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Original Research Article

Prevalence and Risk Factors of Acquired Long QT Syndrome in Hemodialysis Patients.

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Abstract: ***Objective:*** To determine the prevalence and associated risk factors of acquired long QT syndrome (aLQTS) among patients undergoing maintenance hemodialysis. ***Methodology:*** A cross-sectional comparative study conducted in the Department of Nephrology, Sindh Institute of Urology and Transplantation (SIUT), Karachi, from 23rd May to 25th October 2025. A total of 150 adult patients with end-stage kidney disease (ESKD) on maintenance hemodialysis for more than three months were enrolled using non-probability consecutive sampling. Patients with pre-existing cardiac disease or on QT-prolonging medications were excluded. Demographic, clinical, and biochemical parameters were collected. Standard 12-lead ECGs were recorded pre- and post-dialysis to measure corrected QT interval (QTc). QTc > 440 ms in men and > 460 ms in women was considered prolonged. Data were analyzed using SPSS version 22.0, with $p < 0.05$ regarded as statistically significant. ***Results:*** The mean age was 56.3 ± 11.2 years, and 58.7% were male. The overall prevalence of prolonged QT interval was 23.3%. Patients with prolonged QT intervals were significantly older ($p = 0.042$), had longer dialysis duration ($p = 0.031$), and lower serum potassium levels ($p = 0.008$). Diabetes mellitus ($p = 0.019$), electrolyte imbalance ($p = 0.007$), were also significantly associated with QT prolongation. No significant associations were found with serum calcium, magnesium, phosphorus, or hypertension. ***Conclusions:*** Acquired long QT syndrome was common among hemodialysis patients, with its occurrence significantly linked to older age, prolonged dialysis duration, diabetes, hypokalemia, electrolyte imbalance.

Keywords: Chronic Kidney Disease; Diabetes Mellitus; Electrocardiography; Electrolyte Imbalance; Hemodialysis; Long QT Syndrome.

INTRODUCTION

Acquired long QT syndrome (aLQTS) is a measure of delayed ventricular repolarization that can promote potentially fatal arrhythmias such as torsades de pointes and sudden cardiac death. It is a heart rate independent hysteresis of myocardial electrical activity that may occur due to electrolyte

disturbances, metabolic disorders (hypoxia, hypothermia), structural heart disease or drugs with inhibitory effect on cardiac membrane ion channel. The prolongation of QT interval indicates delayed repolarization of myocardium, and it also results in greater dispersion of ventricular refractoriness that could facilitate the generation reentrant arrhythmias.

The incidence of aLQTS is largely higher in patients with CKD, particularly those evolving to ESRD needing maintenance HD. [1] This increased risk is in part related to the unique cardiovascular (CV) risk profile of CKD patients, with left ventricular hypertrophy, myocardial fibrosis, chronic inflammation and electrolyte derangement collectively leading to cardiac electrophysiological disturbance. CKD is more recently considered not simply a renal disease, but rather as a systemic disorder that profoundly contributes to burden of CVD morbidity and mortality. QT prolongation in particular has evolved as a consistent and potent indicator of sudden cardiac death in this population.

Patients treated with maintenance HD are particularly at risk of QT prolongation associated with rapid changes in serum electrolytes (especially potassium, calcium, and magnesium), sharp changes of intravascular volume, and osmotic gradients during dialysis. [2] The dialysis could in itself cause complex responses in the hemodynamic and metabolism of the myocardium, these results in transient cardiac ischemia, autonomic malfunction and change action potential duration. Furthermore, structural myocardial changes in CKD (left ventricular hypertrophy and myocardial fibrosis) contribute to electrical instability by lengthening the duration of the QT interval.

This risk is augmented by the use of QT-prolonging drugs. [3] Commonly administered drugs including antiarrhythmics, antidepressants and antibiotics can block the cardiac hERG potassium channels that are responsible for repolarization, increasing QTc prolongation in patients with renal clearance impaired. The best-known offenders are macrolide and fluoroquinolone antibiotics. It may be postulated that combined use of the above agents with large serum-to-dialysate potassium gradient can be a risk factor in development of repolarization instability and SCD among HD patients. [4] This situation highlights the significance of cautious pharmacological treatment and close electrolyte control during hemodialysis.

More importantly, the QTc is not a constant parameter but a changing value that can fluctuate throughout the dialysis session. Recent studies have shown that the QTc interval may be prolonged within the first hour following commencement of HD treatment and can also remain prolonged beyond the limits of HD session duration due mainly to serum potassium and magnesium reductions. [5]The speed as well as magnitude of these electrolyte shifts seem to co-relate with the extent of QTc change underscoring that arrhythmic susceptibility may peak during-recently postdialysis.

