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Research Article

Assessment of Renal Function, Mineral Metabolism Disturbances, and Anti-Platelet Therapy in Chronic Kidney Disease: Clinical Significance of Urea, Creatinine, Calcium, Phosphorus, Alkaline Phosphatase, and Platelet Modulation

Dr. Rahul R. Kunkulol[1], Dr. Kiran S Suryawanshi[2*], Dr. Vitthal S Shinde[3], Dr. M Prasanna Chandra[4], Mr. Vaibhav Deshmukh[5], Dr Ankur Chandra[6]

[1]Professor & HOD, Dept. of Pharmacology, Dr Balasaheb Vikhe Patil, Rural medical college, Pravara Institute of Medical Sciences, Loni, Ahilyanagar, Maharashtra.

[2*]Assistant Professor, Dept. of Biochemistry, Dr Balasaheb Vikhe Patil, Rural medical college, Pravara Institute of Medical Sciences, Loni, Ahilyanagar, Maharashtra.

[3]Professor, Dept. of Biochemistry, Dr Balasaheb Vikhe Patil, Rural medical college, Pravara Institute of Medical Sciences, Loni, Ahilyanagar, Maharashtra.

[4]Professor & HOD, Dept. of Biochemistry, Dr Balasaheb Vikhe Patil, Rural medical college, Pravara Institute of Medical Sciences, Loni, Ahilyanagar, Maharashtra.

[5]Tutor, Dept. of Biochemistry, Dr Balasaheb Vikhe Patil, Rural medical college, Pravara Institute of Medical Sciences, Loni, Ahilyanagar, Maharashtra.

[6]Tutor, Dept. of Biochemistry, Dr Balasaheb Vikhe Patil, Rural medical college, Pravara Institute of Medical Sciences, Loni, Ahilyanagar, Maharashtra.

Corresponding Author: Dr. Kiran S Suryawanshi

Email: kiransuryawanshi901@gmail.com

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ABSTRACT

Introduction: Chronic kidney disease (CKD) is associated with biochemical disturbances and increased cardiovascular risk. Antiplatelet therapy is widely used in CKD, but its impact on outcomes remains uncertain.

Aim: To evaluate biochemical derangements in CKD patients and correlate them with antiplatelet therapy outcomes for prognostic stratification.

Materials and Methods: A prospective observational study was conducted in 100 CKD patients. Serum urea, creatinine, calcium, phosphorus, and alkaline phosphatase were measured. Patients were stratified into four groups: aspirin, clopidogrel, dual therapy, and no antiplatelet therapy. Clinical outcomes including cardiovascular events, bleeding, and hospitalization were analyzed. Statistical associations were assessed using chi-square test.

Results: Elevated serum urea (>45 mg/dL) was observed in 50% of patients, creatinine (>1.3 mg/dL) in 70%, hypocalcemia in 28%, hyperphosphatemia in 44%, and high ALP in 45%. Cardiovascular events occurred in 20% of aspirin and clopidogrel groups, 25% in dual therapy, and 40% in untreated patients. Dual therapy was associated with higher bleeding (30%) and hospitalization (50%). Elevated urea, creatinine, hypocalcemia, and hyperphosphatemia were significantly correlated with adverse outcomes ($p<0.05$).

Conclusion: CKD patients exhibit marked biochemical disturbances that strongly predict cardiovascular and bleeding complications. Antiplatelet monotherapy reduces cardiovascular risk compared to no therapy, while dual therapy increases bleeding. Integrating biochemical markers with pharmacological outcomes may enhance prognostic stratification and guide individualized management in CKD.

Keywords: Chronic Kidney Disease, Biochemical Markers, Antiplatelet Therapy.

INTRODUCTION

Chronic kidney disease (CKD) is a global public health problem, affecting approximately 8–16% of the population worldwide and contributing significantly to morbidity and mortality[1]. The progressive decline in renal function leads to accumulation of nitrogenous waste products such as urea and creatinine, along with disturbances in mineral metabolism including calcium, phosphorus, and alkaline phosphatase[2]. These biochemical derangements are collectively referred to as CKD–mineral and bone disorder (CKD-MBD), which is strongly associated with cardiovascular

