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Letter to Editor

Biochemistry Section

Correspondence on Alterations in Electrolyte and Renal Profile in Chronic Kidney Disease Patients with Co-existing Cardiovascular Complications: A cross-sectional Study

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Dear Editor,

We read with great interest the study titled "Alterations in Electrolyte and Renal Profile in Chronic Kidney Disease (CKD) Patients with co-existing cardiovascular complications: A cross-sectional Study" [1]. The authors have addressed an important and clinically relevant interface between CKD and Cardiovascular Disease (CVD), a combination that contributes substantially to morbidity and mortality, particularly in low and middle-income countries. However, a few issues merit consideration to appropriately contextualise the findings and their applicability to clinical practice and public health policy.

1. Study Design and Causal Inference

As agreed by the authors the cross-sectional design inherently limits causal interpretation. While the manuscript frequently implies that electrolyte disturbances and biochemical abnormalities contribute to cardiovascular complications, the temporal relationship cannot be established. The exclusion of patients on "prolonged dialysis" without a precise definition (e.g., months on maintenance haemodialysis or peritoneal dialysis) is ambiguous. Various dialysis prescriptions and targets set for ultrafiltration significantly influence the levels of sodium and potassium. In CKD, electrolyte changes may be both causes and consequences of cardiovascular dysfunction, shared comorbidities, or treatment effects. Longitudinal cohort designs are better suited to explore directionality and risk prediction [2].

2. Definition and Heterogeneity of Cardiovascular Disease (CVD)

The manuscript does not clearly define CVD beyond "clinical or diagnostic evidence." CVD encompasses heterogeneous entities each with distinct electrolyte and biomarker profiles. Lack of stratification by CVD subtype limits internal validity and may dilute clinically meaningful associations for biomarkers such as high-sensitivity Troponin I (hs TnI), which is chronically elevated in CKD even in the absence of acute coronary syndromes [3]. Inclusion of eGFR could have depicted a better understanding of the renal function and CKD progression across groups.

3. Confounding by age, CKD severity, and treatment

Patients with CVD were significantly older, yet no multivariable adjustment was performed. Age is a strong and independent confounder for both CKD progression as well as cardiovascular outcomes. Similarly, CKD stage, duration of disease, blood pressure control, diabetes status, and medication use were not adjusted for, despite their known effects on serum electrolytes, urea, and inflammatory markers [4]. Thus, reliance solely on unadjusted t-tests risks overestimating group differences and limits the result interpretability.

4. Clinical Significance versus Statistical Significance

Although statistically significant, the absolute differences in serum sodium (~2 mEq/L) and potassium (~0.2 mEq/L) between groups were modest and largely within the reference ranges. The manuscript does not adequately address whether these differences can be translated into meaningful clinical risk or therapeutic decision-making. Current KDIGO and cardiology guidelines emphasise outcome-driven thresholds rather than small biochemical variations [5].

5. Biomarker Interpretation in CKD

Troponin elevation in CKD often reflects reduced renal clearance, chronic myocardial strain, or left ventricular hypertrophy rather than acute ischaemia. Without echocardiographic or functional correlation, the interpretation of hs-troponin as a marker of "cardiovascular complication severity" may be misleading [3]. The highly significant increase in hs TnI in CVD patients in this study has not been addressed.

6. Public Health and Community Medicine Perspective

The study's hospital-based design limits generalisability. CKD in the community is often undiagnosed and presents in earlier stages with different biochemical profiles. Additionally, socioeconomic status, nutritional intake, salt consumption, access to care, and health-system factors which are highly relevant in the Indian context, were not assessed. These factors are critical for translating findings into prevention strategies and health-policy planning.

Nevertheless, the study reinforces integrated cardio-renal care. Future research should prioritise multicentric, longitudinal designs with standardised CVD definitions, CKD staging, and multivariable adjustment to identify clinically actionable biomarkers and risk thresholds. Integration of community-based screening and risk stratification models would further enhance public-health relevance. While the study contributes to cardio-renal interactions, cautious interpretation is warranted. Distinguishing statistical association from clinical causality, and biochemical variation from actionable risk, is essential to avoid over-medication and to guide evidence-based management in CKD populations.

Thanks.

No response was received from the authors of the original article.

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