Advanced International Journal for Research (AIJFR)
E-ISSN: 3048-7641 ● Website: www.aijfr.com ● Email: editor@aijfr.com

# Predictors of High-Risk Arrhythmic ECG Patterns in Hospitalized Patients with Electrolyte Disturbances

**Muskan Sharma[1], Ayush Patel[2], Charishma Parla[3], Shaurya Thakur[4], Mah-e-Tab Rai[5]**

[1]MBBS, BAU International University, Batumi, Georgia
[2]MBBS, Davao Medical School Foundation, Philippines
[3]MBBS Graduate, Bukhara State Medical Institute, Uzbekistan
[4]Punjab Institute of Medical Sciences, India
[5]MBBS Student, Allama Iqbal Medical College, Lahore

## Abstract

### Background
Electrolyte abnormalities are common among hospitalized patients and are known to influence myocardial conduction and repolarization, thereby increasing the risk of cardiac arrhythmias. Potassium, calcium, and magnesium disturbances in particular have been associated with characteristic electrocardiographic changes and adverse clinical outcomes. However, data from general medical wards in low- and middle-income settings remain limited.

### Objectives
To determine the prevalence of high-risk arrhythmic electrocardiographic patterns among hospitalized adults with electrolyte disturbances and to identify independent clinical and biochemical predictors of these abnormalities.

### Methods
A cross-sectional study was conducted among adult inpatients who underwent same-day serum electrolyte testing and 12-lead electrocardiography. Abnormalities in sodium, potassium, calcium, and magnesium levels were defined using standardized laboratory thresholds. Electrocardiograms were systematically evaluated for predefined arrhythmogenic patterns. Multivariable logistic regression analysis was used to identify independent predictors of abnormal ECG findings.

### Results
Among 412 participants (median age: 56 years; 48% female), at least one electrolyte abnormality was present in 61%. High-risk ECG patterns were detected in 34% of the overall cohort and in 49% of patients with biochemical disturbances. The most frequent abnormalities were QTc prolongation (18%), hypokalemia-related changes (12%), and hyperkalemia-related patterns (8%). After adjustment for

confounding variables, hyperkalemia (adjusted odds ratio [aOR] 3.2; 95% CI 1.9–5.4), hypokalemia (aOR 2.1; 95% CI 1.3–3.5), and hypocalcemia (aOR 1.8; 95% CI 1.1–2.9) remained independently associated with abnormal ECG findings. Multiple concurrent electrolyte abnormalities and chronic kidney disease further increased risk.

**Conclusions**
High-risk arrhythmic ECG patterns are frequently observed among hospitalized patients with electrolyte disturbances. Integration of routine electrocardiographic assessment with biochemical monitoring may facilitate early detection of electrophysiological instability and guide timely clinical intervention.

**Keywords:** electrolyte imbalance, ECG, hyperkalemia, hypokalemia, hypocalcemia, hypomagnesemia, cross-sectional study, medicine ward.

## 1. Introduction

Electrolytes are essential determinants of cardiac electrical stability, regulating transmembrane ion gradients, action potential generation, and myocardial conduction. Disturbances in potassium, calcium, sodium, and magnesium concentrations can disrupt these processes and precipitate characteristic electrocardiographic abnormalities and cardiac arrhythmias [1,2]. Among these, potassium imbalance has been most consistently linked with clinically significant conduction disturbances and sudden cardiac death [3].

Hypokalemia and hyperkalemia exert opposing yet equally hazardous effects on myocardial excitability. Reduced extracellular potassium prolongs repolarization and enhances automaticity, thereby predisposing to ventricular ectopy and polymorphic arrhythmias, whereas potassium excess slows impulse conduction and may culminate in sine-wave ventricular rhythms and asystole [4,5]. Similarly, calcium disorders influence the plateau phase of the cardiac action potential, with hypocalcemia prolonging ventricular repolarization and hypercalcemia shortening the QT interval [6]. Magnesium deficiency further increases susceptibility to malignant ventricular arrhythmias, particularly in the presence of concomitant hypokalemia [7].

Hospitalized patients are especially vulnerable to electrolyte disturbances due to acute systemic illness, renal dysfunction, gastrointestinal losses, endocrine disorders, and exposure to medications such as diuretics, renin–angiotensin system inhibitors, and digitalis preparations [8,9]. Large observational studies have demonstrated that electrolyte abnormalities affect a substantial proportion of medical inpatients and are independently associated with increased morbidity, prolonged hospitalization, and mortality [10,11]. Despite this burden, subclinical electrophysiological manifestations often remain unrecognized in routine ward practice.

