Koşuyolu Heart Journal

Koşuyolu Heart J 2025;28(2):47–53
DOI: 10.51645/khj.2025.500

Original Article

Hemodialysis and Risk of Sudden Cardiac Death

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Abstract

Objectives: Chronic kidney disease (CKD) is a significant global health concern affecting millions of individuals worldwide. Cardiac arrhythmias are highly prevalent in CKD patients, with sudden cardiac death (SCD) representing a substantial cause of mortality, accounting for approximately 25% of all deaths in this patient group. Our hypothesis was that hemodialysis (HD) patients have an increased risk of cardiac arrhythmias and SCD.

Methods: This single-center observational study enrolled 219 participants: 109 patients undergoing HD and 110 in the control group. A 12-lead resting electrocardiogram (ECG) was performed before and after dialysis in all patients on HD and once in the control groups.

Results: The T peak-T end (Tp-e) interval duration (p=0.001), Tp-e/QT ratio (p=0.001), and Tp-e/corrected QT (QTc) ratio (p=0.001) were significantly higher in the HD patients than in the control group. There was no significant difference in Tp-e dispersion (p=0.806) between the groups. Correlation analysis revealed significant correlations between parathyroid hormone levels and the Tp-e interval (p=0.001), Tp-e/QT ratio (p=0.017), Tp-e/QTc ratio (p=0.006), and QTc interval (p=0.020) in HD patients. QT, QTc, and Tp-e durations, as well as QT/QRS, QTc/QRS, Tp-e/QT, and Tp-e/QT ratios, were found to be higher in post-dialysis ECGs than in pre-dialysis ECGs.

Conclusion: The resting ECG findings were analyzed in patients with HD and a predisposition to arrhythmias, and SCD was identified in this cohort.

Keywords: Arrhythmias; end-stage renal disease; hemodialysis; sudden cardiac death.

Hemodiyaliz ve Ani Kardiyak Ölüm Riski

Özet

Amaç: Kronik böbrek hastalığı (KBH), dünya çapında milyonlarca bireyi etkileyen önemli bir küresel sağlık sorunudur. Kardiyak aritmiler KBH hastalarında oldukça yaygındır ve ani kardiyak ölüm, bu hasta grubundaki tüm ölümlerin yaklaşık %25'ini oluşturan önemli bir mortalite nedenini temsil etmektedir. Hipotezimiz, hemodiyaliz hastalarında kardiyak aritmi ve ani kardiyak ölüm riskinin artmış olduğudur.

Gereç ve Yöntem: Bu tek merkezli gözlemsel çalışmaya 219 katılımcı dahil edilmiştir: 109 HD uygulanan hasta ve 110 kontrol grubu. HD uygulanan tüm hastalarda diyalizden önce ve sonra, kontrol gruplarında ise bir kez 12 derivasyonlu istirahat EKG'si çekilmiştir.

Bulgular: Tp-e süresi (p=0,001), Tp-e/QT oranı (p=0,001) ve Tp-e/QTc oranı (p=0,001) HD hastalarında kontrol grubuna göre anlamlı derecede yüksekti. Gruplar arasında Tp-e (d) (p=0,806) açısından anlamlı bir fark yoktu. Korelasyon analizi HD hastalarında paratiroid hormon düzeyleri ile Tp-e aralığı (p=0,001), Tp-e/QT oranı (p=0,017), Tp-e/QTc oranı (p=0,006) ve QTc aralığı (p=0,020) arasında anlamlı korelasyonlar olduğunu ortaya koydu. QT, QTc ve Tp-e sürelerinin yanı sıra QT/QRS, QTc/QRS, Tp-e/QT ve Tp-e/QT oranları diyaliz sonrası EKG'lerde diyaliz öncesi EKG'lere göre daha yüksek bulunmuştur.

Sonuç: HD hastalarında istirahat EKG bulguları analiz edilmiş ve bu kohortta aritmilere ve ani kardiyak ölüme yatkınlık tespit edilmiştir.

