Original Article

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Biochemistry Section

Alterations in Electrolyte and Renal Profile in Chronic Kidney Disease Patients with Coexisting Cardiovascular Complications: A Cross-sectional Study

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ABSTRACT

Introduction: Chronic Kidney Disease (CKD) is often complicated by coexisting Cardiovascular Disease (CVD), which increases morbidity and influences biochemical markers. Electrolyte imbalances contribute to various systemic complications and elevate the risk of cardiovascular disease.

Aim: To compare the electrolyte and renal function profiles of chronic kidney disease patients based on the presence or absence of coexisting cardiovascular disease.

Materials and Methods: A hospital-based cross-sectional study conducted was conducted in the Department of Biochemistry, Dr. S. S. Tantia Medical College, in collaboration with Jan Sewa Hospital, Sri Ganganagar, Rajasthan, India,from November 2023 to July 2025. conducted on 150 CKD patients—50 with CVD (Group-A) and 100 without (Group-B) Biochemical markers, including electrolytes (Na^+, K^+, Cl^+), renal function tests (urea, creatinine, uric acid), inflammatory markers {C-reactive Protein (CRP), procalcitonin, high-senstivity troponin I (hs-Trop I)} and proteinuria indicators were measured.

Results: The CKD patients with CVD were significantly older (58.2±15.65 vs. 49.23±12.71 years; p<0.001). They had lower serum sodium levels (p<0.001) and higher potassium levels (p=0.03). The hs-Trop I and CRP levels were markedly elevated in the CVD group (p<0.001). Urea levels were also significantly higher (83.35±15.65 vs. 68.1±13.51; p<0.001). Serum Sodium S.Na. levels were notably lower in CKD patients with CVD (139.09±2.76 mEq/L) compared to those without CVD (141.2±3.04 mEq/L) (p=0.001). Similarly, Serum Potassium S.K. levels were slightly higher in the CVD group (4.52±0.64 mEq/L) compared to the non CVD group (4.3±0.50 mEq/L) and this difference was statistically significant (p=0.03). However, the difference in Serum Chloride S.Cl. levels was not statistically significant (p=0.21).

Conclusion: Coexisting cardiovascular disease in CKD patients is associated with distinct alterations in electrolyte balance, increased inflammation and worsened renal function. Monitoring these biomarkers is crucial for the effective management of CKD patients with cardiovascular co-morbidities.

Keywords: Cardiovascular abnormalities, C-reactive protein, Electrolytes imbalance, High sensitivity assay, Renal insufficiency, Renal parameters

INTRODUCTION

Chronic Kidney Disease (CKD) is a progressive condition characterised by a gradual decline in kidney function over time, eventually leading to End-Stage Renal Disease (ESRD). Globally, CKD affects approximately 10-15% of the population and represents a major public health concern due to its high morbidity, mortality and economic burden [1]. As kidney function deteriorates, significant alterations occur in the body's internal environment, particularly affecting electrolyte balance and renal biomarkers such as urea and creatinine [2].

Electrolyte imbalances are common in CKD and may involve disturbances in sodium, potassium, calcium, phosphate and magnesium levels. These imbalances can lead to systemic complications, including neuromuscular symptoms, bone mineral disorders and increased cardiovascular risk [3]. Furthermore, CKD patients are highly susceptible to CVD, which remains the leading cause of death in this population [4]. The pathophysiological relationship between CKD and CVD—often referred to as the "cardiorenal syndrome"—is multifactorial and involves fluid overload, electrolyte abnormalities, chronic inflammation, endothelial dysfunction and oxidative stress [5].

In addition, endothelial dysfunction, arterial stiffness and arteriosclerosis can affect the renal vasculature. In these patients, haemodynamic changes, including even mild systolic and diastolic dysfunction, may play a significant role [6].

