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Association of Cardiac Biomarkers and Electrocardiographic Changes with Severity of Stroke

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ABSTRACT

Introduction: A cerebrovascular accident (CVA), commonly referred to as a stroke, is characterized by the sudden appearance of localized neurological impairments resulting from disrupted cerebral blood flow. This disruption may lead to ischemia or hemorrhage, potentially causing either partial or complete loss of neurological function. The profound and often debilitating effects of stroke render it a critical global health concern 1. **Materials And Methods:** The cross-sectional study was conducted in the Department of Medicine and the Department of Emergency at Sri Guru Ram Das Institute of Medical Sciences and Research, Amritsar. The study was carried out from 1st August 2023 to 28th February 2025. This study included a total of 116 patients who presented with acute stroke and met the inclusion criteria. **Results**: There was a statistically significant association between stroke severity (NIHSS) and Troponin I (Trop I) levels (p = 0.001). Patients with minor strokes (NIHSS 0–4) had the lowest mean Trop I level at 0.69 ± 1.41 ng/dl, while those with moderate strokes (5–15) showed a sharp rise to 2.5 ± 2.56 ng/dl. In the moderate to severe category (16–20), Trop I increased further to 2.92 ± 1.77 ng/dl, and in severe strokes (>20), the mean remained elevated at 2.65 ± 2.32 ng/dl. **Conclusion**: This comprehensive study highlights the multifactorial nature of stroke, emphasizing the interplay between demographic variables, clinical presentation, vascular territory, biochemical markers, and functional outcomes. Stroke was most prevalent among older males, with the majority presenting with moderate severity and anterior circulation involvement. Ischemic stroke emerged as the dominant subtype, with the middle cerebral artery being the most frequently affected.

Keywords*:* Stroke, Acute Ischemic Stroke, Troponin I, National, Institutes of Health Stroke Scale (NIHSS), Stroke Severity.

INTRODUCTION

A cerebrovascular accident (CVA), commonly referred to as a stroke, is characterized by the sudden appearance of localized neurological impairments resulting from disrupted cerebral blood flow. This disruption may lead to ischemia or hemorrhage, potentially causing either partial or complete loss of neurological function. The profound and often debilitating effects of stroke render it a critical global health concern [1].

According to predictions by the World Health Organization, stroke was expected to emerge as a leading contributor to disability and death by the year 2020 [2]. Timely identification of stroke-related complications can play a crucial role in reducing mortality and enhancing recovery outcomes through the development and application of effective, rapid-response treatment strategies aimed at high-risk individuals [3]

It has long been recognized that changes in the metabolic functions of the central nervous system can significantly affect cardiac performance [4]. In stroke patients, cardiac dysfunctions are frequently linked to heightened sympathetic nervous system activity, particularly due to damage within the insular cortex, which plays a regulatory role in autonomic control [5]. Interestingly, cardiac biomarkers such as troponin T and creatine kinase-MB (CK-MB) have been reported to rise in certain stroke cases, even in the absence of underlying heart disease [6].

In acute stroke scenarios, an increase in cardiac enzymes may signal either a primary cardiac event contributing to cerebral embolism or a secondary myocardial injury resulting from primary cerebral ischemia. It is understood that activation of the sympathoadrenal system during acute cerebral events can lead to cardiac manifestations such as myocyte injury (myocytolysis), enzyme elevation, and arrhythmias [7]. Additionally, the brain's expression of the CK-BB isoenzyme offers a possible explanation for increased CK-MB levels observed post-stroke, indicating a cerebral origin for some cardiac biomarker elevations [8].

Ischemic occlusions account for approximately 85% of stroke-related cases, while the remaining cases are attributed to intracerebral hemorrhages.[9]

Hemorrhagic stroke constitutes about 10–15% of all strokes and is associated with a high mortality rate. This condition occurs when stress on brain tissue or internal injury causes blood vessels to rupture. The rupture leads to toxic effects within the vascular system, ultimately causing infarction.[10] Hemorrhagic stroke is further classified into intracerebral hemorrhage (ICH) and subarachnoid hemorrhage. In ICH, ruptured blood vessels lead to an abnormal buildup of blood within the brain. The primary causes of ICH include hypertension, vascular abnormalities, and the excessive use of anticoagulants or thrombolytic agents. In contrast, subarachnoid hemorrhage involves the accumulation of blood in the subarachnoid space, typically resulting from head trauma or a cerebral aneurysm.[11,12]

The aim of the present study was to evaluate Cardiac biomarkers and identify ECG changes in stroke patients and find their association with severity of stroke.