Anahtar sözcükler: Aritmi; son dönem böbrek hastalığı; hemodializ; ani kardiyak ölüm.

Cite This Article: Mağden K, Baytugan NZ. Hemodialysis and Risk of Sudden Cardiac Death. Koşuyolu Heart J 2025;28(2):47–53

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Submitted: January 16, 2025
Revised: March 04, 2025
Accepted: March 24, 2025
Available Online: August 28, 2025

Introduction

Chronic kidney disease (CKD) is a global health problem that affects millions of people worldwide. Cardiovascular disease is the leading cause of death among hemodialysis (HD) patients, accounting for approximately 43% of the deaths in this patient group.[1] Furthermore, cardiac arrhythmias are common in CKD patients, and sudden cardiac death (SCD) accounts for approximately 25% of all deaths in these patients.[2]

HD is a treatment modality used in patients with CKD. It facilitates the removal of waste products and excess fluid from blood. Previous research has demonstrated that HD can exert a range of effects on the cardiovascular system. One such effect is the impact of HD on cardiac repolarization, which may be evaluated through the analysis of resting electrocardiogram (ECG) parameters. Previous research has demonstrated that HD sessions result in an increase in arrhythmic substrate, as evidenced by alterations in the ECG.[3,4]

Various ECG parameters may be employed to evaluate the risk of arrhythmia and SCD. These include the QT, corrected QT (QTc), T peak-T end (Tp-e) intervals, and Tp-e dispersion [Tp-e (d)].[5] The recently proposed Tp-e/QT ratio is a more accurate measure of ventricular repolarization than QT, QTc, and Tp-e interval.[6] This ratio is unaffected by alterations in heart rate (HR), thus offering a more precise assessment of repolarization.

Only a limited number of studies have evaluated the value of resting 12-lead ECG as a screening tool in HD patients. The objective of the present study was to evaluate ECG parameters to compare arrhythmia substrates in patients undergoing HD.

Materials and Methods

Study Population

This single-center observational study enrolled 219 participants: 109 patients undergoing HD and 110 in the control group. Standard 12-lead resting ECG was performed before and after dialysis in all patients on HD, and transthoracic echocardiography (TTE) was performed in all patients. A detailed physical examination, a history of chronic disease, and medical treatment were performed. Patients with atrial fibrillation, hypertrophic cardiomyopathy, aortic stenosis, left ventricular ejection fraction (EF) <30, and age <18 years were excluded. All participants were informed of the purpose and protocol of the study in detail and were included after informed consent forms were obtained. The study design, protocol, and inclusion criteria are shown in Figure 1. Ethics committee approval for the study was obtained from the local ethics committee on November 14, 2024 (approval number 2024–124). This study was conducted in accordance with the Declaration of Helsinki guidelines.

ECG Examination

The ECGs were recorded at amplitudes of 0.1 mm/mV and speeds of 25 mm/h and 50 mm/h. After scanning, the ECG signals were transmitted to a computer, and the wave voltage of each ECG signal was measured. The QT interval is defined as the temporal period between the initiation of the QRS complex, the conclusion of the T-wave, and the reversion to the isoelectric line.[7] P-wave dispersion (PW-d) was determined by calculating the difference between the maximum and minimum

