




Assessment of Serum B-Arestin Level in Acromegaly Patients with Diabetes Mellitus

Hiba Ghassan Rajab ¹, Shatha Mj Al-Khateeb ¹, Abbas M. Rahmah ²

¹ Department of Biochemistry, College of Medicine, Mustansiriya University, Baghdad, Iraq.

² National Diabetes Center, Mustansiriya University, Baghdad, Iraq.

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Correspondence Hiba Ghassan Rajab

Email: Heba-ghassan@uomustansiriya.edu.iq

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Abstract

Background: Acromegaly is a rare endocrine disorder, with an annual incidence of 4 cases per million and a prevalence of 40 cases per million. The condition results from a pituitary adenoma that secretes growth hormone.

Objectives: The objective of this study is to assess β -arrestin levels in patients with acromegaly, comparing those with and without diabetes mellitus with a control group.

Patients and Methods: This is a case-control study involving 100 patients with acromegaly registered at the National Diabetes Center, Mustansiriya University, and 76 controls. All recruited participants have given oral consent to participate in the study, which was conducted from February to August 2024.

Results: the age distribution differences were not statistically significant ($p=0.156$). The gender distribution showed 47 females and 53 males in the acromegaly group. The β -arrestin level was higher among patients compared to controls, and showed the highest level in acromegaly and DM (16.86 ± 6.35 ng/ml), followed by acromegaly without DM (11.12 ± 4.78 ng/ml), controls with DM (6.62 ± 2.83 ng/ml), and controls without DM (2.93 ± 0.98 ng/ml).

Conclusion: High prevalence of diabetes among female patients with acromegaly. Elevated serum levels of β -arrestin were observed in patients with acromegaly, with higher levels noted in those with diabetes mellitus compared to non-diabetic acromegaly patients and control groups.

Keywords: Acromegaly. B-Arestin, pituitary adenoma, Diabetes mellitus.

Introduction

Acromegaly is an uncommon endocrine condition marked by increased levels of insulin-like growth factor 1 and growth hormone (1), usually caused by a somatotroph adenoma of the pituitary gland (2). Patients with macroadenomas make up 70% of the patient population, while microadenomas affect 30% of patients (3). With an annual incidence of 4-6 per million and a range of 34 to 137 per million, its incidence and prevalence are gender-neutral (4). In particular, when GH and IGF-1 levels are chronically raised, elevated IGF-1 levels lead to somatic growth and metabolic consequences, which in turn increase morbidity and death (5,6). When a patient has suspected acromegaly, biochemical tests, such as a random serum IGF-1 test and GH level after a glucose load, are used to confirm the diagnosis (7,8). Surgical resection is the usual course of treatment after a pituitary magnetic resonance imaging (MRI) scan to check for signs of an adenoma. When a macroadenoma is removed, almost half of the patients do not recover and need to have more surgery, medicine, or radiation (9). Acromegaly is

characterized by multiorgan/system dysfunction and progressively progressing somatic deformity, primarily affecting the face and extremities (10). Insulin-like growth factor-1 (IGF-1) is released from the liver in response to growth hormone. This alters gene transcription, impairs diastolic ventricular filling, and induces cardiomyocyte hypertrophy. Patients with acromegaly die primarily from cardiovascular issues (11). β -arrestin proteins, namely β -arrestin 1 and β -arrestin 2, are intracellular proteins that modulate G protein-coupled receptor (GPCR) signalling, including somatostatin receptors (SSTRs), which are essential in the management of acromegaly (12). β -Arrestins, are multifunctional intracellular proteins that play crucial roles in regulating a wide array of cellular signalling pathways and physiological functions (13), their synthesis is controlled by genes located on chromosomes 7 and 11, respectively (14), they are proteins with ubiquitous expression throughout the body, which have the ability to directly interact with a large number of cellular partners including the G protein-coupled receptors (GPCRs), and due to their critical role of β arrestins in regulating GPCRs, they have contribute to multiple aspects of GPCR signalling, trafficking and downregulation (15). A major advantage in the treatment of acromegaly is the increased expression of somatostatin receptor 2 (SSTR2) in the involved adenoma, which serves as the primary target for somatostatin analogs. A novel contributor develops in the field, namely β -arrestin, which is being identified as a regulator of SSTR2 function (16). The acromegaly is diagnosed in adolescent patients with excessive linear growth by assessing of excessive growth hormone. In adults, acromegaly should be diagnosed in patients exhibiting acral enlargement or characteristic facial features, as well as in those presenting with a combination of symptoms, signs, or conditions associated with acromegaly, including recurrent headaches, hyperhidrosis,

