Original Article

Prognostic Value of QTc Interval and Other Ventricular Repolarization Parameters in Acute Stroke

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Abstract

Background: **The electrocardiogram (ECG) is routinely used in stroke evaluation, although interactions within the brain–heart axis remain incompletely understood. The prognostic relevance of the QTc interval and other ventricular repolarization markers has gained increasing interest in stroke populations.**

Objectives: **To evaluate the association of QTc interval, QTc dispersion (QTc-d), Tpeak–end interval (Tp-e), and Tp-e/QTc ratio with neurological disability and in-hospital mortality in acute stroke.**

Methods: **This retrospective cohort included patients admitted with acute stroke between January 2018 and April 2020. QTc was analyzed as both a continuous and categorical variable, using a cutoff of 480 ms. Functional disability was assessed using the modified Rankin Scale (mRS) and categorized as no disability (mRS ≤ 2) or disability (mRS > 2). Sample size calculation indicated a minimum of 482 patients (85% power; 5% significance), based on functional outcome differences between prolonged and normal QTc. A 5% significance level was used for all analyses.**

Results: **A total of 565 patients were included (50.1% male; mean age 65 ± 13 years). Of these, 280 (49.6%) had mRS > 2, and 69 (12.5%) died during hospitalization. Higher QTc values were associated with worse neurological outcomes in univariable (HR 1.01; 95% CI, 1.00–1.01; p < 0.001) and multivariable analyses adjusted for age and NIHSS (HR 1.009; 95% CI, 1.00–1.01; p = 0.001). QTc > 480 ms was also associated with disability in univariable (HR 2.43) and multivariable models (HR 2.67). QTc was not associated with in-hospital mortality.**

Conclusion: **QTc prolongation, particularly QTc > 480 ms, was independently associated with unfavorable neurological outcomes.**

Keywords: **Stroke; Electrocardiography; Cross-Sectional Studies.**

Introduction

Acute stroke remains one of the leading causes of death and disability worldwide.[1-4] A study across 204 countries reported that, in 2021, the global prevalence, incidence, mortality, and disability-adjusted life years (DALYs) for stroke were 1,099.3, 141.5, 87.5, and 1,886.2 per 100,000 people, respectively.[4] In the United States, approximately 9.4 million adults aged ≥20 years reported having had a stroke.[1,2] Between 2017 and 2020, the overall prevalence was 3.3%.[2] Each year, nearly 795,000 individuals experience a new or recurrent stroke – 610,000 first-ever events and 185,000 recurrences.[1]

The electrocardiogram (ECG) is a routine component of stroke evaluation. A baseline ECG is recommended in acute ischemic stroke, provided it does not delay initiation of therapy.[5] Beyond supporting etiological assessment, the ECG aids in detecting underlying cardiovascular disease.[5,6] Importantly, patients with acute stroke frequently present with ECG abnormalities – including ST-segment, T-wave, and QTc changes—even in the absence of structural heart disease, as demonstrated in a systematic review.[6-11]

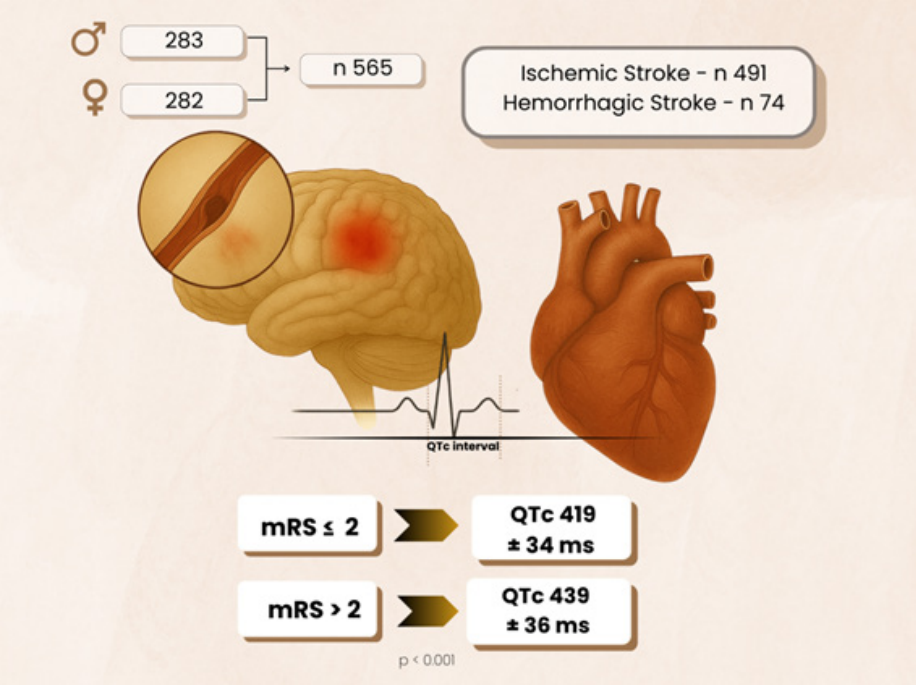
These abnormalities are primarily attributed to fluctuations in autonomic regulation of the heart.[12,13] Interactions within the brain–heart axis remain poorly understood, although growing evidence supports a complex and bidirectional relationship between the central nervous system and cardiovascular function.[14] The brain–heart axis is mainly mediated by the autonomic nervous system, particularly through the central autonomic network, which integrates cortical and subcortical structures such as the insular cortex, amygdala, and hypothalamus to regulate cardiac activity.[12,13,15]

