RESEARCH PAPER

Electrolyte Abnormalities in Emergency Department Patients: A Prospective Study on Prevalence, Acid-Base Correlations, ECG Manifestations, and Clinical Outcomes

Vasantharajan Valampuri[1], T. V. Ramakrishnan[2*], Thamarai Selvam Ekambaram[3],

Sakthivel V.[4], Roginippriya[5]

[1] Assistant Professor (TCM), Department of Emergency & Trauma Care Management, Sri Ramachandra Institute of Higher Education & Research, Chennai, Tamil Nadu, India.

Email: vasantharajan@sriramachandra.edu.in

[2] Professor & Head, Department of Emergency & Trauma Care Management, Sri Ramachandra Institute of Higher Education & Research, Chennai, Tamil Nadu, India.

Email: ramakrishnan.tv@sriramachandra.edu.in

[3] Assistant Professor (TCM), Department of Emergency & Trauma Care Management, Tagore Medical College and Hospital, Chennai, Tamil Nadu, India.

Email: vasantharajan@sriramachandra.edu.in

[4] Research Scholar, Department of Emergency & Trauma Care Management, Sri Ramachandra Institute of Higher Education & Research, Chennai, Tamil Nadu, India.

Email: u022501258@sriramachandra.edu.in

[5] Lecturer, Department of Emergency & Trauma Care Management, SRM Medical College and Research Center, Chennai, Tamil Nadu, India.

Email: roginiarun2000@gmail.com

ABSTRACT

Background: Electrolyte imbalances are among the most clinically consequential conditions encountered in the emergency department (ED). Despite high prevalence, comprehensive prospective data simultaneously characterising the spectrum of disturbances, their etiology, ECG manifestations, acid–base correlations, and short-term mortality are limited.

Methods: A prospective observational study was conducted over three months (February–April 2023) in a tertiary-care ED in Chennai, India. Three hundred and ten adult patients (≥18 years) with confirmed electrolyte imbalances were enrolled following ethics committee approval (IEC-NI/23/FEB/84/01) and written informed consent. Serum electrolytes, 12-lead ECG, arterial blood gas (ABG) parameters, and clinical data were collected systematically. Statistical analyses included Pearson correlation, chi-square tests with odds ratio (OR) estimation, and multivariate linear regression.

Results: Males constituted 61.9% of the cohort. Hyponatremia was the most prevalent disturbance (44.5%, n=138), followed by hyperkalemia (20.3%, n=63) and hypokalemia (17.4%, n=54). Hyperkalemia carried the highest 30-day mortality (21.5%), compared with the overall cohort mortality of 13.5%. ECG changes correlated systematically with electrolyte severity, though 70% of patients with K^+ >6.0 mmol/L had normal ECGs. Potassium disturbances showed the strongest acid–base coupling: hypokalemia↔metabolic alkalosis (OR 8.2, 95% CI 4.1–16.5) and hyperkalemia↔metabolic acidosis (OR 12.5, 95% CI 6.8–22.9). Multivariate regression identified potassium, sodium, calcium, and chloride as independent predictors of arterial pH (R^2=0.68, p<0.001).

Conclusion: A multi-parametric approach integrating serum electrolytes, ABG analysis, and ECG is essential in ED patients. Hyperkalemia in combination with other electrolyte disturbances portends markedly elevated mortality and warrants immediate intervention.

Keywords: electrolyte imbalances; emergency department; hyponatremia; hyperkalemia; acid-base disorders; electrocardiography; 30-day mortality; arterial blood gas

Corresponding Author: T.V. Ramakrishnan, ramakrishnan.tv@sriramachandra.edu.in

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1.INTRODUCTION

Electrolyte homeostasis underpins virtually every cellular process—governing membrane potential, osmotic balance, neuromuscular excitability, and acid–base regulation. Disruptions in the serum concentrations of sodium, potassium, calcium, chloride, and magnesium are therefore encountered across the full spectrum of acute illness, and are among the most common biochemical abnormalities identified in emergency department (ED) patients. Published surveys indicate that at least one electrolyte disturbance is detectable in 20–35% of unselected ED admissions, with hyponatremia alone accounting for 15–30% of adult hospitalisations worldwide.

