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Serum Asprosin level in acromegaly patient: does Diabetes has an impact on it?

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Abstract The anterior pituitary gland's overproduction of growth hormone is the cause of acromegaly. Chronic hyperglycemia brought on by abnormalities in insulin secretion or action is a hallmark of diabetes mellitus type 2, a group of metabolic disorders . Asprosin, a newly discovered adipocyte produced from profibrillin-1, promotes hepatic glucose release during fasting. Diabetic and pre-diabetic patients show increased asprosin levels, whereas acromegaly patients exhibit significantly lower serum asprosin levels. Estimating the levels of acromegaly and its relationship to the other parameters is the aim of the current investigation. The National Diabetic Center at Mustansyrriah University recruited sixty patients (31 with diabetic acromegaly and 29 with acromegaly without diabetes), and thirty control groups (diabetic without acromegaly) were examined there for the biochemical study's criteria. Biochemical indicators such as asprosin, GH, IGF-1, responsiveness to octreotide, and LDL and triglyceride levels were altered in acromegalic patients. According to the current study, asprosin levels are higher in patients with acromegaly than in those without the disease. This is true whether or not a patient has acromegaly. Patients with acromegaly had higher levels of growth hormone and IGF-1, and non-diabetic acromegaly responds better to octreotide than diabetic acromegaly. Diabetes non-acromegaly was reported to have lower LDL and triglyceride levels than diabetic patient with acromegaly.

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Keywords: *Acromegaly; asprosin; Growth hormone; Insulin like growth factor-1; response to octreotide; LDL ; triglyceride levels*

1. INTRODUCTION

The chronic overproduction of growth hormone (GH) is the hallmark of acromegaly, a disorder that leads to insulin resistance, glucose intolerance, and eventually diabetes (1). In addition to aiding in the decrease of lipid buildup, the long-term stimulation of lipolysis by GH is essential for the development of insulin resistance and prediabetes/diabetes (2). Because of this, acromegaly is a unique instance of extreme insulin resistance combined with decreased body fat. When the improved function of pancreatic beta cells is insufficient to offset the ongoing insulin resistance, diabetes frequently appears as a late consequence as a result of impaired glucose metabolism in acromegaly (3). Twenty to thirty-five percent of people with acromegaly have diabetes at the time of diagnosis (4). Additionally, higher levels of (IGF-1) in diabetic patients than in those with normal glucose metabolism or impaired glucose tolerance suggest that diabetes is linked to disease control in acromegaly (5). Asprosin is adipokine that belongs to the caudamin subclass of protein hormones(6). It is synthesized from the C-terminal cleavage of profibrillin-1, a precursor protein encoded by the FBN1 gene on chromosome 15q21.1 (7).Asprosin is primarily secreted by white adipose tissue (WAT), which is a significant source of various adipokines(8). There are elevated amounts of asprosin. in conditions such as type 2 diabetes mellitus (T2DM) and obesity, highlighting its association with insulin resistance(9). It acts by stimulating hepatic glucose release and activating appetite-promoting neurons in the hypothalamus. Interestingly, Patients with acromegaly had reduced serum asprosin levels , perhaps as a result of how elevated growth hormone levels affect the production of adipokines and the function of adipose tissue (10). Thus, understanding asprosin's role could provide insights into the metabolic dysregulation observed in acromegaly and related disorders(11).

2. Materials and Methods

2.1 Patient's selection

Ninety respondents' medical records were enrolled in this study between October 2023 and March 2024. This study included 60 acromegaly patients who were registered at Mussansiriyah University's National Diabetes Center (NDC). They were separated into two categories: (29) non-diabetic acromegaly and (31) diabetic with acromegaly. Thirty diabetic patients without acromegaly made up the control group. As determined by the responsible endocrinologist, all enrolled patients with acromegaly were receiving monthly injectable

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long-acting Octreotide (20 or 30 mg). Age, age at onset, length of acromegaly, pituitary adenoma size, octreotide response using adenoma size regression , decrease in serum IGF-1 level to normal age and gender matched level, and reduction in serum GH level to less than 2.5 ng/ml, which satisfies the criteria for controlling acromegaly, are all included in the study. Serum GH and IGF-1 were measured using the chemiluminescence immune assay (CLIA), asprosin was measured using the ELISA, and (HbA1c) was measured using a point-of-care device (DCA vantage) in order to determine any relationship with the factors listed above and to look for any signs of diabetes in the data. Additionally, the lipid profile (HDL, LDL, VLDL, TG, and cholesterol) is measured by biochemical analysers.

