

## Original Research Article

# ELECTROCARDIOGRAPHIC ABNORMALITIES IN ACUTE STROKE AND THEIR ASSOCIATION WITH STROKE SUBTYPES

Boeni Divya<sup>1</sup>, Muppavaram Pranay<sup>2</sup>, Kollabathulla Kamal Chand<sup>3</sup>

<sup>1</sup>Undergraduate Medical Student, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, Telangana, India.

<sup>2</sup>Assistant Professor, Department of Physiology, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, Telangana, India.

<sup>3</sup>Professor and Head, Department of Physiology, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, Telangana, India.

Received : 28/10/2025  
Received in revised form : 11/12/2025  
Accepted : 30/12/2025

## Corresponding Author:

Dr. Muppavaram Pranay,  
Department of Physiology, Kamineni  
Academy of Medical Sciences and  
Research Centre Hyderabad,  
Telangana, India.  
Email: mpranay96@gmail.com

DOI: 10.70034/ijmedph.2026.1.53

Source of Support: Nil,  
Conflict of Interest: None declared

Int J Med Pub Health  
2026; 16 (1); 296-300

## ABSTRACT

**Background: Objective:** To evaluate the frequency and pattern of electrocardiographic (ECG) abnormalities in patients with different types of stroke and assess their association with stroke subtype.

**Materials and Methods:** A cross-sectional observational study was conducted among 78 patients diagnosed with acute stroke at a tertiary care hospital from August 2024 to January 2025. Stroke was classified as ischaemic, haemorrhagic or transient ischaemic attack (TIA) based on neuroimaging. Standard 12-lead ECGs were analysed for abnormalities including left ventricular hypertrophy (LVH), ST-segment elevation, T-wave changes, QT prolongation and rhythm disturbances. Data were analysed using Microsoft Excel and GraphPad Prism. Comparisons across stroke subtypes were performed using Chi-square and Fisher's exact tests, with  $p < 0.05$  considered statistically significant.

**Results:** Among the 78 patients, 70.5% had ischaemic stroke, 23.1% haemorrhagic stroke and 6.4% TIA. ECG abnormalities were present in 82% of cases, the most frequent being left atrial enlargement (26.9%), ST-segment elevation (25.6%) and left ventricular hypertrophy (24.4%). LVH demonstrated a statistically significant association with stroke subtype ( $p = 0.0008$ ), with higher prevalence in haemorrhagic and TIA cases. Other ECG findings did not differ significantly between stroke groups.

**Conclusion:** ECG abnormalities are highly prevalent among patients with acute stroke, reflecting neurogenic myocardial injury and autonomic dysfunction. Routine ECG evaluation should be incorporated into acute stroke assessment to identify patients at increased risk of cardiac complications.

**Keywords:** Stroke; Electrocardiography; Ischemic Stroke; Hemorrhagic Stroke; Autonomic Nervous System Diseases.

## INTRODUCTION

Stroke remains one of the leading causes of mortality and long-term disability worldwide, contributing to nearly 10% of total deaths and 5% of all disability-adjusted life years.<sup>[1]</sup> It encompasses a heterogeneous group of cerebrovascular disorders characterised by the sudden loss of neurological function due to either vascular occlusion or rupture. The two principal categories, ischaemic stroke and haemorrhagic stroke, differ in pathophysiology but share systemic consequences, notably involving the cardiovascular system.

Cardiac disturbances following acute stroke are increasingly recognised as part of the stroke-heart syndrome, a condition encompassing a spectrum of cardiac complications triggered by cerebrovascular events.<sup>[2-4]</sup> Experimental and clinical evidence indicates that stroke may impair cardiac function even in the absence of traditional risk factors such as hypertension, diabetes, or coronary artery disease.<sup>[3,5,6]</sup> These disturbances often occur within the first few days of onset, underscoring the importance of early cardiac monitoring in stroke units.<sup>[7]</sup>

Electrocardiographic (ECG) abnormalities are among the most frequently encountered cardiac