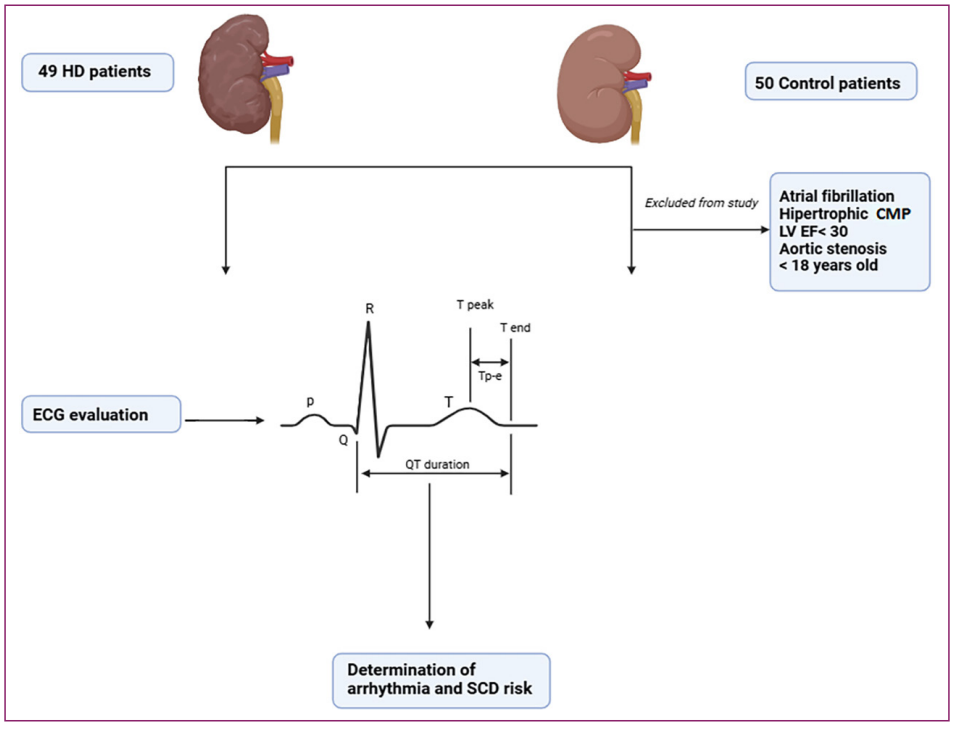

Figure 1. The study design, protocol, and inclusion criteria.

HD: Hemodialysis; CMP: Cardiomyopathy; LV: Left ventricule; EF: Ejection fraction; ECG: Electrocardiogram; SCD: Sudden cardiac death.

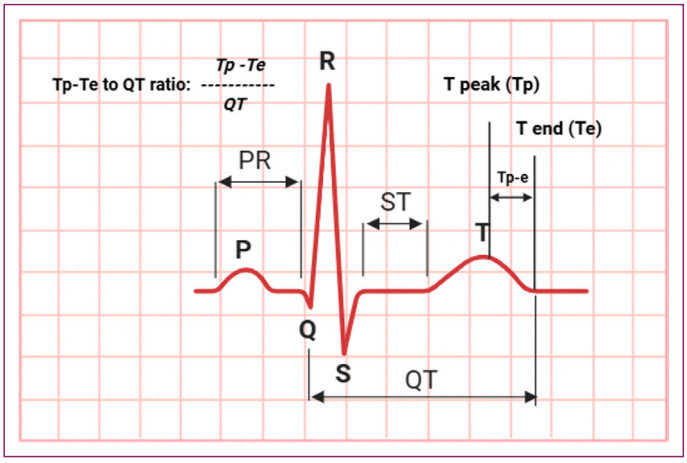

Figure 2. ECG parameters.

QT: QT duration; PR: PR duration.

P-wave durations.[8] The ECG data were analyzed, with particular attention paid to baseline intervals, including HR, QRS duration, PR duration, QT and QTc interval, Tp-e interval, Tp-e (d), Tp-e/QT, and Tp-e/QTc ratio (Fig. 2). The QT interval was measured manually on three occasions per lead using ECG calipers and a magnifying glass to ensure accuracy. The mean values from these measurements were used in the analysis. The QTc interval was calculated using the Bazett formula (QTc = QT/√[RR interval]). The Tp-e interval, which is the time from the peak to the end of the T-wave, was measured using precordial leads. The Tp-e/QT and Tp-e/QTc ratios were calculated as part of the analysis to provide insight into the repolarization phase. These measurements were performed over three consecutive beats to ensure consistency and reliability of the results.[9] For additional analyses, the mean values of these measurements were recorded. The intra- and interobserver variances for the measurements were <3%. TTE imaging was performed in the parasternal and apical planes, and all images were obtained using standard techniques according to current guidelines.[10]

