



Proarrhythmic Electrocardiographic Abnormalities in Chronic Kidney Disease Stages 3–5: An Observational Study

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ABSTRACT:

Background: Chronic kidney disease (CKD) is associated with increased cardiovascular morbidity, mortality, and sudden cardiac death. Disturbances in cardiac electrical repolarization contribute to arrhythmogenesis, but data on proarrhythmic electrocardiographic (ECG) abnormalities across CKD stages are limited. **Objectives:** To assess the prevalence and pattern of proarrhythmic ECG abnormalities in patients with CKD stages 3–5 and evaluate their association with disease severity. **Methods:** This hospital-based cross-sectional study included 160 adults with CKD stages 3, 4, and 5. Patients with structural heart disease, reduced left ventricular systolic function, prior arrhythmias, or use of QT-prolonging drugs were excluded. Standard 12-lead ECGs were analyzed for corrected QT interval (QTc), QTc dispersion, P-wave dispersion, Tp–e interval, and Tp–e/QTc ratio. Statistical significance was set at $p < 0.05$. **Results:** The study population showed a male predominance, with half of the participants in CKD stage 5. All ECG parameters increased significantly with advancing CKD stage. Mean QTc, QTc dispersion, P-wave dispersion, Tp–e interval, and Tp–e/QTc ratio were significantly higher in stages 4 and 5 than in stage 3 ($p < 0.001$). The prevalence of abnormal ECG findings also rose progressively with worsening renal function. **Conclusion:** Proarrhythmic ECG abnormalities are common in CKD and increase with disease severity. Comprehensive ECG assessment may serve as a simple, non-invasive tool for identifying CKD patients at increased risk of arrhythmias and sudden cardiac death.

MAIN POINTS

- Proarrhythmic ECG abnormalities are common in CKD stages 3–5, even in patients without structural heart disease or prior arrhythmias.
- Advancing CKD severity is associated with progressive worsening of ECG markers, including QTc interval,

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QTc dispersion, P-wave dispersion, Tp-e interval, and Tp-e/QTc ratio.

- Patients with CKD stages 4 and 5 showed significantly higher values of all proarrhythmic ECG parameters compared with those in stage 3 ($p < 0.001$).
- The prevalence of abnormal ECG findings increased with declining renal function, suggesting greater electrical instability and arrhythmic susceptibility in advanced CKD.
- Comprehensive ECG assessment may serve as a simple, non-invasive method for arrhythmic risk stratification in patients with moderate to advanced CKD.

INTRODUCTION:

Chronic kidney disease (CKD) is a major global health problem and is associated with a disproportionately high burden of cardiovascular morbidity and mortality (1,2). Cardiovascular disease accounts for a substantial proportion of deaths among patients with CKD, with sudden cardiac death representing one of the leading causes, particularly in advanced stages of renal dysfunction (3). Importantly, the excess cardiovascular risk in CKD cannot be fully explained by traditional risk factors or by structural heart disease alone, suggesting that CKD-specific mechanisms contribute significantly to arrhythmogenesis.

CKD is increasingly recognized as a proarrhythmic condition. Patients with impaired renal function exhibit a higher prevalence of both atrial and ventricular arrhythmias compared to the general population, even in the absence of overt coronary artery disease or significant systolic dysfunction (4). Consensus statements and observational studies have emphasized that declining kidney function is closely associated with abnormalities in cardiac electrical conduction and repolarization, which predispose patients to malignant arrhythmias and sudden cardiac death (5,6).

Electrocardiography remains a simple, widely available, and cost-effective tool for the assessment of cardiac electrical activity. Among conventional ECG markers, prolongation of the corrected QT (QTc) interval has long been associated with ventricular arrhythmias and adverse cardiovascular outcomes. In patients with CKD, acquired long QT syndrome is commonly observed and has been shown to correlate with worsening renal function and increased mortality risk (7–9). However, QTc duration alone reflects global repolarization delay and does not adequately capture the regional heterogeneity of repolarization that underlies arrhythmogenesis.

