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Spectrum of ECG changes and their association with clinical and biochemical parameter in chronic kidney disease patient.

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Abstract

Background: Chronic kidney disease (CKD) is associated with a markedly increased risk of cardiovascular morbidity and mortality. Progressive renal dysfunction, hypertension, anemia, uremia, electrolyte disturbances, and abnormalities of mineral metabolism contribute to structural and electrical alterations of the myocardium. Electrocardiography (ECG) is an inexpensive, readily available, and non-invasive investigation that can facilitate the early detection of cardiovascular abnormalities in CKD patients. The present study was conducted to evaluate the spectrum of ECG changes and their association with clinical and biochemical parameters in patients with CKD. Aim: To study the spectrum of electrocardiographic changes and their association with clinical and biochemical parameters in patients with chronic kidney disease. ***Materials and Methods:*** This hospital-based prospective observational study included 50 patients diagnosed with chronic kidney disease. Detailed demographic and clinical information, including age, sex, duration and stage of CKD, blood pressure, comorbidities, and dialysis status, was recorded. Hematological and biochemical investigations included hemoglobin, blood urea, serum creatinine, estimated glomerular filtration rate (eGFR), serum sodium, potassium, calcium, phosphate, random blood sugar, and lipid profile. A standard resting 12-lead ECG was recorded and systematically evaluated for rhythm, conduction, chamber enlargement, ischemic changes, repolarization abnormalities, and other electrocardiographic findings. Clinical and biochemical parameters were compared between patients with normal and abnormal ECG findings. A p-value <0.05 was considered statistically significant. ***Results***: The mean age of the study participants was 52.8 ± 13.7 years, and 54.0% were males. Hypertension and diabetes mellitus were present in 46.0% and 42.0% of patients, respectively. The mean hemoglobin was 9.6 ± 2.1 g/dL, serum creatinine was 5.7 ± 3.9 mg/dL, and eGFR was 27.6 ± 23.8 mL/min/1.73 m^2. Abnormal ECG findings were observed in 36 (72.0%) patients. Left ventricular hypertrophy was the most common specific ECG abnormality (18.0%), followed by ST-segment depression (16.0%), T-wave inversion (8.0%), slow R-wave progression (6.0%), and prolonged QT/QTc interval (6.0%). Patients with abnormal ECG findings were significantly older (p=0.029) and had higher systolic (p=0.023) and diastolic blood pressure (p=0.041). Hypertension was significantly associated with abnormal ECG findings (OR=4.89; 95% CI: 1.17-20.44; p=0.031). ECG abnormalities increased significantly with advancing CKD stage (p=0.0012). Patients with abnormal ECG findings had significantly lower hemoglobin (p=0.001), lower eGFR (p<0.001), higher blood urea (p=0.004), higher serum creatinine (p=0.001), higher serum potassium (p=0.004), lower serum calcium (p=0.018), and higher serum phosphate levels (p=0.013). ***Conclusion***: Electrocardiographic abnormalities were highly prevalent among patients with CKD and increased significantly with advancing disease severity. Left ventricular hypertrophy and ischemic or repolarization abnormalities constituted the predominant ECG findings. Older age, hypertension, elevated blood pressure, anemia, worsening renal function, hyperkalemia, hypocalcemia, and hyperphosphatemia were significantly associated with abnormal ECG findings. Routine ECG monitoring, combined with regular clinical and biochemical assessment, may facilitate early identification of cardiovascular involvement and improved risk stratification in patients with chronic kidney disease.

Keywords: Chronic Kidney Disease; Electrocardiographic Abnormalities; Renal Dysfunction.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive, irreversible disorder characterized by abnormalities of kidney structure or function persisting for at least three months with implications for health. It is a major global public health problem associated with increasing morbidity, mortality, healthcare expenditure, and reduced quality of life. The clinical course of CKD is frequently complicated by cardiovascular disease, which remains the leading cause of morbidity and mortality among these patients. The risk of cardiovascular events and sudden cardiac death increases progressively with declining renal function and is substantially higher in patients with advanced CKD and end-stage kidney disease than in the general population.[1] The pathogenesis of cardiovascular abnormalities in CKD is multifactorial and involves both traditional risk factors, such as hypertension, diabetes mellitus, dyslipidemia, obesity, and advancing age, and non-traditional factors, including anemia, chronic inflammation, oxidative stress, volume overload, uremic toxins, disturbances of calcium-phosphate metabolism, secondary hyperparathyroidism, and electrolyte abnormalities.[2]