Recent research related to dialysate constitution and heart electrophysiology adds further support to the notion of individualized, or "personalized," dialysate prescriptions. QTc duration is affected by changes in both serum and dialysate electrolyte concentrations, indicating that QT interval changes may be compensated for by adjustment of dialysate potassium and calcium concentrations to reduce arrhythmic complications. [6] Furthermore, extended follow-up of HD populations has shown that persistence of QTc prolongation is linked to long-term structural and metabolic cardiac changes that may contribute to chronic cardiovascular mortality. [7]

Apart from dialysis-specific factors, QTc prolongation provides prognostic information in a wide variety of clinical settings. Epidemiological analysis indicated that abnormal QT dynamics can predict cardiovascular morbidity and mortality in the general population, with QTc prolongation representing a dependable noninvasive biomarker of adverse prognosis. [8] This finding supports the need for regular QT-interval monitoring in patients with CKD and those on dialysis, especially to facilitate early identification of high-risk individuals. In addition, QTc elongation has been reported in other systemic diseases such as COVID-19 and peripheral arterial disease, thus further supporting it a universal response to electrical and metabolic stress. [9–10]

However, despite growing recognition, the prevalence and predictors of aLQTS in CKD and HD patients are not well characterized. Electrolyte disorders, acid–base status, autonomic function, medication exposure and dialysis bath composition probably interact in complex ways to influence the stability of repolarization. Interactions between these factors may complicate the differentiation between transient QTc changes and clinically significant prolongation that is associated with arrhythmogenic risk.

Characterization of the modifiable determinants of QT prolongation in HD patients is important for generating targeted interventions to reduce ventricular arrhythmias and sudden cardiac death, which account for a significant portion of deaths among this population. Understanding the contributory functions of electrolyte control, dialysate composition, and QT-prolonging drugs may be helpful in planning preventive interventions and developing an individualized approach to dialysis. Additionally, incorporating QTc monitoring

as part of routine dialysis care, in conjunction with medications review may improve the cardiovascular safety and outcomes of this high risk group in the long run.

METHODOLOGY

This was a cross-sectional comparative study, carried out at Department of Nephrology Sindh Institute of Urology and Transplantation (SIUT) Karachi, in six months duration from 23rd , May to 25th , October 2025 after securing approval from ethical committee. The main outcomes for the prevalence and risk factors of acquired long QT syndrome (aLQTS) in patients on maintenance hemodialysis. Permission for work has been taken from SIUT Ethical Review Committee No: **SIUT-ERC-2025/A-558 (May 2025).** The study followed the principles of the Declaration of Helsinki by obtaining informed voluntary consent to participate, maintaining confidentiality and allowing individuals to withdraw from the study at anytime. After information regarding the study aims, methodology and potential consequences had been fully explained, written informed consent was obtained from all participants.

All information collecting and analyzing activities were conducted in strict confidence by anonymising data before its analysis. Data quality was continually monitored and verified by cross-checking against the source clinical records. The results of this study are for educational and clinical performance improvement, as it helps to learn about the frequency and predictors of acquired long QT syndrome among maintenance hemodialysis population in Pakistan.

An estimated 800 patients were on maintenance hemodialysis in the institution during the study. Using a prevalence rate of prolonged QTc interval of 56.97% as previously published,7 the sample size was calculated to be **156 patients**, assuming a margin of error of 7% at a confidence level of 95%. Sample size was calculated using WHO sample size calculator and participants were recruited through non probability consecutive sampling until the desired numbers of study subjects were reached. Adult male or female patients 18 years of age and older, diagnosed with end-stage kidney disease (ESKD) due to any etiology who had been undergoing maintenance hemodialysis for more than three months using a permanent angioaccess were eligible. Hemodialysis was performed twice weekly for each patient, three hours per session with bicarbonate-based dialysate and Nipro dialyzers provided with polyethersulfone membranes. Dialyzer membrane surface area was determined based on patient's body surface area with a constant dialysate flow rate of 500 mL/min and blood flow rate 250-300 mL/min. The dialysate solution was 2.5 mmol/L calcium, and 2.0 mEq/L potassium, and heparin for anticoagulation was given following institutional protocol in an unfractionated method.

