RESEARCH PAPER

"Comprehensive Evaluation of Hematological, Biochemical and Inflammatory Markers in Relation to Estimated Glomerular Filtration Rate (eGFR) in Pre-Dialysis Chronic Kidney Disease Patients"

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Abstract

Objective: The purpose of this study was to assess the hematological, biochemical, and inflammatory parameters in predialysis patients with CKD and their correlation with an estimated glomerular filtration rate (eGFR).

A cross-sectional study involving 60 participants (40 pre-dialysis CKD patients and 20 healthy controls) was conducted at Health Quest Imaging & Diagnostics, Gurugram, India. Hematological, biochemical, inflammatory markers were analyzed, and eGFR was calculated using the CKD-EPI equation.

Results: Compared with healthy controls, CKD patients exhibited significant hematological, biochemical, inflammatory, and electrolyte abnormalities, including reduced eGFR, elevated ESR and CRP, and significant correlations between eGFR and multiple laboratory parameters ($P < 0.05$). Univariate linear regression analysis identified serum creatinine, blood urea, hemoglobin, ESR, sodium, age, uric acid, and potassium as significant predictors associated with eGFR.

Conclusions: Hematological, biochemical and inflammatory markers have seen to be quite different in pre-dialysis CKD patients than in healthy controls. Most of these abnormalities are linked to lowered eGFR, which is indicative of systemic effects of poor renal function. Raised ESR or CRP also highlights the presence of chronic inflammation in the progression of CKD. Routine assessment of these markers may support early diagnosis and effective CKD management.

Keywords: Chronic Kidney Disease, eGFR; Hematological Parameters, Biochemical Markers; Inflammation .

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Introduction

The urinary system is important in the removal of metabolic waste, and in regulating fluid and electrolyte levels such as sodium, potassium, chloride, calcium, phosphorus and magnesium. [1]. Kidney failure is when the kidneys can no longer remove waste products and keep the body's water and mineral salt (salts) balanced [2]. CKD is a permanent worsening of the renal functioning where kidneys lose much of their filtration ability and physiological potential. Diagnosis of kidney failure is carried out by laboratory examination of kidney functions, and in other cases, by kidney biopsy [3]. The primary issue of the chronic kidney failure patients is a rise in blood acidity and urea and nitrogenous compounds level and a clinical uremia syndrome [4]. The prevalence of chronic kidney disease (CKD) in the Middle East has more recently been reported to be very high, about 100,000 people are affected, and a prevalence of 430 cases per million population (c/million) has been estimated [5]. Chronic kidney disease (CKD) is a progressive disease of

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the kidneys in which the kidneys are damaged over time. Hypertension, diabetes mellitus, and genetic predisposition are the major risk factors. The more a kidney fails, the more likely other problems will occur, including cardiovascular disease and anemia [6]. Early detection and treatment are crucial to mitigate the progression of CKD and improve patient outcomes; CKD is a progressive disease with no cure and high morbidity and mortality that is common in the general adult population, especially in patients with DM and HTN [7]. Increased fluid in the body (which causes swelling), elevated acid levels, elevated potassium and phosphate levels, decreased calcium levels, and anemia in later stages are all possible issues with renal failure [8]. Endstage renal illness is often the consequence of chronic kidney disease (CKD). According to [9], Chronic kidney disease is characterized by kidney damage or a decline in kidney function lasting for three months or longer, and it is more common among individuals with diabetes, high blood pressure, and older adults.

eGFR is a creatinine-based estimate of kidney function, which is a reflection of the glomerular filtration rate (GFR). It is a crucial parameter for the diagnosis and monitoring of CKD progression. [10]. The early diagnosis of chronic kidney disease is often based on the measurement of blood urea and creatinine, but creatinine is an insensitive marker of early renal disease as it does not increase until there is already substantial renal damage (that is, about 40–50% of renal function) [11]. Thus, early kidney injury can go undiagnosed and untreated, causing a delay in diagnosis and treatment. [12]. The study of hematological, biochemical, electrolyte as well as inflammatory parameters in relation to eGFR in pre-dialysis CKD patients is scarce in the Indian population based studies. This comprehensive evaluation might help to better understand the severity of the disease and systemic complications associated with worsening renal function.

Is there a significant correlation between hematological, biochemical, and inflammatory markers and the estimated glomerular filtration rate (eGFR) in patients with chronic kidney disease prior to dialysis compared to healthy individuals?

