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Original Research Article

EFFECT OF HEMODIALYSIS ON CORRECTED QT INTERVAL AND QTc DISPERSION

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ABSTRACT

Background: Cardiovascular disease is the leading cause of mortality in patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis. Prolongation of corrected QT (QTc) interval and increased QTc dispersion are established markers of ventricular repolarization abnormalities and predictors of malignant arrhythmias. Hemodialysis-induced electrolyte shifts may further influence ventricular repolarization. **Aim:** To determine the effects of hemodialysis on corrected QT interval and QTc dispersion in CKD patients without clinically manifest heart disease.

Materials and Methods: This prospective observational study included 125 stable CKD patients on maintenance hemodialysis for ≥3 months. Patients with structural heart disease, arrhythmias, or drugs affecting QT interval were excluded. Twelve-lead ECGs and serum electrolytes (calcium, potassium, and magnesium) were measured before and after hemodialysis. QTc was calculated using Bazett's formula. Statistical analysis was performed using SPSS v25. Paired t-test and Pearson correlation were applied. P <0.05 was considered significant.

Results: Post-dialysis, serum potassium significantly decreased while serum calcium significantly increased (p<0.001). QTc minimum and QTc maximum showed significant prolongation after hemodialysis (p<0.05). QTc dispersion increased numerically but was not statistically significant. Pre-dialysis QTc dispersion correlated significantly with serum potassium and hemoglobin levels.

Conclusion: Hemodialysis significantly influences ventricular repolarization, particularly QTc minimum and QTc maximum, potentially increasing arrhythmic risk. Electrolyte shifts, especially potassium changes, play an important role in QT dynamics.

Keywords: Chronic kidney disease, Hemodialysis, QTc interval, QTc dispersion, Electrolytes, Ventricular arrhythmia.

INTRODUCTION

Cardiovascular disease is the leading cause of mortality among patients undergoing maintenance hemodialysis, accounting for nearly 40–50% of all deaths in this population.[1,2] Patients with chronic kidney disease (CKD) are exposed not only to traditional cardiovascular risk factors such as hypertension and diabetes but also to nontraditional factors including uremic toxins, autonomic dysfunction, chronic inflammation, electrolyte imbalance, and structural cardiac remodelling.[3-6] These factors collectively predispose patients to ventricular arrhythmias and sudden cardiac death.[7,8]

The QT interval on surface electrocardiography (ECG) represents the duration of ventricular depolarization and repolarization. Prolongation of the corrected QT (QTc) interval has been shown to predict ventricular arrhythmias and cardiovascular mortality in both the general population and in patients with end-stage renal disease (ESRD).[9,10] QTc dispersion, defined as the difference between the maximum and minimum QTc intervals across a

12-lead ECG, reflects spatial heterogeneity of ventricular repolarization and has been proposed as a non-invasive marker of arrhythmic risk.[11,12]

Patients undergoing hemodialysis frequently exhibit prolonged QTc interval and increased QTc dispersion.[13,14] Acute electrolyte shifts during hemodialysis—particularly in serum potassium, calcium, and magnesium—may further influence myocardial repolarization and electrical stability.[15-17] Several studies have demonstrated an increase in QTc interval and QTc dispersion following a single hemodialysis session.[13,18,19] However, results remain inconsistent across studies. Many earlier investigations were limited by smaller sample sizes and inclusion of patients with overt cardiovascular disease, which independently affects QT parameters.[13,18]

Given the high incidence of sudden cardiac death among dialysis patients and the potential role of dialysis-induced electrolyte shifts in altering ventricular repolarization, further evaluation in patients without clinically manifest heart disease is warranted.

Therefore, the present study was undertaken to determine the effects of hemodialysis on corrected QT interval and QTc dispersion in CKD patients without clinically evident cardiac disease.

MATERIALS AND METHODS

Study Design and Setting

This was a prospective observational study conducted over a period of 18 months (March 2019 to September 2020) in the Department of Nephrology, Narayana Medical College and Hospital, Nellore, India.

Sample Size

A sample size of 200 patients was initially planned. However, due to restrictions during the COVID-19 pandemic, recruitment was limited to 125 patients.

