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***Original Article***

# The Physiological Effects of Visfatin on Immune Response and Inflammatory Impacts on Nephropathy

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**Abstract**

Obesity triggers the development of adipokines such as leptin, resistin, and visfatin, which have been associated with the development of diabetic nephropathy and other vascular disorders. The main purpose of the current investigation was to identify the physiological impact of visfatin on immunological response and its inflammatory effects on nephropathy. Fifty Iraqi patients with chronic kidney disease (CKD) at various stages, as described by the National Kidney Foundation (NKF) and ranging in age from 48.367.56 to 53.68 8.46 years on average were considered. Prior to the start of the investigation, informed consent was obtained from all participants, and the ethics committee approved the study. Patients were classified into two groups: Group (A) comprised patients with a GFR higher than 60 mL/minute, and Group (B) comprised patients with a GFR of less than 60 mL/min. There was no considerable variance between the groups as regards visfatin, but a highly significant correlation between serum visfatin and CRP was observed. The results of the current investigation indicated that serum visfatin levels are significantly correlated with CRP in CKD patients; it is also correlated with deterioration of kidney function. Moreover, higher visfatin levels were accompanied by increased serum triglyceride and cholesterol levels. These findings would suggest that visfatin may perform an essential function in uremia-related inflammation and may serve as a potential target for treatment and prevention of renal associated complications. Future studies may delineate whether visfatin is a marker of disease activity and severity as well as a predictor of outcome in CKD.

**Keywords:** nephropathy, obesity, visfatin, adipokines, immune response

**Les Effets Physiologiques de la Visfatine sur la Réponse Immunitaire et les Impacts Inflammatoires sur la Néphropathie**

**Résumé:** L'obésité déclenche le développement d'adipokines telles que la leptine, la résistine et la visfatine, qui ont été associées au développement de la néphropathie diabétique et d'autres troubles vasculaires. L'objectif principal de l'enquête actuelle était d'identifier l'impact physiologique de la visfatine sur la réponse immunologique et ses effets inflammatoires sur la néphropathie. Cinquante patients irakiens atteints d'insuffisance rénale chronique (IRC) à divers stades, tels que décrits par la Fondation nationale du rein (FNR) et âgés de 48.367.56 à 53.688.46 ans en moyenne ont été pris en compte. Avant le début de l'enquête, le consentement éclairé de tous les participants a été obtenu et le comité d'éthique a approuvé l'étude. Les patients ont été classés en deux groupes: le groupe (A) comprenait les patients avec un DFG supérieur à 60 ml/min, et le groupe (B) comprenait les patients avec un DFG inférieur à 60 ml/min. Il n'y avait pas de variance considérable entre les groupes en ce qui concerne la visfatine, mais une corrélation hautement significative entre la visfatine sérique et la CRP a été observée. Les résultats de l'enquête actuelle ont indiqué que les taux sériques de visfatine sont significativement corrélés avec la CRP chez les patients atteints d'IRC; ils sont également corrélés à la détérioration de la fonction rénale. De plus, des taux plus élevés de visfatine étaient accompagnés d'une

augmentation des taux sériques de triglycérides et de cholestérol. Ces résultats suggèrent que la visfatine peut jouer un rôle essentiel dans l'inflammation liée à l'urémie et peut servir de cible potentielle pour le traitement et la prévention des complications rénales associées. De futures études pourraient déterminer si la visfatine est un marqueur de l'activité et de la gravité de la maladie ainsi qu'un prédicteur de l'issue de l'IRC.
**Mots-clés:** néphropathie, obésité, visfatine, adipokines, réponse immunitaire

## 1. Introduction

Found in "human peripheral blood lymphocytes, visfatin is predominantly generated by visceral adipose tissue, according to initial discoveries. It is a ubiquitous intracellular protein known as nicotinamide phosphoribosyltransferase (NAMPT)/pre-B cell colony-enhancing factor (PBEF)-1. Its enzymatic foundation and structural characteristics have been determined. "NAMPT is a component of the nicotinamide adenine dinucleotide (NAD+) salvage/recycling system in the nucleus that governs the activities of NAD+-dependent enzymes such as the protein deacetylase sirtuin, which is essential for normal cell function (SIRT) -1" (1). Visfatin has been connected with a potential involvement in vascular dysfunction and inflammation associated with various metabolic diseases, and it has been shown to increase vascular smooth muscle inflammation (2). It may also be connected with the aging-dependent circadian cycle associated with the decrease "of pancreatic cell function in type 2 diabetes, and thus, it may be a useful therapeutic target in the treatment of this condition. Angiotensin II has been shown to be beneficial in the extension of mouse life span. Mice deficient in angiotensin acquired a longevity phenotype, and visfatin production in the kidney was increased in these mice (3).