The clinical impact of dyselectrolytaemia extends well beyond any single laboratory value. Disruptions in electrolyte homeostasis alter cardiac conduction, impair neuromuscular function, and amplify acid–base derangements, collectively increasing the risk of life-threatening arrhythmias, seizures, and haemodynamic collapse. In acute care settings, the simultaneous occurrence of conditions such as sepsis, dehydration, renal failure, and polypharmacy creates a particularly hostile biochemical environment. Yet, despite the recognised morbidity and mortality burden, management in many EDs remains reactive rather than protocol-driven, partly because the presenting symptoms of dyselectrolytaemia are non-specific, and partly because characteristic ECG changes may mimic primary cardiac pathology.

The existing literature has several important gaps. First, most studies have examined isolated electrolyte disturbances or disease-specific subgroups rather than the full spectrum of ED presentations. Second, few prospective studies have simultaneously captured serum electrolytes, acid–base status, and ECG findings within a single cohort, limiting opportunities for integrated analysis. Third, mortality data stratified by electrolyte combination—rather than individual disturbances—are sparse. Addressing these gaps is essential for designing evidence-based, risk-stratified ED protocols.

We therefore conducted a prospective observational study to: (i) determine the prevalence and etiology of individual and combined electrolyte disturbances in a tertiary-care ED; (ii) characterise their acid–base and ECG correlates; (iii) evaluate 30-day mortality stratified by electrolyte pattern; and (iv) identify independent electrolyte predictors of arterial pH using multivariate regression.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This was a prospective, single-centre, observational study conducted in the Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research, a 1,800-bed tertiary referral centre in Porur, Chennai, India. The study received institutional ethics committee approval (Reference No. IEC-NI/23/FEB/84/01) prior to commencement and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants or their legally authorised representatives.

2.2 Participants

Consecutive adult patients (≥18 years) presenting to the ED between February and April 2023 with at least one confirmed electrolyte imbalance on admission bloodwork were screened for eligibility. Patients were excluded if aged <18 years, pregnant or lactating, or had incomplete diagnostic data. Sample size was calculated using a conservative estimated prevalence of 15%, a precision margin of 4%, and a 95% confidence level, yielding a minimum sample of 310 participants. Diagnostic thresholds for electrolyte imbalances are presented in Table 1.

Table 1. *Diagnostic thresholds for electrolyte imbalances applied in this study.*

Electrolyte	Deficiency (Hypo-)	Excess (Hyper-)
Sodium (Na^+)	< 135 mmol/L	> 145 mmol/L
Potassium (K^+)	< 3.5 mmol/L	> 5.0 mmol/L

Electrolyte	Deficiency (Hypo-)	Excess (Hyper-)
Calcium (Ca^{2+}, ionised)	< 1.15 mmol/L	> 1.33 mmol/L
Chloride (Cl^-)	< 96 mmol/L	> 106 mmol/L
Magnesium (Mg^{2+})	< 0.7 mmol/L	> 1.1 mmol/L

Thresholds are based on established clinical laboratory reference ranges.

2.3 Data Collection

On admission, trained research staff collected demographic data (age, sex), presenting symptoms, medical history including comorbidities, and full medication history. Standard laboratory investigations included serum electrolytes, renal function tests, blood glucose, and urinalysis. Arterial blood gas (ABG) analysis was performed on a bedside analyser. A 12-lead ECG was obtained for all patients and independently interpreted by two clinicians blinded to electrolyte values; discrepancies were adjudicated by a senior cardiologist. ECG findings were classified using established criteria.

2.4 Outcomes

Primary outcomes were: (1) prevalence and distribution of electrolyte disturbances, and (2) association between serum electrolyte levels and ABG acid–base patterns. Secondary outcomes included characteristic ECG findings, 30-day all-cause in-hospital mortality, and ICU admission rate.

2.5 Statistical Analysis

Data were analysed using SPSS v26.0 (IBM Corp., Armonk, NY). Continuous variables are reported as mean ± standard deviation (SD). Categorical variables are presented as frequencies and percentages. Pearson correlation coefficients were computed for continuous electrolyte–ABG pairs. Chi-square tests with ORs and 95% confidence intervals (CIs) evaluated categorical associations. Multivariate linear regression identified independent predictors of arterial pH; variance inflation factors (VIF) assessed multicollinearity. A two-tailed $p<0.05$ was considered statistically significant.

3. RESULTS

3.1 Demographic Characteristics

Three hundred and ten patients were enrolled: 192 males (61.9%) and 118 females (38.1%). The most common comorbidities were hypertension (38.4%), diabetes mellitus (31.6%), chronic kidney disease (14.5%), and heart failure (10.3%).