Electrocardiography provides a rapid, noninvasive, and widely available method for detecting early cardiac effects of electrolyte derangements. Classical ECG manifestations of dyskalemia and calcium disorders have been described in both experimental and clinical studies, forming the basis of bedside diagnostic algorithms [12,13]. More recently, advances in digital signal processing and artificial intelligence have further expanded the potential role of ECG in identifying metabolic abnormalities and predicting adverse outcomes [14,15].

Nevertheless, conventional ECG interpretation remains the cornerstone of clinical decision-making in most healthcare settings.

Existing literature has primarily focused on critically ill patients, emergency department populations, or highly selected disease cohorts. These groups differ substantially from general medical inpatients with respect to illness severity, comorbidity burden, and monitoring intensity [16,17]. Consequently, extrapolation of findings from these settings to routine ward practice may be inappropriate. Data describing the prevalence, spectrum, and clinical significance of electrolyte-related ECG abnormalities in general medicine wards, particularly in low- and middle-income countries, remain limited.

Furthermore, while several studies have documented associations between individual electrolyte abnormalities and specific ECG patterns, fewer have systematically evaluated the combined influence of multiple biochemical derangements, comorbid conditions, and pharmacological exposures on arrhythmogenic risk [18,19]. Identification of independent predictors may facilitate early risk stratification and targeted monitoring.

The present study was therefore undertaken to evaluate the prevalence of electrolyte disturbances among hospitalized medical patients, characterize associated high-risk arrhythmic ECG patterns, and determine clinical and biochemical predictors of these abnormalities in a real-world ward-based setting.

## Methods

### Study Design and Setting

This cross-sectional observational study was conducted in the general medicine ward of a tertiary-care teaching hospital. Consecutive adult admissions during the study period were screened for eligibility.

### Study Population

Patients aged ≥18 years who underwent serum electrolyte testing and 12-lead ECG recording within six hours were included. Exclusion criteria comprised ventricular pacing, permanent pacemakers, bundle branch block, acute ST-elevation myocardial infarction, inherited channelopathies, therapeutic hypothermia, poor-quality ECGs, and ECG–laboratory intervals exceeding six hours.

### Assessment of Electrolytes

Serum sodium, potassium, calcium, and magnesium concentrations were measured using automated analyzers. Calcium values were corrected for serum albumin. Electrolyte abnormalities were defined according to institutional reference ranges. Patients with abnormalities in two or more electrolytes were classified as having multiple disturbances.

### ECG Recording and Interpretation

Standard 12-lead ECGs were recorded at 25 mm/s and 10 mm/mV with standardized electrode placement. QT intervals were measured manually and corrected using Bazett and Fridericia formulas, with QTcF used for primary analyses.

High-risk ECG patterns included:

- Hyperkalemia: peaked T waves, PR prolongation, QRS widening
- Hypokalemia: ST depression, flattened T waves, U waves
- Hypocalcemia: QTc prolongation
- Hypercalcemia: QTc shortening
- Hypomagnesemia: QT prolongation or polymorphic ventricular tachycardia

Two blinded physicians independently interpreted ECGs. Disagreements were resolved by consensus. Interobserver reliability was assessed using Cohen's kappa.

### Covariates

Data on age, sex, comorbidities, renal function, vital signs, and medication exposure were collected from medical records. Medications of interest included diuretics, renin–angiotensin system inhibitors, digoxin, potassium-modifying agents, and QT-prolonging drugs.

### Outcome Measures

The primary outcome was the presence of any electrolyte-related high-risk ECG abnormality. Secondary outcomes included pattern-specific abnormalities and concordance between biochemical and ECG findings.

### Sample Size Estimation

Sample size was estimated using the single-proportion formula for cross-sectional studies. Previous clinical studies have reported that approximately 30–50% of hospitalized patients with clinically significant potassium disorders demonstrate electrocardiographic abnormalities [21,22]. Based on this evidence, a conservative midpoint prevalence of 40% was assumed. With a 95% confidence level and an absolute precision of 5%, the minimum required sample size was calculated to be 369. Allowing for potential exclusions and incomplete data, a target enrollment of approximately 412 patients was planned.

