STEMedicine

ORIGINAL RESEARCH ARTICLE

Association Between Serum Potassium Abnormalities and Cardiac Arrhythmias in Medical Inpatients

Dr. Shivaraj Hadimani[1]*, Dr. Pragati Nalkamana[2], Dr. Manish Kumar[3]

[1]Assistant Professor, Department of General Medicine, S.R. Patil Medical College, Badagandi, Bagalkote, Karnataka
[2]House Surgeon, S.S. Institute of Medical Sciences, Davanagere, Karnataka
[3]Assistant Professor, Department of General Medicine, Balvir Singh Tomar Institute of Medical Science Research & Hospital, Jaipur - Delhi Highway, Jaipur, Rajasthan

Corresponding Author: Dr. Shivaraj Hadimani
Email: drshivarajhadimani@gmail.com

Abstract: Background: Serum potassium disturbances can alter cardiac electrophysiology and may increase arrhythmic risk in hospitalized patients. This study evaluated the association between admission potassium abnormalities and newly documented cardiac arrhythmias in medical inpatients.**Methods:** A total of 500 adult medical inpatient encounters were analyzed. Admission potassium was categorized as hypokalemia (<3.5 mmol/L), reference range (3.5–5.4 mmol/L), or hyperkalemia (≥5.5 mmol/L). The primary outcome was a clinically significant new arrhythmia occurring within 72 hours. Penalized logistic regression adjusted for age, cardiovascular disease, and estimated glomerular filtration rate.**Results:**Hypokalemia was present in 57 (11.4%) patients and hyperkalemia in 14 (2.8%). Arrhythmias occurred in 16 patients (3.2%), including 5.3% with hypokalemia, 7.1% with hyperkalemia, and 2.8% with reference potassium. Adjusted associations were not statistically significant for hypokalemia (OR 2.10, 95% CI 0.62–7.08) or hyperkalemia (OR 3.89, 95% CI 0.65–23.43). Hypokalemia was associated with T-wave flattening, prominent U waves, and longer QTc, while peaked T waves were more frequent with hyperkalemia.**Conclusion:** Potassium abnormalities produced characteristic ECG changes but were not independently associated with documented short-term arrhythmias.

Keywords: potassium; hypokalemia; hyperkalemia; cardiac arrhythmia; electrocardiography; medical inpatients

Received:10.03.2026 | Revised:25.03.2026 | Accepted:10.04.2026 | Published:30.05.2026

Citation: Dr. Shivaraj Hadimani *et al* (2026). Association Between Serum Potassium Abnormalities and Cardiac Arrhythmias in Medical Inpatients. STEMed, 7(1): 43-51.

Introduction

Potassium plays an important role in determining cardiac membrane excitability, conduction and repolarization. Even relatively small changes in extracellular potassium can affect the resting membrane potential and the activity of ion channels, leading to conditions in which conduction abnormalities or triggered electrical activity can occur [1]. Therefore, hypokalemia and hyperkalemia are both clinically significant in hospitalized patients, especially if they have structural heart disease, renal dysfunction, other electrolyte abnormalities, or drugs known to affect cardiac electrophysiology.

Hypokalemia may also cause prolonged repolarization, an increase in automaticity, and an increase in atrial and ventricular arrhythmias. Hypokalemia was found in about 17% of acute medical admissions in a prospective cohort, with more severe hypokalemia associated with higher short-term mortality rates [2]. The same findings have been reported in large unselected populations of medical patients, with both low and high serum potassium levels linked to poor hospital outcomes [3]. A U-shaped association between admission potassium and mortality was also seen in a large group of hospitalized adults [4].

This potassium concentration-cardiac rhythm association is well known in cardiovascular

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

diseases. In patients admitted to hospital with acute myocardial infarction, Goyal et al. observed a U-shaped association between potassium and the occurrence of VF or cardiac arrest, and between potassium and mortality [5].

Hypokalemia<3.5 mmol/L was associated with cardiac arrest, and potassium <3.0 mmol/L was associated with new onset atrial fibrillation, but hyperkalemia was more strongly associated with mortality in a large cohort of patients admitted with suspected acute coronary syndrome [6]. Population data also indicate that low potassium is a risk factor for atrial arrhythmogenesis. In the Rotterdam Study, hypokalemia was linked to a 63% higher risk of incident AF and even more strongly in those who had a previous myocardial infarction [7].

Hyperkalemia impairs cardiac conduction by causing progressive depolarization of the membrane and inactivation of sodium channels. Common electrocardiographic changes include peaked T waves, prolonged PR interval, widened QRS, conduction block, bradyarrhythmias, and, at extreme levels, ventricular arrhythmia or asystole. ECG changes, however, do not occur in a consistent and predictable manner and are not directly proportional to the serum potassium level, so clinical risk assessment is based on both biochemical and electrophysiological parameters [8]. Arrhythmic susceptibility may be further modified by coexisting abnormalities like hypomagnesemia, renal impairment, cardiovascular disease, and exposure to QT-prolonging or potassium-altering medications.

