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ORIGINAL ARTICLE

Relationship Between Visfatin Level and Cardiovascular Changes in Hemodialysis Patients

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Abstract

Background: **Cardiovascular alterations such as atherosclerosis, calcification, heart failure, diastolic and systolic dysfunction, and stroke are the primary causes of mortality in people with chronic kidney disease (CKD) and end-stage renal disease (ESRD). Individuals with CKD have been demonstrated to have increased serum levels of visfatin. A new study suggests that high visfatin levels in individuals with CKD accurately predict cardiovascular abnormalities. Chronic renal failure, atherosclerosis, and calcification in the carotid arteries vary in individuals. The researchers set out to look at the correlation of visfatin level and cardiovascular abnormalities in CKD patients. In this case−control comparative study, conventional echocardiogram and carotid duplex assessment were used to evaluate the differences between 40 patients with stage III or stage IV CKD receiving conservative treatment, 40 ESRD patients receiving regular hemodialysis, and 20 participants as a control group.**

Results: **Hemodialysis-dependent carotid atherosclerosis, systolic and diastolic dysfunction, and calcifications are more common in ESRD patients compared with nonhemodialysis-dependent individuals that significantly correlated with high serum visfatin levels compared with healthy control participants. Carotid atherosclerosis, diastolic dysfunction, and systolic dysfunction are all more likely to occur in those with CKD who had increased carotid intima-media thickness ($P = 0.036$) of carotid Doppler, decreased EF % ($P = 0.032$), increased left ventricular mass index ($P = 0.004$), and reversed Early diastolic over late diastolic trans mitral flow (E/A) ratio ($P = 0.003$) demonstrated by echocardiographic and carotid Doppler assessment. Patients with an elevated visfatin level had increased cardiovascular morbidity and mortality and atherosclerosis of the carotid arteries with a high risk of ischemic stroke.**

Keywords: Cardiovascular, Chronic kidney disease, Hemodialysis, Visfatin

1. Introduction

Chronic renal failure increased in frequency in the last decades, making it a serious threat to public health. Early diagnosis, prediction, and slowing progression of chronic kidney disease (CKD) are essential to alter cardiovascular sequelae and decrease mortality. There is a wide variety of factors that might influence the decline in kidney function seen in early CKD, including systemic inflammation, oxidative stress, and endothelial dysfunction.[1]

Human plasma concentrations of the extracellular protein visfatin (eNAMPT) extracellular nicotinamide phosphoribosyl transferase were measured at 10−282 ng/ml. Adipocytokines were associated with detectable levels of eNAMPT in their supernatants. However, data on eNAMPT's abundance compared with its intracellular counterpart is scant. The extracellular version accounts for around 1% of the total NAMPT, according to the lone research on the patient.[2]

A higher risk of dying from cardiovascular causes is correlated with elevated visfatin levels in those

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with chronic renal failure. There was a rise in visfatin levels in atherosclerotic patients.[3]

Patients with uremia have been reported to have elevated levels of visfatin, which has been linked to endothelial dysfunction. However, visfatin itself may provide some cellular protection. The availability of energy, cellular function, and viability are all profoundly affected by visfatin activity. In mice, visfatin has been shown to have direct cardioprotective benefits. Because of this, secondary variables like raised resistin or reduced high-density lipoprotein (HDL) cholesterol may be responsible for most of the negative consequences seen with elevated visfatin levels. As a result, it is quite doubtful that visfatin's preventive properties will be enough to counteract these dangers.[4]

2. Participants and methods

This study was a case–control comparative study conducted in Al Hussein University Hospital. One hundred participants were studied: 40 CKD patients of stage III and stage IV on conservative medical treatment, 40 patients on regular hemodialysis for more than 1 year, and 20 apparently healthy controls matched for age and sex with previous groups; all participants agreed and signed a consent.

2.1. Inclusion criteria

Patients in stages III and IV of CKD received conservative medical therapy, and those who have been on regular hemodialysis for at least a year due to end-stage renal disease (ESRD).

2.2. Exclusion criteria

Patients under the age of 18 years and those above the age of 60 years, individuals with rheumatic congenital, valvular heart disorders, ischemic heart disease at rest, any serious heart rhythm disorder or pericardial illness, inflammatory bowel disease, rheumatoid arthritis, acute or chronic inflammatory conditions, and CKD stage I or II.

2.3. Methods

Full medical history, physical examination, and lab testing, including a full blood count, C-reactive protein, hemoglobin A1C, lipid profile, total serum calcium, phosphorus, and parathyroid hormone were performed on all patients. Serum visfatin levels were evaluated using an enzyme-linked immunosorbent assay immunoassay.