MATERIALS AND METHODS

The cross-sectional study was conducted in the Department of Medicine and the Department of Emergency at Sri Guru Ram Das Institute of Medical Sciences and Research, Amritsar. The study was carried out from 1st August 2023 to 28th February 2025. This study included a total of 116 patients who presented with acute stroke and met the inclusion criteria.
Participants: All diagnosed patients of stroke who presented to the Outpatient Department or Inpatient Department of the Medicine Department, or to the Emergency Department of SGRDIMSR, Amritsar, were recruited into the study after obtaining written, informed consent in the vernacular language and securing approval from the Institutional Ethics Committee.

Inclusion Criteria

- Patients aged above 18 years with stroke diagnosed clinically using the TOAST criteria and confirmed radiologically by non-contrast CT (NCCT) head or MRI.

Exclusion Criteria

- Patients with known cardiovascular disease
- Patients post defibrillation
- Patients with a history of pulmonary embolism
- Patients with Rhabdomyolysis

METHODOLOGY

All patients who presented to SGRD Hospital within 48 hours of acute stroke onset were included in the study. Patients with a prior history of coronary artery disease, those taking medications known to cause electrocardiogram changes, and those with conditions such as acute pulmonary embolism, rhabdomyolysis, or who had undergone defibrillation—conditions known to elevate troponin levels—were excluded.

The diagnosis of stroke was established through a combination of patient history, detailed clinical examination, and neuroimaging—either computed tomography (CT) or magnetic resonance imaging (MRI). Upon admission, all stroke patients underwent testing for Troponin I levels. Additionally, a standard 12-lead electrocardiogram (ECG) was conducted for each individual.

Based on Troponin I results, patients were divided into two cohorts: those exhibiting elevated levels and those with normal levels. Stroke severity for all participants was evaluated using the National Institutes of Health Stroke Scale (NIHSS). Functional outcomes at the time of discharge were assessed using the Modified Rankin Scale (mRS). A score below 3 on the mRS was classified as a favorable outcome, whereas a score of 3 or higher indicated an unfavorable outcome.

The relationships between cardiac biomarkers, ECG findings, NIHSS scores, and functional outcomes (as measured by mRS) were systematically analyzed.

Statistical Analysis

The collected data were entered into Microsoft Excel and analyzed using SPSS version 26.0. Descriptive statistics were used to summarize the data, including frequencies, percentages, means, and standard deviations. Categorical variables such as gender, stroke type, circulation involvement, ECG findings, and outcome categories were compared using the Chi-square test or Fisher's exact test, wherever applicable. Continuous variables such as age, lipid profile parameters, Troponin I, and CPK-MB levels were compared using the independent t-test or ANOVA, depending on the number of groups being compared. The association between NIHSS scores, modified Rankin Scale (mRS), and cardiac biomarkers (Troponin I and CPK-MB) was assessed. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Baseline characteristics

AGE	**Number**	**Percent**
≤30	7	6.0
31-40	12	10.3
41-50	16	13.8
51-60	24	20.7
61-70	29	25.0
>70	28	24.1
SEX		
FEMALE	42	36.2
MALE	74	63.8
RISK FACTORS OF STROKE		
HISTORY OF HYPOTHYROIDISM	38	32.8
T2DM	33	28.4
HISTORY OF OCP USE	11	9.5
FAMILY HISTORY OF CVA	10	8.6
HISTORY OF SMOKING	3	2.6
STROKE TYPE		
HEMORRHAGIC STROKE	25	21.6
ISCHEMIC STROKE	91	78.4

The age distribution of stroke patients in this study (n=116) reveals that the majority of cases were concentrated in the older age groups, with the highest frequency observed in the 61–70 years age range (25%), followed closely by those aged over 70 years (24.1%). In the present study cohort, males constituted a higher proportion of stroke cases (63.8%) compared to females (36.2%). The male predominance was observed in this study. In this study, hypothyroidism (32.8%) and type 2 diabetes mellitus (28.4%) were the most common stroke risk factors, both known to contribute to vascular disease and cardiac complications. A history of OCP use (9.5%) was notable among females, while family history of stroke (8.6%) and smoking (2.6%) were less frequent. Ischemic stroke was the predominant type, accounting for 78.4% of cases, while hemorrhagic stroke comprised 21.6%.