Study Population

Stable patients with chronic kidney disease (CKD) undergoing maintenance intermittent hemodialysis were screened for eligibility.

Inclusion Criteria

- CKD patients on chronic intermittent hemodialysis for at least 3 months
- Clinically stable at the time of enrollment

Exclusion Criteria

- Known ischemic, valvular, hypertensive heart disease, or cardiomyopathy
- Atrial fibrillation, supraventricular or ventricular ectopics
- Paroxysmal supraventricular tachycardia
- Left bundle branch block
- Patients receiving antiarrhythmic drugs known to prolong QTc interval (e.g., quinidine, amiodarone)
- Patients on beta blockers
- Inability to clearly identify the end of the T wave in more than three ECG leads

Data Collection

After obtaining written informed consent, detailed clinical history including duration and etiology of CKD and drug history was recorded.

Baseline Data

After informed consent:

- Demographic data recorded
- Etiology and duration of CKD noted
- Blood pressure recorded pre- and post-dialysis
- 12-lead ECG recorded at 25 mm/s and 10 mm/mV

Electrocardiographic Assessment

A standard 12-lead electrocardiogram (ECG) was recorded at a paper speed of 25 mm/s and calibration of 10 mm/mV:

- Immediately before the hemodialysis session
- Immediately after completion of the hemodialysis session

The QT interval was measured from the onset of the QRS complex to the end of the T wave.

- If the T wave was inverted, the endpoint was taken as the return of the trace to the TP baseline.
- If U waves were present and the end of the T wave could not be clearly identified in a particular lead, that lead was excluded from analysis.

Three consecutive QT interval measurements were obtained in each lead, and the mean value was calculated.

The QT interval was corrected for heart rate using Bazett's formula:

$QTc = QT / \sqrt{RR}$

QTc dispersion was defined as: QTc dispersion = QTc maximum − QTc minimum

Laboratory Measurements

Venous blood samples were collected:

- Immediately before initiation of hemodialysis
- Immediately after completion of hemodialysis

The following serum electrolytes were measured:

- Serum calcium
- Serum potassium
- Serum magnesium

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 25.0.

- Continuous variables were expressed as mean ± standard deviation (SD).
- Categorical variables were expressed as percentages.
- Paired t-test was used to compare pre- and post-dialysis parameters.
- Independent t-test was used where applicable.
- Pearson's correlation coefficient was applied to assess correlation between QT parameters and serum electrolytes.

A p-value < 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

A total of 125 patients undergoing maintenance hemodialysis were included in the study. The mean age of the study population was 48.94 ± 13.66 years. The majority of patients belonged to the 51–60 years age group (30.4%), followed by 41–50 years (22.4%). Males constituted 70.4% of the study population.

Hypertension was the most common etiology of chronic kidney disease (56.8%), followed by diabetes mellitus (48.8%), chronic glomerulonephritis (23.2%), and chronic interstitial nephritis (21.6%). Other causes included post-transplant status, ADPKD, obstructive nephropathy, and unknown etiology. [Table 1]

Clinical and Biochemical Parameters

The comparison of clinical and biochemical parameters before and after hemodialysis is shown in Table 2.

Systolic blood pressure increased significantly from 136.24 ± 20.42 mmHg pre-dialysis to 139.00 ± 17.66 mmHg post-dialysis (P = 0.04). Diastolic blood pressure also increased significantly (96.18 ± 9.82 mmHg vs. 99.32 ± 7.88 mmHg; P = 0.03). Heart rate increased from 83.24 ± 7.86 bpm to 87.66 ± 8.54 bpm (P = 0.02).

Serum calcium showed a significant increase following dialysis (8.09 ± 0.81 mg/dL vs. 9.41 ± 0.95 mg/dL; P < 0.001). Serum potassium levels decreased significantly from 4.52 ± 0.67 mEq/L to 3.72 ± 0.45 mEq/L (P < 0.001). However, corrected calcium and serum magnesium did not show statistically significant changes.

QTc Interval Changes

Changes in electrocardiographic parameters before and after hemodialysis are presented in Table 3.