Although there is a large body of evidence from the myocardial infarction, heart failure, and chronic kidney disease populations, the link between admission potassium abnormalities and newly identified arrhythmias in larger medical inpatient populations is less established. Potassium disturbances are often mild in hospitalized patients, and their independent effect may be difficult to distinguish from the effect of underlying disease, renal function, cardiovascular comorbidities, and variations in rhythm monitoring.

This study was thus designed to examine the relationship between admission potassium abnormalities and newly recognised clinically important cardiac arrhythmias in adult medical inpatients. Secondary objectives were to characterize the ECG changes associated with potassium, assess relevant clinical and biochemical covariates, and evaluate the robustness of the potassium–arrhythmia relationship under sensitivity analyses.

Materials and Methods

Study design and setting

A retrospective observational analysis was conducted on a structured cohort of 500 adult medical inpatient encounters from November 2025 to March 2026. The clinical setting for the above mentioned research protocol was S.R. Patil Medical College, Badagandi, Bagalkote, Karnataka. The 600 candidate records were screened against prespecified criteria and 519 were eligible; the first 500 eligible encounters in chronological order were analyzed.

Eligibility criteria

Records were eligible for inclusion if the patient was 18 years old or older, had an index serum potassium level obtained within 24 hours of admission, an interpretable baseline electrocardiogram (ECG) obtained within 6 hours of the potassium level, and adequate follow-up documentation to determine the primary outcome. Records were excluded if the potassium measured was invalid or hemolyzed, if the patient had an active arrhythmia at or before the index sampling, if there was insufficient rhythm documentation, if the event chronology was uncertain, or if the record was a duplicate. There was no exclusion for having a history of arrhythmia, but no active baseline rhythm disturbance.

Exposure and clinical evaluations

The main exposure was serum potassium at index sampling, categorized as hypokalemia (less than 3.5 mmol/L), study reference range (3.5–5.4 mmol/L), or hyperkalemia (≥5.5 mmol/L). Baseline variables included demographic, admission diagnosis, comorbidities, pre-index medication exposure, vital signs, renal function, magnesium and other electrolyte levels, and ECG findings. Estimated glomerular filtration rate was estimated with the race-free CKD-EPI creatinine equation from 2021. The corrected calcium was calculated by adding $0.8 \times [4 - \text{serum albumin (g/dL)}]$ to the total calcium (mg/dL).

Outcome definition

The primary outcome was the development of a clinically significant cardiac arrhythmia within 72 hours after the index potassium collection or before discharge or death if earlier. The first qualifying rhythm disturbance was counted for each record. Atrial fibrillation/flutter, supraventricular tachycardia, ventricular tachycardia or fibrillation, high-grade atrioventricular block, and clinically significant bradyarrhythmias were eligible events. Arrhythmia events were not defined as isolated sinus tachycardia, premature beats, QTc prolongation or potassium-related T-wave or U-wave changes. ECG and telemetry findings were analyzed separately, alongside monitoring intensity, potassium correction, transfer to the ICU

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

and in-hospital mortality.

Statistical analysis

Data for continuous variables were presented as mean (standard deviation) or median [interquartile range] and analyzed by Welch analysis of variance or Kruskal–Wallis test as appropriate. Categorical variables were presented as n (%) and compared with Pearson's chi-square or Fisher's exact test, with Monte Carlo estimation for sparse multiway tables. The proportions of arrhythmias were reported with Wilson 95% confidence intervals, absolute risk differences and odds ratios relative to the reference potassium category. Univariable logistic regression was used to assess candidate clinical and medication-related factors. Since the count of events was limited, the prespecified adjusted analysis was performed using Jeffreys-prior penalized logistic regression with separate hypokalemia and hyperkalemia indicators, age (per 10 years), cardiovascular disease (coronary artery disease and/or heart failure) and estimated glomerular filtration rate (per 10 mL/min/1.73 m^2). Adjusted odds ratios were presented with approximate Wald 95% confidence intervals (CIs) and two-sided p-values. Sensitivity analyses considered any potassium abnormality, more complete rhythm surveillance, exclusion of eGFR <30 mL/min/1.73 m^2, and borderline-high potassium (5.1–5.4 mmol/L). Missing covariate observations were handled by available-case analysis without imputation; the primary adjusted model included 500 records. Statistical significance was defined as $p<0.05$.