Aim of the Study

The aim of this study was to compare the revelance of haematological, biochemical and inflammatory markers with estimated glomerular filtration rate in patients with pre-dialysis chronic kidney disease with healthy individuals.

Material and Methods

Ethical Committee Approval

The research study was approved by the Ethics Decision Committee of Health Quest and Diagnostic in June 2025 with Decision number 00198 .

Study Area and Design

The study involved 60 participants (40 patients with CKD and 20 healthy controls) from Health Quest Imaging & Diagnostics, Gurugram, India, from June to November 2025. This was an exploratory, cross-sectional study that enrolled all eligible CKD patients who presented to the center during the study period in a consecutive manner. The CKD group consisted of 25 males and 15 females (mean age 31–82 years) and the control group comprised 10 males and 10 females (mean age 27–81 years). Standardised conditions were observed for the collection of venous blood samples. Sample

Collection Sample

Collection

Demographic and clinical data—such as age, gender, blood pressure, blood type, and serological status regarding Hepatitis B virus (HBV), Hepatitis C virus (HCV), and Human Immunodeficiency Virus (HIV)—were recorded. Venous blood samples (total volume: 5 mL) were collected from both chronic kidney disease patients and a group of healthy individuals (control group). The collected blood samples were aliquoted into three separate tubes: 1 mL was placed in an EDTAcontaining tube for hematological analysis (white blood cells, red blood cells, platelet count, hemoglobin, and hematocrit); 2 mL of whole blood was used to measure the erythrocyte sedimentation rate (ESR); and 3 mL of whole blood was transferred to a plain tube containing gel (without anticoagulant) for biochemical analysis. Following clot formation, the samples were centrifuged at 3000 rpm for 10 minutes; the separated serum was then collected and stored at 2–4°C pending biochemical analysis.

Blood pressure measurements were obtained using a calibrated sphygmomanometer after participants had rested in a seated position for at least 5 minutes. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded according to standard clinical procedures.

Procedures Experiments Laboratory

Estimation of the Haematological Parameters: Complete blood count (CBC) was done to assess hematological parameters such as red blood cells (RBC), white blood cells (WBC), platelets, hemoglobin and hematocrit. All measurements were performed with an Erba H560 Hematology Analyzer, following the manufacturer's instructions.

Estimation of Biochemical Parameters:

Estimate of the Blood Glucose level: Enzymatic procedures are chosen because they are easier, more precise, and more specific. The most used technique for estimating glucose among them is the GOD-POD approach.

Estimation of the Erythrocyte Sedimentation Rate: Westergren Method Widely used method for ESR estimation, its common method that we are going to use in today laboratory

Estimation of the Blood Urea: by using Berthelot Method kit (DELTA UREA-BERTHELOT Company)

Estimation of the CREATININE Concentration by Using JAFFE KINETIC Method

Estimation of the Glomerular Filtration Rate: by using CKD-EPI 2021 Equation is: eGFR=142×min(Scr/k,1)α×max(Scr/k,1)−1.200×0.9938 Age×(1.012 if female)

Estimation of the Uric acid in the serum / plasma by using Uricase method

Estimation of the Sodium and Potassium Level in the Serum by using Electrolyte test kit

Estimation of the Chloride level in the serum / Plasma by using SBIO KIT (Thiocyanate Method) invitro diagnostic .

Estimation of the C –Reactive Protein : Serum C-reactive protein (CRP) levels were quantitatively determined using an automated latex-enhanced immunoturbidimetric assay (ERBA Diagnostics), in accordance with the manufacturer's instructions.

Statistical Analysis: Continuous variables were expressed as mean ± SD. Normality was assessed using the Shapiro–Wilk test. Comparisons between two groups were performed using the independent samples t-test, while comparisons across CKD stages were performed using one-way ANOVA (or Kruskal–Wallis test for nonnormally distributed data). Spearman's rank correlation was used to assess associations between laboratory parameters and eGFR. Univariate linear regression analysis was subsequently performed to identify

Results

variables significantly associated with eGFR. Variables with $P < 0.05$ were considered candidates for further multivariable analysis. A two-sided P-value < 0.05 was considered statistically significant.

Table 1. Demographic Characteristics of the Study Participants According to Age Group and Sex

Age Group (Years)	Males, n (%)	Females, n (%)	Total, n (%)
20–39	6 (10.0)	4 (6.7)	10 (16.7)
40–59	12 (20.0)	8 (13.3)	20 (33.3)
60–79	15 (25.0)	11 (18.3)	26 (43.3)
≥80	2 (3.3)	2 (3.3)	4 (6.7)
Total	35 (58.3)	25 (41.7)	60 (100.0)

Data are presented as number (n) and percentage (%). Percentages were calculated relative to the total study population (N = 60). Age groups represent the combined distribution of CKD patients and healthy controls.