manifestations after stroke. The mechanisms are multifactorial and are thought to involve autonomic dysregulation, catecholamine surge, and neurogenic myocardial injury resulting from insult to cortical and subcortical centres, particularly the insular cortex, hypothalamus, and brainstem nuclei that regulate cardiac autonomic tone.<sup>[3,4]</sup> Classical ECG changes include T-wave inversion, QTc prolongation, U waves, and rhythm disturbances such as sinus tachycardia and atrial fibrillation. Historical observations by Byer et al. and Burch et al. described these distinctive “cerebrogenic ECG patterns,” suggesting a strong neurocardiac connection. Subsequent systematic reviews have reaffirmed that acute stroke can precipitate dynamic ECG alterations through central-autonomic pathways.<sup>[8-10]</sup> The clinical implications of these findings are substantial. ECG changes in acute stroke have been linked to adverse short-term outcomes, increased cardiac morbidity, and even mortality.<sup>[9,10]</sup> Yet, the frequency, pattern, and prognostic significance of these abnormalities may vary across populations and stroke subtypes. In India, where stroke incidence continues to rise, comprehensive data on such neurocardiac interactions remain scarce. Hence, the present study was undertaken to describe and compare electrocardiographic abnormalities across different types of stroke, both ischaemic and haemorrhagic. Through systematic observation and analysis, this cross-sectional study aims to elucidate the spectrum of ECG changes in acute stroke, thereby enhancing understanding of brain–heart interactions and emphasising the role of routine ECG evaluation in comprehensive stroke management.

## MATERIALS AND METHODS

### Study Design and Setting

This cross-sectional observational study was conducted jointly by the Departments of Physiology and Neurology at a tertiary care teaching hospital in South India. The study period extended from August 2024 to January 2025. Ethical approval was obtained from the Institutional Ethics Committee (Protocol No.EC/NEW/INST/2023/TE/0350), and the study adhered to the principles of the Declaration of Helsinki (2013 revision).

### Study Participants

A total of 78 patients admitted with a diagnosis of acute stroke were enrolled after obtaining informed consent from patients or their legal guardians. The diagnosis and classification of stroke were confirmed by a consultant neurologist using clinical evaluation and neuroimaging (CT/MRI). Patients were categorised into ischaemic stroke, haemorrhagic stroke, and transient ischaemic attack (TIA) based on neuroimaging findings and in accordance with the American Heart Association/American Stroke Association (AHA/ASA) guidelines for cerebrovascular disease.

### Inclusion Criteria

Adults aged  $\geq 18$  years with radiologically confirmed acute stroke (ischaemic, haemorrhagic, or TIA). Patients with complete clinical and electrocardiographic records.

### Exclusion Criteria

History of pre-existing cardiac disease, electrolyte disturbances, or arrhythmias.

Patients on antiarrhythmic drugs or medications known to affect ECG parameters.

Cases with structural heart disease, prior myocardial infarction, or conduction abnormalities documented before the stroke event.

### Electrocardiographic Recording and Analysis

All patients underwent a standard 12-lead electrocardiogram (ECG) recording within 48 hours of hospital admission. ECGs were recorded using a digital ECG machine (Manufacturer: PAGE writer; Model: TC20) with standard calibration at 25 mm/s and 10 mm/mV. Recordings were interpreted independently by two qualified observers blinded to stroke subtype.

The following ECG parameters were analysed: sinus tachycardia, left ventricular hypertrophy (LVH), prolonged QT interval, abnormal R-wave progression, ST-segment elevation, left atrial enlargement (LAE), heart block, and T-wave abnormalities. QT intervals were corrected for heart rate using Bazett’s formula. Discrepancies in interpretation were resolved by consensus.

### Data Collection and Management

Clinical and demographic data were obtained from medical records. Each patient’s stroke type and ECG parameters were entered into a coded database using Microsoft Excel (Microsoft Corp., USA) to ensure confidentiality. No identifiable patient information was included in the analysis.

### Statistical Analysis

Data were tabulated and analysed using GraphPad Prism (version 9) and Microsoft Excel. Descriptive statistics were expressed as frequencies and percentages. Comparative analysis of ECG abnormalities across stroke types was performed using the Chi-square test and Fisher’s exact test as appropriate. A p-value  $< 0.05$  was considered statistically significant.

### Ethical Considerations

This study was conducted following approval from the Institutional Ethics Committee (IEC No.EC/NEW/INST/2023/TE/0350).

Informed written consent was obtained from all participants or their legally authorised representatives. All patient data were anonymised to preserve confidentiality.

### Reporting Guideline Compliance

The study was conducted and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.

