

Research Article

# **ECG Patterns in Patients with Electrolyte Imbalances: Findings from a Cross-Sectional** Study

Muskan Sharma¹, Ayush Patel², Charishma Parla³, Shaurya Thakur⁴, Mah-e-Tab Rai⁵, Pranjal Dubey<sup>6</sup>

- <sup>1</sup>Student, BAU International University, Batumi, Georgia
- <sup>2</sup>Student, Davao Medical School Foundation, Davao City, Philippines
- <sup>3</sup>Student, Bukhara State Medical Institute named after Abu Ali Ibn Sino
- <sup>4</sup>Student, Punjab Institute of Medical Sciences, India
- <sup>5</sup>Student, Allama Iqbal Medical College, Lahore
- <sup>6</sup>Student, Eras Lucknow Medical College and Hospital

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### INFO

#### **Corresponding Author:**

Ayush Patel, Davao Medical School Foundation, Davao City, Philippines

#### E-mail Id:

drayushpatel15@gmail.com

Orcid Id:

https://orcid.org/0009-0004-5953-9358

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### A B S T R A C T

Background: Electrolyte disturbances are common in hospitalised medical patients and may cause characteristic electrocardiographic (ECG) abnormalities that signal risk for malignant arrhythmias. Evidence from realworld medicine wards in low and middleincome settings remains limited.

Objective: To determine the prevalence and patterns of ECG changes associated with electrolyte imbalances among adult patients and to identify clinical predictors of ECG abnormalities.

Main measures: Presence and type of electrolyte imbalance (sodium, potassium, calcium, magnesium; predefined cutoffs) and predefined ECG abnormalities (e.g., peaked T waves, U waves, QTc prolongation).

*Analysis:* Descriptive statistics;  $\chi^2$  tests; multivariable logistic regression for predictors of any ECG abnormality, adjusting for demographics, comorbidities, and relevant medications.

Results: Among 412 patients (median age 56 years; 48% female), 61% had ≥1 electrolyte imbalance. ECG abnormalities consistent with any electrolyte effect were present in 34% overall and in 49% of those with imbalances. Common patterns included QTc prolongation (18%), U waves/ST depression (hypokalaemiapattern, 12%), and peaked T waves/QRS widening (hyperkalaemiapattern, 8%). In adjusted models, hyperkalaemia (aOR 3.2; 95% CI 1.9-5.4), hypokalaemia (aOR 2.1; 1.3-3.5), and hypocalcemia (aOR 1.8; 1.1-2.9) were independently associated with ECG abnormalities. Polyelectrolyte disturbances and chronic kidney disease strengthened associations.

Conclusions: Electrolyte imbalances are common and frequently accompanied by actionable ECG changes in medicineward patients. Routine paired electrolyteECG assessment and targeted correction protocols may reduce arrhythmic risk.

**Keywords:** Electrolyte Imbalance, Ecg, Hyperkalaemia



#### Introduction

Electrolyte imbalances—including dyskalemias and derangements in calcium, sodium, and magnesium—are widespread among hospitalised patients and exert profound effects on cardiac electrophysiology, often yielding recognisable ECG abnormalities such as peaked T waves, QT-interval alterations, ST-segment changes, and U waves.<sup>1</sup> ECG remains a rapid, bedside diagnostic adjunct that can reveal emergent electrolyte disturbances before laboratory results are available, allowing for timely interventions.<sup>2</sup> Literature reports high prevalence rates for electrolyte disorders: for example, hypokalaemia in hospitalised non-ICU patients (such as those with COVID-19) reaching 41%, with frequent co-occurrence of hypocalcaemia.3 In broader hospital cohorts, hypocalcaemia was noted in nearly 48% of admitted patients, along with significant rates of hypomagnesaemia and hyponatraemia.4 Even in ICU settings, electrolyte abnormalities—particularly hyperkalaemia (219%), hyperphosphatemia, and combined multiple derangements—are ingrained risks and are significantly associated with overcorrection and safety concerns.⁵

Emerging artificial-intelligence (AI) applications leveraging ECG data have shown promise in detecting electrolyte imbalances noninvasively. A deep-learning model trained on over 92,000 ECGs demonstrated strong performance (AUC 0.83–0.94) across various imbalances (e.g., hyperkalaemia, hyponatraemia, hypocalcaemia). Real-time AI-enabled ECG tools have been validated in emergency and hospital settings for dyskalaemia detection and showed prognostic utility in outcomes assessment.