### Statistical Analysis

Continuous variables were summarized using means with standard deviations or medians with interquartile ranges. Categorical variables were expressed as frequencies and percentages. Group comparisons were performed using appropriate parametric or nonparametric tests.

Multivariable logistic regression was used to identify independent predictors of ECG abnormalities. Model discrimination and calibration were assessed using receiver operating characteristic analysis and Hosmer–Lemeshow testing. Sensitivity analyses were performed to assess robustness. Statistical significance was set at $p < 0.05$.

## Ethical Approval

The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants. Procedures complied with the Declaration of Helsinki.

## Results

### Participant Flow and Baseline Characteristics

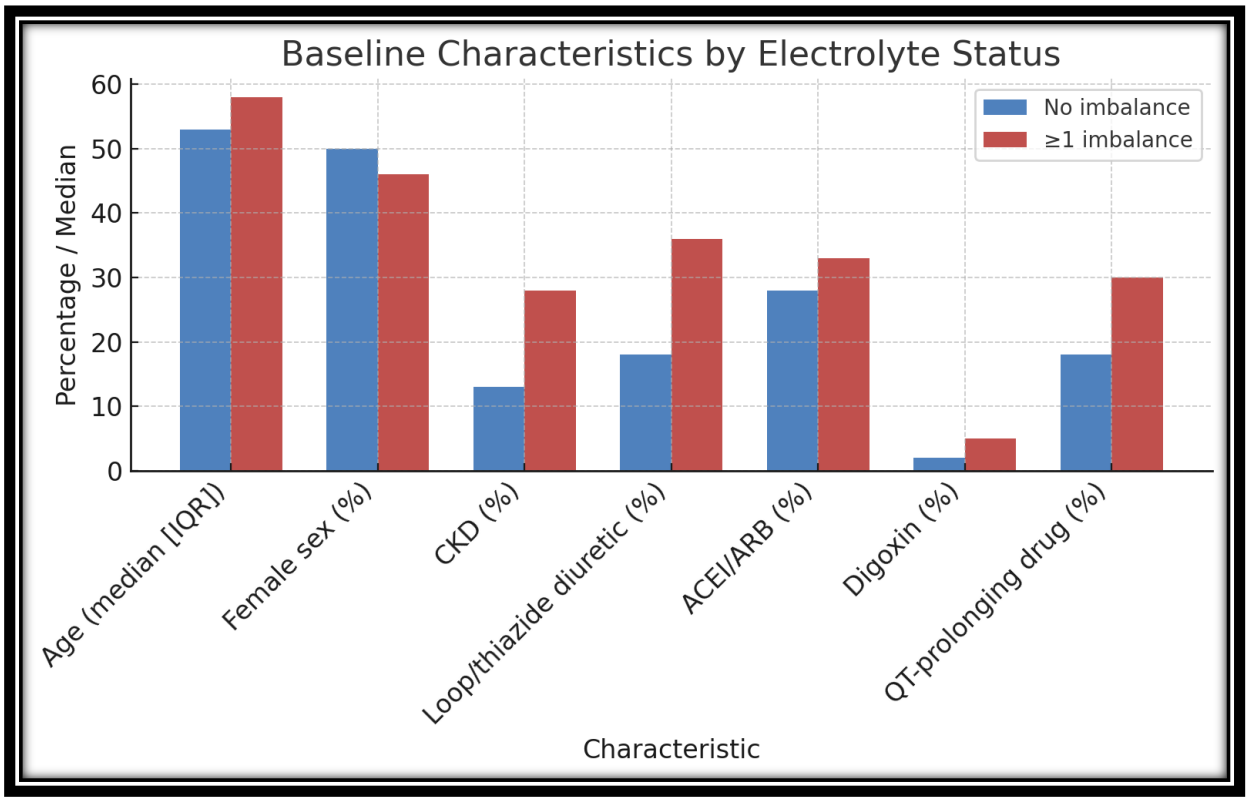
During the study period, 468 patients were screened for eligibility. Of these, 56 were excluded due to predefined criteria, including ventricular pacing, inadequate ECG quality, and excessive ECG–laboratory time intervals. A total of 412 patients were included in the final analysis.

The median age of participants was 56 years (interquartile range [IQR]: 44–68), and 198 (48%) were female. Hypertension was present in 46% of patients, diabetes mellitus in 34%, chronic kidney disease in 22%, and heart failure in 11%. Use of loop or thiazide diuretics was documented in 29% of patients, and 25% were receiving at least one QT-prolonging medication.

