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Assessment of pathological and biochemical investigations in CKD patients on hemodialysis

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Abstract:

Chronic kidney disease (CKD) is a progressive disorder characterized by gradual loss of renal function, leading to metabolic, hematological and biochemical disturbances. Hemodialysis, a common renal replacement therapy, can influence these parameters, impacting patient outcomes. This cross-sectional study included 110 CKD patients undergoing maintenance hemodialysis and 101 age- and sex-matched healthy controls. Demographic data were collected and blood samples were analyzed for hematological indices and biochemical indices. The CKD patients had higher age and significantly elevated glycemic and renal parameters. Hematological assessment showed anemia, increased RDW, leukocytosis and altered platelet indices. Biochemical evaluation revealed hypoalbuminemia, dyslipidemia, disturbed mineral and iron metabolism and elevated CRP compared to controls (all $p < 0.05$). The CKD and hemodialysis are associated with profound metabolic, hematological and inflammatory alterations, showing the need for comprehensive laboratory monitoring to improve clinical management.

Keywords: Chronic kidney disease (CKD), hemodialysis, CBC, protein profile, lipid profile, renal function tests, inflammation, dyslipidemia

Background:

Chronic kidney disease (CKD) is a progressive and irreversible condition characterized by gradual loss of renal function, leading to the accumulation of metabolic waste products and disturbances in fluid, electrolyte and acid-base balance [1]. CKD represents a significant public health concern worldwide due to its increasing prevalence and association with high morbidity and mortality. Patients with advanced CKD often require renal replacement therapy, such as hemodialysis, to maintain metabolic stability and sustain life [2]. CKD is frequently associated with multiple metabolic and hematological abnormalities. Impaired renal function leads to elevated levels of urea, creatinine and uric acid, reflecting reduced excretory capacity [3]. Hemodialysis patients often exhibit anemia, altered red blood cell indices and immune cell dysfunction due to impaired erythropoiesis, chronic inflammation and oxidative stress [4]. Additionally, protein-energy malnutrition is common, reflected in lower serum albumin and total protein levels, while lipid metabolism is frequently disturbed, with dyslipidemia contributing to increased cardiovascular risk [5]. Several studies have showed the interplay between CKD, inflammation and cardiovascular disease. Parameters such as C-reactive protein (CRP), ferritin and iron-binding capacity are often altered, reflecting chronic inflammation and iron metabolism disturbances [6]. Glycemic dysregulation is also commonly observed, especially in diabetic CKD patients, with elevated fasting blood sugar, postprandial blood sugar and HbA1c levels [7]. With this background this study aimed to evaluate complete blood count (CBC), protein profile, lipid profile and renal function tests (RFTs) in CKD patients undergoing maintenance hemodialysis and to compare these parameters with age- and gender-matched healthy controls. The findings provide insight into the systemic effects of CKD and the impact of hemodialysis on these metabolic and hematological parameters [8]. Therefore, it is of interest to design the assessment of CBC, Protein Profile, Lipid Profile and Renal Function Tests in CKD Patients on Hemodialysis.

Methods:

This study was conducted as a comparative cross-sectional analysis to assess complete blood count (CBC), protein profile, lipid profile and renal function tests (RFTs) in patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis. A total of 211 participants were enrolled, including 110 CKD patients and 101 age- and gender-matched healthy individuals (controls). Participants were selected using purposive sampling based on predefined inclusion and exclusion criteria. Inclusion criteria for cases comprised adult patients diagnosed with CKD stage 4-5 who had been on maintenance hemodialysis for at least three months. Exclusion criteria included patients with acute kidney injury, recent infections, malignancy, autoimmune disorders, or those receiving immunosuppressive therapy. After obtaining informed consent, demographic data including age, sex and relevant clinical history were recorded for all participants. Venous blood samples were collected under aseptic conditions and processed to measure hematological parameters (haemoglobin, red blood cell count, haematocrit, MCV, MCH, MCHC, RDW-CV, WBC count with differential, platelets, PDW, PCT, MPV), biochemical markers (total protein, albumin, globulin, urea, creatinine, uric acid, calcium, phosphorus, iron, TIBC, ferritin, CRP), lipid profile (total cholesterol, triglycerides, HDL) and glycemic indices (fasting blood sugar, postprandial blood sugar, HbA1c) using standardized laboratory methods. Hematological parameters were measured using an automated haematology analyzer, whereas biochemical and lipid parameters were assessed through automated biochemistry analysers with appropriate quality controls. Data were compiled and results were expressed as mean $\pm$ standard deviation for continuous variables and as frequency and percentage (n, %) for categorical variables. Statistical comparisons between case and control groups were performed using independent t-tests or chi-square tests where appropriate, with p-values < 0.05 considered statistically significant. The methodology adhered to institutional ethical guidelines and the study protocol was approved by the Institutional Ethics Committee.