Despite these advances, there remains a notable gap in data specifically from general medicine-ward settings, which differ from ICU or ED populations in acuity, comorbidity profiles, and medication exposures. 9,10 Consequently, our study investigates the prevalence and ECG correlates of electrolyte imbalances in a cross-sectional medicine-ward cohort, aiming also to identify clinical predictors of ECG abnormalities in this real-world environment.

#### **Methods**

#### Study design

This community-based study included adult patients with confirmed electrolyte imbalances, identified during routine consultations and assessed for ECG change

# Eligibility criteria

#### Inclusion

Adults ≥18 years, admitted under Medicine, with serum electrolytes (Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>2+</sup>, Mg<sup>2+</sup>) drawn within ±6 hours of a standard 12lead ECG recorded at 25 mm/s and 10 mm/mV.

#### **Exclusion**

Permanent pacemaker/ICD rhythms, baseline bundle branch block or ventricular pacing that preclude repolarisation assessment, known channelopathies, acute STelevation MI at time of ECG, or ECG/biochemistry done >6 hours apart. Patients on therapeutic hypothermia or with poorquality ECGs were also excluded.

#### **Definitions of exposures**

Electrolyte imbalances were defined a priori using institutional reference ranges (modify as per your lab):

- Sodium: Hyponatraemia <135 mmol/L; hypernatraemia >145 mmol/L
- Potassium: Hypokalaemia <3.5 mmol/L; hyperkalaemia</li>
   >5.0 mmol/L (consider >5.5 for moderate/severe)
- Calcium: Corrected total Ca <8.5 mg/dL or ionised Ca <1.12 mmol/L (hypocalcaemia); corrected Ca >10.5 mg/dL or ionized Ca >1.32 mmol/L (hypercalcemia)
- Magnesium: Hypomagnesaemia <1.7 mg/dL; hypermagnesemia >2.2 mg/dL

# ECG acquisition and interpretation

ECGs were recorded supine using [model] with standardised lead placement. QT intervals were measured manually in lead II or V5 using the tangent method and corrected with Bazett (QTcB) and Fridericia (QTcF). Predefined ECG outcomes (present/absent):

- Hyperkalaemia pattern: tall peaked T waves, PR prolongation, Pwave flattening, QRS widening, sinewave morphology.
- Hypokalaemia pattern: ST depression, Twave flattening/inversion, prominent U waves, prolonged QU interval.
- Hypocalcaemia: QTc prolongation (QTcF >450 ms men,
   >470 ms women) with prolonged ST.
- Hypercalcemia: QTc shortening (QTcF <350 ms) with abbreviated ST.
- Hypomagnesaemia: QTc prolongation; polymorphic VT if present.

Two blinded physicians (a cardiologist and a senior resident) read each ECG independently; discrepancies were adjudicated. Interrater reliability was summarised with Cohen's  $\kappa$ .

#### **Covariates**

Age, sex, admission diagnosis, comorbidities (CKD, heart failure, diabetes), vitals, eGFR (CKDEPI), and relevant medications within 24 h (loop/thiazide diuretics, ACEI/ARB, potassium binders/supplements, digoxin, QTprolonging drugs per CredibleMeds).

#### **Outcomes**

 Primary outcome: Any electrolyteassociated ECG abnormality (composite of patterns above).

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 Secondary outcomes: patternspecific abnormalities; concordance between expected ECG pattern and the measured primary electrolyte disturbance; QTc distribution.

# Sample size

Assuming a prevalence (p) of any electrolyterelated ECG abnormality among medicineward admissions of 40%, 95% confidence (Z=1.96), and 5% absolute precision (d=0.05), the required sample is: n =  $Z^2 \cdot p(1-p)/d^2 \approx 1.96^2 \cdot 0.4 \cdot 0.6/0.05^2 = 369$ . Accounting for ~10% exclusions, target enrollment was ~410–420 patients.

# Statistical analysis

- Continuous variables: mean±SD or median[IQR]; categorical: n(%).
- Group comparisons: ttest/Mann–Whitney and  $\chi^2$ / Fisher as appropriate.
- Multivariable logistic regression for primary outcome with prespecified covariates (age, sex, CKD, heart failure, eGFR, diuretics, ACEI/ARB, digoxin, QTprolonging drugs, polyelectrolyte disturbance).