In the context of acute stroke, disruption of these regulatory pathways leads to autonomic imbalance, typically characterized by sympathetic overactivation and parasympathetic withdrawal. This dysregulation can result in a catecholamine surge, contributing to myocardial injury, arrhythmias, and abnormalities in ventricular repolarization, including QTc prolongation.[12,14]

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Central Illustration: Prognostic Value of QTc Interval and Other Ventricular Repolarization Parameters in Acute Stroke



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Additionally, stroke-related cardiac dysfunction is influenced by multiple interconnected mechanisms, including activation of the hypothalamic–pituitary–adrenal axis, systemic inflammatory responses, and neurogenic myocardial stunning.[14] Despite advances in understanding these pathways, the precise mechanisms linking specific electrocardiographic changes – such as QTc prolongation – to neurological injury severity and clinical outcomes remain incompletely elucidated.[15] Recent evidence in neurocardiology has further reinforced the relevance of these interactions in acute stroke.[12,15,16]

Several studies have suggested that prolonged QTc is associated with poor prognosis in the acute phase of stroke.[2,15,17-22] Increased QTc dispersion – defined as the difference between the maximum and minimum QTc values on a single ECG – has also been associated with poor prognosis after stroke, as demonstrated in a systematic review. Moreover, the Tpeak–end interval (Tp-e) and the Tp-e/QTc ratio, proposed markers of transmural dispersion of ventricular repolarization, have been investigated in acute stroke.[11,23-26] However, most studies had small sample sizes, and cutoff values for prognostic risk varied substantially.

The role of prolonged cardiac monitoring during hospitalization in guiding treatment decisions for secondary stroke prevention remains uncertain.[5,27] This study aimed to evaluate the prognostic value of QTc prolongation and other ventricular repolarization parameters in predicting neurological outcomes and in-hospital mortality in patients with acute stroke.

Methods

Study Design

This was a retrospective cohort study including consecutive patients aged >18 years with acute stroke (ischemic or hemorrhagic) admitted to a tertiary teaching hospital in southern Brazil between January 2018 and April 2020. Patients were identified at hospital admission and followed throughout hospitalization until discharge or death.

Clinical and demographic data were obtained from electronic medical records. Electrocardiographic parameters were measured from baseline ECGs performed at admission. Outcomes were assessed longitudinally during hospitalization, including functional status at discharge using the modified Rankin Scale (mRS) and in-hospital mortality.

The study was conducted in accordance with the Declaration of Helsinki and was approved by the institutional Research Ethics Committee. Written informed consent was obtained from all patients or their legal representatives at the time of data collection by the team responsible for maintaining the institutional database.

Electrocardiogram analysis

Standard 12-lead ECGs were recorded using a digital system at a paper speed of 25 mm/s and an amplitude of 10 mm/mV. All recordings were saved as PDF files and analyzed independently by two trained investigators using EP Calipers software (EP Studio Software). QT and QTc intervals were measured in lead D2, and QTc dispersion was measured across all 12 leads. The Tp-e interval was measured in the six precordial leads (V1–V6) (Figure 1).