Table No 2. Distribution of CKD Patients According to eGFR Stages

CKD Stage	eGFR (mL/min/1.73 m²)	Number of Patients (n)	Percentage (%)
G1	≥ 90	0	0.0
G2	60–89	1	2.5
G3a	45–59	3	7.5
G3b	30–44	11	27.5
G4	15–29	9	22.5
G5	< 15	16	40.0
Total	—	40	100.0

eGFR was calculated using the CKD-EPI equation. CKD stages were classified according to KDIGO guidelines.

Table 3.Comparison of Hematological, Biochemical, Inflammatory, and Electrolyte Parameters According to CKD Stage

Parameter	G2 (≥60)(n=1)	G3a (45–59)(n=3)	G3b (30–44)(n=11)	G4 (15–29)(n=9)	G5 (<15)(n=16)
Hemoglobin (g/dL)	12.79	6.60	9.84	9.93	9.08
ESR (mm/hr)	11.25	44.00	39.92	46.50	47.83
Blood Urea (mg/dL)	25.47	52.00	74.70	113.34	135.36
Serum Creatinine (mg/dL)	0.81	1.31	1.78	3.21	6.20
Sodium (mmol/L)	139.90	136.00	134.67	136.70	134.92
Potassium (mmol/L)	4.11	5.00	4.72	4.68	4.62
Chloride (mmol/L)	106.20	110.00	106.42	106.60	107.58
CRP Positive, n (%)	0 (0.0%)	0 (0.0%)	10 (90.9%)	9 (100.0%)	16 (100.0%)

CKD stages were classified according to the KDIGO guidelines based on estimated glomerular filtration rate (eGFR).

Continuous variables are presented as mean values; CRP is presented as number (percentage) of positive cases.

ESR: erythrocyte sedimentation rate; CRP: C-reactive protein.

Table No 4 . Clinical Characteristics of CKD Patients

Parameter	CKD Patients (N=40)
Diabetes Mellitus	31 (77.5%)
Hypertension	33 (82.5%)
Systolic Blood Pressure (mmHg)	148.2 ± 10.2
Diastolic Blood Pressure (mmHg)	92.4 ± 6.8

Clinical characteristics of the CKD patients are summarized in Table 3. Among the 40 CKD patients, 31 (77.5%) had diabetes mellitus and 33 (82.5%) had hypertension, with mean systolic and diastolic blood pressure values of 148.2 ± 10.2 mmHg and 92.4 ± 6.8 mmHg, respectively .

Haematological Parameters:

Table 5 and Figures (1–3) compare hematological parameters (RBC, WBC, Hb, PCV, and PLT) between predialysis CKD patients and healthy controls.

Parameter	Healthy Controls (N=20)	CKD Patients (N=40)	P-value
RBCs ($\times 10^6/\mu L$)	4.05 ± 0.43	3.05 ± 0.58	< 0.05

WBCs ($\times 10^3/\mu L$)	7.10 ± 1.44	14.00 ± 2.02	< 0.01
Hemoglobin(g/dL)	13.59 ± 1.20	7.56 ± 0.61	< 0.001
Packed Cell Volume (%)	40.37 ± 0.73	27.23 ± 1.10	< 0.001
Platelets ($\times 10^3/\mu L$)	303.1 ± 10.3	120.1 ± 9.41	< 0.01

Data are presented as mean ± SD. Statistical comparisons were performed using an independent samples t-test, with $p < 0.05$ considered statistically significant.

