

Prevalence and Severity of Metabolic Acidosis in Patients on Maintenance Hemodialysis: A Cross-Sectional Study from Western India

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ABSTRACT

Background: Metabolic acidosis is a modifiable complication of kidney failure that promotes protein catabolism and bone loss. Indian data on its burden in maintenance hemodialysis, where twice-weekly schedules are common, are scarce.

Methods: In this cross-sectional study at a tertiary care centre in Udaipur, India, 120 consecutive adults on maintenance hemodialysis underwent pre-dialysis venous blood gas analysis at the mid-week session. Metabolic acidosis (serum bicarbonate <22 mmol/L) was graded as mild (17.0–21.9), moderate (12.0–16.9) or severe (<12.0 mmol/L). Prevalence was compared across dialysis frequency, residual urine output and diet, and acid-base and electrolyte parameters across severity groups.

Results: Mean age was 50.7 ± 14.9 years; 63.3% were men and 90.0% were dialysed twice weekly. Metabolic acidosis was present in 81 patients (67.5%; 95% CI 58.7–75.2%): mild in 37.5%, moderate in 20.0% and severe in 10.0%. Mean bicarbonate was 19.6 ± 4.8 mmol/L.

Blood pH fell (7.42 to 7.23) and the anion gap rose (7.8 to 17.7 mEq/L) from no to severe acidosis (both $p < 0.001$). Acidosis was more frequent with twice-weekly than thrice-weekly dialysis (72.2% vs 25.0%; $p = 0.002$) and in patients early in their dialysis course (median vintage 9 vs 36 months); bicarbonate correlated with dialysis vintage ($\rho = 0.53$; $p < 0.001$).

Conclusion: Two-thirds of patients on maintenance hemodialysis had metabolic acidosis, and nearly one-third had moderate or severe acidosis. Twice-weekly dialysis and the first year of dialysis identify patients at highest risk. Routine pre-dialysis bicarbonate monitoring and timely correction are warranted.

List of Abbreviations

ANOVA	: Analysis of Variance
AV	: Arteriovenous
CI	: Confidence Interval
CKD	: Chronic Kidney Disease
IQR	: Interquartile Range
KDIGO	: Kidney Disease: Improving Global Outcomes
MHD	: Maintenance Hemodialysis
pCO ₂	: Partial Pressure of Carbon Dioxide
SD	: Standard Deviation

Keywords: Metabolic Acidosis, Maintenance Hemodialysis, Serum Bicarbonate, Prevalence, Anion Gap, Chronic Kidney Disease

Introduction

chronic kidney disease (CKD) is a growing non-communicable disease burden in India. A recent meta-analysis of community-based surveys estimated a pooled adult prevalence of 13.2%, rising from 11.1% in surveys conducted in 2011–2017 to 16.4% in those conducted in 2018–2023 [1]. Many of these patients progress to kidney failure, and maintenance hemodialysis (MHD) remains the most widely used form of kidney replacement therapy in the country.

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Chronic metabolic acidosis is a near-universal complication of kidney failure. The failing kidney cannot excrete the daily diet-derived acid load or regenerate filtered bicarbonate, and intermittent hemodialysis corrects this only transiently: bicarbonate rises towards the dialysate concentration during each session and falls again as acid accumulates between sessions [2,3].

Acidosis is not a passive laboratory finding. It activates the ubiquitin-proteasome pathway in skeletal muscle, impairs the anabolic actions of insulin and insulin-like growth factor-1, and mobilises buffer from bone, thereby promoting muscle wasting, bone disease and protein-energy wasting [4-7].

Current KDIGO guidance defines metabolic acidosis in CKD as a serum bicarbonate persistently below 22 mmol/L and recommends monitoring and treatment to keep bicarbonate within the normal range [8]. Because oral alkali therapy is inexpensive and correction has been associated with clinical benefit, identifying the patients who are acidotic is the first practical step [9].

Indian data in patients on MHD are limited. A single study from Pune reported metabolic acidosis in 62.9% patients on MHD [10,11]. Indian dialysis practice differs from that in most Western units: twice-weekly schedules are common for resource reasons, lengthening the interdialytic interval over which acid accumulates. Larger Indian studies describing the prevalence and severity of acidosis, and the patient groups in which it clusters, are needed. We therefore studied adults on MHD at a tertiary care centre in Udaipur, Rajasthan, to determine the prevalence and severity of metabolic acidosis.