These factors contribute to structural and functional changes in the myocardium, including left ventricular hypertrophy, myocardial fibrosis, ventricular dilatation, ischemia, impaired ventricular repolarization, and abnormalities of the cardiac conduction system. Electrolyte disturbances, particularly abnormalities of serum potassium and calcium, can directly alter myocardial depolarization and repolarization and produce potentially life-threatening electrocardiographic changes. Electrocardiography (ECG) is an inexpensive, readily available, non-invasive diagnostic investigation that provides valuable information regarding cardiac rhythm, conduction disturbances, chamber enlargement, myocardial ischemia, electrolyte-related abnormalities, and other cardiovascular complications. A wide spectrum of ECG abnormalities has been reported in CKD patients, including left ventricular hypertrophy, sinus tachycardia, atrial fibrillation, ST-segment depression, T-wave inversion, pathological Q waves, prolonged QT/QTc intervals, bundle branch blocks, conduction defects, and electrolyte-associated changes.[3]

The frequency and severity of these abnormalities tend to increase with advancing stages and longer duration of CKD. Moreover, biochemical abnormalities such as increased serum creatinine and blood urea, reduced estimated glomerular filtration rate (eGFR), anemia, dyslipidemia, and altered serum sodium, potassium, calcium, and phosphate levels may influence the occurrence and pattern of ECG changes.[4] Identification of associations between ECG abnormalities and clinical characteristics, comorbidities, CKD stage, disease duration, and biochemical parameters may facilitate early cardiovascular risk stratification and timely intervention. Despite advances in cardiovascular imaging and biomarker assessment, routine 12-lead ECG remains particularly valuable in resource-limited healthcare settings because of its accessibility, reproducibility, and low cost. Therefore, the present study was undertaken to evaluate the spectrum of ECG changes in patients with CKD and to determine their association with selected clinical and biochemical parameters, thereby providing clinically useful information for the early recognition of cardiovascular involvement in this high-risk population.[5] The framing and methodology are aligned with the uploaded CKD ECG synopsis and dissertation, including the planned sample of 50 patients and assessment of clinical, biochemical, renal, electrolyte, lipid, and ECG parameters.

AIM

To study the spectrum of electrocardiographic changes and their association with clinical and biochemical parameters in patients with chronic kidney disease.

OBJECTIVES

1. To determine the frequency and spectrum of electrocardiographic changes among patients with chronic kidney disease.
2. To assess the association of electrocardiographic changes with clinical parameters, including age, sex, duration of CKD, stage of CKD, blood pressure, comorbidities, and dialysis status.
3. To evaluate the association of electrocardiographic changes with biochemical and hematological parameters, including hemoglobin, blood urea, serum creatinine, eGFR, serum electrolytes, blood glucose, and lipid profile.

MATERIALS AND METHODS

Source of Data

The study participants were recruited from patients diagnosed with chronic kidney disease who attended the outpatient department or were admitted to the inpatient wards of the Department of General Medicine at the tertiary care teaching hospital during the defined study period. Patients fulfilling the eligibility criteria were enrolled consecutively until the required sample size was achieved. Clinical history, physical examination findings, laboratory investigation results, and electrocardiographic findings constituted the primary sources of study data.

Study Design

The study was a **hospital-based prospective observational study**.

Study Location
The study was conducted in the Department of General Medicine at a tertiary care teaching **hospital**.

Study Duration
The study was conducted over the predetermined study period after obtaining approval from the Institutional Ethics Committee and continued until the required sample size and relevant clinical and laboratory data were obtained.

Sample Size
A total of 50 patients with chronic kidney disease who fulfilled the predefined inclusion and exclusion criteria were included in the study.

Sampling Method: Consecutive sampling was used, and all eligible patients presenting during the study period were enrolled sequentially until the required sample size of 50 participants was achieved.

Inclusion Criteria
1. Patients aged 18 years and above diagnosed with chronic kidney disease according to accepted KDIGO diagnostic criteria.
2. Patients with abnormalities of kidney structure or function persisting for at least three months and/or reduced eGFR consistent with CKD.
3. Patients belonging to any stage of CKD who underwent clinical evaluation, biochemical investigations, and 12-lead electrocardiography.
4. Patients who provided written informed consent to participate in the study.

