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Research Article

PREVALENCE AND SEVERITY OF METABOLIC ACIDOSIS IN PATIENTS WITH CHRONIC KIDNEY DISEASE ON MAINTENANCE HAEMODIALYSIS: A CROSS-SECTIONAL STUDY

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Abstract

Background: Metabolic acidosis is a common complication of advanced chronic kidney disease (CKD) and may persist despite maintenance haemodialysis. Persistent acidosis is associated with protein catabolism, muscle wasting, bone disease, inflammation, and increased cardiovascular risk. Assessment of its prevalence and severity among patients receiving maintenance haemodialysis is therefore clinically important.

Aim: To assess the prevalence and severity of metabolic acidosis among patients with CKD receiving maintenance haemodialysis and to evaluate its association with selected demographic and biochemical parameters.

Methodology: A hospital-based cross-sectional study was conducted among 150 adult patients with CKD receiving maintenance haemodialysis. Demographic characteristics, duration of dialysis, comorbidities, and biochemical parameters were recorded. Pre-dialysis venous blood samples were assessed for serum bicarbonate and other relevant laboratory parameters. Metabolic acidosis was defined as serum bicarbonate <22 mEq/L. Severity was categorized as mild (18–21.9 mEq/L), moderate (15–17.9 mEq/L), and severe (<15 mEq/L).

Results: Among 150 patients, 82 (54.7%) were male and 68 (45.3%) were female, with a mean age of 52.8 ± 12.4 years.

Metabolic acidosis was identified in 68 (45.3%) patients. Mild, moderate, and severe acidosis were observed in 42 (28.0%), 21 (14.0%), and 5 (3.3%) patients, respectively. Patients with longer dialysis duration had a higher prevalence of metabolic acidosis. Diabetes mellitus and lower serum albumin were also more frequent among patients with metabolic acidosis. The mean serum bicarbonate concentration was significantly lower among patients with metabolic acidosis than those without acidosis (18.9 ± 2.4 vs. 24.3 ± 2.1 mEq/L; $p<0.001$).

Conclusion: Metabolic acidosis remains a frequent biochemical abnormality among patients receiving maintenance haemodialysis, with a substantial proportion demonstrating moderate or severe acidosis. Regular monitoring of serum bicarbonate and identification of potentially modifiable contributing factors may help improve metabolic control and reduce complications associated with persistent acidosis.

Keywords: Chronic kidney disease, metabolic acidosis, haemodialysis, serum bicarbonate, renal failure, dialysis.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive disorder characterized by persistent abnormalities of kidney structure or function and is associated with substantial morbidity and mortality. As kidney function declines, the ability to maintain acid-base homeostasis becomes impaired because of reduced ammonium production and impaired urinary acid excretion. Consequently, metabolic acidosis becomes increasingly common in advanced CKD. [1,2]

Metabolic acidosis in CKD is characterized by a reduction in serum bicarbonate concentration and is particularly important in patients with advanced kidney failure. In patients undergoing maintenance haemodialysis, the acid-base balance is influenced by residual kidney function, dietary acid load, dialysis prescription, interdialytic interval, and the bicarbonate concentration of the dialysate. [3,4]

Persistent metabolic acidosis has several adverse biological effects. Chronic acid retention may stimulate skeletal muscle protein degradation, impair bone mineral metabolism, increase inflammation, and contribute to nutritional abnormalities. These effects may be particularly important in haemodialysis patients, who are already at increased risk of protein-energy wasting and cardiovascular complications. [5,6]

The prevalence of metabolic acidosis among haemodialysis patients varies considerably between studies because of differences in patient characteristics, dialysis adequacy, timing of blood sampling, dietary patterns, and definitions used for acidosis. Serum bicarbonate is commonly used as a practical marker of metabolic acid-base status in routine dialysis practice. [7]

Dialysis-related factors also influence serum bicarbonate levels. Adequate dialysis removes accumulated acids and provides bicarbonate or bicarbonate-generating capacity, while inadequate dialysis, high dietary acid load, and poor

residual renal function may contribute to persistent acidosis. Conversely, excessive bicarbonate exposure during dialysis may predispose to post-dialysis alkalosis. [8]

Metabolic acidosis has also been associated with adverse clinical outcomes in patients with CKD. Lower serum bicarbonate concentrations have been linked with increased risks of hospitalization, cardiovascular disease, and mortality in several observational studies, although the relationship may be influenced by nutritional and inflammatory status. [9]

International recommendations emphasize the importance of monitoring serum bicarbonate in patients with advanced CKD and dialysis-dependent kidney failure. Identification of patients with persistent low bicarbonate levels allows clinicians to review dialysis adequacy, dietary factors, residual renal function, and other potential causes. [10]

Despite the clinical importance of metabolic acidosis, its prevalence and severity may vary between dialysis populations. Local assessment is therefore useful for understanding the burden of this complication and identifying patient groups who require closer monitoring.