Results:

Table 1 illustrates the comparison of demographic variables between groups. The demographic analysis showed that CKD patients on hemodialysis had a higher mean age (46.7±12.8 years) compared to controls (41.6±13.5 years, $p=0.005$). Female participants accounted for 43.6% of cases and 31.7% of controls, while males represented 56.4% and 68.3%, respectively, with no significant difference in sex distribution ($p=0.074$). **Table 2** shows the comparison of glycemic parameters between groups. The glycemic parameters were significantly elevated in CKD patients, with HbA1c of 7.3±1.1% versus 5.5±0.5% in controls ($p<0.001$), fasting blood sugar 98.5±19.8 mg/dL versus 84.9±7.7 mg/dL ($p<0.001$) and postprandial blood sugar 197.0±33.2 mg/dL versus 97.9±19.6 mg/dL ($p<0.001$). **Table 3** indicates comparison of renal parameters between groups. The renal function tests revealed markedly higher urea (84.0±40.1 mg/dL vs 19.0±5.2 mg/dL, $p<0.001$), creatinine (5.8±2.5 mg/dL vs 0.7±0.1 mg/dL, $p<0.001$) and uric acid (8.3±9.8 mg/dL vs 5.4±1.1 mg/dL, $p=0.003$) in CKD patients compared to controls. **Table 4** shows that comparison of hematological parameters between groups. Hematological assessment showed significant anemia and altered blood indices in cases, with lower hemoglobin, RBC count, hematocrit, MCV, MCH and MCHC (all $p<0.001$), higher RDW-CV (16.3±2.7% vs 14.0±1.4%, $p<0.001$), increased WBC and neutrophils and reduced lymphocytes ($p<0.001$). Platelet indices PDW (16.2±0.6 vs 12.9±2.0, $p<0.001$) and MPV (10.1±1.3 vs 9.4±1.6, $p=0.001$) were also elevated, while monocytes, eosinophils, platelets and PCT showed no significant differences. **Table 5** indicates that comparison of biochemical and lipid parameters between groups. The biochemical and lipid profiles revealed lower total protein (6.4±1.0 g/dL vs 7.0±0.7 g/dL, $p<0.001$) and albumin (3.3±0.7 g/dL vs 3.9±0.8 g/dL, $p<0.001$) in cases, with globulin similar ($p=0.427$). CKD patients exhibited dyslipidemia with higher total cholesterol (166.4±42.5 mg/dL vs 139.7±19.8 mg/dL, $p<0.001$), triglycerides (172.2±64.9 mg/dL vs 74.8±13.4 mg/dL, $p<0.001$) and lower HDL (33.6±11.3 mg/dL vs 45.3±8.1 mg/dL, $p<0.001$). Mineral and iron parameters were altered, with reduced calcium and iron, increased phosphorus, decreased TIBC and elevated ferritin (all $p<0.001$). Inflammatory marker CRP was significantly higher in cases (59.2±85.1 mg/L vs 2.4±0.7 mg/L, $p<0.001$).

