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Assessment of the Factors Influencing the Procession of Chronic Kidney Disease and their Relationship with Biochemical Markers and Blood Pressure

Aalia Othman Farhan *

Department of Laboratory and Clinical Sciences, Collage of Pharmacy, Tikrit University. Tikrit,Iraq

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Abstract

Chronic kidney disease (CKD) is a progressive condition, along with the gradual deterioration of the kidney functioning and systemic inflammation, oxidative stress, and hemodynamic disorders. A cross-sectional study with a comparative design was performed on 120 subjects which comprised 80 participants with CKD diagnosis and 40 seemingly healthy controls. Demographic, clinical, and biochemical measurements were studied, such as serum creatinine, urea, uric acid, estimated glomerular filtration rate (eGFR), C-reactive protein (CRP), malondialdehyde (MDA), and reduced glutathione (GSH) along with lipid profile, liver functioning tests, and blood pressure. The findings revealed that CKD patients recorded a substantial increase in creatinine, urea, uric acid, CRP and MDA when compared to controls with a significant decrease in eGFR and GSH ($p < 0.001$). The values of blood pressure were intimately high and had an inverse correlation with eGFR and systolic pressure, the values of which were strongly negative ($r = -0.68$,$p= 0.01$). Moreover, liver enzymes (ALT, AST) and lipid rates (cholesterol, triglycerides) were raised, and the level of albumin and HDL cholesterol was lower. Analysis at stages showed gradual increases in creatinine, CRP, and MDA with a decrease in GSH and hemoglobin at each of CKD stages. These results show that inflammatory problems, oxidative stress, and hemodynamic imbalance are major factors that lead to the development of CKD. The synergistic increase in CRP and MDA, and the reduction in GSH and eGFR is an expression of the interactions between oxidative and inflammatory mechanisms of deterioration of the kidney. The timely detection and surveillance of these biomarkers can offer useful prognostic data and be used to inform the antioxidant and anti-inflammatory therapeutic interventions to decelerate chronic kidney disease development.

Keyword: Chronic kidney disease; Blood Pressure; Biochemical measurements; Hemodynamic disorders

1. Introduction

Chronic Kidney Disease (CKD) is a major international societal health concern, impacting an estimated 850 million individuals across the world that is, more than one in ten people across the world, and its prevalence is constantly growing, especially in developing and middle-income nations [1]. CKD is a progressive condition which is gradual deterioration of the glomerular filtration rate (GFR) consequential to structural and functional destruction of nephrons [2]. The disease is usually asymptomatic in the early phases of the condition and thus gets a late diagnosis causing greater chances of progressing to the End-Stage Renal Disease (ESRD), which necessitates renal replacement therapy as dialysis or kidney transplantation [3]. New statistics also underscore the huge economic and social burden of CKD that makes it one of the most expensive chronic diseases because of the high cost of treatment and its extensive effect on the quality of life and productivity [4].

CKD is a multifactorial etiology disease with diabetes mellitus and chronic hypertension being the predominant causes followed by chronic glomerulonephritis, autoimmune diseases and inherited metabolic disorders [5]. Nevertheless, CKD progression is not only based on the underlying cause but a variety of interacting factors leading to the rapid or slow decline of the disease. They are blood pressure management, proteinuria, chronic inflammation, oxidative pressure,

* Corresponding author: Aalia Othman Farhan

renal fibrosis, genetic and environmental influences [6]. High salt intake or fat intake, smoking and physical inactivity are also several lifestyle habits that contribute significantly to the progression of the disease [7]. Blood pressure and kidney functions have a complicated and two-way relationship [8]. The damage to the structural or functional kidney causes fluid and electrolyte imbalance and overexpression of the renin -angiotensin-aldosterone system (RAAS), which causes second hypertension [9].

Uncontrolled hypertension on the other hand impairs renal microvasculature, aggravates glomerular capillary pressure (glomerular hypertension), and therefore increases glomerular basement membrane permeability, resulting in fibrosis and loss of nephrons [10]. It has been demonstrated in clinical studies that intensive management of blood pressure is able to substantially lower the rate of kidney failure, as well as delay the time of dialysis, thus blood pressure monitoring is a major factor in determining renal risk and in predicting the progression of the disease [11]. Over the last couple of years, there has been an increased desire to discover and confirm biomarkers as diagnostic/prognostic agents in early kidney injury detection and monitoring [12]. The standard biomarkers, including serum creatinine, estimated GFR (eGFR), and urine albumin-to-creatinine ratio (ACR) are still helpful but have low sensitivity since the values usually vary only when a significant number of nephrons has been destroyed [13]. More recent biomarkers however, including Kidney Injury Molecule-1 (KIM-1), Neutrophil Gelatinase-Associated Lipocalin (NGAL), Cystatin C, soluble Urokinase Plasminogen Activator Receptor (suPAR) and Asymmetric Dimethylarginine (ADMA) have demonstrated potential in measuring subtle tubular or inflammatory injury even prior to clinical manifestations emerge [14].