hypertension, sleep apnoea, oligomenorrhea, arthralgia, carpal tunnel syndrome, and type 2 diabetes mellitus (17). A gap of several years between the development of symptoms and diagnosis is typical. An extended duration between disease onset and diagnosis correlates with increased all-cause mortality and a greater prevalence of comorbidities, underscoring the significance of early detection and timely intervention (18).

Patients and Methods

Study design: This is case-control study, involving consecutive male and female adult patients with acromegaly recruited from February to august 2024.

Subjects: From the National Diabetes Center, Mustansiriyah University, which is a specialized center that offer diabetes care, treatment, and public health awareness, and known to collect a large number of acromegaly patients, totally about 300 patients with acromegaly were registered, these patients have fully written medical records and they attend the center every month to receive their somatostatin analogue injection and conduct required investigations according to the request of the endocrinologist. According to the treatment guidelines, all the patients were advised to have transsphenoidal hypophysectomy as the gold standard therapy except those who were unfit for surgery or not willing to do surgery. In this situation medical therapy by somatostatin analogue was the primary treatment. Out of 300 acromegaly patients, about 100 have visited the center during the period of the study, and totally about 176 individuals were included in the proposed study, then the participants were divided into two main groups, Group 1 included 100 patients with acromegaly, and Group 2 included 76 participants without acromegaly as controls. All the participants were given a comprehensive study description, and written informed consent was obtained from each participant.

Inclusion criteria: patients with acromegaly who registered at the national diabetes center during the study period, in their prespecified scheduled visits (individuals aged 30-70 years).

Exclusion criteria: which include the Patients with neuro-inflammatory or immune disorders and inflammation in the central nervous system and Pregnant or breastfeeding individuals may be excluded due to potential hormonal fluctuations that can affect β -arrestin levels, as well as ethical considerations.

Measurements of outcome: All patients underwent fasting for a duration of 8 to 12 hours. During the appointment, blood samples were collected in the morning prior to 9 a.m. A total of 5 ml of venous blood was withdrawn and centrifuged at 3000 rpm for 10 minutes.

Hormonal analysis: GH and IGF-1. The sera were subsequently analyzed for growth hormone and insulin like growth factor-1, both GH and IGF-1 were quantified using a fully automated device based on the Electrochemiluminescence Immunoassay (ECLIA) principle (Cobas E411) by roche, which based on conversion of electrical energy to light which proceeded by converting electrochemical reactions to luminescence, these reactions are products of generation from a stable precursor on electrode surface (19).

Estimation of blood glucose levels: Fasting blood glucose was determined by using an enzymatic colorimetric method, for the fasting blood glucose, the hydrogen peroxide is created when glucose oxidase breaks down glucose and then combines with a material that produces color. The blood glucose level is reflected in the color's intensity (Spin react, Spain) (20),

Estimation of HbA1c levels: the Roche Tinaquant® HbA1c Gen. 3 kit uses a turbidimetric inhibition immunoassay to measure HbA1c levels in whole blood. After the red blood cells are broken down, the test uses antibodies that specifically bind to the glycosylated hemoglobin. Then synthetic substance was added to compete

with HbA1c for these antibodies. The amount of turbidity formed in the solution decreases as more HbA1c is present. This reduction in turbidity is measured photometrically and is inversely proportional to the HbA1c concentration (21).