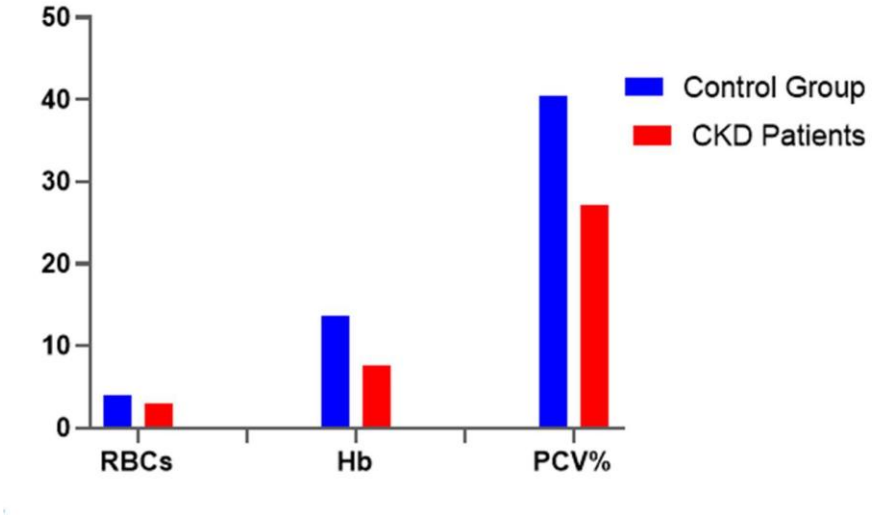

Figure -1 RBCs, Hb and PCV levels in CKD patients compared with healthy control

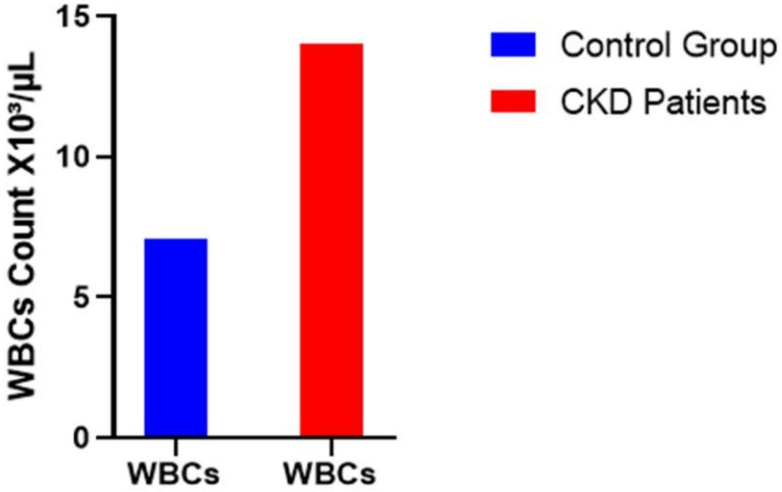

Figure -2 WBCs levels in CKD patients compared with healthy controls

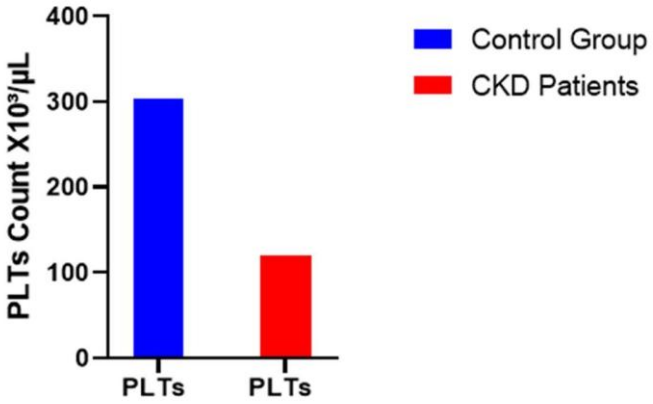

Figure -3 Platelets levels in CKD patients compared with healthy controls

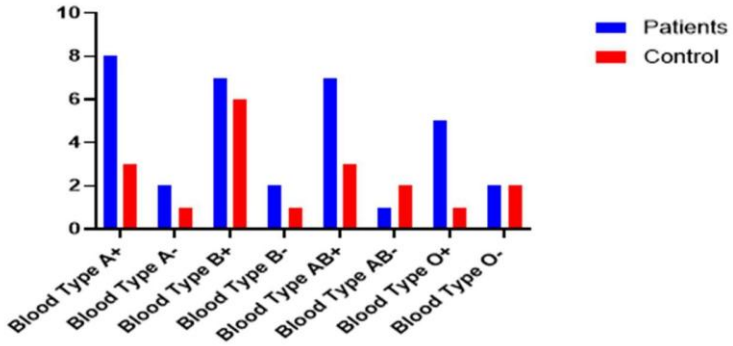

Figure No-4 Distribution of the blood group between the CKD patients and Healthy group

Biochemical Parameters:

Table 6 and Figure (5- 8) present the comparison of biochemical parameters between pre-dialysis CKD patients and healthy control.