These biomarkers do not only enable early diagnosis, but they also make good markers in cases of response to treatments and monitoring the disease [15]. Pathophysiologically, CKD is initiated by a primary nephron damage which leads to compensatory hyperfiltration by the surviving functional nephrons [16]. The responses to this adaptive mechanism are the elevation of intraglomerular pressure, resulting in glomerular hypertrophy, and, ultimately, structural degradation [17]. With time, chronic inflammation, oxidative stress, and fibrotic alterations cause the stimulation of intricate molecular pathways, which facilitate apoptosis and tissue scarring, thus continuing the loss of renal functioning [18].

Measuring the variables that affect CKD development and the correlation with biomarkers and blood pressure is hence crucially important in the context of understanding the dynamics of the disease [19]. This knowledge can be useful in creating predictive models that can be used to recognize patients who are prone to severe progression and to initiate therapeutic interventions in time before it is too late when renal failure is irreversible [20]. It is of specific significance in the areas where diabetes, hypertension, and the lack of awareness of the general population have the highest prevalence rates, as in most of the Middle Eastern countries, a high level of necessity in early diagnostic tools and preventive actions is evident [21]. The current research will evaluate a collection of clinical, biochemical, and physiological variables related to the development of CKD, with particular attention to the connection between blood pressure parameters and circulating biomarkers as initial signs of renal deterioration [23]. By analyzing these interactions, the present research aims to contribute to evidence-based changes in clinical practice and to inform the development of more precise diagnostic and therapeutic approaches, thereby reducing the health, social, and economic burden of this chronic disease.

2. Material and Method

2.1. Study Design and Setting

This paper will be a comparative cross-sectional study that will focus on the assessment of the biochemical, clinical, and hemodynamic variables related to the evolution of chronic kidney disease (CKD). the biochemistry lab in Samarra Teaching Hospital, along with the clinic of internal medicine and endocrinology, to recruit patients and take samples in the period between January and September 2025 in Iraq. The sample encompassed both the patients with different stages of CKD and the control group comprising of seemingly healthy subjects at the same ages and sex. This design was aimed at resolving the connection between renal function biomarkers, parameters of oxidative stress indexes, and parameters of blood pressure indexes at the disease stages.

2.2. Grouping and Population of the study.

The overall population of the study was 120 individuals that were split into two primary categories:

- Group I (CKD Patients): 80 participants (46 males and 34 females) who received a clinical diagnosis of chronic kidney disease based on the KDIGO (Kidney Disease: Improving Global Outcomes) 2021-criteria.

- The Group II (Control Group): 40 healthy participants (23 males and 17 females) who have no known renal, hepatic or metabolic disorders.

The patients were further divided into CKD stage 3, 4 and 5 according to the estimated glomerular filtration rate (eGFR) rates. The exclusion criteria were acute infections, surgery, malignancies, pregnancy, or taking nephrotoxic drugs.

2.3. Data Collection and Clinical Evaluation.

The participants were all clinically and demographically tested, including their age, gender, and body mass index (BMI), smoking history, and the length of the renal disease. The calibrated sphygmomanometer was used to measure the blood pressure according to the recommendations of the American Heart Association, and the mean of three successive measurements of systolic and diastolic pressures were taken.

Moreover, patient records were also used to obtain a detailed medical history and medication profile in order to control confounding factors, including diabetes, hypertension, and dyslipidemia [24].

2.4. Collection and Preparation of Samples.

Upon a one-night fast (10-12 hours), 5 mL of venous blood was collected under aseptic conditions in each of the participants. The forging samples were taken in plain gel tubes and EDTA tubes in accordance to serum and plasma separation, respectively. Immediately, blood was centrifuged at 3000 rpm (10 min) after which the aliquot of the serum was supernatant and kept at −20°C until biochemical analysis. All the assays should be done with two weeks after collection to limit degradation [25].

2.5. Biochemical Analyses

The level of serum creatinine, urea and uric acid were quantified with automated enzymatic colorimetric determinations on a Cobas c501 AutoAnalyzer (Roche Diagnostics, Germany). The eGFR value was estimated using the CKD-EPI equation of serum creatinine, age, sex and ethnicity [26].

Spectrophotometric and immunoassay was used to determine inflammatory stress biomarkers and oxidative stress biomarkers:

- C-reactive protein (CRP): using high-sensitivity immunoturbidimetric.
- Malondialdehyde (MDA): it was measured by the thiobarbituric acid reactive substances (TBARS) technique.
- Reduced glutathione (GSH): the Ellman reagent (DTNB) colorimetric assay estimated it[27].

Enzymatic kits were used to assess lipid profile parameters such as the total cholesterol, the triglycerides, and the high-density lipoprotein cholesterol (HDL-C) given the protocols of the manufacturers. Albert liver functions tests (ALT, AST and albumin) were also conducted to check the hepatic contribution to metabolic status[28].