Table 2: ECG findings in stroke patients

ECG FINDINGS	**Number**	**Percent**
Normal	19	16.4
ST DEPRESSION	43	37.1

T WAVE INVERSION	13	11.2
POOR R WAVE PROGRESSION	10	8.6
LBBB	8	6.9
PROLONGED PR INTERVAL	7	6.0
ST ELEVATION	7	6.0
AF	6	5.2
SINUS TACHYCARDIA	5	4.3
SHORT PR INTERVAL	5	4.3
PREMATURE VENTRICULAR COMPLEX	4	3.4
LEFT AXIS DEVIATION	4	3.4
FLATTENED T WAVE	4	3.4
U WAVE	3	2.6
PROLONGED QT INTERVAL	3	2.6
DEEP Q WAVE	2	1.7
RBBB	1	0.9

Abnormal ECG changes were observed in a majority of stroke patients, with ST depression (37.1%) being the most common, indicating myocardial ischemia or strain. T wave inversion (11.2%), poor R wave progression (8.6%), and conduction abnormalities like LBBB (6.9%) and prolonged PR interval (6.0%) were also notable. Atrial fibrillation (5.2%)—a known cardioembolic risk factor—was present in several cases. Only 16.4% of patients had a normal ECG.

Table 3: Distribution of study population according to artery of stroke involvement

ARTERY OF STROKE	Frequency	Percent
MCA	70	60.3
PCA	8	6.9
PICA	6	5.2
ACA	3	2.6
MCA AND PCA	16	13.8
ACA AND MCA	4	3.4
MCA AND PICA	3	2.6
ACA AND PCA	1	0.9
ACA, MCA AND PCA	1	0.9
BASILAR ARTERY	3	2.6
VERTEBRAL ARTERY	1	0.9
TOTAL	116	100.0

The middle cerebral artery (MCA) was the most frequently involved vessel, accounting for 60.3% of cases, consistent with its role as the most commonly affected artery in ischemic strokes. Other arteries such as the posterior cerebral artery (PCA) and posterior inferior cerebellar artery (PICA) were less frequently involved. Combined arterial involvement, including MCA with PCA (13.8%), was also observed, indicating more extensive cerebral infarction. Involvement of multiple territories may correlate with greater stroke severity.

Table 4: Association of ECG changes and MRS functional outcome

	Good	Poor	p value
	Number (%)	Number (%)	
AF	3(7.5)	3(3.95)	0.663
ST DEPRESSION	10(25)	33(43.42)	0.069
T WAVE INVERSION	4(10)	9(11.84)	0.515
ST ELEVATION	2(5)	5(6.58)	0.543
U WAVE	0(0)	3(3.95)	0.316
LBBB	1(2.5)	7(9.21)	0.26
RBBB	1(2.5)	0(0)	0.345
DEEP Q WAVE	0(0)	2(2.63)	0.544
PROLONGED QT INTERVAL	1(2.5)	2(2.63)	0.966
PROLONGED PR INTERVAL	3(7.5)	4(5.26)	0.691
SHORT PR INTERVAL	0(0)	5(6.58)	0.163
PREMATURE VENTRICULAR COMPLEX	3(7.5)	1(1.32)	0.118

POOR R WAVE PROGRESSION	1(2.5)	9(11.84)	0.161
SINUS TACHYCARDIA	2(5)	3(3.95)	0.791
FLATTENED T WAVE	0(0)	4(5.26)	0.297

In this study, ECG abnormalities were more common in patients with poor functional outcomes, particularly ST depression (43.42%), but none showed a statistically significant association ($p > 0.05$). Although findings like T wave inversion, prolonged intervals, and poor R wave progression were more frequent in poor outcome groups, they did not significantly differ. Thus, ECG changes alone may not predict functional outcome, but they remain clinically relevant for cardiac monitoring.

