Original Article

A study on the electrocardiographic findings in acute stroke, a case controlled study in a tertiary hospital in Eastern India

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Abstract

Context: In cerebrovascular accident (CVA) patients, electrocardiogram (ECG) changes may or may not be due to underlying cardiac illness. **Aims:** This study was done on CVA patients without underlying cardiac illness and diabetes, to evaluate the incidence and patterns of ECG changes in acute stroke. **Settings and Design:** Prospective nonrandomized case–control study in a tertiary hospital. **Subjects and Methods:** Ninety-seven CVA patients as case and 97 patients' age and sex matched, attending preanasthetic check-up without cardiac illness and diabetes were taken as control. ECG computed tomography brain, magnetic resonance imaging brain (in inconclusive situations) were done. **Statistical Analysis Used:** Chi-square test and Levine test using appropriate software IBM SPSS Version 22. **Results:** Among 97 CVA subjects, 80 had hemorrhage and 17 had infarcts. 55 lesions were situated in the right hemisphere and 42 in the left hemisphere. ECG changes were present in 89.6% patients (87 of 97). Among control 22.6% (22 of 97) had new ECG changes ($P < 0.01$). The most common ECG changes were prolonged QT_c interval (78/97) 80.4%, increased QT_c dispersion (QT_{cD}) (66/97) 71%, and ST-T changes (16/97) 16.5%. Hemorrhagic strokes had more QT_c prolongation (71/80) (81%) than ischemic CVA (7/17) (41%) ($P < 0.001$) QT_{cD} was more with hemorrhage (63/80 = 79%) than with ischemia (6/17 = 35.3%) ($P < 0.001$) QT_{cD} increase had increased mortality (29/69 = 42%) than with no QT_{cD} increase (1/28 = 3.5%) ($P < 0.001$). Ischemic CVA had more ST-T changes (8/17 = 47%) than hemorrhage (8/80 = 10%) ($P < 0.01$). **Conclusions:** This study showed increased incidence of ECG changes following CVA. QT_c and QT_{cD} prolongation were more in hemorrhagic CVA while ST-T changes were more in ischemic CVA. Increased QT_{cD} were associated with increased short-term mortality.

Key words: Clinical significance, electrocardiographic changes, stroke

INTRODUCTION

Changes in electrocardiogram (ECG) in stroke may reflect deranged central nervous system (CNS) influences on cardiac autonomic function. There might also be an actual concomitant myocardial injury that may or may not be due to underlying cardiac disease.[1] Lateralization studies indicate that destruction of areas adjacent to the right insular cortex has specially marked cardiac effects.[2]

Several studies done on the development of stroke and new ECG changes, its relation with the type of stroke and prognosis with variable results.[3,4]

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This present study was done on CVA patients without underlying cardiac illness and diabetes, to evaluate the incidence & and patterns of ECG changes in acute stroke.

The present study aims at detecting new ECG changes such as QT dispersion, QT duration, ST-T changes, its relation with the type of stroke and prognosis.

SUBJECTS AND METHODS

Ninety-seven stroke patients admitted to General Medicine wards during February 2013–October 2014 were selected for the study. Patients with history of diabetes, Ischemic Heart disease, arrhythmias/previous ECGs showing arrhythmias, ingestion of drugs known to produce a QT prolongation or predispose to arrhythmias including antiarrhythmics, presence of dyselectrolytemia were excluded from the study. Ninety-seven patients' age and sex matched, attending preanasthetic check-up for a disease other than cardiac illness and diabetes were taken as control. Detailed clinical history and examination and imaging with a noncontrast computed tomography (CT) scan of brain and magnetic resonance imaging brain (if CT brain inconclusive), ECG recording-12 lead with long lead II within 24 h of admission, laboratory tests like blood glucose, electrolytes, etc., were done. Outcome at the end of hospital stay was considered as short-term.