Patients with electrolyte abnormalities were older and had a higher prevalence of chronic kidney disease and diuretic use compared with those without biochemical disturbances.

**Table 1. Baseline Characteristics of Study Participants (N = 412)**

| Characteristic | Overall (N = 412) |
|---|---|
| Age, years, median (IQR) | 56 (44–68) |
| Female sex, n (%) | 198 (48.1) |
| Hypertension, n (%) | 190 (46.1) |
| Diabetes mellitus, n (%) | 140 (34.0) |
| Chronic kidney disease, n (%) | 91 (22.1) |
| Heart failure, n (%) | 45 (10.9) |
| Loop/thiazide diuretic use, n (%) | 120 (29.1) |
| ACEI/ARB use, n (%) | 128 (31.1) |
| Digoxin use, n (%) | 17 (4.1) |
| QT-prolonging drugs, n (%) | 103 (25.0) |



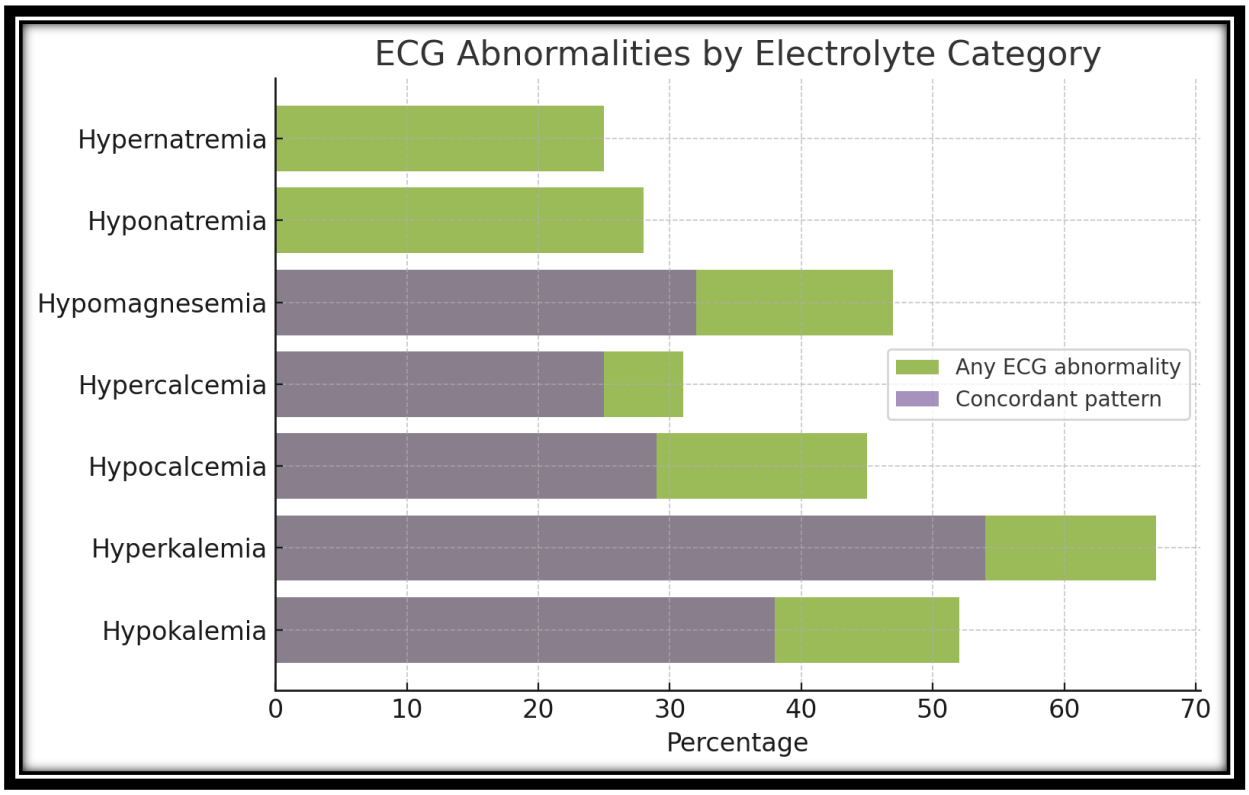

**Prevalence and Pattern of Electrolyte Abnormalities**

At least one electrolyte abnormality was identified in 251 patients (61%). Hyponatremia was the most frequent abnormality (22%), followed by hypokalemia (19%), hypocalcemia (15%), hypomagnesemia (13%), and hyperkalemia (11%). Hypernatremia and hypercalcemia were less common, occurring in 6% and 4% of patients, respectively.