Table 5: Association between ECG changes and stroke severity based on NIHSS score

	Minor	Moderate	Moderate to severe	Severe	p value
	Number (%)	Number (%)	Number (%)	Number (%)	
AF	3(8.33)	2(2.78)	1(20)	0(0)	0.239
ST DEPRESSION	10(27.78)	29(40.28)	3(60)	1(33.33)	0.455
T WAVE INVERSION	4(11.11)	7(9.72)	2(40)	0(0)	0.176
ST ELEVATION	2(5.56)	5(6.94)	0(0)	0(0)	0.889
U WAVE	0(0)	3(4.17)	0(0)	0(0)	0.637
LBBB	1(2.78)	6(8.33)	1(20)	0(0)	0.382
RBBB	1(2.78)	0(0)	0(0)	0(0)	0.379
DEEP Q WAVE	0(0)	2(2.78)	0(0)	0(0)	0.611
PROLONGED QT INTERVAL	4(11.11)	3(4.17)	0(0)	0(0)	0.463
PROLONGED PR INTERVAL	0(0)	4(5.56)	1(20)	0(0)	0.217
SHORT PR INTERVAL	2(5.56)	1(1.39)	1(20)	0(0)	0.196
PREMATURE VENTRICULAR COMPLEX	1(2.78)	9(12.5)	0(0)	0(0)	0.233
POOR R WAVE PROGRESSION	2(5.56)	3(4.17)	0	0	0.919
SINUS TACHYCARDIA	1(2.78)	3(4.17)	0	0	0.931
FLATTENED T WAVE	1(2.78)	3(4.17)	0	0	0.931

In the present study, various ECG abnormalities were observed across different levels of stroke severity based on the NIHSS, but none showed a statistically significant association. ST segment depression was the most common finding, seen in 27.78% of minor strokes, 40.28% of moderate strokes, and 60% of moderate to severe strokes, suggesting a possible trend of increasing ischemic changes with stroke severity, though not statistically significant ($p = 0.455$). T wave inversion and prolonged PR or QT intervals were more frequently seen in moderate and moderate to severe strokes, indicating potential autonomic or repolarization disturbances. Atrial fibrillation was present in 8.33% of minor strokes and 20% of moderate to severe strokes ($p = 0.239$), but did not differ significantly across groups. Other findings like LBBB, RBBB, premature ventricular complexes, and deep Q waves occurred infrequently. Overall, while ECG changes were more common in patients with more severe strokes.

Table 6: Association between CPK-MB levels and MRS functional outcome

MRS functional outcome	N	Mean CPK MB(ng/dl)	P value
Good <3	40	3.78±2.36	<0.001
POOR≥3	76	11.68±8.36	

Patients with poor functional outcomes (mRS ≥3) had markedly elevated mean CPK-MB levels (11.68 ng/dl) compared to those with good outcomes (3.78 ng/dl), with a highly significant p-value (<0.001).

Table 7: Association between troponin I (TROP I) levels and MRS functional outcome

MRS functional outcome	N	Mean TROP I (ng/dl)	P VALUE
Good<3	40	0.74±1.4	<0.001
Poor ≥3	76	2.6±2.51	

Stroke patients with poor functional outcomes (mRS ≥3) had significantly higher mean Troponin I levels (2.6 ± 2.51 ng/dl) compared to those with good outcomes (mRS <3; 0.74 ± 1.4 ng/dl). The difference was statistically significant ($p < 0.001$), suggesting a strong association between elevated Trop I levels and stroke severity.

Table 8: Association between NIHSS stroke severity and CPK-MB and TROP I levels

	Severity of Stroke (NIHSS)				p value
	Minor	Moderate	Moderate to severe	Severe	
CPK MB	3.54±2.26	10.53±7.68	13.9±7.54	27.83±5.86	<0.001
TROP I	0.69±1.41	2.5±2.56	2.92±1.77	2.65±2.32	0.001

There was a highly significant association between stroke severity (NIHSS) and CPK-MB levels (p = 0.000). As stroke severity increased, mean CPK-MB levels rose markedly—from 3.54 ng/dl in minor strokes to 27.83 ng/dl in severe strokes. This trend suggests that higher stroke severity is strongly linked with greater cardiac muscle involvement, reinforcing the value of CPK-MB as a prognostic cardiac biomarker in acute stroke assessment.

