



Corrected QT dispersion in patients with acute cerebrovascular disease: A cross-sectional study

¹Dr. Sumant R Gosavi, ²Dr. Vikas S Gupta

¹ Assistant Professor, Department of General Medicine, MGM Medical College and Hospital, Kamothe, Navi Mumbai

² Assistant Professor, Department of paediatrics, MGM Medical College and Hospital, Kamothe, Navi Mumbai

OPEN ACCESS

Key Words

Acute cerebrovascular disease; stroke; corrected QT dispersion; QTc; NIHSS; electrocardiography; ventricular repolarization

Corresponding Author

Dr. Sumant R Gosavi, Assistant Professor, Department of General Medicine, MGM Medical College and Hospital, Kamothe, Navi Mumbai

Email: sumantgosavi@googlemail.com

Received: 09 Sep 2026

Accepted: 30 Sep 2026

Published: 02 Oct 2026

Copy Right: MAK HILL Publications

ABSTRACT

Background: Acute cerebrovascular disease (CVD) may produce cardiovascular autonomic disturbances and electrocardiographic abnormalities, including altered ventricular repolarization. Corrected QT dispersion (QTcd), the difference between the maximum and minimum corrected QT intervals across the 12-lead electrocardiogram, has been studied as a marker of heterogeneity of ventricular repolarization. **Objective:** To assess QTcd in patients presenting with acute CVD and determine its relationship with stroke severity measured by the National Institutes of Health Stroke Scale (NIHSS). **Methods:** This cross-sectional study included 60 patients admitted with acute CVD within 48 hours of symptom onset. Patients with recurrent stroke, transient ischemic attack, heart failure, diabetes mellitus, structural heart disease, coronary artery disease, hypertensive heart disease, arrhythmias or conduction abnormalities, electrolyte abnormalities, renal or hepatic dysfunction, and use of QT-prolonging drugs were excluded. A standard 12-lead ECG was obtained on admission. QT intervals were manually measured in all leads and corrected using Bazett's formula. QTcd was calculated as QTcmax minus QTcmin; >70 ms was considered abnormal. Stroke severity was assessed using NIHSS. Data were analyzed using SPSS, correlation tests, t tests and one-way ANOVA. **Results:** The mean age was 60.25±12.33 years; 39 (65.0%) were men. Infarction accounted for 52 (86.7%) cases and haemorrhage for 8 (13.3%). The mean QTcmax, QTcmin and QTcd were 433.45±34.22 ms, 328.38±33.90 ms and 104.75±15.34 ms, respectively. QTcd showed a strong positive correlation with NIHSS score ($r=0.775$, $p<0.001$), while QTcmax also correlated positively with NIHSS ($r=0.471$, $p<0.001$). QTcmin was not significantly correlated with NIHSS ($r=0.119$, $p=0.364$). QTcd was higher in hemorrhagic stroke than infarction (121.13±15.08 vs 102.23±13.87 ms; $p=0.001$). Across NIHSS severity categories, mean QTcd increased from 78.0 ms in the minor-stroke category to 108.67±13.73 ms in the severe-stroke category (ANOVA $p<0.001$). **Conclusion:** In this study population, QTcd was above the predefined abnormal threshold in all patients and was strongly associated with clinical stroke severity. Admission ECG assessment may therefore provide useful information for identifying patients with greater neurological severity who may warrant close cardiac monitoring.

INTRODUCTION

Acute cerebrovascular disease (CVD), comprising predominantly ischemic and hemorrhagic stroke, is an important cause of neurological morbidity and mortality [1]. Although the primary pathology occurs within the brain, acute cerebral injury may also produce significant cardiovascular manifestations. The central nervous system and cardiovascular system are closely interconnected through the autonomic nervous system, with the hypothalamus and brainstem playing important roles in cardiovascular regulation [2]. Disturbance of these pathways during acute cerebrovascular disease may result in abnormalities of cardiac autonomic function and electrocardiographic changes [3].