When one has metabolic syndrome as with diabetes, the kidney is a primary target organ. Despite the fact that the kidney suffers as a result of hyperglycemia, no one has been able to determine what function it plays in glucose metabolism and insulin resistance. However, it has lately been recognized that the kidney performs an essential role in glucose metabolism and that individuals with chronic renal disease commonly have problems in glucose metabolism. Animal studies have also provided significant evidence for the synthesis and use of glucose by the kidneys (4). With the advent of novel antidiabetic medicines, researchers have recently begun to look into the function of the kidney in glucose metabolism and energy balance. Several investigations have demonstrated "that adipose tissue is a highly metabolic organ with pleuripotent activities that extend far beyond the simple storage of energy."It is now understood to be an endocrine organ that secretes a large variety of bioactive proteins called adipokines. These proteins have important roles in a variety of processes, including energy balance, glucose and lipid metabolism, insulin resistance, inflammation, and atherosclerosis. Adipocyte-secreted active molecules including leptin, resistin, and adiponectin, as well as inflammatory-cell-produced cytokines like tumor necrosis factor (TNF)-, "interleukin (IL)-6, macrophage/monocyte chemoattractant protein (MCP)-1", and interleukin (IL)-1, are among them (5).

"Visfatin is a ubiquitous intracellular enzyme also known as nicotinamide phosphoribosyltransferase (NAMPT)/pre-B cell colony-enhancing factor (PBEF)-1 and is one of the identified adipokines. It was first discovered in lymphocytes, and thereafter, its enzymatic biochemical foundation as well as its structural characteristics were determined. This pathway controls the activities of NAD+-dependent enzymes, such as the protein deacetylase sirtulin (SIRT)-1, by rescuing and recycling nicotinamide adenine dinucleotide (NAD+) from the nucleus and recycling it back into the cell (6). NAMPT, "a single-gene enzyme that catalyzes the conversion of nicotinamide (NAM) to nicotinamide mononucleotide

(NMN) which is then transformed into NAD+ by nicotinamide mononucleotide adenylyltransferase (NMNAT)-1, "is a rate-limiting enzyme that catalyzes the conversion of nicotinamide mononucleo-sodium adenosine dinucleotide (NAD+) is a coenzyme that participates in a variety of metabolic and redox activities. It is consumed as an ADP-ribose donor with the subsequent release of NAM as a byproduct. NAMPT has been demonstrated to be a key NAD biosynthesis enzyme in mammalian cells, as it controls the activity of the SIRT-1 enzyme (7). The current study sought to identify the physiological impacts of visfatin on the immune response as well as the inflammatory consequences of nephropathy.

## 2. Material and Methods

Fifty Egyptian "patients at different stages of CKD defined according to the National Kidney Foundation" (NKF) with a mean age of 48.36±7.56 to 53.68±8.46 years were investigated. Informed consent from the patients and approval from the relevant ethics committee were obtained prior to the start of the study. All patients were arranged in two groups: Group (A) comprised patients with GFR greater than 60 ml/min, and Group (B) was composed of patients with GFR less than 60 ml/min. Glomerular filtration rates (GFRs) were determined by calculating GFR from serum creatinine levels using the Cockcraft and Gault (C&G) formula.

Cockcraft-Gault GFR = (140-age) X (Wt. in kg) X (0.85 if female) / (72 X Cr)

Wt = weight, Cr = serum creatinine in mg/dL. The estimated glomerular filtration rate (EGFR) is expressed as mL/min/1.73 m 2.

### 2.1. Exclusion Criteria

Patients with a history of inflammatory disease (infection, sepsis), malignancy, liver disease, collagen disease, steroid use, or surgery within one month prior to admission, and those with diabetes or coronary artery disease were excluded from the study.