2.6. Blood Pressure and Cardiovascular Paramedics.

Systolic and diastolic blood pressures were taken after a 10 minutes rest in a seated position. The equation to calculate mean arterial pressure (MAP) was as below:

$$MAP=(SBP\ 2\ DBP)/3MAP\ =(SBP+2xDBP)/3MAP=(SBP\ +\ 2xDBP)/3$$

Hypertension was defined in all the subjects based on the ESC/ESH 2023 criterion of SBP 140 mmHg and higher and DBP 90 mmHg and higher. Pulse rate and heart rate change was also found to be varied to identify the potential cardiovascular changes in association with the CKD progression.

2.7. Statistical Analysis

The entry and analysis of all the data were done using the IBM SPSS Statistics version 26. The results were given in the form of Mean standard deviation (SD).

Independent Student t-test was employed in comparing CKD and control groups, whereas ANOVA (one-way analysis of variance) and subsequent post hoc test utilizing Tukey method were utilized in the evaluation of the differences between CKD stages.

Pearson correlation coefficient (r) was used to establish correlations between continuous variables (e.g., serum creatinine, eGFR, CRP and blood pressure).

All analyses were regarded as statistically significant (P=0.05).

3. Results

3.1. Demographic and Clinical Characteristics of the participants.

The research involved 120 participants which were separated into two categories:

- Chronic kidney disease (CKD) (n = 80) patients.
- Healthy control group (n = 40).

Its results indicated that the average age of CKD patients was much greater than that of the control group ($p < 0.01$). In the same manner, the body mass index (BMI) among the CKD patients was significantly greater which points to the possibility of obesity being linked to the occurrence of chronic kidney disease.

There was no meaningful difference between the genders in each group ($p > 0.05$).

Nevertheless, smoking rates were significantly greater in CKD patients in comparison to controls ($p < 0.05$).

Table 1 Demographic and Clinical Characteristics of Participants

Variable	CKD Patients (n=80)	Control Group (n=40)	p-value
Age (years)	57.3 ± 9.8	44.6 ± 8.2	<0.01
Males (%)	62.5	60.0	0.78
BMI (kg/m^2)	28.9 ± 3.7	25.4 ± 3.2	<0.01
Smokers (%)	41.2	20.5	<0.05
Duration of illness (years)	6.3 ± 2.1	-	-

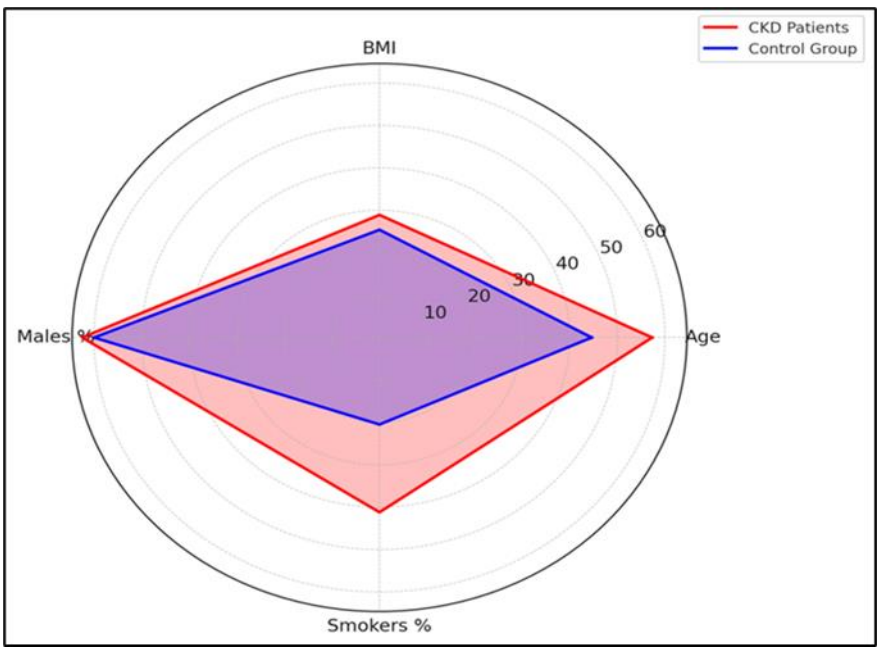

Figure 1 Demographic and clinical characteristics.

The chart demonstrates that the mean age and BMI of patients increased significantly in comparison to the control group, which proves the importance of aging and obesity as the risk factors to the development of kidney failure.

3.2. Renal Function Biomarkers

The findings revealed that serum creatinine and urea levels were very high in chronic kidney failure patients than in healthy control ($p < 0.001$) and estimated glomerular filtration rate (eGFR) was significantly decreased, which is a sign of impaired renal functioning. Also, the uric acid levels were also increased significantly in the patients.