Results

Study population and potassium distribution. The cohort comprised 500 adult medical inpatient records, with a mean age of 53.4 (SD 16.1) years; 292 (58.4%) were male. Serum potassium concentrations were within the study reference range (3.5–5.4 mmol/L) in 429 (85.8%) patients, while 57 (11.4%) had hypokalemia and 14 (2.8%) had hyperkalemia. Most abnormalities were mild. Type 2 diabetes, gastrointestinal fluid-loss disorders, and lower serum magnesium concentrations were more frequent in the hypokalemia group. Baseline characteristics and potassium-severity categories are summarized in Tables 1 and 2.

Occurrence and patterns of cardiac arrhythmias. New clinically significant cardiac arrhythmias were documented within 72 hours in 16 (3.2%) patients. Arrhythmia occurrence was numerically higher in both potassium-abnormality groups than in the reference group, although the overall association was not statistically significant (Fisher's exact p=0.257). Atrial fibrillation was the most frequent arrhythmia subtype, and the other documented events were distributed across supraventricular, ventricular, and bradyarrhythmic categories (Table 2 and Table 3; Figure 1).

Electrocardiographic findings. Flattened T waves, prominent U waves, and longer corrected QT intervals were more common in hypokalemia. Peaked T waves were more frequently documented in hyperkalemia. Other ST–T abnormalities also varied across potassium categories, while differences in mean QRS and PR intervals were not statistically significant (Table 3).

Regression analysis. Univariable logistic regression showed imprecise positive associations for hypokalemia, hyperkalemia, and several cardiovascular risk factors. In the penalized multivariable model adjusted for age, cardiovascular disease, and estimated glomerular filtration rate, neither potassium category was independently associated with a statistically significant increase in documented arrhythmias. All adjusted confidence intervals included unity (Table 4; Figure 2).

Sensitivity analyses and hospital outcomes. The direction of the potassium-category associations was broadly consistent across the sensitivity analyses, although estimates remained imprecise. ICU transfer and potassium-directed treatment were more frequent in the hyperkalemia and/or hypokalemia groups; in-hospital mortality did not differ significantly across potassium categories. These outcomes and monitoring differences are detailed in Table 5.

Table 1. Baseline clinical characteristics by serum potassium category

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

Characteristic	Overall (n=500)	Reference (n=429)	Hypokalemia (n=57)	Hyperkalemia (n=14)	Statistic / test	p-value
Age (years), mean (SD)	53.4 (16.1)	53.3 (16.1)	54.8 (17.2)	52.1 (13.1)	Welch F=0.27	0.769
Male sex	292 (58.4%)	251 (58.5%)	31 (54.4%)	10 (71.4%)	Fisher exact	0.530
BMI (kg/m²), mean (SD)	24.0 (3.9)	24.1 (3.9)	23.8 (3.8)	22.2 (4.6)	Welch F=1.21	0.313
Hypertension	138 (27.6%)	119 (27.7%)	14 (24.6%)	5 (35.7%)	χ^2=0.73	0.695
Type 2 diabetes mellitus	104 (20.8%)	81 (18.9%)	22 (38.6%)	1 (7.1%)	Fisher exact	0.002
Gastrointestinal illness/fluid loss	51 (10.2%)	36 (8.4%)	15 (26.3%)	0 (0.0%)	Fisher exact	<0.001
Coronary artery disease	53 (10.6%)	45 (10.5%)	8 (14.0%)	0 (0.0%)	Fisher exact	0.364
Congestive heart failure	50 (10.0%)	44 (10.3%)	6 (10.5%)	0 (0.0%)	Fisher exact	0.660
Chronic kidney disease	37 (7.4%)	34 (7.9%)	1 (1.8%)	2 (14.3%)	Fisher exact	0.093
Acute kidney injury at index	33 (6.6%)	30 (7.0%)	1 (1.8%)	2 (14.3%)	Fisher exact	0.123
Loop diuretic exposure	50 (10.0%)	45 (10.5%)	5 (8.8%)	0 (0.0%)	Fisher exact	0.580
ACE inhibitor exposure	75 (15.0%)	70 (16.3%)	3 (5.3%)	2 (14.3%)	Fisher exact	0.064
Insulin exposure	73 (14.6%)	55 (12.8%)	17 (29.8%)	1 (7.1%)	Fisher exact	0.005
QT-prolonging medication	77 (15.4%)	61 (14.2%)	12 (21.1%)	4 (28.6%)	Fisher exact	0.126
Magnesium (mg/dL), mean (SD)	2.0 (0.3)	2.0 (0.3)	1.7 (0.2)	2.0 (0.3)	Welch F=22.88	<0.001
Creatinine (mg/dL), mean (SD)	1.1 (0.4)	1.1 (0.4)	1.1 (0.4)	1.2 (0.5)	Welch F=0.47	0.627
eGFR (mL/min/1.73 m²), mean (SD)	77.5 (26.2)	77.6 (26.3)	76.9 (24.9)	75.5 (30.1)	Welch F=0.05	0.954
Albumin (g/dL), mean (SD)	3.6 (0.4)	3.6 (0.4)	3.7 (0.4)	3.7 (0.5)	Welch F=0.96	0.396

Values are n (%) or mean (SD). Between-group comparisons used Welch's analysis of variance, Pearson's χ^2 test, or Fisher's exact test as appropriate. BMI was available for 485, magnesium for 488, and albumin for 493 records. ACE, angiotensin-converting enzyme; eGFR, estimated glomerular filtration rate.