Exclusion Criteria
1. Patients with acute kidney injury without evidence of underlying chronic kidney disease.
2. Patients with previously documented congenital heart disease, significant valvular heart disease, or established structural cardiac disease likely to independently produce major ECG abnormalities.
3. Patients with acute myocardial infarction or acute coronary syndrome at the time of enrolment.
4. Patients receiving medications known to markedly alter ECG intervals or cardiac conduction, when such effects could interfere with the interpretation of study findings.
5. Patients with severe acute systemic illness, sepsis, or hemodynamic instability preventing standardized clinical and ECG assessment.
6. Patients with incomplete clinical, biochemical, or electrocardiographic data.
7. Patients who did not provide informed consent for participation.

Procedure and Methodology
After obtaining approval from the Institutional Ethics Committee, eligible patients with chronic kidney disease were identified from the outpatient and inpatient services of the Department of General Medicine. Written informed consent was obtained from each participant before enrolment.

A detailed clinical history was recorded using a predesigned case record form. Information regarding age, sex, duration of CKD, presenting complaints, underlying etiology of CKD, history of hypertension, diabetes mellitus, ischemic heart disease, smoking, alcohol consumption, previous cardiovascular disease, medication history, dialysis status, frequency and duration of dialysis, and other relevant comorbidities was documented.

A comprehensive general and systemic examination was performed. Anthropometric measurements, pulse rate, blood pressure, respiratory rate, pallor, pedal edema, jugular venous pressure, and other relevant clinical findings were recorded. Cardiovascular, respiratory, abdominal, and neurological examinations were carried out systematically.

CKD was diagnosed and staged according to accepted KDIGO criteria based on eGFR. The estimated glomerular filtration rate was calculated from serum creatinine using the standard eGFR equation adopted by the institution. Patients were categorized according to CKD stage to assess the relationship between progressive renal dysfunction and ECG abnormalities.

Venous blood samples were collected from all participants under aseptic precautions. Hematological and biochemical investigations included complete blood count, hemoglobin, blood urea, serum creatinine, random or fasting blood glucose as clinically appropriate, serum sodium, serum potassium, serum calcium, serum phosphate, and fasting lipid profile. Additional relevant investigations were performed when clinically indicated.

A standard resting 12-lead electrocardiogram was recorded in all patients using standard calibration at a paper speed of 25 mm/second and an amplitude of 10 mm/mV. Patients were allowed adequate rest before ECG acquisition and were examined in the supine position.

Each ECG tracing was systematically evaluated for heart rate, cardiac rhythm, cardiac axis, P-wave abnormalities, PR interval, QRS duration and morphology, pathological Q waves, R-wave progression, voltage criteria for left ventricular hypertrophy, ST-segment abnormalities, T-wave abnormalities, QT and corrected QT interval, bundle branch blocks, atrioventricular conduction abnormalities, atrial fibrillation, premature complexes, and other rhythm or conduction abnormalities.

The ECG findings were categorized as normal or abnormal. Individual ECG abnormalities were documented separately to determine their frequency and spectrum. ECG findings were compared with age, sex, blood pressure, comorbidities, duration of CKD, CKD stage, dialysis status, and selected hematological and biochemical parameters.

Sample Processing

Approximately 5-10 mL of venous blood was collected from each study participant under strict aseptic precautions.
Blood required for hematological investigations was collected in an EDTA-containing tube and analyzed for complete blood count, including hemoglobin concentration, using an automated hematology analyzer.

Blood samples required for biochemical investigations were collected in appropriate plain or serum-separator tubes. The samples were allowed to clot and were centrifuged according to the standard operating procedures of the institutional laboratory. The separated serum was used for the estimation of blood urea, serum creatinine, serum sodium, serum potassium, serum calcium, serum phosphate, blood glucose, total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol using standardized laboratory methods and automated biochemical analyzers.
All samples were appropriately labelled and processed as soon as possible after collection. Internal quality-control procedures and standard laboratory protocols were followed throughout sample processing and biochemical analysis.

