. (30)	p-value
Apelin (pg/mL)	367.18±140.33	221.42±126.94	203.89±117.28	≤ 0.05
Elabela (ng/mL)	0.78±0.44	0.75±0.43	0.73±0.42	NS
Insulin(μIU/mL)	32.7±14.98	9.10±0.95	7.2±0.64	≤ 0.05
HOMA IR	7.8 ±0.62	2.1±0.05	1.5±0.03	≤ 0.05

Correlation analysis: Table 4 presents the outcomes of the correlation analysis conducted among numerical variables.

Table 4. Correlation Analysis between Variables.

		BMI	WHR	TC	TG	HDL	LDL	FBS	Insulin	HOMA-IR	Apelin	Ela
BMI	R	1										
	P											
WHR	R	.460	1									
	P	.041										
TC	R	.429	.176	1								

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	P	.059	.457									
TG	R	.457*	.525*	.309	1							
	P	.043	.017	.185								
HDL	R	.084	-.260	-.098	-.288	1						
	P	.726	.268	.682	.218							
LDL	P	.306	.127	.958*	.161	-.293	1					
	R	.190	.594	.000	.499	.210						
FBS	P	.227	.287	-.172	.398	-.237	.200	1				
	R	.335	.220	.467	.082	.315	.397					
Insulin	P	.093	.144	-.207	.042	-.067	.199	.090	1			
	R	.697	.545	.382	.859	.779	.400	.705				
HOMAIR	P	.078	.280	-.265	.268	-.172	.281	.675**	.793**	1		
	R	.743	.232	.259	.253	.469	.231	.001	.000			
Apelin	P	.760*	.348	-.038	-.286	.169	.070	.304*	.234	.005	1	
	R	.000	.133	.872	.221	.476	.771	.035	.321	.985		
Ela	P	.171	-.154	.195	-.156	-.120	.192	.011	.524	-.416	.327	1
	R	.471	.516	.409	.511	.615	.417	.965	.199	.068	.159	

The ROC curve analysis: According to the results of association analysis, the ROC curve analysis was used to examine whether apelin has the potential effect on obesity and metabolic disease. The area under curve (AUC) was 0.937 in Figure 1 and 0.711 in Figure 2. The area under curve for apelin showed higher values as shown in Figure 1, more than in Figure 2. Therefore, a high level of apelin expression in adipose tissue is associated with obesity and a metabolic syndrome (Table 5).

Table 5. ROC curve analysis of apelin in the MetS in both the obesity and non-obesity groups.

Area Under the Curve					
Test Result Variable(s): Apelin					
	Area	Std. Errora	Asymptotic Sig.b	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Figure 1	.93	.049	.000	.841	1.000
Figure 2	.71	.111	.046	.493	.928

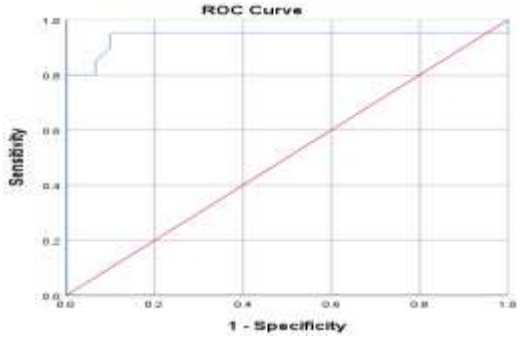

Figure 1. ROC curve of Apelin in obese-MetS patients.

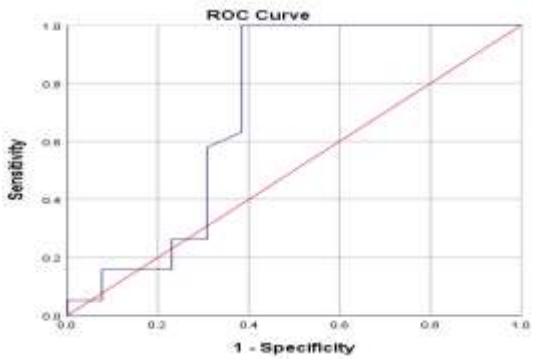

Figure 2. ROC curve of Apelin in non-obese- MetS patients.