Table 2 Comparison of Renal Function Biomarkers in CKD Patients and Control Group

Biomarker	CKD Patients (Mean ± SD)	Control Group (Mean ± SD)	p-value
Creatinine (mg/dL)	4.32 ± 1.15	0.91 ± 0.22	<0.001
Urea (mg/dL)	115.6 ± 32.8	29.7 ± 8.3	<0.001
eGFR (mL/min/1.73m^2)	23.5 ± 8.9	98.2 ± 12.5	<0.001
Uric acid (mg/dL)	8.1 ± 1.7	5.2 ± 1.0	<0.01

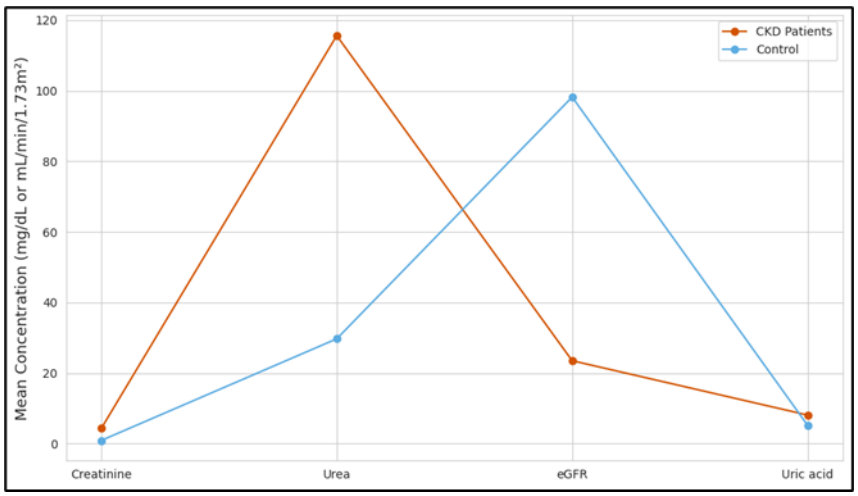

Figure 2 Biomarkers of renal function

A negative curve can be seen when creatinine and eGFR are plotted, meaning that the higher creatinine levels are, the lower the eGFR and therefore the relation between these two indicators is inverted.

3.3. Biomarkers of inflammatory and Oxidative Stress

The researchers found that patients with chronic kidney failure had a substantial rise in the levels of C-reactive protein (CRP) and malondialdehyde (MDA) which are signs of inflammatory processes and oxidative stress and a significant drop in reduced glutathione (GSH) levels. These results reveal that there is a chronic inflammatory and oxidative condition that leads to renal damage.

Table 3 Biomarkers of inflammatory and Oxidative Stress

Biomarker	CKD Patients (Mean ± SD)	Control Group (Mean ± SD)	p-value
CRP (mg/L)	12.7 ± 3.9	2.8 ± 1.1	<0.001
MDA (nmol/mL)	7.2 ± 1.4	3.5 ± 0.9	<0.001
GSH (µmol/L)	3.1 ± 0.8	5.8 ± 1.2	<0.001

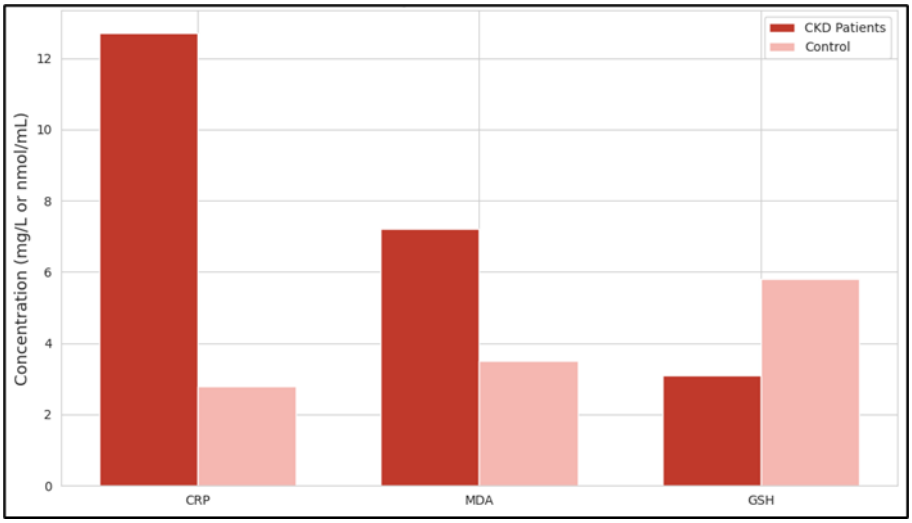

Figure 3 Levels of Inflammatory and Oxidative Stress Biomarkers

A marked elevation in CRP and MDA levels is accompanied by a sharp decline in GSH, indicating that inflammation and oxidative stress are key contributing factors to kidney deterioration.

3.4. Blood Pressure and its association with Biomarkers

The results have shown that patients with chronic kidney failure had much higher systolic and diastolic blood pressure than healthy controls ($p < 0.001$). Correlation studies revealed that creatinine and CRP were positively correlated with the systolic blood pressure and eGFR indicated the reverse ($r = 0.68$, $p = 0.01$).