Statistical Analysis

All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) (version 20.0; IBM SPSS Corp., Armonk, NY, USA). The acquired data were assessed for normality using the Kolmogorov–Smirnov test. Continuous variables with normal distribution were expressed as mean± standard deviation, and non-normal distribution as median and interquartile range. Student's t-test was used for two-group evaluations of normally distributed variables, one-way analysis of variance was used for comparisons of three groups and above, and the Games–Howell test was used to determine the group causing the difference. The data were displayed as the median and interquartile range. Pearson's correlation was employed to establish a relationship between the quantitative data. Regression analysis was employed to identify the independent predictors of ECG changes. The Chi-square test was used for comparisons, and the data for categorical variables are presented as percentages. A significance level of p<0.05 was established.

Table 1. The baseline demographic, clinical, and laboratory characteristics of the patients

	Groups				p
	Hemodialysis (n=109)		Control (n=110)		
	n	%	n	%	
Female	38	34.8	43	39	0.222
Age	60.1 (29–88)		62.4 (32–82)		0.457
Smoking	17	15.6	21	19	0.681
DM	34	31.1	23	20.9	0.027
HT	42	38.5	17	363	0.559
HL	18	16.5	15	13.6	0.201
CAD	14	12.8	17	15.4	0.293
Clinical condition					
SBP (mm/Hg)	144±22		125±29		0.021
DBP (mm/Hg)	89±13		82±16		0.043
BSA (m^2)	2.0±0.4		2.1±0.5		0.302
BMI (kg/m^2)	23±2.7		24±3.8		0.207
Laboratory values					
Hgb (g/dL)	10.3±3.1		14.05±2.2		0.001
Hematocrit	29.2 (19.3–43.5)		39.5 (25.5–49.2)		0.001
WBC×10^3 (µL)	6.23 (4.77–14.77)		7.40 (3.87–13.79)		0.550
Plt×10^3 (µL)	148±101		247±112		0.001
Glucose (mg/dL)	118 (70–355)		110 (73–321)		0.321
Creatinine (mg/dL)	5.88±3.9		1.1±0.9		0.001
BUN (mg/dL)	44±20		21.5±10.3		0.001
e-GFR (ml/min/1.73 m^2)	15.6±10.9		72.13±15.4		0.001
PTH (pg/mL)	312.3±51.1		37.2±15.1		0.001
Ca (mg/L)	7.3±2.1		8.8±1.4		0.001
Mg (mEq/L)	1.8±0.3		2.1±0.4		0.275
K (mEq/L)	4.9±0.8		4.0±0.4		0.025
hs-CRP (mg/L)	7.7 (1.1–19.7)		6.8 (0.3–20.1)		0.191
AST (mg/dL)	28±12		25±10		0.603
ALT (mg/dL)	30±9		27±10		0.351
D-dimer (ng/mL)	0.9 (0.3–3.5)		0.8 (0.3–3.2)		0.796
Uric acid (mg/L)	7.2 (2.4–11.9)		3.4 (1.9–8.8)		0.001

DM: Diabetes mellitus; HT: Hypertension; HL: Hyperlipidemia; CAD: Coronary artery disease; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; BSA: Body surface area; BMI: Body mass index; Hgb: Hemoglobine; WBC: White blood cell; Plt: Platelet; BUN: Blood urea nitrogen; e-GFR: estimated glomerular rate; PTH: Parathyroid hormone; Ca: Calcium; Mg: Magnesium; K: Potassium; hs-CRP: High sensitivity C-reactive protein; AST: Aspartate transaminase; ALT: Alanine aminotransferase.