Assessment of serum β -Arrestin using ELISA: Serum Human β -Arrestins concentration were determined by using β -Arrestins ELISA kit (BT LAB/china-Cat.No E0398Hu), the principle was an Enzyme-Linked Immunosorbent Assay sandwich ELISA, in which the plate was pre coated with human β -Arrestins antibody, then β -Arrestins that present in the sample was added and bind to antibodies coated on the wells, then the yellow compound was produced whose absorption intensity could be measured at a wavelength of 450 nanometers (22).

Statistical Analysis

The statistical package for the social science program (SPSS) version 20 was used to analyses the data. Chi-square test for gender distribution, One-way ANOVA for age comparisons across groups, Post-hoc Tukey test for multiple comparisons, Kolmogorov-Smirnov test for normality of age distribution, Bonferroni correction applied for multiple comparisons, 95% confidence intervals calculated for mean ages. Significance differences were considered at $p < 0.05$.

Results

Demographic and clinical characteristics: The results in Table 1 show that, out of 100 patients with acromegaly have been enrolled in this study, 53 males and 47 females, and out of 76 healthy participants without acromegaly 42 males and 34 females and equal distribution of DM and non-DM cases in acromegaly group (50:50), nearly equal distribution in control group (48.7% DM vs 51.3% non-DM) and no significant difference in diabetes distribution between acromegaly and control groups, pituitary MRI had to be requested to verify the presence of Microadenoma (> 10 mm) or macroadenoma (≥ 10 mm in largest

diameter), macroadenoma was found in (74%) of the patients. The results in Table 2 show that Significant difference in gender distribution between DM and non-DM groups in acromegaly

patients (p=0.023) but higher proportion of females in DM groups across both acromegaly and control populations and more balanced gender distribution in control groups.

Table 1. Gender distribution and disease status of the participants.

	Parameters	Acromegaly (n=100)	Control (n=76)	p-value
Gender	Female, n (%)	47 (47.0%)	34 (44.7%)	0.765
	Male, n (%)	53 (53.0%)	42 (55.3%)	
Diabetes Status	DM, n (%)	50 (50.0%)	37 (48.7%)	0.861
	Non-DM, n (%)	50 (50.0%)	39 (51.3%)	

Table 2. Study population demographics by group and diabetes status.

Characteristic	Acromegaly with DM	Acromegaly without DM	Control with DM	Control without DM	P value
Gender, n (%)					0.023 *
Female	30 (60.0%)	17 (34.0%)	19 (51.4%)	15 (38.5%)	
Male	20 (40.0%)	33 (66.0%)	18 (48.6%)	24 (61.5%)	
Age Groups, n (%)					0.156
30-40 years	8 (16.0%)	15 (30.0%)	9 (24.3%)	12 (30.8%)	
41-50 years	28 (56.0%)	27 (54.0%)	20 (54.1%)	19 (48.7%)	
>50 years	14 (28.0%)	8 (16.0%)	8 (21.6%)	8 (20.5%)	

Serum biomarkers levels among the study groups:

The results in Table 3 show that β -arrestin levels showed significant differences between all groups, Acromegaly with DM showed the highest levels (16.86 ± 6.35 ng/mL), significantly higher than acromegaly without DM (11.12 ± 4.78 ng/mL, $t=5.10$, $p<0.001$). Both control groups showed markedly lower levels (DM: 6.62 ± 2.83 ng/mL; without DM: 2.93 ± 0.98 ng/mL), suggesting β -arrestin might be a potential biomarker for acromegaly, particularly in the presence of diabetes as shown in Figure 1 and 2. HbA1C levels were significantly higher in both diabetic groups (Acromegaly with DM: $8.47 \pm 1.64\%$; Control with DM: $7.30 \pm 0.90\%$)

compared to non-diabetic groups (Acromegaly without DM: $5.33 \pm 1.43\%$; Control without DM: $5.36 \pm 0.95\%$). Notably, acromegaly patients with DM showed significantly higher HbA1C levels compared to controls with DM ($t=4.27$, $p<0.001$), as shown in Figure (3). The results in table 3 showed that IGF-1 levels were markedly elevated in both acromegaly groups (with DM: 403.24 ± 168.18 ng/mL; without DM: 409.44 ± 184.63 ng/mL) compared to control groups (with DM: 242.89 ± 53.04 ng/mL; without DM: 247.50 ± 47.40 ng/mL; $p<0.001$ for both comparisons). There was no significant difference between the two acromegaly groups ($p>0.05$). GH levels were significantly elevated in both acromegaly groups

