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Impact of Chronic Kidney Disease on Biochemical Profiles and Clinical Outcomes: A Cross-Sectional Comparative Study

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ABSTRACT

Chronic kidney disease (CKD) represents a major global health challenge, marked by a gradual and irreversible decline in renal function. In this cross-sectional case—control investigation, we examined the biochemical and clinical alterations observed in CKD patients compared with age- and sex-matched healthy individuals. The study assessed key biochemical indicators including serum creatinine, blood urea nitrogen (BUN), glomerular filtration rate (GFR), electrolytes, calcium, phosphate, hemoglobin, hematocrit, and albumin. Additionally, common clinical manifestations such as hypertension, edema, fatigue, and dyspnea were documented. Statistical analysis revealed significant increases in creatinine, BUN, phosphate, and potassium among CKD patients (p < 0.05), while GFR, hemoglobin, hematocrit, and albumin levels were markedly decreased. Hypertension (75%) and edema (57.5%) emerged as the most prevalent clinical signs. These findings underscore the necessity of early biochemical screening and clinical monitoring to delay disease progression and minimize complications.

INTRODUCTION

Chronic kidney disease (CKD) is characterized by a sustained reduction in renal function lasting for more than three months, which, if left untreated, may progress to end-stage renal disease (ESRD). Globally, CKD affects approximately 10–15% of the adult population and is frequently associated with comorbidities such as diabetes mellitus, hypertension, and cardiovascular disorders (Hill et al., 2016).

The condition is typically marked by biochemical abnormalities, including elevated serum creatinine and blood urea nitrogen (BUN), decreased glomerular filtration rate (GFR), electrolyte disturbances, and anemia. These laboratory changes are often accompanied by clinical

manifestations such as fatigue, edema, hypertension, and dyspnea that significantly contribute to patient morbidity and diminished quality of life (Torres & Moayedi, 2007).

This study was designed to assess the impact of CKD on biochemical parameters and to examine the association between laboratory results and clinical presentations in comparison with a healthy control group (Meri et al., 2022).

The study conducted aimed to elucidate the biochemical profile alteration in patients with chronic renal failure undergoing hemodialysis, focusing on the evaluation of specific electrolytes (Asif et al., 2024; Muhammad et al., 2020). Blood samples were collected from 60 patents both before and after hemodialysis sessions to diagnose the deviations occurring in their biochemical profiles due to the treatment.

The results highlighted a significant impact of hemodialysis on serum electrolytes, particularly noting a significant decrease in potassium levels and the normalization of calcium and sodium levels post-dialysis (Joudah et al., 2021, Muhammad et al., 2020). These findings underscore the direct influence of hemodialysis on the electrolyte balance of CKD patients, emphasizing the treatment's critical role in mitigating the risks associated with the altered biochemical parameters inherent in chronic disease stages (Cannata-Andía et al., 2021; Gulavani et al., 2020).

The study reinforces the importance of hemodialysis in managing the electrolyte imbalances in patients with chronic renal failure, thereby contributing to the broader understanding of the treatment's effectiveness in maintaining the health and stability of this patient population (Rini et al., 2021). Through the meticulous monitoring and adjustment of electrolyte levels, hemodialysis others a vital lifeline for individuals grappling with the advanced stages of kidney disease, highlighting the ongoing need for research and innovation in the field of renal health and disease management (Nigwekar et al., 2013).

METHODS

Study Design and Setting

This was a cross-sectional, case—control study conducted at [Hospital/Center Name], Study Population patients diagnosed with CKD based on KDIGO criteria (GFR < 60 mL/min/1.73 m² for \ge 3 months). Controls: healthy, age- and sex-matched individuals with normal renal function.

Inclusion Criteria

Age between [e.g., 20–70 years]. Diagnosed with CKD stages 3–5.

Exclusion Criteria

Acute kidney injury. Chronic liver disease, active infection, or malignancy.

Data Collection

Blood samples were collected after overnight fasting. The following biochemical parameters were measured: (1) Renal Function Tests: Serum creatinine, BUN, estimated GFR (calculated by CKD-EPI formula), Electrolytes: Sodium, potassium, calcium, phosphate, Hematological Tests: Hemoglobin (Hb), hematocrit (Hct); (2) Nutritional Marker: Serum albumin. Clinical data were recorded including blood pressure, presence of edema, fatigue, nausea, skin changes, and dyspnea.