Results

The study was conducted with 219 participants, consisting of 109 HD patients; 71 men (65.2%), 38 women (34.8%) and 110 control group; 67 men (61%) and 43 women (39%) with mean age of 61.2 years (range: 29–88) were included in the study. The baseline parameters of the study population are demonstrated in Table 1. The prevalence of diabetes mellitus was more common in the HD group (p=0.027). Age, smoking status, coronary artery disease, hyperlipidemia, body mass index, and body surface area did not differ between the groups (Table 1). Serum high sensitivity C-reactive protein (hs-CRP), white blood count, D-dimer, magnesium, aspartate transaminase, alanine aminotransferase, and fasting glucose levels were found to be similar in both groups. Serum creat-

Table 2. ECG parameters according to groups

	Hemodialysis (n=109)		Control (n=110)	p
	Pre-dialysis	Post-dialysis		
Heart rate	76±16	75±17	70±12	0.061
PW-d	43.7±14.4	49.3±15.5	34.4±10.1	0.001
PR (msn)	163±16	162±14	150±16	0.001
QRS (msn)	94±9	94±10	92±10	0.551
QT (msn)	380.7±33.2	388.6±42.3	371.3±28.8	0.176
QTc (msn)	409.5±28.6	418.3±34.2	385.6±21.1	0.017
QT/QRS	4.13±0.52	4.15±0.44	4.07±0.53	0.858
QTc/QRS	4.35±0.63	4.53±0.77	4.11±0.75	0.001
Tp-e (msn)	94.3±9.3	99.2±8.4	83.5±6.9	0.001
Tp-e/QT	0.24±0.01	0.25±0.02	0.21±0.02	0.001
Tp-e/QTc	0.23±0.02	0.24±0.03	0.20±0.02	0.001
Tp-e (d) (msn)	16±4	16±5	15±4	0.806
Left ventricular ejection fraction (%)	61.1±8.3		64.1±8.7	0.271
Left ventricular end-diastolic dimension (mm)	50.7±4.7		48.6±5.2	0.378
Left ventricular end-systolic diameter (mm)	34.7±6.1		32.5±5.4	0.767
IVS-d (mm)	11.4±1.7		10.2±0.8	0.072
PW-d (mm)	11.4±1.5		10.3±0.7	0.154

ECG: Electrocardiogram; PW-d: P-wave dispersion; PR: PR duration; QRS: QRS duration; QT: QT duration; QTc: Corrected QT; Tp-e: Tp-Te duration; IVS-d (mm): Interventricular septum diastolic diameter; PW-d: (mm): Posterior wall diastolic diameter.

Table 3. Multivariate linear regression analysis in HD patients (n=109)

	B coefficient		p
Tp-e interval	Hemoglobin hypertension	0.205	0.013
Tp-e/QTc ratio	Platelet	0.115	0.033
	Age	0.382	0.002
Tp-e/QT ratio	Male gender	0.180	0.001
	Hematocrit	0.202	0.012
	Age	0.223	0.007
QTc/QRS ratio	hs-CRP	0.117	0.010
	Hypertension	0.297	0.002

HD: Hemodialysis; QTc: Corrected QT; QRS: QRS duration.

inine, blood urea nitrogen, parathyroid hormone, potassium, and uric acid levels were higher in the HD patients; however, serum hemoglobin, hematocrit, platelet, calcium, and estimated glomerular filtration rate levels were lower in HD patients (Table 1).

ECG and TTE Results

Basal HR (p=0.061), QT (p=0.176), QRS (p=0.551), and QTc duration (p=0.176) were similar between the groups; also, PR (p=0.001) duration was significantly longer in HD patients compared to the control group (Table 2). Furthermore, Tp-e duration (p=0.001), Tp-e/QT ratio (p=0.001), Tp-e/QTc ratio (p=0.001), and PW-d (p=0.001) were significantly higher in HD patients. There was no difference in Tp-e (d) (p=0.806) between the groups (Table 2).

QT, QTc, and Tp-e durations as well as QT/QRS, QTc/QRS, Tp-e/QT and Tp-e/QT ratios were found to be higher in post-dialysis ECGs compared to pre-dialysis ECGs and shown in Table 2. However, HR, PR, QRS duration, and Tp-e (d) value did not change after HD (Table 2).