disease (CVD) and increased mortality[3]. Cardiovascular disease remains the leading cause of death in CKD patients, with mechanisms involving endothelial dysfunction, vascular calcification, platelet abnormalities, and chronic inflammation[4]. Elevated serum phosphorus and reduced calcium levels have been linked to vascular calcification and adverse cardiovascular outcomes, while high alkaline phosphatase reflects increased bone turnover and systemic inflammation[5]. Thus, biochemical markers provide important insights into both renal dysfunction and cardiovascular risk. Antiplatelet therapy, particularly aspirin and clopidogrel, is widely used for secondary prevention of cardiovascular events. In CKD patients, however, the balance between thrombotic risk reduction and bleeding complications remains controversial[6]. Meta-analyses suggest that antiplatelet agents reduce major cardiovascular events but increase bleeding risk, necessitating individualized therapy[7]. Comparative studies of aspirin and clopidogrel in CKD populations highlight differences in efficacy and safety, with dual therapy offering enhanced platelet inhibition but at the cost of higher bleeding rates[8]. Given the dual burden of biochemical disturbances and cardiovascular risk in CKD, integrating biochemical markers with antiplatelet therapy outcomes may provide a more comprehensive prognostic framework. This study therefore evaluates renal function and mineral metabolism parameters in CKD patients, correlates them with antiplatelet therapy outcomes, and explores their utility in risk stratification.

Aim

To evaluate biochemical disturbances in CKD patients and correlate them with antiplatelet therapy outcomes for prognostic risk stratification.

Objectives

1. To evaluate renal function and mineral metabolism disturbances in CKD patients using serum urea, creatinine, calcium, phosphorus, and alkaline phosphatase.
2. To assess the impact of anti-platelet drug therapy on clinical outcomes in CKD patients.
3. To correlate biochemical disturbances with anti-platelet drug response, focusing on cardiovascular risk reduction and bleeding complications.
4. To determine feasibility of integrating biochemical and pharmacological markers for prognostic stratification in CKD.

Material and Methods

This was a prospective observational study conducted in patients diagnosed with chronic kidney disease (CKD) attending the Department of Nephrology and Medicine. A total of 100 patients were enrolled after obtaining informed consent.

Inclusion Criteria

Adult patients (>18 years) with established CKD based on clinical and biochemical parameters. **Exclusion Criteria**

Patients with acute kidney injury, those on dialysis, and individuals with known hepatic or metabolic bone disorders.

Biochemical Assessment:

Venous blood samples were collected under aseptic precautions. Serum urea was estimated using the diacetyl monoxime method, and serum creatinine was measured by the Jaffe's kinetic method. Serum calcium was determined by the Arsenazo III method, phosphorus by the molybdate UV method, and alkaline phosphatase (ALP) by the p-nitrophenyl phosphate kinetic method. All assays were performed using automated analyzers, with internal quality control maintained. Reference ranges

1. urea 15–45 mg/dL
2. creatinine 0.6–1.3 mg/dL
3. calcium 8.5–10.5 mg/dL
4. phosphorus 2.5–4.5 mg/dL
5. ALP 44–147 IU/L[12].

Therapy Groups

Patients were stratified into four groups based on antiplatelet therapy: aspirin monotherapy, clopidogrel monotherapy, dual therapy (aspirin + clopidogrel), and no antiplatelet therapy. Clinical outcomes including cardiovascular (CV) events, bleeding episodes, and hospitalization duration (>7 days) were recorded during follow-up.

Statistical Analysis

Data were expressed as frequencies and percentages. Associations between biochemical parameters and clinical outcomes were analyzed using chi-square (χ^2) test. A p-value <0.05 was considered statistically significant. Statistical analysis was performed using SPSS software version 22.0.

OBSERVATION AND RESULT

Table 1: Biochemical Parameters in CKD Patients

Sr No	Variables	Number of cases n	Percentage %
1	**Serum Urea**		
	a. <15 mg/dL	12	12 %
	b. 15–45 mg/dL	38	38 %
	c. >45 mg/dL	50	50 %
2	**Serum Creatinine**		
	a. <0.6 mg/dL	5	5 %
	b. 0.6–1.3 mg/dL	25	25 %
	c. >1.3 mg/dL	70	70 %
3	**Serum Calcium**		
	a. <8.5 mg/dL	28	28 %
	b. 8.5–10.5 mg/dL	42	42 %
	c. >10.5 mg/dL	30	30 %
4	**Serum Phosphorus**		
	a. <2.5 mg/dL	20	20 %
	b. 2.5–4.5 mg/dL	36	36 %
	c. >4.5 mg/dL	44	44 %
5	**Alkaline Phosphatase**		
	a. <44 IU/L	15	15 %
	b. 44–147 IU/L	40	40 %
	c. >147 IU/L	45	45 %

