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ACID–BASE STATUS AND DYSELECTROLYTEMIA IN CHILDREN UNDER FIVE YEARS HOSPITALISED WITH ACUTE GASTROENTERITIS AT A TERTIARY ARMED FORCES HOSPITAL IN NORTH INDIA: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Acute gastroenteritis (AGE) produces combined acid–base and electrolyte disturbances whose frequency and relationship to clinical dehydration are poorly characterised in Indian children.

Objective: To describe acid–base status and dyselectrolytemia in under-five children hospitalised with AGE and to examine their relationship with the severity of dehydration.

Methods: Prospective observational study at Command Hospital (Central Command), Lucknow, from September 2022 to February 2024, including 72 children aged 2–60 months. Dehydration was graded by WHO criteria. Venous blood obtained before rehydration was analysed for sodium, potassium, chloride, pH, bicarbonate, standard base excess and pCO_2; the anion gap was calculated and a repeat sodium and potassium was available. Fisher's exact, Mann–Whitney and Kruskal–Wallis tests were used; correlation, logistic regression and ROC analyses were exploratory.

Results: Mean age was 26.3 ± 16.6 months and 55.6% were boys. Dehydration was absent in 41 (56.9%), some in 28 (38.9%) and severe in 3 (4.2%). Dyselectrolytemia affected 27 children (37.5%): hyponatraemia 20.8%, hypernatraemia 5.6%, hypokalaemia 2.8% and hyperkalaemia 6.9%. Low bicarbonate (<22 mmol/L) occurred in 19 (26.4%) and metabolic acidosis in 12 (16.7%), including all three severely dehydrated children. Dehydrated children had lower pCO_2 (40.2 vs 45.6 mmHg; p<0.001), higher stool frequency (p=0.002) and chloride (p=0.043), and more often low bicarbonate (38.7% vs 17.1%; OR 2.95, 95% CI 1.02–8.53; p=0.058). Sodium, potassium and chloride abnormalities were not associated with dehydration grade. Potassium fell after rehydration (4.37 to 4.14 mmol/L; p=0.015) and 20.8% were hypokalaemic on repeat testing.

Conclusion: About one in six children had metabolic acidosis and over a third an electrolyte derangement, poorly predicted by dehydration grade. Selective blood-gas and electrolyte testing and post-rehydration potassium monitoring are advisable; larger multicentre studies are needed.

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INTRODUCTION

Diarrhoeal disease remains one of the principal infectious causes of morbidity and death in children under five years of age worldwide, with the greatest burden in South Asia and sub-Saharan Africa [1–4]; it still accounted for roughly 9% of all deaths in children under five years in 2019 [3]. Rotavirus, enterotoxigenic Escherichia coli, Shigella and other enteric pathogens continue to drive severe disease despite progress in vaccination, sanitation and case management [4,5]. Oral rehydration solution (ORS) and zinc supplementation have averted a very large number of deaths, but a substantial minority of children are still hospitalised with significant dehydration [6–8], and India

continues to record a high national burden of under-five diarrhoea in its household surveys [9].

Watery stool is rich in bicarbonate, sodium, potassium and chloride, so progressive faecal loss predisposes to non-anion-gap metabolic acidosis, hypokalaemia and disturbances of plasma sodium [10,11]. Vomiting adds loss of hydrogen and chloride ions, and hypovolaemia may induce hypoperfusion, lactic acidosis, ketosis and pre-renal kidney injury, which raise the anion gap and impair renal acid excretion [11,12] (Figure 1). The net acid–base and electrolyte phenotype is therefore heterogeneous, and is further modulated by the composition of fluids ingested before admission, including hypotonic home fluids and incorrectly prepared ORS [13,14].

Plasma sodium is the dominant determinant of tonicity, and both hypo- and hypernatraemia carry neurological risk if mismanaged [15–18]; hospital-acquired hyponatraemia from hypotonic intravenous fluids is a recognised iatrogenic hazard [19]. Guidelines recommend that routine biochemistry is unnecessary in most children with mild gastroenteritis but is indicated when dehydration is moderate–severe, when intravenous therapy is required or when the clinical picture is atypical [7,8,20]. However, bedside signs correlate imperfectly with biochemical derangement [21,22], and local data describing the combined electrolyte and acid–base picture, including the anion gap and respiratory compensation, are scarce for Indian children, particularly in tertiary military hospitals that serve a defined, referral-based population.

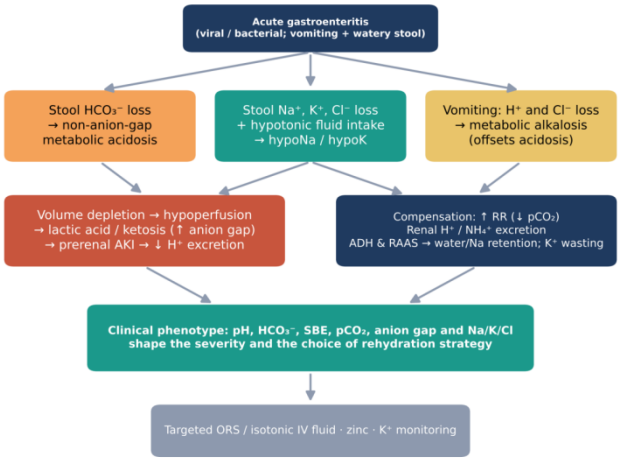


Figure 01: Conceptual model of the mechanisms linking acute gastroenteritis to acid–base and electrolyte disturbance, the compensatory responses, and the resulting therapeutic implications.

We therefore conducted a prospective observational study with two objectives: (i) to describe acid–base status and dyselectrolytemia in children under five years admitted with AGE, and (ii) to examine the relationship of these disturbances with the severity of clinical dehydration. We additionally explored clinical correlates of metabolic acidosis and dyselectrolytemia and the change in sodium and potassium after rehydration.

