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Original Research Article

Evaluation of Visfatin Levels and Some Immunological Parameters in Chronic Kidney Disease Patients

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Abstract: Chronic kidney disease (CKD) represents one of the major non-communicable diseases affecting populations worldwide. It is characterized by a gradual decline in renal function resulting from persistent structural or functional abnormalities of the kidneys, eventually leading to impaired filtration capacity and multiple systemic complications. Persistent renal dysfunction is accompanied by immune dysregulation and chronic inflammation, leading to altered production of several cytokines and inflammatory mediators., Increasing evidence indicates that chronic inflammation plays a central role in CKD progression, while circulating visfatin has emerged as an inflammatory adipokine closely linked to metabolic and immune alterations in these patients, cellular stress responses and metabolism. This investigation was designed to quantify serum concentrations of visfatin, interleukin-17A, beta2-microglobulin, and interleukin-22 in individuals with chronic kidney disease and to compare these biomarkers with those measured in healthy participants. A case-control study was performed between January and June 2025 involving 90 participants, including 60 clinically diagnosed CKD patients and 30 apparently healthy controls., serum samples were collected to measure the level of visfatin, interleukin 17A, beta2-microglobulin and interleukin 22 by ELIZA technique, which is based on the Sandwich ELIZA principle. The results of the study showed a significant increase in the level of visfatin in patients with CKD (7.25 ± 0.6 pg/ml) compared to controls (3.6 ± 0.3 pg/ml). The results of the study also showed a highly significant increase in the level of interleukin 17A, beta 2 microglobulin and interleukin 22 in patients with CKD (24. 39 ± 7.2 pg/ml, 6.86 ± 1.7 ng/ml, and 185.7 ± 20.5 pg/ml) respectively, compared with controls (3.22 ± 0.77 pg/ml, 2.53 ± 0.74 ng/ml, and 89.4 ± 15.3 pg/ml) respectively. These findings indicate that elevated serum levels of visfatin, interleukin-17A, beta2-microglobulin, and interleukin-22 are closely associated with chronic kidney disease and may serve as useful biomarkers reflecting immune activation and inflammatory status in affected patients.

Keywords: Chronic Kidney Disease, Interleukin 17A, Interleukin 22, Beta2-Microglobulin.

INTRODUCTION

Chronic kidney disease (CKD) is one of the most prevalent chronic disorders worldwide, affecting millions of individuals and representing a substantial burden on healthcare systems. As renal function progressively declines, patients become more susceptible to kidney failure, cardiovascular complications, and premature mortality. By the fourth decade of the 21st century it will be the fifth leading cause of death in the world, and awareness of this disease and its risks is often low, both from patients themselves and people who care for them [1].

Hypertension is recognized as a major modifiable risk factor for both chronic kidney disease and cardiovascular disorders. Persistent elevation of blood pressure accelerates renal damage and contributes to disease progression, claiming the lives of about 40% of the world's population of people in most countries in the world [2]. Clinically, hypertension is diagnosed according to sustained elevations in systolic and/or diastolic blood pressure above the recommended diagnostic thresholds established by international guidelines [3]. Hypertension secondary to an underlying etiology such as CKD is often asymptomatic until severe chronic disorders develop, and blood pressure measurement is a diagnostic procedure to determine the etiology and assess other risk factors for cardiovascular health [4].

Excessive salt intake and obesity also contribute to kidney damage, and these factors can be controlled through lifestyle changes by reducing salt intake, smoking cessation, fitness and high protein intake [5]. Visfatin is a multifunctional adipokine involved in numerous biological processes, including metabolic regulation, inflammatory signaling, immune responses, and cellular adaptation to physiological stress [6]. It can stimulate the expression of pro-inflammatory cytokines, and Previous investigations have reported elevated circulating visfatin concentrations in several inflammatory disorders, including chronic kidney disease, suggesting its involvement in disease pathogenesis [7]. Visfatin has also been implicated in regulating immune cell activity by enhancing cytokine production and modulating the function of T lymphocytes and other inflammatory cells, it increases blood pressure by inhibiting endothelial nitric oxide production, and increased ROS production contributes to the development of vascular fibrosis, glomerular damage and increased salt retention [8]. Beta2-microglobulin is a low-molecular-weight protein that reflects immune activation and renal function. Its serum concentration increases as glomerular filtration declines, making it a useful biomarker in patients with impaired kidney function [9]. Beta2-microglobulin is a hallmark of kidney dysfunction due to hypertension, as studies have shown that its level in the urine is elevated in people with hypertension [10]. Interleukin-22 is an immunoregulatory cytokine involved in maintaining epithelial integrity and promoting tissue repair. In kidney disease, altered IL-22 expression has been associated with inflammatory responses and renal injury [11]. Interleukin 22 possesses anti-inflammatory properties in a wide range of tissues, including kidney tissue, as it can suppress the production of chemokines and cytokines associated with inflammation, thereby reducing the immune response and minimizing tissue damage [12]. Based on these observations, the present study was designed to investigate circulating visfatin together with selected immunological biomarkers in patients with chronic kidney disease and to compare their levels with those observed in healthy individuals.