QTc was defined as the interval from the onset of the QRS complex to the end of the T wave. It was calculated using Bazett's formula, the Hodges formula for heart rates >100 bpm, and the Fridericia formula for heart rates <50 bpm.[28] The mean QTc across all 12 leads was analyzed as a continuous variable. QTc prolongation was defined as >480 ms, based on the cutoff with optimal sensitivity and specificity for outcome prediction in this cohort.

Additional parameters included QTc dispersion, Tp-e interval, and the Tp-e/QTc ratio. QTc dispersion was defined as the difference between the maximum and minimum QTc values on the 12-lead ECG.[11,23,29-31] The Tp-e interval was calculated by subtracting the QT peak from the QT interval in each precordial lead.[23,30-32] ECGs were considered valid for Tp-e analysis if at least 5 of the 6 precordial leads were analyzable and the T-wave amplitude was ≥2 mm.[26] The Tp-e/QTc ratio was calculated using Tp-e in lead V5 and QTc in lead D2.[26,32]

Patients with atrial fibrillation, bundle branch block, pacemaker rhythm, electrolyte disturbances, or prior use of QT-prolonging drugs were excluded (Figure 2). Intra- and interobserver variability was assessed in a subset of 20 ECGs.

The sample size of 20 ECGs used for assessment of intra- and interobserver variability was defined a priori based on methodological precedents from previous reproducibility studies of QT-based electrocardiographic parameters. Prior investigations evaluating manual measurements of QT, QTc dispersion, and Tp-e intervals typically employed subsets ranging from 10 to 30 tracings, which have been shown to provide stable estimates of intraclass correlation coefficients while ensuring feasibility for detailed manual analysis. Therefore, a sample of 20 ECGs was considered adequate to balance methodological rigor and practical measurement workload.

Outcomes

Neurological disability was assessed at admission and discharge using the modified Rankin Scale (mRS).[5,27] Patients were classified as non-disabling or mildly disabling (mRS ≤2) versus disabling (mRS >2) (Central Figure). For patients who died, the last recorded score before death was considered the discharge mRS. The secondary outcome was all-cause in-hospital mortality.

Statistical analysis

Continuous variables were assessed for normality using the Shapiro–Wilk test and visual inspection of histograms. Variables with a normal distribution are reported as mean ± standard deviation, whereas non-normally distributed variables are reported as median and interquartile range (25^{th}-75^{th} percentile). Categorical variables are presented as absolute and relative frequencies (n and %).