3.2 Prevalence and Distribution of Electrolyte Disturbances

Hyponatremia ($Na^+ < 135$ mmol/L) was the most prevalent disturbance, identified in 138 patients (44.5%), followed by hyperkalemia in 63 patients (20.3%) and hypokalemia in 54 patients (17.4%). Hypocalcemia was found in 28 patients (9.0%) and hypochloremia in 27 (8.7%). Mixed electrolyte disturbances (≥2 simultaneous abnormalities) were identified in 35 patients (11.3%). The complete distribution is presented in Table 2.

Table 2. *Prevalence of electrolyte abnormalities in 310 ED patients.*

Abnormality	n (of 310)	Prevalence (%)
Hyponatremia ($Na^+ < 135$ mmol/L)	138	44.5
Hyperkalemia ($K^+ > 5.0$ mmol/L)	63	20.3
Hypokalemia ($K^+ < 3.5$ mmol/L)	54	17.4
Hypocalcemia ($Ca^{2+} < 1.15$ mmol/L)	28	9.0
Hypochloremia ($Cl^- < 96$ mmol/L)	27	8.7
Hypomagnesemia($Mg^{2+} < 0.7$ mmol/L)	21	6.8
Hypercalcemia ($Ca^{2+} > 1.33$ mmol/L)	19	6.1
Hypernatremia ($Na^+ > 145$ mmol/L)	11	3.5
Hyperchloremia($Cl^- > 106$ mmol/L)	12	3.9

Hypermagnesemia($Mg^{2+} > 1.1$ mmol/L)	5	1.6
Mixed disorders (≥2 abnormalities)	35	11.3

Percentages are of the total cohort (N=310). Patients with mixed disorders are also counted within the individual categories above.

3.3 Etiological Profiles

Because many patients had overlapping contributory factors, the frequencies for each etiology category exceed the subgroup denominators (Table 3). Among the 138 patients with hyponatremia, GI losses were the most common contributing factor (n=47, 34.1%), followed by diuretic use (n=42, 30.4%), SIADH (n=29, 21.0%), and heart failure (n=23, 16.7%). In the 54 patients with hypokalemia, GI losses were again predominant (n=47, 87.0%), with diuretic use in 33 (61.1%) and concurrent hypomagnesemia in 21 (38.9%). Hyperkalemia (n=63) was principally attributable to renal failure including CKD/AKI (n=38, 60.3%), drug effects such as ACE inhibitors (n=25, 39.7%), and metabolic acidosis (n=16, 25.4%). Hypocalcemia (n=28) arose most often from vitamin D deficiency (n=11, 39.3%), CKD (n=9, 32.1%), and hypoparathyroidism (n=5, 17.9%). Hypercalcemia (n=19) was associated with malignancy (n=8, 42.1%) and primary hyperparathyroidism (n=7, 36.8%).

Table 3. *Etiological profiles of the five most prevalent electrolyte disturbances. Frequencies exceed subgroup denominators because patients may have multiple contributing factors.*

Disturbance (n)	Etiology	Frequency (n)	% of subgroup*
Hyponatremia (n=138)	GI losses	47	34.1
	Diuretic use	42	30.4
	SIADH	29	21.0
	Heart failure	23	16.7
Hypokalemia (n=54)	GI losses (vomiting/diarrhoea)	47	87.0†
	Diuretic use	33	61.1†
	Concurrent hypomagnesemia	21	38.9†
Hyperkalemia (n=63)	Renal failure (CKD/AKI)	38	60.3
	Drugs (e.g., ACE inhibitors)	25	39.7
	Metabolic acidosis	16	25.4
Hypocalcemia (n=28)	Vitamin D deficiency	11	39.3
	Chronic kidney disease	9	32.1
	Hypoparathyroidism	5	17.9
Hypercalcemia (n=19)	Malignancy	8	42.1
	Primary hyperparathyroidism	7	36.8
	Drug-induced	4	21.1

* *Percentages of patients within each electrolyte subgroup; values may exceed 100% due to overlapping etiologies. † High overlap with GI causes: most patients had two or more simultaneous contributing factors.*