There was a statistically significant association between stroke severity (NIHSS) and Troponin I (Trop I) levels (p = 0.001). Patients with minor strokes (NIHSS 0–4) had the lowest mean Trop I level at 0.69 ± 1.41 ng/dl, while those with moderate strokes (5–15) showed a sharp rise to 2.5 ± 2.56 ng/dl. In the moderate to severe category (16–20), Trop I increased further to 2.92 ± 1.77 ng/dl, and in severe strokes (>20), the mean remained elevated at 2.65 ± 2.32 ng/dl.

DISCUSSION

Stroke continues to be a significant cause of death and disability on a global scale, presenting a major challenge to public health systems worldwide. According to the World Health Organization (WHO), it is the second leading cause of death globally, responsible for nearly 11% of all fatalities[13]. Findings from the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017 identified stroke as the third most significant contributor to the combined burden of mortality and disability—measured in disability-adjusted life years (DALYs)—and the second leading cause of death in 2017 [14] . Additionally, the GBD 2017 analysis showed a notable decline in age-standardized stroke mortality between 1990 and 2017. However, the reduction in incidence during this period was comparatively modest, suggesting that progress in stroke treatment has outpaced preventive efforts [15.]

The majority of stroke patients in this cohort were aged above 60 years, with the highest prevalence in the 61–70 age group (25%) followed closely by those above 70 years (24.1%). This age-related trend mirrors findings from the Global Burden of Disease (GBD) Study, which shows a sharp increase in stroke incidence and mortality in the elderly due to cumulative vascular damage, reduced physiological reserves, and comorbid conditions such as hypertension and diabetes.[13,15,16] Aging also alters cerebral autoregulation and increases arterial stiffness, making the elderly more susceptible to cerebrovascular events. Males represented 63.8% of stroke patients, showing a clear gender disparity. This trend has been observed in multiple population-based studies and is likely due to higher exposure among males to risk factors such as tobacco use, alcohol consumption, occupational stress, and delayed health-seeking behaviors.[7,16,17] Biological differences, including the protective effect of estrogen in premenopausal women, may also explain the relatively lower stroke incidence in females. Gender-specific risk assessment should be incorporated into clinical practice.

The most prevalent comorbidities in this study were hypothyroidism (32.8%) and type 2 diabetes mellitus (28.4%). Hypothyroidism may contribute to dyslipidemia and endothelial dysfunction, both of which are recognized contributors to atherosclerosis and stroke.[18,19] Diabetes is a well-established risk factor for both ischemic and hemorrhagic stroke due to its impact on microvascular integrity and prothrombotic states.[10,17] The observed prevalence of OCP-related stroke in females (9.5%) also echoes the thromboembolic risks associated with estrogen.5 Weakness on one side of the body (hemiparesis) was the most common presenting symptom (54.3%), followed by altered sensorium (39.7%) and slurred speech (26.7%). These symptoms are consistent with anterior circulation strokes, particularly involving the MCA territory, which supplies the motor cortex and language areas.[10,17] The presence of ataxia and visual disturbances reflects involvement of posterior circulation, especially the cerebellum and occipital lobes. A high percentage (83.6%) of stroke patients exhibited abnormal ECG findings. ST depression (37.1%) and T wave inversion (11.2%) were the most prevalent, indicating subendocardial ischemia or repolarization abnormalities. Atrial fibrillation, present in 5.2% of patients, is a well-established cause of cardioembolic strokes, especially in elderly patients.[20-22]