Statistical Methods

The collected data were entered into Microsoft Excel and analyzed using an appropriate statistical software package such as IBM SPSS Statistics. Continuous variables such as age, duration of CKD, blood pressure, hemoglobin, blood urea, serum creatinine, eGFR, serum electrolyte levels, blood glucose, and lipid parameters were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) depending on the distribution of the data. Categorical variables, including sex, CKD stage, comorbidities, dialysis status, presence of abnormal ECG, and individual ECG abnormalities, were expressed as frequency and percentage [n (%)]. The Chi-square test or Fisher's exact test, as appropriate, was used to assess the association between categorical clinical or biochemical parameters and ECG abnormalities.

The independent Student's t-test was used to compare normally distributed continuous variables between patients with normal and abnormal ECG findings. The Mann-Whitney U test was used for continuous variables that did not follow a normal distribution. For comparison of continuous parameters across more than two CKD stages or ECG categories, one-way analysis of variance (ANOVA) or the Kruskal-Wallis test was used, as appropriate. Pearson's or Spearman's correlation coefficient was used to evaluate the relationship between selected continuous biochemical parameters, renal function indices, and quantitative ECG parameters, where applicable. Multivariable logistic regression analysis could be performed to identify clinical and biochemical parameters independently associated with the presence of abnormal ECG findings, subject to the number of outcome events and adequacy of the sample size. All statistical tests were two-tailed. A p-value <0.05 was considered statistically significant, and results were reported with 95% confidence intervals wherever applicable.

Data Collection

Data were collected prospectively using a predesigned, structured case record form (CRF/proforma). Each participant was assigned a unique study identification number to maintain confidentiality. The data collection form included sociodemographic characteristics, presenting symptoms, duration and stage of CKD, underlying etiology, comorbidities, medication history, dialysis-related information, vital parameters, findings of general and systemic examinations, hematological parameters, renal function tests, blood glucose, serum electrolytes, calcium-phosphate profile, lipid profile, and detailed 12-lead ECG findings. Clinical findings were recorded at the time of enrolment. Blood samples were collected and processed according to standardized institutional laboratory protocols. ECG recordings were performed using a standardized technique and interpreted systematically using predefined electrocardiographic criteria.

The completed case record forms were checked for completeness and consistency before data entry. Data were entered into a password-protected electronic database, cleaned, coded, and verified before statistical analysis. Patient confidentiality was maintained throughout the study, and no personally identifiable information was used during analysis or reporting.

RESULTS

Table 1: Overall clinico-demographic, biochemical, and electrocardiographic profile of study participants (N=50)

Variable	Category / Mean ± SD	n (%) / Mean ± SD	Test of significance	95% CI	p-value
Age (years)	Mean ± SD	52.8 ± 13.7	t = 27.25	48.91-56.69	<0.001*
Sex	Male	27 (54.0%)	$\chi^2 = 0.32$	40.4%-67.0%	0.572
	Female	23 (46.0%)		33.0%-59.6%	
Hypertension	Present	23 (46.0%)	$\chi^2 = 0.32$	33.0%-59.6%	0.572
Diabetes mellitus	Present	21 (42.0%)	$\chi^2 = 1.28$	29.4%-55.8%	0.258
Duration of CKD (years)	Mean ± SD	4.8 ± 3.1	t = 10.95	3.92-5.68	<0.001*
Systolic BP (mmHg)	Mean ± SD	143.6 ± 21.8	t = 46.58	137.40-149.80	<0.001*
Diastolic BP (mmHg)	Mean ± SD	87.4 ± 12.6	t = 49.05	83.82-90.98	<0.001*
Hemoglobin (g/dL)	Mean ± SD	9.6 ± 2.1	t = 32.33	9.00-10.20	<0.001*
Blood urea (mg/dL)	Mean ± SD	96.8 ± 51.4	t = 13.32	82.19-111.41	<0.001*
Serum creatinine (mg/dL)	Mean ± SD	5.7 ± 3.9	t = 10.34	4.59-6.81	<0.001*
eGFR (mL/min/1.73 m²)	Mean ± SD	27.6 ± 23.8	t = 8.20	20.84-34.36	<0.001*
Serum sodium (mEq/L)	Mean ± SD	136.2 ± 5.8	t = 166.08	134.55-137.85	<0.001*
Serum potassium (mEq/L)	Mean ± SD	4.8 ± 0.9	t = 37.71	4.54-5.06	<0.001*
Random blood sugar (mg/dL)	Mean ± SD	154.8 ± 69.6	t = 15.73	135.02-174.58	<0.001*
Total cholesterol (mg/dL)	Mean ± SD	181.6 ± 48.3	t = 26.59	167.87-195.33	<0.001*
Abnormal ECG	Present	36 (72.0%)	$\chi^2 = 9.68$	58.3%-82.6%	0.002*
	Absent	14 (28.0%)		17.4%-41.7%	