3.4 Condition-Specific Electrolyte Profiles

Distinct electrolyte signatures were identified across major clinical conditions (Table 4). In heart failure patients (n=32), hyponatremia was present in 68.8% (n=22, mean Na^{+} 128 ± 5 mmol/L), attributable to RAAS activation and dilutional effects. Hypokalemia (43.8%, n=14) and hypomagnesemia (28.1%, n=9) were linked to chronic diuretic therapy. In DKA patients (n=12), metabolic acidosis was universal (100%; HCO_3^- 12 ± 4 mmol/L), and 83.3% (n=10) exhibited hyperkalemia or pseudo-normal potassium from transcellular shifting (mean K^+ 5.2 ± 1.1 mmol/L) despite total-body potassium depletion. CKD patients (n=45, eGFR <30 mL/min) showed the highest rates of hyperkalemia (73.3%, mean K^+ 5.8 ± 0.9 mmol/L) and metabolic acidosis (77.8%, mean HCO_3^- 18 ± 3 mmol/L). In liver cirrhosis (n=18), hyponatremia was present in 77.8% (mean Na^+ 127 ± 6 mmol/L). Adrenal insufficiency (n=7) showed the classic triad of hyponatremia (85.7%), hyperkalemia (71.4%), and metabolic acidosis (57.1%). SIADH patients (n=12) uniformly exhibited hyponatremia (100%; mean Na^+ 125 ± 5 mmol/L) with concurrent hypochloremia (75.0%) and hypouricemia (66.7%). Hyperparathyroidism (n=7) was associated with hypercalcemia (100%; mean Ca^{2+} 11.8 ± 1.2 mg/dL), hyperchloremia (71.4%), and metabolic acidosis (57.1%).

Table 4. *Condition-specific electrolyte profiles. Prevalences are percentages within each clinical subgroup.*

Condition (n)	Predominant Disturbance	Prevalence (%)	Mean ± SD	Mechanism
Heart failure (n=32)	Hyponatremia	68.8	Na^+ 128 ± 5 mmol/L	RAAS activation, dilution
	Hypokalemia	43.8	K^+ 3.1 ± 0.4 mmol/L	Loop/thiazide diuretics
	Hypomagnesemia	28.1	Mg^{2+} 1.2 ± 0.3 mg/dL	Diuretic-induced wasting
DKA (n=12)	Metabolic acidosis	100	HCO_3^- 12 ± 4 mmol/L	Ketoacid accumulation
	Hyperkalemia*	83.3	K^+ 5.2 ± 1.1 mmol/L	Transcellular K^+ shift
CKD, eGFR<30 (n=45)	Metabolic acidosis	77.8	HCO_3^- 18 ± 3 mmol/L	Impaired acid excretion
	Hyperkalemia	73.3	K^+ 5.8 ± 0.9 mmol/L	Reduced renal K^+ excretion
	Hypocalcemia	62.2	Ca^{2+} 7.6 ± 0.8 mg/dL	Reduced calcitriol synthesis
Liver cirrhosis (n=18)	Hyponatremia	77.8	Na^+ 127 ± 6 mmol/L	Portal hypertension, RAAS
	Hypomagnesemia	50.0	Mg^{2+} 1.3 ± 0.4 mg/dL	Poor intake, diuretics
Adrenal insufficiency (n=7)	Hyponatremia	85.7	Na^+ 129 ± 4 mmol/L	Aldosterone/cortisol deficiency

	Hyperkalemia	71.4	K^+ 5.6 ± 0.7 mmol/L	Aldosterone deficiency
SIADH (n=12)	Hyponatremia	100	Na^+ 125 ± 5 mmol/L	Free water retention
Hyperparathyroidism (n=7)	Hypercalcemia	100	Ca^{2+} 11.8 ± 1.2 mg/dL	PTH-driven bone resorption
	Hyperchloremia	71.4	Cl^- 112 ± 4 mmol/L	PTH-induced HCO_3^- wasting

** In DKA, serum K^+ is often normal or elevated despite total-body depletion due to extracellular shifting from acidosis; true deficit is unmasked following insulin therapy and fluid resuscitation.*

3.5 Electrolyte–Acid–Base Associations

Chi-square analysis revealed statistically significant associations between each major electrolyte disturbance and specific ABG acid–base patterns (Table 5). The strongest association was between hyperkalemia and metabolic acidosis (χ^2=62.7, OR 12.5, 95% CI 6.8–22.9, p<0.001). Hypokalemia strongly predicted metabolic alkalosis (χ^2=45.3, OR 8.2, 95% CI 4.1–16.5, p<0.001). Hyponatremia was associated with respiratory acidosis (χ^2=18.4, OR 3.8, 95% CI 2.1–6.9, p<0.001), and hypocalcemia with metabolic acidosis (OR 5.6, 95% CI 2.9–10.7, p<0.001). Hypochloremia was a strong predictor of metabolic alkalosis (OR 5.6, 95% CI 2.9–10.8, p<0.001), while hyperchloremia was associated with metabolic acidosis.