The distribution of biochemical markers among CKD patients revealed significant derangements. Half of the patients (50%) had elevated serum urea levels above 45 mg/dL, while 38% were in the moderate range (15–45 mg/dL), and only 12% had values below 15 mg/dL. Similarly, serum creatinine was markedly raised in the majority, with 70% showing values above 1.3 mg/dL, 25% within the normal range, and just 5% below 0.6 mg/dL. Calcium levels were relatively preserved, with 42% in the normal range, though 28% had hypocalcemia (<8.5 mg/dL) and 30% showed hypercalcemia (>10.5 mg/dL). Phosphorus disturbances were evident, with 44% having hyperphosphatemia (>4.5 mg/dL), 36% within normal limits, and 20% hypophosphatemic. Alkaline phosphatase was elevated in 45% of patients, normal in 40%, and low in 15%.

Table 2: Clinical Outcomes by Antiplatelet Therapy

Sr No	Therapy Group	CV Events (n, %)	Bleeding (n, %)	Hospitalization >7 days (n, %)
1	**Aspirin (n=40)**	8 (20%)	5 (12.5%)	12 (30%)
2	**Clopidogrel (n=30)**	6 (20%)	4 (13.3%)	9 (30%)
3	**Dual Therapy (A+C) (n=20)**	5 (25%)	6 (30%)	10 (50%)
4	**No Antiplatelet Therapy (n=10)**	4 (40%)	1 (10%)	6 (60%)

Clinical outcomes varied across therapy groups. Among aspirin users (n=40), cardiovascular (CV) events occurred in 20%, bleeding in 12.5%, and prolonged hospitalization (>7 days) in 30%. Clopidogrel users (n=30) showed similar CV event rates (20%) and slightly higher bleeding (13.3%), with 30% requiring extended hospitalization. Dual therapy (aspirin + clopidogrel, n=20) was associated with higher complication rates, with CV events in 25%, bleeding in 30%, and hospitalization in 50%. Patients without antiplatelet therapy (n=10) had the worst outcomes, with CV events in 40%, bleeding in 10%, and hospitalization in 60%.

Table 3: Biochemical Disturbances and Clinical Events

Sr No	Parameter	CV Events (n=23)	p-value	Bleeding (n=16)	p-value
1	High Urea (>45)	9 (39%)	0.03	4 (25%)	0.05 (S)
2	High Creatinine (>1.3)	7 (30%)	0.02	3 (19%)	0.04 (S)
3	Low Calcium (<8.5)	4 (17%)	0.01	3 (19%)	0.02 (S)
4	High Phosphorus (>4.5)	2 (9%)	0.04	3 (19%)	0.05 (S)
5	High ALP (>147)	1 (4%)	0.05	3 (19%)	0.07 (NS)

Correlation analysis demonstrated significant associations between biochemical abnormalities and adverse outcomes. High serum urea (>45 mg/dL) was linked to 39% of CV events (p=0.03) and 25% of bleeding episodes (p=0.05). Elevated creatinine (>1.3 mg/dL) was associated with 30% of CV events (p=0.02) and 19% of bleeding (p=0.04). Hypocalcemia (<8.5 mg/dL) contributed to 17% of CV events (p=0.01) and 19% of bleeding (p=0.02). Hyperphosphatemia (>4.5 mg/dL) was implicated in 9% of CV events (p=0.04) and 19% of bleeding (p=0.05). High ALP (>147 IU/L) showed weaker associations, with 4% CV events (p=0.05) and 19% bleeding (p=0.07, NS).

Table 4: Prognostic Markers and Risk Stratification

Sr No	**Prognostic Marker**	**High-Risk Outcome (n, %)**	**Low-Risk Outcome (n, %)**	**χ^2 Value**	**p-value**
1	**Elevated Serum Urea (>45 mg/dL)**	20 (40%)	30 (60%)	4.8	**0.03** (S)
2	**Elevated Creatinine (>1.3 mg/dL)**	22 (31%)	48 (69%)	5.2	**0.02** (S)
3	**Hypocalcemia (<8.5 mg/dL)**	15 (54%)	13 (46%)	6.1	**0.01** (S)
4	**Hyperphosphatemia (>4.5 mg/dL)**	18 (41%)	26 (59%)	4.5	**0.04** (S)
5	**High ALP (>147 IU/L)**	16 (36%)	29 (64%)	3.9	**0.05** (S)
6	**Dual Antiplatelet Therapy**	5 (25%)	15 (75%)	2.1	**0.14** (NS)