Table 2. Potassium abnormality severity and primary arrhythmia outcome — Panel A: Potassium classification

Potassium category	Severity subgroup	Patients, n	Percentage of total (%)
Reference	3.5–5.4 mmol/L	429	85.8
Hypokalemia	Mild (3.0–3.4)	43	8.6
Hypokalemia	Moderate (2.5–2.9)	11	2.2
Hypokalemia	Severe (<2.5)	3	0.6
Hyperkalemia	Mild (5.5–5.9)	10	2.0
Hyperkalemia	Moderate (6.0–6.4)	3	0.6
Hyperkalemia	Severe (≥6.5)	1	0.2

Table 2 (continued). Panel B: Incident arrhythmias by serum potassium category

Potassium group	Patients	Arrhythmias n (%)	Risk (95% CI), %	Risk diff. vs ref.	Unadjusted OR (95% CI)	Fisher p
Reference	429	12 (2.8%)	2.8 (1.6–4.8)	Reference	Reference	—
Hypokalemia	57	3 (5.3%)	5.3 (1.8–14.4)	+2.5 pp	1.93 (0.53–7.06)	0.402
Hyperkalemia	14	1 (7.1%)	7.1 (1.3–31.5)	+4.3 pp	2.67 (0.32–22.12)	0.345
Overall	500	16 (3.2%)	—	—	—	0.257*

*CI, confidence interval; pp, percentage points. Pairwise p-values compare each abnormal potassium group with the reference group using Fisher's exact test; *overall Fisher's exact test for the 2×3 contingency table. Odds ratios use the reference potassium group as comparator.*

Table 3. Arrhythmia subtypes and admission ECG characteristics — Panel A: First documented arrhythmia subtype

Arrhythmia subtype	Reference	Hypokalemia	Hyperkalemia	Total
Atrial fibrillation	1	3	1	5
Atrial flutter	1	0	0	1
Supraventricular tachycardia	3	0	0	3
Nonsustained ventricular tachycardia	2	0	0	2
Sustained ventricular tachycardia	2	0	0	2
Ventricular fibrillation	1	0	0	1
Clinically significant bradyarrhythmia	2	0	0	2
High-grade atrioventricular block	0	0	0	0

Counts represent the first qualifying arrhythmia event for each affected record; total events n=16. Subtype-specific inferential tests were not undertaken because of small event numbers.

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

Table 3 (continued). Panel B: Baseline electrocardiographic findings

ECG characteristic	Overall	Reference	Hypokalemia	Hyperkalemia	Statistic / test	p-value
Peaked T waves	9 (1.8%)	5 (1.2%)	2 (3.5%)	2 (14.3%)	Fisher exact	0.009
Flattened T waves	23 (4.6%)	8 (1.9%)	15 (26.3%)	0 (0.0%)	Fisher exact	<0.001
Prominent U waves	21 (4.2%)	7 (1.6%)	13 (22.8%)	1 (7.1%)	Fisher exact	<0.001
ST–T abnormalities	72 (14.4%)	54 (12.6%)	14 (24.6%)	4 (28.6%)	Fisher exact	0.013
Conduction abnormality	30 (6.0%)	25 (5.8%)	2 (3.5%)	3 (21.4%)	Fisher exact	0.057
QRS duration (ms), mean (SD)	95.3 (12.6)	94.9 (12.6)	97.0 (12.9)	101.1 (11.8)	Welch F=2.30	0.118
QTc interval (ms), mean (SD)	434.9 (26.3)	432.0 (25.2)	455.8 (25.7)	437.6 (24.9)	Welch F=21.12	<0.001
PR interval (ms), mean (SD)	159.3 (19.7)	159.1 (19.7)	159.6 (20.4)	162.1 (19.4)	Welch F=0.16	0.851

Values are n (%) or mean (SD). Fisher's exact p-values are for exact 2×3 tests; Welch's F is reported for continuous measures. QTc was documented in 495 records. Isolated ECG morphologic abnormalities were not considered incident arrhythmias.