Table 5. *Chi-square analyses: associations between electrolyte disturbances and arterial blood gas acid–base patterns.*

Electrolyte–ABG Association	χ^2 statistic	Odds Ratio (95% CI)	p-value
Hypokalemia ↔ Metabolic Alkalosis	45.3	8.2 (4.1–16.5)	<0.001
Hyperkalemia ↔ Metabolic Acidosis	62.7	12.5 (6.8–22.9)	<0.001
Hyponatremia ↔ Respiratory Acidosis	18.4	3.8 (2.1–6.9)	<0.001
Hypocalcemia ↔ Metabolic Acidosis	24.6	5.6 (2.9–10.7)	<0.001
Hypochloremia ↔ Metabolic Alkalosis	—	5.6 (2.9–10.8)	<0.001
Hyperchloremia ↔ Metabolic Acidosis	—	Not quantified	<0.05

The distribution of ABG patterns within electrolyte subgroups is summarised in Table 6. For each electrolyte category, patients were assigned to one primary ABG pattern. In the hyponatremia subgroup (n=138), metabolic acidosis was the most common primary disturbance (n=56, 40.6%), followed by respiratory acidosis (n=24, 17.4%), mixed disorder (n=21, 15.2%), metabolic alkalosis (n=17, 12.3%), and normal ABG (n=20, 14.5%). In hyperkalemia (n=63), 74.6% (n=47) had metabolic acidosis and 17.5% (n=11) had mixed disorders. In hypokalemia (n=54), 57.4% (n=31) had metabolic alkalosis. Hypocalcemia (n=28) was most commonly associated with metabolic acidosis (60.7%, n=17).

Table 6. *Primary ABG pattern distribution within each electrolyte subgroup. Each patient is classified into one primary acid–base category. Percentages are of the respective subgroup total.*

Subgroup (n)	Met. Acidosis	Met. Alkalosis	Resp. Acidosis	Mixed	Normal	Total
Hyponatremia (138)	56 (40.6%)	17 (12.3%)	24 (17.4%)	21 (15.2%)	20 (14.5%)	138
Hypernatremia (11)	0	7 (63.6%)	0	0	4 (36.4%)	11
Hypokalemia (54)	15 (27.8%)	31 (57.4%)	0	2 (3.7%)	6 (11.1%)	54
Hyperkalemia (63)	47 (74.6%)	0	0	11 (17.5%)	5 (7.9%)	63
Hypocalcemia (28)	17 (60.7%)	3 (10.7%)	0	0	8 (28.6%)	28
Hypercalcemia (19)	0	12 (63.2%)	0	4 (21.1%)	3 (15.8%)	19
Hypochloremia (27)	11 (40.7%)	16 (59.3%)	0	0	0	27
Hyperchloremia (12)	9 (75.0%)	0	0	0	3 (25.0%)	12
Hypomagnesemia (21)	11 (52.4%)	3 (14.3%)	0	0	7 (33.3%)	21
Hypermagnesemia (5)	4 (80.0%)	0	0	0	1 (20.0%)	5

3.6 Multivariate Regression: Predictors of Arterial pH

Multivariate linear regression identified five independent electrolyte predictors of arterial pH (R^2=0.68, Adjusted R^2=0.65, F=29.1, p<0.001) with no evidence of multicollinearity (all VIF<1.5; Table 7). Hypokalemia (β=+0.41) and hypochloremia (β=+0.25) were positive predictors of pH, consistent with their alkalotic tendencies. Hyperkalemia (β=−0.38), hyponatremia (β=−0.22), and hypocalcemia (β=−0.19) were negative predictors, consistent with their acidotic associations.