MATERIALS AND METHODS

Study design, setting and reporting

This was a single-centre, prospective, hospital-based observational study conducted in the Department of Paediatrics, Command Hospital (Central Command), Lucknow-a tertiary-care armed forces hospital in North India-from September 2022 to February 2024. The report follows the STROBE statement for observational studies [23].

Participants

Consecutive children aged under five years admitted with acute gastroenteritis (acute onset of loose watery stools, with or without vomiting and fever, of <14 days' duration) and with a complete history, examination and investigations were eligible. Exclusion criteria were: refusal of guardian consent; age above five years; dyselectrolytemia attributable to post-surgical or chemo-radiation status; clinical picture suggesting malabsorption; operated short-bowel syndrome; and loose stools due to purgative use. Participant flow is shown in Figure 02.

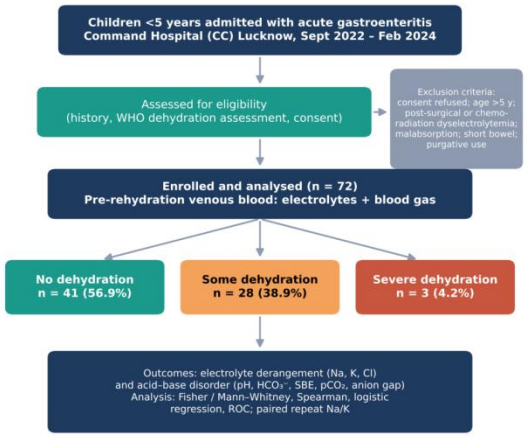


Figure 02: STROBE-style flow of participants and analytical plan. Dehydration was graded using WHO criteria at admission.

Sample Size

The sample size was calculated with the single-proportion formula $n = Z^2 \cdot p(1-p)/d^2$, assuming an expected prevalence of dyselectrolytemia of 25% from prior Indian data, 95% confidence ($Z = 1.96$) and an absolute precision of 10%, yielding $n = 72$. The study was therefore powered to estimate overall prevalence, not to detect differences between dehydration strata; all subgroup comparisons are exploratory.

Clinical Assessment

Demographic data, duration (days) and frequency of loose stools, stool volume and consistency, number of vomiting episodes, fever, pre-hospital ORS use and its method of preparation were recorded on a structured proforma. Pulse rate, respiratory rate and SpO_2 were measured at admission. Dehydration was graded as no dehydration, some dehydration or severe dehydration according to WHO criteria (general condition, eyes, thirst, skin pinch and oral mucosa) [7]. Management followed WHO/ESPGHAN principles: low-osmolarity ORS for all children, intravenous isotonic fluids for those with some/severe dehydration judged unable to be rehydrated orally, zinc supplementation, and ondansetron as required [7,8,24].

Laboratory measurements and definitions

Before starting rehydration, venous blood was drawn for serum sodium, potassium and chloride and for blood-gas analysis (pH, HCO_3^-, standard base excess [SBE], pCO_2, pO_2). The anion gap (AG) was calculated as $Na^+ - (Cl^- + HCO_3^-)$ without potassium. A repeat sodium and

potassium measurement was recorded for every child. Operational definitions are in Table 1. Because the pO_2 values (median 55 mmHg) are characteristic of venous or capillary rather than arterial blood, interpretation of pH and pCO_2 allowed for the known small venous–arterial differences (venous pH ≈0.03–0.04 lower and pCO_2 ≈4–6 mmHg higher) [25], and a sensitivity analysis with a lower pH threshold was performed. The sampling site and the interval to the repeat measurement were not recorded on the proforma (see Limitations).

Table 01: Operational definitions used in this study

Variable	Definition (primary analysis)
Hyponatraemia / hypernatraemia	Serum Na^+ <135 / >145 mmol/L
Hypokalaemia / hyperkalaemia	Serum K^+ <3.5 / >5.0 mmol/L
Hypochloraemia / hyperchloraemia	Serum Cl^- <98 / >107 mmol/L
Dyselectrolytemia	Any of the six abnormalities above
Acidaemia	pH <7.35 (sensitivity analysis: pH <7.30)
Low bicarbonate	HCO_3^- <22 mmol/L
Base deficit	Standard base excess ≤ −3 mmol/L
Metabolic acidosis	pH <7.35 and HCO_3^- <22 mmol/L
Raised anion gap	AG >16 mmol/L (conservative threshold allowing for analyser variability)
Respiratory acidosis (apparent)	pH <7.35, pCO_2 >45 mmHg and HCO_3^- ≥22 mmol/L
Expected pCO_2 in metabolic acidosis	1.5 × HCO_3^- + 8 (± 2) mmHg (Winter's formula) [26]
Dehydration	WHO grade: none / some / severe; analysed as three groups and as none vs some/severe

Reference thresholds are standard paediatric laboratory cut-offs.

Statistical analysis

Continuous data are presented as mean ± SD (and median [IQR] where skewed); categorical data as n (%). Because only three children had severe dehydration, the primary contrast was none (n = 41) versus some/severe (n = 31), with three-group Kruskal–Wallis and ordinal Spearman tests reported for completeness. Categorical associations used Fisher's exact test; continuous variables between two groups used the Mann–Whitney U test. Paired admission/repeat Na^+ and K^+ were compared with the Wilcoxon signed-rank test. Unadjusted odds ratios (Haldane–Anscombe correction, Woolf 95% CI) were computed for pre-specified clinical correlates, and a multivariable logistic model for low bicarbonate included four predictors (dehydration, age ≤12 months, stool frequency, respiratory rate) to respect the events-per-variable limit. Discrimination of clinical variables for some/severe dehydration was summarised by the area under the ROC curve. Two-sided $p<0.05$ was considered statistically significant; no adjustment for multiplicity was made and subgroup findings are hypothesis-generating. Analyses used Python (SciPy/NumPy).