METHODOLOGY

Study Design& Setting

A hospital-based cross-sectional observational study was conducted among patients with CKD receiving maintenance haemodialysis. The study was conducted in the Department of Nephrology/Dialysis Unit of a tertiary care hospital. The study was conducted over a period of 6 months**.**

Study Population

The study population consisted of 150 adult patients with CKD receiving regular maintenance haemodialysis.

Inclusion Criteria

Patients were included if they:

- Were aged ≥18 years.
- Had established CKD requiring maintenance haemodialysis.
- Had been receiving haemodialysis regularly for at least 3 months.
- Were willing to participate in the study.
- Provided informed consent.

Exclusion Criteria

Patients were excluded if they:

- Had acute kidney injury.
- Had acute metabolic or respiratory illness at the time of assessment.
- Had active sepsis.
- Had severe hepatic failure.
- Were receiving sodium bicarbonate therapy specifically for an acute acid-base disorder.

- Had incomplete clinical or laboratory records.
- Refused consent.

Data Collection

After obtaining informed consent, demographic and clinical information was collected using a structured proforma. Data included age, sex, duration of CKD, duration of haemodialysis, diabetic status, hypertension, and other relevant comorbidities.

Pre-dialysis venous blood samples were collected before a scheduled haemodialysis session. Serum bicarbonate and other biochemical parameters were measured using standard laboratory methods.

Definition of Metabolic Acidosis

For the present study, metabolic acidosis was defined as:

Serum bicarbonate <22 mEq/L.

Severity was categorized as:

Severity	Serum bicarbonate
No acidosis	≥22 mEq/L
Mild	18–21.9 mEq/L
Moderate	15–17.9 mEq/L
Severe	<15 mEq/L

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequency and percentage. The Chi-square test was used to assess associations between categorical variables. Independent sample *t*-test was used for comparison of continuous variables between two groups. A **p-value <0.05** was considered statistically significant.

Ethical Considerations

Institutional Ethical Committee approval was obtained before commencement of the study. Written informed consent was obtained from all participants. Patient confidentiality was maintained throughout the study.

RESULTS

A total of **150 patients with CKD receiving maintenance haemodialysis** were included in the study.

Table 1. Distribution of Study Participants According to Age and Sex

Variable	Category	Number (n)	Percentage (%)
Age	18–30 years	8	5.3
	31–40 years	18	12.0
	41–50 years	38	25.3
	51–60 years	51	34.0
	>60 years	35	23.4
Total		**150**	**100.0**
Sex	Male	82	54.7
	Female	68	45.3
Total		**150**	**100.0**

Among the 150 participants, 82 (54.7%) were male and 68 (45.3%) were female. The majority of patients belonged to the 51–60-year age group, comprising 51 (34.0%) participants, followed by the 41–50-year group with 38 (25.3%). The mean age of the study population was approximately 52.8 ± 12.4 years. (Table 1)

Table 2. Distribution According to Duration of Maintenance Haemodialysis

Duration of haemodialysis	Number (n)	Percentage (%)
3–12 months	46	30.7
13–36 months	59	39.3
>36 months	45	30.0

Total	**150**	**100.0**

The largest proportion of patients, 59 (39.3%), had been receiving maintenance haemodialysis for 13–36 months. Forty-six patients (30.7%) had been on haemodialysis for 3–12 months, while 45 (30.0%) had received dialysis for more than 36 months. (Table 2)

Table 3. Prevalence of Metabolic Acidosis According to Serum Bicarbonate

Serum bicarbonate category	Number (n)	Percentage (%)
≥22 mEq/L – No acidosis	82	54.7
18–21.9 mEq/L – Mild acidosis	42	28.0
15–17.9 mEq/L – Moderate acidosis	21	14.0
<15 mEq/L – Severe acidosis	5	3.3
Total	**150**	**100.0**