Parameter	Healthy Group (N =20)	CKD Group (N =40)	Statistical Test	P-value
Blood Urea (mg/dL)	25.38 ± 1.63	90.3 ± 5.0	Independent samples t-test	< 0.001
Serum Creatinine (mg/dL)	0.77 ± 0.01	3.69 ± 0.53	Independent samples t-test	< 0.001
Uric Acid (mg/dL)	4.51 ± 0.20	7.82 ± 0.64	Independent samples t-test	< 0.001
Blood Glucose (mg/dL)	96.8 ± 0.72	239.0 ± 1.70	Independent samples t-test	< 0.001

Data are expressed as mean ± SD. Differences between groups were analyzed using an independent samples t-test. Statistical significance was set at $p < 0.05$.

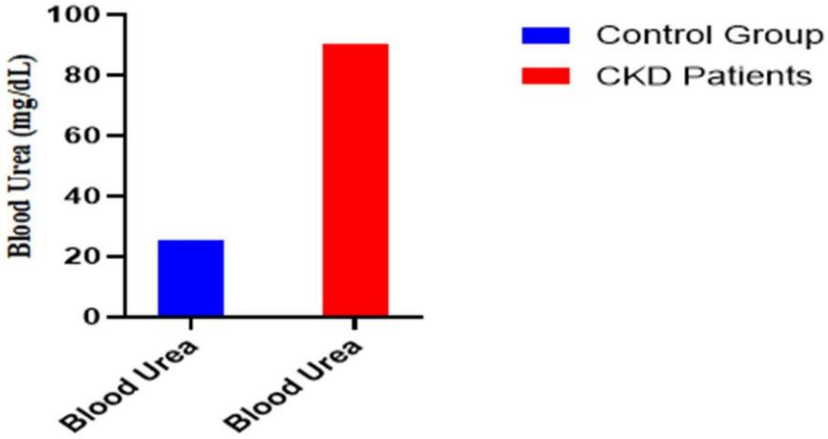

Figure -5 B.Urea levels in CKD patients compared with healthy controls

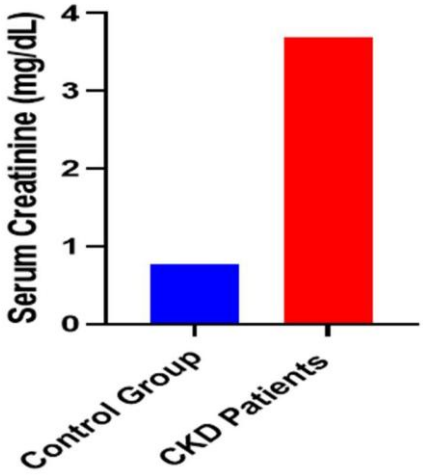

Figure -6 Creatinine Levels in CKD patients compared with healthy controls

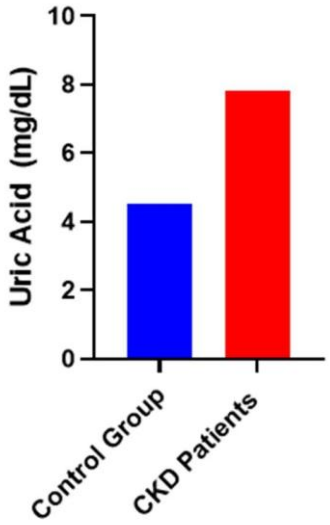

Figure-7 Uric Acid levels in CKD patients compared with healthy controls

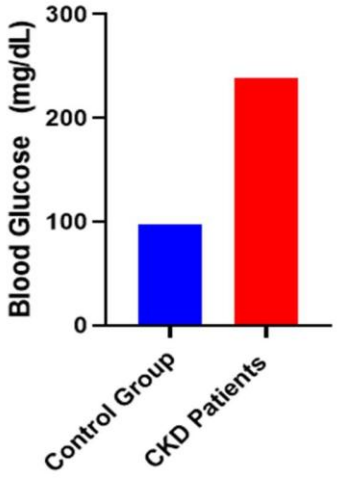

Figure-8 Blood Glucose levels in CKD patients compared with healthy controls

Table 7 and Figures)9–11(present the comparison of electrolyte levels between pre-dialysis CKD patients and healthy controls.

Parameter (mmol/L)	Healthy Controls (Mean ± SD)	CKD Patients (Mean ± SD)	P-value
Sodium (Na^+)	144.0 ± 1.2	132.3 ± 1.4	0.001 >
Potassium (K^+)	3.3 ± 0.7	5.7 ± 2.8	0.001 >

Chloride (Cl^-)	105.0 ± 2.3	108.0 ± 3.1	0.001

Data are expressed as mean ± SD. Statistical significance was determined using an independent samples t-test ($p < 0.05$).