Table 4. Univariable and penalized multivariable regression for incident cardiac arrhythmia

Candidate predictor	Unadjusted OR (95% CI)	Wald z	p-value	Adjusted OR (95% CI)	Wald z	p-value
Hypokalemia vs reference	1.93 (0.53–7.06)	0.99	0.320	2.10 (0.62–7.08)	1.19	0.232
Hyperkalemia vs reference	2.67 (0.32–22.12)	0.91	0.362	3.89 (0.65–23.43)	1.48	0.138
Age, per 10 years	0.97 (0.71–1.33)	−0.17	0.865	0.91 (0.64–1.29)	−0.55	0.584
Cardiovascular disease	1.48 (0.47–4.69)	0.66	0.506	1.68 (0.54–5.21)	0.90	0.369
eGFR, per 10 mL/min/1.73 m²	0.95 (0.79–1.16)	−0.48	0.634	0.94 (0.76–1.15)	−0.62	0.537
Low serum magnesium (<1.7 mg/dL)	1.04 (0.29–3.71)	0.05	0.957	—	—	—
QT-prolonging medication	2.60 (0.88–7.71)	1.73	0.085	—	—	—
Previous arrhythmia history	2.63 (0.72–9.64)	1.46	0.144	—	—	—
Sepsis on admission	2.00 (0.55–7.27)	1.06	0.291	—	—	—
Loop diuretic exposure	1.30 (0.29–5.88)	0.34	0.736	—	—	—

The adjusted model used Jeffreys-penalized logistic regression because of sparse outcome events (16/500) and included potassium category, age, cardiovascular disease, and eGFR. Adjusted 95% CIs and p-values are approximate penalized-Wald estimates. Low-magnesium univariable analysis used 488 records; other univariable and all adjusted analyses used 500. Unadjusted p-values are Wald tests; they may differ from the Fisher exact comparisons in Table 2.

Table 5. Sensitivity analyses and hospital course — Panel A: Sensitivity analyses

Analysis	Exposure	N	Events	Adjusted OR (95% CI)	p-value
Any potassium abnormality vs reference	Any abnormality	500	16	2.08 (0.65–6.67)	0.216
More complete rhythm surveillance	Hypokalemia	341	13	1.33 (0.28–6.34)	0.724
More complete rhythm surveillance	Hyperkalemia	341	13	2.36 (0.27–20.65)	0.439
Excluding eGFR <30	Hypokalemia	487	15	2.17 (0.58–8.05)	0.249
Excluding eGFR <30	Hyperkalemia	487	15	2.89 (0.34–24.57)	0.332
Borderline-high K (5.1–5.4 mmol/L)	Borderline-high	429	12	0.85 (0.10–6.91)	0.877

Sensitivity analyses used conventional logistic regression adjusted for age, cardiovascular disease, and eGFR; these estimates are distinct from the penalized principal model. Borderline-high potassium was compared with

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

3.5–5.0 mmol/L.

Table 5 (continued). Panel B: Clinical course and rhythm monitoring across potassium categories

Variable	Overall (n=500)	Reference (n=429)	Hypokalemia (n=57)	Hyperkalemia (n=14)	Test	p-value
ICU transfer	45 (9.0%)	36 (8.4%)	5 (8.8%)	4 (28.6%)	Fisher exact	0.049
In-hospital mortality	5 (1.0%)	4 (0.9%)	1 (1.8%)	0 (0.0%)	Fisher exact	0.537
Potassium correction given	77 (15.4%)	54 (12.6%)	19 (33.3%)	4 (28.6%)	Fisher exact	<0.001
Repeat ECG performed	327 (65.4%)	274 (63.9%)	40 (70.2%)	13 (92.9%)	Fisher exact	0.048
Telemetry monitoring used	164 (32.8%)	137 (31.9%)	23 (40.4%)	4 (28.6%)	Fisher exact	0.451

Values are n (%). P-values are exact 2×3 Fisher tests across potassium categories. Correction and ICU transfer were evaluated as post-index clinical events.

