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The Effect of Visfatin Levels on Blood Pressure and Cardiac Risk Factors in Patients with Acromegaly

Ömer Faruk Kuzu[1], Emre Hoca[2], Evrim Çakır[3], Hayriye Esra Ataoğlu[2]

[1]Çankırı State Hospital, Clinic of Medical Oncology, Çankırı, Türkiye
[2]University of Health Sciences Türkiye, Haseki Training and Research Hospital, Clinic of Internal Medicine, İstanbul, Türkiye
[3]University of Health Sciences Türkiye, Taksim Training and Research Hospital, Clinic of Endocrinology, İstanbul, Türkiye

ABSTRACT

Introduction: Acromegaly is a hormonal disorder characterized by gradual somatic changes that influence multiple bodily organs and systems. Cardiovascular complications are the primary cause of mortality in individuals with this condition. Visfatin is one of the adipocytokines whose role in obesity and metabolic diseases is controversial. This study aimed to investigate the correlation between visfatin levels and various laboratory and cardiometabolic parameters in patients diagnosed with acromegaly.

Methods: The study included 28 patients diagnosed with acromegaly, forming the patient group. A control group of 27 patients, without a history of chronic disease, was also enrolled from the patient population attending our hospital. Echocardiographic findings and 24-hour blood pressure holter results of all participants were recorded. Blood samples were obtained from the control group and the patients.

Results: Patients with acromegaly exhibited a higher proportion of females, fasting blood glucose levels, growth factor-1 levels, and left ventricular end-diastolic diameter compared to the control group. Conversely, visfatin levels were found to be lower in acromegaly patients. No correlation was identified between visfatin levels and blood pressure or other biochemical parameters in these patients. However, a positive correlation was observed specifically between left atrium diameter and visfatin levels.

Conclusion: Our study revealed that only a limited number of parameters showed a significant association with visfatin levels. Further research with a larger patient cohort is necessary to confirm these findings.

Keywords: Acromegaly, blood pressure, cardiovascular complications, visfatin

Introduction

Acromegaly is an endocrine disease with progressive somatic disorders affecting many organs and systems in the body (1). Cardiovascular system involvement, driven by elevated insulin-like growth factor-1 (IGF-1) and growth hormone (GH), is the leading cause of mortality in these patients, accounting for 60% of deaths (2). The remaining 25% of patients do not survive due to respiratory system problems, and 15% are lost due to malignancy. The most important determinant of mortality is the control of GH levels (3). The heightened risk of cardiovascular disease in acromegalic patients is linked to the frequent presence of atherosclerotic risk factors like hypertension and diabetes; however, cardiovascular involvement in acromegaly can manifest even in the absence of other risk factors (4). Cardiac hypertrophy and cardiomyopathy, directly or indirectly induced by high GH levels through IGF-1, are among the most prevalent cardiac complications in acromegaly patients (1,4). While heart abnormalities may emerge in the early stages of acromegaly, their prevalence tends to rise as patients grow older (5,6). At the time of diagnosis, echocardiographic left ventricular ejection fraction is often normal in the vast majority of patients (55-78%), but microscopic structural defects or subclinical echocardiographic findings are more likely to be present in these patients (7,8).

Adipose tissue serves various roles, including mechanical cushioning, heat production, energy and fat-soluble vitamin storage, and acting hormonally by releasing adipocytokines that regulate metabolism, eating behavior, insulin response, and inflammation (9-11). Visceral adipose tissue, located around major abdominal organs, holds greater significance than subcutaneous adipose tissue in the development of obesity-related conditions such as type 2 diabetes (T2D), cardiovascular pathologies, and diseases of the lung, liver, and kidneys, as well as cancer (11,12).

Adipocytes synthesize some adipocytokines, and some are synthesized by stromal-vascular adipose tissue components, such as preadipocytes, endothelial cells, lymphocytes, macrophages, and fibroblasts (9).