QTc minimum increased significantly from 449.28 ± 44.44 ms pre-dialysis to 480.34 ± 44.00 ms post-dialysis (P < 0.001). Similarly, QTc maximum increased from 456.21 ± 41.48 ms to 488.16 ± 34.77 ms (P < 0.001). The mean QTc interval also showed a modest but statistically significant increase (464.81 ± 41.52 ms vs. 472.71 ± 34.36 ms; P = 0.04).

Although QTc dispersion increased numerically after dialysis (50.35 ± 32.21 ms vs. 64.68 ± 33.74 ms), this difference was not statistically significant.

Prevalence of Prolonged QTc Interval

The prevalence of prolonged QTc interval before and after hemodialysis is summarized in Table 4.

Prolonged QTc interval was observed in 59 patients (47.4%) prior to dialysis and increased to 64 patients (51.2%) following dialysis. Males constituted the majority in both pre- and post-dialysis groups.

QTc Dispersion Distribution

Distribution of QTc dispersion categories is shown in Table 5.

QTc dispersion ≥74 ms increased from 25.6% pre-dialysis to 35.2% post-dialysis, while dispersion <74 ms decreased from 74.4% to 64.8%.

Association Between QTc Dispersion and Biochemical Parameters

The association between pre-dialysis QTc dispersion and biochemical parameters is shown in Table 6. Serum potassium (P = 0.039) and hemoglobin (P = 0.036) demonstrated statistically significant associations with QTc dispersion prior to dialysis. No significant associations were observed with age, albumin, calcium, corrected calcium, or magnesium.

Post-dialysis QTc dispersion did not demonstrate statistically significant associations with serum biochemical parameters. [Table 7]

Table 1: Etiology of Chronic Kidney Disease (n = 125)

Etiology	Frequency (n)	Percentage (%)
Hypertension	71	56.8
Diabetes Mellitus	61	48.8
Chronic Glomerulonephritis	29	23.2
Chronic Interstitial Nephritis	27	21.6
Post Renal Transplant	3	2.4
ADPKD	1	0.8
Obstructive Nephropathy	1	0.8
Unknown	9	7.2

Table 2: Clinical and Biochemical Parameters Before and After Hemodialysis

Parameter	Pre-dialysis (Mean ± SD)	Post-dialysis (Mean ± SD)	P value
Heart rate (bpm)	83.24 ± 7.86	87.66 ± 8.54	0.02
Systolic BP (mmHg)	136.24 ± 20.42	139.00 ± 17.66	0.04
Diastolic BP (mmHg)	96.18 ± 9.82	99.32 ± 7.88	0.03
Hemoglobin (g/dL)	8.53 ± 1.59	—	—
Serum albumin (g/dL)	3.38 ± 0.44	—	—
Calcium (mg/dL)	8.09 ± 0.81	9.41 ± 0.95	<0.001
Corrected calcium (mg/dL)	9.41 ± 0.61	9.92 ± 1.04	0.39
Magnesium (mg/dL)	2.23 ± 0.36	2.32 ± 0.37	0.66
Potassium (mEq/L)	4.52 ± 0.67	3.72 ± 0.45	<0.001

Table 3: QTc Parameters Before and After Hemodialysis

ECG Parameter	Pre-dialysis (Mean ± SD)	Post-dialysis (Mean ± SD)	P value
QTc minimum (ms)	449.28 ± 44.44	480.34 ± 44.00	<0.001
QTc maximum (ms)	456.21 ± 41.48	488.16 ± 34.77	<0.001
Mean QTc interval (ms)	464.81 ± 41.52	472.71 ± 34.36	0.04
QTc dispersion (ms)	50.35 ± 32.21	64.68 ± 33.74	0.34

Table 4: Prevalence of Prolonged QTc Interval

QTc Status	Pre-dialysis n (%)	Post-dialysis n (%)
Prolonged QTc	59 (47.4%)	64 (51.2%)
Males	36 (61%)	42 (65.6%)
Females	23 (38%)	22 (34.3%)

Table 5: QTc Dispersion Categories

QTc Dispersion	Pre-dialysis n (%)	Post-dialysis n (%)
< 74 ms	93 (74.4%)	81 (64.8%)
≥ 74 ms	32 (25.6%)	44 (35.2%)