Materials and Methods

Study Design and Setting

This cross-sectional study used enrolment data from a single-centre prospective study conducted over 12 months in the dialysis unit of the Department of Nephrology, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India. All patients were dialysed in the same unit with bicarbonate-based dialysate during standard 4-hour sessions.

Ethics

The study was approved by the Institutional Ethics Committee of Geetanjali Medical College and Hospital and conducted in accordance with the Declaration of Helsinki. Written informed consent, in English or Hindi, was obtained from every patient. Data were de-identified using coded study numbers.

Participants

Adults aged 18 years or above with CKD stage 5 (KDIGO 2024 criteria) [8] on a stable twice- or thrice-weekly MHD schedule for at least one month were eligible. Patients were excluded if they had acute kidney injury or uncertain chronicity; were on peritoneal dialysis or had a functioning transplant; were pregnant; had an acute intercurrent illness at enrolment (e.g. sepsis, acute coronary syndrome, acute gastrointestinal illness); had a non-renal cause of metabolic acidosis (e.g. diabetic ketoacidosis, lactic acidosis, drug-induced acidosis, diarrhoeal bicarbonate loss); had cognitive impairment precluding consent.

Data Collection

A structured proforma was used to record age, sex, education, marital status, socioeconomic class (modified Kuppuswamy scale) [12], smoking, alcohol use and diet. Clinical data included the primary cause and duration of CKD, dialysis vintage, dialysis frequency, vascular access, dry weight, target ultrafiltration, residual urine output (anuric <100 mL/24 h, oliguric 100–500 mL/24 h, preserved >500 mL/24 h), comorbidities and current medications.

Timing of Sampling

To account for cyclical changes in acid-base status between sessions, blood was sampled before the mid-week dialysis session. This avoids both the post-weekend acidaemia of the first session and post-dialytic alkalinisation.

Acid-Base and Electrolyte Measurements

A pre-dialysis venous sample was analysed on a point-of-care blood gas analyser for pH, bicarbonate and pCO₂. Serum sodium, potassium, chloride and ionised calcium were measured on the same sample, and the anion gap was calculated as sodium – (chloride + bicarbonate). Patients were classified by measured bicarbonate as having no acidosis (≥ 22.0 mmol/L) or mild (17.0–21.9 mmol/L), moderate (12.0–16.9 mmol/L) or severe (<12.0 mmol/L) metabolic acidosis.

Statistical Analysis

Data were analysed in Python 3 using the SciPy and stats models libraries. Continuous variables are presented as mean $\pm$ SD and median (IQR), and categorical variables as number (%). Normality was assessed by the Shapiro-Wilk test and histograms. The 95% CI of the prevalence of acidosis was calculated by the Wilson method. Acid-base and electrolyte parameters were compared across the four severity groups by one-way ANOVA and, because several variables were not normally distributed, also by the Kruskal-Wallis test. Prevalence of acidosis by dialysis frequency, residual urine output and diet was compared by the chi-square or Fisher's exact test, and bicarbonate between two groups by the Mann-Whitney U test. The correlation of bicarbonate with dialysis vintage was assessed by Spearman's ρ . All tests were two-sided, with $p < 0.05$ considered significant.

Results

Study Population

Of 121 patients screened, one (aged 14 years) was excluded, and 120 were enrolled and analysed. Mean age was 50.74 ± 14.90 years and 76 (63.3%) were men (Table 1). Hypertensive nephrosclerosis (32.5%) and diabetic nephropathy (30.0%) were the leading causes of CKD. Median dialysis vintage was 12.0 months (IQR 7.0–42.8). Most patients were dialysed twice weekly (90.0%) through an arteriovenous fistula (72.5%), and hypertension was near-universal (96.7%).

Prevalence and Severity of Metabolic Acidosis

Metabolic acidosis was present in 81 of 120 patients, a prevalence of 67.5% (95% CI 58.7–75.2%) (Figure 1). It was mild in 45 (37.5%), moderate in 24 (20.0%) and severe in 12 (10.0%); moderate-to-severe acidosis (bicarbonate <17 mmol/L) was therefore present in 36 patients (30.0%). Mean bicarbonate was 19.59 ± 4.77 mmol/L, and blood pH was below 7.35 in 61 patients (50.8%).