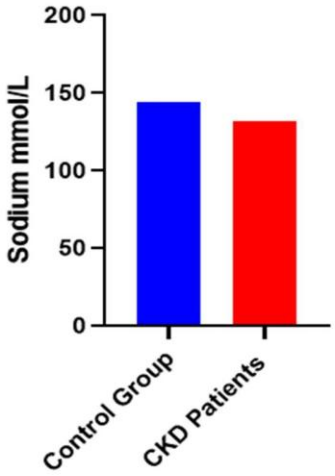

Figure -9 Sodium levels in CKD patients compared with healthy controls

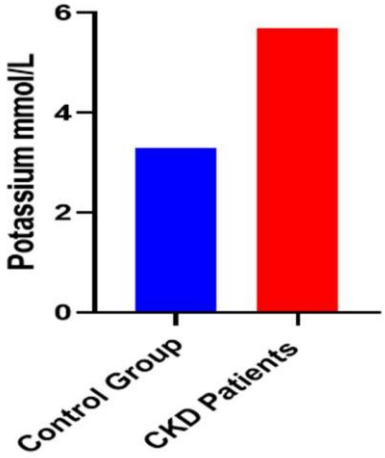

Figure-10 Potassium levels in CKD patients compared with healthy controls

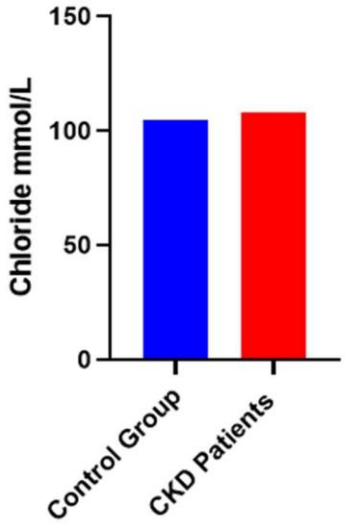

Figure -11 Chloride level in CKD patients compared with healthy control

Table 8 and Figure)12–14 (present the comparison of inflammatory markers between pre-dialysis CKD patients and healthy controls.

Parameter (mmol/L)	Healthy Controls (Mean ± SD)	CKD Patients (Mean ± SD)	P-value
Sodium (Na^+)	144.0 ± 1.2	132.3 ± 1.4	0.001 >
Potassium (K^+)	3.3 ± 0.7	5.7 ± 2.8	0.001 >
Chloride (Cl^-)	105.0 ± 2.3	108.0 ± 3.1	0.001

Data are expressed as mean ± SD. ESR: erythrocyte sedimentation rate; CRP: C-reactive protein. Statistical significance was set at $p < 0.05$.

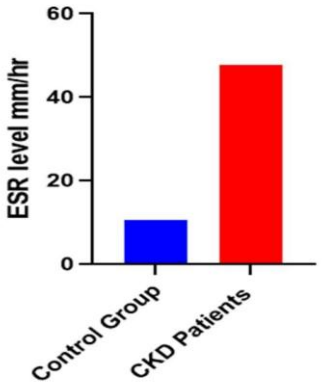

Figure -12 ESR levels in CKD patients compared with healthy controls

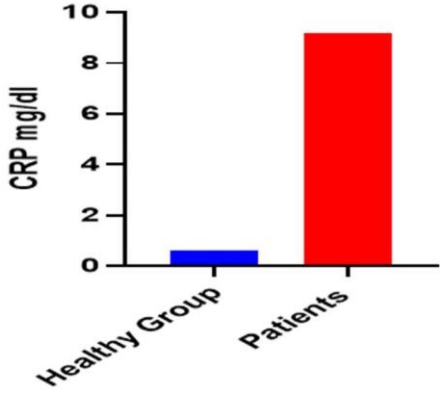

Figure -13 CRP levels in CKD patients compared with healthy controls

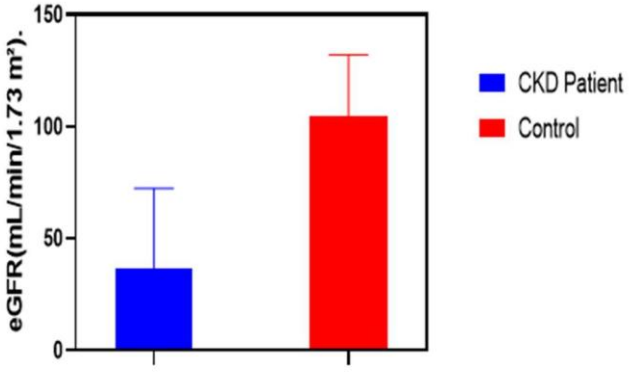

Figure-14 eGFR levels in CKD patients compared with healthy controls

Table No - 9 Correlation between Rate eGFR and Hematological Parameters.