### 2.2. Additional Tests

In addition to a thorough history-taking and physical examination, abdominal ultrasonography and laboratory tests were performed. These included serum urea, creatinine, serum triglycerides, total cholesterol, potassium (k), phosphorus (ph), calcium (Ca), sodium (Na), and C-reactive protein (CRP). the participants' demographic information, including age, gender, presence of renal failure, and time from diagnosis of renal failure, was taken into consideration. Height, hip circumference, weight, and waist circumference were all measured during the physical examination, and the results were recorded. The "formula for calculating body mass index" (BMI) is as follows: BMI = weight (kg)/height2 (m).

### 2.3. Sampling

After waking up in the morning, each patient had five milliliters of venous blood taken from them. "The leftover sample was placed into a disposable plastic tube and allowed to clot for a few minutes before being centrifuged at 1200 rpm for 10-15 minutes to separate the serum from the other components. The serum was then stored frozen at -20 ℃ until used. Serum visfatin concentration was measured using an enzyme linked immunosorbant test (ELISA) (EIAAB® Human Visfatin Enzyme Immunoassay, NO o638H).

### 2.4. Statistical Design

SPSS version 15 was utilized to code and statistically analyze the data. The mean and standard deviation (SD) of quantitative data was computed to determine the central tendency and dispersion of the data, and frequency of occurrence was estimated to assess qualitative data. The study groups were compared using the chi-square test (X2) for qualitative data, the student t test for comparing quantitative parametric data between the two groups, the Mann-Whitney U test for comparing nonparametric data between the two groups, and Spearman's correlation coefficient to assess the relationship between two variables used. The significance threshold was set at a *p*-value of less than 0.05 (2 tailed). Tables and graphs were used to present the findings.

### 2.5. Statistical Analysis

SPSS version 18.0 was employed to conduct the statistical analyses. The student t-test or Mann Whitney U-test (if necessary) was utilized to compare the two groups. "Chi-square and Fisher's exact tests were employed in the analysis of 2x2 contingency tables" when dealing with non-numerical information. When dealing with regularly distributed data, the Pearson correlation test was employed, and when dealing with non-normally distributed data, the Spearman's rho correlation test was utilized. The groups were compared using the student t-test or, if necessary, one-way or multiple variance analysis (ANOVA). When there were more than two groups to compare and the data was not normally distributed, Kruskal Wallis-H analysis was used. The Tukey HSD test was employed to make post-hoc comparisons. To investigate the variables that influence the amount of serum visfatin, a Cox regression analysis was done. A p-value of less than 0.05 was considered statistically significant.

## 3. Results

For gender, age, and BMI, there was no statistically considerable variance between the two populations **(**Table 1; Figure 1 and Figure 2**)**. However, there was a high statistically significant variance between the two groups in urea and creatinine **(**Table 2; Figure 3 and Figure 4**)**. Highly significant variances were also seen between the two groups in terms of potassium, calcium, and CRP, all of which were quite significant **(**Table 3).

There was no statistically significant variance between the two groups when it came to visfatin (Table 4); however, a highly significant correlation was found between serum visfatin and CRP (Table 5). The relationship seen in univariate analyses was only apparent in multivariate analyses between visfatin and IL-6 (Table 6).

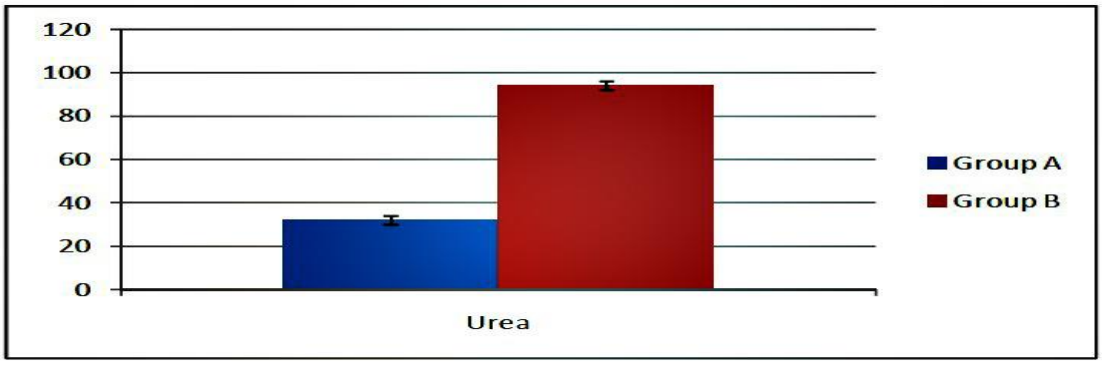


**Figure 1.** Urea concentration in the experimental groups. Group **(A)** patients with GFR greater than 60 ml/min; Group **(B)** patients with GFR less than 60 ml/min.