Table 1: Comparison of demographic variables between groups (n=211)

Parameter	Case (n=110)	Control (n=101)	Total (n=211)	P-value
Age (years)	46.7±12.8	41.6±13.5	44.2±13.4	0.005
Female	48 (43.6%)	32 (31.7%)	80 (37.9%)	0.074
Male	62 (56.4%)	69 (68.3%)	131 (62.1%)	

Table 2: Comparison of glycemic parameters between groups

Parameter	Case (Mean ± SD)	Control (Mean ± SD)	p-value
HbA1c	7.3±1.1	5.5±0.5	<0.001
FBS	98.5±19.8	84.9±7.7	<0.001
PPBS	197.0±33.2	97.9±19.6	<0.001

Table 3: Comparison of renal parameters between groups

Parameter	Case (Mean±SD)	Control (Mean±SD)	p-value
Urea	84.0±40.1	19.0±5.2	<0.001

Creatinine	5.8±2.5	0.7±0.1	<0.001
Uric Acid	8.3±9.8	5.4±1.1	0.003

Table 4: Comparison of hematological parameters between groups

Parameter	Case (Mean ± SD)	Control (Mean ± SD)	p-value
Hemoglobin	8.7±2.1	13.6±1.2	<0.001
RBC	3.5±0.8	5.0±0.6	<0.001
HCT	28.7±8.0	41.5±4.0	<0.001
MCV	79.7±8.9	84.8±8.1	<0.001
MCH	24.8±3.7	27.8±2.7	<0.001
MCHC	31.0±3.2	32.6±1.0	<0.001
RDW-CV	16.3±2.7	14.0±1.4	<0.001
WBC	9689.5±5175.3	7701.6±1991.2	<0.001
Neutrophils	75.5±11.7	60.9±8.8	<0.001
Lymphocytes	15.6±12.8	28.4±8.1	<0.001
Monocytes	7.4±7.2	6.9±2.1	0.477
Eosinophils	3.3±3.6	3.2±2.5	0.827
Platelets	245818.2±124148.7	268917.0±85702.6	0.120
PCT	0.4±1.6	0.3±0.1	0.377
PDW	16.2±0.6	12.9±2.0	<0.001
MPV	10.1±1.3	9.4±1.6	0.001

Table 5: Comparison of biochemical and lipid parameters between groups

Parameter	Case (Mean±SD)	Control (Mean±SD)	p-value
Total Protein	6.4±1.0	7.0±0.7	<0.001
Albumin	3.3±0.7	3.9±0.8	<0.001
Globulin	3.0±0.5	3.0±0.2	0.427
Total Cholesterol	166.4±42.5	139.7±19.8	<0.001
Triglycerides	172.2±64.9	74.8±13.4	<0.001
HDL	33.6±11.3	45.3±8.1	<0.001
Calcium	7.9±1.3	9.1±1.3	<0.001
Phosphorus	5.7±2.4	2.0±0.7	<0.001
Iron	61.7±35.4	78.8±26.8	<0.001
TIBC	226.9±76.8	287.0±48.6	<0.001
Ferritin	453.5±338.6	182.8±52.7	<0.001
CRP	59.2±85.1	2.4±0.7	<0.001

Discussion:

In this study, the mean age of CKD patients on hemodialysis (46.7±12.8 years) was significantly higher than that of healthy controls (41.6±13.5 years, $p=0.005$), showing that CKD patients tended to be older compared with the healthy population. This finding is similar to the observation by Ekbote *et al.*, who reported a similar demographic pattern with CKD patients having a mean age in the mid-40s, supporting the trend of CKD prevalence increasing with age due to progressive renal deterioration over time [9]. Regarding sex distribution, males represented 56.4% of CKD cases and females 43.6%, although this difference was not statistically significant ($p=0.074$). This male predominance is in agreement with several previously published studies. For instance, in a study by Siddiqui *et al.*, 76.2% of CKD patients were male with a mean age of 55.8±13.49 years, showing similar male preponderance among CKD populations [10]. Similarly, in a large hemodialysis cohort analysed by Jiang *et al.*, male patients accounted for 60.7% of participants, further corroborating the higher representation of males among CKD populations undergoing dialysis [11]. However, the sex distribution in CKD can vary by population and study setting; global epidemiological data show that while CKD prevalence increases with age in both sexes, there is often a higher proportion of females in general CKD populations, especially in non-dialysis cohorts, as reported by Deng *et al.* in a global burden analysis where females comprised a large portion