Serum hemoglobin (p=0.013) and hypertension (HT) (p=0.033) were found to be independent predictors of an increased Tp-e interval in the multivariate linear regression analysis for HD patients (Table 3). In addition, age (p=0.001) and platelets (p=0.002) all independently predicted a higher Tp-e/QTc ratio. Hematocrit (p=0.007), age (p=0.018), and male gender (p=0.012) were found to be independent predictors of elevated Tp-e/QT ratio. Moreover, the QTc/QRS ratio was found to be independently predicted by HT (p=0.002) and hs-CRP levels (p=0.010) (Table 3). According to correlation analysis, significant correlations were found between serum parathyroid hormone levels and QTc duration (r=0.179, p=0.020), Tp-e (r=0.255, p=0.001), Tp-e/QT ratio (r=0.287, p=0.017), and Tp-e/QTc ratio (r=0.301, p=0.006) in HD patients (Fig. 3a-d).

TTE results showed no difference between the groups in left ventricular diastolic and end-systolic diameters, septum and posterior wall thicknesses, and EF values (Table 1).

Discussion

The present study evaluated resting ECG findings in HD patients, identifying a predisposition to ventricular and supraventricular arrhythmias and SCD. The study revealed that the QTc interval, PW-d, QTc/QRS, Tp-e, Tp-e/QT, and Tp-e/QTc ratios, which are utilized to assess the risk of ventricular and supraventricular arrhythmias, were significantly elevated in CKD patients compared to the control group. Furthermore, HD treatment exacerbates this risk. Patients with CKD are at considerable risk of mortality, with a median survival period of approximately 3 years. The risk of death within the 1st year is estimated to be 20%,[11] and the 5-year mortality rate for this group was approximately 65%. Despite numerous