Address for Correspondence: Emre Hoca Asst. Prof., MD, University of Health Sciences Türkiye, Haseki Training and Research Hospital, Clinic of Internal Medicine, İstanbul, Türkiye
E-mail: emrehoca89@gmail.com **ORCID ID:** orcid.org/0000-0003-4232-7362

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Adipocytokines exert autocrine, paracrine, and endocrine effects on the body (13,14). Changes in adipose tissue disrupt the regulation mechanisms of adipocytokines and predispose individuals to many metabolic disorders such as inflammation of adipose tissue, insulin resistance, chronic systemic inflammation, and endothelial dysfunction (14,15). Visfatin belongs to the group of adipocytokines, though its involvement in metabolic conditions remains debated. Visfatin, secreted predominantly from visceral adipose tissue, exerts proinflammatory effects and activates multiple molecular signaling pathways, thereby contributing to endothelial dysfunction-an important early mechanism in the pathogenesis of atherosclerosis (16). These effects are thought to have significant implications for the development of cardiovascular diseases. Many studies have reported that visfatin levels are also associated with insulin resistance, diabetes and metabolic syndrome (17,18). Furthermore, some studies have shown that visfatin levels in patients with acromegaly may be associated with metabolic disorders, independent of the type of treatment (19).

In this study, we investigated how visfatin levels impact blood pressure, cardiac functions, and other clinical indicators in individuals with acromegaly.

Methods

Study Participants and Laboratory Evaluation

The study commenced following approval from the Ethics Committee of University of Health Sciences Türkiye, Haseki Training and Research Hospital (approval number: 388, date: 22.06.2016). Twenty-eight patients who were being followed up in the Endocrinology Outpatient Clinic of University of Health Sciences Türkiye, Haseki Training and Research Hospital with the diagnosis of acromegaly were included in our study by signing informed consent forms. The control group comprised 27 patients, free of chronic disease history, who presented to our hospital and signed informed consent forms. Detailed echocardiographic findings of the patient and control groups were recorded in consultation with cardiologists who have at least 10 years of echocardiographic experience, and the 24-hour blood pressure holter results of all participants were monitored. Blood samples for fasting blood glucose, insulin, alanine aminotransferase, creatinine, C-reactive protein (CRP), IGF-1, and visfatin were taken after fasting for at least 12 hours. First, the samples were stored at optimal room temperature for 30 minutes; then centrifuged at 4000 rpm for 10 minutes, and kept at -40 °C (for visfatin analysis) until the analysis day. Visfatin levels were determined by enzyme-linked immunosorbent assay (ELISA) method using Sun Red brand, (antibody-coated 96-well plate human visfatin, Shanghai, Sunred Biological Technology Co., China) ELISA kit, and absorbance values were determined on ELx800 (Biomedical Technologies Inc., USA) microplate reading device (Bio-Tek Instruments, INC.). Duration of medication use for acromegaly was recorded.

Statistical Analysis

IBM SPSS Statistics (version 25) software was used to analyze the data obtained in this study. Descriptive statistics are given as mean ± standard deviation, minimum and maximum and median. The normality of continuous variables was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests. For variables exhibiting normal distribution, the Independent Sample t-test was applied, while the Mann-Whitney U test was used for variables not conforming to a normal distribution. Categorical variables were presented as numbers and percentages (%), and the chi-square test or Fisher's exact test was used for intergroup comparisons when the expected cell count was <5. Pearson or Spearman correlation analyses were utilized to ascertain relationships between variables. Statistical significance was set at $p<0.05$. Comparative analyses of the related groups were performed to evaluate the relationship between biochemical parameters, cardiovascular measurements, and clinical data. The results were presented in tables, and statistically significant differences were indicated.