In India, data on the alterations of serum electrolytes and renal profiles in CKD patients with cardiovascular disease are limited. Existing studies describe mild renal hypoperfusion and congestion, accompanied by subclinical inflammation and neurohormonal activation, which contribute to tubular damage, glomerular and cardiac fibrosis and proteinuria. Previous research also suggests that subclinical abnormalities in cardiac structure are associated with long-term kidney function decline [7-8].

Therefore, the present study aims to evaluate and compare the electrolyte and renal profiles of CKD patients with and without cardiovascular disease.

MATERIALS AND METHODS

The present was a hospital-based cross-sectional study conducted in the Department of Biochemistry, Dr. S. S. Tantia Medical College, in collaboration with Jan Sewa Hospital, Sri Ganganagar, Rajasthan, India, from November 2023 to July 2025. The study protocol was approved by the Institutional Ethics Committee of Dr. S. S. Tantia Medical College (Ref. No. TU/IEC/2024/30). Written informed consent was obtained from all participants before their inclusion.

Inclusion criteria: Patients clinically diagnosed with CKD who were willing to participate in the study were included. Individuals aged above 20 years, of either gender, who provided written informed consent personally or through their legally authorised representative were eligible.

Exclusion criteria: Patients undergoing prolonged dialysis, those with congenital renal malformations or end-stage renal failure, pregnant or lactating women and individuals unwilling to participate were excluded.

Sample size calculation: The sample size was calculated using previously reported prevalence estimates of CKD in South Asia. A recent systematic review by Hasan M et al., reported a CKD prevalence of 10.6% to 23.3%. A conservative prevalence (p=10%) was selected to avoid overestimation and ensure representativeness [9].

Using the formula:

$$n=\frac{(t^2 \times p(1-p))}{m^2},$$

where, t=1.96 (95% confidence level), p=0.10 and m=0.05,

The sample size was calculated as:

$$n=\frac{(1.96)^2 \times 0.10(1-0.10)}{(0.05)^2}=138.$$

To account for potential dropouts, a total of 150 patients aged >18 years were included.

The participants were categorised into two groups:

Group-A (CKD with CVD): A total of 50 patients with clinically confirmed CKD and coexisting cardiovascular disease.

Group-B (CKD without CVD): A total of 100 patients diagnosed with CKD but without any clinical or diagnostic evidence of cardiovascular disease.

Study Procedure

A detailed clinical history, demographic profile (age, sex) and laboratory findings were recorded using a predesigned case record form. Venous blood and spot urine samples were collected under aseptic conditions. Biochemical analyses were performed in the central biochemistry laboratory using standard protocols and validated automated analysers.

The following biochemical parameters were estimated: Serum Electrolytes: Sodium (Na^+), Potassium (K^+), Chloride (Cl^+) (measured using Ion-selective electrode).

Renal function tests: Blood urea (Urease hypochlorite), Serum creatinine (Jaffe's method), Serum uric acid {Uricase-Peroxidase (POD)}.

Protein parameters: Total protein (Biuret method), Serum albumin {Bromocresol Green (BCG) dye method}.

Inflammatory and cardiac markers: C-reactive protein (Latex-enhanced turbidimetric immunoassay), High-sensitivity Troponin I (Enzyme-Linked Fluorescent Assay).

Urinary parameters: Urinary microalbumin, Urinary creatinine and Albumin-to-Creatinine Ratio (Pyrogallol method).

STATISTICAL ANALYSIS

Data were compiled and analysed using Statistical Package for the Social Sciences (SPSS) software version 21.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were expressed as Mean±Standard Deviation (SD). The independent t-test was used to compare means between the two groups. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 150 CKD patients were included in the present study, comprising 50 patients with coexisting CVD and 100 patients without CVD. The primary objective was to compare electrolyte and renal parameters between these two groups.

The mean age of CKD patients with CVD (58.2±15.65 years) was significantly higher than that of patients without CVD (49.23±12.71 years; p <0.001), indicating an age-related trend in CVD prevalence among CKD individuals. The age and gender distribution of both groups is shown in [Table/Fig-1,2]. The CVD group showed a male predominance 33 males (66%), whereas the non CVD group had an equal gender distribution.