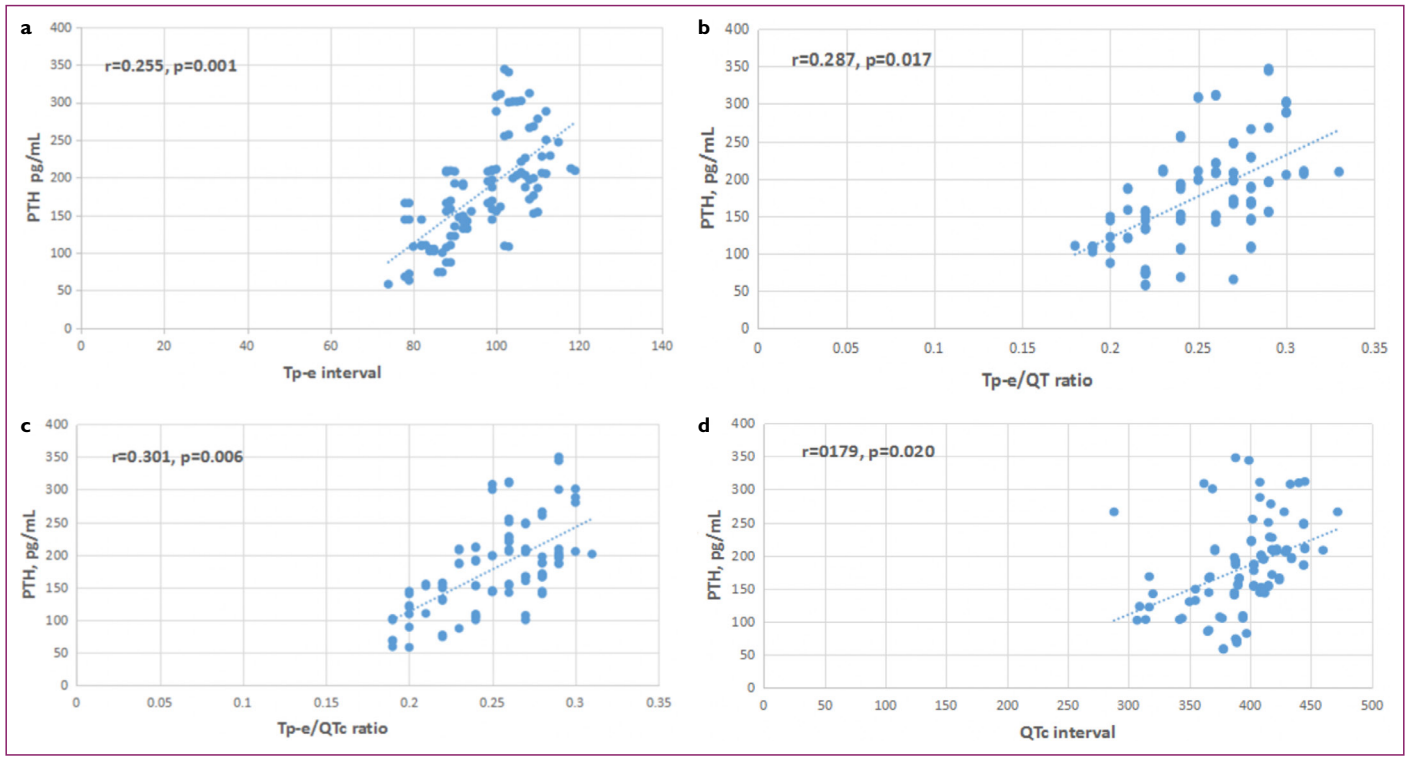

Figure 3. (a) Correlation analysis between left ventricular (LV) mass index and T peak-T end (Tp-e) interval, (b) correlation analysis between LV mass index and Tp-e/corrected QT (QTc) ratio, (c) correlation analysis between LV mass index and Tp-e/QT ratio, (d) correlation analysis between LV mass index and QTc duration.

PTH: Parathyroid hormone.

advancements in general medical care over the past decade, this rate has not significantly improved.[12,13] CVD remains the leading cause of mortality in patients with CKD, and a significant proportion of the mortality (25–40% of all deaths) is due to SCD.[12] Many patients on HD have comorbidities, such as coronary artery disease, vascular calcification, heart failure, or left ventricular hypertrophy (LVH), which predispose them to arrhythmias. In addition, patients with diabetes mellitus, older age, high ultrafiltration rates, and hypotension during HD are at risk for SCD.[13] In the US Renal Data System database, two-thirds of cardiac deaths were attributed to arrhythmias, accounting for 26% of the mortality. Similarly, in the HD study and the German diabetes dialysis study, 22% and 26% of deaths, respectively, were due to SCD.[14,15] The EVOLVE study, the largest randomized dialysis study, enrolled 3,883 HD patients, and 25% of the deaths were sudden.[16] Malignant cardiac arrhythmias are likely to be more common in the early years of dialysis due to hypotension during dialysis, post-dialysis metabolic changes, and the presence of subclinical ischemia. In the later stages of dialysis, bradyarrhythmias may become more common owing to degeneration of the conduction system with the use of cardiac medications. HD can cause significant electrolyte shifts, intravascular volume, blood pressure, hypotension, and changes in fluid balance, all of which may influence cardiac electrophysiology. HD can alter the body's pH and cause metabolic alkalosis, potentially leading to arrhythmias. HD can cause changes in the serum potassium, calcium, and magnesium levels. Hypokalemia is known to prolong QT intervals.[17] Hyperkalemia is a common complication of renal failure. Severe cases may lead to various cardiac arrhythmias, including conduction blocks, asystoles, and ventricular arrhythmias. This physiological condition shortens the myocyte action potential duration, thereby affecting myocardial refractoriness, whereas hyperkalemia can shorten the QT interval.[17] The increased arrhythmia risk observed on post-dialysis ECGs in this study corroborates previous literature findings, indicating an increased risk of SCD in HD. ECG measurement of the QT interval is an assessment of ventricular electrical activity, which is determined by the phases of depolarization and repolarization. Changes in the QT interval can be hereditary (e.g., due to genetic mutations) or acquired (e.g., due to pharmaceutical interventions). These changes can lead to malignant ventricular arrhythmias and SCD. Several techniques, including QT, QTc, and transmural dispersion of repolarization (TDR), have been used to measure myocardial repolarization.[18] Increased QT and QTc are well-established markers for arrhythmogenic substrates and are linked to both ventricular arrhythmias in CKD.[19,20] TDR is reflected by the Tp-e interval, Tp-e/QT ratio, and Tp-e (d) on the ECG. The Tp-e/QT ratio of the interval was established as a new arrhythmia index that provides an estimate of the repolarization distribution relative to the total repolarization time. It eliminates inter-individual variation in HR variability and QT interval. This represents the TDR, which is the difference in repolarization times across the ventricular wall. The voltage gradient between the subendocardial and subepicardial myocardium is represented by the T