To address this limitation, greater attention has been directed toward ECG markers that reflect dispersion of repolarization. QTc dispersion, defined as the difference between the maximum and minimum QTc intervals across ECG leads, serves as an indicator of spatial heterogeneity of ventricular repolarization. Increased QTc dispersion has been associated with ventricular arrhythmias and sudden cardiac death in patients with CKD and those undergoing dialysis, independent of QTc prolongation itself (10,11).

Atrial electrical abnormalities also contribute significantly to arrhythmic risk in CKD. P-wave dispersion reflects inhomogeneous atrial conduction and has been associated with an increased risk of atrial fibrillation, an arrhythmia that is highly prevalent in CKD and contributes to stroke, heart failure, and mortality (12–14). Structural and functional cardiac changes commonly seen in CKD, including left ventricular hypertrophy, atrial enlargement, and myocardial fibrosis, promote atrial electrical remodeling and conduction delay, thereby increasing atrial arrhythmic susceptibility (15,16).

More recently, markers of transmural ventricular repolarization have gained prominence in arrhythmic risk assessment. The interval from the peak to the end of the T wave (Tp-e) reflects dispersion of repolarization across myocardial layers and has been shown to be associated with ventricular tachyarrhythmias and sudden cardiac death (17,18). The Tp-e/QTc ratio, which adjusts Tp-e for overall QT duration, has been proposed as a more stable and heart rate-independent marker of ventricular electrical instability, particularly in clinical settings such as CKD where both QT prolongation and repolarization heterogeneity coexist (17).

Multiple interrelated mechanisms contribute to these electrocardiographic abnormalities in CKD. Electrolyte disturbances, accumulation of uremic toxins, autonomic imbalance, chronic volume overload, anemia, and hypertension collectively promote myocardial fibrosis, ion channel dysfunction, and heterogeneity of electrical conduction (6,15,16). These pathophysiological processes intensify with advancing renal dysfunction, providing a biological basis for the progressive increase in proarrhythmic ECG abnormalities across CKD stages.

Despite increasing recognition of these associations, studies evaluating a comprehensive panel of proarrhythmic

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ECG markers across different stages of CKD remain limited, particularly in the Indian population. Most available studies focus on isolated ECG parameters or are restricted to dialysis populations, limiting their applicability across the spectrum of CKD (19,20). Given the rising burden of CKD in India and its cardiovascular complications, systematic evaluation of both conventional and advanced ECG markers across CKD stages is clinically relevant.

Therefore, the present study was undertaken to evaluate the prevalence and pattern of proarrhythmic electrocardiographic abnormalities—including QTc interval, QTc dispersion, P-wave dispersion, Tp-e interval, and Tp-e/QTc ratio—in patients with CKD stages 3–5, and to assess their association with disease severity.

MATERIAL AND METHODS:

Study Design and Setting: This hospital-based cross-sectional analytical study was conducted over 18 months in tertiary care teaching hospital.

Study Population and Sampling: Adult patients diagnosed with chronic kidney disease (CKD) stage 3 or higher who attended the General Medicine Department during the study period were screened for eligibility. CKD staging was performed according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines based on estimated glomerular filtration rate. Eligible participants were recruited by convenience sampling until the required sample size was achieved.

The sample size was calculated to be 160 patients based on data reported by Yehia et al. Using a mean corrected QT interval (QTc) of 479.6 ms, a standard deviation of 41.11 ms, a precision of 10 ms, and a 95% confidence level, the sample size was estimated using the formula for estimation of a mean.

Patients aged 18 years and above with CKD stages 3, 4, or 5, including those receiving maintenance hemodialysis, were included in the study. Patients receiving medications known to prolong the QT interval or precipitate arrhythmias, those with structural heart disease, impaired left ventricular systolic function (ejection fraction <50%), previously documented arrhythmias, or acute illnesses that could influence electrocardiographic parameters were excluded.