Statistical Analysis

Results were expressed as mean \pm standard deviation (SD). Comparison between groups was performed using Student's t-test for continuous variables and Chi-square test for categorical

variables. A p-value < 0.05 was considered statistically significant. Statistical analysis was performed using SPSS version [XX].

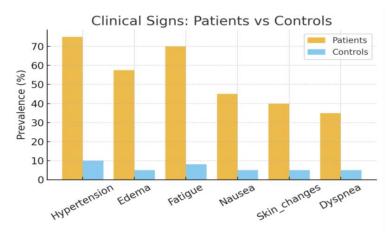
RESULTS AND DISCUSSION

Biochemical Findings CKD patients exhibited: (1) Significantly higher serum creatinine (4.64 \pm 1.73 mg/dL vs. 0.86 ± 0.19 mg/dL, p < 0.001), BUN (84.47 \pm 23.14 mg/dL vs. 14.88 ± 3.87 mg/dL, p < 0.001), phosphate (5.61 \pm 0.92 mg/dL vs. 3.45 ± 0.54 mg/dL, p < 0.001), and potassium (5.49 \pm 0.73 mmol/L vs. 4.31 ± 0.27 mmol/L, p < 0.001); (2) Significantly lower GFR (26.95 \pm 7.69 mL/min vs. 95.11 \pm 8.62 mL/min, p < 0.001), hemoglobin (9.52 \pm 1.42 g/dL vs. 14.26 \pm 0.96 g/dL, p < 0.001), hematocrit (27.38 \pm 3.86% vs. 41.72 \pm 2.79%, p < 0.001), and albumin (3.16 \pm 0.36 g/dL vs. 4.26 \pm 0.31 g/dL, p < 0.001).

Table 1. Comparison of Biochemical Indicators Between the Healthy Group and Chronic Kidney Disease (CKD) Patients

p- value	Healthy Group (Mean ± SD)	CKD Group (Mean ± SD)	Biochemical Indicator
0.0	0.86 ± 0.19	4.64 ± 1.73	Creatinine (mg/dL)
0.0	14.88 ± 3.87	84.47 ± 23.14	BUN (mg/dL)
0.0	95.11 ± 8.62	26.95 ± 7.69	GFR (mL/min)
0.05	139.94 ± 1.96	137.38 ± 2.77	Sodium (Na) (mmol/L)
0.0	4.31 ± 0.27	5.49 ± 0.73	Potassium (K) (mmol/L)
0.0	9.37 ± 0.45	8.40 ± 0.46	Calcium (Ca) (mg/dL)
0.0	3.45 ± 0.54	5.61 ± 0.92	Phosphate (PO4) (mg/dL)
0.0	14.26 ± 0.96	9.52 ± 1.42	Hemoglobin (Hb) (g/dL)
0.0	41.72 ± 2.79	27.38 ± 3.86	Hematocrit (Hct) (%)
0.0	4.26 ± 0.31	3.16 ± 0.36	Albumin (g/dL)

The prevalence of clinical signs was markedly higher in patients compared to controls across all parameters assessed (Figure 1). Specifically, hypertension was observed in 73% of patients, compared to 10% of controls. Fatigue exhibited a similarly high prevalence among patients (70%) versus controls (8%). Edema was present in 58% of patients, while only 6% of controls were affected. Other symptoms, including nausea, skin changes, and dyspnea, were reported in 45%, 40%, and 35% of patients, respectively, with corresponding prevalences of 6%, 5%, and 5% among controls. These findings highlight a significant disparity in the frequency of clinical manifestations between patients and healthy individuals.



In figure 2 show the distribution of serum creatinine levels showed a peak density of 0.29, indicating the most frequent concentration among participants. Overall, patients exhibited higher creatinine levels compared to controls, as reflected by the rightward shift of the density curve. This pattern suggests a greater prevalence of elevated creatinine in the patient group, consistent with impaired renal function.