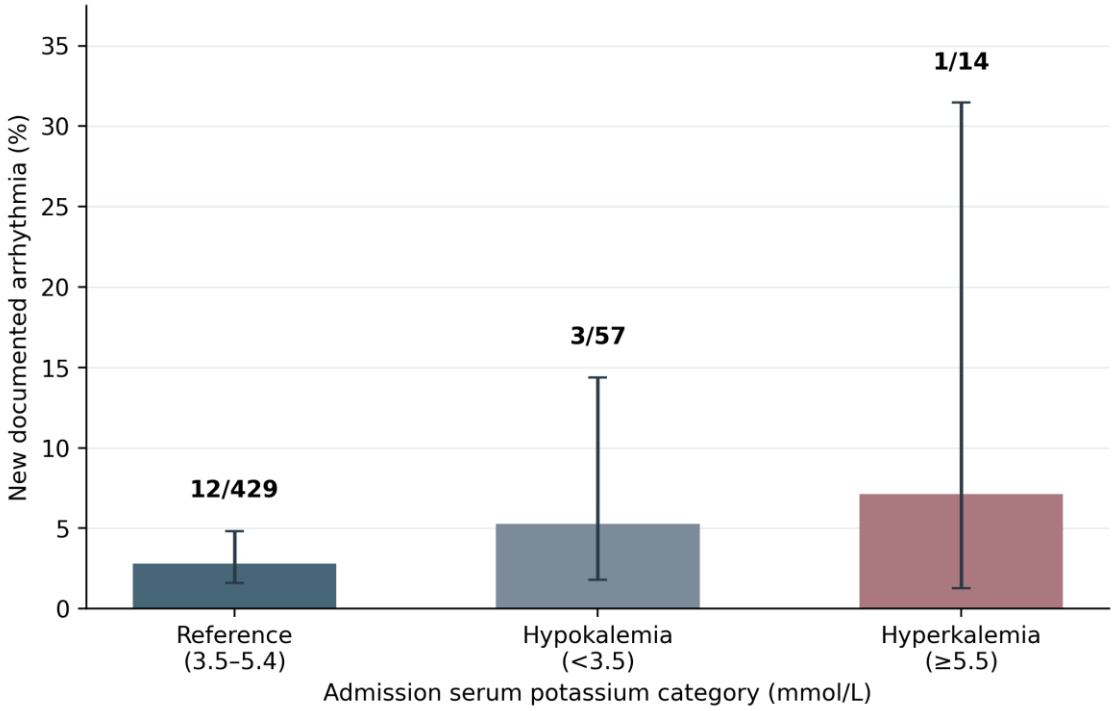

Figure 1. New documented cardiac arrhythmias according to admission serum potassium category.

Bars show the proportion of affected patients; error bars represent Wilson 95% confidence intervals. Numerators and denominators are displayed above the bars.

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

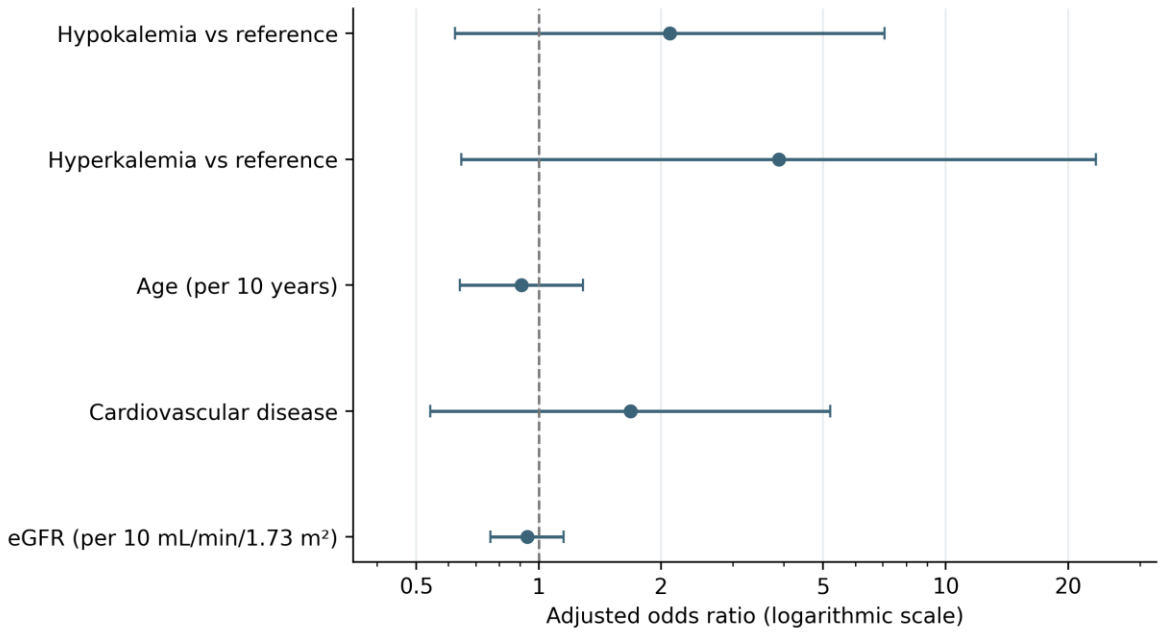

Figure 2. Adjusted associations of serum potassium category and selected baseline characteristics with incident arrhythmia.

Symbols indicate adjusted odds ratios from the penalized logistic model; horizontal lines indicate approximate 95% confidence intervals. The vertical dashed line denotes an odds ratio of 1.