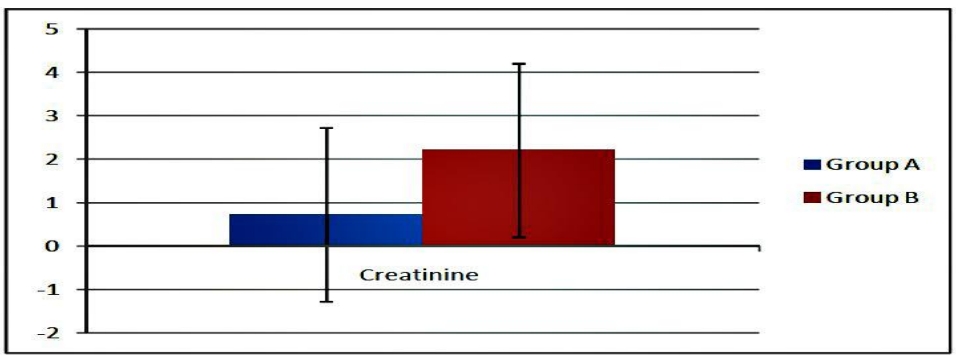


**Figure 2.** Creatinine concentration in the experimental groups. Group **(A)** patients with GFR greater than 60 ml/min; Group **(B)** patients with GFR less than 60 ml/min.

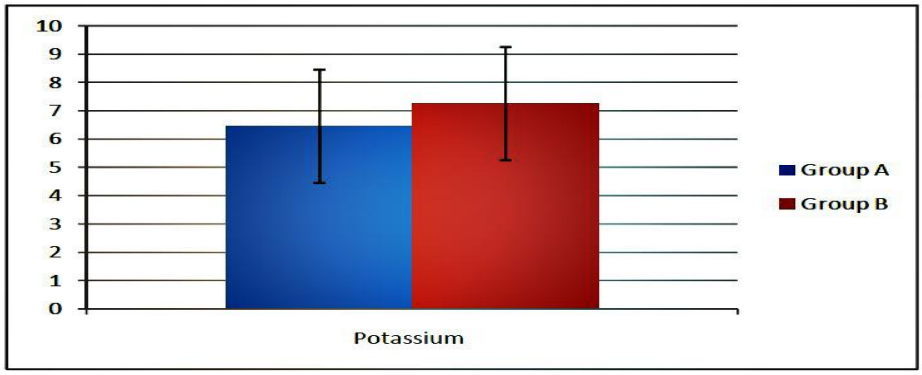


**Figure 3.** Potassium concentration in the experimental groups. Group **(A)** patients with GFR greater than 60 ml/min; Group **(B)** patients with GFR less than 60 ml/min.

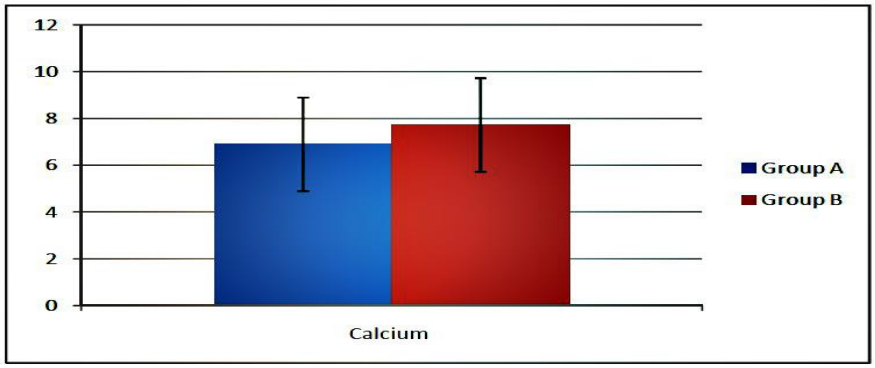


**Figure 4.** Calcium concentration in the expearimental groups. Group **(A)** patients with GFR greater than 60 ml/min; Group **(B)** patients with GFR less than 60 ml/min.