of CKD cases across age groups [12]. In our study, CKD patients on hemodialysis had significantly elevated glycemic parameters compared to healthy controls. The mean HbA1c in cases was $7.3 \pm 1.1\%$, notably higher than the control group's $5.5 \pm 0.5\%$ ($p < 0.001$) and both fasting blood sugar (98.5 ± 19.8 mg/dL versus 84.9 ± 7.7 mg/dL, $p < 0.001$) and postprandial blood sugar (197.0 ± 33.2 mg/dL versus 97.9 ± 19.6 mg/dL, $p < 0.001$) were significantly raised. These findings reflect impaired glycemic control in CKD patients, which is consistent with existing evidence that glucose and insulin metabolism are profoundly disrupted in advanced CKD and that patients frequently experience both hyperglycemia and hypoglycemia due to altered renal handling and metabolic changes associated with kidney failure. Patel *et al.* reported that patients with end-stage kidney disease often exhibit wide glycemic fluctuations and that HbA1c remains the preferred biomarker despite limitations in this population (where a target range of approximately 7–8% is considered acceptable due to these complexities) [13]. For renal function parameters, our CKD patients showed markedly higher urea (84.0 ± 40.1 mg/dL vs 19.0 ± 5.2 mg/dL, $p < 0.001$) and creatinine (5.8 ± 2.5 mg/dL versus 0.7 ± 0.1 mg/dL, $p < 0.001$) compared with controls, which is a well-established finding in CKD research. Elevated serum urea and creatinine are standard indicators of reduced kidney excretory capacity and numerous studies have documented similar trends; for example, Hsiao *et al.*, found that serum urea and creatinine levels were significantly higher in CKD patients than in healthy controls, reinforcing these parameters' clinical utility in diagnosing and monitoring renal dysfunction [14]. Serum uric acid was also significantly elevated in cases (8.3 ± 9.8 mg/dL vs 5.4 ± 1.1 mg/dL, $p = 0.003$). Elevated uric acid is frequently observed in CKD due to decreased renal excretion and is linked to both the presence and progression of CKD. Silva *et al.* reported that high serum uric acid correlates positively with CKD and inversely with estimated glomerular filtration rate, supporting our observation that hyperuricemia coexists with declining renal function [15]. In the present study, CKD patients on hemodialysis demonstrated significant hematological abnormalities compared with healthy controls. Mean hemoglobin (8.7 ± 2.1 g/dL vs 13.6 ± 1.2 g/dL; $p < 0.001$), RBC count ($3.5 \pm 0.8 \times 10^6/\mu\text{L}$ vs $5.0 \pm 0.6 \times 10^6/\mu\text{L}$; $p < 0.001$) and hematocrit ($28.7 \pm 8.0\%$ vs $41.5 \pm 4.0\%$; $p < 0.001$) were markedly reduced in cases, reflecting the high prevalence of anemia in CKD, which is well supported by the literature. For example, Lacson *et al.* [16] reported that anemia was a predominant manifestation in CKD patients, with significantly reduced hemoglobin and RBC counts attributed mainly to inadequate erythropoietin production in advanced disease stages. Moreover, in a study by Ogolla *et al.* [17], hemoglobin levels progressively decreased with advancing CKD stage, while RDW increased significantly, underlining the impact of CKD on erythropoiesis and red cell morphology. Consistent with our findings, RDW-CV was elevated in cases ($16.3 \pm 2.7\%$ versus $14.0 \pm 1.4\%$, $p < 0.001$), which has previously been associated with inflammation and adverse outcomes in CKD populations [18]. White blood cells, including total WBC count and neutrophil percentages, were significantly higher in CKD patients, while