(With DM: 6.23 ± 3.42 ng/mL; without DM: 6.19 ± 2.31 ng/mL) compared to control groups (with DM: 1.46 ± 0.79 ng/mL; without DM: 1.26 ± 0.61 ng/mL; $p < 0.001$ for both comparisons).

Table 3. β -arrestin, HbA1C, IGF-1, GH and FBG across the study groups.

Parameter	Acromegaly with DM	Acromegaly without DM	Control with DM	Control without DM	P value
β -arrestin (ng/mL)	$16.86 \pm 6.35^*$	$11.12 \pm 4.78^\dagger$	$6.62 \pm 2.83^\ddagger$	2.93 ± 0.98	<0.001
HbA1C (%)	$8.47 \pm 1.64^*$	5.33 ± 1.43	$7.30 \pm 0.90^\dagger$	5.36 ± 0.95	<0.001
IGF-1 (ng/mL)	$403.24 \pm 168.18^*$	$409.44 \pm 184.63^*$	242.89 ± 53.04	247.50 ± 47.40	<0.001
GH (ng/mL)	$6.23 \pm 3.42^*$	$6.19 \pm 2.31^*$	1.46 ± 0.79	1.26 ± 0.61	<0.001
FBG (mg/dL)	$201.05 \pm 79.96^*$	$150.32 \pm 63.29^\dagger$	$157.43 \pm 22.98^\ddagger$	104.79 ± 38.41	<0.001

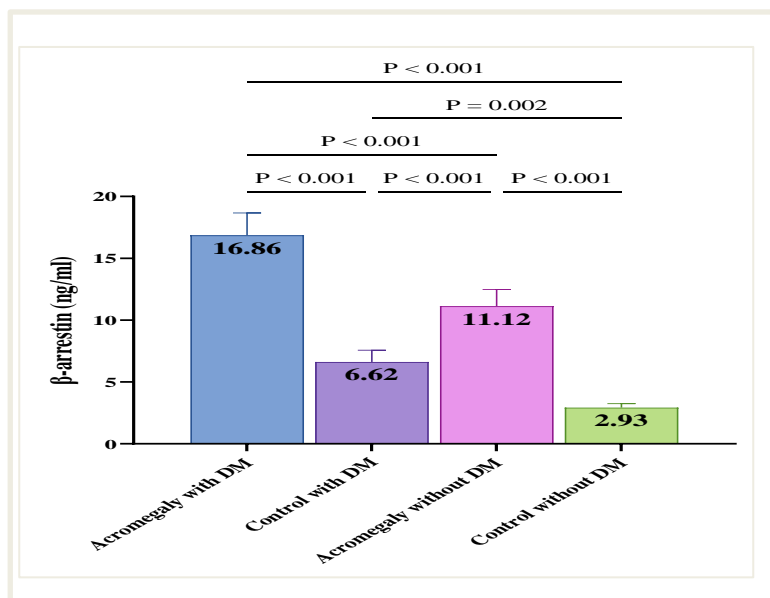


Figure 1. β -arrestin distribution across the study groups.