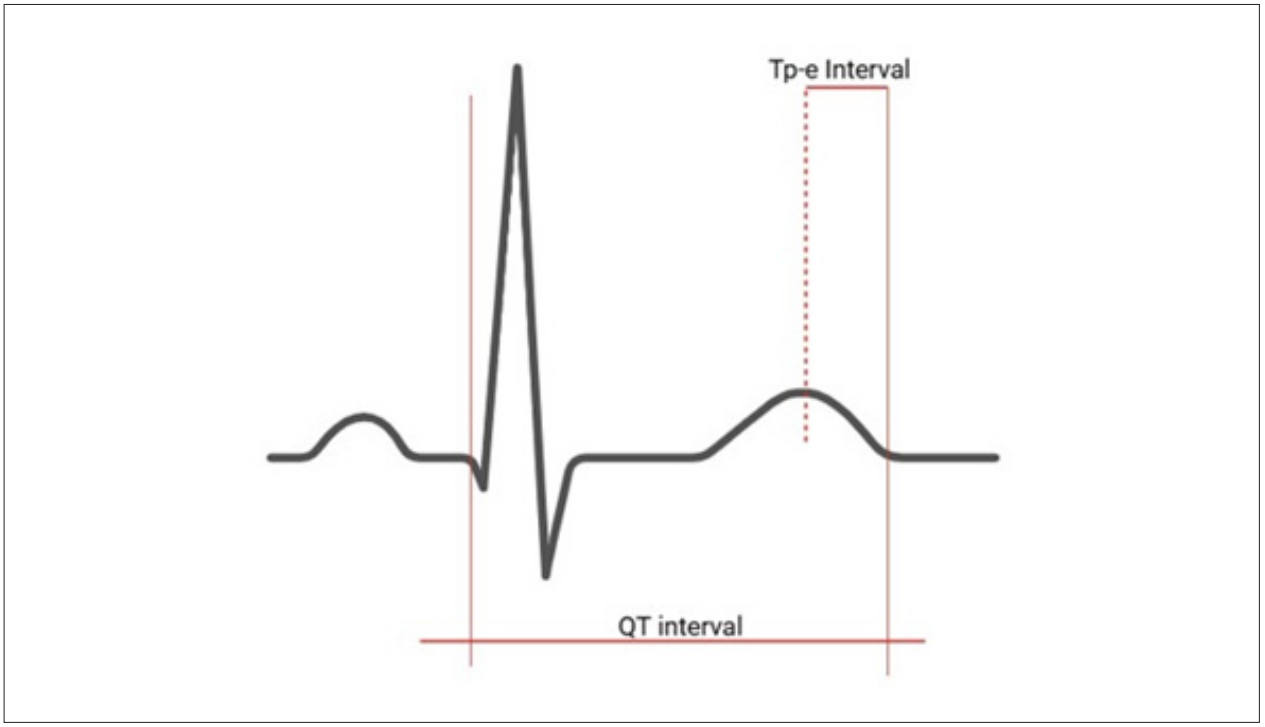


Figure 1 – *Schematic representation of QT and Tp-e intervals on an electrocardiographic waveform*

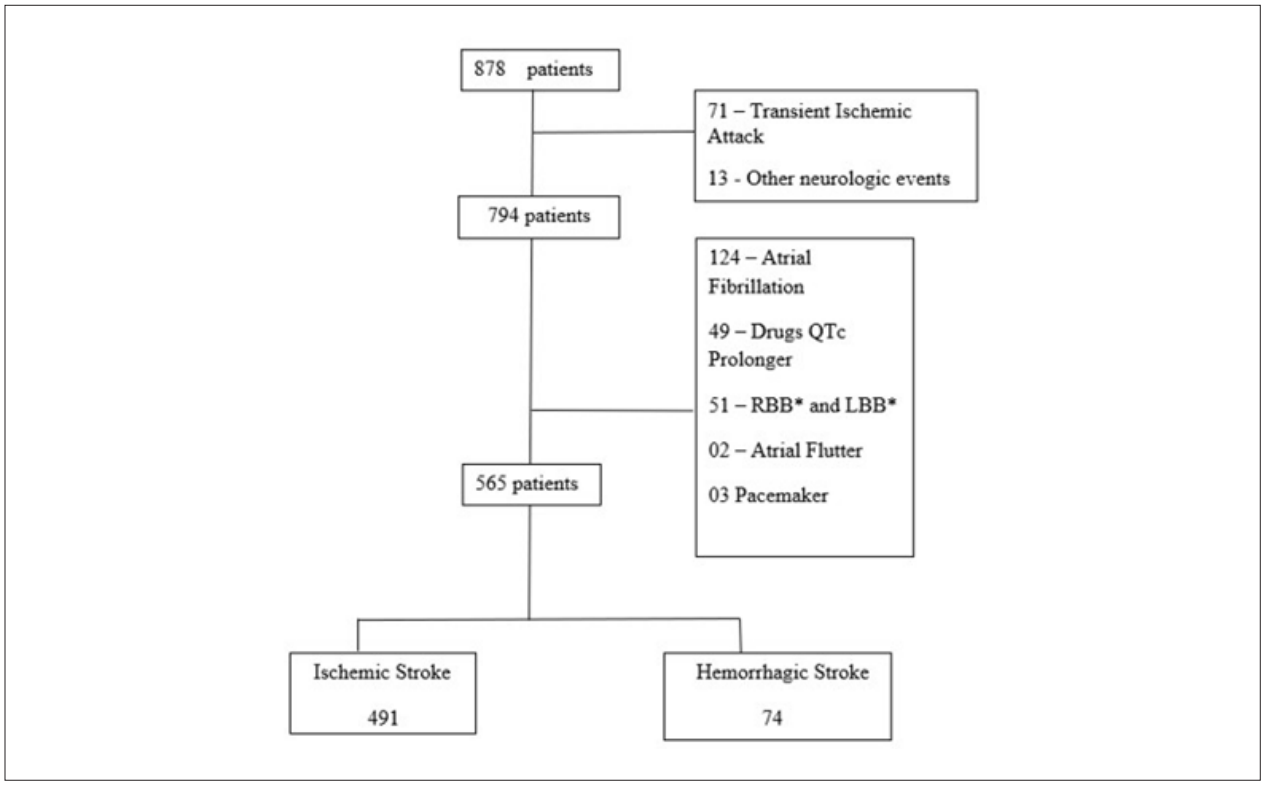


Figure 2 – *Study flow diagram; RBB: right bundle block, LBB: left bundle block*

For comparisons between groups, the unpaired (independent-samples) Student's t-test was applied for normally distributed continuous variables, and the Mann–Whitney U test was used for non-normally distributed continuous variables. The chi-square test was used for categorical variables.