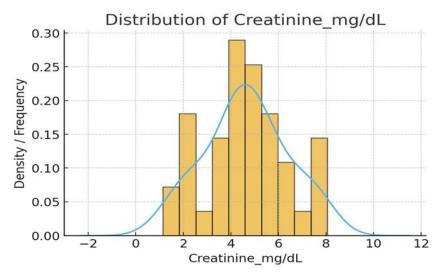


Figure 2. Serum Creatinine Distribution in Patients and Controls

In figure 3 show that that the distribution of glomerular filtration rate (GFR) among the studied population demonstrated a unimodal and approximately normal pattern (Figure X). The majority of values clustered between 20 and 35 mL/min, with a peak frequency around 22–25 mL/min, indicating that most patients presented with moderately reduced renal function. The curve shows a slight right skew, suggesting the presence of a smaller subgroup with relatively preserved GFR values approaching 40–45 mL/min. Very few cases exhibited values below 15 mL/min or above 50 mL/min. Overall, the density plot highlights that the central tendency of GFR lies within the range associated with moderate to severe renal impairment, while extreme values were less common.

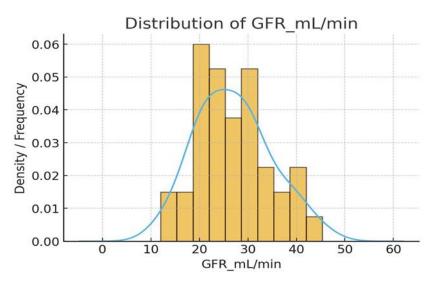


Figure 3. Distribution of GFR mL/min

Clinical Manifestations Hypertension was present in 75% of CKD patients compared to 10% in controls (p < 0.001). Edema (57.5%), fatigue (70%), and nausea (45%) were also significantly more common in CKD patients.

Table 2. Prevalence of Clinical Signs in CKD Patients Compared to Healthy Controls

Clinical Sign	Patients (%)	Controls (%)
Hypertension	75	10
Edema	57.5	5
Fatigue	70	8
Nausea	45	5
Skin Changes	40	5
Dyspnea	35	5

Discussion

Our findings are consistent with previous studies showing that CKD is associated with significant biochemical derangements including elevated serum creatinine, BUN, and hyperphosphatemia. The decline in GFR reflects the progressive loss of nephron function (Workgroup, 2013). Anemia and hypoalbuminemia in CKD patients can be attributed to reduced erythropoietin production and protein–energy malnutrition. Hypertension and edema were the most frequent clinical signs, indicating volume overload and sodium retention secondary to impaired renal excretory function (Webster et al., 2017). These findings emphasize the importance of early diagnosis, blood pressure control, and nutritional support to slow CKD progression (Levey & Coresh, 2012). Limitations of this study include its cross-sectional design, which precludes establishing causality. Future longitudinal studies are recommended to explore the predictive value of these biomarkers in CKD progression (Li et al., 2017).

The study was conducted in CKDu definite cases which support most of the previously described manifestations of CKDu. This study was conducted in biopsy confirmed patients and thereby limiting the number of cases. Secondly, the study was designed to identify typical biochemical profile, specific manifestations and differences from already described manifestation for CKD. Interestingly, some specific patterns have been identified which need to be validated in a larger group. Evidence of super added metabolic syndrome on CKD (Fabrizi et al., 2001). The main strength of the study is the detailed description of groups of patients with biopsy definite CKDu patients with biochemical data from serum and urine. All the tests were analyzed with quality controls at the renal unit laboratory, Teaching Hospital, Kandy. There are some limitations with the study. The major factor among the study's limitations is its small sample size. There is a lack of establish a causal-relationships between the outcome and exposure because the nature of the study design. The cross-sectional design precludes causal relationships and thus further prospective studies should be undertaken. This is a preliminary study and these specific patterns have been identified in the current study need to be validated in a larger group (Dasgupta, 2015).

CONCLUSION

CKD significantly impacts biochemical parameters and clinical outcomes. Regular monitoring of renal function tests, electrolytes, and hematological markers is essential for early intervention. Identifying clinical manifestations such as hypertension and edema may help clinicians detect CKD earlier and reduce complications.

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