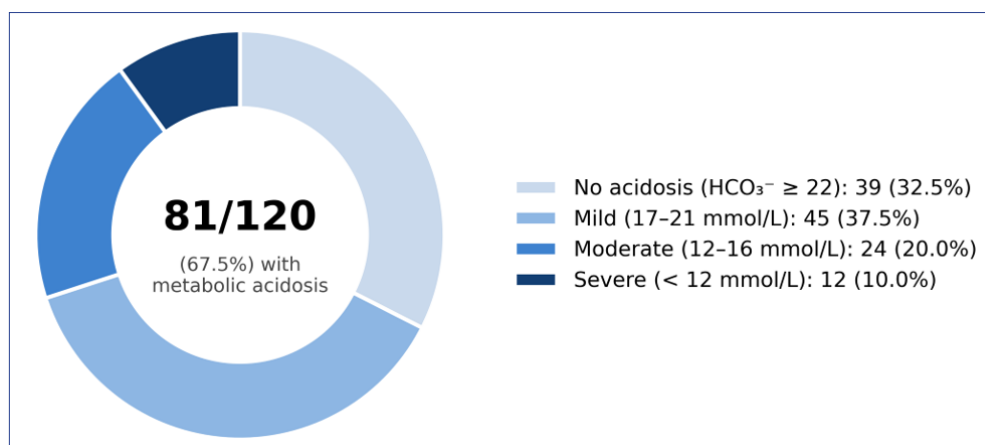


Figure 1: Prevalence and severity of metabolic acidosis (n = 120). Severity graded by pre-dialysis serum bicarbonate: mild 17.0–21.9, moderate 12.0–16.9, severe <12.0 mmol/L

Table 1: Baseline Demographic and Clinical Characteristics (N = 120)

Characteristic	Value
Age, years, mean $\pm$ SD (median)	50.74 $\pm$ 14.90 (52.50)
Male sex, n (%)	76 (63.3)
Married, n (%)	104 (86.7)
Education: secondary or above, n (%)	110 (91.7)
Socioeconomic class (modified Kuppaswamy): upper / upper-middle / middle / lower-middle, n (%)	3 (2.5) / 34 (28.3) / 60 (50.0) / 23 (19.2)
Never smoked, n (%)	88 (73.3)
Never consumed alcohol, n (%)	70 (58.3)
Vegetarian diet, n (%)	66 (55.0)
Cause of CKD: hypertensive nephrosclerosis / diabetic nephropathy / unknown / chronic glomerulonephritis / obstructive uropathy / other, n (%)	39 (32.5) / 36 (30.0) / 15 (12.5) / 13 (10.8) / 13 (10.8) / 4 (3.3)
Duration of CKD, years, median (IQR)	5.0 (3.0–7.0)
Dialysis vintage, months, median (IQR)	12.0 (7.0–42.8)
Dialysis frequency: twice / thrice weekly, n (%)	108 (90.0) / 12 (10.0)
Vascular access: AV fistula / permanent catheter / temporary catheter, n (%)	87 (72.5) / 20 (16.7) / 13 (10.8)
Residual urine output: anuric / oliguric / preserved, n (%)	29 (24.2) / 64 (53.3) / 27 (22.5)
Dry weight, kg, mean $\pm$ SD	57.44 $\pm$ 9.95
Target ultrafiltration, L/session, mean $\pm$ SD	2.60 $\pm$ 0.84
Hypertension / diabetes mellitus / coronary artery disease, n (%)	116 (96.7) / 37 (30.8) / 23 (19.2)
Prior stroke / thyroid disease / failed kidney transplant, n (%)	16 (13.3) / 11 (9.2) / 6 (5.0)
Erythropoiesis-stimulating agent / antihypertensives, n (%)	114 (95.0) / 117 (97.5)
Phosphate binders / vitamin D analogues, n (%)	120 (100) / 120 (100)

Blood pH fell progressively from 7.42 ± 0.07 in patients without acidosis to 7.23 ± 0.11 in severe acidosis, and the anion gap rose from 7.80 ± 2.62 to 17.68 ± 3.40 mEq/L (both $p < 0.001$ on ANOVA and Kruskal-Wallis tests) (Table 2). pCO_2 was lowest in moderate acidosis (32.83 ± 6.30 mmHg; $p = 0.002$). Serum sodium was modestly lower in more acidotic patients (130.69 to 127.42 mEq/L; $p = 0.024$), whereas potassium, chloride and ionised calcium did not differ significantly across groups.