ECG – Paper speed was 25 mm/s. 1 mm = 1 mV. Following parameters were evaluated:

- QT_c – Corrected QT calculated using Bazett formula[5] – $QT_c = QT/\sqrt{(RR)}$. A cut-off of 460 ms (females) and 440 ms (males) was used-values above considered increased for the purposes of this study
- QT_c dispersion (QT_{cD}) – Corrected QT dispersion calculated first by calculating the longest QT_c in each lead and then calculating the difference between the highest and lowest among the 12 QT_c calculated thus. A lot of variabilities exist regarding normal values of QT_{cD}. However, values ranging from 10 to 71 ms have been noted.[6] A QT_{cD} value >80 ms have been used to define "increased QT dispersion" for this study
- ST-T changes – following changes were considered:
  - ST elevation
  - ST depression
  - Tall T waves (>10 mV)
  - T wave inversion
  - Nonspecific ST-T changes.

- Conduction disturbances – following conduction disturbances were considered:
  - 1°/2°/Complete heart block
  - Right bundle branch block/left bundle branch block
  - Bi-fascicular block
  - Stroke volume assessment.

The ABC/2 formula[7] was used for calculating approximate stroke volume in "ml." It was used for both infarction and hemorrhage.

A = longest diameter in X-axis (cm)
B = longest diameter in Y-axis (cm)
C = (number of slices × slice thickness in cm) (cm)

Slices with lesion volume <25% of the maximum volume slice were excluded from the calculation.

The volume was approximated to the nearest whole no.

RESULTS

This study was completed with a sample $n = 97$ with 65 males and 32 females. Most patients (86) belong to 40–79 years age group. Ninety-seven patients' age and sex matched, attending preanasthetic check-up for a disease other than cardiac illness and diabetes were taken as control. Regarding hemispheric localization, there was slight right hemispherical predominance over left hemisphere (55 vs. 42). Regarding nature, there was overwhelmingly larger number of hemorrhagic cerebrovascular accident (CVA) as opposed to ischemic CVA (80 vs. 17). Among the specific lesions basal ganglia lesions topped the list (30%) closely followed by thalamic (18%). In them also hemorrhage was the predominant lesion.

ECG changes were found in (87/97) 89.6% of subjects – only 10.4% did not have any ECG changes. Among control 22 out of 97 (22.6%) had ECG changes. Regarding the ECG changes the two most predominant ECG changes were prolonged QT_c and increased QT_{cD}. (78/97) 80.4% of the sample had increased QT_c while nearly (66/97) 71% had increased QT_{cD}. ST-T changes were found to be (16/97) 16.5%. The ECG changes are given in [Table 1].

Increased QT_c and QT_{cD} values and ST-T change in patients of right and left hemispheric CVA and in hemorrhagic and ischemic CVA were compared and statistical analysis were done using Chi-square test [Tables 2 and 3].

Levine's test for equality of variances showed no increase in QT_c with increase of stroke volume but the increase in QT_{cD} with an increase of stroke volume ($P = 0.001$). There is no significant increase in ST-T with increased stroke size.

Table 1: Frequency of electrocardiographic changes

ECG changes	Percentage of sample
Sinus tach	10
Sinus brad	5
RAD	4
LAD	15
↑ QT_c	80.4
↑ QT_{cD}	71
ST-T	16.5

ECG: Electrocardiographic, RAD: Right axis deviation, LAD: Left axis deviation, QT_{cD}: QT_c dispersion

Table 2: Increased QT_c and QT_c dispersion values and ST-T change in patients of right and left hemispheric cerebrovascular accident and their statistical significance

Parameters	Right hemisphere %	Left hemisphere %	*P*
Increased QT_c	80.3 (45/56)	80.4 (33/41)	0.599
Increased QT_{cD}	64.2 (36/56)	80.5 (33/41)	0.09
ST-T changes	21.4 (12/56)	9.7 (4/41)	0.4

QT_{cD}: QT_c dispersion

Table 3: Increased QT_c and QT_c dispersion values and ST-T change in patients of hemorrhagic and ischemic cerebrovascular accident and their statistical significance