Results

When the groups were evaluated in terms of age, the mean age of the control group was 48.4±14.8 years, and for acromegaly patients, it was 45.0±9.7 years. No significant difference was observed between the two groups ($p=0.305$). The female sex ratio was lower in the control group than in acromegaly patients ($p=0.010$). Fasting glucose and IGF-1 values were higher in acromegaly patients ($p=0.001$ and $p<0.001$, respectively). Body mass index was statistically significantly higher in acromegaly patients compared to the control group ($p=0.034$). Regarding other anthropometric measurements, there was no significant difference between the control group and acromegaly patients, Table 1.

No significant difference was observed in blood pressure measurements between the two groups (Table 2).

The visfatin level was 17.8±29.3 ng/mL in the whole group (acromegaly + control). The median, minimum, and maximum values of visfatin were 9.0, 5.4, and 191.1, respectively. There was no difference in visfatin values between genders in acromegaly patients ($p=0.105$), but visfatin level was lower in acromegaly patients compared to the control group ($p=0.015$) (Table 3), (Graphic 1).

In the cardiological evaluations of acromegaly patients, the mean left ventricular end-diastolic diameter (LVEDD) was statistically significantly higher than that of the control group ($p<0.001$). In addition, the incidence of left ventricular hypertrophy was higher in patients with acromegaly compared to the control group ($p=0.048$) (Table 4).

No significant correlation was found between visfatin and IGF-1 levels ($p=0.060$). There was a positive correlation between visfatin levels and left atrium diameter ($p=0.023$). There was no correlation between visfatin levels and other demographic findings or laboratory findings. There was a positive correlation between IGF-1 and CRP and ejection fraction, and a negative correlation between IGF-1 and left ventricular end-systolic diameter ($p=0.002$, $p=0.026$, $p=0.043$, respectively) (Table 5).

No correlation was observed between visfatin levels, IGF-1 levels, and 24-hour blood pressure parameters (Table 6).

Table 1. Comparison of demographic data, anthropometric measurements and laboratory parameters between patients with acromegaly and control group

		Acromegaly		Control		
		n	%	n	%	p
Gender	Female	21	75	11	40.7	**0.010**
	Male	7	25	16	59.3	
		Mean ± SD	Min-max (median)	Mean ± SD	Min-max (median)	p
Age (years)		45±9.7		48.4±14.8		0.305
Disease duration (years)		8.7±6.1	0-25 (9)			
Sandostatin treatment duration (months)		23.0±29.2	0-84 (6)			
Somatuline treatment duration (months)		8.7±19.4	0-72 (0)			
Dostinex treatment duration (months)		21.3±24.3	0-78 (12)			
Height (cm)		163.6±8.5	148-185 (162)	167.7±11.0	150-186 (167)	0.131
Weight (kg)		80.4±12.4	56-100 (81)	79.2±13.3	50-100 (78)	0.729
BMI (kg/m^2)		29.8±4.3	23-37 (29)	27.5±5.7	19-43 (27.0)	0.094
Hip circumference (cm)		111.1±7.5	97-127 (110)	106.4±11.8	89-141 (104)	0.082
Waist circumference (cm)		99.7±10.8	82-117 (101.5)	97.8±13.5	74-133 (97)	0.574
Glucose (mg/dL)		118.1±35.3	77-216 (107.5)	96.4±11.6	76-130 (94)	**0.001**
Insulin (uIU/mL)		7.6±4.4	2.1-18.1 (6.4)	7.0±4.3	2.6-23.8 (6.41)	0.545
CRP (mg/L)		3.3±3.8	0.2-17.8 (2.1)	3.1±4.1	0.3-22.5 (2.1)	1.000
ALT (U/L)		23.1±24.8	8-144 (16)	24.9±20.3	7-113 (17)	0.425
Creatinine (mg/dL)		0.69±0.23	0.39-1.59 (0.65)	0.74±0.15	0.42-0.96 (0.71)	0.293
IGF-1 (ng/mL)		295.4±261.0	1.55-1142 (234)	118.5±45.4	35.6-215 (117)	**<0.001**
HOMA-IR		2.3±1.7	0.61-1.87 (1.88)	1.5±0.8	0.56-3.48 (1.39)	**0.034**