The **Inclusion criteria:** acromegaly patients that diagnosed and were on Octreotide monthly injection for at least for one-year duration.(30-70) years in age

Exclusion Criteria: Smoking, History of renal impairment, Presence of cardio metabolic disorder, pregnant women

Statistical analysis. In order to determine whether there are any differences between the patients and control group, the variables under study are arranged in a master table that is beautifully created and prepared for (statistical package for social sciences version 27). The patients' biochemical characteristics were examined based on their age and gender. All of the participants in the study were separated based on their body weight, length of acromegaly, and utilization of Gamma-knife therapy. GH, IGF1, glycated Hb, and any variations between the patient and control groups. The relaxant statistical test is used to examine the correlation between Asprosin, GH, and IGF1 and pateint age, weight, and duration.

3. RESULTS

The baseline characteristics of the patients (diabetic and non-diabetic acromegaly) and control group (diabetic non-acromegaly) are displayed in Table 1. Asprosin levels are lower in the sick group than in the control group. The patient group's asprosin level is 16.41 ± 4.79, while the control group's is 17.32 ± 9.35. Acromegalic patients' (FBG) is 138 ± 40.57, while controls' is 181.73 ± 38.48; the difference is extremely significant (p value <0.001). (IGF-1) is 498.43 ± 234.94 in patients and 210.37 ± 39.22 in controls; the difference is highly significant <0.001.(GH) is 6.90 ± 13.96 in acromegalic patients and 1.43 ± 0.63 in the control group. LDL is 132.14 ± 38.35 in acromegalic patients and 93.03 ± 18.22 in the control group. The distinction is quite important. p <0.001.and the control group's triglyceride is 144.13 ± 14.96, whereas the acromegalic patient's is 196.53 ± 45.01. With a p value less than 0.001, the difference is extremely significant.

Table 1: *Comparison of patients with acromegaly(with and without diabetes with patients with diabetes (without acromegaly)*

	Acromegaly(with and without DM)	Diabetes Mellitus(without acromegaly)	P value
Number	60	30	
Male/Female	32/28	18/12	
Age (Years)	50.87±11.94	52.07±11.56	0.651
Weight (Kg)	94.35±11.90	80.27±16.27	<0.001
FBG	138.00±40.57	181.73±38.48	<0.001
HBA1C	6.73±1.53	9.00±1.70	<0.001
Asprosin	16.41±4.79	17.32±9.35	0.538
High density lipoprotein	39.15±6.33	42.73±2.75	<0.001
Low density lipoprotein	132.14±38.35	93.03±18.22	<0.001
Very low density lipoprotein	37.81±8.37	33.87±4.32	0.018
Cholesterol	225.15±47.50	199.67±21.61	0.006
Triglyceride	196.53±45.01	144.13±14.96	<0.001
GH	6.90±13.96	1.43±0.63	0.035
IGF-1	498.43±234.94	210.37±39.22	< 0.001

The baseline characteristics of the pateints with diabetic acromegaly and the control group with diabetic non-acromegaly are displayed in Table 2. Asprosin levels are lower in the sick group than in the control group. The patient group's asprosin level is 16.49 ± 3.35, while the control group's is 17.32 ± 9.35. Acromegalic individuals have a fasting blood glucose (FBG) of 161.74 ± 40.40, while controls have an FBG of 181.73±38.48. In acromegaly diabetic patients, growth hormone (GH) is 7.51 ± 13.54, while in the control group it is 1.43 ± 0.63. Insulin-like growth factor-1 (IGF-1) is 524.13 ± 249.89 in patients and 210.37 ± 39.22 in controls; the difference is highly significant <0.001. LDL is 146.01 ± 24.81

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in acromegaly patients and 93.03 ± 18.22 in the control group. The distinction is quite important. p <0.001. Additionally, the triglyceride levels in the control group are 144.13 ± 14.96 and the acromegalic patient is high at 196.53 ± 45.01; the difference is extremely significant (p value <0.001).

