

# Electrocardiographic Changes and Serum Troponin Levels in Patients with Acute Stroke, A prospective cohort study from Sudan

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## Research Article

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# Abstract

## Background:

Electrocardiographic changes and elevated serum troponin are frequent findings in acute stroke . They may reflect what is known as the neuro-genic myocardial injury . However, as stroke and cardiac disease share the same risk factors. Coexistence of the two is highly susceptible.

## Objectives:

To determine the electrocardiographic changes and serum troponin level in acute stroke patients and to correlate these changes to the anatomical location and pathological type of the stroke.

## Methods:

A Prospective cohort study conducted at the national center of neurological Science, from January to December 2019 Study was done at the neurological center in Sudan , The National Center of Neurological sciences. All cases presented with acute stroke during the study period were included. Non probability sample, with total coverage during study period. 50 patients were included in the study. Data were analyzed by using (SPSS) version 25

12 standard ECG were performed in the first hours of admission. 2 samples from each patient were obtained for serum troponin with at least 8 hours apart.

## Results:

All patients had wide variants of ECG changes. But tachycardia was the most frequent one identified in 54% of patients(27/50). Half of them were found to have an anterior circulation stroke. 14% of patients (7/50) have positive troponin, ECG changes identified in all of patients who represent positive troponin 100% (7 patients).

Moreover, anterior circulation stroke was recognized in all patients with positive troponin I marker.

## Conclusion:

This study suggest that ECG abnormalities in patients with acute stroke are very common, especially tachycardia. The site of lesion appear to play major factor as a cause of genesis of arrhythmia. Concomitant cardiac diseases may present. Serum troponin elevation may play a role in diagnosing neuro-cardiogenic injury but, ECG appears to be more sensitive and familial.

# Introduction

Cardiovascular abnormalities are common after stroke. The Disorders of central nervous system cause a wide array of cardiovascular system dysfunctions ranging from electrocardiogram changes and transient

myocardial dysfunction to sudden death.(1)

Cardiovascular effects of stroke are modulated by concomitant or preexistent cardiac disease and may be related to pathological type of CVA disease and its localization(2). It is essential to distinguish whether cardiovascular abnormalities are caused by stroke, unrelated to it, or in the course of cardiac arrhythmia, that leads to stroke. The distinction is often difficult because preexisting cardiac abnormalities are highly prevalent among stroke(3).

There is considerable clinical and experimental evidence that stimulation of certain areas in brain such as insular cortex can induce cardiac dysfunction in form of myocytolysis, enzyme elevation and arrhythmias (4). Furthermore, increase sympathetic activity thought to be contributory, inducing reversible cardiac myocardium damage through catecholamine & cortisol surge.(5)

Early detection and management of cardiac complications post stroke may carry prognostic value, as cardiac abnormalities can greatly increase the morbidity and mortality of patients with intracranial pathologies.(6) Stroke brings high morbidity and mortality rate; it is a major health problem world widely. An estimated 17.5 million people died from CVDs in 2012 representing 31% of all global deaths. An estimated 6.7 million were due to stroke (7). In Europe it is the second mortality cause accounting for 10% of all death cause in men and 15% in women (8). Several studies have demonstrated that cardiac dysfunction may occur after vascular brain injuries without any evidence of primary heart disease. Autopsy showed sub endothelial hemorrhage and ischemic changes that are not related to coronary arteries distribution. In addition, Autonomic dysfunction is common after acute stroke. This is evident by impairment of physiological regulation of heart rate and blood pressure, namely decrease heart rate variability(HRV), and impaired baroreceptor(9). The variability in heart rate is indicative of the heart's ability to adjust to circulatory changes. Several studies have reported decreased HRV in patients with stroke, not only in the acute phase but also at 1 to 6 months after stroke.

Autonomic dysfunction causing increased surge of catecholamine and cortisol. Catecholamine excess over activates the B adrenergic receptors, over activation of these receptors leads to tonic opening of Ca channels causing impairment in sequestration of intracellular Ca<sup>2+</sup>, which is necessary for relaxation of cardiac muscles. Sequestration of intracellular Ca<sup>2+</sup> prolongs repolarization period and hence, prolonged contraction of cardiac muscle which leads to cell damage and death. (9)

Elevated cardiac markers after stroke support the autonomic imbalance theory and not necessarily indicate that myocardial injury occurs. In a study CK, CK-MB, myoglobins, were found to be elevated for more sympathetic tone following right hemispheric stimulation, 3 days after stroke and returns to normal in the 4th day.[11]

Troponin T that is more specific for myocardial injury was in reference range. In another study conducted in 730 patients with acute stroke, without known heart or renal disease and no clinical ECG or echocardiography changes suggestive of CAD, found that 7% of patients had elevated troponin level. Several studies have reported elevated troponin T level can predict

A recent study mentioned the level of cardiac troponin in patients with stroke and the pattern of acute or chronic elevation with clinical conditions can help to classify the coronary causes and neurogenic heart syndrome morbidity and mortality after acute stroke at 30 days to 6 month.