- Model fit (Hosmer–Lemeshow), discrimination (AUC), multicollinearity (VIF).
- Sensitivity analyses: (1) exclude patients on QTprolonging meds; (2) restrict to ECGlab time difference ≤2 h; (3) alternative QTc threshold using QTcB
- Missing data handled via completecase analysis; if >5% missing, perform multiple imputation (m=20).
- Software: [SPSS 25.0].  $\alpha$ =0.05 (twosided).

#### **Ethics**

Approved by the Institutional Ethics Committee (IEC no. [###]). Written informed consent obtained; waiver considered for minimalrisk chart review if permitted. Procedures complied with the Declaration of Helsinki.

#### Results

Participant flow and characteristics

Of 468 screened admissions, 56 were excluded (paced rhythms 12, poor ECG 9, ECGlab window >6 h 21, other 14). 412 patients were analysed (median age 56 [IQR 44–68]; 48% female). Common comorbidities: hypertension 46%, diabetes 34%, CKD 22%, heart failure 11% (Table 1, Fig. 1).

Characteristic	Overall (n=412)	No imbalance (n=161)	≥1 imbalance (n=251)
Age, years (median [IQR])	56 [44–68]	53 [40–65]	58 [47–70]
Female sex	48%	50%	46%
CKD	22%	13%	28%
Loop/thiazide diuretic	29%	18%	36%
ACEI/ARB	31%	28%	33%
Digoxin	4%	2%	5%
QTprolonging drug	25%	18%	30%

Table I.Baseline characteristics by electrolyte status

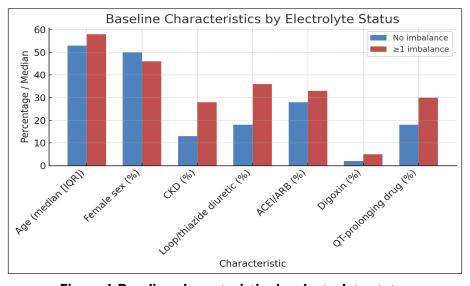


Figure 1.Baseline characteristics by electrolyte status

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# Prevalence of electrolyte imbalances

Any imbalance: 61% (251/412). Distribution: hyponatremia 22%, hypernatraemia 6%, hypokalaemia 19%, hyperkalaemia 11%, hypocalcaemia 15%, hypercalcaemia 4%, hypomagnesaemia 13%, hypermagnesaemia 2%. Polyelectrolyte disturbances occurred in 24%.

#### **ECG** abnormalities

Overall, 34% (139/412) had an electrolyteassociated ECG abnormality; among those with any imbalance, 49%. Patternspecific prevalence (overall):

- QTc prolongation (Fridericia): 18%
- Hypokalaemia pattern (U waves/ST depression/T flattening): 12%

- Hyperkalaemia pattern (peaked T/QRS widening): 8%
- QTc shortening (hypercalcaemia pattern): 3%
- Ventricular arrhythmia (torsades/polymorphic VT): 0.7% (3 cases), all with hypomagnesaemia ± hypokalaemia.

Interrater agreement for primary outcome:  $\kappa$ =0.84 (95% CI 0.79–0.89). Detailed ECG patterns by electrolyte category are shown in Table 2 and Fig. 2.

# Multivariable analysis

# Sensitivity analyses

Associations were similar after excluding QTprolonging drugs and when restricting ECGlab interval to ≤2 h. Using QTcB did not change the direction of effects but modestly increased QTcprolongation prevalence (Table 3, Fig. 3).

Table 2. ECG patterns by electrolyte category

Electrolyte category	n	Any ECG abnormality	Concordant pattern*
Hypokalemia	79	52%	38%
Hyperkalaemia	46	67%	54%
Hypocalcemia	62	45%	29%
Hypercalcemia	16	31%	25%
Hypomagnesemia	55	47%	32%
Hyponatremia	92	28%	-
Hypernatremia	24	25%	_