Metabolic acidosis was present in **68 of 150 patients (45.3%)**. Mild acidosis was the most common severity category, occurring in 42 (28.0%) patients. Moderate acidosis was observed in 21 (14.0%), while severe acidosis was present in 5 (3.3%) patients. More than half of the study population, 82 (54.7%), had serum bicarbonate ≥22 mEq/L. (Table 3)

Table 4. Association Between Duration of Haemodialysis and Metabolic Acidosis

Duration of haemodialysis	No acidosis n (%)	Metabolic acidosis n (%)	Total	p-value
3–12 months	31 (67.4)	15 (32.6)	46	
13–36 months	34 (57.6)	25 (42.4)	59	
>36 months	17 (37.8)	28 (62.2)	45	**0.015**
Total	**82 (54.7)**	**68 (45.3)**	**150**	

A significant association was observed between duration of haemodialysis and metabolic acidosis (p=0.015). The prevalence of metabolic acidosis increased from 32.6% among patients receiving dialysis for 3–12 months to 62.2% among those undergoing dialysis for more than 36 months. (Table 4)

Table 5. Association of Diabetes Mellitus and Hypertension with Metabolic Acidosis

Comorbidity	No acidosis n (%)	Metabolic acidosis n (%)	Total	p-value
Diabetes mellitus	35 (42.7)	39 (57.3)	74	**0.014***

No diabetes mellitus	47 (61.8)	29 (38.2)	76	
Hypertension	58 (51.3)	55 (48.7)	113	0.203
No hypertension	24 (64.9)	13 (35.1)	37	

*P value <0.05 significant using Chi square Test

Metabolic acidosis was significantly more frequent among patients with diabetes mellitus compared with those without diabetes (57.3% vs. 38.2%, p=0.014). Although metabolic acidosis was numerically more common among patients with hypertension, the association was not statistically significant (p=0.203). (Table 5)

Table 6. Comparison of Biochemical Parameters Between Patients With and Without Metabolic Acidosis

Biochemical parameter	**No acidosis (n=82) Mean ± SD**	**Acidosis (n=68) Mean ± SD**	**p-value**
Serum bicarbonate (mEq/L)	24.3 ± 2.1	18.9 ± 2.4	**<0.001***
Serum albumin (g/dL)	3.8 ± 0.4	3.5 ± 0.5	**<0.001***
Blood urea nitrogen (mg/dL)	68.4 ± 17.2	75.8 ± 19.4	**0.018***
Serum Creatinine (mg/dL)	7.1 ± 1.6	7.5 ± 1.8	0.146
Serum Potassium (mEq/L)	4.7 ± 0.6	5.0 ± 0.7	**0.006***
Haemoglobin (g/dL)	9.6 ± 1.2	9.2 ± 1.3	0.061

*P value <0.05 significant using Student T Test

Patients with metabolic acidosis had significantly lower serum bicarbonate and albumin concentrations than patients without acidosis (p<0.001 for both). Blood urea nitrogen and serum potassium were also significantly higher in the acidosis group. Serum creatinine and haemoglobin did not differ significantly between the two groups. (Table 6)

Table 7. Overall Severity of Metabolic Acidosis Among Study Participants

Category	**Number (n)**	**Percentage (%)**
No metabolic acidosis	82	54.7
Mild metabolic acidosis	42	28.0
Moderate metabolic acidosis	21	14.0

Severe metabolic acidosis	5	3.3

Overall, 68 (45.3%) patients had metabolic acidosis. Of these, mild acidosis constituted the largest proportion (61.8% of patients with acidosis), followed by moderate acidosis (30.9%) and severe acidosis (7.4%). Thus, although most affected patients had mild acidosis, approximately one-third had moderate-to-severe metabolic acidosis. (Table 7)

Multivariable logistic regression demonstrated that diabetes mellitus, longer duration of haemodialysis, higher BUN, higher serum potassium and lower serum albumin were independent factors associated with metabolic acidosis**.** Patients with diabetes had approximately **2-fold higher odds** of metabolic acidosis compared with non-diabetic patients (OR 2.01, 95% CI: 1.04–3.88; p=0.038). Patients receiving haemodialysis for >36 months had approximately 2.7-fold higher odds compared with those receiving dialysis for 3–12 months (OR 2.74, 95% CI: 1.22–6.16; p=0.015).BUN >70 mg/dL was independently associated with metabolic acidosis (OR 2.08, p=0.029), as was serum potassium ≥5.0 mEq/L (OR 2.17, p=0.030).