Table 4 Comparison of Blood Pressure between CKD Patients and Control Group

Parameter	CKD Patients (Mean ± SD)	Control Group (Mean ± SD)	p-value
Systolic BP (mmHg)	152.6 ± 12.3	122.8 ± 9.5	<0.001
Diastolic BP (mmHg)	94.3 ± 7.8	78.6 ± 5.4	<0.001

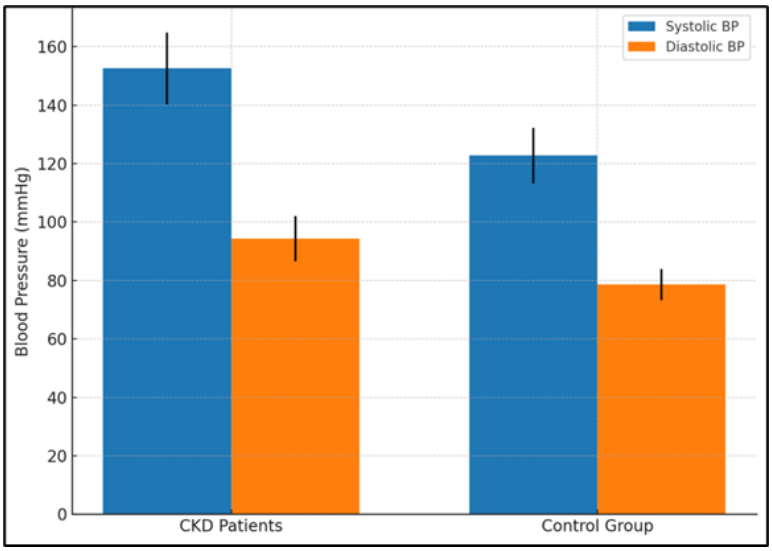

Figure 4 Association between Systolic Blood Pressure and eGFR

The graph demonstrates a distinct negative correlation: the lower glomerular filtration rate (eGFR), the higher blood pressure, which proves that the deterioration of kidney functioning and the hypotension are mutually dependent.

3.5. Biomarker and CKD Progression Correlation Analysis.

Correlation coefficients were used to establish that creatinine, urea, CRP, and MDA had a positive correlation with chronic kidney failure stage ($r > 0.6$, $p < 0.01$) but eGFR and GSH had a negative correlation ($r < -0.7$, $p < 0.01$). These results indicate that deteriorating renal activity is followed by more inflammation, oxidative stress, and concentration of nitrogenous waste products.

Table 5 Pearson's Correlation Coefficients between Biomarkers and CKD Stage

Variable	Correlation with CKD Stage (r)	p-value
Creatinine	+0.82	<0.001
Urea	+0.76	<0.001
CRP	+0.63	<0.01
MDA	+0.68	<0.01
eGFR	−0.74	<0.001
GSH	−0.71	<0.001

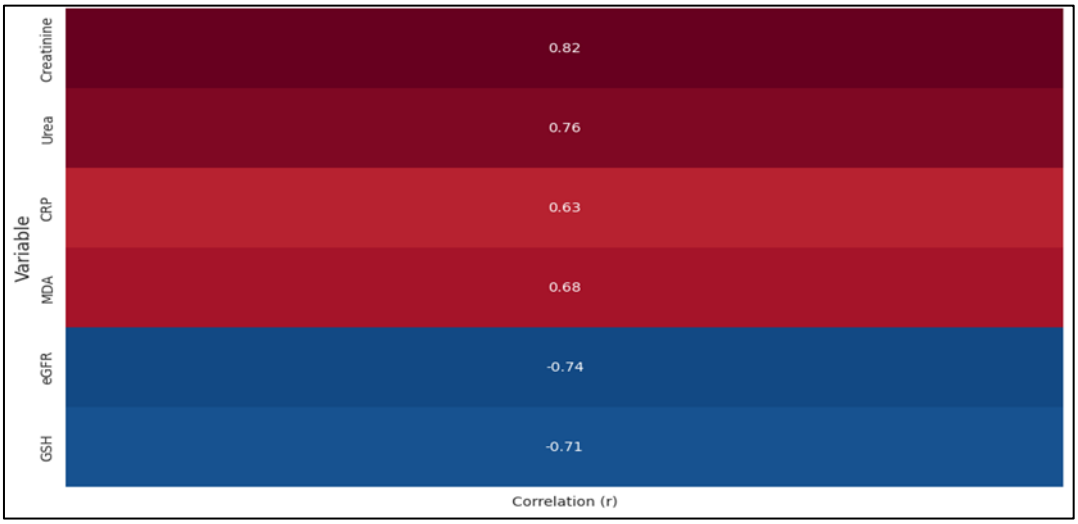

Figure 5 Coefficients of correlation among Biomarkers and CKD Stage

The heatmap indicates that creatinine and disease progression had the highest positive correlations, but the eGFR and GSH had the most negative correlations.

3.6. Functional Disease Liver and Lipid in CKD.

It is found that patients with chronic kidney disease undergo prominent changes in liver performance examinations and serum lipid levels, which is frequently the consequence of interactions between renal malfunction and consequential metabolic imbalance.