## RESULTS

A total of 78 patients were included in the study, comprising 55 (70.5%) with ischaemic stroke, 18 (23.1%) with haemorrhagic stroke, and 5 (6.4%) with transient ischaemic attack (TIA) (Table 1, Figure 1). The mean age of participants was  $59.8 \pm 12.1$  years in the ischaemic group,  $58.3 \pm 16.6$  years in the haemorrhagic group, and  $64.0 \pm 10.3$  years in the TIA group. Males constituted approximately two-thirds (67%) of the overall cohort, with the highest male predominance seen among haemorrhagic stroke patients. [Table 1]  
Electrocardiographic (ECG) abnormalities were present in 82% of patients, while 18% showed normal

tracings. The most common abnormalities were left atrial enlargement (26.9%), ST-segment elevation (25.6%), left ventricular hypertrophy (24.4%), and T-wave abnormalities (21.8%). [Table 2, Figure 2]  
When stratified according to stroke subtype, LVH was observed in 12.7% of ischaemic, 55.6% of haemorrhagic, and 40% of TIA patients, showing a statistically significant association ( $\chi^2 = 10.84$ ,  $p = 0.0008$ ). Other ECG parameters such as ST-segment elevation, LAE, and T-wave changes showed no significant differences between stroke types ( $p > 0.05$ ). [Table 3, Figure 3] Sinus tachycardia and prolonged QT interval were infrequent overall. No correlation was found between patient age or sex and the occurrence of ECG abnormalities.

**Table 1: Baseline Characteristics of the Study Population (n = 78)**

Variable	Ischemic Stroke (n = 55)	Hemorrhagic Stroke (n = 18)	Transient Ischemic Attack (n = 5)
Mean age (years)	59.8 $\pm$ 12.1	58.3 $\pm$ 16.6	64.0 $\pm$ 10.3
Age range (years)	26–83	27–91	55–80
Male : Female ratio	33 : 22 (60% : 40%)	16 : 2 (88.9% : 11.1%)	1 : 4 (20% : 80%)

Table 1. Baseline characteristics of the study population Describes demographic and clinical characteristics of the 78 patients included in the

study, including age distribution, sex, and stroke subtype classification (ischaemic, haemorrhagic, and transient ischaemic attack.

**Table 2: Frequency of Electrocardiographic (ECG) Abnormalities in All Stroke Patients (n = 78)**

ECG Finding	Frequency (n)	Percentage (%)
Left atrial enlargement (LAE)	21	26.9
ST-segment elevation	20	25.6
Left ventricular hypertrophy (LVH)	19	24.4
T-wave abnormality	17	21.8
Abnormal R-wave progression	13	16.7
Sinus tachycardia	8	10.3
Heart block	5	6.4
Prolonged QT interval	5	6.4

Table 2. Frequency of electrocardiographic abnormalities among stroke patients Shows the distribution of ECG abnormalities observed in the study population, including left atrial enlargement,

ST-segment elevation, left ventricular hypertrophy, T-wave abnormalities, QT prolongation, and rhythm disturbances.

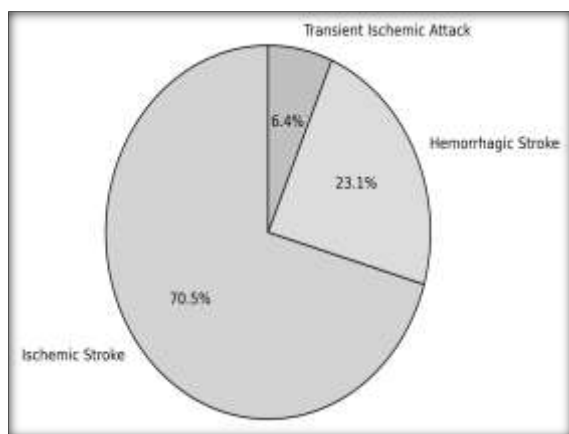
**Table 3: Comparison of ECG Abnormalities Across Stroke Subtypes**

ECG Finding	Ischemic Stroke (%)	Hemorrhagic Stroke (%)	Transient Ischemic Attack (%)	$\chi^2$ / Fisher's p-value
ST elevation	27.3	27.8	20.0	0.3977
Left atrial enlargement	25.5	33.3	20.0	0.7565
Abnormal R-wave progression	27.3	16.7	20.0	0.3433
T-wave abnormality	21.8	22.2	20.0	0.9943
Left ventricular hypertrophy	12.7	55.6	40.0	0.0008*
Sinus tachycardia	9.1	5.6	40.0	0.0700
Heart block	5.5	0.0	0.0	0.8055
Prolonged QT interval	3.6	16.7	0.0	0.1222

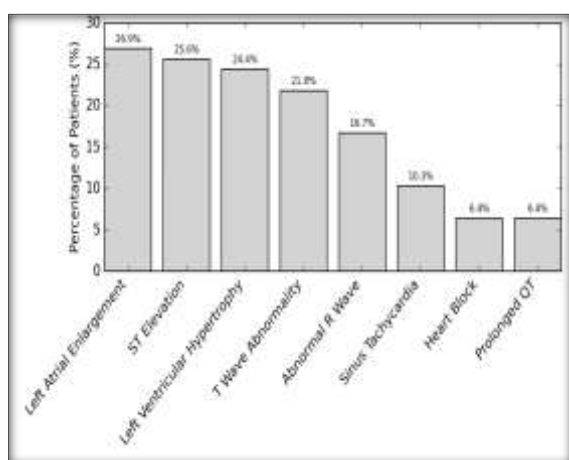
\*Significant at  $p < 0.05$

Table 3. Comparison of ECG abnormalities across stroke subtypes Presents the association between specific ECG findings and stroke subtype (ischaemic,