Univariable Cox regression analysis included clinical variables previously associated with stroke outcomes. Multivariable Cox regression was used to assess the association between ECG parameters and these clinical variables.

Statistical analyses were performed using IBM SPSS Statistics, version 29.0.1.0. Sample size calculation, based on 85% power and a 5% significance level, indicated a minimum of 482 patients, derived from differences in functional outcomes (mRS ≤2 vs >2) between patients with prolonged versus normal QTc, as reported by Stead et al.[9]

The secondary outcome (in-hospital mortality) was analyzed using a parametric model with log-normal distribution, selected based on Akaike Information Criterion (AIC) and visual fit to Kaplan–Meier curves. Analyses were performed in R version 4.4.3 using the survfit and survreg functions from the survival package. Graphs were generated with ggplot2 and ggsurvplot.

Results

Between January 2018 and April 2020, 878 patients with suspected stroke were admitted to hospital. Stroke (ischemic or hemorrhagic) was confirmed in 794 cases. After applying exclusion criteria, the final study sample comprised 565 patients, with a mean age of 65 ± 13 years and 50.1% were male (Figure 2 and central figure). Of these, 491 patients had ischemic stroke and 74 had hemorrhagic stroke (Central Figure).

At discharge, 285 patients (50.4%) had a favorable outcome (mRS ≤ 2), whereas 280 patients (49.6%) had disabling stroke (mRS > 2) (Table 1 and Central Figure). The most common comorbidities were hypertension, diabetes, smoking, prior stroke, and coronary artery disease.

The mean QTc interval was significantly shorter in patients with favorable outcomes compared with those with disabling stroke (419 ± 34 ms vs. 439 ± 36 ms; $p < 0.001$) (Figure 3 and Central Figure). Patients with disabling stroke were generally older. The median length of hospital stay was 9 days (IQR 6–15). Patients with favorable outcomes (mRS ≤ 2) had a shorter hospital stay than those with disabling stroke (7 [4–12] vs. 11.5 [7–18] days).

The intraclass correlation coefficient (ICC) was 0.93 (95% CI, 0.839–0.973) for intraobserver measurements of electrocardiographic variables and 0.87 (95% CI, 0.69–0.94) for interobserver measurements ($p < 0.05$).

Primary Outcome: Functional Disability

In univariable Cox analysis (Table 2), age, National Institutes of Health Stroke Scale (NIHSS) score, mean QTc (continuous), and QTc > 480 ms were significantly associated with an increased risk of disabling stroke. In contrast, QTc dispersion,

Table 1 – Baseline Clinical, Demographic, and Electrocardiographic Characteristics According to Functional Outcome at Discharge (mRS ≤ 2 vs mRS > 2)

Variable	All Patients (n = 565)	mRS ≤ 2 (n = 285)	mRS > 2 (n = 280)	p Value
Male, n (%)	283 (50.1%)	150 (52.6%)	133 (47.4%)	0.275
Age, mean ± SD (years)	65 ± 13	63 ± 12	67 ± 12	0.001
Length of stay, days, (median [IQR])	9 [6-15]	7 [4-12]	11.5 [7-18]	<0.001
Hypertension, n (%)	403 (71.6%)	196 (68.8%)	207 (73.9%)	0.103
Diabetes mellitus, n (%)	157 (27.8%)	74 (26.0%)	83 (29.6%)	0.189
Previous stroke, n (%)	129 (22.8%)	9 (3.2%)	120 (42.1%)	0.025
Smoking (current or former), n (%)	150 (26.4%) / 91 (16.1%)	83 (29.1%) / 67 (23.5%)	67 (23.9%) / 41 (14.6%)	0.201
Coronary artery disease, n (%)	97 (17.2%)	49 (17.2%)	48 (17.1%)	0.538
Alcohol use (current / former), n (%)	34 (6.0%) / 15 (2.7%)	15 (5.3%) / 8 (2.8%)	19 (6.8%) / 7 (2.5%)	0.735
NIHSS ≥ 16, n (%)	73 (12.9%)	5 (1.8%)	68 (24.3%)	<0.001
Ischemic stroke, n (%)	491 (86.9%)	260 (91.2%)	231 (82.5%)	0.003
Hemorrhagic stroke, n (%)	74 (13.1%)	25 (8.8%)	49 (17.5%)	0.003
In-hospital mortality, n (%)	69 (12.2%)	1 (0.4%)	68 (24.3%)	<0.001
QTc interval, mean ± SD (ms)	425 ± 36	419 ± 34	439 ± 36	<0.001
QTc > 480 ms, n (%)	37 (6.5%)	11 (3.9%)	26 (9.3%)	0.017
QTc dispersion, mean ± SD (ms)	24 ± 29	19 ± 28	53 ± 22	<0.001
Tp-e mean ± SD (ms)	79.10 ± 20.46	77.77 ± 20.7	80.45 ± 20.18	0.152
Tp-e / QTc mean ± SD	0.20 ± 0.09	0.20 ± 0.10	0.20 ± 0.08	0.827