Data Collection and Electrocardiographic Assessment: After obtaining written informed consent, demographic and clinical data, including age, sex, CKD stage, comorbidities, and relevant laboratory parameters related to renal and electrolyte status, were recorded using a structured proforma.

A standard 12-lead electrocardiogram (ECG) was obtained from all participants in the resting supine position using a calibrated ECG machine. ECG recordings were analyzed manually using standardized techniques. The evaluated parameters included corrected QT interval (QTc), calculated using Bazett's formula; QTc dispersion, defined as the difference between maximum and minimum QTc values across ECG leads; P-wave dispersion, defined as the difference between maximum and minimum P-wave duration; Tp-e interval, measured from the peak to the end of the T wave; and the Tp-e/QTc ratio.

QTc prolongation was defined as >450 ms in males and >460 ms in females. QTc dispersion >50 ms, P-wave dispersion >40 ms, Tp-e interval >100 ms, and Tp-e/QTc ratio >0.25 were considered abnormal according to established literature.

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee of Aarupadai Veedu Medical College and Hospital. Written informed consent was obtained from all participants before enrollment, and the study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using statistical software. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were expressed as frequency and percentage.

Comparisons of continuous ECG parameters across CKD stages were performed using one-way ANOVA or Kruskal–Wallis test, as appropriate. Associations between categorical variables were analyzed using the Chi-square test or Fisher's exact test. A p-value <0.05 was considered statistically significant.

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Normality was assessed using the Shapiro–Wilk test. Cases with incomplete ECG data were excluded from final analysis.

Bias control: Selection bias was minimized by enrolling consecutive eligible patients during the study period. Measurement bias was reduced by standardized ECG acquisition under resting conditions. ECG measurements were performed using predefined criteria.

RESULTS:

A total of 160 patients with chronic kidney disease stages 3–5 were included in the analysis. The study population predominantly comprised individuals aged 41–60 years, with the largest proportion in the 51–60 years age group (27.5%), and showed a male predominance (66.9%). Half of the cohort consisted of patients with CKD stage 5, while stages 3 and 4 accounted for 25% each (Table 1). The mean corrected QT interval (QTc) increased significantly with advancing CKD stage, from 443.75 ± 21.61 ms in stage 3 to 471.18 ± 14.20 ms in stage 4 and 471.50 ± 26.40 ms in stage 5 ($p < 0.001$) (Table 2). QTc prolongation was observed in 59.4% of patients overall and was significantly more frequent in advanced CKD stages, with 82.5% of stage 4 and 68.8% of stage 5 patients exhibiting prolonged QTc compared to 17.5% in stage 3 ($p < 0.001$) (Table 3). Repolarization dispersion parameters, including QTc dispersion, P-wave dispersion, Tp–e interval, and Tp–e/QTc ratio, demonstrated a statistically significant progressive increase across CKD stages ($p < 0.001$ for all), with the highest mean values observed in stage 5 disease (Table 4). Correspondingly, the prevalence of abnormal ECG markers increased with CKD severity; high-risk QTc dispersion was present in 65.0% of stage 5 patients, abnormal P-wave dispersion in 60.0%, prolonged Tp–e interval in 56.3%, and high-risk Tp–e/QTc ratio in 85.0%, all showing significant associations with CKD stage ($p < 0.001$) (Table 5). Overall, a consistent and statistically significant trend of increasing proarrhythmic electrocardiographic abnormalities was observed with advancing CKD stage (Table 6).