The analysis of ROC curves was used to determine the efficacy of apelin in the MetS in both the obesity

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and non-obesity groups. Serum apelin performed better in the obese-MetS group (AUC = 0.937) than in the non-obese-MetS group (AUC =0.711), with a higher cut-off value (388 pg/mL) in the obese-MetS group than in the non-obese-MetS group (239 pg/mL, respectively). Furthermore, apelin is a more accurate indicator of obesity in the obese-MetS group than in the non-obese MetS group, according to our observations.

Discussion

The conditions insulin resistance, hyperglycemia, dyslipidemia, and hypertension are linked to obesity, especially excess visceral adiposity. These conditions are collectively referred to as the "metabolic syndrome." Type 2 diabetes mellitus (T2DM) and cardiovascular diseases are made more likely to occur by these metabolic disorders, which also lead to elevated rates of morbidity and mortality (8). According to the data from this study, BMI and WHR increase more in the obese MetS group than in the non-obese MetS group and the healthy group. This result agrees with Masquio et al., (9) who found that obesity subjects with MetS have shown significant differences in body weight, BMI, and WHR compared to non-obese subjects with MetS. Adiponectin is associated with central obesity and is produced by adipocyte tissue, indicating that its levels are correlated with adipocyte mass (10). Since all patients with metabolic syndrome had central obesity, the levels were significantly increase than in subjects with healthy group.

The result of this study was in agreement with study by Gallagher EJ et al,(11) according to which low HDL and elevated TG are components of the metabolic syndrome's dyslipidemia profile. There was a 2.5 relative risk of coronary disease between people with TG levels in the top third of the population and people with TG levels in the bottom third of the population, according to a meta-analysis of prospective studies. Lipid profiles are significantly impacted by the onset of IR. With the loss of its inhibition of hormone-sensitive lipase, IR causes an increase in the amount of FFA produced by adipocytes. Furthermore, there is a decrease in endothelial lipoprotein lipase activity, which both add to the rise in free fatty acids in circulation (12). Hepatic TG increased as a result of increased FFA inflow into the liver and insulin-stimulated hepatic lipogenesis. Due to the liver's increased influx of free fatty acids (FFAs) and insulin's stimulation of hepatic lipogenesis, the liver produced more triglycerides, which were stored in the liver and resulted in steatosis and very low-density lipoprotein. Because the TG is stored in the liver, it is produced as VLDL and steatosis (13). Transferring cholesterol esters from HDL to VLDL is made possible by the cholesterol ester transferase protein produced by adipocytes (14). The low HDL and elevated TG levels associated with the metabolic syndrome are caused by the liver's absorption of HDL and subsequent production of VLDL due to increased HDL clearance by the kidney (15).

As the information in Table 3 showed, there was a highly significant difference in Apelin, insulin, and HOMA IR in the Obese Patient with MetS group compared to the Non-Obese Patient with MetS group and the Control group. While no significant difference levels found in Elabela concentration between patients them self and with control group. This result agrees with the findings of Mutlak, S.S., et al., (16), which discovered that the group with MetS had higher serum apelin levels than the group without MetS .

Apelin is thought to be a significant predictive biomarker for metabolic disorders. Additionally, the outcome concurs with research conducted by other scientists (17). Which demonstrated that MetS had higher apelin levels than age-matched controls. According to their findings, apelin levels and IR were positively correlated. Furthermore, the outcome is consistent with Angelova, P., et al., (18).

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A comparison of the apelin levels in the obese patients and the control group in their study indicates a significant increase in apelin levels. Moreover, apelin levels decrease following insulin sensitizer therapy in individuals with low body mass index. Apelin synthesis occurs in other locations, such as the vascular endothelium, which may conceal less secretion from fat tissue. This is most likely the cause of the observed phenomenon. However, following weight loss, apelin expression is shown to be reduced (19). Increased apelin levels in humans and animals have been linked to a number of metabolic disorders, according to some research, but not all of them. Apelin is now known to be a useful adipokine that has anti-diabetic and anti-obesity properties, which makes it a potentially useful therapeutic target for a number of metabolic disorders. When compared to normal controls, apelin levels have been shown in certain studies to be higher in insulin-resistant individuals and morbidly obese people with type 2 diabetes (20). Human endocardial endothelial cells and vascular endothelial cells have been found to contain apelin. It has been demonstrated that apelin significantly increases heart rate and contraction in the heart. apelin produces peripheral vasodilatation through a mechanism that is dependent on nitric oxide (NO). Apelin inhibits the electrical activity of vasopressin-releasing neurons in the hypothalamus, indicating a potential role in the control of vascular tone (21). An elevated fat diet in rats causes an increase in the expression of apelin in the subcutaneous adipose tissue (SAT). Both significantly obese patients having gastric banding and mice fed a high-fat diet had higher plasma apelin levels. ApeLIN levels have recently been shown to decrease in obese women following a 12-week period of diet-induced weight loss (22).