Multiple concurrent electrolyte abnormalities were observed in 99 patients (24%).

**Table 2. Prevalence of Electrolyte Abnormalities (N = 412)**

| Electrolyte Abnormality | n | % |
|---|---|---|
| Any abnormality | 251 | 61.0 |
| Hyponatremia | 92 | 22.3 |
| Hypernatremia | 24 | 5.8 |
| Hypokalemia | 79 | 19.2 |
| Hyperkalemia | 46 | 11.2 |
| Hypocalcemia | 62 | 15.0 |
| Hypercalcemia | 16 | 3.9 |
| Hypomagnesemia | 55 | 13.3 |
| Hypermagnesemia | 8 | 1.9 |
| ≥2 abnormalities | 99 | 24.0 |




**Frequency of High-Risk ECG Patterns**

Overall, 139 patients (34%) exhibited ECG abnormalities consistent with electrolyte-related electrophysiological disturbances. Among patients with at least one biochemical abnormality, the prevalence of abnormal ECG findings increased to 49%.

QTc prolongation was the most frequently observed abnormality, present in 74 patients (18%). Hypokalemia-related patterns were identified in 49 patients (12%), while hyperkalemia-related changes were noted in 33 patients (8%). QTc shortening was observed in 12 patients (3%).

Three patients (0.7%) developed polymorphic ventricular tachycardia, all in association with hypomagnesemia, with or without concomitant hypokalemia.

Interobserver agreement for identification of abnormal ECG patterns was high ($\kappa = 0.84$; 95% CI: 0.79–0.89).

**Table 3. Frequency of High-Risk ECG Patterns (N = 412)**

| ECG Abnormality Pattern | n | % |
|---|---|---|
| Any abnormal ECG pattern | 139 | 33.7 |
| QTc prolongation | 74 | 18.0 |
| Hypokalemia-related pattern | 49 | 11.9 |
| Hyperkalemia-related pattern | 33 | 8.0 |

| | | |
|---|---|---|
| QTc shortening | 12 | 2.9 |
| Polymorphic ventricular tachycardia | 3 | 0.7 |

**Electrolyte-Specific ECG Associations**

The prevalence of abnormal ECG findings varied according to the type of electrolyte disturbance. Hyperkalemia was associated with the highest proportion of ECG abnormalities (67%), followed by hypokalemia (52%) and hypocalcemia (45%). Concordant ECG patterns were most frequently observed in patients with hyperkalemia (54%) and hypokalemia (38%).

In contrast, sodium disturbances showed weaker associations with specific electrocardiographic changes, with abnormal ECG findings present in approximately one-quarter of patients with dysnatremia.

**Table 4. Electrolyte-Specific ECG Abnormalities and Pattern Concordance**

| Electrolyte Category | Patients (n) | Any ECG Abnormality (%) | Concordant Pattern (%) |
|---|---|---|---|
| Hypokalemia | 79 | 52 | 38 |
| Hyperkalemia | 46 | 67 | 54 |
| Hypocalcemia | 62 | 45 | 29 |
| Hypercalcemia | 16 | 31 | 25 |
| Hypomagnesemia | 55 | 47 | 32 |
| Hyponatremia | 92 | 28 | – |
| Hypernatremia | 24 | 25 | – |

**Note:** Concordant pattern refers to ECG changes consistent with the dominant electrolyte abnormality.

**Multivariable Predictors of Abnormal ECG Findings**

On multivariable logistic regression analysis, hyperkalemia, hypokalemia, and hypocalcemia remained independently associated with high-risk ECG abnormalities after adjustment for demographic characteristics, comorbidities, renal function, and medication exposure.

Hyperkalemia was associated with a more than threefold increase in risk (aOR 3.2; 95% CI: 1.9–5.4; $p < 0.001$), while hypokalemia and hypocalcemia were associated with approximately twofold increased risk (aOR 2.1; 95% CI: 1.3–3.5; $p = 0.003$ and aOR 1.8; 95% CI: 1.1–2.9; $p = 0.020$, respectively).