Age (in years)	Group-A (n=50) n (%)	Group-B (n=100) n (%)
21-30	0	5 (5%)
31-40	8 (16%)	32 (32%)
41-50	10 (20%)	12 (12%)
51-60	11 (22%)	32 (32%)
61-70	5 (10%)	16 (16%)
71-80	12 (24%)	3 (3%)
81-90	4 (8%)	0

[Table/Fig-1]: Frequency distribution of age of CKD with CVD patients and CKD without CVD patients.

Gender	Group-A (n=50)	Group-B (n=100)
Male	33 (66%)	50 (50%)
Female	17 (34%)	50 (50%)

[Table/Fig-2]: Frequency distribution of gender of CKD with CVD patients and CKD without CVD patients.

The electrolyte levels: serum sodium 139.09±2.76 mEq/L, potassium 4.52±0.64 mEq/L and chloride 100.67±3.44 mEq/L is shown in [Table/Fig-3]. Serum sodium levels were significantly lower in the CVD group (139.09±2.76 mEq/L) compared to the non CVD group (141.2±3.04 mEq/L; p<0.001). Serum potassium was slightly elevated in the CVD group (4.52±0.64 vs. 4.3±0.50 mEq/L; p=0.03). The variation in serum chloride was not statistically significant (p=0.21).

Parameters	Group-A (n=50)	Group-B (n=100)	p-value
Age (in years)	58.2±15.65	49.23±12.71	<0.001
S. Na (mEq/L)	139.09±2.76	141.2±3.04	<0.001
S. K (mEq/L)	4.52±0.64	4.3±0.50	0.03
S.Cl (mEq/L)	100.67±3.44	99.84±3.92	0.21
Hs-Trop I (ng/L)	65.64±20.62	12.17±5.03	<0.001
CRP (mg/L)	20.58±7.09	12.65±4.93	<0.001
TP (g/dL)	5.13±0.6	5.67±0.58	<0.001
Alb (g/dL)	3.37±0.37	3.44±0.46	0.32
U. Microalb (mg/24hr)	187.42±73.95	183.61±62.06	0.70
U. Creat (mg/24hr)	89.17±19.85	87.35±19.04	0.59
Urinary microprotein Creatinine ratio	2.18±1.04	2.22±0.88	0.78
Urea (mg/dL)	83.35±15.65	68.1±13.51	<0.001
S. Creat (mg/dL)	2.81±0.91	2.62±0.82	0.19
Uric Acid (mg/dL)	7.05±1.16	7.08±1.73	0.90

[Table/Fig-3]: Showing comparative analysis of biochemical parameters of CKD with CVD patients and CKD without CVD patients.
Alb: Albumin Creat: Creatinine TP: Total protein

Cardiac and inflammatory markers were elevated in the CVD group. High-sensitivity Troponin I averaged 65.64±20.62 ng/L and CRP 20.58±7.09 mg/L, significantly higher than in the non CVD group (12.17±5.03 ng/L and 12.65±4.93 mg/L respectively; p=0.001). Nutritional indicators revealed lower total protein levels in the CVD group (5.13±0.6 g/dL vs. 5.67±0.58 g/dL; p=0.001), while serum albumin differences were not statistically significant (p=0.32).

Urinary microalbumin (187.42±73.95 mg/24 hr), urinary creatinine (89.17±19.85 mg/24 hr) and the albumin-to-creatinine ratio (2.18±1.04) indicated moderate to severe proteinuria. Renal function markers—urea (83.35±15.65 mg/dL) and creatinine (2.81±0.91 mg/dL)—were elevated, indicating significant renal impairment. Urea levels were significantly higher in CVD patients than in non CVD