Prognostic stratification revealed that elevated serum urea (>45 mg/dL) was associated with 40% high-risk outcomes (χ^2=4.8, p=0.03). Elevated creatinine (>1.3 mg/dL) showed 31% high-risk outcomes (χ^2=5.2, p=0.02). Hypocalcemia (<8.5 mg/dL) was strongly predictive, with 54% high-risk outcomes (χ^2=6.1, p=0.01). Hyperphosphatemia (>4.5 mg/dL) was linked to 41% high-risk outcomes (χ^2=4.5, p=0.04). Elevated ALP (>147 IU/L) also showed significant association, with 36% high-risk outcomes (χ^2=3.9, p=0.05). In contrast, dual antiplatelet therapy did not reach statistical significance (χ^2=2.1, p=0.14).

DISCUSSION

The present study demonstrated that CKD patients exhibit significant biochemical disturbances, particularly elevated serum urea, creatinine, phosphorus, and alkaline phosphatase, along with hypocalcemia. These abnormalities were strongly associated with adverse cardiovascular and bleeding outcomes. Elevated urea and creatinine reflected impaired renal clearance, while disturbances in calcium

and phosphorus metabolism highlighted the presence of CKD–mineral and bone disorder (CKD-MBD), a well-recognized contributor to vascular calcification and cardiovascular risk[3,5]. Similar findings were reported by Mehmood et al., who observed that derangements in urea and creatinine were prevalent in CKD patients and correlated with disease severity[1]. Suryawanshi et al. also emphasized that metabolic alterations in calcium and phosphorus are central to CKD progression and complications[2].

Our results showed that hypocalcemia was significantly associated with high-risk outcomes, consistent with the role of calcium-phosphorus imbalance in vascular calcification and endothelial dysfunction[3,5]. Elevated phosphorus levels were linked to cardiovascular events, supporting prior evidence that hyperphosphatemia accelerates vascular stiffness and increases mortality in CKD patients[3]. Elevated alkaline phosphatase, though less strongly correlated, may reflect increased bone turnover and systemic inflammation, as highlighted in CKD-MBD literature[5].

With respect to antiplatelet therapy, aspirin and clopidogrel monotherapy were associated with reduced cardiovascular events compared to no therapy, whereas dual therapy increased bleeding risk without significant prognostic benefit. These findings align with meta-analyses showing that antiplatelet agents reduce major cardiovascular events but increase bleeding risk in CKD patients[7]. Farhana et al. compared aspirin and clopidogrel in CKD and noted similar efficacy in cardiovascular protection, though clopidogrel was associated with slightly higher bleeding rates[6]. Choi et al. further demonstrated that clopidogrel monotherapy may be as effective as aspirin in high-risk populations, but dual therapy carries a higher bleeding burden[8].

Pathophysiologically, CKD patients are predisposed to both thrombotic and hemorrhagic complications. Uremia impairs platelet adhesion and aggregation, while endothelial dysfunction promotes thrombosis[4]. Elevated urea and creatinine contribute to platelet dysfunction and altered coagulation pathways, explaining the dual risk observed in our cohort. Hypocalcemia and hyperphosphatemia exacerbate vascular calcification, increasing cardiovascular risk, while antiplatelet therapy modifies platelet activation but simultaneously predisposes to bleeding. Thus, the interplay of biochemical derangements and pharmacological interventions defines the complex risk profile in CKD.

Overall, our findings reinforce that biochemical markers such as urea, creatinine, calcium, and phosphorus are not only indicators of renal dysfunction but also prognostic determinants of cardiovascular and bleeding outcomes. Antiplatelet therapy provides benefit in reducing cardiovascular events, but careful selection is required to balance bleeding risk. Integrating biochemical and pharmacological markers may therefore enhance risk stratification and guide individualized therapy in CKD patients.

CONCLUSION

CKD patients exhibit significant biochemical disturbances, particularly elevated urea, creatinine, phosphorus, and alkaline phosphatase, along with hypocalcemia, which are strongly associated with adverse cardiovascular and bleeding outcomes. Antiplatelet therapy, especially monotherapy, reduces cardiovascular risk compared to no therapy, though dual therapy increases bleeding complications. Integrating biochemical markers with pharmacological outcomes offers a feasible approach for prognostic stratification and individualized management in CKD.

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