Data handling and ethics

Two apparent decimal-transcription errors in the dataset (one pH recorded as 728, corrected to 7.28; one repeat potassium recorded as 32, corrected to 3.2) were corrected before analysis; stool frequency recorded as a range was converted to its mid-point. The study was approved by the Institutional Ethics Committee of Command Hospital (CC), Lucknow, before enrolment, and written informed consent was obtained from the parent or guardian of every child (ethics certificate IEC/37/2022), in accordance with the Declaration of Helsinki and ICMR guidelines.

RESULTS

Participant characteristics

Seventy-two children were enrolled (Figure 2). The mean age was 26.3 ± 16.6 months (median 24, IQR 12–36; range 2–60); 29.2% were aged ≤12 months and 56.9% ≤24 months. Forty children (55.6%) were boys (Figure 3). Loose-stool frequency had a median of 6.5 per day (IQR 5.0–8.6) for a mean duration of 3.2 ± 1.6 days; 41 children (56.9%) vomited (median 5 episodes among those vomiting) and 13 (18.1%) had fever. Mean pulse rate was 109.1 ± 10.7/min, respiratory rate 28.8 ± 5.4/min and SpO_2 97.5 ± 1.0% (Table 02).

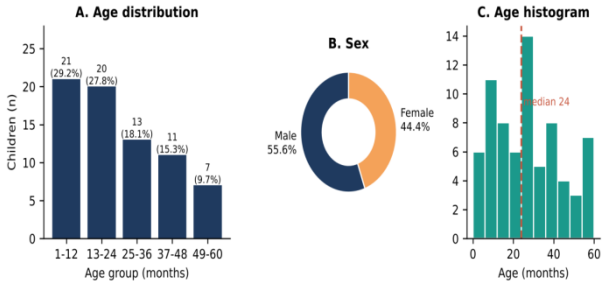


Figure 03: Demographic profile. (A) Age distribution in 12-month bands; (B) sex distribution; (C) histogram of age with median (dashed).

Table 02: Baseline clinical characteristics of 72 children with acute gastroenteritis, overall and by dehydration status

Characteristic	All (n=72)	No dehydration (n=41)	Some/severe (n=31)	p
Demographics				
Age, months (mean ± SD)	26.3 ± 16.6	25.8 ± 16.6	27.1 ± 16.8	0.698
Age ≤12 months, n (%)	21 (29.2 %)	13 (31.7%)	8 (25.8%)	—
Male sex, n (%)	40 (55.6 %)	24 (58.5%)	16 (51.6%)	0.635
Diarrhoea history				
Stool frequency, per day (mean ± SD)	7.9 ± 4.1	6.6 ± 3.3	9.5 ± 4.5	0.002
Duration, days (mean ± SD)	3.2 ± 1.6	3.3 ± 1.8	2.9 ± 1.2	0.528

Vomiting present, n (%)	41 (56.9 %)	20 (48.8%)	21 (67.7%)	0.150
Fever present, n (%)	13 (18.1 %)	11 (26.8%)	2 (6.5%)	0.032
Examination at admission				
Pulse rate, /min (mean ± SD)	109.1 ± 10.7	106.4 ± 8.2	112.7 ± 12.6	0.052
Respiratory rate, /min (mean ± SD)	28.8 ± 5.4	27.8 ± 5.4	30.1 ± 5.2	0.055
SpO_2, % (mean ± SD)	97.5 ± 1.0	97.5 ± 1.0	97.5 ± 1.1	0.734
Dry oral mucosa, n (%)	32 (44.4 %)	8 (19.5%)	24 (77.4%)	<0.0 01
Sick-looking child, n (%)	11 (15.3 %)	2 (4.9%)	9 (29.0%)	0.007
Treatment				
IV fluid given, n (%)	28 (38.9 %)	0 (0.0%)	28 (90.3%)	<0.0 01

p: Mann–Whitney U (continuous) or Fisher's exact test (categorical), none vs some/severe. All children received ORS, zinc and ondansetron as required; 9 received paracetamol.

Severity of dehydration

Dehydration was absent in 41 children (56.9%), "some" in 28 (38.9%) and severe in 3 (4.2%). Dry oral mucosa was present in 32 (44.4%) and 11 (15.3%) appeared sick; both were concentrated in the dehydrated groups (Table 2, Figure 4). Stool frequency was the one history variable clearly higher in dehydrated children (mean 9.5 vs 6.7 per day, p=0.002; AUC for some/severe dehydration 0.71), whereas respiratory rate (AUC 0.63) and pulse rate (AUC 0.63) discriminated only modestly (Figure 12).

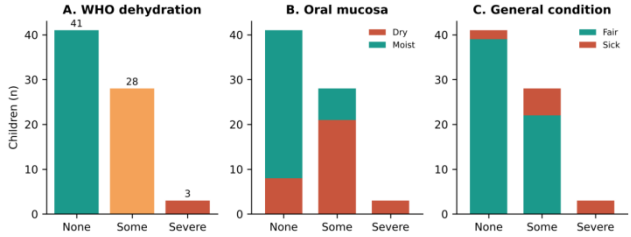


Figure 04: Clinical severity at admission. (A) WHO dehydration grade; (B) oral mucosa and (C) general condition stratified by dehydration grade.

Serum electrolytes

At admission mean serum sodium was 137.3 ± 3.7 mmol/L (range 131–150), potassium 4.37 ± 0.44 mmol/L (3.1–5.3) and chloride 102.7 ± 2.9 mmol/L (95–110) (Table 3). Twenty-seven children (37.5%) had at least one electrolyte abnormality, most often an isolated sodium disturbance (16 children; combined abnormalities in 3). Hyponatraemia was present in 15 (20.8%) and hypernatraemia in 4 (5.6%); hypokalaemia in 2 (2.8%) and hyperkalaemia in 5 (6.9%); hypochloraemia and hyperchloraemia in 2 each (2.8%) (Table 04, Figure 05).