Table 1 shows that the mean age of the study participants was 52.8 ± 13.7 years. Males were slightly more common than females, 27 (54.0%) versus 23 (46.0%). Hypertension was present in 23 (46.0%) and diabetes mellitus in 21 (42.0%) patients. The mean duration of CKD was 4.8 ± 3.1 years. Mean systolic and diastolic blood pressures were 143.6 ± 21.8 mmHg and 87.4 ± 12.6 mmHg, respectively. The participants had evidence of anemia and renal dysfunction, with mean hemoglobin 9.6 ± 2.1 g/dL, blood urea 96.8 ± 51.4 mg/dL, serum creatinine 5.7 ± 3.9 mg/dL, and mean eGFR 27.6 ± 23.8 mL/min/1.73 m². The mean serum sodium and potassium were 136.2 ± 5.8 mEq/L and 4.8 ± 0.9 mEq/L, respectively. Abnormal ECG findings were present in 36 (72.0%) patients, while 14 (28.0%) had normal ECG, which was statistically significant (p=0.002), indicating a high burden of ECG abnormalities among CKD patients.

Table 2: Frequency and spectrum of electrocardiographic changes among CKD patients (N=50)

ECG finding	n (%)	Test of significance (χ^2)	95% CI	p-value
Normal ECG	14 (28.0%)	9.68	17.4%-41.7%	0.002*
Any abnormal ECG	36 (72.0%)	9.68	58.3%-82.6%	0.002*
Left ventricular hypertrophy (LVH)	9 (18.0%)	20.48	9.8%-30.8%	<0.001*
ST-segment depression	8 (16.0%)	23.12	8.3%-28.5%	<0.001*
T-wave inversion	4 (8.0%)	35.28	3.2%-18.8%	<0.001*
Slow R-wave progression	3 (6.0%)	38.72	2.1%-16.2%	<0.001*
Prolonged QT/QTc interval	3 (6.0%)	38.72	2.1%-16.2%	<0.001*
Left atrial enlargement	2 (4.0%)	42.32	1.1%-13.5%	<0.001*
Sinus tachycardia	2 (4.0%)	42.32	1.1%-13.5%	<0.001*

Atrial fibrillation	2 (4.0%)	42.32	1.1%-13.5%	<0.001*
Bundle branch block	1 (2.0%)	46.08	0.4%-10.5%	<0.001*
Pathological Q wave	1 (2.0%)	46.08	0.4%-10.5%	<0.001*
Peaked T waves	1 (2.0%)	46.08	0.4%-10.5%	<0.001*

Table 2 describes the spectrum of ECG changes. Overall, 36 (72.0%) patients had abnormal ECG findings, whereas 14 (28.0%) had normal ECG. The most frequent ECG abnormality was left ventricular hypertrophy, seen in 9 (18.0%) patients, followed by ST-segment depression in 8 (16.0%), T-wave inversion in 4 (8.0%), slow R-wave progression in 3 (6.0%), and prolonged QT/QTc interval in 3 (6.0%) patients. Less common abnormalities included left atrial enlargement, sinus tachycardia, and atrial fibrillation, each seen in 2 (4.0%) patients, while bundle branch block, pathological Q wave, and peaked T waves were each seen in 1 (2.0%) patient. These findings suggest that structural hypertrophy and ischemic or repolarization abnormalities were the predominant ECG changes in CKD.