To evaluate carotid intima-media thickness (CIMT), peak systolic velocity (PSV), atheromatous plaques, systolic and diastolic dysfunction, left ventricular mass (LVM), left ventricular mass index (LVMI), left ventricular ejection fraction (LVEF), Interventricular septal thickness (IVST), Left ventricular posterior wall thickness (LVPWT), and Early diastolic over late diastolic trans mitral flow (E/A) ratio, carotid Doppler studies, and conventional echocardiographic assessment were performed on all participants. Each patient who will have an operation has given their written informed consent for that procedure. Everything was done in accordance with the rules set out by the Ethics Committee of Al-Azhar University.

Data analysis was performed using SPSS, Statistical Package for the Social Sciences, version 24, USA. Quantitative data was given as frequency and percentage, whereas qualitative data was provided as an open text. When looking at a set of numbers, the average is the number that falls in the center. It is determined by dividing the total number of numbers in the set by the sum. The SD quantifies the degree to which a set of numbers differs from the mean (SD). A smaller SD indicates that the values are more closely clustered around the mean, as opposed to the opposite, which would be the case if the SD were larger. The correlation strength between both data sets was assessed using Pearson's correlation coefficient (r).

3. Results

There is an increase in serum visfatin in CRF patients undergoing maintenance hemodialysis; however, we recommend further research into the correlation between serum visfatin and the various factors influencing cardiovascular changes, as well as the study of serum visfatin before and after hemodialysis. Determining the causal relationship between visfatin in CRF patients and cardiovascular changes, as well as studying visfatin expression within renal tissues, may clarify its definite CVS complications in CKD and be best accomplished through serial measurements taken at the onset of CKD and again during progressively declining stages of renal dysfunction.

Basic demographic data ($N = 100$) showed no significant difference between groups in terms of age, sex, BMI, duration of kidney disease (groups 1 and 2 only), and primary renal disease (groups 1 and 2 only) ($P = 0.05$) (Table 1).

Laboratory measurements ($N = 100$) show a statistically significant positive correlation between

Table 1. Comparison of Demographic data between CKD, HD and control group.

	CKD ($N = 40$)	HD ($N = 40$)	Control ($N = 20$)	P value
Age, years				0.753*
Mean ± SD	44.2 ± 5.6	45.3 ± 6.6	43.9 ± 2.4	
Range	30–60	32–55	31–57	
Sex				0.073**
Male	18 (45)	16 (40)	10 (50)	
Female	22 (55)	24 (60)	10 (50)	
BMI, kg/m^2				0.195*
Mean ± SD	37.3 ± 2.9	37 ± 3.1	36.8 ± 3.3	
Range	31.5–44.2	32–45	31–43	
Duration, years				0.089***
Mean ± SD	4.6 ± 1.0	5.1 ± 1.2	–	
Range	5–10	6–12	–	
Primary renal disease				0.467**
Unknown	10 (25)	9 (22.5)	–	
DN	12 (30)	10 (25)	–	
HN	7 (17.5)	9 (22.5)	–	
Postrenal	5 (12.5)	4 (10)	–	
GN	4 (10)	5 (12.5)	–	
ADPKD	2 (5)	1 (2.5)	–	

CKD, chronic kidney disease; ADPKD, Adult polycystic kidney disease; GN; Glomerulonephritis; DN, Diabetic nephropathy; HD, Hemodialysis.

visfatin levels in the three groups and hs-C-reactive protein (CRP), glycated hemoglobin (HbA1c), triglycerides (TGs), total cholesterol, LDL, urea, and creatinine. However, a statistically significant negative correlation was found between visfatin levels in the three groups and HDL cholesterol. Neither group found a significant relation between visfatin levels and hemoglobin, calcium, phosphorus, and Parathyroid hormone (PTH) levels (Table 2).

Correlation between visfatin levels and laboratory parameters ($N = 100$) shows a statistically significant positive correlation between visfatin levels in the three groups and CRP, HbA1c, TGs, total cholesterol, LDL, urea, and creatinine. However, there was a significant negative relationship between HDL cholesterol and visfatin levels in the three groups. Hemoglobin, calcium, phosphorus, and PTH levels were not significantly correlated with visfatin levels in either group (Table 3).