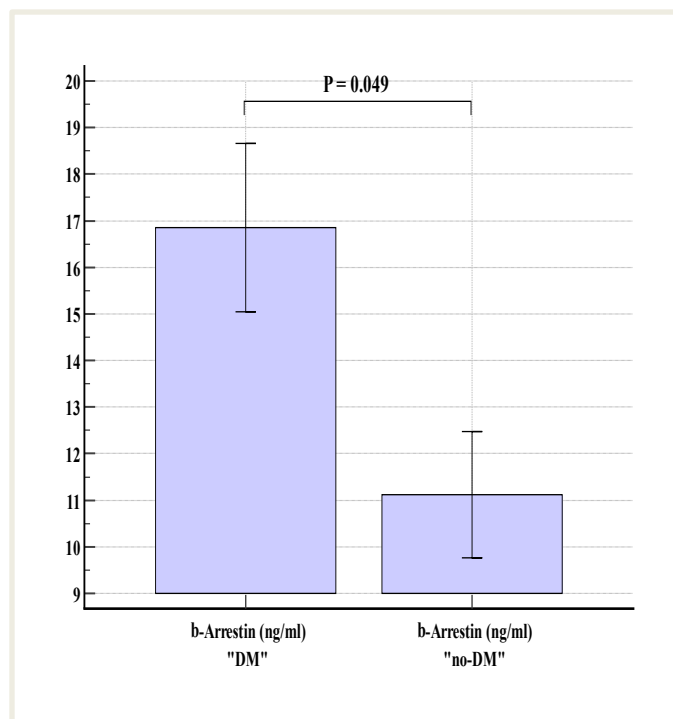


Figure 2. β -arrestin in acromegaly patient.

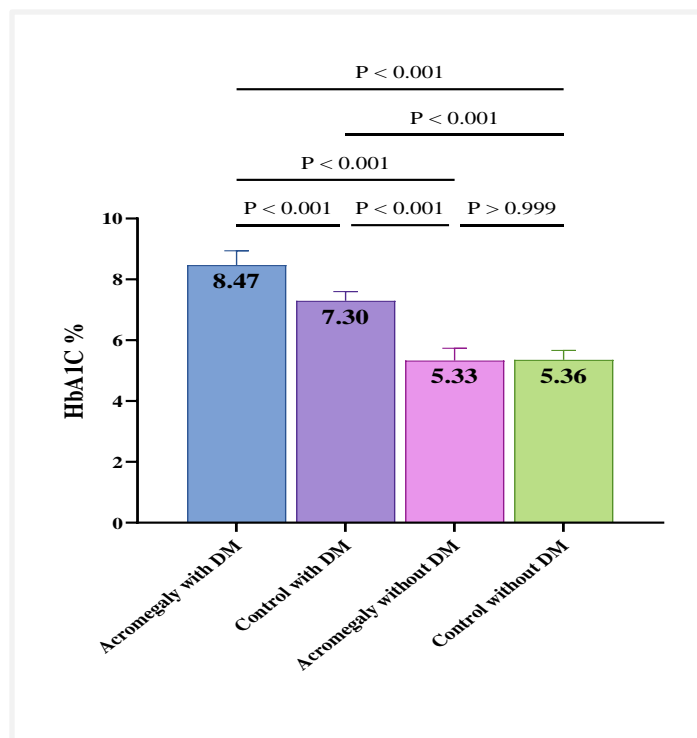


Figure 3. HbA1C distribution across the study groups.

Discussion

According to the results that related to the gender distribution in the study groups, it may indicate that gender may play a role in the association between diabetes and acromegaly, a condition known to affect metabolic processes, this significant gender imbalance in the DM group within the acromegaly patient may indicates a potential gender-related risk to the development of diabetes in acromegaly. This finding may suggest that females with acromegaly have an increased chance of metabolic problems, such as diabetes. Several variables may contribute to this, including hormonal impacts, variations in body composition, or other gender-specific factors that increase insulin resistance in females with acromegaly. Furthermore, variations in lifestyle variables, genetic predisposition, or the pathophysiology of acromegaly between males and females may responsible for this gender differences, which is in line with (23). But the results that related to control group shows a more balanced gender distribution in both DM and

non-DM subgroups, this result suggests that in the absence of acromegaly, gender may have a lower impact on the onset of diabetes. The variation between acromegaly and control groups may suggest that acromegaly alters the typical gender-specific risk factors for diabetes. And according to the age distribution, middle-aged adults (41-50 years) comprised the largest subgroup across all categories and similar age distribution patterns between acromegaly and control groups, and no significant difference in age distribution between DM and non-DM groups, this indicates that acromegaly, diabetes mellitus (DM), and associated health issues in the study population may be more common identified within this age group. The age distribution pattern is consistent in both the acromegaly and control groups, indicating that acromegaly in our sample is not age-dependent and affects individuals throughout a wide age range, similarly to the general population. The results that related β -arrestin, it suggests that β -arrestin levels may correlate with diabetes mellitus status in patients with