**Table 1.** Demographic data

| Variable | | Group A (n=25) | Group B (n=25) | P |
|---|---|---|---|---|
| **Gender** | Male | 13 | 16 | 0.4094 |
| | Female | 12 | 9 | |
| **Age** | | 47.35±7.52 | 53.68± 8.46 | 0.5684 |
| **BMI** | | 29.2±5.0 | 30.9±4.9 | 0.9220 |

**Table 2.** Hematological and biochemical data of patients according to groups

| Variable | Group A(n=25) | Group B(n=25) | P |
|---|---|---|---|
| **Urea(mg/dl)** | 32.0±7.5 | 94.0±39.6 | 0.001 |
| **Creatinine(mg/dl)** | 0.72±0.14 | 2.2±1.0 | 0.001 |
| **e-GFR(ml/min/1.73m2)** | 99±14 | 33±15 | .7381 |

**Table 3.** Comparison of groups regarding clinical and biochemical markers (mean ± SD)

| Variable | Group A (No. = 25) | Group B (No. = 25) | P |
|---|---|---|---|
| **Total cholesterol (mg/Dl)** | 207±60 | 227±68 | .5445 |
| **Low-density lipoprotein (mg/dL)** | 127±45 | 141±55 | .3322 |
| **High-density lipoprotein (mg/dL)** | 45±10 | 42±9 | .6098 |
| **Triglyceride (mg/dL)** | 165±82 | 219±98 | .3887 |
| **Sodium (mmol/L)** | 136.92±2.87 | 137.04± 2.99 | 0.8426 |
| **Potassium (mmol/L)** | 6.45± 1.19 | 7.25±0.60 | 0.001* |
| **Calcium (mg/dL)** | 6.89± 1.44 | 7.72±.48 | .001* |
| **Phosphorus (mg/dL)** | 3.4±0.6 | 4.1±0.9 | .05237 |
| **CRP (mg/L)** | 3.80±1.89 | 11.40±4.19 | .001* |

**Table 4.** Visfatin levels in groups

| Variable | Group A (n=25) | Group B (n=25) | P |
|---|---|---|---|
| **Visfatin (ng/ml)** | 2.6±1.2 | 2.7±1.1 | .6733 |

**Table 5.** Correlation coefficient of serum visfatin with CRP

| Variable | Correlation Coefficient | P-value | Significance |
|---|---|---|---|
| **CRP** | 0.905 | 0.000 | HS |

HS (Highly Significant)

**Table 6.** Visfatin-related parameters in multivariate analysis in patients of both groups

| | Group A (n=25) | | | Group B (n=25) | | |
|---|---|---|---|---|---|---|
| **Variable** | **B** | **Beta** | **P** | **B** | **Beta** | **P** |
| **IL-6** | -0.068 | -0.121 | 0.417 | 0.070 | 0.380 | 0.006 |
| **TNF-α** | 0.06 | 0.07 | 0.791 | -0.004 | -0.098 | 0.435 |

## 4. Discussion

Inflammatory diseases like acute lung damage, sepsis, and rheumatoid arthritis are associated with a rise in visfatin levels. Thus, visfatin is regarded as a proinflammatory adipokine through either a compensatory reaction or an epiphenomenon, depending on how it is produced. It is noteworthy that visfatin is virtually exclusively expressed "in visceral adipose tissue more than subcutaneous adipose tissue important. Several adipokines that are produced by adipocyte tissue have been investigated in relation to insulin resistance and the metabolic syndrome, which encompasses, among other things, obesity, glucose intolerance, and dyslipidemia (8). In addition, elevated visfatin levels were seen in patients undergoing hemodialysis, and a significant connection between visfatin levels and all phases of chronic renal disease was discovered (9). Previous research has shown that endothelial "dysfunction in early diabetic nephropathy is related to altered levels of the protein visfatin in" the bloodstream. One investigation revealed that visfatin activated endothelial nitric oxide synthase (eNOS) via Akt and mitogen-activated protein (MAP) kinase and

MCP-1 and also improved endothelial cell function, angiogenesis, and the development of atherosclerosis. As a result, there is disagreement between clinical research and experimental evidence on the involvement of visfatin in diabetic nephropathy. It is uncertain whether this condition is caused largely by direct vascular abnormalities or secondary to the existence of visceral obesity and a dysregulated metabolic milieu, both of which are common in many diabetic patients. The cause of this phenomena is unknown (10).