Echocardiographic measurements ($N = 100$) showed a statistically significant positive correlation between visfatin levels in the three groups and LVM, LVMI, IVST, LVPWT, and E/A. However, a statistically significant negative correlation was found between visfatin levels in the three groups and LVEF and fractional shortening (FS). In addition, by running Spearman's correlation analysis, a significant association was found between visfatin levels and the development of cardiac calcifications (Table 4).

Correlation between visfatin levels and echocardiographic parameters ($N = 100$) shows a

Table 2. Comparison of laboratory data between CKD, HD and control group.

	CKD ($N = 40$)	HD ($N = 40$)	Control ($N = 20$)	P value
Visfatin, ng/ml	30.2 ± 4.5	37.3 ± 9.1	23.2 ± 5.6	0.001
CRP, mg/l	20.4 ± 3.3	30.5 ± 5.8	2.5 ± 1.1	0.003
Hemoglobin, g/dl	10.6 ± 1.3	10.4 ± 2.5	11.6 ± 1.7	0.021
HbA1c, %	7.7 ± 1.3	8.1 ± 1.5	6.2 ± 0.5	0.043
TG, mg/dl	155 ± 20	206 ± 29	118 ± 23	0.002
Cholesterol, mg/dl	160 ± 34	162 ± 37	145 ± 35	0.048
LDL, mg/dl	88 ± 30	97 ± 32	70 ± 15	0.033
HDL, mg/dl	40 ± 6	32 ± 4	54 ± 7	0.028
Calcium, mg/dl	9.5 ± 0.8	10.2 ± 0.9	9.1 ± 0.5	0.047
Phosphorus, mg/dl	4.1 ± 0.4	3.0 ± 1.2	4.5 ± 0.7	0.032
Intact PTH, pg/ml	170 ± 57	197 ± 50	40 ± 12	0.000
Urea, mg/dl	70.5 ± 33.2	94.2 ± 29.6	33.5 ± 8.4	0.006
Creatinine, mg/dl	5.6 ± 1.3	9.6 ± 2.1	0.7 ± 0.3	0.001

CKD, chronic kidney disease; CRP, C-reactive protein; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglycerides.

Table 3. Correlation between visfatin level and laboratory parameters in CKD, HD and control group.

	CKD ($N = 40$)		HD ($N = 40$)		Control ($N = 20$)	
	r	*P* value	*r*	*P* value	*r*	*P* value
CRP, mg/l	0.33	0.005	0.25	0.004	0.30	0.001
Hemoglobin, g/dl	−0.04	0.875	−0.03	0.744	−0.05	0.812
HbA1c, %	0.22	0.005	0.32	0.002	0.35	0.023
TG, mg/dl	0.41	0.001	0.44	0.005	0.35	0.001
Cholesterol, mg/dl	0.35	0.043	0.38	0.023	0.27	0.014
LDL, mg/dl	0.22	0.036	0.32	0.044	0.19	0.021
HDL, mg/dl	−0.54	0.001	−0.57	0.007	−0.63	0.038
Calcium, mg/dl	0.05	0.954	0.12	0.831	0.09	0.189
Phosphorus, mg/dl	−0.01	0.571	−0.04	0.674	−0.07	0.354
Intact PTH, pg/ml	0.06	0.091	0.11	0.088	0.14	0.067
Urea, mg/dl	0.21	0.012	0.44	0.024	0.21	0.005
Creatinine, mg/dl	0.31	0.004	0.35	0.007	0.42	0.037

CKD, chronic kidney disease; CRP, C-reactive protein; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglycerides.

Table 4. Comparison of echocardiographic data in CKD, HD, and control group.

	CKD ($N = 40$)	HD ($N = 40$)	Control ($N = 20$)	*P* value
LVM, g	204 ± 30	215 ± 45	142 ± 70	0.002
LVMI, g/m^2	95 ± 23	105 ± 16	84 ± 35	0.004
LVEF, %	50 ± 10	45 ± 9	65 ± 26	0.032
FS, %	50 ± 15	45 ± 11	62 ± 18	0.033
IVST, mm	11 ± 2	12 ± 1	9 ± 3	0.012
LVPWT, mm	10 ± 4	11 ± 3	8 ± 2	0.008
E/A ratio	2.2 ± 0.1	2.5 ± 0.2	1.2 ± 0.5	0.003
Calcifications	10 (25%)	16 (40%)	0 (0%)	0.001

CKD, chronic kidney disease; FS, fractional shortening; LVEF, left ventricular ejection fraction; LVM, left ventricular mass; LVMI, left ventricular mass index.

Table 5. Correlation between visfatin level and echocardiographic data in CKD, HD, and control group.