Table 03: Admission laboratory parameters (n = 72)

Parameter	Mean ± SD	Median (IQR)	Range
Sodium, mmol/L	137.3 ± 3.7	136 (135–139)	131–150
Potassium, mmol/L	4.37 ± 0.44	4.3 (4.1–4.7)	3.1–5.3
Chloride, mmol/L	102.7 ± 2.9	102 (100–105)	95–110
pH	7.32 ± 0.07	7.32 (7.29–7.39)	7.20–7.49
Bicarbonate, mmol/L	23.3 ± 3.3	23.2 (21.7–25.7)	16.0–32.0
Standard base excess, mmol/L	−3.9 ± 2.1	−4.0 (−5.0 to −3.0)	−11.0 to 3.0
pCO_2, mmHg	43.3 ± 6.8	42.0 (38.8–46.5)	29.7–63.0
pO_2, mmHg	54.2 ± 7.4	55.0 (48.0–60.0)	40.0–70.0
Anion gap, mmol/L	11.4 ± 4.9	11.8 (8.0–15.0)	0.0–22.0
Repeat sodium, mmol/L	136.7 ± 2.9	—	130–146
Repeat potassium, mmol/L	4.14 ± 0.68	—	2.0–5.6

Negative standard base excess values denote base deficit.

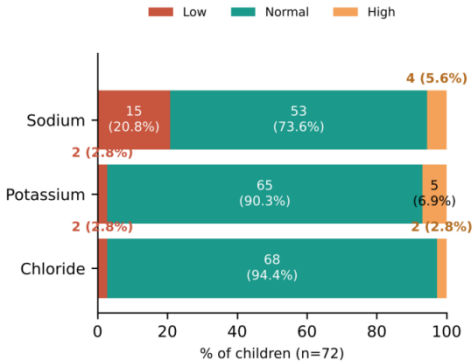


Figure 05: Prevalence of low, normal and high serum sodium, potassium and chloride at admission (n = 72).

Table 04: Pattern of electrolyte derangement and its distribution by dehydration status

Abnormality	All, n (%)	No dehydration (n=41)	Some/severe (n=31)	Fisher p
Hyponatraemia (Na^+ <135)	15 (20.8 %)	9 (22.0%)	6 (19.4%)	1.000
Hypernatraemia (Na^+ >145)	4 (5.6%)	2 (4.9%)	2 (6.5%)	1.000
Hypokalaemia (K^+ <3.5)	2 (2.8%)	1 (2.4%)	1 (3.2%)	1.000
Hyperkalaemia (K^+ >5.0)	5 (6.9%)	2 (4.9%)	3 (9.7%)	0.646

Hypochloraemia (Cl^- <98)	2 (2.8%)	1 (2.4%)	1 (3.2%)	1.000
Hyperchloraemia (Cl^- >107)	2 (2.8%)	0 (0.0%)	2 (6.5%)	0.182
Any dyselectrolytemia	27 (37.5%)	14 (34.1%)	13 (41.9%)	0.624

The 31 children with some/severe dehydration include 3 with severe dehydration, none of whom had a sodium, potassium or chloride abnormality.

No electrolyte abnormality was significantly more frequent in dehydrated children (Table 04; all p>0.18). Only two children were hypokalaemic (one in each stratum), so no inference about potassium and dehydration is possible. Mean sodium, potassium and chloride did not differ materially between strata, although chloride was higher in dehydrated children (Table 06).

Acid–base status

Mean pH was 7.32 ± 0.07, bicarbonate 23.3 ± 3.3 mmol/L, SBE −3.9 ± 2.1 mmol/L and pCO_2 43.3 ± 6.8 mmHg. Forty-nine children (68.1%) had pH <7.35 (19, 26.4%, had pH <7.30), 19 (26.4%) had bicarbonate <22 mmol/L and 56 (77.8%) had a base deficit ≤ −3 mmol/L (Figure 6). Twelve children (16.7%) met the criterion for metabolic acidosis; their mean bicarbonate was 18.8 mmol/L and mean anion gap 16.1 mmol/L, six having a raised gap (>16). Among all 19 children with low bicarbonate, 12 had a normal and 7 a raised anion gap, consistent with predominantly hyperchloraemic (stool-bicarbonate-loss) acidosis with a lactate/ketone contribution in a minority. Sixteen children (22.2%) had pH <7.35 with pCO_2 >45 mmHg and normal bicarbonate, and four had mixed low bicarbonate with high pCO_2 (Table 05).

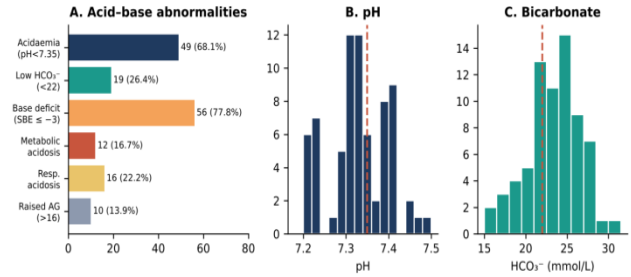


Figure 06: Acid–base profile at admission. (A) Prevalence of acid–base abnormalities (categories overlap); (B) distribution of pH, dashed line 7.35; (C) distribution of bicarbonate, dashed line 22 mmol/L.