Electrocardiographic abnormalities observed during acute stroke may range from ST-segment and T-wave changes to abnormalities of ventricular repolarization and cardiac rhythm. These cardiovascular alterations have been described as part of the cardiac consequences of acute cerebral injury [4]. The occurrence of cardiac dysrhythmias in patients with acute cerebrovascular disease is of particular clinical interest because they may complicate the acute course of stroke. Consequently, assessment of cardiac electrical activity is an important component of the clinical evaluation of patients with acute cerebrovascular disease [5].

QT interval dispersion (QTd) is defined as the difference between the maximum and minimum QT intervals measured across the standard 12-lead electrocardiogram [6]. It has been used as an electrocardiographic measure of the heterogeneity of ventricular repolarization. Increased QT dispersion indicates greater variation in ventricular repolarization among different regions of the myocardium and has been investigated as a marker of electrical instability. Since the QT interval is influenced by heart rate, correction for heart rate is required [7]. Corrected QT dispersion (QTcd) can therefore be assessed as the difference between the maximum and minimum corrected QT intervals [8]. The severity of neurological impairment in acute stroke can be assessed using the National Institutes of Health Stroke Scale (NIHSS). The NIHSS provides a standardized clinical assessment of neurological deficits and allows the clinical severity of stroke to be quantified [9]. While previous studies have examined QT dispersion in relation to the type or anatomical characteristics of stroke, assessment of the relationship between QTcd and clinically determined stroke severity may provide additional

information regarding the cardiovascular response to acute cerebral injury [10].

QTcd is obtained from a routine, non-invasive, and widely available electrocardiographic examination. Therefore, assessment of QTcd may provide a simple method for identifying alterations in ventricular repolarization in patients with acute cerebrovascular disease. However, the clinical significance of QTcd in relation to the severity of acute cerebrovascular disease requires further evaluation [10].

Therefore, the present study was undertaken to assess corrected QT dispersion in patients with acute cerebrovascular disease and to determine its relationship with the severity of stroke as assessed by the National Institutes of Health Stroke Scale. The study also evaluated the association of QTcd with selected clinical characteristics, including age, sex, smoking status, type of cerebrovascular lesion, and side of the lesion.

AIM AND OBJECTIVES

- 1.To study corrected QT dispersion (QTcd) in patients with acute cerebrovascular disease.
- 2.To correlate QTcd with the severity of acute cerebrovascular disease based on the NIHSS.

MATERIALS AND METHODS

Study design

This was a cross-sectional study conducted in the Department of General Medicine. The study was conducted over a period of two years. Institutional Ethics Committee approval was obtained.

Study population and sample

60 patients admitted to the medical intensive care unit and medical wards with acute cerebrovascular disease were included. Patients presenting with acute cerebrovascular accident within 48 hours of symptom onset were eligible.

Eligibility criteria

Patients with recurrent CVA, transient ischemic attack, heart failure, diabetes mellitus, structural heart disease, coronary artery disease, hypertensive heart disease, arrhythmias or conduction disturbances, electrolyte abnormalities, renal or hepatic dysfunction, or treatment with drugs known to prolong the QT interval were excluded.

Clinical and ECG assessment

After informed consent from the patient or relative, history and clinical examination were performed. Routine laboratory investigations, including renal function tests and electrolytes, echocardiography and plain CT brain were performed. A standard 12-lead ECG was obtained on admission. QT intervals

were measured manually in all leads and corrected for heart rate using Bazett's formula: $QT_c = QT/VRR$, with QT and RR expressed in seconds. QTcmax was the maximum corrected QT interval and QTcmin the minimum corrected QT interval among the measured leads. QTcd was calculated as QTcmax – QTcmin in milliseconds. QTcd >70 ms was considered abnormal.

Assessment of stroke severity

Stroke severity was assessed using the NIHSS. In this study, NIHSS categories were defined as: 1–4, minor stroke; 5–15, moderate stroke; 16–20, moderate-to-severe stroke; and 21–42, severe stroke.