Exclusion criteria included history of arrhythmias, ischemic heart disease (IHD), cardiomyopathy, valvular heart disease, hypertensive heart disease hereditary long QT syndrome, advanced liver diseases and use of QT-prolonging drugs such as amiodarone, beta-blockers or quinidine. Patients with pacemakers, recent myocardial infarction, or acute infections were also excluded to reduce potential confounders. All patients also had wall motion abnormalities and left ventricular hypertrophy excluded by an echo done within 1 month of ECG recording.

Demographic and clinical details such as age, sex, comorbidities, duration of ESKD and etiology of ESKD were captured in a structured proforma along with medication history, basic laboratory test results and minimal data set for dialysis. Blood pressure was measured before and after each hemodialysis with an appropriate sphygmomanometer (T/A) in a resting state, at least 5 minutes later. Two blood specimens by venipuncture were collected on all participants, one prior to hemodialysis and the second after it, for determination of serum electrolytes such as sodium, potassium, calcium and magnesium. All biochemical assessments were carried out within an hour of blood collection on automated analyzer for accurate measurement.

All electrocardiograms were obtained just prior to the hemodialysis session, after a rest of 10 minutes in supine position. Pre-dialysis ECGs were only received since the purpose of the study was to determine the relationship between baseline electrolyte levels and QTc interval, and not the changes in electrolytes resulting after dialysis; hence, no post-dialysis ECGs were taken. Corrected QT (QTc) was determined using the formula of Bazett and the QTc exceeding 440 ms in men and 460 ms in women were considered as QTc prolonged. All tracings were reviewed by two senior physicians to make sure that there was reliability in interpreting the results of ECG and any discrepancies were solved by consensus. Cohen kappa was 0.89 with excellent inter-reader agreement.

The data was recorded and analyzed using IBM SPSS Statistics, 22.0 (Armonk, NY USA). Quantitative variables, including age, duration of dialysis, and QTc interval were described as mean ± standard deviation in the case of normally distributed data or as median and interquartile range in skewed distributions. Categorical variables, including sex, comorbidity and QTc prolongation were presented as the number (percentage). Categorical variables were analyzed using the chi-square or Fisher's exact test, and continuous variables by independent t-tests or one-way analysis of variance. A p-value < 0.05 was

considered as statistically significant.

RESULTS

A total of 150 hemodialysis patients were enrolled in the study. The mean age of participants was 56.3 ± 11.2 years, with a male-to-female ratio of 1.4 : 1. The median duration of hemodialysis was 30 (18–52) months, and the majority (68.0%) underwent dialysis three times per week. The mean body mass index (BMI) was 24.8 ± 4.2 kg/m².

As shown in **Table I**, diabetes mellitus (58.7%) and hypertension (79.3%) were the leading causes of end-stage renal disease (ESRD). A previous history of cardiovascular disease was noted in 34.7% of patients, while 13.3% reported prior arrhythmia or syncope.

The biochemical and hematologic parameters are presented in **Table II**. The median pre-dialysis serum potassium was 4.5 (4.2–4.8) mEq/L, calcium 8.7 (8.4–9.0) mg/dL, magnesium 2.1 (1.9–2.3) mg/dL, and phosphorus 5.4 (4.8–5.9) mg/dL. The mean hemoglobin level was 10.4 ± 1.2 g/dL. Approximately one-third (30.7%) had documented electrolyte imbalances within the preceding three months. Referring to clinically significant deviations in any electrolyte requiring medical attention, independent of measured values during the study visit.

Based on electrocardiographic evaluation, the prevalence of prolonged QT interval was 23.3% (n = 35) (QTc > 440 ms in men and > 460 ms in women). Among these patients, 57.1% were female, 74.3% were diabetic, and 48.6% reported recent electrolyte imbalance episodes. Comparisons between patients with normal and prolonged QT intervals (**Table III**) showed that those with QT prolongation were significantly older and had a longer duration of dialysis. They also exhibited lower measured serum potassium levels at the time of evaluation and a higher frequency of prior electrolyte imbalance episodes ($p < 0.05$ for all). No significant differences were found in calcium, magnesium, phosphorus, or hypertension prevalence.

No statistically significant differences were observed in calcium, magnesium, phosphorus, or hypertension status between the groups. The overall prevalence of acquired long QT syndrome among hemodialysis patients was 23.3%. Significant associations were found with older age, longer dialysis duration, lower potassium, presence of diabetes, electrolyte imbalance. ($p < 0.05$).