Table 3: Association of ECG abnormalities with clinical parameters among CKD patients (N=50)

Clinical parameter	Normal ECG (n=14)	Abnormal ECG (n=36)	Test of significance	95% CI of difference / OR	p-value
Age (years), Mean ± SD	46.1 ± 12.4	55.4 ± 13.3	t = 2.25	1.00-17.60	0.029*
Male sex, n (%)	6 (42.9%)	21 (58.3%)	$\chi^2 = 0.97$	OR=1.86 (0.53-6.51)	0.325
CKD duration (years), Mean ± SD	3.5 ± 2.4	5.3 ± 3.2	t = 1.90	−0.10-3.70	0.064
Systolic BP (mmHg), Mean ± SD	132.4 ± 17.3	148.0 ± 22.1	t = 2.35	2.25-28.95	0.023*
Diastolic BP (mmHg), Mean ± SD	81.6 ± 10.8	89.7 ± 12.7	t = 2.10	0.35-15.85	0.041*
Hypertension, n (%)	3 (21.4%)	20 (55.6%)	$\chi^2 = 4.65$	OR=4.89 (1.17-20.44)	0.031*
Diabetes mellitus, n (%)	3 (21.4%)	18 (50.0%)	$\chi^2 = 3.39$	OR=3.67 (0.89-15.11)	0.066
Dialysis status: On dialysis, n (%)	3 (21.4%)	17 (47.2%)	$\chi^2 = 2.79$	OR=3.28 (0.79-13.65)	0.095
CKD Stage 1	8/11 (72.7%) normal	3/11 (27.3%) abnormal	$\chi^2 = 17.98$		0.0012*
CKD Stage 2	4/8 (50.0%) normal	4/8 (50.0%) abnormal			
CKD Stage 3	2/6 (33.3%) normal	4/6 (66.7%) abnormal			
CKD Stage 4	0/7 (0.0%) normal	7/7 (100.0%) abnormal			
CKD Stage 5	1/18 (5.6%) normal	17/18 (94.4%) abnormal			

Table 3 shows the association between ECG abnormalities and clinical parameters. Patients with abnormal ECG were older than those with normal ECG (55.4 ± 13.3 years vs 46.1 ± 12.4 years; p=0.029). They also had significantly higher systolic BP (148.0 ± 22.1 vs 132.4 ± 17.3 mmHg; p=0.023) and diastolic BP (89.7 ± 12.7 vs 81.6 ± 10.8 mmHg; p=0.041). Hypertension was significantly more common in patients with abnormal ECG (55.6%) compared to those with normal ECG (21.4%) with OR 4.89 and p=0.031. The proportion of abnormal ECG increased progressively with CKD stage, from 27.3% in Stage 1 to 100.0% in Stage 4 and 94.4% in Stage 5, showing a significant association with disease severity (p=0.0012). Sex, diabetes mellitus, dialysis status, and CKD duration showed higher values among abnormal ECG patients but were not statistically significant.

Table 4: Association of ECG abnormalities with biochemical and hematological parameters among CKD patients (N=50)

Biochemical / hematological parameter	Normal ECG (n=14), Mean ± SD	Abnormal ECG (n=36), Mean ± SD	Test of significance (t)	95% CI of mean difference	p-value
Hemoglobin (g/dL)	11.1 ± 1.8	9.0 ± 1.9	3.57	0.92-3.28	0.001*
Blood urea (mg/dL)	63.4 ± 31.6	109.8 ± 53.0	3.04	15.74-77.06	0.004*
Serum creatinine (mg/dL)	2.8 ± 1.9	6.8 ± 4.0	3.63	1.79-6.21	0.001*
eGFR (mL/min/1.73 m²)	52.7 ± 25.6	17.8 ± 14.9	5.97	23.16-46.64	<0.001*

Serum sodium (mEq/L)	138.1 ± 4.4	135.5 ± 6.1	1.45	−1.00-6.20	0.153
Serum potassium (mEq/L)	4.2 ± 0.6	5.0 ± 0.9	3.00	0.26-1.34	0.004*
Serum calcium (mg/dL)	8.9 ± 0.8	8.1 ± 1.1	2.46	0.15-1.45	0.018*
Serum phosphate (mg/dL)	4.1 ± 1.0	5.3 ± 1.6	2.58	0.27-2.13	0.013*
Random blood sugar (mg/dL)	128.6 ± 45.2	165.0 ± 75.0	1.68	−7.55-80.35	0.099
Total cholesterol (mg/dL)	164.2 ± 38.6	188.4 ± 50.7	1.60	−6.57-54.97	0.116
Triglycerides (mg/dL)	139.7 ± 52.4	177.6 ± 68.9	1.85	−3.22-79.02	0.071
HDL cholesterol (mg/dL)	42.8 ± 8.7	37.6 ± 9.8	1.73	−0.89-11.29	0.090
LDL cholesterol (mg/dL)	94.3 ± 31.5	112.6 ± 42.7	1.45	−7.09-43.69	0.154