Apigenin-angiotensin receptor-like 1 (APJ) has two ligands. There are two: apelin and elabela/toddler (ELA) (23). APLNR and APJ are apelin receptors that control the biological activity of the peptide hormones APELIN (APLN) and ELABELA (ELA, Toddler, apela). ELA and APLN, two agonists of APLNR, have the ability to alter a number of intracellular signaling pathways, including PKA (protein kinase A) and adenyl cyclase (AC).

Toddler, also referred to as apelin or elabela, is a peptide consisting of 54 amino acids, which includes a secretory signal. Two research groups have recently discovered a mature form of elabela, which comprises 32 amino acids (24). The function of this peptide in relation to adipose tissue metabolism remains unclear. But given the information in the literature and how it affects metabolic pathways, like the control of SIRT3-mediated oxidative stress inhibition through Foxo3a deacetylation or the inverse relationship between blood glucose level and ELA, it is reasonable to believe that APLN and ELA are also involved in the metabolism of adipose tissue (25). The findings of this study concur with the first one conducted by Yeniel N. et al., (24). Which discovered that the Elabela concentrations in the obese and control groups did not differ significantly. Despite the fact that Elabela levels did not differ between obese participants in this study, the paucity of research on Elabela's function in metabolism or obesity makes it incorrect to draw firm conclusions. We think that more thorough research will provide more important details regarding Elabela's involvement in obesity.

The two primary underlying risk factors for metabolic disturbances are obesity and insulin resistance (IR), which also contribute to the rise in other risk factors like dyslipidaemia, hypertension, and hyperglycaemia (26). Another indicator of insulin resistance is an increase in hepatic fat brought on by an excess of free fatty acid influx to hepatocytes as a result of dysfunctional lipolysis brought on by insulin resistance. Visceral or hepatic fat cannot be accurately estimated by BMI or waist

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circumference alone. Selective loss of body fat, severe insulin resistance, and non-alcoholic fatty liver disease are characteristics of genetic or acquired lipodystrophies, suggesting that obesity in and of itself is not the primary cause of the metabolic syndrome. Rather, the cluster of abnormalities associated with the metabolic syndrome may be primarily caused by lipotoxicity and ectopic fat deposition. The outcome concurred with Widjaja, Nur Aisiyah, and others (27). Insulin resistance occurs when insulin is unable to inhibit lipolysis and Fox01, which influences how insulin signalling regulates gluconeogenesis and glycogenolysis, but instead triggers rapamycin complex-1 (mTORC1). The overproduction of VLDL and decreased clearance of VLDL are the results of insulin's inability to suppress Fox01, which raises the expression of apoCIII and microsomal triglyceride transfer protein (MTTP) (28).

The present study, demonstrated a positive correlation between serum plasma apelin levels and BMI. Comparable results were found in another study conducted by Zaki, Moushira, et al., (29) .Indicating that apelin plays a part in the etiology of obesity. Furthermore, additional research (30). The apelin levels were considerably higher in obese individuals than in control subjects, and they showed a positive correlation with BMI. Therefore, it seems that apelin concentration is significantly influenced by obesity. Furthermore, the significantly elevated apelin and insulin levels in obese MetS raise the possibility that apelin homeostasis is compromised. It could also imply that elevated insulin levels lead to an increase in apelin blood concentrations, as another study has also suggested. The results also showed a negative correlation between apelin and glucose, which is consistent with a study by Saadi, H.A.H., et al. (20). This shows that apelin may have significant therapeutic implications for metabolic syndrome. According to a study, apelin levels and fasting blood glucose (FBG) were negatively correlated. There was no correlation found between any of the parameters and Elabela levels. Further investigation is necessary to clarify the relationship between Elabela, insulin resistance, and the metabolism of glucose in individuals who do not have diabetes. This finding is consistent with a study conducted by Yeniel N. et al., (24). Which reported that there was no correlation between Elabela levels and other numerical variables. This might be because Elabela cannot sufficiently penetrate the blood-brain barrier to affect human nutritional centers.