Table 8. Binary Logistic Regression Analysis of Factors Associated with Metabolic Acidosis (n=150)

Variable	Reference category	Odds Ratio (OR)	95% CI	p-value
Age >50 years	≤50 years	1.28	0.67–2.45	0.451
Male sex	Female	1.12	0.59–2.14	0.725
Diabetes mellitus	No diabetes	**2.01**	**1.04–3.88**	**0.038***
Hypertension	No hypertension	1.39	0.64–3.01	0.399
Haemodialysis duration >36 months	3–12 months	**2.74**	**1.22–6.16**	**0.015***
Haemodialysis duration 13–36 months	3–12 months	1.49	0.70–3.17	0.299
BUN >70 mg/dL	≤70 mg/dL	**2.08**	**1.08–4.01**	**0.029***
Creatinine >7.5 mg/dL	≤7.5 mg/dL	1.21	0.63–2.32	0.566
Potassium ≥5.0 mEq/L	<5.0 mEq/L	**2.17**	**1.08–4.36**	**0.030***
Albumin <3.5 g/dL	≥3.5 g/dL	**2.31**	**1.12–4.76**	**0.023***
Haemoglobin <10 g/dL	≥10 g/dL	1.38	0.72–2.65	0.328
Sodium <135 mEq/L	≥135 mEq/L	1.56	0.61–4.00	0.352
Calcium <8.5 mg/dL	≥8.5 mg/dL	1.47	0.71–3.06	0.298
Phosphate >5.5 mg/dL	≤5.5 mg/dL	1.59	0.81–3.13	0.181

OR = Odds Ratio; CI = Confidence Interval; BUN = Blood Urea Nitrogen.

Patients with serum albumin <3.5 g/dL had approximately **2.3-fold higher odds** of metabolic acidosis (OR 2.31, 95% CI: 1.12–4.76; p=0.023).Age, sex, hypertension, serum creatinine, haemoglobin, sodium, calcium and phosphate were **not independently associated** with metabolic acidosis. (Table 8)

DISCUSSION

The present study evaluated the prevalence and severity of metabolic acidosis among 150 patients receiving maintenance haemodialysis. Metabolic acidosis, defined as a serum bicarbonate concentration <22 mEq/L, was identified in 68 (45.3%) patients. Mild, moderate and severe acidosis were observed in 28.0%, 14.0% and 3.3% of the study population, respectively. This finding demonstrates that metabolic acidosis remains a frequent biochemical abnormality among patients receiving maintenance haemodialysis. Vashistha et al. reported that uremic metabolic acidosis remains prevalent among patients with advanced CKD and dialysis dependence and may have important prognostic implications. [11]

In the present study, a significant association was observed between duration of haemodialysis and metabolic acidosis. The prevalence increased from 32.6% among patients receiving dialysis for 3–12 months to 62.2% among those undergoing dialysis for more than 36 months (p=0.015). Longer dialysis duration may be associated with loss of residual renal function, greater comorbidity burden and differences in dialysis adequacy. Kovesdy et al. demonstrated that serum bicarbonate abnormalities are associated with important clinical outcomes in patients with CKD, supporting the importance of monitoring acid-base status during progressive kidney disease. [12]

The present study also demonstrated a significant association between diabetes mellitus and metabolic acidosis. Acidosis was present in 57.3% of diabetic patients compared with 38.2% of patients without diabetes (p=0.014). Diabetes may contribute indirectly through greater disease severity and associated metabolic and nutritional abnormalities. Bommer et al., in a large haemodialysis cohort, demonstrated that predialysis serum bicarbonate concentrations were associated with morbidity and mortality, emphasizing the clinical importance of acid-base status in dialysis patients. [13]

Patients with metabolic acidosis had significantly lower serum albumin concentrations than those without acidosis (3.5 ± 0.5 vs. 3.8 ± 0.4 g/dL, p<0.001). This finding may indicate an association between metabolic acidosis and poor nutritional status or protein-energy wasting. Chen and Abramowitz described metabolic acidosis as an important complication of CKD with potential effects on nutritional and metabolic processes and emphasized its relationship with CKD progression. [14]

The present study showed significantly higher blood urea nitrogen and serum potassium concentrations among patients with metabolic acidosis. Mean serum potassium was 5.0 ± 0.7 mEq/L in the acidosis group compared with 4.7 ± 0.6 mEq/L in patients without acidosis (p=0.006). Acid-base and electrolyte disturbances commonly coexist in advanced CKD and end-stage kidney disease. Raphael et al. demonstrated that bicarbonate concentration is associated with kidney function and survival, highlighting the importance of considering serum bicarbonate as part of the broader

metabolic profile of patients with CKD. [15]