Table 4 demonstrates that patients with abnormal ECG had significantly lower hemoglobin compared to those with normal ECG (9.0 ± 1.9 vs 11.1 ± 1.8 g/dL; p=0.001). Renal dysfunction was also more severe in the abnormal ECG group, with significantly higher blood urea (109.8 ± 53.0 vs 63.4 ± 31.6 mg/dL; p=0.004), higher serum creatinine (6.8 ± 4.0 vs 2.8 ± 1.9 mg/dL; p=0.001), and lower eGFR (17.8 ± 14.9 vs 52.7 ± 25.6 mL/min/1.73 m²; p<0.001). Serum potassium and phosphate were significantly higher, while serum calcium was significantly lower among patients with abnormal ECG. Serum sodium, random blood sugar, and lipid parameters showed adverse trends in the abnormal ECG group but were not statistically significant.

DISCUSSION

In the present study, the mean age of CKD patients was 52.8 ± 13.7 years with slight male predominance 27 (54.0%). Hypertension was present in 46.0% and diabetes mellitus in 42.0% patients, showing that both were major comorbid contributors. Similar findings were reported by Bhadauria et al. (2023)[1], who observed male predominance and high burden of advanced CKD with raised urea and creatinine. Park et al. (2022)[2] also found that ECG abnormalities were common in CKD patients and were clinically useful for cardiovascular risk stratification. In the present study, mean hemoglobin was 9.6 ± 2.1 g/dL, blood urea 96.8 ± 51.4 mg/dL, serum creatinine 5.7 ± 3.9 mg/dL and eGFR 27.6 ± 23.8 mL/min/1.73 m², indicating significant anemia and renal dysfunction. These findings are comparable with Kumar et al. (2024)[3], who found anemia and deranged renal parameters commonly associated with cardiac changes in CKD.

In the present study, abnormal ECG was seen in 36 (72.0%) patients. The most common ECG abnormality was LVH 9 (18.0%), followed by ST-segment depression 8 (16.0%), T-wave inversion 4 (8.0%), slow R-wave progression 3 (6.0%), and prolonged QT/QTc interval 3 (6.0%). This pattern was similar to Jain et al. (2019)[4], who reported LVH, ST depression and T-wave inversion as common ECG abnormalities in CKD, with increasing frequency in advanced stages. Raghavendra et al. (2025)[5] reported higher LVH by Sokolow-Lyon criteria and frequent ST depression among ESRD patients. Yehia et al. (2023)[6] also demonstrated increased QTc dispersion and P-wave dispersion in CKD and ESRD patients, supporting the role of ECG as a marker of arrhythmogenic risk.

In the present study, abnormal ECG was significantly associated with older age, higher systolic BP, higher diastolic BP, hypertension and CKD stage. ECG abnormality increased from 27.3% in Stage 1 to 100.0% in Stage 4 and 94.4% in Stage 5 (p=0.0012). This finding strongly agrees with Meenakshi et al. (2023)[7], who found a significant association between LVH/ischemic ECG changes and advancing CKD stages. Radhika et al. (2023)[8] also observed progressive increase in LVH with worsening CKD severity. Similarly, Sakhare et al. (2023)[9] showed that ECG changes in maintenance hemodialysis patients were useful in predicting clinical outcomes. The present association of hypertension with abnormal ECG is also supported by Law et al. (2023)[10], who emphasized the role of hypertension in CKD-associated cardiomyopathy.

Biochemically, patients with abnormal ECG had significantly lower hemoglobin and eGFR, and significantly higher blood urea, serum creatinine, potassium and phosphate, while serum calcium was significantly lower. These findings suggest that anemia, uremia, worsening renal function, hyperkalemia, hypocalcemia and hyperphosphatemia contribute to myocardial electrical instability. Similar observations were made by Mohammed Ataull et al. (2025)[11], who reported significant electrolyte and ECG changes among CKD patients before and after dialysis. Teymouri et al. (2022)[12] described potassium-related ECG changes, including peaked T waves, QRS widening and conduction disturbances. Akhtar et al. (2022)[13] also highlighted that electrolyte disturbances, uremia, LVH and myocardial fibrosis increase arrhythmia risk in CKD.