***Table 2**: Comparison of biological markers between diabetic patients with acromegaly and those with diabetes but without acromegaly*

	Acromegaly with Diabetes		Diabetes		
	Mean	SD	Mean	SD	P Value
Age	54.45	12.07	52.07	11.56	0.434
Weight	95.97	9.81	80.27	16.27	<0.001
GH	7.51	13.54	1.43	0.63	0.017
IGF1	524.13	249.89	210.37	39.22	<0.001
Asprosin	16.49	3.35	17.32	9.35	0.644
HbA1c	7.85	1.29	9.00	1.70	0.004
FBG	161.74	42.40	181.73	38.48	0.005
High density lipoprotein	41.10	5.79	42.73	2.75	0.164
low density lipoprotein	146.01	24.81	93.03	18.22	< 0.001
Very low density lipoprotein	40.02	8.37	33.87	4.32	< 0.001
Cholesterol	248.16	36.61	199.67	21.61	< 0.001
TG	212.65	40.57	144.13	14.96	< 0.001

Result in table 3 showed that : One third of the enrolled subjects were harboring microadenoma and the remaining two thirds were harboring macroadenoma and the presence of diabetic is not reflected to the type of harbored adenoma (p= 1.0844).

Regarding type of current therapy, all of patients were using Octreotide injections monthly, some of them have story of hypophysectomy or γ-knife radiosurgery or both.

It was discovered that the response was better in non-diabetic acromegalic compared to acromegaly with diabetes. The percentage of respondents was 75.9 in non-diabetic acromegaly and 54.8% in diabetic acromegaly; therefore, the non-respondents were 24.1% in non-diabetic acromegaly and 45.2% in diabetic acromegaly. The p value was 0.0031, suggesting that non-diabetic acromegaly had a higher probability of adenoma regression in response to octreotide than diabetic acromegaly. Diabetes is observed to have a negative effect on the response to octreotide because it blunts the response by causing adenoma regression. This is true for IGF-1 and GH, with p values for IGF-1 and GH being 0.0037 and 0.017, respectively.

***Table 3** Adenoma size, response of Long-acting repeatable octreotide (LAR) on adenoma regression and (IGF-1, GH) levels in acromegalic diabetics versus acromegalic non-diabetics.*

		Acromegaly (n=60)				*P* value
		Diabetics (n=31)		Non-Diabetics (n=29)		
		No	%	No	%	
Size of adenoma	**Microadenoma**	7	22.6	4	13.8	1.0844
	Macroadenoma	24	77.4	25	86.2	
Response of adenoma	**No response**	14	45.2	7	24.1	0.0031
	Response	17	54.8	22	75.9	
Response of IGF1	**No response**	19	61.3	7	24.1	0.0037
	Response	12	38.7	22	75.9	
Response of GH	**No response**	19	61.3	8	27.6	0.0170
	Response	12	38.7	21	72.4	
Statistical analyses were done using independent sample t-test. The P ≤ 0.05 considered to be significant.						

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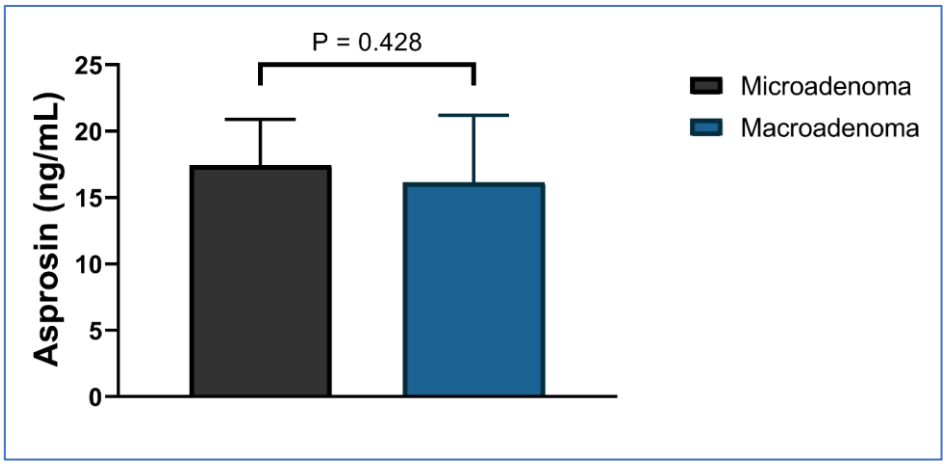