acromegaly, with higher levels possibly suggesting an exacerbated metabolic disruption caused by diabetes. Since β -arrestin is a protein that participates in cell signaling pathways which may affect insulin resistance and glucose metabolism, and in acromegaly, where the permanent elevation of the level of GH and IGF-1 already alter metabolic balance, the increase in the β -arrestin levels might contribute to an additional effect, which could contribute to insulin resistance and glucose dysregulation, this may elucidate the significant elevation of β -arrestin levels in acromegaly patients with diabetes mellitus compared to those without the condition. The explanation for that is elevated levels of β -arrestin are frequently a result of the combined impacts of excess growth hormone (GH) and IGF-1 that observed in acromegaly, which overstimulate cell signaling pathways regulated by β -arrestin, and the inflammatory condition and insulin resistance that present in these patients lead to amplify this effect, alongside β -arrestin providing a crucial function in regulating insulin signaling and addressing metabolic stress. So, the presence of these interconnecting conditions requires an increased demand for β -arrestin, leading to elevated levels compared to patients with only diabetes mellitus or healthy individuals, these findings are consistent with findings conducted by Al-Hakeim (24), in addition the study by Anderson demonstrates that the Increase in plasma β -arrestin levels may be attributed to the leakage of β -arrestin from the tissues (25), and according to the results that related HbA1c, the elevation in the level of HbA1c in patients with acromegaly and diabetes may suggest poor glycemic control, and this may be attributable to distinct physiological changes linked to acromegaly, that include insulin resistance and modified glucose metabolism. In acromegaly, excess growth hormone enhances gluconeogenesis and lipolysis, that may lead to complicate

hyperglycemia, while the increase in the level of IGF-1 may lead to additional impairment of glucose regulation, which is in line with (26). The result of serum level of IGF-1 suggesting that diabetes status does not significantly impact IGF-1 levels in acromegaly, these findings indicate that increased IGF-1 levels are significantly correlated with acromegaly, despite diabetes mellitus status. This correlates with the pathophysiology of acromegaly, in which excessive growth hormone (GH) promotes higher IGF-1 expression. The elevated level of IGF-1 certainly contributes to several metabolic effects observed in acromegaly, including insulin resistance, which may increase the chance of developing diabetes mellitus. Interestingly, the similar IGF-1 levels in acromegaly patients with and without DM suggest that high IGF-1 alone may not fully account for the onset of DM in these patients. Other factors, such as disease duration, lifestyle, genetic predispositions, or the degree of insulin resistance, may also influence DM development in acromegaly, these findings were in line with (27). The results of the serum level of GH indicate that the elevated level of GH is the hallmark of acromegaly patient, and the similarity between the two acromegaly groups suggests that diabetes status does not significantly affect GH levels in acromegaly, and the significant difference in GH levels between acromegaly and control groups shows the importance of GH as a critical diagnostic and monitoring indicator in the treatment of acromegaly. Consistent GH monitoring in acromegaly patients, in conjunction with IGF-1, may be beneficial for evaluating disease activity and recognizing individuals at high metabolic risk.

Conclusion

High prevalence of macroadenoma (>1 cm) observed in 74% of acromegaly patients emphasizes the commonality of larger pituitary adenomas in this patient population, which may have implications for disease severity and management. High prevalence of

diabetes in female acromegaly patients. The serum levels of β -arrestin were found to be elevated in patients with acromegaly, higher levels observed in those with DM compared to non-DM acromegaly patients and control groups. The patient with acromegaly and diabetes mellitus exhibit high HbA1C compared to diabetic control group. Prospective studies can help to understand how serum β -arrestin levels change over time, especially before and after treatments. This could show whether Filamin A levels are linked to the progression or improvement of the disease. Studying β -arrestin levels in other pituitary disorders, like Cushing's disease or non-functioning pituitary adenomas, could help to understand if these changes are specific to acromegaly or part of a broader issue with pituitary function.