Table 05: Acid–base classification of 72 children at admission

Category	N (%)	Comment
Acidaemia (pH <7.35)	49 (68.1%)	26.4% (n=19) if pH <7.30
Low bicarbonate (<22 mmol/L)	19 (26.4%)	10 (13.9%) if <20 mmol/L
Base deficit (SBE ≤ −3 mmol/L)	56 (77.8%)	Mean SBE −3.9 ± 2.1
Metabolic acidosis (pH <7.35 + HCO_3^- <22)	12 (16.7%)	6 with AG >16
Normal-AG acidosis (HCO_3^- <22, AG ≤16)	12 (16.7%)	Of 19 low-bicarbonate children
Raised-AG acidosis (HCO_3^- <22, AG >16)	7 (9.7%)	Lactate/ketones likely
Apparent respiratory acidosis	16 (22.2%)	Likely venous/crying artefact (see text)
Mixed low HCO_3^- and high pCO_2	4 (5.6%)	Inadequate compensation
Alkalaemia (pH >7.45)	2 (2.8%)	—

Applying Winter's formula to the 12 acidotic children, observed pCO_2 equalled the expected value (±2 mmHg) in three, was higher (inadequate compensation or venous sampling) in eight and lower (additional respiratory alkalosis) in one. By contrast the three severely dehydrated children all had pH 7.20–7.23, HCO_3^- 16.0–18.0 mmol/L, SBE −5.1 to −6.0 mmol/L and pCO_2 29.7–35.0 mmHg, a textbook compensated metabolic acidosis, with anion gaps of 15.0–18.8 mmol/L (Figure 07).

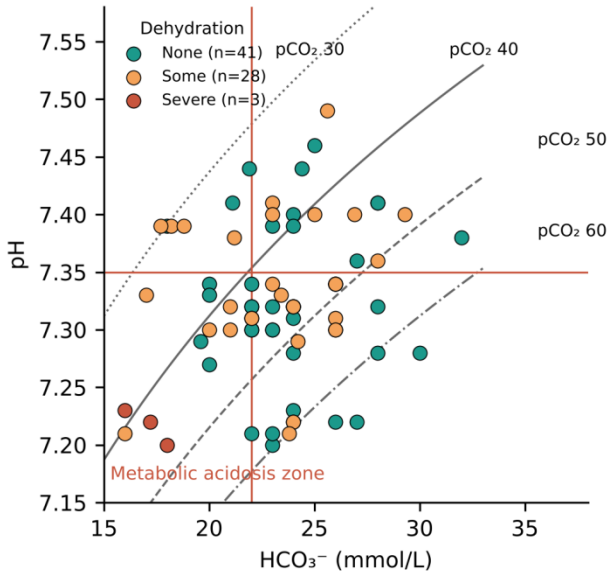


Figure 07: Davenport-style pH–bicarbonate plot with pCO_2 isobars (Henderson–Hasselbalch). Red lines mark pH 7.35 and HCO_3^- 22 mmol/L; points in the lower-left quadrant meet the definition of metabolic acidosis. The three severely dehydrated children cluster at low bicarbonate with low pCO_2.

Acid–base and electrolyte indices by dehydration grade

Table 06: Admission acid–base and electrolyte indices by WHO dehydration grade (mean ± SD)

Parameter	None (n=41)	Some (n=28)	Severe (n=3)	Kruskal–Wallis p	Spearman ρ (p)
pH	7.32 ± 0.07	7.34 ± 0.06	7.22 ± 0.02	0.025	0.01 (0.922)
Bicarbonate, mmol/L	23.9 ± 2.9	23.0 ± 3.4	17.1 ± 1.0	0.017	−0.21 (0.082)
SBE, mmol/L	−3.7 ± 2.0	−3.8 ± 2.3	−5.7 ± 0.5	0.069	−0.18 (0.128)
pCO_2, mmHg	45.6 ± 5.0	41.2 ± 7.4	31.6 ± 3.0	<0.001	−0.49 (<0.001)
Anion gap, mmol/L	11.2 ± 4.9	11.1 ± 5.0	16.3 ± 2.2	0.156	0.06 (0.621)
Sodium, mmol/L	137.2 ± 3.8	137.3 ± 3.8	139.3 ± 2.3	0.333	0.10 (0.393)
Potassium, mmol/L	4.35 ± 0.41	4.37 ± 0.49	4.67 ± 0.42	0.451	0.09 (0.433)
Chloride, mmol/L	102.1 ± 2.5	103.2 ± 3.3	106.0 ± 1.0	0.030	0.27 (0.021)

Severe group n=3: its estimates are imprecise and drive the three-group tests. For none vs some/severe: HCO_3^- 23.9 vs 22.4 (p=0.182), pCO_2 45.6 vs 40.2 (p<0.001), Cl^- 102.1 vs 103.5 (p=0.043), pH 7.32 vs 7.33 (p=0.588).

Bicarbonate was lowest and pCO_2 lowest in the severe group, producing a significant three-group difference for both (Table 6, Figure 8). With the three severely dehydrated children pooled with those with some dehydration, the bicarbonate difference was attenuated (23.9 vs 22.4 mmol/L, p=0.182) but low bicarbonate became more frequent (12/31, 38.7% vs 7/41, 17.1%; OR 2.95, 95% CI 1.02–8.53; Fisher p=0.058). pCO_2 fell progressively with dehydration (ρ = −0.49, p<0.001), consistent with ventilatory compensation of metabolic acidosis, and chloride rose (ρ = 0.27, p=0.021). Sodium, potassium, pH and anion gap did not differ across grades.

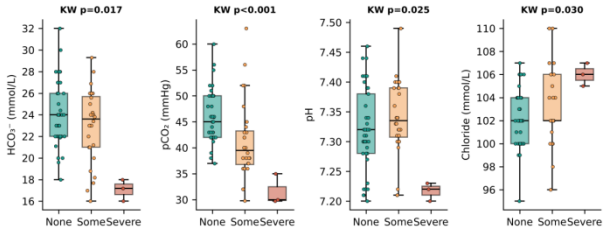


Figure 08: Distribution of bicarbonate, pCO_2, pH and chloride by dehydration grade (box: median and IQR; points: individual children; KW = Kruskal–Wallis).