BMI: Body mass index, CRP: C-reactive protein, ALT: Alanine aminotransferase, IGF-1: Insulin-like growth factor 1, Min: Minimum, Max: Maximum, SD: Standard deviation, HOMA-IR: Homeostasis model assessment of insulin resistance

Table 2. Evaluation of blood pressures of acromegaly patients and control group

	Acromegaly		Control		
24-hour BP findings (mmHg)	Mean ± SD	Min-max (median)	Mean ± SD	Min-max (median)	p
Maximum systolic BP	154.9±25.6	113-213 (159)	159.5±25.7	109-204 (161)	0.482
Maximum diastolic BP	111.9±30.3	55-191 (106)	108.9±23.3	67-169 (108)	0.656
Minimum systolic BP	88.8±10.8	71-118 (87)	89.2±11.4	65-118 (90)	0.875
Minimum diastolic BP	51.9±10.3	40-82 (50)	55.9±13.1	30-78 (58)	0.190
Mean systolic BP	117.2±13.1	94-145 (113)	121.4±11.8	96-151 (120)	0.184
Mean diastolic BP	75.3±11.1	55-100 (75)	76.8±8.6	56-97 (77)	0.542

BP: Bood pressure, Min: Minimum, Max: Maximum, SD: Standard deviation

Table 3. Comparison of visfatin levels between acromegaly patients and control group

	Acromegaly		Control		
Visfatin (ng/mL)	Mean ± SD, (n)	p	Mean ± SD, (n)		
Male	11.09±8.42, (21)	0.105	12.41±12.71, (11)		
Female	12.69±16.56, (7)		32.52±49.49, (16)		
	Mean ± SD, (n)	Min-max (median)	Mean ± SD, (n)	Min-max (median)	p
All patients	11.5±10.7	5.4-50.2 (7.75)	24.3±39.7	5.7-191.1 (9.5)	**0.015**

Min: Minimum, Max: Maximum, SD: Standard deviation

Tablo 4. Evaluation of echocardiographic findings in acromegaly patients and control group

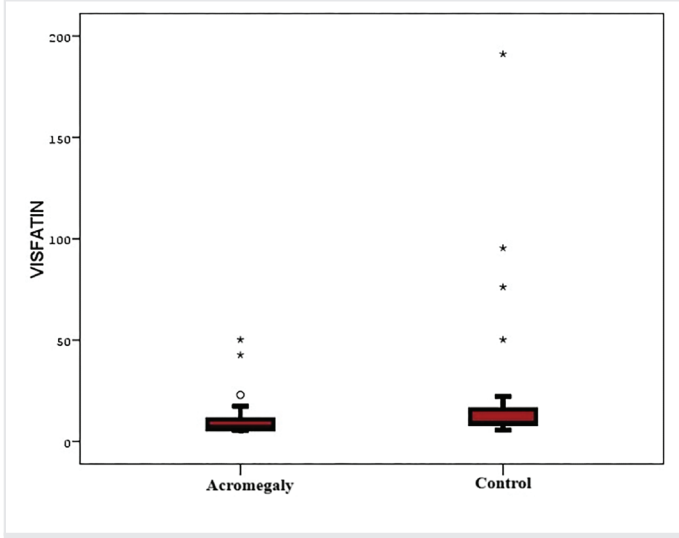
	Acromegaly		Control		
	Mean ± SD	Min-max (median)	Mean ± SD	Min-max (median)	p
Ejection fraction (%)	59.0±3.6	45-65 (60)	59.8±2.6	50-65 (60)	0.447
LVESD (mm)	38.5±5.4	27-50 (41)	36.5±7.5	24-50 (34)	0.197
LVEDD (mm)	51.0±3.4	45-60 (52)	45.3±7.4	30-52 (48)	**<0.001**
IVS thickness (mm)	11.5±0.9	9-13 (12)	11.1±1.1	9-13 (11)	0.169
PW thickness (mm)	11.1±0.9	8-12 (11)	10.8±1.0	8-12 (11)	0.173
	n	%	n	%	p
Left ventricular hypertrophy (+)	14	43.8	5	19.2	**0.048**

LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, IVS: Interventricular septum, PW: Posterior wall, Min: Minimum, Max: Maximum, SD: Standard deviation

Table 5. Association of visfatin and IGF-1 levels with other findings in patients with acromegaly

	Visfatin		IGF-1	
	rho	p	rho	p
IGF-1	-0.359	0.060		
Disease duration	-0.198	0.311	0.053	0.773
Sandostatin duration	-0.200	0.316	-0.025	0.895
Somatuline duration	-0.266	0.179	0.139	0.455
Dostinex duration	-0.332	0.091	0.104	0.577
Height	-0.335	0.082	0.104	0.571
Weight	-0.078	0.695	0.031	0.868
BMI	0.237	0.224	-0.128	0.486
Hip circumference	0.231	0.237	-0.112	0.542
Waist circumference	0.031	0.877	-0.102	0.578
Glucose	-0.021	0.914	0.278	0.124
Insulin	-0.099	0.623	0.341	0.061
CRP	0.069	0.727	-0.518	**0.002**
ALT	-0.117	0.570	0.004	0.984
Creatinine	-0.190	0.333	-0.128	0.484
Ejection fraction	0.330	0.086	-0.392	**0.026**
LVESD	-0.316	0.102	0.359	**0.043**
LVEDD	-0.241	0.217	0.337	0.059
IVS thickness	0.105	0.593	-0.247	0.173
PW thickness	0.004	0.983	-0.042	0.821
Left atrium diameter	0.427	**0.023**	-0.064	0.727
Right ventricle diameter	0.170	0.387	0.015	0.934

IGF-1: Insulin-like growth factor 1, BMI: Body mass index, CRP: C-reactive protein, ALT: Alanine aminotransferase, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, IVS: Interventricular septum, PW: Posterior wall



Graphic 1. Visfatin levels in acromegaly patients and control group

Table 6. Association of visfatin and IGF-1 levels with blood pressure values in patients with acromegaly

	Visfatin		IGF-1	
	rho	p	rho	p
Maximum systolic BP	0.321	0.110	-0.017	0.931
Maximum diastolic BP	0.352	0.078	-0.156	0.419
Minimum systolic BP	0.228	0.262	0.097	0.615
Minimum diastolic BP	0.059	0.777	0.192	0.319
Mean systolic BP	0.198	0.332	0.144	0.456
Mean diastolic BP	0.137	0.504	0.102	0.600

IGF-1: Insulin-like growth factor 1, BP: Bood pressure

Discussion

A limited number of studies in the existing literature suggest that serum visfatin levels might be linked to vascular inflammation, atherosclerosis, and increased vascular resistance. In our current investigation, we explored the relationship between visfatin levels and inflammatory markers, echocardiographic findings, and metabolic parameters.

Visfatin is an adipocytokine that is useful in demonstrating inflammation and endothelial damage. In a study conducted in Malaysia, circulating visfatin levels were found to be higher in metabolic syndrome, obesity and T2D (18). In a study conducted with acromegaly patients by Piskinpasa et al. (20), visfatin levels were associated with glycaemic dysregulation. Similarly, in the study conducted by Erten (21),

it was reported that visfatin levels may be predictive for metabolic risk. Conversely, Filippatos et al. (22) observed an increase in visfatin levels in patients with metabolic disorders compared to healthy controls. Some other studies, however, reported lower visfatin levels in patients with metabolic syndrome or obesity than in those without. The precise mechanism accounting for this variability in visfatin levels remains unclear (22).