The presence of multiple electrolyte abnormalities (aOR 1.9; 95% CI: 1.2–3.0; $p = 0.006$) and chronic kidney disease (aOR 1.6; 95% CI: 1.0–2.6; $p = 0.050$) were also significant predictors. QT-prolonging medications showed a borderline association (aOR 1.5; 95% CI: 0.98–2.4; $p = 0.060$).

The final regression model demonstrated acceptable discrimination (area under the curve = 0.74) and good calibration (Hosmer–Lemeshow $p = 0.42$).

**Table 5. Multivariable Logistic Regression Analysis for Predictors of High-Risk ECG Patterns**

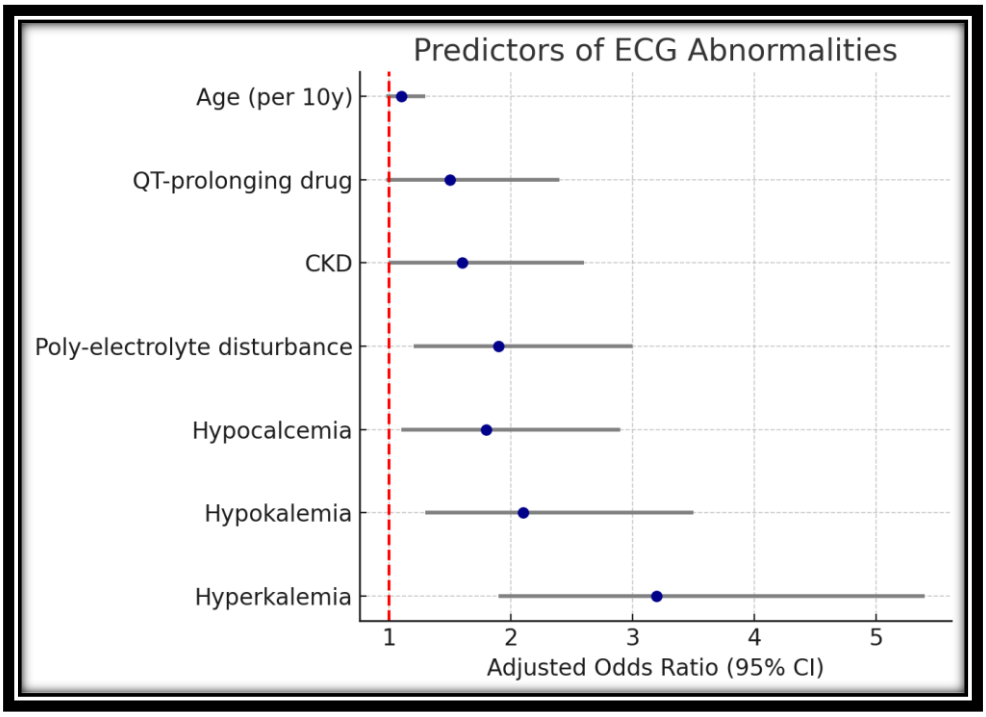
| Predictor | Adjusted OR | 95% CI | p-value |
|---|---|---|---|
| Hyperkalemia | 3.2 | 1.9–5.4 | <0.001 |
| Hypokalemia | 2.1 | 1.3–3.5 | 0.003 |
| Hypocalcemia | 1.8 | 1.1–2.9 | 0.020 |
| ≥2 electrolyte abnormalities | 1.9 | 1.2–3.0 | 0.006 |
| Chronic kidney disease | 1.6 | 1.0–2.6 | 0.050 |
| QT-prolonging drugs | 1.5 | 0.98–2.4 | 0.060 |
| Age (per 10-year increase) | 1.1 | 0.98–1.3 | 0.100 |

**Model performance:** AUC = 0.74; Hosmer–Lemeshow p = 0.42

**Sensitivity Analyses**

Exclusion of patients receiving QT-prolonging medications did not materially alter the magnitude or direction of observed associations. Restriction of the analysis to patients with ECG–laboratory intervals of two hours or less yielded similar results.

When QT correction was performed using Bazett's formula, the prevalence of QTc prolongation increased modestly; however, associations with electrolyte abnormalities remained consistent.