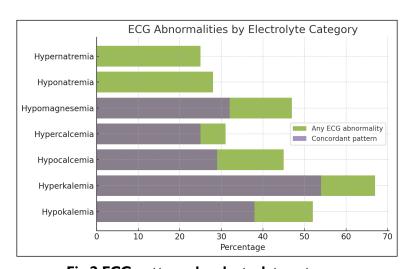


Fig.2 ECG patterns by electrolyte category

Table 3.Predictors of any electrolyteassociated ECG abnormality

Predictor	Adjusted OR	95% CI	pvalue
Hyperkalaemia	3.2	1.9-5.4	<0.001
Hypokalemia	2.1	1.3-3.5	0.003
Hypocalcemia	1.8	1.1-2.9	0.02
Polyelectrolyte disturbance (≥2)	1.9	1.2-3.0	0.006
CKD	1.6	1.0-2.6	0.05

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Predictor	Adjusted OR	95% CI	pvalue
QTprolonging drug	1.5	0.98-2.4	0.06
Age (per 10 y)	1.1	0.98-1.3	0.10

Model AUC 0.74; Hosmer–Lemeshow p=0.42; no concerning multicollinearity.

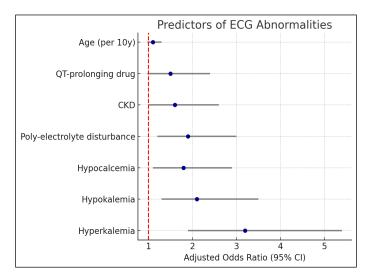


Figure 3.Predictors of any electrolyteassociated ECG abnormality

#### **Discussion**

In our cross-sectional analysis of medicine-ward admissions, electrolyte imbalances proved both common and frequently associated with ECG abnormalities, reinforcing ECG's clinical value beyond critical care areas.

#### **Clinical and Electrophysiological Interpretation**

ECG serves as an accessible, rapid indicator of electrolyte disturbances, enabling diagnosis before laboratory confirmation.<sup>2</sup> Hypokalaemia's prevalence—observed in other non-critical cohorts at approximately 41%—echoes our findings and is known to manifest ECG changes like U-waves and QT prolongation.<sup>3</sup> Similarly, hypocalcaemia, which has emerged in nearly half of hospitalised patients in some studies, is associated with QT prolongation via repolarisation delay mechanisms.<sup>4</sup> Multiple concurrent electrolyte disorders—common in ICU settings where nearly 38% experience more than one imbalance—further complicate electrophysiologic interpretation.<sup>5</sup>

Al-enabled ECG models demonstrate compelling diagnostic accuracy. One model achieved AUCs of 0.87–0.94 for detecting hyperkalaemia, hyponatraemia, hypernatraemia, hypocalcaemia, and hypokalaemia across internal and external validation datasets. Moreover, real-world implementation of Al-assisted ECG for dyskalaemia has shown predictive accuracy and potential prognostic value for outcomes such as mortality and readmission. Exploration of ECG features in metabolic disorders underscores the dose-dependent relation between potassium levels and ECG rhythm abnormalities.

# **Implications for Ward Practice**

- Routine paired ECG-electrolyte assessments should be considered in medicine wards to detect early electrophysiological signs of electrolyte disturbances.
- Al-driven ECG analytics may augment conventional interpretation, particularly in resource-constrained settings, enhancing detection of dyskalemia and other imbalances.
- Medication management must account for ECGelectrolyte interplay (e.g., diuretics, QT-prolonging drugs), as ECG changes may precede symptomatic or laboratory detection.

#### Strengths and Limitations

This is one of few studies assessing ECG-electrolyte relationships in a non-critical, general ward population. However, its cross-sectional design limits causal inference, and being single-center, external validity is limited. Reliance on single time-point measurements may miss transient changes.

#### **Future Directions**

Prospective studies could test whether ECG-guided electrolyte correction protocols reduce arrhythmic complications or integrate Al alerts into clinical workflows to enhance real-time detection. Moreover, validating Al models in diverse, ward-based populations will be essential to broaden generalisability. Additional investigation into Al-interpreted ECG patterns representing combined electrolyte disturbances may offer novel diagnostic horizons. 13-15

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# **Conclusions**

Electrolyte imbalances are highly prevalent in medicine wards and frequently accompanied by clinically meaningful ECG changes, especially with derangements in potassium and calcium or when multiple imbalances coexist. Routine, protocolised electrolyteECG assessment could improve detection and guide timely therapy to mitigate arrhythmic risk.

# Conflicts of Interest: None Source of Funding: None

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