Conclusions

The metabolic syndrome should be considered when assessing patients who are overweight or obese, as it may help identify those who are at a higher risk of developing type 2 diabetes and cardiovascular disease (CVD) in the future. The current study highlights the crucial role of apelin in MetS in both non-obese and obese MetS, as well as in clinical and biochemical markers associated with obesity. When it comes to MetS in obesity, serum apelin exhibits a high degree of predictive accuracy, outperforming matched non-obese individuals with MetS. This implies that serum apelin might be useful in MetS and obesity-related comorbidity settings, both clinically and therapeutically. Taking everything into account, the data points to APLN and ELA as potent adipocyte metabolism modulators. Nevertheless, since the effects of ELA are still unclear, more investigation is required.

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Ethical clearance: The Research Ethics Committee of AL-Nahrain University's Department of Chemistry, College of Sciences, approved this study, and in accordance with the ethical guidelines of the Declaration of Ethical Committee of the College (2024NC690). Written consent was obtained from all patients before inclusion.

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Conflict of interest: None.

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تقييم الأبلين والإيلابيلا وبعض المؤشرات الحيوية لدى المرضى المصابين بأمراض التمثيل الغذائي المرتبطة بالسمنة

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الملخص

الخلفية: تم اكتشاف ببتيد جديد يُعرف باسم ايلابيلا ؛ يعمل بشكل مشابه للأبيلين ويعمل من خلال مستقبلات الابلين.

الأهداف: يهدف هذا البحث إلى مقارنة خصائص وأدوار الأبلين والايلابيلا البيولوجية لدى مرضى متلازمة التمثيل الغذائي الذين يعانون من السمنة وأولئك الذين لا يعانون من السمنة.

المرضى والطرق: شملت الدراسة المقطعية ٩٠ مشاركًا تتراوح أعمارهم بين ٢٠ و ٤٥ عامًا، تم جمع عيناتهم من مستشفى اليرموك / الكرخ / بغداد، من أبريل إلى يوليو / ٢٠٢٤. تم تصنيفهم إلى ٣ مجموعات: المجموعة الاولى تشمل مرضى متلازمة التمثيل الغذائي المصابين بالسمنة (٣٠)، والمجموعة الثانية تشمل مرضى متلازمة التمثيل الغذائي غير المصابين بالسمنة (٣٠)، والمجموعة الثالثة تشمل مجموعة التحكم (٣٠). تم إجراء القياسات الأنثروبومترية والمعايير لجميع مجاميع الدراسة، والتي تتضمن: قياس نسبة الكلوكوز في الدم الصائم ومستوى الدهون بواسطة عملية إنزيمية باستخدام طرق قياس الطيف الضوئي، في حين تم تقييم الأنسولين والأبلين والإيلابيلا بواسطة اختبار الممتز المناعي المرتبط بالإنزيم.

النتائج: تظهر النتيجة فرقًا كبيرًا في مؤشر كتلة الجسم ونسبة الخصر إلى الورك ومستوى الدهون ونسبة مصل الأبلين والأنسولين و-HOMA IR، في حين لم يكن هناك فرق كبير بين مجموعات الدراسة في مستويات الإيلابيلا. أيضًا وجد ارتباط إيجابي بين الأبلين ومؤشر كتلة الجسم ونسبة مصل الابيلين والأنسولين. مستويات الأبلين لها قيمة تشخيصية أعلى للسمنة من متلازمة التمثيل الغذائي على التوالي.

الاستنتاج: تشير الأدلة إلى أن الابيلين والايلابيلا من المنظمات القوية لعملية التمثيل الغذائي للخلايا الدهنية. ومع ذلك، هناك حاجة إلى مزيد من البحث لأن تأثير الايلابيلا لا يزال غير معروف.

الكلمات المفتاحية: الأبلين، **APJ**، الإيلابيلا، متلازمة التمثيل الغذائي، السمنة.

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