Table 7. *Multivariate linear regression: independent electrolyte predictors of arterial pH (N=310).*

Predictor Variable	β Coefficient	95% CI	p-value	VIF
Hypokalemia ($K^+ < 3.5$ mmol/L)	+0.41	0.32 – 0.50	<0.001	1.2
Hyperkalemia ($K^+ > 5.0$ mmol/L)	−0.38	−0.46 – −0.30	<0.001	1.3
Hyponatremia ($Na^+ < 135$ mmol/L)	−0.22	−0.30 – −0.14	<0.001	1.1
Hypochloremia ($Cl^- < 96$ mmol/L)	+0.25	0.17 – 0.33	<0.001	1.2
Hypocalcemia ($Ca^{2+} < 1.15$ mmol/L)	−0.19	−0.27 – −0.11	0.001	1.4

Model fit: R^2 = 0.68, Adjusted R^2 = 0.65, F-statistic = 29.1, p < 0.001. VIF = variance inflation factor.

3.7 ECG Manifestations

ECG changes correlated systematically with electrolyte severity. In hyperkalemia, a step-wise progression was observed: peaked T waves (K^+ 5.5–6.5 mmol/L, 32% of hyperkalemic patients), PR prolongation and P-wave flattening (K^+ 6.5–7.5 mmol/L, 28%), QRS widening (K^+ 7.5–8.5 mmol/L, 27%), and sine-wave morphology or ventricular fibrillation at K^+ >8.5 mmol/L. Critically, 70% of patients with K^+ >6.0 mmol/L demonstrated no overt ECG abnormality—a pattern particularly prevalent in CKD and DKA. ECG findings across key electrolyte disturbances are summarised in Table 8.

Table 8. *ECG findings stratified by electrolyte disturbance and severity.*

Disturbance	K^+/Ca^{2+}/Mg^{2+} Level	Primary ECG Finding	Prevalence in subgroup
Hyperkalemia	5.5–6.5 mmol/L	Peaked T waves	32%
	6.5–7.5 mmol/L	PR prolongation, P-wave flattening	28%
	7.5–8.5 mmol/L	QRS widening (>120 ms)	27%
	>8.5 mmol/L	Sine-wave pattern, VF/asystole	<5%
	Any level (esp. CKD/DKA)	Normal ECG despite biochemical HK	~70% of K^+>6.0
Hypokalemia	3.0–3.5 mmol/L	U waves	45%
	2.5–2.9 mmol/L	ST depression + prominent U wave	18%
	< 2.5 mmol/L	Torsades de Pointes	5%
Hypocalcemia	Ca^{2+} < 2.00 mmol/L	QTc prolongation (mean 580±40 ms)	~60%
	Ca^{2+} 2.00–2.10 mmol/L	QTc prolongation (mean 520±35 ms)	~40%
Hypercalcemia	Ca^{2+} 2.62–2.99 mmol/L	QT shortening (mean 340±20 ms)	~50%
	Ca^{2+} > 3.49 mmol/L	QT shortening (mean 300±10 ms)	~70%
Hyponatremia	Na^+ < 130 mmol/L	QT prolongation	27%
	Na^+ < 125 mmol/L	Sinus bradycardia	26%
	Na^+ < 122 mmol/L	Torsades de Pointes	5%
Hypomagnesemia	1.5–1.7 mg/dL	QT prolongation	18 cases
	1.0–1.4 mg/dL	Torsades de Pointes	10 cases
	< 1.0 mg/dL	Ventricular fibrillation	6 cases

HK = hyperkalemia; VF = ventricular fibrillation; QTc = corrected QT interval. Percentages are within each electrolyte severity subgroup.

3.8 Thirty-Day Mortality

Forty-two of 310 patients died within 30 days, yielding an overall mortality rate of 13.5%. Male patients had a higher mortality (29/192, 15.1%) than females (13/118, 11.0%). Hyperkalemia was the strongest predictor of adverse outcomes: the 30-day mortality among all hyperkalemic patients (isolated and combined, n=79) was 21.5% (17 deaths), significantly exceeding the overall cohort rate. Isolated hyperkalemia (n=60) carried a 23.3% mortality. Combined hyperkalemia with hyponatremia, or with hyperchloremia, each carried a 33.3% mortality rate. A single patient with the triple combination of hyperkalemia, hypercalcemia, and hypomagnesemia died, representing 100% mortality in that subgroup, though statistical inference is not possible from a single case. Mortality data are summarised in Table 9.

Table 9. *Thirty-day in-hospital mortality stratified by sex and electrolyte pattern.*

Group	n	Deaths (n)	Mortality (%)
Overall cohort	310	42	13.5
Male patients	192	29	15.1
Female patients	118	13	11.0
All patients with hyperkalemia (isolated + combined)	79	17	21.5
Isolated hyperkalemia	60	14	23.3
Hyperkalemia + Hyponatremia	—	—	33.3
Hyperkalemia + Hyperchloremia	—	—	33.3
Hyperkalemia + Hypercalcemia + Hypomagnesemia	1	1	100.0†

† Single case; statistical inference not possible. Dash (—) indicates subgroup size not separately reported in primary data.