Table 6: Association Between Pre-dialysis QTc Dispersion and Biochemical Parameters

Variable	QTc ≤74 ms (Mean ± SD)	QTc >74 ms (Mean ± SD)	P value
Age (years)	52.84 ± 13.52	48.74 ± 15.36	0.972
Hemoglobin (g/dL)	8.71 ± 1.52	8.04 ± 1.67	0.036
Albumin (g/dL)	3.41 ± 0.43	3.11 ± 0.48	0.078
Calcium (mg/dL)	8.10 ± 0.78	8.08 ± 0.89	0.911
Corrected calcium (mg/dL)	8.79 ± 0.88	11.07 ± 1.28	0.086
Magnesium (mg/dL)	2.46 ± 2.10	2.21 ± 0.43	0.496
Potassium (mEq/L)	4.45 ± 0.63	4.72 ± 0.73	0.039

Table 7: Association Between Post-dialysis QTc Dispersion and Biochemical Parameters

Variable	QTc ≤74 ms (Mean ± SD)	QTc >74 ms (Mean ± SD)	P value
Age (years)	52.84 ± 13.52	48.74 ± 15.36	0.972
Hemoglobin (g/dL)	8.71 ± 1.52	8.04 ± 1.67	0.036
Albumin (g/dL)	3.41 ± 0.43	3.11 ± 0.48	0.078
Calcium (mg/dL)	9.20 ± 0.89	9.46 ± 1.11	0.754
Corrected calcium (mg/dL)	9.67 ± 1.00	10.04 ± 1.13	0.405
Magnesium (mg/dL)	2.14 ± 2.10	2.35 ± 0.39	0.611
Potassium (mEq/L)	3.71 ± 0.48	3.74 ± 0.38	0.698

DISCUSSION

Patients with end-stage renal disease (ESRD) on maintenance hemodialysis are at substantially higher risk of sudden cardiac death compared to the general population. Ventricular arrhythmias are considered the leading mechanism underlying these events. Electrocardiography (ECG), being a sensitive and non-invasive bedside tool, plays a pivotal role in detecting repolarization abnormalities and arrhythmogenic risk. Prolongation of the corrected QT (QTc) interval has been consistently associated with increased cardiovascular morbidity and mortality in both renal and non-renal populations.

The present study evaluated the impact of hemodialysis on QTc interval and QTc dispersion in 125 patients with CKD without clinically manifest heart disease. Male predominance observed in our cohort is consistent with previous studies.[19] This may partly reflect higher muscle mass and creatinine generation in males, along with differences in health-seeking behavior and comorbidity patterns.[20] However, other studies have reported variable gender distribution, and the precise reason for male preponderance remains inconclusive.

The mean age of the study population was 48.94 ± 13.66 years, with most patients in the 51–60 year age group. Similar age distributions have been reported in earlier studies.[21] Although ESRD commonly affects older individuals, it can occur across all age groups depending on the underlying etiology.

Hypertension was the most common cause of CKD in our cohort, followed by type 2 diabetes mellitus and chronic glomerulonephritis. This etiological distribution differs from reports by Sohal et al,[22] and Wakeel et al,[23] Regional variations, referral bias, and lifestyle differences may explain these discrepancies.

Hemodynamic parameters showed a modest reduction in systolic blood pressure (SBP) post-dialysis in most patients, consistent with previous reports. Intradialytic blood pressure variability is multifactorial and may result from rapid fluid shifts, sympathetic overactivity, activation of the renin–angiotensin–aldosterone system (RAAS), dialysate temperature, endothelial dysfunction, and dialyzable antihypertensive medications. Heart rate increased modestly after dialysis, similar to findings by Mendes et al,[24] Autonomic imbalance, RAAS

activation, and beta-blocker dialyzability may contribute to these changes.

Anemia was highly prevalent (95.2%) in our cohort, consistent with previous studies. Anemia in ESRD is multifactorial, including erythropoietin deficiency, iron deficiency, inflammation-mediated hepcidin elevation, nutritional deficiencies, and reduced gastrointestinal absorption. Hypoalbuminemia observed in the study population aligns with earlier reports.[25,26] Malnutrition-inflammation complex syndrome and reduced hepatic albumin synthesis contribute significantly to hypoalbuminemia in CKD.