Table 1. Baseline demographic and clinical characteristics of the study population (n = 160)

Variable	Category	n (%)
Age group (years)	18–30	9 (5.6)
	31–40	26 (16.3)
	41–50	43 (26.9)
	51–60	44 (27.5)
	61–70	33 (20.6)
	71–80	5 (3.1)
Sex	Male	107 (66.9)
	Female	53 (33.1)
CKD stage	Stage 3	40 (25.0)
	Stage 4	40 (25.0)
	Stage 5	80 (50.0)

Table 2. Comparison of corrected QT interval (QTc) across CKD stages

CKD Stage	Mean $\pm$ SD (ms)	Minimum–Maximum (ms)
Stage 3	443.75 ± 21.61	408–486
Stage 4	471.18 ± 14.20	419–495
Stage 5	471.50 ± 26.40	410–520
p-value	<0.001	

Table 3. Distribution of QTc prolongation across CKD stages

QTc classification	Stage 3 n (%)	Stage 4 n (%)	Stage 5 n (%)	Total n (%)
Normal QTc	33 (82.5)	7 (17.5)	25 (31.3)	65 (40.6)
Prolonged QTc	7 (17.5)	33 (82.5)	55 (68.8)	95 (59.4)
p-value	<0.001			

Table 4. Comparison of repolarization dispersion parameters across CKD stages

Parameter	Stage 3	Stage 4	Stage 5	p-value
QTc dispersion (ms)	46.08 ± 17.83	67.05 ± 19.02	72.38 ± 29.01	<0.001
P-wave dispersion (ms)	31.82 ± 12.50	42.35 ± 12.79	44.61 ± 14.25	<0.001
Tp–e interval (ms)	90.72 ± 10.89	103.05 ± 8.99	102.33 ± 11.96	<0.001
Tp–e/QTc ratio	0.21 ± 0.02	0.24 ± 0.02	0.25 ± 0.04	<0.001

Table 5. Distribution of abnormal ECG markers across CKD stages

ECG parameter	Stage 3 n (%)	Stage 4 n (%)	Stage 5 n (%)	Total n (%)	p-value
High-risk QTc dispersion	8 (20.0)	23 (57.5)	52 (65.0)	83 (51.9)	<0.001

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Abnormal P-wave dispersion	9 (22.5)	22 (55.0)	48 (60.0)	79 (49.4)	<0.001
Prolonged Tp-e interval	7 (17.5)	19 (47.5)	45 (56.3)	71 (44.4)	<0.001
High-risk Tp-e/QTc ratio	25 (62.5)	40 (100)	68 (85.0)	133 (83.1)	<0.001

Table 6. Summary of electrocardiographic parameters indicating increasing arrhythmic risk with advancing CKD stage

ECG marker	Observed trend across CKD stages	Statistical significance
Corrected QT interval (QTc)	Increase from 443.75 ms to 471.50 ms	p < 0.001
QTc dispersion	Increase from 46.08 ms to 72.38 ms	p < 0.001
P-wave dispersion	Increase from 31.82 ms to 44.61 ms	p < 0.001
Tp-e interval	Increase from 90.72 ms to 102.33 ms	p < 0.001
Tp-e/QTc ratio	Increase from 0.21 to 0.25	p < 0.001

DISCUSSION:

The present study included 160 patients with CKD stages 3–5, with a predominance of males (66.9%) and a majority of patients in the middle-aged to elderly groups. This demographic pattern mirrors observations from large epidemiological and hospital-based CKD studies, where male sex and advancing age are consistently associated with higher CKD prevalence and progression (1,2,19). The high proportion of CKD stage 5 patients (50%) reflects the tertiary care referral pattern of the study setting and is comparable to Indian hospital-based studies that have reported a similar predominance of advanced CKD among admitted patients (19,20). This distribution provided an appropriate framework to examine stage-wise electrocardiographic changes associated with worsening renal function.

In the present study, the mean QTc interval showed a significant increase with advancing CKD stage, rising from 443.75 ± 21.61 ms in stage 3 to 471.50 ± 26.40 ms in stage 5 (Table 2). QTc prolongation was observed in 59.4% of the total cohort and was significantly more frequent in CKD stages 4 and 5 compared to stage 3 (Table 3). Liu et al. reported that acquired long QT syndrome is common in CKD and is closely related to declining renal function, although specific stage-wise QTc values were not uniformly reported (7). In a large population-based analysis, Malik et al. demonstrated that prolonged QTc was independently associated with mortality in individuals with reduced kidney function, supporting the clinical significance of QTc prolongation observed in the present study (8). Similarly, Deo et al. reported that longer QT intervals were associated with increased mortality risk among older adults with CKD, reinforcing the relevance of the observed QTc prolongation in advanced stages (9).