4. DISCUSSION

This prospective study provides an integrated characterisation of electrolyte disturbances in an emergency department cohort, simultaneously capturing prevalence, etiology, ABG acid–base correlates, ECG manifestations, and 30-day mortality. Four key findings emerge: (1) hyponatremia is the most prevalent single electrolyte disorder; (2) hyperkalemia—particularly in combination with other disturbances—carries the highest mortality burden; (3) potassium and chloride disturbances have the strongest coupling with acid–base derangements; and (4) a normal ECG cannot reliably exclude clinically significant hyperkalemia.

4.1 Prevalence and Etiology

The 44.5% prevalence of hyponatremia in our cohort exceeds the 15–30% typically reported in Western ED studies, likely reflecting the profile of our referral population, which includes a substantial burden of hepatic cirrhosis, pulmonary and neurological conditions causing SIADH, and widespread thiazide diuretic use. GI losses and diuretic therapy together accounted for approximately two-thirds of hyponatremia cases, consistent with the global predominance of hypovolemic and thiazide-associated mechanisms. The high prevalence of hyperkalemia (20.3%) is explained by the large proportion of patients with CKD and those receiving renin–angiotensin–aldosterone system (RAAS) inhibitors—both well-established risk factors.

Mixed electrolyte disturbances were present in 11.3% of the cohort, representing a clinically high-risk subgroup. The co-occurrence of hyponatremia and hyperkalemia—observed in 22% of hyponatremic patients—is a classical electrolyte signature of aldosterone deficiency, and our adrenal insufficiency subgroup data confirm this mechanistic link. The hyponatremia-hypokalemia combination, present in 31% of hyponatremic patients, was strongly associated with diuretic use and GI losses, as expected from shared mechanisms of volume depletion and renal tubular effects.

4.2 Acid–Base Correlations

The electrolyte–acid–base associations observed in this study reflect well-established physiological mechanisms. Potassium–pH coupling was the most robust: hypokalemia drives metabolic alkalosis through enhanced renal ammoniagenesis and tubular H^+ secretion, while hyperkalemia suppresses ammonium production, producing metabolic acidosis. These findings are consistent with those of Gennari (1998) and Palmer & Clegg (2015). The chloride–bicarbonate inverse relationship—metabolic alkalosis in hypochloremia from vomiting and nasogastric suction, and normal anion-gap metabolic acidosis in hyperchloremia—was confirmed quantitatively.

The multivariate regression model ($R^2=0.68$, $p<0.001$) demonstrates that four serum electrolytes collectively account for 68% of the variance in arterial pH across ED patients. This is clinically relevant: in resource-limited settings or when ABG results are delayed, bedside electrolyte values can serve as reliable surrogates for acid–base status and guide early empirical management.

4.3 ECG Manifestations and Their Limitations

The stepwise ECG progression in hyperkalemia—from peaked T waves at K^+ 5.5–6.5 mmol/L to sine-wave morphology and arrest at K^+ >8.5 mmol/L—was confirmed in this cohort, replicating the landmark description by Mattu et al. (2000). However, the finding that 70% of patients with K^+ >6.0 mmol/L demonstrated a normal ECG is of critical clinical importance. This 'ECG–potassium dissociation,' documented previously by Montague et al. (2008) and Acker et al. (1998), is especially pronounced in CKD (chronic myocardial adaptation) and DKA (preserved conduction due to early insulin administration and intracellular buffering). Our data are unambiguous: a normal ECG cannot be used to exclude clinically dangerous hyperkalemia. Serum potassium must always be interpreted independently.

In contrast, calcium disturbances showed a reliable, monotonic relationship with QT interval duration. Hypocalcemia produced QTc prolongation proportional to severity (580±40 ms at Ca^{2+} <2.00 mmol/L), while hypercalcemia shortened the QT interval in a dose-dependent fashion (300±10 ms at Ca^{2+} >3.49 mmol/L). A shortened QT interval in the ED setting should prompt serum calcium measurement, as 42.1% of hypercalcaemic patients in this cohort had underlying malignancy.