Statistical analysis

Data were entered and analyzed using SPSS. Continuous variables were summarized as mean and standard deviation. Pearson correlation was used to examine relationships between NIHSS and QT parameters. Independent-samples t tests were used for two-group comparisons, and one-way ANOVA was used for comparisons across age and NIHSS categories. A p value <0.05 was considered statistically significant.

RESULTS

Table 1. Baseline demographic and clinical characteristics of the study population.

Characteristic	n	%
Age 30–40 years	4	6.7
Age 41–60 years	28	46.7
Age 61–80 years	25	41.7
Age >80 years	3	5.0
Male	39	65.0
Female	21	35.0
Smoker	20	33.3
Non-smoker	40	66.7
Infarction	52	86.7
Hemorrhage	8	13.3
Left-sided lesion	42	70.0

Characteristic	n	%
Right-sided lesion	18	30.0

Among the 60 participants, 28 (46.7%) were aged 41–60 years and 25 (41.7%) were aged 61–80 years. Thirty-nine (65.0%) participants were male and 21 (35.0%) female. Twenty participants (33.3%) were smokers. Cerebral infarction was present in 52 (86.7%) participants and cerebral hemorrhage in 8 (13.3%). The lesion was left-sided in 42 (70.0%) and right-sided in 18 (30.0%) participants.

Table 2. Overall NIHSS and corrected QT parameters.

Parameter	Mean ± SD	Range
NIHSS score	25.95 ± 7.38	4–38
QTcmax (ms)	433.45 ± 34.22	354–505
QTcmin (ms)	328.38 ± 33.90	275–397
QTcd (ms)	104.75 ± 15.34	75–142

QT parameters and NIHSS The overall mean NIHSS score was 25.95±7.38. The mean QTcmax was 433.45±34.22 ms, QTcmin was 328.38±33.90 ms, and QTcd was 104.75±15.34 ms (range 75–142 ms). Thus, all 60 participants had QTcd values above the predefined abnormal threshold of 70 ms.

Table 3. Correlation between NIHSS and QT parameters.

Variable vs NIHSS	Pearson r	p value	Interpretation
QTcd	0.775	<0.001	Strong positive correlation
QTcmax	0.471	<0.001	Positive correlation
QTcmin	0.119	0.364	Not statistically significant

QTcd demonstrated a strong positive correlation with NIHSS ($r=0.775$, $p<0.001$). The corresponding coefficient of determination was $R^2=0.6007$. QTcmax also correlated positively with NIHSS ($r=0.471$, $p<0.001$; $R^2=0.2217$). QTcmin did not show a significant correlation with NIHSS ($r=0.119$, $p=0.364$).

Table 4. QTcd according to type of cerebrovascular disease.

Stroke type	n	QTcd mean $\pm$ SD (ms)	p value
Infarction	52	102.23 $\pm$ 13.87	
Hemorrhage	8	121.13 $\pm$ 15.08	0.001

Mean QTcd was significantly higher among patients with hemorrhagic stroke than among those with infarction (121.13 $\pm$ 15.08 vs 102.23 $\pm$ 13.87 ms; $p=0.001$). NIHSS was also higher in hemorrhagic stroke (32.50 $\pm$ 6.46 vs 24.94 $\pm$ 7.04; $p=0.006$).

Table 5. QTcd according to NIHSS severity category. One-way ANOVA: $F=8.201$, $p<0.001$.

NIHSS category	n	QTcd mean $\pm$ SD (ms)
1–4 (minor)	1	78.00
5–15 (moderate)	3	80.00 $\pm$ 6.25
16–20 (moderate-to-severe)	8	93.88 $\pm$ 10.01
21–42 (severe)	48	108.67 $\pm$ 13.73
Total	60	104.75 $\pm$ 15.34

Other subgroup findings QTcd did not differ significantly by sex (male 102.56 $\pm$ 15.72 ms vs female 108.81 $\pm$ 14.07 ms; $p=0.134$), smoking status (smokers 103.35 $\pm$ 16.67 ms vs non-smokers 105.45 $\pm$ 14.79 ms; $p=0.621$), or lesion laterality (left-sided 104.02 $\pm$ 15.54 ms vs right-sided 106.44 $\pm$ 15.16 ms; $p=0.580$). QTcd differed significantly across age groups (ANOVA $F=3.172$, $p=0.031$).