Parameters	Hemorrhagic stroke %	Ischemic stroke %	*P*
QT_c prolongation	81 (71/80)	41 (7/17)	<0.001
Increased QT_{cD}	79 (63/80)	35.3 (6/17)	<0.001
ST-T changes	10 (8/80)	47 (8/17)	<0.001

QT_{cD}: QT_c dispersion

In our study, mortality was calculated using Fisher's exact test. QT_c prolongation associated mortality (28/78 = 36%) was not significantly more than in no QT_c prolongation (2/19 = 11%) ($P = 0.045$). It was found that QT_{cD} increase was associated with significantly increased mortality (29/69 = 42%) than with no QT_{cD} increase (1/28 = 3.5% mortality) ($P < 0.001$). Difference in mortality in ST-T change (4/16 = 25%) and no ST-T change (26/55 = 47%) ($P = 0.4$) were not significant.

Our study found significant mortality associated with QT_{cD} in much shorter term.

DISCUSSION

The study was done with the aim of grossly exploring ECG findings in acute stroke with special reference to QT dispersion, QT duration, and ST-T changes and its relation with the type of stroke, hemispheric localization and size with short-term prognosis.

This study was completed with a sample $n = 97$ with 65 males and 32 females. Regarding hemispheric localization of CVA, there was slight right hemispherical predominance over left hemisphere (55 vs. 42). Regarding nature, there was overwhelmingly larger number of hemorrhages as opposed to infarction (80 vs. 17). As the study was done in a tertiary care hospital, may be, hemorrhagic CVA with more overt clinical manifestations were admitted more. Among the specific lesions basal ganglia lesions topped the list (30%) followed by thalamic (18%). In them also, hemorrhage was the predominant lesion.

Regarding the distribution of ECG findings, ECG changes were found in 89.6% of subjects – only 10.4% did not have any ECG changes. Among control 22.6% (22 of 97) had new ECG changes. ECG changes were significantly higher in CVA patients ($P < 0.01$). This is also reflected in other studies like Bozluolcay *et al.*, where the frequency of the ECG changes observed in patients with cerebral infarct was 62.1% while it was 29.9% in the control group ($P < 0.0001$).[8] Ebrahim *et al.* found similar figures in his study of electrocardiographic changes in acute ischemic stroke where he found ECG changes in 68% of the subjects as compared to 30% of control.[9] Goldstein noted in his study with 150 patients 138 (92%) showed ECG abnormalities which is quite high.[3]

Changes in ECG in acute stroke may reflect deranged CNS influences on cardiac autonomic function in the form of autonomic neural stimulation from the hypothalamus or elevated circulating catecholamines. There might also be an actual concomitant myocardial injury that may or may not be due to underlying cardiac disease.[1] In our study, assessment for correlation between new ECG change and catecholamine level could not be done.

Regarding the ECG changes the two most predominant ECG changes were prolonged QT_c and increased QT_{cD}. 80.4% of the sample had increased QT_c while nearly 71% had increased QT_{cD}: Goldstein noted in his study of 180 patients, QT prolongation (68 patients, 45%), ischemic changes (59, 35%)[3] Fure *et al.* found the most frequent ECG changes were: Prolonged QT_c 36.0%,[4] quite less compared to our study. A huge body of contemporary work has been done on exploring the importance of an increased QT_{cD} in acute stroke – its presence, correlation to stroke size, stroke site and relation to mortality. The importance of QT_{cD} stems from the fact that QT-dispersion represents inter-lead variability of QT interval and reflects the heterogeneity of myocardial repolarization. QT dispersion was originally proposed as an index of the spatial dispersion of ventricular recovery times. Attempts to characterize and quantify the nonhomogeneousity of ventricular repolarization from

the surface ECG using precise mathematical methods, such as principal component analysis of the T wave, can be traced back to the 1960s.[10] In our study, mortality was calculated using Fisher's Exact Test. It was found in our study that QT_c prolongation was not associated with significantly increased mortality (36%) compared with no QT_c prolongation (11%) ($P = 0.045$).