QTc dispersion increased significantly across CKD stages in the present study, from 46.08 ± 17.83 ms in stage 3 to 72.38 ± 29.01 ms in stage 5 (Table 4). High-risk QTc dispersion was present in 65.0% of stage 5 patients compared to 20.0% in stage 3 (Table 5). Narayan et al. demonstrated that increased QT dispersion is associated with ventricular arrhythmias in CKD patients, emphasizing its role as a marker of repolarization heterogeneity (11). Genovesi et al. also highlighted the association between prolonged QT parameters and sudden death in hemodialysis patients, although dispersion values varied depending on clinical context (10). The marked increase in QTc dispersion observed in advanced CKD in the present study is consistent with the concept that worsening renal dysfunction is associated with increasing spatial heterogeneity of ventricular repolarization.

The present study demonstrated a significant increase in P-wave dispersion with advancing CKD stage, with mean values rising from 31.82 ± 12.50 ms in stage 3 to 44.61 ± 14.25 ms in stage 5 (Table 4). Abnormal P-wave dispersion was observed in 60.0% of CKD stage 5 patients (Table 5). Although most large CKD epidemiological studies have focused on atrial fibrillation incidence rather than direct P-wave dispersion measurements, CKD has been consistently shown to be associated with atrial electrical and structural remodeling (12–14). Wiggins et al. and Alonso et al. reported higher rates of incident atrial fibrillation in patients with reduced kidney function, suggesting underlying atrial conduction abnormalities (12,13). The increased P-wave dispersion observed in the present study likely reflects this atrial electrical instability associated with progressive CKD.

The Tp-e interval increased significantly with CKD severity in the present study, from 90.72 ± 10.89 ms in stage 3 to 102.33 ± 11.96 ms in stage 5 (Table 4). Prolonged Tp-e interval was observed in more than half of CKD stage 5 patients (56.3%) (Table 5). Çiçek et al. demonstrated that patients with CKD had significantly prolonged Tp-e intervals compared to controls and that increased Tp-e was associated with a higher risk of ventricular arrhythmias, although exact stage-wise values varied (17). Suzuki et al. similarly reported that prolonged Tp-e interval was associated with ventricular arrhythmias in CKD patients (18). The progressive increase in Tp-e interval observed in the present study supports the role of transmural repolarization abnormalities in the arrhythmogenic substrate of advanced CKD.

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In the present study, the Tp-e/QTc ratio increased significantly from 0.21 ± 0.02 in CKD stage 3 to 0.25 ± 0.04 in stage 5 (Table 4), with high-risk ratios observed in 85.0% of stage 5 patients (Table 5). Çiçek et al. highlighted the utility of the Tp-e/QTc ratio as a relatively stable index of ventricular repolarization heterogeneity in CKD, demonstrating higher values in patients at increased arrhythmic risk (17). Although absolute cut-off values vary across studies, the consistently higher Tp-e/QTc ratios in advanced CKD observed in the present study align with previously reported associations between elevated Tp-e/QTc and ventricular arrhythmias.

All evaluated electrocardiographic markers—QTc interval, QTc dispersion, P-wave dispersion, Tp-e interval, and Tp-e/QTc ratio—demonstrated a statistically significant and progressive increase from CKD stage 3 to stage 5. This uniform pattern supports the concept that CKD progression is associated with cumulative electrical remodeling of the myocardium, driven by electrolyte disturbances, uremic toxins, autonomic dysfunction, and structural cardiac changes (4–6,15,16).

The findings of the present study reinforce existing evidence that CKD is a proarrhythmic state and highlight the value of comprehensive ECG assessment across CKD stages for identifying patients at increased arrhythmic risk.

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