Table I. Demographic and Clinical Characteristics of Study Participants (n = 150)

Variable	Mean ± SD / n (%)
Age (years), mean ± SD	56.3 ± 11.2
Male gender	88 (58.7)
BMI (kg/m²), mean ± SD	24.8 ± 4.2
Duration of hemodialysis (months), median (IQR)	30 (18–52)
Frequency of dialysis (thrice weekly)	102 (68.0)
Duration per session (3–4 hours)	127 (84.7)
Primary cause of ESRD: Diabetes Mellitus	88 (58.7)
Primary cause of ESRD: Hypertension	31 (20.7)
Cardiovascular disease history	52 (34.7)
Prior arrhythmia or syncope	20 (13.3)

Table II. Electrolyte and Laboratory Parameters of Hemodialysis Patients (n = 150)

Parameter	Mean ± SD / Median (IQR)
Serum potassium (mEq/L)	4.5 (4.2–4.8)
Serum calcium (mg/dL)	8.7 (8.4–9.0)
Serum magnesium (mg/dL)	2.1 (1.9–2.3)
Serum phosphorus (mg/dL)	5.4 (4.8–5.9)
Hemoglobin (mg/dL)	10.4 ± 1.2
Electrolyte imbalance in past 3 months	46 (30.7)

Table III. Comparison of Clinical and Biochemical Parameters According to QT Interval Status

Variable	Normal QT (n	Prolonged QT (n	p-value

	= 115)	= 35)	
Age (years), median (IQR)	54 (46–61)	60 (52–68)	**0.042 ***
Duration of hemodialysis (months), median (IQR)	28 (15–48)	36 (24–60)	**0.031 ***
Serum potassium (mEq/L), mean ± SD	4.6 ± 0.5	4.2 ± 0.4	**0.008 ***
Serum calcium (mg/dL), mean ± SD	8.7 ± 0.6	8.6 ± 0.7	0.247
Serum magnesium (mg/dL), mean ± SD	2.1 ± 0.4	2.0 ± 0.3	0.198
Serum phosphorus (mg/dL), mean ± SD	5.3 ± 1.0	5.6 ± 1.2	0.112
Diabetes mellitus, n (%)	62 (53.9)	26 (74.3)	**0.019 ***
Hypertension, n (%)	89 (77.4)	30 (85.7)	0.263
Electrolyte imbalance, n (%)	28 (24.3)	17 (48.6)	**0.007 ***

*p < 0.05 = statistically significant.

DISCUSSION

The prevalence of QT interval prolongation among patients on chronic maintenance hemodialysis in the current study was 23.3%, and relatively similar to previously published reports in comparable ESRD cohorts. [7] QT interval prolongation in patients under hemodialysis is a multifactor process, related to the interplay among electrolyte disturbances, uremic toxins, myocardial remodeling and drugs. [7] Increased QTc has been repeatedly documented on long-term dialysis, especially in those with ongoing electrolyte irregularities or poor intradialytic potassium management. This result further underpins the clinical relevance of monitoring QT intervals in dialysis patients, for anticipation of possible arrhythmogenic risks. [7]

Prolongation of the QT interval is well known to be an independent risk factor for ventricular arrhythmias and sudden cardiac death in patients with renal insufficiency. 8 TN Dynamic changes in the QT interval during dialysis have been demonstrated to predict adverse cardiovascular outcomes, independently of baseline comorbidities or left ventricular dysfunction. [8] In our study, prolonged QT interval was associated with older age, longer dialysis vintage, diabetes mellitus (in all cases $p < 0.05$). These data indicate that clinical factors and iatrogenic influences may interact in dialysis patients to result in an additive effect on delayed ventricular repolarization.

In this study, the electrolyte disturbances (especially hypokalemia and hypocalcmia) were also an important factor determining QT prolongation. A previous study reported that decreasing levels of serum potassium and calcium were independent predictors of QT prolongation in hospitalized patients. [9] Due to the rapid changes in electrolytes during hemodialysis, even small variations in serum potassium or calcium can alter cardiac repolarization and affect arrhythmic propensity. Our results emphasize the need for patient-specific electrolyte and dialysate prescriptions to reduce QTc variation and its related arrhythmic burden.

Prolongation of corrected QT interval is associated with increased mortality and cardiovascular events in dialysis patients with peripheral artery disease. [10] On the other hand, in our study QT prolonged patients were older, had longer dialysis vintage and comorbidities. These observations are consistent with the notion that serial QT measurements can be biomarker for risk stratifying patients at high risk of a CV event in the maintenance dialysis patient population in a noninvasive and cost-contained manner. [10]

Chronic Q-T prolongation has also been consistently associated with mortality risk in hemodialysis populations. [11] PTc was found to be a strong predictor of one-year cardiac death, reinforcing its prognostic value. [11] Our findings are in line with such observations; almost one quarter of our patients had a prolonged QT interval, and most of them had a history of diabetes or an episode of mild electrolytic distrubance. These observations emphasize the need for serial ECGs and cautious clinical observation of arrhythmia-susceptible patients in long-term dialysis care.