Discussion

This study of 500 medical inpatient encounters found that clinically significant arrhythmias were present in 3.2% within 72 hours. The hypokalemia and hyperkalemia groups had higher event rates than the potassium in the reference range group, but these rates were not statistically significant after adjustment for age, cardiovascular disease, and renal function. Potassium-related electrocardiographic changes, however, were clearly present: hypokalemia was associated with T-wave flattening, prominent U waves and longer QTc intervals, while peaked T waves were more common with hyperkalemia. The results of this study indicate that dyskalemia can have electrophysiological effects that can be recognized, although it is not easily demonstrated as an independent association with short-term clinically documented arrhythmia.

The direction of the hypokalemia association is the same as in the large ICU analysis by Phillips et al. [9]. In 20,665 cardiac ICU admissions, potassium levels of 3.0–3.4 mEq/L were linked to a higher use of antiarrhythmic treatment than potassium levels of 4.0–5.0 mEq/L (HR 1.23, 95% CI 1.01–1.51). This was also true of the medical and surgical ICUs, where there was a similar association with 69,714 medical and surgical ICU admissions (HR 1.26, 95% CI 1.09–1.45); potassium levels in the range of 3.5–3.9 mEq/L were not significantly different from the reference range. Most of the hypokalemia in our cohort was mild, and only 14 patients had potassium levels below 3.0 mmol/L, which could account for the weaker association.

Not all hospital-based evidence supports the aggressive normalization of potassium levels for the sole purpose of preventing arrhythmia. Harkness et al. [10] studied 1,338 hypokalemic patient-days in telemetry monitored patients who had neither acute coronary syndrome nor a prior history of arrhythmia. There were only 22 arrhythmic events, representing 1.6% of patient-days. The rate of arrhythmias was 1.0% of uncorrected versus 2.6% of successfully corrected patient-days, and multivariable analysis did not show a significant protective effect of correction. This low absolute event frequency is similar to our results and highlights the potential for challenges in determining clinically meaningful differences in non-highly selected cardiovascular populations.

There is a greater association in cohorts with a higher prevalence of ventricular arrhythmic disease, however. Laslett et al. [11] found a difference in the LYTE-VT study between 140 admissions with sustained ventricular tachycardia or fibrillation and 140 heart failure controls. Hypokalemia was present in 35.7% of VT/VF cases compared to 12.9% of controls; severe hypokalemia was present in 13.6% of VT/VF cases compared to 2.7% of controls. This stronger association compared to our study may be due to the fact that the cardiac population was selected and that the potassium disturbances were more severe in the arrhythmic cases.

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

Our results are similar to those of the multicentre study by Kildegaard et al. [12] which comprised 79,599 patients in the emergency department. Around 40% of hypokalemic patients had at least one ECG abnormality potentially related to potassium; T-wave flattening, ST depression and QTc prolongation occurred in 27%, 16% and 14%, respectively. Higher crude risks of ICU admission and ventricular arrhythmia or cardiac arrest were associated with hypokalemia, but the predictive value of individual ECG abnormalities was limited for short-term outcomes. Likewise, the hypokalemic group had significantly more flattened T waves, U waves, and an approximately 24-ms longer mean QTc, but clinically significant arrhythmias were still rare.

The highest numerical arrhythmia rate was seen in hyperkalemia, but only 14 encounters were exposed, leading to a wide adjusted confidence interval. Montague et al. [13] showed that ECG changes are not a sufficient indicator of risk. Of 90 patients with potassium ≥6.0 mmol/L, 48% had renal failure and 80% had potassium <7.2 mmol/L. The incidence of ECG changes was related to the potassium level, but ECG interpretation was not sensitive enough and no reliable threshold for the T-wave to diagnose hyperkalemia could be set. The increased prevalence of peaked T waves in hyperkalemia that we found is therefore physiologically plausible, but should not be considered a reliable indicator of arrhythmic risk.

In dialysis populations, the role of coexisting electrolyte and physiological factors is further highlighted. Edwards et al. [14] followed 60 patients with a history of hyperkalemia on hemodialysis for a period of time. Twenty-nine patients had hyperkalemia, 11 had bradycardia, and 50 had ventricular ectopy, with neither bradycardia nor ventricular ectopy being clearly related to measured electrolyte levels. These observations highlight that arrhythmogenesis is not just a single serum potassium measurement.

Likewise, Roy-Chaudhury et al. [15] used implantable loop recorders in 66 hemodialysis patients and observed 1,678 clinically significant events in 44 patients during six months. The majority of events were bradycardias (1,461 episodes), 14 were asystolic events, and only one sustained VT; 41% of participants had AF. The frequency of the events was different depending on the dialysis cycle, but potassium was not the most important biochemical predictor. These more general data confirm the concept of dyskalemia as a part of an arrhythmogenic substrate that also encompasses underlying cardiac disease, renal dysfunction, magnesium status, medication exposure, acute illness, and monitoring intensity.