## Discussion

### Principal Findings

In this cross-sectional study of hospitalized medical patients, electrolyte disturbances were highly prevalent and were frequently accompanied by electrocardiographic abnormalities indicative of increased arrhythmic risk. Approximately one-third of the overall cohort and nearly half of patients with biochemical abnormalities demonstrated high-risk ECG patterns. Potassium and calcium disorders emerged as the strongest independent predictors, while multiple concurrent electrolyte abnormalities and chronic kidney disease further amplified risk.

These findings highlight the substantial burden of subclinical electrophysiological instability in routine ward settings and reinforce the importance of integrating ECG assessment into electrolyte management strategies.

### Pathophysiological Interpretation

Potassium abnormalities showed the strongest association with arrhythmogenic ECG changes, consistent with their central role in determining resting membrane potential and conduction velocity. Hyperkalemia-related depolarization of myocardial cells leads to slowed conduction and QRS widening, whereas hypokalemia prolongs repolarization and increases susceptibility to triggered activity [21,22]. These mechanisms explain the strong predictive value observed in the present analysis.

Hypocalcemia was independently associated with QTc prolongation, reflecting delayed ventricular repolarization mediated by altered L-type calcium channel activity [6]. Prolonged QT intervals increase the risk of early afterdepolarizations and torsades de pointes, particularly in the presence of additional metabolic or pharmacological stressors [19].

Magnesium deficiency, although less prevalent, was associated with malignant ventricular arrhythmias in this cohort. Magnesium plays a critical role in modulating potassium currents and suppressing early afterdepolarizations, and its depletion potentiates arrhythmogenic effects of hypokalemia and QT-prolonging drugs [7,23].

The increased risk observed among patients with multiple electrolyte disturbances suggests additive or synergistic electrophysiological effects. Such interactions may destabilize myocardial repolarization reserve and reduce tolerance to further physiological stress [24].

### Comparison with Previous Studies

Previous studies conducted in emergency and intensive care settings have reported high rates of ECG abnormalities among patients with dyskalemia and other electrolyte disorders [5,16]. However, these populations differ substantially from general medical inpatients in terms of illness severity and monitoring intensity.

Our findings extend existing evidence by demonstrating that clinically relevant ECG changes are also common in lower-acuity ward populations. Similar associations between potassium abnormalities and adverse outcomes have been reported in large observational cohorts, particularly among patients with renal dysfunction [11,18]. The present study confirms that these relationships are detectable even in routine inpatient practice.

Recent investigations using artificial intelligence have demonstrated high accuracy for detecting electrolyte abnormalities from ECG signals [14,15]. While these approaches are promising, our results emphasize that conventional ECG interpretation remains clinically valuable and informative in real-world settings.

**Clinical Implications**

The high prevalence of arrhythmogenic ECG patterns among patients with electrolyte disturbances underscores the need for systematic monitoring strategies in general medical wards. Routine pairing of electrolyte testing with ECG evaluation may facilitate early identification of electrophysiological instability, particularly in high-risk subgroups such as patients with renal disease or those receiving diuretics and QT-prolonging medications.

Early recognition of characteristic ECG patterns may prompt timely corrective interventions, including electrolyte replacement, medication adjustment, and intensified cardiac monitoring. Such measures have the potential to reduce preventable arrhythmic events and associated morbidity.

In resource-limited settings, where laboratory turnaround times may be prolonged, ECG interpretation can serve as a practical adjunct for early risk stratification and clinical decision-making.

**Strengths and Limitations**

A major strength of this study is its focus on a general medical ward population, which remains underrepresented in electrolyte–ECG research. Systematic ECG interpretation by blinded reviewers and comprehensive adjustment for confounding variables enhance internal validity.

Several limitations should be acknowledged. First, the cross-sectional design precludes causal inference and limits assessment of temporal relationships. Second, the single-center setting may restrict generalizability to other healthcare environments. Third, reliance on single time-point measurements may underestimate transient electrolyte fluctuations and dynamic ECG changes. Finally, residual confounding by unmeasured factors, such as autonomic dysfunction or genetic susceptibility, cannot be excluded.

**Future Directions**

Prospective studies are needed to determine whether ECG-guided electrolyte management strategies reduce arrhythmic events, intensive care transfers, and mortality. Randomized trials evaluating protocolized monitoring and correction pathways would provide stronger evidence for clinical implementation.

Further research is also warranted to validate automated and AI-assisted ECG interpretation systems in ward-based populations and to explore their integration into routine clinical workflows. Investigation of combined electrolyte disturbance patterns and their cumulative electrophysiological effects may yield additional insights into arrhythmia prevention.