Clinical correlates of acidosis and dyselectrolytemia

In unadjusted analyses (Table 7, Figure 9), metabolic acidosis was more frequent in children with ≥8 stools/day (7/20 vs 5/52; OR 4.80, 95% CI 1.37–16.84; p=0.016) and numerically more frequent with vomiting and with some/severe dehydration (OR ≈3–4, p=0.06–0.11); it was less frequent in infants ≤12 months (1/21 vs 11/51; OR 0.26). Dyselectrolytemia was associated with a respiratory rate ≥30/min (17/29 vs 10/43; OR 4.47, 95% CI 1.64–12.19; p=0.003), a finding that, given multiple comparisons, requires replication. In a multivariable model for low bicarbonate, only the direction of association for dehydration (adjusted OR 2.68, 95% CI 0.82–8.72, p=0.102) and respiratory rate (aOR 1.10 per breath/min, 0.98–1.23, p=0.101) was suggestive; age ≤12 months (aOR 0.74) and stool frequency (aOR 0.99) were not independently associated (Table 8; Figure 10 shows the full correlation structure). Hyponatraemia was not associated with dehydration (OR 0.87, p=1.0), age, stool frequency or vomiting.

Table 07: Unadjusted associations of clinical features with acid–base and electrolyte outcomes

Exposure	Events if exposed	Events if unexposed	OR (95% CI)	Fisher p
Outcome: low bicarbonate (HCO_3^- <22 mmol/L)				
Some/severe dehydration	12/31	7/41	2.95 (1.02–8.53)	0.058
Age ≤12 months	5/21	14/51	0.86 (0.28–2.69)	1.000
Stool frequency ≥8/day	7/20	12/52	1.80 (0.60–5.38)	0.374
Vomiting present	12/41	7/31	1.38 (0.48–3.96)	0.596
RR ≥30/min	11/29	8/43	2.60 (0.91–7.42)	0.101
Fever present	4/13	15/59	1.36 (0.39–4.80)	0.734
Outcome: metabolic acidosis				
Some/severe dehydration	8/31	4/41	3.01 (0.86–10.56)	0.109
Age ≤12 months	1/21	11/51	0.26 (0.04–1.53)	0.161
Stool frequency ≥8/day	7/20	5/52	4.80 (1.37–16.84)	0.016
Vomiting present	10/41	2/31	3.93 (0.91–17.07)	0.057
RR ≥30/min	7/29	5/43	2.33 (0.69–7.88)	0.204
Fever present	2/13	10/59	1.02 (0.22–4.69)	1.000

Outcome: any dyselectrolytemia				
Some/severe dehydration	13/31	14/41	1.38 (0.54–3.57)	0.624
Age ≤12 months	6/21	21/51	0.59 (0.20–1.73)	0.424
Stool frequency ≥8/day	9/20	18/52	1.54 (0.55–4.31)	0.429
Vomiting present	16/41	11/31	1.15 (0.45–2.99)	0.810
RR ≥30/min	17/29	10/43	4.47 (1.64–12.19)	0.003
Fever present	8/13	19/59	3.21 (0.97–10.67)	0.062

OR with Haldane–Anscombe correction; exploratory, unadjusted for multiplicity.

Table 08: Multivariable logistic regression for low bicarbonate (HCO_3^- <22 mmol/L; 19 events)

Predictor	Adjusted OR	95% CI	p
Some/severe dehydration	2.68	0.82–8.72	0.102
Age ≤12 mo	0.74	0.21–2.65	0.645
Stool freq (per +1/day)	0.99	0.86–1.14	0.867
RR (per +1/min)	1.10	0.98–1.23	0.101

Model limited to four predictors (≈5 events per variable); estimates are unstable and presented as exploratory.

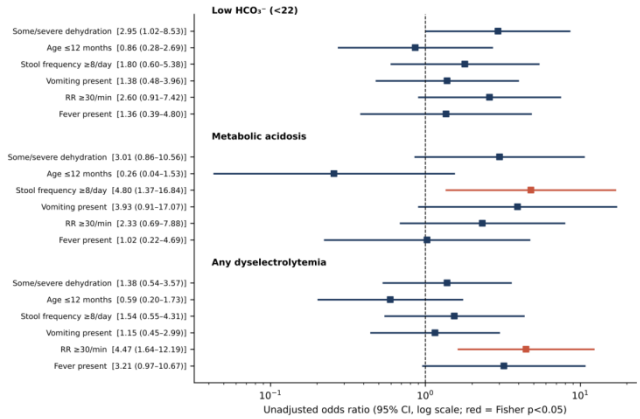


Figure 09: Forest plot of unadjusted odds ratios (95% CI, logarithmic scale) for low bicarbonate, metabolic acidosis and any dyselectrolytemia. Red = Fisher p<0.05.

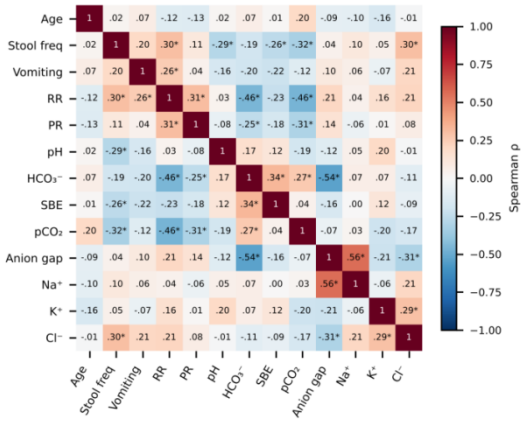


Figure 10: Spearman correlation matrix of clinical, acid–base and electrolyte variables (*p<0.05). Note the expected arithmetic coupling of the anion gap with sodium and bicarbonate, and the inverse relation of bicarbonate with respiratory rate (ρ = −0.46).