Variable	Correlation Coefficient (ρ)	P-value	Interpretation
RBCs ($\times 10^6/\mu L$)	-0.232	0.406	Not Significant

WBCs ($\times 10^3/\mu L$)	-0.618	0.014	Significant*
Hemoglobin (g/dL)	-0.225	0.421	Not Significant
Hematocrit (%)	0.331	0.228	Not Significant
Platelets ($\times 10^3/\mu L$)	0.076	0.790	Not Significant

Table No-10 Correlation between (eGFR) and Renal Failure tests

Variable	Correlation Coefficient (ρ)	P - value	Interpretation
Urea (mg/dL)	-0.629	0.012	Significant inverse correlation
Creatinine(mg/dL)	-0.753	0.001	Strong significant inverse correlation
Uric Acid (mg/dL)	-0.694	0.004	Significant inverse correlation

Table No – 11 Correlation between (eGFR) and Inflammatory Markers

Variable	Correlation Coefficient (ρ)	P-value	Interpretation
ESR (mm/hr)	-0.523	0.046	Significant inverse correlation*
CRP (mg/dL)	-0.681	0.005	Strong significant inverse correlation**

Table 12. Linear Regression Analysis of Factors Associated with Estimated Glomerular Filtration Rate (eGFR) in Pre-Dialysis Chronic Kidney Disease Patients

Independent Variable	Direction of Association with eGFR	Pvalue	Statistical Interpretation
Serum Creatinine	Negative	<0.001	Highly significant
Blood Urea	Negative	<0.001	Highly significant
Erythrocyte Sedimentation Rate (ESR)	Negative	<0.001	Highly significant
Hemoglobin	Positive	<0.001	Highly significant
Sodium	Positive	<0.001	Highly significant
Age	Negative	<0.001	Significant
Uric Acid	Negative	<0.001	Highly significant
Potassium	Negative	0.003	Significant
White Blood Cell Count (WBC)	Negative	0.081	Not statistically significant
Platelet Count	Positive	0.578	Not statistically significant

Data were analyzed using univariate linear regression with estimated glomerular filtration rate (eGFR) as the dependent variable. A two-tailed P-value < 0.05 was considered statistically significant. Variables demonstrating statistical significance in the univariate analysis were considered candidates for inclusion in the subsequent multiple linear regression model.