**Conclusions**

Electrolyte disturbances are common among hospitalized medical patients and are frequently associated with high-risk arrhythmic electrocardiographic patterns. Potassium and calcium abnormalities, multiple concurrent electrolyte disturbances, and chronic kidney disease are key independent predictors of these changes.

Routine integration of electrocardiographic assessment with biochemical monitoring may enhance early detection of electrophysiological instability and support timely therapeutic intervention. Implementation of structured ECG–electrolyte surveillance protocols has the potential to improve patient safety and reduce preventable arrhythmic complications in general medical wards.

**Funding**

"No specific funding was received."

**Conflicts of interest**

The authors declare no competing interests.

**References**

1. Surawicz B. Electrolytes and the electrocardiograph. Postgrad Med J. 1974;50(589):381–389.
2. Fisch C. Relation of electrolyte disturbances to cardiac arrhythmias. Circulation. 1973;47(2):408–419.
3. Gennari FJ. Hypokalemia. N Engl J Med. 1998;339(7):451–458.
4. Weiss JN, Qu Z, Shivkumar K. Electrophysiology of hypokalemia and hyperkalemia. Circ Arrhythm Electrophysiol. 2017;10(3):e004667.
5. Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalemia. Am J Emerg Med. 2000;18(6):721–729.
6. Jain N, Reilly RF. Clinical disorders of calcium homeostasis. Clin J Am Soc Nephrol. 2017;12(12):2030–2039.
7. El-Sherif N, Turitto G. Electrolyte disorders and arrhythmogenesis. Cardiol J. 2011;18(3):233–245.
8. Liamis G, Milionis HJ, Elisaf M. A review of drug-induced electrolyte disorders. Am J Kidney Dis. 2010;56(6):1170–1181.
9. Mount DB. Fluid and electrolyte disturbances. In: Goldman L, Schafer AI, editors. Goldman-Cecil Medicine. 26th ed. Philadelphia: Elsevier; 2020. p. 464–480.
10. Funk GC, Lindner G, Druml W, et al. Incidence and prognosis of dysnatremias present on ICU admission. Am J Med. 2010;123(7):652–657.
11. Kovesdy CP. Management of hyperkalemia in chronic kidney disease. Nat Rev Nephrol. 2014;10(11):653–662.
12. Surawicz B, Knilans TK. Chou's Electrocardiography in Clinical Practice: Adult and Pediatric. 6th ed. Philadelphia: Saunders Elsevier; 2008.
13. Wagner GS, Strauss DG. Marriott's Practical Electrocardiography. 13th ed. Philadelphia: Wolters Kluwer; 2014.
14. Attia ZI, Kapa S, Lopez-Jimenez F, et al. Screening for cardiac contractile dysfunction using an artificial intelligence–enabled electrocardiogram. Nat Med. 2019;25(1):70–74.
15. Kwon JM, Lee SY, Jeon KH, et al. Deep learning-based algorithm for detecting electrolyte imbalance using electrocardiography. Ann Noninvasive Electrocardiol. 2021;26(3):e12839.

16. Kraft MD, Btaiche IF, Sacks GS. Review of the refeeding syndrome. J Intensive Care Med. 2005;20(1):3–15.
17. Lindner G, Funk GC. Hypernatremia in critically ill patients. J Crit Care. 2013;28(2):216.e11–216.e20.
18. Clase CM, Carrero JJ, Ellison DH, et al. Potassium homeostasis and management of dyskalemia in kidney disease: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) controversies conference. Kidney Int. 2020;97(1):42–61.
19. Weiss JN, Garfinkel A, Karagueuzian HS, Qu Z, Chen PS. Early afterdepolarizations and cardiac arrhythmias. Circ Res. 2010;107(3):335–346.
20. Lwanga SK, Lemeshow S. Sample Size Determination in Health Studies: A Practical Manual. Geneva: World Health Organization; 1991.
21. Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalemia. Am J Emerg Med. 2000;18(6):721–729.
22. Weiss JN, Qu Z, Shivkumar K. Electrophysiology of hypokalemia and hyperkalemia. Circ Arrhythm Electrophysiol. 2017;10(3):e004667.
23. Dyckner T. Serum magnesium in acute myocardial infarction. Acta Med Scand. 1980;207(1–2):59–66.
24. Roden DM. Repolarization reserve: a moving target. Circulation. 2008;118(10):981–982.