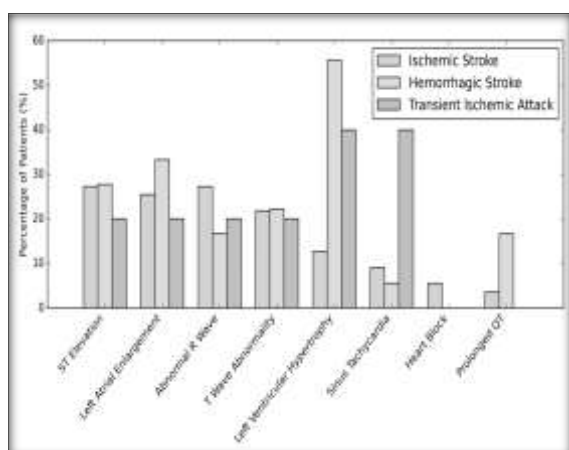
haemorrhagic, and TIA), analysed using Chi-square or Fisher's exact test. Statistically significant values ( $p < 0.05$ ) are highlighted.



**Figure 1: Distribution of Stroke subtypes** Pie chart showing the proportion of patients with ischaemic stroke (70.5%), haemorrhagic stroke (23.1%), and transient ischaemic attack (6.4%)



**Figure 2: Frequency of electrocardiographic abnormalities among stroke patients** Bar graph depicting the frequency of ECG abnormalities, including left atrial enlargement (26.9%), ST-segment elevation (25.6%), left ventricular hypertrophy (24.4%), T-wave changes, prolonged QT interval, and rhythm disturbances



**Figure 3: Association between left ventricular hypertrophy and stroke subtype** Clustered bar chart illustrating the distribution of LVH across ischaemic, haemorrhagic, and TIA patients. LVH was significantly more common in haemorrhagic stroke and TIA ( $p = 0.0008$ )

## DISCUSSION

This cross-sectional study demonstrates that ECG abnormalities are highly prevalent in patients with acute stroke, irrespective of subtype, highlighting the close interplay between cerebral and cardiac physiology. The overall prevalence of ECG changes in this study (82%) is comparable with earlier reports ranging from 60% to 90%, supporting the hypothesis that acute cerebrovascular injury can elicit secondary cardiac electrophysiological disturbances through autonomic and neurohumoral mechanisms.<sup>[3,4,8]</sup>

### Spectrum of ECG Abnormalities

The most common findings in our cohort were left atrial enlargement, ST-segment elevation, LVH, and T-wave inversion, consistent with previous studies.<sup>[5-7,9]</sup> The predominance of repolarisation changes such as T-wave inversion and QT prolongation in stroke patients has been attributed to excessive sympathetic discharge following insular and hypothalamic injury. Experimental studies by Chen et al. demonstrated catecholamine-mediated myocardial injury following cerebral infarction, further linking the brain-heart axis to these ECG phenomena.<sup>[3]</sup>

In our study, LVH showed a statistically significant association with stroke subtype, being more frequent among haemorrhagic and TIA groups. This may reflect pre-existing hypertensive cardiac remodelling, which predisposes individuals to both haemorrhagic cerebrovascular events and ECG voltage changes. Similar patterns of cardiac involvement have been reported by Hjalmarsson et al., who observed that QT prolongation and elevated cardiac troponin levels predicted poorer outcomes following stroke.<sup>[9]</sup>

### Neurogenic Myocardial Injury

Autonomic imbalance resulting from damage to the insular cortex is considered central to neurogenic cardiac dysfunction. Studies have identified the right insula as a sympathetic control centre and the left insula as parasympathetic; lesions affecting either can produce arrhythmias or repolarisation abnormalities. Our findings of frequent ST and T-wave changes, along with QT prolongation, support this autonomic hypothesis.

### Clinical Implications

Recognition of ECG abnormalities in stroke patients has crucial implications. These changes may precede overt cardiac events and can aid in risk stratification and early intervention. Routine ECG screening in all stroke admissions can help identify patients at risk of arrhythmias, myocardial injury, or autonomic dysfunction, thereby improving prognosis and reducing in-hospital morbidity.