DISCUSSION

The present study evaluated corrected QT dispersion

in 60 patients presenting with acute cerebrovascular disease within 48 hours of symptom onset and examined its relationship with neurological severity. The principal finding was a strong positive association between QTcd and NIHSS score. The mean QTcd was 104.75 $\pm$ 15.34 ms, and every participant had a QTcd above the predefined threshold of 70 ms.

The demographic profile was broadly comparable with earlier studies. The mean age in the present study was 60.25 $\pm$ 12.33 years, compared with 68 $\pm$ 12 years in the study by Afsar et al. [11], 61.8 $\pm$ 16.1 years in the study by Chao et al. [12], and 62 $\pm$ 10.7 years in the study by Alabd et al. [13]. Men constituted 65% of the present study population, similar to the male proportions reported in the cited studies.

Cerebral infarction predominated in the present study, accounting for 86.7% of cases, while hemorrhagic stroke accounted for 13.3%. A predominance of ischemic stroke has also been reported in previous studies evaluating electrocardiographic and QT/QTc dispersion changes in acute cerebrovascular disease. Jain et al. [14] reported infarction in 61.5% of their acute neurological event cohort compared with 34.6% with hemorrhagic stroke.

The mean QTcd in the present study was higher than the values reported by Afsar et al. [11] (56 $\pm$ 19 ms) and Alabd et al. [13] (62 $\pm$ 21 ms), but lower than the mean reported by Chao et al. [12] in acute hemorrhagic stroke (132 $\pm$ 51.9 ms). Direct numerical comparisons should be interpreted cautiously because the study populations, stroke subtypes, inclusion criteria and methods of QT measurement differed. Unlike several cited studies, the present study did not include a healthy control group; therefore, the study demonstrates the distribution of QTcd within the acute CVD cohort rather than providing a case-control estimate of the excess QTcd attributable to stroke.

A notable finding was the significantly greater QTcd among patients with hemorrhagic stroke compared with infarction. Our results showed mean QTcd values of 121.13 $\pm$ 15.08 ms and 102.23 $\pm$ 13.87 ms, respectively. Hemorrhagic stroke also had a higher mean NIHSS score. Thus, the difference in QTcd between stroke subtypes may be related, at least in part, to differences in neurological severity, although the present study was not designed to establish an independent effect of stroke subtype.

The strongest statistical finding was the correlation between QTcd and NIHSS ($r=0.775$, $p<0.001$). QTcd increased progressively across the NIHSS severity categories, with the highest mean value in the

severe-stroke group. This supports the study hypothesis that greater clinical neurological impairment is accompanied by greater inter-lead variability in ventricular repolarization. These ECG changes may be related to disturbances in autonomic cardiovascular regulation following acute brain injury, including sympathetic hyperactivity, reduced parasympathetic activity and abnormal baroreceptor function.

The findings are consistent with earlier studies. Afsar et al. [11] reported increased QT dispersion in patients with acute stroke without known cardiac disease. The present study extends these observations by relating QTcd to NIHSS, a clinical measure of stroke severity, rather than relying solely on imaging characteristics.

Clinical implications

Because QTcd can be derived from a standard 12-lead ECG, its measurement may provide an inexpensive additional marker of altered ventricular repolarization in acute CVD. In this study, higher QTcd was associated with greater NIHSS severity. The finding supports careful ECG assessment and cardiac monitoring during the acute phase of stroke, particularly in patients with more severe neurological impairment. However, the cross-sectional design does not establish that QTcd predicts arrhythmia, mortality or functional outcome.