A few drawbacks should be noted. There were only 16 arrhythmic events and hyperkalemia was rare, which reduced the precision of the statistics and resulted in large confidence intervals even with penalized regression. Severe dyskalemia was not well assessed as most potassium abnormalities were mild. The rhythm surveillance was not consistent, and so some events may have been missed if they were asymptomatic or transient. The risk may also have been modified by potassium correction after the index measurement. However, the results of the sensitivity analyses and the distinct potassium-related ECG patterns support the biological plausibility of the observed associations.

Conclusion

The ECG changes were associated with potassium abnormalities, and the short-term arrhythmia rates were higher, but neither hypokalemia nor hyperkalemia was independently associated with documented arrhythmia after adjustment. Results indicate that potassium should be considered in the context of other clinical and electrophysiologic parameters, and not as a standalone predictor of inpatient arrhythmic events.

References

1. Weiss JN, Qu Z, Shivkumar K. Electrophysiology of hypokalemia and hyperkalemia. *CircArrhythmElectrophysiol.* 2017;10(3). **doi:10.1161/CIRCEP.116.004667.**
2. Jensen HK, Brabrand M, Vinholt PJ, Hallas J, Lassen AT. Hypokalemia in acute medical patients: risk factors and prognosis. *Am J Med.* 2015;128(1):60–67.e1. **doi:10.1016/j.amjmed.2014.07.022.**
3. Conway R, Creagh D, Byrne DG, O'Riordan D, Silke B. Serum potassium levels as an outcome determinant in acute medical admissions. *Clin Med (Lond).* 2015;15(3):239–243. **doi:10.7861/clinmedicine.15-3-239.**
4. Thongprayoon C, Cheungpasitporn W, Hansrivijit P, et al. Admission serum potassium levels in hospitalized patients and one-year mortality. *Medicines (Basel).* 2020;7(1):2. **doi:10.3390/medicines7010002.**
5. Goyal A, Spertus JA, Gosch K, et al. Serum potassium levels and mortality in acute myocardial infarction. *JAMA.* 2012;307(2):157–164. **doi:10.1001/jama.2011.1967.**
6. Faxén J, Xu H, Evans M, Jernberg T, Szummer K, Carrero JJ. Potassium levels and risk of in-hospital arrhythmias and mortality in patients admitted with suspected acute coronary syndrome. *Int J Cardiol.* 2019;274:52–58. **doi:10.1016/j.ijcard.2018.09.099.**
7. Krijthe BP, Heeringa J, Kors JA, Hofman A, Franco OH, Witteman JCM, Stricker BHC. Serum potassium levels and the risk of atrial fibrillation:

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

the Rotterdam Study. *Int J Cardiol.* 2013;168(6):5411–5415. **doi:10.1016/j.ijcard.2013.08.048.**
8. Montford JR, Linas S. How dangerous is hyperkalemia? *J Am Soc Nephrol.* 2017;28(11):3155–3165. **doi:10.1681/ASN.2016121344.**
9. Phillips CT, Wang J, Celi LA, Zhang Z, Feng M. Association of hypokalemia with an increased risk for medically treated arrhythmias. *PLoS One.* 2019;14(6). **doi:10.1371/journal.pone.0217432.**
10. Harkness W, Watts P, Kopstein M, Dziadkowiec O, Hicks G, Scherbak D. Correcting hypokalemia in hospitalized patients does not decrease risk of cardiac arrhythmias. *Adv Med.* 2019;2019:4919707. **doi:10.1155/2019/4919707.**
11. Laslett DB, Cooper JM, Greenberg RM, et al. Electrolyte abnormalities in patients presenting with ventricular arrhythmia (from the LYTE-VT Study). *Am J Cardiol.* 2020;129:36–41. **doi:10.1016/j.amjcard.2020.04.051.**
12. Kildegaard H, Brabrand M, Forberg JL, Platonov P, Lassen AT, Ekelund U. Prevalence and prognostic value of electrocardiographic abnormalities in hypokalemia: a multicenter cohort study. *J Intern Med.* 2024;295(4):544–556. **doi:10.1111/joim.13757.**
13. Montague BT, Ouellette JR, Buller GK. Retrospective review of the frequency of ECG changes in hyperkalemia. *Clin J Am Soc Nephrol.* 2008;3(2):324–330. **doi:10.2215/CJN.04611007.**
14. Edwards JC, Mosman A, Hauptman PJ, et al. Arrhythmia in chronic hemodialysis as a function of predialysis electrolytes and interdialytic interval. *Hemodial Int.* 2023;27(1):45–54. **doi:10.1111/hdi.13057.**
15. Roy-Chaudhury P, Tumlin JA, Koplan BA, et al. Primary outcomes of the Monitoring in Dialysis Study indicate that clinically significant arrhythmias are common in hemodialysis patients and related to dialytic cycle. *Kidney Int.* 2018;93(4):941–951. **doi:10.1016/j.kint.2017.11.019.**

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.