Parameters		Age	Na	K	Cl	U. Microprotein	U.Creatinine	U/C Ratio
Age	r-value	1	-.269	0.179	0.015	0.134	0.110	0.063
	p-value		0.059	0.215	0.918	0.354	0.449	0.662
Na	r-value		1	0.196	-.032	0.173	-0.111	0.244
	p-value			0.172	0.826	0.229	0.441	0.088
K	r-value			1	-.289	0.398**	0.004	0.354
	p-value				0.042	0.004	0.977	0.012
Cl	r-value				1	-0.266	0.093	-0.261
	p-value					0.062	0.519	0.067
U. Microprotein	r-value					1	0.352	0.686
	p-value						0.012	<0.001
U. Creatinine	r-value						1	-.353
	p-value							0.012
U/C Ratio	r-value							1
	p-value							

Correlation is significant at the 0.05 level

[Table/Fig-4]: Correlation of biochemical parameters of CKD with CVD.
CKD: Chronic kidney disease CVD: Cardiovascular disease U/C: Urea-Creatinine ratio

patients (83.35±15.65 vs. 68.1±13.51 mg/dL; p=0.001). Uric acid levels were also elevated (7.05±1.16 mg/dL), reflecting reduced clearance.

Correlation analysis showed important associations between biochemical parameters in patients with CKD. Serum potassium exhibited a positive correlation with urinary microalbumin (r=0.398**, p=0.004) [Table/Fig-4].

DISCUSSION

The present study highlights significant biochemical differences between CKD patients with and without coexisting CVD. The findings indicate that electrolyte and renal alterations are substantial risk factors in CKD progression and are closely associated with increased cardiovascular complications.

The data demonstrate that age is a significant risk factor for cardiovascular complications in CKD patients. This aligns with the 2012 Italian population data, which reported that 22% of CKD patients with CVD were over 65 years of age and 7% were over 80 years, suggesting that advanced age correlates strongly with cardiovascular disease occurrence [10].

In the present study, serum sodium levels were significantly lower in CKD patients with CVD, while serum potassium levels were marginally higher—both statistically significant findings. Serum chloride levels did not differ significantly between the groups (p=0.21). The lower sodium and higher potassium levels observed in the CVD group suggest altered fluid-electrolyte homeostasis, possibly related to cardiac dysfunction, diuretic therapy, or tubular impairment. Several studies indicate that hyperkalaemia is common and closely associated with cardiovascular and all-cause mortality, particularly in patients with CKD and heart disease, due to impaired renal potassium excretion [11-12].

The present study also found significantly higher blood urea levels in the CVD group, suggesting impaired nitrogenous waste clearance possibly caused by reduced renal perfusion or advanced CKD. Previous research has shown that urea acts as a direct or indirect uraemic toxin, contributing to cardiovascular disease [13-14]. Laville SM et al., reported that serum urea levels predict cardiovascular outcomes in patients with moderate to advanced CVD [13]. Uraemia—a build-up of small molecules in the blood from decreased renal excretion—has also been identified as a nontraditional cardiovascular risk factor contributing to vascular changes in chronic renal failure patients [15].

The correlation analysis demonstrated several meaningful associations between biochemical parameters. Serum potassium showed a positive correlation with urinary microalbumin (r=0.398; p=0.004). The role of albuminuria in promoting hyperkalaemia has not been widely studied; however, the findings suggest that patients with higher albuminuria also experience higher rates of hyperkalaemia. This is supported by other studies and carries important clinical relevance, as patients with high albuminuria often benefit from renin-angiotensin system inhibitor therapy—which also increases the risk of hyperkalaemia [16-17].

Limitation(s)

The present study has several limitations that may restrict the generalisability of the findings. The cross-sectional design does not allow assessment of causal relationships between biochemical alterations and cardiovascular complications. Additionally, variations in treatment regimens, dietary habits and duration of CKD among participants were not controlled and may have influenced biochemical parameters. Future multicentric longitudinal studies with larger sample sizes are recommended to validate these findings.

CONCLUSION(S)

The CKD patients with coexisting cardiovascular disease exhibited more pronounced electrolyte disturbances, elevated inflammatory markers and greater renal impairment than those without cardiovascular disease. These findings emphasise the need for early detection, continuous monitoring and comprehensive management of this high-risk population.

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