	CKD ($N = 40$)		HD ($N = 40$)		Control ($N = 20$)	
	r	*P* value	*r*	*P* value	*r*	*P* value
LVM, g	0.24	0.004	0.27	0.044	0.34	0.031
LVMI, g/m^2	0.25	0.001	0.33	0.004	0.22	0.008
LVEF, %	−0.23	0.006	−0.42	0.011	−0.15	0.036
FS, %	−0.51	0.041	−0.55	0.045	−0.25	0.021
IVST, mm	0.45	0.023	0.47	0.022	0.25	0.007
LVPWT, mm	0.42	0.037	0.42	0.049	0.39	0.004
E/A ratio	0.34	0.011	0.41	0.047	0.65	0.008
Calcifications	0.25	0.002	0.32	0.003	0.29	0.038

CKD, chronic kidney disease; FS, fractional shortening; LVEF, left ventricular ejection fraction; LVM, left ventricular mass; LVMI, left ventricular mass index.

statistically significant positive correlation between visfatin levels in the three groups and LVM, LVMI, IVST, LVPWT, and E/A (Table 5). However, a statistically significant negative correlation was found between visfatin levels in the three groups and LVEF and FS. In addition, by running Spearman's correlation analysis, a significant association was found between visfatin levels and the development of cardiac calcifications (Fig. 1).

Atherosclerotic parameters ($N = 100$) showed a statistically significant positive correlation between visfatin levels in the three groups and SBP, DBP, PSV, and CIMT. By running Spearman correlation analysis, a significant association was found between visfatin levels and the development of atherosclerotic plaques (Table 6).

Correlation between visfatin levels and atherosclerotic parameters ($N = 100$) showed a statistically

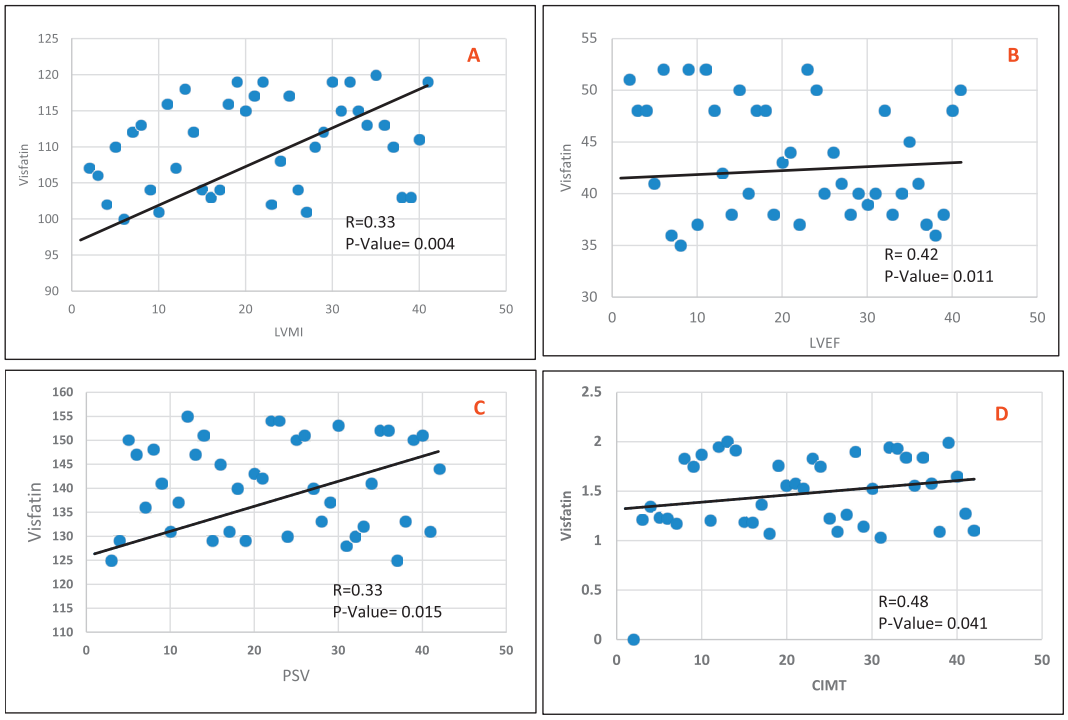

Fig. 1. (A–D) Positive significant correlation between visfatin and left ventricular mass index (LVMI) (r = 0.33, P = 0.004), left ventricular ejection fraction (LVEF) (r = 0.42, P = 0.011), peak systolic velocity (PSV) (r = 0.33, P = 0.015), and carotid intima-media thickness (CIMT) (r = 0.48, P = 0.041).