Other studies found that visfatin levels were lower in patients with metabolic syndrome or obesity than in those without (23,24). However, the mechanism by which this variability in visfatin levels occurs has not yet been clearly elucidated.

In our study, visfatin levels were lower in patients with acromegaly compared with the control group. This may be because IGF-1 elevation leads to a decrease in the level of visfatin secreted from visceral adipose tissue by causing lipolysis in the long-term. The fact that the mean duration of acromegaly in the patients in our study was 8.7 years supports this long-term effect.

The cardiovascular effects of visfatin are multifaceted. A study by Lovren et al. (25) showed that visfatin had proangiogenic effects and was associated with increased endothelial nitric oxide synthesis, potentially conferring protective vascular effects. Conversely, Yu et al. (26) demonstrated that visfatin increased cardiac fibroblast proliferation and accelerated type 1 and type 3 collagen production in myocardial tissue, leading to myocardial fibrosis over time. This profibrotic effect may increase oxidative stress in cardiomyocytes and predispose cardiomyocytes to ischemic conditions and cardiac insufficiency.

Despite the known cardiovascular complications of acromegaly, including left ventricular hypertrophy (which was more prevalent in our patient group) and increased LVEDD, we found no statistically significant correlation between visfatin levels and most echocardiographic parameters. However, a notable positive correlation was observed between visfatin levels and left atrium diameter, suggesting a possible relationship between visfatin and atrial remodeling.

The lack of correlation between visfatin and most cardiovascular parameters may be explained by the fact that cardiovascular complications in acromegaly are primarily driven by direct GH/IGF-1 effects rather than adipocytokine-mediated mechanisms (1,4). Additionally, the cardiovascular side effects of visfatin occur mostly through inflammatory processes (15), and since our patients were predominantly treatment-controlled with suppressed disease activity, significant inflammation and associated cardiovascular changes may not have developed.

Our analysis of CRP levels as an inflammatory marker did not reveal a statistically significant relationship between CRP and visfatin levels in acromegaly patients. However, we found a negative correlation between IGF-1 and CRP levels, suggesting complex inflammatory regulation in this disease. Interestingly, there was no difference in CRP levels between acromegaly patients and controls, which may indicate that inflammatory processes were not prominently active in our treatment-controlled cohort.

Very few studies have evaluated the relationship between visfatin levels and metabolic parameters in patients with acromegaly. The prevalence of acromegaly varies between 40 and 70 per million. Therefore, there were only 28 patients with acromegaly in our hospital. This was the most significant limitation of our study. In addition, patients were receiving treatment for acromegaly, and most were under control. This may have prevented cardiovascular complications and possible metabolic changes from occurring. Our failure to find a clear relationship between visfatin levels and other parameters may be explained by these conditions. Therefore, studies with a larger number of patients, including untreated patients, and using follow-up data, may contribute to the literature.

Study Limitations

One main limitation was that there were only 28 patients with acromegaly in our hospital. In addition, since all patients were receiving treatment for acromegaly, cardiometabolic complications that typically develop secondary to acromegaly did not develop.

Conclusion

In such rare cases, evaluating the visfatin level with the predicted values may not be meaningful. More extensive studies are required to elucidate the significant effects of visfatin levels in more common diseases such as diabetes and hepatosteatosis. It would be more appropriate to perform such a study in treatment-naive patients.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Ethics Committee of University of Health Sciences Türkiye, Haseki Training and Research Hospital (approval number: 388, date: 22.06.2016).

Informed Consent: All patients received information regarding the study's details and provided written informed consent.

Footnotes

Authorship Contributions: Surgical and Medical Practices - E.Ç., H.E.A.; Concept - E.Ç., H.E.A.; Design - E.H., E.Ç., H.E.A.; Data Collection or Processing - Ö.F.K., E.H., H.E.A.; Analysis or Interpretation - E.H., E.Ç., H.E.A.; Literature Search - Ö.F.K., E.H.; Writing - Ö.F.K., E.H.

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