### Limitations

This study was hospital-based and limited by modest sample size. Cardiac biomarkers such as troponin or echocardiographic parameters were not assessed. A longitudinal follow-up could provide better insight into the prognostic value of ECG changes. Nevertheless, the inclusion of multiple stroke

subtypes and use of standardised ECG interpretation strengthen the findings.

## CONCLUSION

Electrocardiographic abnormalities are common across all forms of stroke and reflect the neurocardiac interactions triggered by cerebral injury. Left atrial enlargement, ST-segment elevation, and LVH were the predominant findings, with LVH showing a significant association with stroke subtype. Routine ECG evaluation in stroke patients is recommended to facilitate early detection of cardiac involvement and guide integrated management.

**Acknowledgements:** The authors express sincere gratitude to the Departments of Physiology and Neurology, of a tertiary care teaching hospital in South India, for their guidance, technical support, and encouragement throughout this study.

All authors read and approved the final manuscript.

**Conflict of Interest:** The authors declare no conflict of interest related to this work.

**Ethical Approval:** The study protocol was reviewed and approved by the Institutional Ethics Committee of a tertiary care teaching hospital in India (Protocol No. EC/NEW/INST/2023/TE/0350). All procedures performed were in accordance with the ethical standards of the institutional research committee and the 2013 revision of the Declaration of Helsinki. Written informed consent was obtained from all participants or their legally authorised representatives prior to inclusion in the study.

**Funding and License Declaration:** This research received no external funding from any governmental, commercial, or non-profit agency.

This is an open-access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International License (CC BY-NC-SA 4.0), which permits non-commercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited, and derivative works are licensed under identical terms.

## REFERENCES

1. Pandian JD, Gall SL, Kate MP, Silva GS, Akinyemi RO, Ovbiagele BI, et al. Prevention of stroke: a global perspective. *Lancet*. 2018;392(10154):1269–78. [https://doi.org/10.1016/S0140-6736\(18\)31269-8](https://doi.org/10.1016/S0140-6736(18)31269-8) PMID: 30319114
2. Committee of the Chinese Society for Integrated Chinese and Western Medicine, First Aid Medicine Branch. Expert consensus on Chinese and Western medicine emergency treatment for acute ischemic stroke in China. *Chinese Critical Care Medicine*. 2018;30(3):193–200. <https://doi.org/10.3760/cma.j.issn.2095-4352.2018.03.002>
3. Chen Z, Venkat P, Seyfried D, Chopp M, Yan T, Chen J. Brain–Heart Interaction: Cardiac Complications After Stroke. *Circulation Research*. 2017;121(4):451–68. <https://doi.org/10.1161/CIRCRESAHA.117.311170> PMID: 28775014
4. Vogt JF, Nolte CH, Doehner W, Hachinski V, Endres M. Stroke–Heart Syndrome: Clinical Presentation and Underlying Mechanisms. *Lancet Neurology*. 2018;17(12):1109–20. [https://doi.org/10.1016/S1474-4422\(18\)30336-3](https://doi.org/10.1016/S1474-4422(18)30336-3) PMID: 30509695
5. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: The Framingham Study. *Stroke*. 1991;22(8):983–8. <https://doi.org/10.1161/01.STR.22.8.983> PMID: 1866765
6. Kolin A, Norris JW. Myocardial damage from acute cerebral lesions. *Stroke*. 1984;15(6):990–3. <https://doi.org/10.1161/01.STR.15.6.990> PMID: 6506127
7. Prosser J, MacGregor L, Lees KR, Diener HC, Hacke W, Davis SM. Predictors of early cardiac morbidity and mortality after ischemic stroke. *Stroke*. 2007;38(8):2295–302. <https://doi.org/10.1161/STROKEAHA.106.471813> PMID: 17569877
8. Khechinashvili G, Asplund K. Electrocardiographic changes in patients with acute stroke: a systematic review. *Cerebrovascular Diseases*. 2002;14(2):67–76. <https://doi.org/10.1159/000064733> PMID: 12187009
9. Hjalmarsson C, Bokemark L, Fredriksson S, Antonsson J, Shadman A, Andersson B. Can prolonged QTc and cTnT level predict prognosis of stroke? *International Journal of Cardiology*. 2012;155(3):414–7. <https://doi.org/10.1016/j.ijcard.2010.10.042> PMID: 21093074
10. Fure B, Bruun Wyller T, Thommessen B. Electrocardiographic and troponin T changes in acute ischaemic stroke. *Journal of Internal Medicine*. 2006;259(6):592–7. <https://doi.org/10.1111/j.1365-2796.2006.01639.x> PMID: 16704560