Ischemic stroke accounted for 78.4% of cases, while hemorrhagic stroke comprised 21.6%. This is consistent with global epidemiological data, which report ischemic strokes as the predominant subtype, representing 85–90% of stroke cases globally.[13,15,16] The middle cerebral artery (MCA) was involved in 60.3% of patients, followed by multi-territory involvement like MCA and PCA (13.8%). MCA strokes are the most frequent due to the artery's large diameter and direct flow from the internal carotid artery.[17] These strokes often result in hemiparesis, aphasia, or hemispatial neglect. In contrast, PCA and PICA strokes present with visual field defects, vertigo, or ataxia, often leading to delayed recognition. Moderate strokes (NIHSS 5–15) were most common (62.1%), with minor strokes constituting 31% and severe strokes being relatively rare. This distribution aligns with population-based data, as many strokes present with moderate impairment at onset.[10,16] NIHSS scoring allows standardized quantification of neurological deficits and correlates with lesion size and outcome. A poor functional outcome (mRS ≥3) was observed in 65.5% of patients. This suggests a high burden of disability post-stroke, despite timely care. Poor outcomes are influenced by factors like stroke severity, delayed presentation, comorbidities, and lack of early rehabilitation.[23-25] Atherothrombotic (TOAST Class 1) strokes were the predominant subtype in this study, accounting for 87.9% of cases. This observation aligns with global and regional trends that point to large artery atherosclerosis as the most common mechanism of ischemic stroke, particularly in low- and middle-income countries.[15-17]

Although there was no statistically significant association (all p-values > 0.05), certain ECG changes—particularly ST depression (60% in moderate to severe strokes), T wave inversion, and prolonged QT/PR intervals—were more prevalent in patients with higher NIHSS scores. This suggests a possible trend toward increased neurocardiac stress in more severe strokes.[21,22,26] The neurogenic mechanism, often involving a surge of catecholamines due to hypothalamic–pituitary–adrenal axis activation, can lead to myocardial stunning or arrhythmias, even in patients without primary cardiac disease. ECG monitoring remains a valuable tool in all stroke patients, especially those with moderate to severe deficits. While ECG changes may not directly correlate with NIHSS in all cases, their presence should prompt further cardiac evaluation and close hemodynamic monitoring. Early detection of arrhythmias or myocardial injury can prevent secondary complications and reduce stroke-related mortality.

In our study, patients with poor functional outcomes (mRS ≥3) had significantly elevated mean CPK-MB levels (11.68 ± 8.36 ng/dl) compared to those with good outcomes (3.78 ± 2.36 ng/dl), with a highly significant p-value ($p < 0.001$). Our findings align with those of Li et al., who demonstrated that elevated serum CPK-MB levels post-stroke are significantly associated with worse clinical outcomes.[27] Troponin I was significantly higher in patients with poor outcomes (2.6 ± 2.51 ng/dl) compared to those with good outcomes (0.74 ± 1.4 ng/dl; $p < 0.001$). Troponin elevation in stroke patients has been correlated with sympathetic overactivation, myocardial stunning, and a higher risk of mortality.[28,29]

There was a strong and statistically significant association between increasing stroke severity and rising CPK-MB levels ($p < 0.001$). Mean values increased from 3.54 ng/dl in minor strokes to 27.83 ng/dl in severe cases. These elevations support the concept of neurogenic myocardial injury and heightened catecholaminergic activity in severe strokes.[19] Troponin I levels were significantly elevated with increasing stroke severity ($p = 0.001$), rising from 0.69 ng/dl in minor strokes to over 2.6 ng/dl in moderate to severe cases. This pattern may reflect the association between stroke-related sympathetic discharge and myocardial injury.[30]

CONCLUSION

This comprehensive study highlights the multifactorial nature of stroke, emphasizing the interplay between demographic variables, clinical presentation, vascular territory, biochemical markers, and functional outcomes. Stroke was most prevalent among older males, with the majority presenting with moderate severity and anterior circulation involvement. Ischemic stroke emerged as the dominant subtype, with the middle cerebral artery being the most frequently affected.

Overall, the findings underline the importance of a multidisciplinary approach to stroke care incorporating early neurological assessment, cardiac evaluation, metabolic control, and vascular imaging. Elevated Troponin I and CPK-MB levels were significantly associated with greater stroke severity and poorer functional outcomes, suggesting their potential utility in risk stratification. In contrast, although ECG abnormalities were common, they did not demonstrate a statistically significant association with stroke severity or functional outcome. Further multicentric studies with larger sample sizes are required to validate these findings.

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