The prognostic relevance of serum bicarbonate has been demonstrated in several observational studies. Chen and Melamed reviewed the relationship between serum bicarbonate and mortality in CKD and highlighted the potential clinical significance of abnormal bicarbonate concentrations. [16] Similarly, Abramowitz et al. demonstrated that oral sodium bicarbonate therapy can modify serum bicarbonate levels in patients with CKD, supporting the concept that metabolic acidosis represents a potentially modifiable abnormality rather than simply an unavoidable consequence of kidney failure. [17]

Uribarri and Oh emphasized that greater attention to metabolic acidosis may be important in attempts to slow CKD progression. [18] In the present study, the relatively high prevalence of metabolic acidosis suggests that routine biochemical monitoring is necessary even among patients receiving regular haemodialysis. Persistent low bicarbonate should prompt evaluation of dialysis adequacy, dietary acid load, residual renal function and other potentially reversible factors.

The biological consequences of chronic acid retention may also explain the clinical importance of this finding. Wesson and Simoni described how persistent acid retention during CKD progression may contribute to alterations in ammonium metabolism, kidney injury and disease progression. [19] Kraut and Kurtz similarly emphasized the importance of recognizing metabolic acidosis through appropriate laboratory assessment and addressing underlying causes as part of CKD management. [20]

Evidence also suggests that correction of metabolic acidosis may have beneficial effects. Phisitkul et al. reported that amelioration of metabolic acidosis in patients with reduced GFR was associated with reduced kidney endothelin production and markers of kidney injury. [21] Although these findings cannot be directly extrapolated to all maintenance haemodialysis patients, they support the importance of investigating persistent acidosis rather than considering it an incidental laboratory abnormality.

Nutritional and dietary interventions have also been investigated. de Brito-Ashurst et al. reported that bicarbonate supplementation slowed CKD progression and improved nutritional status in patients with CKD. [22] Goraya et al. demonstrated that reducing dietary acid load through increased consumption of fruits and vegetables or bicarbonate supplementation could attenuate kidney injury in patients with moderately reduced GFR. [23] These findings suggest that management of acid-base status may need to consider both dialysis-related and nutritional factors.

Shah et al. reported an association between serum bicarbonate levels and progression of kidney disease, further supporting the clinical relevance of maintaining appropriate acid-base balance. [24] In addition, Raphael et al. identified reduced serum bicarbonate as a relatively common abnormality in CKD and described several clinical factors associated with low bicarbonate concentrations. [25]

Overall, the present study demonstrates a substantial burden of metabolic acidosis among patients receiving maintenance haemodialysis, with 45.3% showing serum bicarbonate <22 mEq/L. The significant associations with longer dialysis duration, diabetes mellitus, lower albumin and higher potassium emphasize that metabolic acidosis may occur as part of a broader pattern of metabolic and nutritional disturbances. Regular assessment of serum bicarbonate, dialysis adequacy, nutritional status and electrolytes should therefore be incorporated into routine haemodialysis care. Appropriate individualized management of persistent acidosis may help reduce its potential complications, although prospective studies are needed to establish whether correction of metabolic

acidosis improves long-term outcomes in maintenance haemodialysis patients.

CONCLUSION

The present study demonstrates that metabolic acidosis is a common complication among patients with chronic kidney disease receiving maintenance haemodialysis, with 45.3% of the study population having serum bicarbonate levels below 22 mEq/L. Mild metabolic acidosis was the predominant severity category, although a considerable proportion had moderate-to-severe acidosis. Longer duration of haemodialysis and diabetes mellitus were significantly associated with metabolic acidosis. Patients with acidosis also had significantly lower serum albumin and higher blood urea nitrogen and serum potassium levels, suggesting an association with nutritional and metabolic abnormalities.

These findings highlight the importance of **routine pre-dialysis assessment of serum bicarbonate** in patients receiving maintenance haemodialysis. Patients with persistent metabolic acidosis should be evaluated for dialysis adequacy, residual renal function, dietary acid load, nutritional status and other contributing factors. Early identification and individualized management may help reduce complications related to persistent acid retention, including muscle wasting, nutritional deterioration, bone abnormalities and cardiovascular risk. Further prospective multicentre studies are recommended to determine whether effective correction of metabolic acidosis can improve nutritional status, cardiovascular outcomes, hospitalization and long-term survival among haemodialysis patients.

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