Multiple studies have found "that plasma visfatin levels were raised in humans and animals" suffering from nephropathy (11). The main purpose of the current investigation was to identify the physiological impacts of visfatin on the immune response as well as the inflammatory consequences of nephropathy. Based on the results of the current investigation, no statistically significant variances were seen between the two groups in gender, age, or BMI.

Sakin, Sahin (12) showed that there was no statistically significant variance between the two groups regarding gender, age, or body mass index (BMI) in their study. Rehab et al. (13) found no statistically significant variance between the two groups in their study in gender, but that there was a statistically considerable variance between the two groups when it came to age. The current findings revealed high statistically significant variances between the two groups in terms of urea" and creatinine, as was demonstrated by Mohammed, Ebrahem (13) and Sakin, Sahin (12) in their investigations. Substantial variances between the two groups in terms of potassium, calcium, and CRP, all of which were statistically significant, were also found in the current study.

Sakin, Sahin (12) demonstrated a significant variance between the two groups in their study in potassium, but not in calcium. Mohammed, Ebrahem (13) found no statistically significant variance between the two groups in terms of potassium and calcium, but they did find a significant difference in phosphorus and a very considerable difference in CRP between the two groups. No statistically significant difference between the two groups was seen regarding visfatin in the current study or by Sakin, Sahin (12) in their study. However, Mohammed, Ebrahem (13) observed a highly significant variance between the two groups in their study. Yilmaz, Saglam (14) studied patients with chronic kidney disease (CKD) ranging from stage 1 to stage 5, "and they discovered that patients with stage 3-5 CKD had higher levels of visfatin in comparison with subjects with stage 1-2 CKD and controls; however, no statistically significant variance was seen between the controls and the subjects with stage 1-2 CKD. As previously stated, the reason for this observation could be related to decreased clearance as a result of low glomerular filtration in renal damage. Another possible explanation could be excessive release from progressively damaged renal cells in advanced stages, as it is proposed to be located intracellularly and released upon cell lysis (15).

In 3023 older individuals, Kocelak, Olszanecka-Glinianowicz (16) found no connection between visfatin levels and GFR. Despite the fact that they separated patients into two groups based on GFR (patients with GFR below 60 and patients with GFR over 60 ml/min/m2), no statistically considerable variance was detected "between the groups in terms of visfatin concentrations. They did not, however, involve diabetic individuals in their study. According to the findings of the current investigation, there is a highly significant correlation between" serum visfatin and CRP. According to Mohammed, Ebrahem (13), there were favorable associations between serum cholesterol, triglycerides, CRP, and serum visfatin levels in the blood. According to Kato, Odamaki (17), visfatin may not directly represent atherosclerotic alterations in cardiovascular and renal patients, but rather the overall inflammatory condition in these patients. This study demonstrated that the relationship seen in univariate analyses was only evident between visfatin and IL-6 when multivariate analyses were performed. After repeating the correlation study with each group

independently, Sakin, Sahin (12) discovered that there was a highly positive association between visfatin and IL-6 levels in Group III,, which was also favorably connected with microalbuminuria. There were several limitations to this study, such as the limited number of cases in both groups.

The current investigation displayed that "serum visfatin levels are significantly correlated with CRP in CKD" patients, and it is also correlated with deterioration of kidney function. Moreover, higher visfatin levels were accompanied by increased serum triglyceride and cholesterol levels. These findings would suggest that visfatin may perform an essential function in uremia-related inflammation and may serve as a potential target for treatment and prevention of renal associated complications. Future studies may delineate whether visfatin is also a marker of disease activity and severity as well as a predictor of outcome in CKD.

## Authors' Contribution

Study concept and design: R. M. Sh. O.
Acquisition of data: R. M. Sh. O.
Analysis and interpretation of data: R. M. Sh. O.
Drafting of the manuscript: R. M. Sh. O.
Critical revision of the manuscript for important intellectual content: R. M. Sh. O.
Statistical analysis: R. M. Sh. O.
Administrative, technical, and material support: R. M. Sh. O.

## Ethics

Informed consent from the patients and approval from the relevant ethics committee were obtained prior to the start of the study.

## Conflict of Interest

The authors declare that they have no conflict of interest.