Values are presented as mean ± standard deviation, median [interquartile range], or n (%), as appropriate; abbreviations: mRS: modified Rankin Scale; NIHSS: National Institutes of Health Stroke Scale; QTc: corrected QT interval; Tp-e: Tpeak–end interval

Tp-e interval, and the Tp-e/QTc ratio were not significantly associated with neurological outcome.

In multivariable Cox regression (Table 2), age, NIHSS, mean QTc, and QTc > 480 ms remained independently associated with a higher risk of functional disability at discharge.

Secondary Outcome: In-Hospital Mortality

In univariable Cox analysis (Table 3), neither QTc nor other repolarization parameters were significantly associated with in-hospital mortality. In multivariable Cox analysis (Table 3), only the NIHSS score was independently associated with increased in-hospital mortality.

Discussion

In this study, we demonstrated that a prolonged QTc interval was significantly associated with worse neurological outcomes in patients with acute stroke (Central Figure). Other repolarization parameters – such as QTc dispersion, Tp-e interval, and the Tp-e/QTc ratio – were not associated with neurological outcomes or in-hospital mortality.

Previous studies have reported similar findings. A recent systematic review and meta-analysis further supported the association between QTc prolongation and adverse outcomes after acute stroke.[11] Stead et al.,[9] in a cohort of 345 patients, found that longer QTc intervals were associated with poorer functional outcomes at 90 days, using gender-specific cutoffs of 450 ms for women and 430 ms for men.[9] In 2016, Hromádka et al. reported that QTc prolongation 48 hours after stroke onset was associated with worse neurological prognosis in a small cohort of 69 patients.[10] Other studies using a 440 ms cutoff also reported poor neurological outcomes during the acute phase.[16]

In contrast, our study adopted a higher QTc cutoff of 480 ms, commonly used as a threshold for increased risk of malignant arrhythmias. Even with this more conservative threshold, QTc prolongation remained associated with worse neurological outcomes, supporting its role as a prognostic marker in acute stroke.

These findings suggest that QTc prolongation may reflect more extensive neurological injury, potentially mediated by disturbances in the brain–heart axis.[9,14,33,34] Ventricular repolarization abnormalities after stroke have been described for more than two decades.[13,18,21,33,35] Our study adds to this body of knowledge by demonstrating these associations in a mixed population of ischemic and hemorrhagic stroke and by using a higher QTc cutoff.

The observed changes are likely related to autonomic dysregulation, myocardial stunning, and catecholamine-mediated