Wong *et al.*[11] showed in his study that the increased QT_c measured from any lead of the ECG (except aVR) was associated with increased death from any cause. A prolonged QT_c in limb lead III and chest lead V6 carried the highest relative risk of cardiac death (a 3.1-fold increase). After adjusting for overt ischemic heart disease, pulse pressure, glucose, and cholesterol, a prolonged QT_c in lead V6 was associated with a relative risk of cardiac death of 2.8 (95% confidence interval [CI] 1.1–7.3) ($P = 0.028$). If the QT_c in V6 exceeded 480 ms, then the specificity of predicting cardiac death within 5 years after the stroke was 94%.[11] In our study, we did not compare the magnitude of QT_c prolongation with mortality, but patients with prolonged QT_c did not have higher mortality in short-term.

This was in contrast to the study of Wong *et al.*[11] The frequency of QT_{cD} in our study was to the tune of 72%. It was the predominant finding in both hemorrhagic and ischemic strokes, but it was significantly more in hemorrhagic CVA (79%) than ischemic strokes (35.3%) ($P < 0.001$). Although, left hemispheric CVA had more QT_{cD} than right hemispheric CVA (80.5% vs. 64.2%), it was not statistically significant ($P = 0.09$).

In our study, it was found that QT_{cD} increase was associated with increased mortality (42%) than with no QT_{cD} increase (3.5% mortality) ($P < 0.001$).

Bicakci *et al.* also conducted mortality studies related to QT_{cD} in acute stroke patients. She involved two groups of patients-one with death (Group II) at end of hospital stay and the other with survival (Group I) and ventured to find the relative prevalence of ECG changes in both. It was found that corrected QT and QT_{cD} were prolonged in both groups; but particularly Group II had relatively higher QT_{cD} compared to Group I.[12]

Lazar *et al.* found QT_{cD} was higher in patients with intercerebral hemorrhage as compared to ischemic CVA and transient ischemic attack (70 ms vs. 53 ms vs. 48 ms, respectively; $P = 0.03$). Increasing QT_{cD} was associated with lower functional outcomes on all 3 scales (all $P < 0.05$) and with higher mortality ($P = 0.02$). On multivariate analysis, other independent predictors of worse outcome were QT_{cD} (odds ratio, 1.35; 95% CI, 1.08 1.68) and a trend toward age (odds ratio, 1.07; 95% CI, 0.99 1.16). On age-adjusted logistic regression, mortality increased by odds ratio of 1.28 and 95% CI of 1.02 1.61 for every 10 ms increase in QT_{cD}. QT_{cD} is an independent predictor of functional outcome and mortality following acute neurological events. In this setting, QT_{cD} reflects neurological injury as well as underlying heart disease.[13] The findings on QT_{cD} sits very well with the findings of our study. However, in our study, the exact cause of death could not be determined. Further well-randomized control studies are required in this field.

Few studies have been done with a view to have an approximation of the particular cortical areas associated with ECG anomalies namely QT_{cD} and QT_c in view of its importance as electrocardiological substrate for fatal ventricular arrhythmias. Eckardt *et al.*[14] attempted to localize cortical areas where cerebrovascular damage was associated with prolonged QT_{cD}. In his study with 40 subjects, he found that patients with involvement of the insular cortex, the QT dispersion is significantly longer than in those without insular involvement. In our study, localization could not be done to that extent. But in our study, there was no significant hemispheric predilection for ECG changes.

New onset QT_c prolongation found at the time of stroke may be an indicator of underlying ischemic heart disease. Mortality in those subset of stroke patients with prolongation of QT_c may thus be a composite marker of underlying cardiac disease compounded by the prospect of developing fatal arrhythmias due to the prolonged QT_c. Findings of such sort were revealed in the study by Familoni *et al.*[15]

Total ST-T changes were seen in 16.5% of patients but it was quite high in ischemic CVA group - 8 of 17 (47%). This is similar to Ebrahim *et al.*[9] who found ischemic changes, ST-T changes and abnormal T waves to be the predominant findings in his series of ischemic CVA with changes in 98 (37.4%) patients and 16 (15.7%) controls. Our study was close with. Fure *et al.* where he found the most frequent ECG changes were: ST depression 24.5%, T wave inversion 17.8%.[4] Higher incidence of ST-T changes in ischemic CVA may be due to some underlying ischemic heart disease which we could not study.