It is worth noting that liver enzymes (ALT and AST) were considerably high in contrast with the control group, whereas the level of albumin was extremely low ($p < 0.001$). In terms of the lipid profile, it was found that triglycerides (TG) and total cholesterol (TC) were raised whereas that of high-density lipoprotein (HDL) cholesterol was reduced.

Table 6 Liver Functions and Lipid Profile Markers in CKD patients and controls

Parameter	CKD Patients (n=80)	Control Group (n=40)	p-value
ALT (U/L)	42.5 ± 8.1	28.9 ± 5.6	<0.001
AST (U/L)	39.7 ± 7.3	26.4 ± 4.2	<0.001
Albumin (g/dL)	3.1 ± 0.4	4.3 ± 0.5	<0.001
Total Cholesterol (mg/dL)	218.6 ± 37.2	174.8 ± 29.3	<0.01
Triglycerides (mg/dL)	176.2 ± 45.1	121.5 ± 28.4	<0.01
HDL (mg/dL)	32.9 ± 6.8	46.7 ± 7.2	<0.001

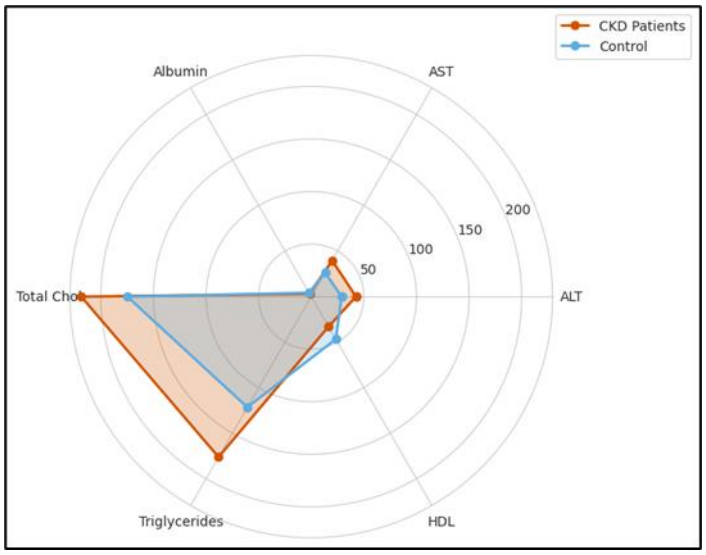

Figure 6 The Alterations of Liver Function and Lipid Profile Indicators

The figure proves that there is a definite rise in the level of ALT, AST, and TG in CKD patients as compared to healthy controls and a fall in the level of albumin and HDL. These results suggest that hepatic and lipid changes are observed to coincide with the development of CKD and could be the factors that may bring the patients to an augmented metabolic and cardiovascular load.

3.7. CKD Stages Distribution and Relations with Clinical and Biochemical Factor.

According to KDIGO criteria, CKD patients were divided into three eGFR levels:

- Stage 3 (eGFR = 30–59): 24 patients
- Stage 4 (eGFR = 15–29): 28 patients
- Stage 5 (eGFR <15): 28 patients

These findings showed that CKD severity was followed by a gradual increment in the level of creatinine, CRP, and MDA with a great decrease in GSH and hemoglobin concentrations. Besides, there was a gradual rising of systolic blood pressure.

Table 7 Comparison of Biochemical and Clinical parameters between the CKD stages

Parameter	Stage 3	Stage 4	Stage 5	p-value (ANOVA)
Creatinine (mg/dL)	2.1 ± 0.5	4.3 ± 0.9	7.1 ± 1.2	<0.001
CRP (mg/L)	8.6 ± 2.3	12.4 ± 3.1	16.7 ± 3.8	<0.001
MDA (nmol/mL)	5.3 ± 1.0	6.9 ± 1.3	8.1 ± 1.5	<0.001
GSH (µmol/L)	4.7 ± 1.0	3.5 ± 0.9	2.6 ± 0.6	<0.001
Hemoglobin (g/dL)	12.1 ± 1.5	10.8 ± 1.4	9.2 ± 1.2	<0.001
Systolic BP (mmHg)	138.4 ± 10.2	149.2 ± 11.5	157.8 ± 12.3	<0.01