Figure 1: *The comparison between the levels of Asprosin in acromegaly patients with microadenoma and patients with macroadenoma. Independent sample T- test. With P value ≤ 0.05 considered to be significant.*

It was found that asprosin value is higher in microadenoma than in macroadenoma with p value = 0.428.

4. DISCUSSION

For this investigation, we measured the asprosin levels in the blood of 60 patients and 30 control participants. According to the findings, those with type 2 diabetes who did not have acromegaly had greater levels, which is consistent with Gong et al (12). In line with Farrag et al., they were also more prevalent in acromegalic diabetic patients than in acromegalic non-diabetic individuals; however, the difference was not statistically significant (13). Furthermore, regardless of whether they had acromegaly or not, diabetics had greater serum asprosin levels, which is consistent with Alsajri et al. (14). Additionally, we discovered that the blood level of asprosin is higher in microadenoma than macroadenoma in individuals with acromegaly P (value = 0.428). This finding could be attributed to a number of reasons pertaining to tumour biology, hormone regulation, and metabolic status. According to Mishra et al. (15), diabetic individuals without acromegaly had lower levels of LDL, VLDL, cholesterol, and TG than those with acromegaly. This is likely because diabetic patients without acromegaly are more compliant with statin treatment. According to this study, individuals with acromegaly have greater amounts of IGF-1 and growth hormones than those without the condition. This result is consistent with the Aggarwal et al. study's findings (16). Additionally, this is consistent with the Varaldo et al. study's findings (17). Glucose intolerance results from insulin resistance in the liver and peripheral tissues, which is brought on by an increase in GH levels. Acromegaly patients showed hyperglycemia, elevated fasting blood sugar, and elevated blood sugar two hours after the glucose tolerance test, and a significant difference in HbA1c between the acromegaly and control groups (diabetic non-acromegaly). Additionally, the percentage of respondents who had non-diabetic acromegaly was 75.9 percent, while the percentage of non-diabetic acromegaly respondents was 54.8%. The non-respondents had non-diabetic acromegaly at 24.1% and diabetic acromegaly at 45.2%, with the p value being 0.0031. Therefore, non-diabetic acromegaly is thought to have a higher possibility than diabetic acromegaly of adenoma regression in response to octreotide. In line with MacFarlane et al. (18) and Ahmed et al. (19), the presence of diabetes is found to have a negative impact on the response to octreotide by blunting the respons by adenoma regression to octreotide. This is also true for IGF-1 and GH, with p values for IGF-1 and GH = 0.0037 and 0.017, respectively.

5. CONCLUSION

Out of the 270 patients registered at the National Diabetic Centre (NDC), sixty (60) patients with acromegaly were chosen.30 individuals with non acromegaly diabetes were chosen to serve as a pathological control group. Of the 60 acromegaly individuals who were purposefully chosen, 31 have diabetes (50% have a HbA1c < 7). Hypophysectomy lowers the risk of developing diabetes. Serum asprosin levels in diabetic non-acromegaly participants rise more than in those with diabetes acromegaly. Additionally, the asprosin level was lower in comparable study groups (diabetic and non-diabetic) than in the group (diabetic without acromegaly). This suggests that the presence of either acromegaly, diabetes, or both has no effect on asprosin levels (p≥0.05).Instead of acromegaly, the diabetic is the primary player in this marker (asprosin). Octreotide users who do not have a hypophysectomy are more likely to have poorly managed diabetes. The risk of developing diabetes is unrelated to the kind of pituitary adenoma

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