Change in sodium and potassium after rehydration

Mean sodium changed little between admission and the repeat sample (137.3 to 136.7 mmol/L; Wilcoxon p=0.346): 12 of 15 hyponatraemic and all four hypernatraemic children were normonatraemic on repeat. By contrast, mean potassium fell from 4.37 to 4.14 mmol/L (mean change −0.22 mmol/L; p=0.015); all five hyperkalaemic children normalised, but 15 children (20.8%) were hypokalaemic (K^+ <3.5 mmol/L) on repeat, compared with 2 (2.8%) at admission (Figure 11).

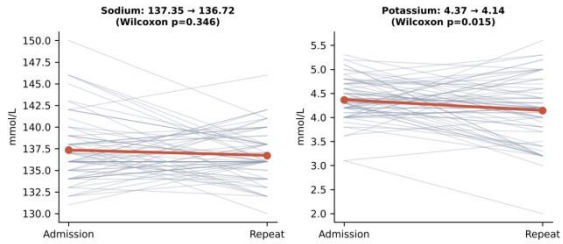


Figure 11: Paired admission and repeat serum sodium (left) and potassium (right). Grey lines: individual children; red: group mean.

Admission clinical variables identified dehydration only modestly: ROC AUCs were 0.77 for low pCO_2, 0.71 for stool frequency, 0.64 for chloride, 0.63 for respiratory rate and 0.63 for pulse rate (Figure 12), underscoring that no single bedside or laboratory marker substitutes for the full WHO assessment.

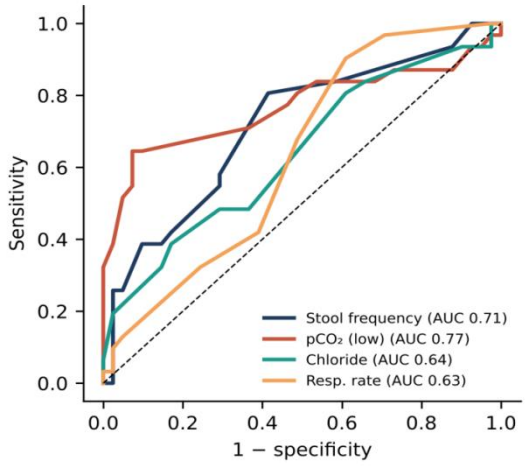


Figure 12: ROC curves of selected admission variables for discriminating some/severe dehydration (n=31) from no dehydration (n=41).

DISCUSSION

Principal findings

In this prospective cohort of 72 hospitalised under-five children with AGE, just over one-third had an electrolyte abnormality -chiefly hyponatraemia-and roughly one in six had metabolic acidosis. All three severely dehydrated children were acidotic with appropriate respiratory compensation, whereas sodium, potassium and chloride abnormalities were unrelated to dehydration grade. Potassium, but not sodium, fell significantly after rehydration, with one in five children hypokalaemic on the repeat sample.

Electrolyte derangements

Hyponatraemia is consistently reported as the commonest dysnatraemia in hospitalised children with gastroenteritis, reflecting faecal sodium loss with replacement by hypotonic fluids and non-osmotic ADH release [15,16,20,27]; our prevalence of 20.8% accords with a range that varies widely between series according to case-mix, definition and prior fluid intake. Hypernatraemia (5.6%) was uncommon, as in other contemporary hospital series where ORS and breastfeeding are widely used [15,27]. Importantly, that sodium abnormalities were independent of dehydration grade supports the established view that serum sodium cannot be inferred from the degree of clinical dehydration and that isonatraemic, hyponatraemic and hypernatraemic dehydration may look alike at the bedside [21,22,27]. Because sodium was normal on repeat in nearly all children treated with isotonic fluids and low-osmolarity ORS, our data are compatible with the safety of this regimen and with guidance to avoid hypotonic maintenance fluids [8,18,19].

Admission hypokalaemia was rare (2.8%), lower than in many series, possibly because acidosis and haemoconcentration, which shift potassium out of cells, can mask total-body depletion [11,28]. The fall in potassium after rehydration-with 15 children hypokalaemic on repeat-is physiologically coherent: correction of acidosis and volume, insulin release with oral glucose, and continued stool losses unmask potassium depletion. This is clinically relevant because unrecognised hypokalaemia may cause ileus, muscle weakness and arrhythmia, and ORS contains only modest potassium. We recommend that potassium be rechecked after rehydration in children treated with IV fluids or with initial values at the low end of normal, although the optimum timing remains to be defined, as the interval to repeat sampling was not recorded here.

Acid–base disturbance

Metabolic acidosis in AGE arises principally from faecal bicarbonate loss, producing a hyperchloraemic, normal-anion-gap pattern, with a superimposed raised-gap component when hypoperfusion generates lactate or starvation ketosis [11,12]. This dichotomy was evident: 12 of 19 children with low bicarbonate had a normal anion gap and 7 a raised gap, and the three severely dehydrated children had gaps at the upper end of, or above, the reference range. Chloride rose with dehydration, as expected with bicarbonate-for-chloride exchange. The lower pCO_2 with increasing dehydration ($\rho = -0.49$) reflects the respiratory drive that mitigates acidaemia; tachypnoea in a dehydrated child should therefore prompt consideration of metabolic acidosis rather than primary respiratory disease [26,28].

Two features of the data warrant cautious interpretation. First, 68% of children had pH <7.35, and 22% had an apparent respiratory acidosis, which is unlikely to reflect true hypoventilation in mostly alert children. Venous or capillary sampling, which yields a lower pH and higher pCO_2 than arterial blood, crying during sampling and the thresholds applied probably explain this [25]; consistent with this, the proportion with pH <7.30 was 26.4% and the bicarbonate-based definition (low HCO_3^-, 26.4%; metabolic acidosis, 16.7%) is the more robust estimate, because bicarbonate is far less sensitive to sampling artefact. Second, the SBE was predominantly negative, indicating mild base deficit in most children (77.8%), which in the presence of normal bicarbonate probably represents the normal-to-mildly-reduced venous range plus the effect of loss of bicarbonate.