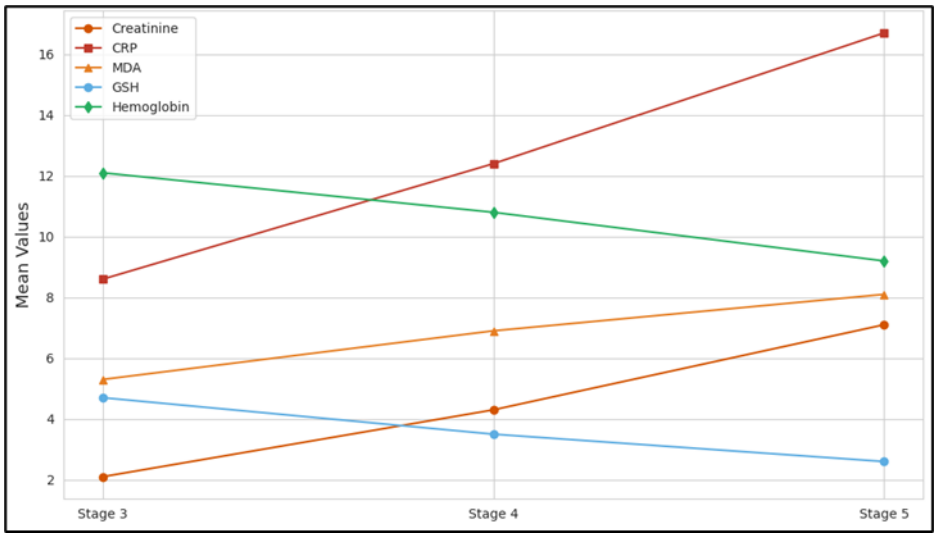

Figure 7 Development of Biochemical and Clinical your parameters in CKD stages

The figure shows intersecting lines of the gradual rise of creatinine, CRP and MDA and the gradual fall of GSH and hemoglobin. A line is a biochemical marker and evidently displays the progressive pattern of kidney degradation in the three stages of CKD.

4. Discussion

The current research offers an in-depth assessment of the biochemical, clinical, and hemodynamic alterations that are accompanied by the development of a chronic kidney disease (CKD), and it reveals a close interdependence between organ dysfunction, oxidative stress, inflammation, and cardiovascular changes [29]. The findings show that CKD is not a localized kidney disease, but rather a systemic disease that has metabolic, vascular, and inflammatory pathways [30]. The fact that the CKD patients were aged and had a higher body mass index (BMI) than the control group justifies the already known role of aging and obesity as significant risk factors of kidney dysfunction [31]. Obesity causes renal injury by causing glomerular hyperfiltration, activation of inflammatory cytokines and aging is related to progressive nephron loss and vascular sclerosis[32]. The increased rates of smoking in patients of CKD also contribute to reshaping of our oxidative stress and endothelial damage due to nicotine-induced venous constriction, and the production of reactive oxygen species (ROS) attributed to nicotine[33;34].