ST-T changes in our study have no predilection for particular hemisphere. It has no significant association with the stroke size. ST-T changes were not associated with increased mortality outcomes–mortality in ST-T change (25%) and no ST-T change mortality (47%) ($P = 0.4$).

CONCLUSION

ECG changes of some nature were found in majority of stroke patients with majority being increased QT Dispersion (QT_{cD}) and increased QTc. While statistically significant correlation with ↑QT_{cD} and mortality was found (in consonance with contemporary studies related to neurological outcomes with QT_{cD} changes), the same couldn't be found for ↑QTc and mortality (in contrast to similar contemporary studies). Inability to quantitavely correlate QT_{cD} with mortality remains a limitation; further studies might illuminate this point as well as identify subsets of stroke patients with high risk ECG changes who might need more intensive cardiologic monitoring.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Davis TP, Alexander J, Lesch M. Electrocardiographic changes associated with acute cerebrovascular disease: A clinical review. Prog Cardiovasc Dis 1993;36:245-60.
2. Oppenheimer SM. Neurogenic cardiac effects of cerebrovascular disease. Curr Opin Neurol 1994;7:20s4.
3. Goldstein DS. The electrocardiogram in stroke: Relationship to pathophysiological type and comparison with prior tracings. Stroke 1979;10:253-9.
4. Fure B, Bruun Wyller T, Thommessen B. Electrocardiographic and troponin T changes in acute ischaemic stroke. J Intern Med 2006;259:592-7.
5. Bazett HC. An analysis of the time relations of electrocardiograms. Heart 1920;11:353-70.
6. Alabd AA, Fouad A, Abdel-Nasser R, Nammas W. QT interval dispersion pattern in patients with acute ischemic stroke: Does the site of infarction matter? Int J Angiol 2009;18:177-81.
7. Sims JR, Gharai LR, Schaefer PW, Vangel M, Rosenthal ES, Lev MH, *et al.* ABC/2 for rapid clinical estimate of infarct, perfusion, and mismatch volumes. Neurology 2009;72:2104-10.
8. Bozluolcay M, Ince B, Celik Y, Harmanci H, Ilerigelen B, Pelin Z. Electrocardiographic findings and prognosis in ischemic stroke. Neurol India 2003;51:500-2.
9. Ebrahim K, Mohamadali A, Majid M, Javad A. Electrocardiographic changes in acute iscahemic cerebral stroke. J Appl Res 2012;12:51-8.
10. Horan LG, Flowers NC, Brody DA. Principal Factor waveforms of the thoracic QRS complex. Circ Res 1964;15:131-45.
11. Wong KY, Mac Walter RS, Douglas D, Fraser HW, Ogston SA, Struthers AD. Long QTc predicts future cardiac death in stroke survivors. Heart 2003;89:377-81.
12. Bicakci S, Donmez Y, Ozeren A, Acarturk E. QT dispersion on ECG in acute ischemic stroke and its impact on early prognosis. Neurosciences (Riyadh) 2008;13:366-9.
13. Lazar J, Manzella S, Moonjelly J, Wirkowski E, Cohen TJ. The prognostic value of QT dispersion in patients presenting with acute neurological events. J Invasive Cardiol 2003;15:31-5.
14. Eckardt M, Gerlach L, Welter FL. Prolongation of the frequency-corrected QT dispersion following cerebral strokes with involvement of the insula of Reil. Eur Neurol 1999;42:190-3.
15. Familoni OB, Odusan O, Ogun SA. The pattern and prognostic features of QT intervals and dispersion in patients with acute ischemic stroke. J Natl Med Assoc 2006;98:1758-62.