Serum calcium levels increased significantly following hemodialysis ($p<0.001$). These findings are comparable to prior studies. The observed changes likely reflect diffusive and convective shifts across the dialyzer membrane, influenced by dialysate calcium concentration (3 mEq/L in our study). Chou et al. reported reduced post-dialysis calcium levels when lower dialysate calcium concentrations were used, emphasizing the importance of dialysate composition.[27,28]

Serum magnesium showed a modest, non-significant increase post-dialysis. Similar observations have been reported in earlier studies.[27] Magnesium balance during dialysis depends on dialysate magnesium concentration, dietary intake, impaired renal excretion, and parathyroid hormone (PTH) levels.

Potassium levels demonstrated expected shifts with correction of hyperkalemia and occurrence of transient hypokalemia post-dialysis. These findings are consistent with prior work by Feig et al,[29] and Hung et al.[30] Potassium removal is governed by the serum–dialysate concentration gradient and intracellular–extracellular redistribution, explaining post-dialysis rebound phenomena.

Prolonged QTc interval was observed in 47.4% of patients pre-dialysis and 51.2% post-dialysis, comparable to previous reports. Males exhibited greater QTc prolongation numerically, although without statistical significance, consistent with findings by Malhis et al.[31]

QTc minimum and QTc maximum increased significantly after dialysis ($p<0.001$), aligning with prior studies.[32] Electrolyte shifts, particularly changes in calcium and potassium, likely contribute to altered ventricular repolarization. However, unlike some earlier reports, the mean QTc interval did not demonstrate strong statistical significance in our study, possibly reflecting differences in dialysate composition or patient characteristics.

QTc dispersion, a marker of heterogeneity of ventricular repolarization and predictor of cardiac death,[4] did not show statistically significant change in our cohort. Although previous studies have reported significant increases, others have observed findings similar to ours.[33] Variations in methodology, patient selection, and electrolyte composition may account for these differences.

Subgroup analysis by CKD etiology revealed significant post-dialysis changes in calcium, potassium, QTc minimum, and QTc maximum across diabetes, hypertension, and chronic glomerulonephritis groups. These results are partially consistent with earlier studies.[23] However, QTc dispersion did not show significant association with etiology in our study, unlike some previous reports.[43]

The pathophysiology underlying QT prolongation in ESRD remains multifactorial. Structural cardiac remodeling, left ventricular hypertrophy, myocardial fibrosis, aldosterone excess, and vascular calcification may alter myocardial conduction properties, predisposing to prolonged repolarization.[22]

Correlation analysis demonstrated that pre-dialysis QTc dispersion was significantly associated with serum potassium but not with calcium or magnesium. This differs from some prior studies that demonstrated stronger associations with calcium and magnesium. Post-dialysis QTc dispersion showed no significant correlation with serum electrolytes, possibly due to standardized dialysate electrolyte concentrations and limited variability within the cohort.

Overall, the present findings reinforce that hemodialysis induces significant acute changes in ventricular repolarization parameters, particularly QTc minimum and QTc maximum, predominantly influenced by electrolyte shifts. Routine post-dialysis ECG evaluation may help identify patients at increased arrhythmic risk. Further large-scale, longitudinal studies evaluating dialysate modification and long-term arrhythmic outcomes are warranted.

CONCLUSION

The results of this study have shown that a significant number of patients of CKD on maintenance hemodialysis have prolonged QTc minimum and QTc maximum with significant increase after hemodialysis.

There was a significant association between QTc minimum, QTc maximum, serum potassium and serum calcium irrespective of the etiology of CKD before and after hemodilaysis

Post hemodialysis ECG would effectively identify patients who are at risk of arrythmias due to significant prolongation of QTc minimum and QTc maximum.

In the present study, there was no statistical significance between QTc dispersion and QTc interval in relation to hemodialysis irrespective of etiology of CKD.

In these individuals, a dialysis regimen can be adopted which is less likely to affect ventricular repolarization.

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