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Ethical clearance: The study was conducted after obtaining approval from the scientific committees in the national diabetes center and college of Medicine Mustansiriyah University and in accordance with the ethical guidelines of the Declaration of ethical committee of the College (document no. 1297, date: 7/1/2024), and the objective of the study were explained to the patients and their consent was obtained before starting the blood draw procedure and completing the study.

Conflict of interest: None.

Use of Artificial Intelligence (AI): The authors state they did not use any generative AI tools for creating or editing the manuscript's language.

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تقييم مستوى بيتا-أريستين في مصل الدم لدى مرضى ضخامة الأطراف المصابين بداء السكري

^١ هبة غسان رجب، ^١ شذى الخطيب، ^٢ عباس رحمة

الملخص

الخلفية: ضخامة الأطراف اضطراب غدي نادر، حيث يبلغ معدل الإصابة السنوي ٤ حالات لكل مليون نسمة، ومعدل الانتشار ٤٠ حالة لكل مليون نسمة. وينتج هذا المرض عن ورم غدي في الغدة النخامية يُفرز هرمون النمو.

الأهداف: تهدف هذه الدراسة إلى تقييم مستويات بروتين بيتا-أريستين لدى مرضى ضخامة الأطراف، ومقارنة المرضى المصابين بداء السكري وغير المصابين به مع مجموعة ضابطة.

المرضى والطرق: هذه دراسة حالة-ضابطة شملت ١٠٠ مريض بضخامة الأطراف مسجلين في المركز القومي للسكري بجامعة المستنصرية، و٧٦ شخصًا من المجموعة الضابطة. وقد وافق جميع المشاركين شفهيًا على المشاركة في الدراسة، التي أُجريت في الفترة من فبراير إلى أغسطس ٢٠٢٤.

النتائج: لم تكن فروق توزيع الأعمار ذات دلالة إحصائية ($p=0.156$). أما توزيع الجنس، فقد أظهر ٤٧ أنثى و٥٣ ذكرًا في مجموعة ضخامة الأطراف. كان مستوى بروتين بيتا-أريستين أعلى لدى المرضى مقارنةً بالمجموعة الضابطة، وسُجّل أعلى مستوى له لدى المصابات بداء السكري المصاحب لضخامة الأطراف ($16,86 \pm 6,35$ نانوغرام/مل)، يليه المصابات بضخامة الأطراف غير المصاحبة لداء السكري ($11,12 \pm 4,78$ نانوغرام/مل)، ثم المجموعة الضابطة المصابة بداء السكري ($6,62 \pm 2,83$ نانوغرام/مل)، وأخيرًا المجموعة الضابطة غير المصابة بداء السكري ($2,93 \pm 0,98$ نانوغرام/مل).

الاستنتاج: ارتفاع معدل انتشار داء السكري بين الإناث المصابات بضخامة الأطراف. لوحظ ارتفاع مستوى بروتين بيتا-أريستين في مصل الدم لدى المصابات بضخامة الأطراف، مع مستويات أعلى لدى المصابات بداء السكري مقارنةً بضخامة الأطراف غير المصاحبة لداء السكري والمجموعة الضابطة.

الكلمات المفتاحية: ضخامة الأطراف، بيتا-أريستين، ورم الغدة النخامية، داء السكري.

المؤلف المراسل: هبة غسان رجب

الايمل: Heba-ghassan@uomustansiriyah.edu.iq

تاريخ الاستلام: ٢٤ شباط ٢٠٢٥

تاريخ القبول: ٢٩ حزيران ٢٠٢٥

تاريخ النشر: ٢٥ نيسان ٢٠٢٦

^١ قسم الكيمياء الحيوية، كلية الطب، الجامعة المستنصرية، بغداد، العراق.

^٢ المركز الوطني للسكري، الجامعة المستنصرية، بغداد، العراق.