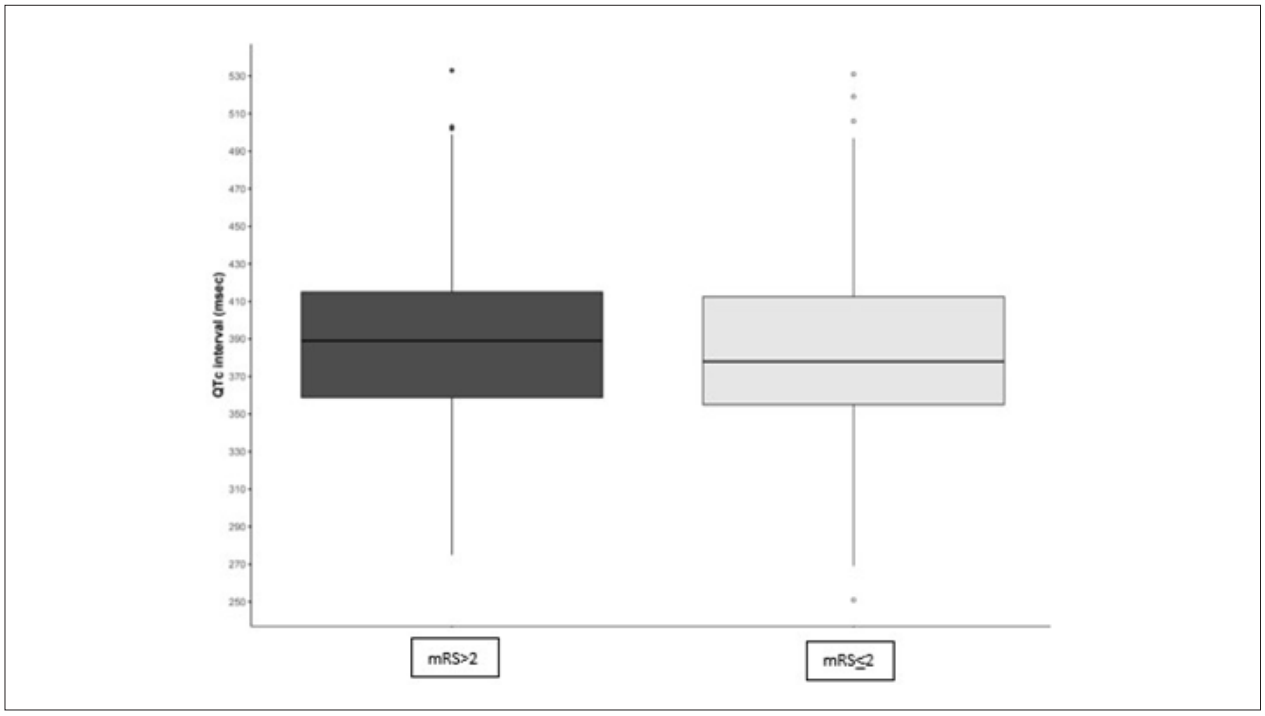


Figure 3 – *Comparison of QTc interval (ms) between patients with non-disabling stroke (mRS ≤ 2) and disabling stroke (mRS > 2). Boxplots represent the median (horizontal line), first quartile (Q1, 25th percentile), and third quartile (Q3, 75th percentile). The interquartile range (IQR) is defined as Q3–Q1. Whiskers extend to values within 1.5 × IQR, and outliers are shown as individual points. Patients with worse functional outcomes exhibited significantly longer QTc intervals at admission (p < 0.001).*

Table 2 – Univariable and multivariable regression analyses for predictors of disabling stroke (mRS > 2) at hospital discharge

Variable	Univariable HR	95% CI	p	Multivariable HR	95% CI	p
Age	1.022	1.008–1.036	<0.001	1.021	1.005–1.038	0.010
Coronary artery disease (CAD)	1.005	0.649–1.556	0.982	1.106	0.545-2.247	0.780
Hypertension	1.231	0.854–1.775	0.265	0.698	0.359-1.359	0.287
Diabetes	1.207	0.835–1.746	0.316	1.136	0.595-2.169	0.700
NIHSS at baseline	18.903	8.009–44.61	<0.001	18.55	7.834–43.95	<0.001
QTc mean	1.010	1.005–1.015	<0.001	1.009	1.003–1.015	0.001
QTc > 480 ms	2.434	1.173–5.048	0.017	2.676	1.136–6.307	0.024
QTc dispersion	1.004	0.998–1.009	0.204	1.011	0.995-1.028	0.168
Tp-e	1.006	0.997–1.015	0.174	0.998	0.982-1.015	0.835
Tp-e/QTc	0.690	0.098–4.869	0.710	0.216	0.004-11.65	0.449

Values are presented as hazard ratios (HR) with 95% confidence intervals (CI). Variables included in the multivariable model were selected based on clinical relevance and/or results from the univariable analysis; Abbreviations: mRS: modified Rankin Scale; HR: hazard ratio; CI: confidence interval; NIHSS: National Institutes of Health Stroke Scale; QTc: corrected QT interval; Tp-e: Tpeak–end interval.

myocardial injury. Some studies have specifically implicated insular cortex involvement in these ECG changes;[12,13,15] however, our study did not evaluate specific brain regions.