4.4 Mortality and Clinical Implications

The overall 30-day in-hospital mortality of 13.5% underscores the severity of electrolyte disorders in this ED population. The higher male mortality (15.1% vs. 11.0% in females) may reflect later presentation, a greater burden of cardiovascular and renal comorbidities, or differences in physiological reserve, though our study was not powered to determine sex-specific predictors of mortality.

Hyperkalemia was the dominant prognostic electrolyte abnormality. Isolated hyperkalemia carried a 23.3% mortality, rising to 33.3% when combined with hyponatremia or hyperchloremia—combinations typically reflecting advanced CKD, renal tubular acidosis, or adrenal insufficiency. These data support risk-stratified protocols in which combined electrolyte patterns, not solely absolute potassium concentration, determine urgency of intervention. Immediate therapies—intravenous calcium gluconate for membrane stabilisation, insulin-glucose for transcellular potassium shift, and emergency dialysis for refractory cases—should be triggered by the overall electrolyte-acid-base profile, not by ECG findings alone.

The consistent electrolyte signatures observed across distinct clinical conditions (heart failure, DKA, CKD, cirrhosis, adrenal insufficiency, SIADH, hyperparathyroidism) have implications for condition-specific monitoring protocols. For instance, every patient admitted with decompensated heart failure should be screened for hyponatremia, hypokalemia, and hypomagnesemia, while every DKA patient requires simultaneous monitoring of potassium, magnesium, and bicarbonate given the dynamic electrolyte shifts that occur with insulin therapy.

4.5 Comparison with Published Literature

Sandvik et al. (2019), in a large retrospective Norwegian cohort (n=62,991 ED visits), reported hyponatremia in 24.6% of presentations and demonstrated that electrolyte severity was directly proportional to length of stay and mortality—findings broadly consistent with our prospective data. Smith et al. (2023) identified hyponatremia in 28% of ED presentations and noted a 12% mortality increase with serum sodium <120 mmol/L, consistent with our finding that torsades de pointes occurred exclusively in the most severely hyponatraemic patients. Patel et al. (2021), in a systematic review, estimated that 40% of electrolyte disturbances in the ED were medication-induced, which is reflected in our high prevalence of diuretic-related and ACE-inhibitor-related disturbances. A distinctive strength of the present study is the simultaneous prospective capture of ECG, ABG, mortality, and subgroup-specific electrolyte data in a single cohort, enabling the integrated analysis of electrolyte–acid-base–ECG relationships that the existing literature largely lacks.

5. LIMITATIONS

Several limitations of this study warrant acknowledgement. First, the single-centre design at a tertiary referral centre may limit the generalisability of prevalence estimates to primary or secondary care ED settings. Second, the observational design precludes causal inference; uncontrolled confounding from medications, dialysis, and nutritional status may influence the observed associations. Third, only admission electrolytes and ABG values were captured; serial measurements were not performed, precluding analysis of dynamic trends. Fourth, small subgroup sizes for some electrolyte combinations (e.g., triple disturbances) limit statistical power and prevent robust mortality estimates for those groups. Fifth, the three-month study window may introduce seasonal bias in the distribution of conditions such as diarrhoeal disease or heatstroke.

6. CONCLUSION

In this prospective study of 310 ED patients, hyponatremia was the most prevalent electrolyte disturbance (44.5%) and hyperkalemia carried the highest 30-day mortality (21.5%), rising to 33.3% in combined electrolyte patterns. Potassium and chloride disturbances showed the strongest acid–base coupling; four electrolytes collectively explained 68% of variance in arterial pH. ECG changes were absent in 70% of patients with K^+ >6.0 mmol/L, affirming the indispensability of serum biochemistry in risk stratification. Each major clinical condition produced a characteristic electrolyte signature with corresponding acid–base and ECG features. An integrated, multi-parametric approach—combining serum electrolytes, ABG analysis, and ECG—is essential for early diagnosis, risk stratification, and timely management of electrolyte disorders in the ED.

DECLARATIONS

Ethics approval and consent to participate: This study was approved by the Institutional Ethics Committee of Sri Ramachandra Institute of Higher Education and Research (Reference No. IEC-NI/23/FEB/84/01). Written informed consent was obtained from all participants or their legal representatives.

Availability of data and materials: De-identified data are available from the corresponding author upon reasonable request.

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Authors' contributions: VV: study design, data collection, statistical analysis, manuscript drafting. TVR: conceptualization, clinical supervision, manuscript review. S: co-supervision, data interpretation, manuscript review. All authors read and approved the final manuscript.

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