Discussion

The present study showed that the prevalence of anemia was significantly higher in patients with chronic kidney disease (CKD) than in healthy controls. The red blood cell (RBC) count, hemoglobin (Hb), packed cell volume (PCV) and hematocrit (HCT) were significantly lower in CKD group than in control group (P<0.05). This was also reflected in a lower count of platelets, but not as significantly (< 0.05). These results agree with the results of other studies that reported anemia is a frequent complication of impaired renal function, and also with the results of McClellan et al. [13] who reported a high prevalence of anemia among CKD patients. Normocytic normochromic anemia is the most common anemia type seen in CKD, which is thought to be due to the decreased production of erythropoietin by the diseased kidneys, since this hormone is responsible for erythropoiesis in the bone marrow [14]. Diabetic nephropathy and hypertension are common conditions associated with CKD in most parts of the world. In the current study, the prevalence of diabetes mellitus was 77.5 % and of hypertension was 82.5 %, indicating that these two conditions play a significant role in the pathogenesis and complication of renal dysfunction. The mean platelet count in the CKD group [120 × 10^3/μL] was not suggestive of general bleeding risk, but there was a significant difference between the platelet counts in the two groups, with the mean count in the CKD group below the level generally considered to be associated with a risk of spontaneous bleeding. This is an area requiring further investigation to understand the implications for clinical practice. White blood cell counts were significantly elevated in the CKD group compared with the control group (P < 0.05), in contrast to previous studies that showed that the total white blood cell count is mildly elevated in patients with CKD. The mean levels of blood urea, creatinine, and uric acid levels were significantly higher in the patients with CKD than in controls (P ≤ 0.05), which is consistent with previous studies [15, 16, 17]. The findings of these studies suggest that urea, creatinine and uric acid are the most abundant nitrogenous wastes excreted by the kidneys and their levels are elevated in CKD due to decreased renal clearance of nitrogenous wastes. Some studies found that serum urea and serum creatinine were both important indicators of acute and chronic renal failure, but others showed that serum creatinine was the better indicator of renal function in all renal insufficiency, because the serum urea concentration would be more affected by the non-renal physiological and dietary factors. [19] The level of blood glucose was also significantly elevated in the CKD patients as compared to the control, which corroborated with studies conducted earlier in the year 2006 and 2011 in India among CKD patients [20, 21, 22] . In addition, there was a significant (P ≤ 0.05) difference in potassium concentration between CKD and control groups. Due to the involvement of kidneys in regulating fluid balance, electrolytes and acid base status, CKD often leads to abnormalities like hyperkalemia, metabolic acidosis and can contribute to complications such as mineral bone disorder, muscle wasting, and vascular calcification [23]. Hyperkalemia is known to be a common and life-threatening electrolyte dysfunction in CKD [24]. The chloride level was also slightly higher in the CKD patients than in the controls. This hyperchloremia can be caused by either a water retention or a loss of free water in excess and may be additionally affected by the decrease of the glomerular filtration rate. The serum sodium levels were lower in the CKD group, however, which may indicate poor sodium control in the kidneys, secondary to dysfunction in the aldosterone control of sodium levels, and lead to increased renal sodium losses [25]. Erythrocyte sedimentation rate (ESR), which is a broad-spectrum inflammatory marker, was also elevated in CKD patients, indicating that there is an acute-phase response to inflammation that is mediated by the presence of more pro-inflammatory cytokines and their receptors in circulation. This agrees with work by Maradit-Kremers et al. 2007 [26] who reported that inflammatory markers, functional status and laboratory indices of immune activation such as ESR are useful for the assessment of disease activity in renal conditions; however, ESR is not disease-specific but remains a convenient, cost-efficient marker of the acutephase response, as reported by Peyton et al. 2015 [27]. CKD patients had significantly higher levels of Creactive protein (CRP) than healthy controls, and were associated with higher levels of serum creatinine, indicating poorer renal function. Increased systemic inflammatory state may be linked to the deteriorating renal function, as indicated by the parallel rise in both CRP and creatinine, possibly because of the build-up of uremic toxins and decreased clearance of inflammatory mediators. Chronic inflammation can, in turn, worsen kidney damage, indicating a two-way relationship between inflammation and kidney dysfunction. Moreover, the group of patients with increased CRP levels had lower estimated glomerular filtration rate (eGFR) levels, consistent with a hypothesis of a link between impaired renal function and increased systemic inflammation due to impaired clearance of inflammatory mediators and uremic toxins. This inverse association highlights the possibility of a bidirectional association between renal function and systemic inflammation and serves as a foundation for further investigation into the connection between lower renal function and higher levels of systemic inflammation. This indicates that CRP might be a valuable indicator of the severity of disease in CKD, but larger prospective studies

are needed for confirmation of its clinical value. [28] The present study further extended the analysis by applying univariate linear regression to identify laboratory parameters associated with renal function. The findings demonstrated that serum creatinine, blood urea, inflammatory markers, and hemoglobin were significantly associated with eGFR, suggesting that deterioration of renal function is influenced by multiple hematological, biochemical, and inflammatory factors rather than a single biomarker.The use of simple linear regression analysis enabled the identification of variables most strongly associated with a decline in the estimated glomerular filtration rate (eGFR) prior to constructing the multiple regression model. The results indicate that markers of renal function—such as creatinine and urea— as well as indicators of inflammation and anemia, are clearly associated with the deterioration of kidney function; this confirms that a decline in eGFR is influenced by multiple interrelated pathological factors rather than by a single biomarker alone.

Conclusions

CKD patients showed significantly reduced RBCs, PCV, Hb, and platelet counts, along with elevated WBC counts, compared with healthy controls ($P \leq 0.05$).

CKD patients exhibited significantly higher blood glucose, urea, creatinine, and uric acid levels, along with significantly lower eGFR values, compared with healthy controls ($P < 0.05$).

Significant electrolyte disturbances were observed in CKD patients, characterized by decreased sodium and increased potassium and chloride levels compared with controls ($P < 0.05$).

On the other side the results showed CKD patients ssignificantly higher ESR and CRP levels than controls, reflecting increased inflammation.

Regression analysis further demonstrated that serum creatinine, blood urea, hemoglobin, ESR, and electrolyte abnormalities were significantly associated with reduced eGFR, highlighting their potential value as laboratory indicators for assessing CKD severity before dialysis

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