wave. Tp corresponds to the end of epicardial repolarization, whereas Te indicates the end of repolarization in the entire myocardium. Consequently, the Tp-e has been identified as a useful indicator for the assessment of the TDR. Prolonged Tp-e and increased TDR are strongly associated with ventricular tachyarrhythmias in various pathologies, including LVH, long QT syndrome, and cardiomyopathy.[21] Atrial fibrillation is a prevalent condition among patients receiving dialysis, with a prevalence estimate ranging from 20% to 50%.[22] The rising risk of stroke among dialysis patients may be mainly caused by undiagnosed paroxysmal atrial fibrillation.[23] This study found that PW-d values were higher in HD patients and increased after dialysis. This finding elucidates the elevated incidence and risk of atrial fibrillation in patients undergoing HD, corroborating previous findings.

HT represents a common initial and significant modifiable risk factor for CKD, with a strong correlation with disease onset and progression. In addition, CKD causes blood pressure to lose its diurnal regularity. Sympathetic overactivity may exacerbate HT, which in turn may worsen renal failure and predispose patients to arrhythmias; however, the exact mechanism underlying QTc prolongation in HT remains unclear. HT may contribute to interstitial fibrosis and glomerulosclerosis, and some reports have suggested that the aldosterone/active renin ratio may be linked to the arrhythmic risk in HT.[24] Specifically, elevated aldosterone levels have been associated with repolarization abnormalities due to an increased density of myocardial capillaries, aberrant matrix proteins, and elevated superoxide levels in the mitochondria. Furthermore, HD may affect the autonomic nervous system, leading to changes in HR and variability, which may in turn affect the QTc interval.[25]

The main limitations of the present study are the relatively small patient sample and the use of experience from a single center. Moreover, the lack of long-term follow-up data for future arrhythmic events constitutes an important gap in the scope of the study. The extent to which subsequent episodes of ventricular or supraventricular arrhythmias occur in this patient population remains unclear. Further studies with a larger number of patients and longer follow-up periods are required to improve the accuracy of the results.

Conclusion

Our study indicates that HD treatment may increase the incidence of arrhythmic events and draws attention to the risk of sudden death in these patients. More careful follow-up and treatment are recommended, especially in patients in the group who may pose a risk of arrhythmia. The Tp-e/QTc interval is an important ECG parameter that can be affected by HD, with implications for the arrhythmia risk in patients undergoing this treatment. Managing arrhythmias during HD requires a multidisciplinary approach that involves nephrologists, cardiologists, and dialysis staff. Regular monitoring and appropriate management strategies are crucial for minimizing cardiovascular risks in these patients.

Disclosures

Ethics Committee Approval: The study was approved by the Kocaeli City Hospital Scientific Research Ethics Committee (no: 2024–124, date: 14/11/2024).

Informed Consent: Informed consent was obtained from all participants.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: No AI technologies utilized.

Author Contributions: Concept – N.Z.B., K.M.; Design – N.Z.B.; Supervision – K.M.; Resource – N.Z.B., K.M.; Materials – K.M.; Data collection and/or processing – K.M.; Data analysis and/or interpretation – N.Z.B.; Literature search – N.Z.B.; Writing – N.Z.B.; Critical review – K.M.

Peer-review: Externally peer-reviewed.

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