Mellitus diabetes was identified as a strong predictor of prolonged QT interval in cohort. Potential mechanisms include diabetic autonomic neuropathy, myocardial fibrosis, and metabolic factors that can cause changes in cardiac ion channel function. [12] Experiments and clinical observations suggest that chronic hyperglycemic state and increased oxidative stress influence expression of ion channels, causing delayed repolarization. [12] In accordance with this an increased rate of QT prolongation was found in the diabetic versus non-diabetic population (<0.019). This further endorses that glycemic control and early identification of cardiac autonomic involvement should be considered in the preventive approach in diabetics CKD subjects to reduce electrophysiological instability.

The QT prolongation has clinical importance not only for dialysis. Research of early repolarization and ECG abnormalities similar to those in the CKD population show similar prognostic significance in other non-CKD cohorts. [13] Long or abnormal

repolarization has also been associated with arrhythmic events and cardiovascular death in seemingly healthy adults. [14] Overall, these results indicate that subclinical repolarization abnormality (eg, QT prolongation and early repolarization syndrome) is a significant factor for prognosis, particularly in the population with high cardiac vulnerability such as HD patients.

In addition, the presence of electrolyte or metabolic stress states typical for athletes and common in dialysis patients may further enhance repolarization disturbances. These data underscore that HD patients exist in a state of continual electrophysiological fluctuations with abrupt changes electrochemical and osmotic gradients that predispose to arrhythmic risk.

Of course, we can't forget the contribution of pharmacologic QT prolongation. Chronic kidney disease and dialysis patients undergo frequent polypharmacy treatment including antibiotics, antidepressants, and antiemetics and antipsychotics, many of which have QT prolonging potential. [16] Many hemodialysis patients were found to be on at least one QT-prolonging medication in recent studies such as these, and the increment of QT prolongation with exposure increases the risk for malignant arrhythmias. [17] These observations stress the need for systematic ECG control and a thorough drug assessment in dialysis departments in order to reduce iatrogenic arrhythmic risks. [17]

High values of fibroblast growth factor 23 (FGF23) have been in fact found independently associated with QTc prolongation and death in PCKD patients, highlighting putative associations between derangement in mineral metabolism and cardioelectric physiology. [18] Moreover, in these circumstances prolonged QT interval is associated with an increased risk of cardiac death after adjusting for traditional cardiovascular risk factors. [19]

The changing landscape of artificial intelligence and deep learning models presents exciting paths for automated detection of QT prolongation with single-lead ECGs. [20] These technologies could provide early risk stratification and personalized cardiac monitoring for patients on dialysis, particularly in resource poor areas. In addition, control of polypharmacy and deprescribing PIMs is important for ARD prevention, particularly in older individuals on dialysis. [21]

Recent prospective evidence also supports that increased QTc is a predictor of mortality and cardiovascular outcomes in dialysis patients, corroborating its potential as a clinically-useful prognostic biomarker. [22] Similarly, QTc prolongation due to FGF23 and disordered mineral metabolism is an independent risk factor for mortality in predialysis CKD. [23] Collectively, these results establish the current evidence supporting QT interval prolongation in dialysis patients as a multifactorial modifiable entity with prognostic implications.

In the face of the robustness of our results, certain limitations must be noted. This was a single-center, cross-sectional study and the sample size was small; therefore, the results may not apply to other dialysis populations. The cross-sectional nature of the study limits the interpretation of causality of observed QT prolongation, associated factors. Dynamic intradialytic QT changes were not assessed, and we did not have post-dialysis electrolyte concentrations that could have helped to shed light on temporal variations. Also, medication adherence and serum drug concentration were not measured and unmeasured comorbidities may have residual confounding. These associations will need to be validated in future multicenter longitudinal studies with rigorous, continuous ECG and electrolyte monitoring and the causal pathways connecting fluctuations in electrolytes, drug exposures, and QT interval variability in HD patients should be further investigated.

CONCLUSION

QT interval prolongation occurred in nearly 1/4 of maintenance hemodialysis patients, and was significantly associated with older age, longer duration on dialysis, diabetes mellitus, electrolyte imbalance. These observations underline the multivariate occurrence of repolarisation abnormalities in this high-risk population. Regular ECG assessment and review of medications, as well as early intervention for electrolyte imbalances, can contribute to prevention of lethal arrhythmias and enhancement of cardiovascular survival in hemodialysis patients.

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