Materials and Methods

Time and Place of the Study

Between January 2025 and June 2025, the study was conducted in the dialysis unit at Al-Ramadi Taeching Hospital. The study population comprised 90 participants, 60 of whom had been diagnosed with chronic kidney disease and 30 of whom were volunteers who appeared healthy and served as the control group.

Sample Collection

Approximately 5 mL of peripheral venous blood was obtained aseptically from each participant using sterile disposable syringes. After letting the blood clot for ten minutes at room temperature in gel test tubes, the blood was separated by centrifuging the tubes for five minutes at 3500 rpm. The blood serum was then kept in Eppendorf tubes at -20°C until sample collection was finished and the required tests were carried out.

Estimation of the Studied Parameters

The level of visfatin, interleukin 17A, beta2-microglobulin and interleukin 22 were estimated by ELIZA technique according to the working method used by Sunlong biotech, which adopts the sandwich ELIZA working principle as shown in Table 1.

Table 1: Shows the Eliza kits used in the study

Kits	Country	Company
Human visfatin ELIZA kit	China	Sunlong
Human interleukin 17A, IL17A ELIZA kit	China	Sunlong
Beta 2 microglobulin, β2 microglobulin ELIZA kit	China	Sunlong
Human interleukin 22, IL22 ELIZA kit	China	Sunlong

Statistical Analysis

Statistical analyses were conducted using SPSS software (Version 27), and the obtained data were expressed as mean ± standard deviation. Non-parametric tests were used due to the non-normal distribution of the data. The Mann-Whitney U test was used to compare continuous variables between the CKD and control groups.

Results

Evaluation of Serum Visfatin Level

The study's findings demonstrated a highly significant increase in visfatin levels in CKD patients when compared to healthy individuals; at a significance level (P=0.003), the level of visfatin in patients was 7.25 ± 0.6 pg/ml, while in healthy individuals it was 3.6 ± 0.3 pg/ml, as Table 2 illustrates.

Table 2: Shows the level of visfatin in the serum of CKD patients and healthy people

Study groups	Level pg/ml	St. deviation	P-value
Patients	7.25	0.6	0.003
Control	3.6	0.3	

Evaluation of Serum Interleukin 17A Level

According to the study's findings, interleukin 17A levels were significantly higher in CKD patients than in healthy individuals at a significance level of P=0.002. According to Table 3, the patient group's interleukin 17A level was 24.39 ± 7.2 pg/ml, whereas the healthy group's level was 3.22 ± 0.77 pg/ml at a significant level P=0.002.

Table 3: Shows the level of interleukin 17A in the serum of CKD patients and control

Study groups	Level pg/ml	St. deviation	P-value
Patients	24.39	7.2	0.002
Control	3.22	0.77	

Evaluation of Beta 2 Microglobulin Level in Blood Serum

The study's findings indicated that patients with chronic kidney disease (CKD) had higher levels of beta 2 microglobulin than healthy individuals at a significance level (P=0.005). The patient group's level was 6.86 ± 1.7 ng/ml. According to Table 4, the level of Beta 2 microglobulin in healthy individuals was 2.53 ± 0.74 ng/ml at a significant level (P=0.005).

Table 4: Shows the level of Beta 2 microglobulin in the blood serum of CKD patients and control

Study groups	Level ng/ml	St. deviation	P-value
Patients	6.86	1.7	0.005
Control	2.53	0.74	

Estimation of Serum Interleukin 22 Level

According to the study's findings, individuals with chronic renal disease had blood serum levels of interleukin 22 that were significantly higher than those of the control group (185.7 ± 20.5 pg/ml). According to Table 5, the interleukin 22 level in the control group was 89.4 ± 15.3 pg/ml at a significant level (P=0.001).