## References

1. Hasegawa K. Novel tubular-glomerular interplay in diabetic kidney disease mediated by sirtuin 1, nicotinamide mononucleotide, and nicotinamide adenine dinucleotide Oshima Award Address 2017. Clin Exp Nephrol. 2019;23(8):987-94.
2. Hognogi LD, Simiti LV. The cardiovascular impact of visfatin - an inflammation predictor biomarker in metabolic syndrome. Clujul Med. 2016;89(3):322-6.
3. Kang YS, Lee MH, Song HK, Kim JE, Ghee JY, Cha JJ, et al. Chronic Administration of Visfatin Ameliorated Diabetic Nephropathy in Type 2 Diabetic Mice. Kidney Blood Press Res. 2016;41(3):311-24.
4. Giri B, Dey S, Das T, Sarkar M, Banerjee J, Dash SK. Chronic hyperglycemia mediated physiological alteration and metabolic distortion leads to organ dysfunction, infection, cancer progression and other pathophysiological consequences: An update on glucose toxicity. Biomed Pharmacother. 2018;107:306-28.
5. Wang T, He C. Pro-inflammatory cytokines: The link between obesity and osteoarthritis. Cytokine Growth Factor Rev. 2018;44:38-50.
6. Dalamaga M, Christodoulatos GS, Mantzoros CS. The role of extracellular and intracellular Nicotinamide phosphoribosyl-transferase in cancer: Diagnostic and therapeutic perspectives and challenges. Metab. 2018;82:72-87.
7. Ratajczak J, Joffraud M, Trammell SA, Ras R, Canela N, Boutant M, et al. NRK1 controls nicotinamide mononucleotide and nicotinamide riboside metabolism in mammalian cells. Nat Commun. 2016;7:13103.
8. Landecho MF, Tuero C, Valenti V, Bilbao I, de la Higuera M, Fruhbeck G. Relevance of Leptin and Other Adipokines in Obesity-Associated Cardiovascular Risk. Nutrients. 2019;11(11).
9. Marouga A, Dalamaga M, Kastania AN, Kroupis C, Lagiou M, Saounatsou K, et al. Circulating resistin is a significant predictor of mortality independently from cardiovascular comorbidities in elderly, non-diabetic subjects with chronic kidney disease. Biomark. 2016;21(1):73-9.
10. Ren J, Wu NN, Wang S, Sowers JR, Zhang Y. Obesity cardiomyopathy: evidence, mechanisms, and therapeutic implications. Physiol Rev. 2021;101(4):1745-807.
11. Kaminski TW, Pawlak K, Karbowska M, Mysliwiec M, Pawlak D. Indoxyl sulfate - the uremic toxin linking hemostatic system disturbances with the prevalence of cardiovascular disease in patients with chronic kidney disease. BMC Nephrol. 2017;18(1):35.
12. Sakin A, Sahin S, Behlul A, Sumnu A, Gursu M, Sakin A, et al. The association of Visfatin levels with metabolic

parameters and inflammation in diabetic nephropathy. East J Med. 2020;25(2):218-24.

13. Mohammed RA, Ebrahem EE, Youssef E, Bayoumy ESJA. Serum visfatin as a biomarker of inflammation in patients with chronic kidney disease.AAMJ. 2012;10(3):2.
14. Yilmaz MI, Saglam M, Carrero JJ, Qureshi AR, Caglar K, Eyileten T, et al. Serum visfatin concentration and endothelial dysfunction in chronic kidney disease. Nephrol Dial Transplant. 2008;23(3):959-65.
15. Vallon V, Thomson SC. The tubular hypothesis of nephron filtration and diabetic kidney disease. Nat Rev Nephrol. 2020;16(6):317-36.
16. Kocelak P, Olszanecka-Glinianowicz M, Owczarek A, Bozentowicz-Wikarek M, Brzozowska A, Mossakowska M, et al. Plasma visfatin/nicotinamide phosphoribosyltransferase (visfatin/NAMPT) concentration is not related to kidney function in elderly subjects. Clin Chem Lab Med. 2015;53(5):793-9.
17. Kato A, Odamaki M, Ishida J, Hishida A. Relationship between serum pre-B cell colony-enhancing factor/visfatin and atherosclerotic parameters in chronic hemodialysis patients. Am J Nephrol. 2009;29(1):31-5.