lymphocyte percentages were lower. This pattern is similar to studies showing leukocyte disturbances in CKD, likely driven by chronic inflammation and immune dysfunction. For instance, Li *et al.*, observed significant alterations in WBC indices among CKD and hemodialysis patients compared with controls, supporting the notion that uremic inflammation contributes to leukocytosis. Platelet indices showed mixed results; while mean platelet count and PCT were not significantly different in our cohort, PDW and MPV were significantly higher in cases ($p < 0.001$ and $p = 0.001$, respectively). Similar trends have been reported where platelet volume indices reflect ongoing inflammation and platelet activation in CKD patients, showing their potential role as markers of disease severity and cardiovascular risk [19]. In the present study, CKD patients on hemodialysis showed significantly lower total protein (6.4 ± 1.0 g/dL vs 7.0 ± 0.7 g/dL; $p < 0.001$) and serum albumin (3.3 ± 0.7 g/dL vs 3.9 ± 0.8 g/dL; $p < 0.001$) compared with healthy controls. Hypoalbuminemia is a well-recognized finding in CKD associated with malnutrition, inflammation and protein loss. In a study by Parhi *et al.* evaluating ESRD patients, serum albumin levels were significantly reduced in CKD patients compared with controls, emphasizing that low albumin is a marker of poor nutritional status and inflammation in this population a pattern also reported by other investigators in similar cohorts [20]. Regarding the lipid profile, our CKD group had higher total cholesterol (166.4 ± 42.5 mg/dL vs 139.7 ± 19.8 mg/dL; $p < 0.001$) and triglycerides (172.2 ± 64.9 mg/dL versus 74.8 ± 13.4 mg/dL; $p < 0.001$), along with significantly lower HDL (33.6 ± 11.3 mg/dL versus 45.3 ± 8.1 mg/dL; $p < 0.001$), showing dyslipidemia. Multiple studies have documented similar dyslipidemic patterns in CKD, with elevated triglycerides and reduced HDL being particularly common. For example, Shruthi *et al.* [21] found significantly higher triglycerides and lower HDL levels in CKD patients compared with controls, consistent with our results and reflecting common lipid abnormalities in renal dysfunction. This dyslipidemia is attributed to impaired lipoprotein metabolism caused by reduced lipoprotein lipase activity and altered HDL synthesis in CKD, as described in mechanistic reviews [22]. Serum iron levels were significantly lower in cases (61.7 ± 35.4 $\mu\text{g/dL}$ vs 78.8 ± 26.8 $\mu\text{g/dL}$; $p < 0.001$), with lower TIBC (226.9 ± 76.8 $\mu\text{g/dL}$ vs 287.0 ± 48.6 $\mu\text{g/dL}$; $p < 0.001$) and markedly higher ferritin (453.5 ± 338.6 ng/mL vs 182.8 ± 52.7 ng/mL; $p < 0.001$). These findings reflect the complex iron metabolism dysfunction in CKD, where ferritin often elevates due to inflammation and iron sequestration, while serum iron and TIBC decrease. A recent study by Thu *et al.* [23] showed that high ferritin and low TIBC were significant predictors of mortality in ESRD patients on hemodialysis, showing the clinical significance of these alterations. Similarly, low iron and TIBC with high ferritin have been linked to functional iron deficiency and inflammation in CKD, similar to immuno-nutritional dysregulation seen in other study [24]. C-reactive protein (CRP) was markedly elevated in cases (59.2 ± 85.1 mg/L versus 2.4 ± 0.7 mg/L; $p < 0.001$), reflecting systemic inflammation, which is common in CKD and linked to adverse outcomes. Elevated CRP with reduced albumin has been reported in multiple

studies, including a cohort of MHD patients where significantly higher CRP and lower albumin were observed in CKD compared to controls, supporting the role of chronic inflammation in CKD pathogenesis.

Conclusion:

CKD patients on hemodialysis exhibited significant metabolic, hematological and biochemical alterations compared with healthy controls. They showed elevated glycemic indices, impaired renal function and anemia with altered red blood cell and leukocyte indices. These findings show the systemic impact of CKD and show the importance of regular monitoring of hematological, biochemical and lipid parameters to optimize patient management and improve clinical outcomes.

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