Table 6. Comparison of doppler data between CKD, HD and control group.

	CKD (*N* = 40)	HD (*N* = 40)	Control (*N* = 20)	*P* value
SPB, mm Hg	155 ± 15	165 ± 10	115 ± 20	0.022
DBP, mm Hg	92 ± 4	95 ± 6	85 ± 5	0.034
PSV, cm/s	125 ± 15	135 ± 20	35 ± 4	0.002
CIMT, mm	1.1 ± 0.3	1.3 ± 0.5	0.7 ± 0.2	0.036
Plaque [*n* (%)]	8 (20)	10 (25)	0	0.016

CIMT, carotid intima-media thickness; CKD, chronic kidney disease; DBP, diastolic blood pressure; PSV, peak systolic velocity; SBP, systolic blood pressure.

Table 7. Correlation between visfatin level and doppler data in CKD, HD, and control group.

	CKD (*N* = 40)		HD (*N* = 40)		Control (*N* = 20)	
	r	*P* value	*r*	*P* value	*r*	*P* value
SPB, mm Hg	0.33	0.024	0.37	0.001	0.18	0.003
DBP, mm Hg	0.16	0.004	0.28	0.014	0.16	0.018
PSV, cm/s	0.39	0.005	0.33	0.015	0.51	0.046
CIMT, mm	0.43	0.031	0.48	0.041	0.37	0.001
Plaque	0.37	0.012	0.46	0.024	0.26	0.008

CIMT, carotid intima-media thickness; CKD, chronic kidney disease; DBP, diastolic blood pressure; PSV, peak systolic velocity; SBP, systolic blood pressure.

significant positive correlation between visfatin levels in the three groups and SBP, DBP, PSV, and CIMT. By running Spearman correlation analysis, a significant association was found between visfatin levels and the development of atherosclerotic plaques (Table 7).

4. Discussion

Due to inadequate renal clearance, it is hypothesized that numerous adipocytokines (adiponectin, leptin, interleukin-6) and tumor necrosis factor-α are elevated in dialysis patients. Therefore, elevated

visfatin levels, along with those of other adipocytokines, may be predicted for this population of patients. Increased mortality and reduced endothelial function have both been linked to hypervisfatinemia in individuals with ESRD.[5]

Based on Murtadha *et al.*[6] Galal et al.[7] revealed that 26 HD patients and 15 control of similar age and sex were analyzed, like our findings. In terms of age, sex, BMI, and time spent with renal disease, no discernible differences were found between the groups. In contrast, Mohammed et al.[8] found a statistically significant variation among patients at various phases. Every patient was split into two groups as follows. Group A: ESRD (Stage 5) or advanced chronic kidney disease; group B: CKD (Stages 3–4) or moderate to severe renal impairment. Relative to age, the CKD group's visfatin levels were determined to be 30.2 ng/ml in CKD, 37.3 ng/ml in HD, and 23.2 ng/ml in control. Significant differences in serum visfatin levels were discovered across the groups (analysis of variance, $P = 0.001$).

Similarly, we found several other laboratory measurements with a statistically significant difference between the groups. Higher concentrations of CRP, HbA1c, triglycerides, total cholesterol, LDL cholesterol, serum calcium, serum phosphorus, intact PTH, serum urea, and creatinine were seen in CKD and HD groups compared with the control group [analysis of variance (ANOVA), $P = 0.05$]. Contrarily, both CKD and HD groups showed lower levels of hemoglobin and HDL cholesterol than the control group did (ANOVA, $P = 0.05$). Forty-five men and 24 women comprised group A; a total of 68 patients with ESRD who had been receiving hemodialysis for 7 months to 15 years (mean 5.57 years); their ages varied from 23 to 75 years (mean 51.24 years), and group B had 22 healthy controls, whose age ranged from 46 to 48 years.

Our findings concurred with those of the Lotfy et al.[9] study (mean 46.67 years). Group A (uremic on hemodialysis) had a significantly higher serum visfatin concentration than group B (controls) (48.95 ng/ml 11.62 vs. 22.65 ng/ml 5.24; $P = 0.001$). Other laboratory markers, such as triglycerides, total cholesterol, LDL cholesterol, serum calcium, serum phosphorus, intact PTH, serum urea, and creatinine, also statistically differed across the groups.