Table 5: Shows the level of interleukin 22 in the blood serum of CKD patients and control

Study groups	Level pg/ml	St. deviation	P-value
Patients	185.7	20.5	0.001
Control	89.4	15.3	

DISCUSSION

The study's findings demonstrated that the patients' and the healthy group's levels of visfatin differed significantly; the patients' level was 7.25 ± 0.6 pg/ml, while the healthy group's level was 3.6 ± 0.3 pg/ml. The study [13], and other studies supported the validity of the current study's findings with high levels of visfatin in CKD. Visceral adipose tissue produces visfatin, which is crucial for a number of metabolic and inflammatory processes as well as insulin control [14]. Because of its anti-inflammatory qualities, visfatin helps control the inflammatory response, which in turn controls the inflammatory response in chronic kidney disease (CKD) [15]. High visfatin levels in CKD patients are due to the inflammatory state associated with decreased kidney function, chronic renal failure, increased inflammation and metabolic disorders seen in patients with renal failure [16]. Elevated visfatin is a marker of inflammation and metabolic problems in patients with chronic kidney disease (CKD) due to its function in inflammation [17]. Monitoring visfatin is a helpful indicator in evaluating the disease and the efficacy of treatment because it has been utilized as a biomarker for CKD complications [18]. Visfatin's involvement in the metabolic and inflammatory conditions linked to chronic kidney disease (CKD) is shown by elevated visfatin levels in CKD patients.

The results of this investigation showed that the interleukin 17A level in the sick group was 24.39 ± 7.2 pg/ml while it was 3.22 ± 0.77 pg/ml in the control group. Interleukin 17A destroys vital organs and greatly increases hypertension by affecting renal cells [8]. The [19], found that rats with systemic IL-17A had systolic blood pressure that was higher than usual at a probability threshold of 0.05. Interleukin 17A has no effect on kidney function metrics or injury, however it did not alter renal function through the levels of urea and creatinine. Antibodies and a genetic strategy were used to block IL-17A in experimentally hypertensive animals resulting in lower blood pressure, and by inhibiting IL-17A, a study [20], reported reduced peripheral organ damage. A study [21, 22], showed that interleukin 17A can be used as an adjunctive therapy for hypertension and peripheral organ dysfunction. Interleukin 17A signaling was also inhibited in hypotension. The current study's findings are consistent with a recent study by (23) that revealed interleukin 17A levels in patients with renal failure are higher than those in healthy individuals. The current study's findings demonstrated that the patient group's level of β2 microglobulin was 6.86 ± 1.7 ng/ml, which was significantly different from the control group's level. Although the control group's β2 microglobulin level was 2.53 ± 0.74 ng/ml, β2 microglobulin plays a mediating role in inflammation in systemic conditions and contributes to atherosclerosis by increasing oxidative activity, causing a chronic inflammatory process, and stimulating macrophages and lymphocytes, which accelerates vascular atherosclerosis [8]. The current study's

results align with the findings of [24], that patients with nephropathy have higher levels of β2 microglobulin than healthy individuals. Because β2 microglobulin is readily filtered by the glomerulus and reabsorbed by the proximal tubule in healthy kidneys, it has also been recognized as a marker of renal function. Evidence suggests that inflammatory markers are positively correlated with high levels of β2 microglobulin, as the level of β2 microglobulin increased with the development of chronic kidney disease (CKD). The results of a study by [25], revealed an association between β2 microglobulin and heart disease. Noted (26) as β2 microglobulin accumulates in peripheral tissues as CKD reaches its final stages glomerular filtration rate is closely related to the level of β2 microglobulin because the kidneys remove β2 microglobulin by glomerular filtration [27].

The current study's findings demonstrated a significant increase in interleukin 22 levels between the CKD group and the healthy group. The CKD group's level was 185.7 ± 20.5 pg/ml, whereas the healthy group's level was 89.4 ± 15.3 pg/ml. The study's findings support the conclusion of [28], that CKD patients have higher levels of interleukin 22 than those healthy individuals. Patients with chronic kidney disease (CKD) have been found to have elevated levels of inflammatory cytokines, such as interleukin 22, which can lead to kidney damage and fibrosis [29]. Interleukin 22, a member of the interleukin 10 cytokine family, is produced immediately after injury from various cells including T-helper cells17 and natural killer cells [30]. The role of interleukin 22 in chronic kidney injury is related to the regulation of inflammation and tissue repair and thus has a protective and anti-inflammatory effect [31, 32], explained that the cause of elevated interleukin 22 in CKD is due to the response of the injured kidney tissue and the body's attempt to repair the damage. Interleukin 22 also plays its role as a mediator in maintaining the epithelial homeostasis of the injured kidney by stimulating tissue regeneration and repair and inhibiting oxidative stress [33]. Elevated levels of interleukin 22 could therefore play a potential role in the inflammatory processes associated with this disease.

CONCLUSIONS

The present study showed significant elevation of interleukin 17A, beta2-microglobulin and interleukin 22 in CKD group compared to control group indicating an association between CKD and immune system. The study also showed elevated level of visfatin in CKD patients compared to controls due to its role in CKD-related inflammation and metabolic disorders.

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