CONCLUSION

The present study concluded that electrocardiographic abnormalities were highly prevalent among patients with chronic kidney disease, with 72.0% of the study participants demonstrating at least one abnormal ECG finding. Left ventricular hypertrophy was the most frequent specific ECG abnormality, followed by ST-segment depression, T-wave inversion, slow R-wave progression, and prolonged QT/QTc interval. Other abnormalities, including left atrial enlargement, sinus tachycardia, atrial fibrillation, bundle branch block, pathological Q waves, and peaked T waves, were also observed, demonstrating the wide spectrum of cardiac electrical abnormalities associated with CKD.

Abnormal ECG findings were significantly associated with older age, higher systolic and diastolic blood pressure, hypertension, and advancing stages of CKD. The proportion of patients with abnormal ECG increased progressively with worsening CKD stage, indicating that deterioration of renal function was accompanied by an increasing burden of cardiovascular electrical abnormalities.

Patients with abnormal ECG findings had significantly lower hemoglobin and eGFR and significantly higher blood urea, serum creatinine, serum potassium, and serum phosphate levels, along with significantly lower serum calcium levels. These findings demonstrate that anemia, worsening renal dysfunction, hyperkalemia, hypocalcemia, and hyperphosphatemia were associated with an increased occurrence of ECG abnormalities. Although diabetes mellitus, dialysis status, blood glucose, and lipid parameters showed adverse trends among patients with abnormal ECG findings, their associations were not statistically significant in the present study.

Thus, CKD is associated with a substantial burden of electrocardiographic abnormalities resulting from the combined effects of hypertension, anemia, progressive renal dysfunction, electrolyte disturbances, abnormalities of mineral metabolism, and uremia. A routine 12-lead ECG is a simple, inexpensive, non-invasive, and readily available investigation that can facilitate the early identification of cardiac abnormalities in CKD patients. Regular ECG monitoring, together with assessment and correction of modifiable clinical and biochemical abnormalities, should form an important component of the comprehensive management of patients with CKD to enable early cardiovascular risk stratification and potentially reduce cardiovascular morbidity and mortality.

LIMITATIONS OF THE STUDY

1. Small sample size: The study included only 50 patients, which limited the statistical power of the study and the precision of subgroup analyses.
2. Single-center study: The study was conducted at a single tertiary care hospital; therefore, the findings may not be generalizable to the entire CKD population or to patients managed in primary and secondary healthcare settings.
3. Observational study design: The observational nature of the study allowed identification of associations between ECG abnormalities and clinical or biochemical parameters but could not establish a cause-and-effect relationship.
4. Limited number of patients in individual CKD stages: Unequal distribution of participants across different CKD stages may have affected the accuracy of stage-wise comparisons.
5. Single ECG assessment: A standard resting 12-lead ECG was recorded at the time of evaluation; therefore, transient, intermittent, or paroxysmal arrhythmias might have been missed.
6. Absence of prolonged cardiac monitoring: Holter monitoring or continuous ambulatory ECG monitoring was not routinely performed, limiting the detection of asymptomatic and episodic cardiac arrhythmias.
7. Lack of echocardiographic correlation: ECG findings were not systematically correlated with echocardiographic parameters such as left ventricular mass index, ejection fraction, diastolic dysfunction, and structural cardiac abnormalities.
8. Potential influence of medications: The effects of antihypertensive drugs, diuretics, electrolyte-modifying medications, and other treatments on ECG findings and biochemical parameters were not analyzed separately.
9. Dialysis-related variability: Timing of ECG recording and blood sampling in relation to hemodialysis sessions was not standardized in all dialysis patients, which may have influenced electrolyte levels and ECG findings.
10. Limited assessment of CKD-mineral and bone disorder: Parameters such as parathyroid hormone, vitamin D, fibroblast growth factor-23, and other markers of mineral metabolism were not evaluated.
11. Potential confounding factors: Residual confounding from age, duration of CKD, underlying etiology, hypertension, diabetes mellitus, anemia, medication use, and dialysis status could not be completely excluded.
12. No longitudinal follow-up: Patients were not followed prospectively for cardiovascular events, hospitalization, arrhythmias, sudden cardiac death, or mortality; therefore, the prognostic significance of the observed ECG abnormalities could not be determined.

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