Table 2: Acid-Base and Electrolyte Parameters by Severity of Metabolic Acidosis, Mean $\pm$ Sd (N = 120)

Parameter	All (n = 120)	None (n = 39)	Mild (n = 45)	Moderate (n = 24)	Severe (n = 12)	ANOVA p	Kruskal-Wallis p
Blood pH	7.35 $\pm$ 0.09	7.42 $\pm$ 0.07	7.34 $\pm$ 0.05	7.31 $\pm$ 0.08	7.23 $\pm$ 0.11	<0.001	<0.001
Bicarbonate, mmol/L	19.59 $\pm$ 4.77	24.84 $\pm$ 1.84	19.74 $\pm$ 1.33	15.10 $\pm$ 1.07	10.90 $\pm$ 0.83	<0.001	<0.001
pCO_2 , mmHg	36.74 $\pm$ 5.92	38.44 $\pm$ 4.17	37.29 $\pm$ 4.81	32.83 $\pm$ 6.30	37.00 $\pm$ 9.93	0.002	0.004

Anion gap, mEq/L	11.64 ± 4.39	7.80 ± 2.62	11.48 ± 2.99	15.15 ± 2.98	17.68 ± 3.40	<0.001	<0.001
Sodium, mEq/L	129.68 ± 3.37	130.69 ± 2.67	129.62 ± 3.61	129.29 ± 3.21	127.42 ± 3.87	0.024	0.042
Potassium, mEq/L	5.01 ± 0.93	4.78 ± 0.95	4.95 ± 0.89	5.32 ± 0.83	5.36 ± 1.06	0.077	0.071
Chloride, mEq/L	98.46 ± 1.48	98.05 ± 1.76	98.40 ± 1.32	99.04 ± 1.04	98.83 ± 1.47	0.054	0.115
Ionised calcium, mmol/L	0.87 ± 0.12	0.87 ± 0.10	0.87 ± 0.12	0.87 ± 0.11	0.82 ± 0.13	0.504	0.455

Severity graded by pre-dialysis serum bicarbonate: none ≥ 22.0 , mild 17.0–21.9, moderate 12.0–16.9, severe <12.0 mmol/L. ANOVA, one-way analysis of variance; $p\text{CO}_2$, partial pressure of carbon dioxide.

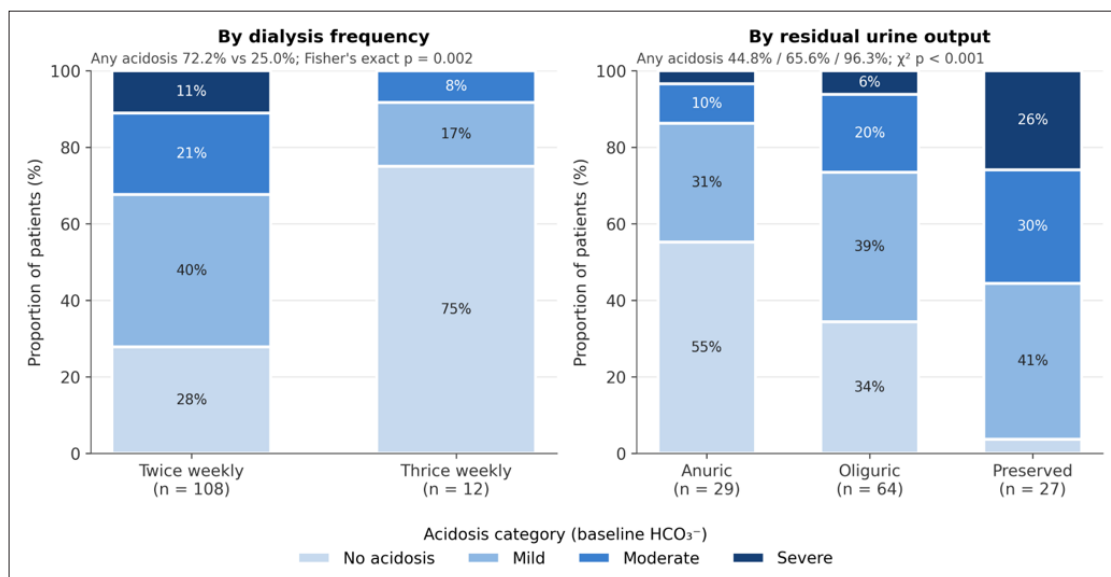


Figure 2: Severity of metabolic acidosis by dialysis frequency (left) and residual urine output (right) ($n = 120$). Anuric <100 mL/24 h; oliguric 100–500 mL/24 h; preserved >500 mL/24 h