Limitations

First, the study had a relatively small sample size of 60 patients and no control group. Second, NIHSS may rate left-sided lesions differently because of the weighting of language-related functions. Third, Bazett's formula was used for heart-rate correction; this formula may overcorrect at higher heart rates and undercorrect at lower heart rates. Fourth, although history, examination, ECG and echocardiography were used to assess cardiac disease, coronary angiography was not performed to exclude occult coronary artery disease.

Conclusion

In this cross-sectional cohort of patients with acute cerebrovascular disease, corrected QT dispersion was above the predefined abnormal threshold in all participants and showed a strong positive correlation with NIHSS score. QTcd was significantly higher in hemorrhagic than infarctive stroke and increased across NIHSS severity categories. These findings indicate an association between clinical stroke severity and altered ventricular repolarization as reflected by QTcd. Standard ECG assessment, together with appropriate cardiac monitoring, may

therefore be useful during the acute management of patients with cerebrovascular disease.

DECLARATIONS

Funding: Source of Funding: Nil.

Ethics Approval: Institutional Ethics Committee approval was obtained for the study.

Patient Consent: Informed consent was obtained from each patient or the patient's relative.

Conflict of Interest: Conflict of Interest: Nil.

Data Availability: The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

REFERENCES

1. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke. *Stroke*. 2019;50(12):e344-e418.
2. Mo J, Li M, Chen X, Li S, Xu Y. Autonomic disturbances in acute cerebrovascular disease. *Neurosci Bull*. 2019;35(1):133-144.
3. Sposato LA, Hilz MJ, Aspberg S, Murthy SB, Bahit MC, Hsieh CY, et al. Cardiovascular autonomic dysfunction after stroke. *Stroke*. 2021;52(6):2188-2202.
4. Faiz KW, Bjerkreim AT, Logallo N, et al. The heart-brain axis: cardiac complications after stroke. *Front Neurol*. 2021;12:626668.
5. Hays AG, Sacco RL, Rundek T. Stroke and ventricular arrhythmias. *J Electrocardiol*. 2013;46(5):416-421.
6. Day CP, McComb JM, Campbell RWF. QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. *Br Heart J*. 1990;63(6):342-344.
7. Malik M, Batchvarov VN. Measurement, interpretation and clinical potential of QT dispersion. *J Am Coll Cardiol*. 2000;36(6):1749-1766.
8. Krishnappa D, Akdemir B, Nantsupawat T, et al. QTc dispersion as a novel marker in identifying patients requiring an epicardial approach for ablation of scar mediated ventricular tachycardia. *Indian Pacing Electrophysiol J*. 2020;20(4):141-146.
9. Lyden PD, Lu M, Levine SR, Brott TG, Broderick J; NINDS rtPA Stroke Study Group. A modified National Institutes of Health Stroke Scale for use in stroke clinical trials: preliminary

- reliability and validity. *Stroke*. 2001;32(6):1310-1317.
10. Lederman Y, Balucani C, Lazar J, Lee V, Arnedo V, Lushbough C, et al. Relationship between QT interval dispersion in acute stroke and stroke prognosis: a systematic review. *J Stroke Cerebrovasc Dis*. 2014;23(10):2469-2475.
 11. Afsar N, Fak AS, Metzger JT, Van Melle G, Kappenberger L, Bogousslavsky J. Acute stroke increases QT dispersion in patients without known cardiac diseases. *Arch Neurol*. 2003;60(3):346-350.
 12. Chao CC, et al. QT dispersion is associated with the severity of neurological impairment and mortality in patients with acute intracerebral hemorrhage. *J Chin Med Assoc*. 2009;72(3):124-128.
 13. Alabd AA, Fouad A, Abdel-Nasser R, Nammass W. QT interval dispersion pattern in patients with acute ischemic stroke: does the site of infarction matter? *Can J Cardiol*. 2009;25(11):e370-e373.
 14. Jain H, Khan M, Raghavendra S, et al. The QT dispersion and QTc dispersion in patients presenting with acute neurological events and its impact on early prognosis. *J Neurosci Rural Pract*. 2016;7(1):65-70.