Despite the association with neurological outcomes, QTc and other repolarization markers were not significantly associated with in-hospital mortality. This contrasts with prior findings, such as those by Stead et al.[9] and Wong et al.,[21,33] who reported increased mortality risk with QTc prolongation at 90 days. A possible explanation is that our study evaluated only in-hospital mortality rather

Table 3 – Univariable and multivariable regression analyses for predictors of in-hospital mortality

Variable	Univariable HR	95% CI	p	Multivariable HR	95% CI	p
Age	1.018	0.998–1.039	0.083	1.020	0.996-1.045	0.104
Coronary artery disease (CAD)	1.018	0.524–1.979	0.958	1.106	0.545-2.247	0.780
Hypertension	1.159	0.550–2.052	0.612	0.698	0.359-1.359	0.287
Diabetes	1.347	0.786–2.308	0.278	1.136	0.595-2.169	0.700
NIHSS at baseline	7.702	3.807–15.58	<0.001	7.328	3.578–15.00	<0.001
QTc mean	1.005	0.998–1.012	0.153	0.998	0.982-1.015	0.835
QTc > 480 ms	1.169	0.439–3.116	0.759	0.916	0.399-2.104	0.837
QTc dispersion	0.999	0.990–1.007	0.796	0.994	0.972-1.017	0.630
Tp-e	0.996	0.982–1.009	0.532	0.998	0.982-1.015	0.835
Tp-e/QTc	0.184	0.004–8.404	0.385	0.216	0.004-11.65	0.449

Values are presented as hazard ratios (HR) with 95% confidence intervals (CI). Variables included in the multivariable model were selected based on clinical relevance and/or results from the univariable analysis; HR: hazard ratio; CI: confidence interval; NIHSS: National Institutes of Health Stroke Scale; QTc: corrected QT interval; Tp-e: Tpeak–end interval

than mid- or long-term outcomes. Other repolarization markers, such as Tp-e and Tp-e/QTc, may reflect acute myocardial events more than neurological injury.[30,31,36] Their prognostic relevance might be more evident with longer follow-up.

This study included 565 patients from a tertiary, stroke-specialized center—one of the largest cohorts to date examining QTc and neurological prognosis. Our findings suggest that QTc analysis, alongside established clinical predictors, may serve as a low-cost, accessible tool to identify patients at higher risk of poor functional recovery, potentially guiding intensive rehabilitation and monitoring.

Limitations

This study has limitations. First, although designed as a retrospective cohort, follow-up was restricted to the in-hospital period, limiting the assessment of long-term outcomes. Second, as a single-center study conducted in a tertiary hospital, the findings may be subject to selection bias and may not be generalizable to other populations or healthcare settings. Third, despite multivariable adjustment, residual confounding cannot be excluded, as unmeasured factors may have influenced the observed associations. Finally, the retrospective use of electronic medical records may be associated with incomplete or inaccurate data, although the relatively large sample size strengthens the robustness of the findings.

Conclusions

In this study, a prolonged QTc interval was significantly associated with worse neurological outcomes in patients with acute stroke. Other repolarization parameters, including QTc dispersion, Tp-e, and the Tp-e/QTc ratio, were not associated with functional outcomes or in-hospital mortality. Prospective studies with longer follow-up are warranted to further investigate these associations. If confirmed, these findings may support the development of prognostic tools based on electrocardiographic parameters and may complement future multimodal prognostic strategies integrating clinical, imaging, and biological markers.

Author Contributions

Conception and design of the research: Santos CBL, Martins SCO, Pimentel M; acquisition of data: Santos CBL, Ferreira Filho SF, Angst M, Martins SCO, Pimentel M; analysis and interpretation of the data: Santos CBL, Ferreira Filho SF, Pimentel M; statistical analysis and critical revision of the manuscript for intellectual content: Santos CBL, Silveira AD, Pimentel M; writing of the manuscript: Santos CBL, Pimentel M.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

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Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Hospital de Clínicas de Porto Alegre under the protocol number 2020-0342. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

During the preparation of this work, the author(s) used ChatGPT 5.5. After using this tool/service, the author(s) reviewed and edited the content as necessary and take full responsibility for the content of the published article.

Availability of Research Data

The individual-level data cannot be made publicly available due to ethical and confidentiality considerations regarding the protection of participants' personal and clinical information. De-identified data may be made available upon reasonable request, subject to approval by the appropriate ethics committee and applicable data protection regulations.

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