Distribution of Acidosis by Patient Characteristics

Acidosis was present in 78 of 108 patients (72.2%) dialysed twice weekly compared with 3 of 12 (25.0%) dialysed thrice weekly (Fisher's exact $p = 0.002$), and mean bicarbonate was 19.09 ± 4.60 versus 24.12 ± 3.98 mmol/L (Mann-Whitney $p < 0.001$) (Figure 2). Acidosis was more frequent with preserved urine output (26/27, 96.3%) than with oliguria (42/64, 65.6%) or anuria (13/29, 44.8%) ($\chi^2 p < 0.001$). This reflected dialysis vintage and schedule rather than residual function: 25 of the 27 patients with preserved urine output had been on dialysis for 12 months or less and all were dialysed twice weekly, whereas 10 of the 12 thrice-weekly patients were anuric. Bicarbonate correlated positively with dialysis vintage (Spearman $\rho = 0.53$, $p < 0.001$); median vintage was 9 months in patients with acidosis and 36 months in those without. Prevalence did not differ between vegetarian (44/66, 66.7%) and non-vegetarian (37/54, 68.5%) patients ($\chi^2 p = 0.98$).

Discussion

In this cohort of 120 adults on MHD, two-thirds had metabolic acidosis and nearly one in three had moderate or severe acidosis. Acidosis clustered in patients dialysed twice weekly and in those early in their dialysis course.

The prevalence of 67.5% closely matches the 62.9% reported from Pune by Sajgure et al., whose cohort also had a similar mean pre-dialysis bicarbonate (20.37 ± 4.94 mmol/L versus 19.59 ± 4.77 mmol/L in the present study) [10,11]. That the

two estimates agree, nearly a decade apart and at different centres, suggests a genuine population-level burden rather than a single-site artefact. The present cohort is more than three times larger, which narrows the uncertainty around this estimate. The prevalence is far higher than the roughly 15% of the overall CKD population with overt acidosis in registry-based estimates [13]; this is expected, because such estimates average across all CKD stages, whereas acid retention is most pronounced in kidney failure [13,14].

The acid-base profile was internally consistent. The progressive rise in anion gap with worsening acidosis indicates retention of unmeasured anions such as sulphate, phosphate and organic acids, the typical pattern of acidosis in advanced kidney failure [2,14]. Half the cohort had a pre-dialysis blood pH below 7.35, showing that acidosis in many patients was not fully compensated before dialysis.

Two modifiable factors stood out. First, acidosis affected 72.2% of twice-weekly but only 25.0% of thrice-weekly patients. Twice-weekly dialysis, common in India for resource reasons, lengthens the interdialytic interval over which non-volatile acid accumulates. Although the thrice-weekly group was small, this supports the dialysis schedule as an important driver of the higher prevalence seen in Indian cohorts. Second, acidosis was concentrated in patients early in their dialysis course: median vintage was 9 months with acidosis and 36 months without, and bicarbonate correlated with vintage ($\rho = 0.53$). This explains the

apparently paradoxical finding that acidosis was commonest in patients with preserved urine output (96.3%). These were mostly recent starters on twice-weekly dialysis, whose residual function could no longer excrete the daily acid load and whose dialysis prescription was still modest. Diet type was not associated with acidosis, despite the lower acid load of plant-predominant diets.

Clinical Implications

Routine monthly measurement of pre-dialysis serum bicarbonate, standardised to the mid-week session, would identify roughly two in three MHD patients with acidosis, a simple and inexpensive target for correction [8]. Patients in their first year of dialysis and those on twice-weekly schedules are practical groups for focused monitoring. In these patients, review of the dialysis prescription, including dialysis frequency, should accompany oral alkali therapy.

Limitations

This study has several limitations. First, acid-base status was assessed from a single mid-week measurement, which may misclassify patients whose bicarbonate fluctuates between sessions. Second, it was a single-centre study with a predominantly twice-weekly schedule, which may limit generalisability to thrice-weekly practice. Third, the thrice-weekly subgroup ($n = 12$) was small, so the comparison by dialysis frequency is imprecise. Fourth, dialysis adequacy (Kt/V), dietary protein intake and dialysate bicarbonate prescription were not analysed, so their contribution to acidosis could not be assessed. Finally, the cross-sectional design identifies associations but cannot establish causes of acidosis.

Conclusion

Metabolic acidosis affected about two-thirds of patients on MHD in this Indian cohort, and nearly one-third had moderate or severe acidosis. It was particularly common with twice-weekly dialysis and in the first year of dialysis. Routine pre-dialysis bicarbonate monitoring should be part of standard MHD care, with attention to these high-risk groups, and multicentre studies are needed to confirm these findings across Indian dialysis practice.

Declarations

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Conflict of Interest: The authors declare no conflict of interest.

Ethical Approval: Approved by the Institutional Ethics Committee, Geetanjali Medical College and Hospital, Udaipur

Informed Consent: Written informed consent was obtained from all participants.

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