Our results conflict with those of Nüsken et al.[10] who found decreased serum visfatin among ESRD patients receiving hemodialysis, despite our patients showing a drop in body fat mass with increased insulin levels. We found a very significant inverse relationship between visfatin and fasting and postprandial blood sugar in our patient population, which may be explained by the higher insulin levels seen in the research by Nüsken and colleagues. Compared with the control group, those with CKD and HD had a greater LVM, LVMI, IVST, LVPWT, and E/A ratio in our echocardiographic analysis (ANOVA, $P = 0.05$).[10]

The FS and LVEF were significantly lower in the CKD and HD groups (ANOVA, $P = 0.05$). Ten (25% CKD) and 16 patients (40%) in the HD group were found to have calcifications. In the nontreated group, no calcifications were seen. Using a Pearson correlation analysis, we discovered that the three groups' visfatin levels were positively correlated with their LVM, LVMI, IVST, LVPWT, and E/A. However, a strong inverse connection was seen between visfatin levels across the three groups and LVEF and FS. Spearman's correlation analysis revealed a statistically significant link between visfatin concentrations and the formation of cardiac calcifications.[11]

PSV, CIMT, and systolic and diastolic blood pressure had all been significantly higher in the CKD and HD groups compared with the control group (ANOVA, $P = 0.05$). Only eight of the CKD patients (20%) and 10 of the HD patients (25%), respectively, were found to have atherosclerotic plaques. In the nontreatment group, no plaques were detected. Visfatin levels were revealed to positively connect with SBP, DBP, PSV, and CIMT across all three groups using Pearson's correlation analysis. Spearman's correlation study showed a statistically significant link between serum visfatin and the onset of atherosclerotic plaques.[11]

The findings of Karakan et al.[11] corroborated our own as they too found that patients were split into three categories based on their serum visfatin levels. Group 1 (34 ng/ml, $n = 22$) was designated as the lowest tertial of low visfatin, whereas group 2 (35–42 ng/ml, $n = 43$) plus group 3 (43 ng/ml, $n = 22$) were classified as the top titers; individuals in visfatin group 3 had higher BMI ($P = 0.00$), total cholesterol ($P = 0.03$), C-reactive protein ($P = 0.03$), homeostasis model evaluation of insulin resistance ($P = 0.03$), and LVMI ($P = 0.02$). Individuals in visfatin group 3 had higher BMI ($P = 0.00$), total cholesterol ($P = 0.03$), C-reactive protein ($P = 0.03$), homeostasis model evaluation of insulin resistance ($P = 0.03$), and LVMI ($P = 0.02$). Individuals in visfatin group 3 had higher BMI ($P = 0.00$), total cholesterol ($P = 0.03$), C-reactive protein ($P = 0.03$),

homeostasis model evaluation of insulin resistance ($P = 0.03$), and LVMI ($P = 0.02$). Independent factors that affected LVMI in the regression analysis were SBP (0.19, $P = 0.05$) and serum visfatin levels (0.74, $P = 0.05$) not only does fat tissue have the potential to release visfatin, but so do other cell types, most notably those involved in inflammation. It may have both local and systemic effects. Perhaps the most noticeable consequence of visfatin is its part in inflammation.[12]

According to research that looked backward, visfatin levels are found to be elevated in situations of chronic or acute inflammation. Besides, visfatin impacts chemotaxis, angiogenesis, fibrosis, and proliferation. Despite the systemic nature of these effects, they manifest mostly in cardiovascular pathology. It may influence the vascular system indirectly by causing problems in other tissues' blood vessels. More research is needed, although some suggest that visfatin may be used to predict cardiovascular problems.[13]

In addition, visfatin exhibits the same modifications in AMI as cardiac troponins. These findings indicate a bright future for visfatin, particularly in treating cardiovascular diseases.[14]

A wide variety of tissues that visfatin may affect, and its actions might manifest themselves in a web of biological pathways that is not always straightforward. Visfatin levels are related to atherosclerotic carotid thickness in individuals with metabolic syndrome and type 2 diabetes mellitus, so it is not surprising that this makes it hard to grasp visfatin well. Both Zhong et al.[3] and Kadoglou et al.[15]

Circulating visfatin levels have been suggested to gauge carotid thickness. Even though certain research has linked visfatin levels to CRP and carotid thickness in people with type 2 diabetes, Takebayashi et al.[16] found no such relationship. However, echocardiographic measurements of epicardial fat thickness in morbidly obese individuals have been shown to correlate positively with blood levels of visfatin[17,18].

Conflict of interest

There is no conflict of interest.

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