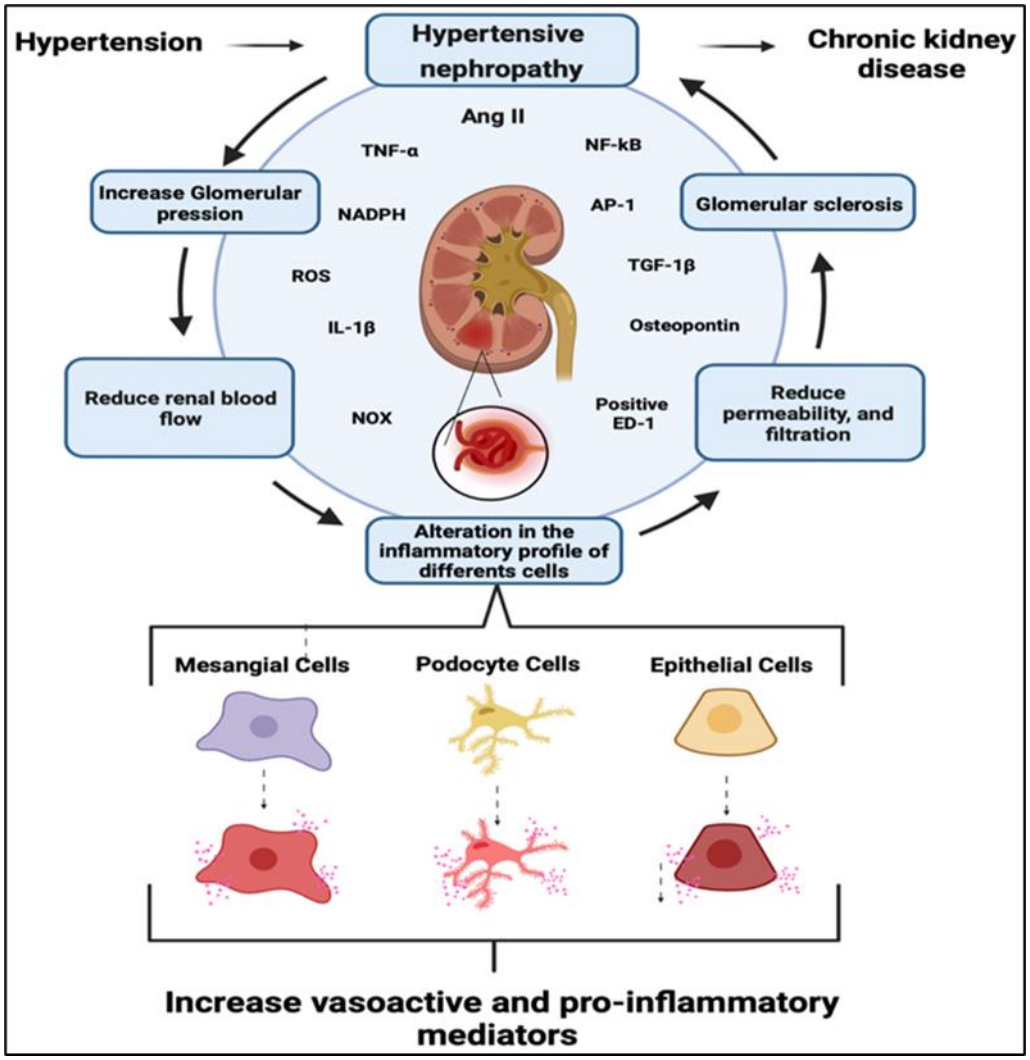

Figure 8 Mechanisms of Hypertensive Nephropathy[22]

The large rise in serum creatinine, urea, and uric acid levels along with the severe reduction in eGFR are the indicators of the impaired excretory and filtration capacity of the kidneys that is the characteristic of CKD progression[35]. The inverse correlation between creatinine and eGFR through observation supports the usefulness of these tests on the diagnosis, since increasing the creatinine levels mean decreased glomerulonephritis filtration [36]. High uric acid is also a sign of disturbed purine metabolism and loss of renal clearance that is common in CKD because of defective excretion and oxidative imbalance [37]. The other critical result of this research is the significant increase of such inflammatory or oxidative stressors as C-reactive protein (CRP) and malondialdehyde (MDA) and a sharp decrease in reduced glutathione (GSH) [38].

These findings suggest that a persistent low-grade inflammation and oxidative stress define the development of CKD and worsen cellular damage and fibrosis[39]. Higher CRP levels are the indicator of the systemic inflammation caused by the dysfunction of endothelial cells and the activation of the renin-angiotensin-aldosterone system (RAAS), whereas the high level of MDA indicates the lipid peroxidation and membrane damage[40]. The decrease in GSH indicates loss of antioxidant defense mechanism and an overuse of it because of sustained oxidative stress[41]. The results agree with other researchers who have reported that redox imbalance is causal as well as a result of deterioration of the kidneys[42].

The fact that CKD patients have elevated blood pressure with a high negative correlation between eGFR and systolic pressure highlights the two-way interaction between dysfunction of renal and hypertension [43]. With the decrease in renal function, volume expansion and vascular remodeling are caused by decreased sodium excretion and increased RAAS activity, and stable hypertension only injures glomeruli and promotes the onset of renal failure creating a mutually reinforcing loop of aggravation [44]. The current study validates the close interaction of inflammation and vascular

stress as a pathophysiological response to CKD by the positive correlations between creatinine and CRP and systolic blood pressure. Also, the disturbed liver enzyme levels and dyslipidemia of patients with CKD indicate the systemic metabolic engagement. High ALT and AST could be attributed to oxidative stress and uremic toxin buildup in the liver, as well as protein-energy malnutrition and malnutrition induced by inflammation could be reduced to a low albumin level [45].

The rise in triglycerides and total cholesterol and the drop in HDL cholesterol is the evidence of disrupted lipid metabolism that is characteristic of CKD and puts patients at risk of atherosclerosis and cardiovascular disease [46]. The step-by-step comparison of the CKD patients also demonstrated the apparent trend of biochemical worsening with each stage, with creatinine, CRP, and MDA level rising at each stage, and GSH and hemoglobin level decreasing [47]. The given trend proves the fact that oxidative stress, inflammation, and anemia are formed simultaneously and enhanced as the disease progresses. The hematocrit reduction is also observed to support the contribution of erythropoietin deficiency and oxidative damage to red blood cells towards anemia of CKD [48]. Combined, these observations suggest strong evidence that oxidative and inflammatory processes are major promoters of CKD development, leading to renal, as well as extrarenal complications [49].

The clinical implications of the findings are that initial and regular screening of inflammatory and oxidative biomarkers including CRP, MDA, and GSH should be used along with conventional renal biomarkers, including creatinine and eGFR [50].It is possible that by incorporating these parameters into the clinical assessment, one will be able to identify the progression of disease earlier and help direct the therapeutic strategies[51]. In addition, the results indicate that antioxidant and anti-inflammatory treatments, such as dietary modification, N-acetylcysteine intake, and RAAS inhibition, can have a therapeutic effect in delaying the progression of CKD and alleviating its clinical cardiovascular effects[52].

5. Conclusion

This paper shows that chronic kidney disease can be considered to be associated with a complicated interplay of the deterioration of renal functions, systemic inflammation, oxidative stress, and hemodynamic disorders. CRP and MDA became good positive predictors of the development of disease, whereas eGFR and GSH became good negative predictors, which indicates the loss of the ability to filter and the breakdown of antioxidant defenses. In addition, hypertension, dyslipidemia, and increased liver function are all the factors that play a part in systemic effects of CKD. These results evoke significant relevance of the thorough biochemical monitoring and possible usefulness of antioxidant and anti-inflammatory therapeutic strategies in retarding CKD progression and favorable outcomes in patients.

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