Dehydration grade and biochemical disturbance

Dehydration grade was associated with low pCO_2, higher stool frequency and higher chloride, and non-significantly with low bicarbonate, but not with abnormalities of sodium, potassium or chloride. This is consistent with the limited accuracy of clinical signs in predicting biochemical status [21,22] and with studies showing that routine electrolyte panels seldom change management in uncomplicated paediatric dehydration but are valuable when dehydration is severe or IV therapy is necessary [8,20]. Our observation that biochemical abnormalities occurred in 34% of children without clinical dehydration is a reminder that testing criteria should not rely on dehydration grade alone. Stool frequency of ≥8 per day, tachypnoea and sick appearance are pragmatic triggers that merit prospective validation (Figure 13).

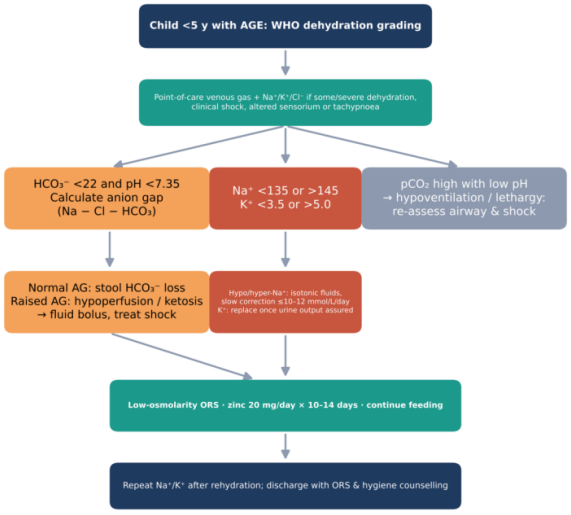


Figure 13: Proposed pragmatic pathway for acid–base and electrolyte assessment of children under five years with acute gastroenteritis, derived from the present findings and published guidance [7,8,12,18,20]. The pathway is a hypothesis for validation and not a substitute for local protocols.

Clinical and public-health implications

- **Targeted testing:** obtain blood gas and electrolytes in children with severe dehydration, shock, altered sensorium, persistent tachypnoea or high stool frequency; routine panels in well-hydrated children are low-yield.
- **Fluids:** low-osmolarity ORS remains first line [10,13,14]; where IV therapy is needed use isotonic fluids to reduce the risk of iatrogenic hyponatraemia [8,19], and limit the rate of sodium correction [17,18].
- **Potassium:** anticipate and recheck potassium after rehydration; replace once urine output is established.
- **Prevention:** zinc supplementation [29,30], correct ORS preparation and promotion of breastfeeding, sanitation and rotavirus immunisation address the upstream burden [5,6,9].

STRENGTHS

Strengths include prospective enrolment, a pre-specified protocol and sample-size rationale, standardised WHO dehydration grading, blood-gas and electrolyte assessment before rehydration, calculation of the anion gap and

respiratory compensation (rarely reported in Indian paediatric series), and a paired repeat sample enabling assessment of change after therapy.

LIMITATIONS

- Single-centre study with a modest sample (n=72), powered for prevalence rather than association; only three severely dehydrated children, so severe-stratum estimates are imprecise and sparse cells were not suited to odds-ratio modelling.
- Blood-gas sample type (venous/capillary), the timing of the repeat Na^+/K^+ sample, urine output, renal function (urea/creatinine), lactate, ketones and pathogen testing were not recorded; magnesium and calcium were not analysed. Interpretation of pH and pCO_2 is therefore approximate.
- Pre-hospital ORS use and its preparation were recorded, but their effects on electrolytes could not be analysed formally; severity grading was clinical and not blinded to laboratory results.
- Exploratory analyses (multiple comparisons, no correction) may yield chance findings; a military referral hospital population may limit generalisability. Clinical outcomes such as length of stay, complications and mortality were not analysed.

CONCLUSION

In hospitalised under-five children with acute gastroenteritis, 37.5% had dyselectrolytemia (predominantly hyponatraemia) and 16.7% had metabolic acidosis, with a compensated metabolic acidosis universal in severe dehydration. Clinical dehydration grade did not predict sodium, potassium or chloride abnormalities, and potassium fell significantly after rehydration. These findings support selective blood-gas and electrolyte testing based on severity, tachypnoea and stool frequency, use of isotonic fluids and low-osmolarity ORS, and potassium surveillance after rehydration. Larger multicentre studies with arterial or standardised venous sampling, timed repeat measurements and clinical outcomes are needed.

Abbreviations: AG, anion gap; AGE, acute gastroenteritis; CI, confidence interval; HCO_3^-, bicarbonate; IQR, interquartile range; IV, intravenous; OR, odds ratio; ORS, oral rehydration solution; pCO_2, partial pressure of carbon dioxide; ROC, receiver operating characteristic; RR, respiratory rate; SBE, standard base excess; STROBE, Strengthening the Reporting of Observational Studies in Epidemiology; WHO, World Health Organization.

ETHICS APPROVAL AND CONSENT

Approved by the Institutional Ethics Committee, Command Hospital (Central Command), Lucknow (certificate number IEC/37/2022); written informed consent was obtained from the parent or guardian of every participant.

FUNDING

Self-funded; no external funding was received.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY

De-identified data are available from the corresponding author on reasonable request.

AUTHOR CONTRIBUTIONS (CREDIT)

Sandeep Bharat Gadade (SBG): conceptualisation, investigation, data collection, writing – original draft. Manish Kumar (MK): data curation, methodology review. Atul Gopal Agrawal (AGA): formal statistical analysis, writing – review and editing, project administration; corresponding author. Vandana Negi (VN): supervision, validation, resources. Umesh Kumar Singh (UKS): clinical supervision, review. Ankita Agrawal (AA): writing – review and editing. All authors read and approved the final manuscript and meet the ICMJE criteria for authorship.

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