However the clinical significance of elevated cardiac bio -markers after acute stroke is inconclusive till now. (9)

Anatomically insular cortex is an interconnecting part of cerebral cortex located within the lateral sulcus of brain. contribute to arrhythmogenesis stimulation of the insular cortex resulted in cardiovascular effect with some lateralization. Stimulation of the right insular cortex more frequently produces a sympathetic effect. And stimulation of left cortical insula produced-parasympathetic(13). Small case reports of isolated insular lesions have suggested that right insular hemorrhagic or ischemic stroke resulted in bradycardia and sometimes a systole. This can be explained by decreased sympathetic tone from right insular lesion and relative parasympathetic over activity. On the contrary, isolated left insular lesion results in decreased HRV possibly because of loss parasympathetic tone and increased basal effect. sympathetic cardiac activity however, assuming that there is complete lateralization of autonomic control from insular cortex is inappropriate because the literature report conflicting results on the topic, some suggest lateralization where others merges against this theory.(10,12)

Our aim is to determine electrocardiographic changes and serum troponin level in patients with acute stroke and to correlate these changes to the anatomical location and pathological type of the stroke.

## Methodology

It is prospective descriptive analytical study was conducted at the national center for neurological science , Khartoum . From February to December 2019 . All cases presented with acute stroke during the study period were included. Non probability sample, with total coverage during study period. 50 patients were included in the study.

### Inclusion criteria:

Sudanese patients above 16 year old presenting with acute stroke within the first 72 hours. Brain imaging and electrocardiography and serum troponin level are mandatory.

### Exclusion criteria:

Patients below 16years old

Hemiplegia because of other neurological problems.

psychological hemiplegia.

Patients with chronic kidney disease or COPD, or congestive cardiac failure.

The patients of acute stroke were identified, and the questionnaire was filled during the first 72 hours: A well-constructed pretested questionnaire which include demographic data, past medical history, examination findings, imaging study .electrocardiographic records, plus troponin status. Data were analyzed by using computer program statistical package of social science (SPSS) version 25. Data were represented by tables and figures. Ethical considerations, verbal consent was taken from patient/co patient after explanation of the study, its nature, the confidential keeping of data and they have the right to quit themselves at any time during study. Ethical clearance, was obtained from the national for neurological science's ethical committee.

## Results

A total of 50 patients with acute stroke ,within 72 hours of symptoms onset were included in this study. ECG is performed to all patients on the first hours of admission, brain imaging and serum troponin I level were studied .The mean age was 70.3+/-6.4 sd, 52%of patients were males (26), 48%were females. Regarding the risk factors, hypertension was found to affect 34 patients (86%). 11 patients are known diabetics (22%).

Hyperlipidemia was identified in 10% .Moreover 10% mentioned history of previous TIA.

13 patients admitted to being smokers (26%), whereas 3 patients consume alcohol.(6%).

6% of the patients have had chronic AF.4% known to have ischemic heart disease, 4% diagnosed with valvular lesions.12%(6) of the patients have no obvious risk factor

Using the Rosier scale the suspected stroke patients were 48/50. 2 patients with scores less than 0 confirmed to have stroke by imaging.

Score 2 Rosier stroke scale: was the most frequent, documented in 22 patients (44%). 15 patients were scoring 3 (30%).Confirmatory imaging was done for 41 patients CT brain scan and MRI brain was done for 9 patients, (82%)&(18%)respectively.

50% (25) patients of stroke was ischemic in nature ..46 %(23)patients was hemorrhagic .subarachnoid hemorrhage found in 2 patients (4%),the temporoparietal region was found to be the most affected area 20% of the patient suffering hemorrhagic stroke in this area ,versus 18% of patients of ischemic stroke .basal ganglia was the second affected area (8%

Hemorrhagic ,8% ischemic stroke followed by the occipitoparietal area (8%,6% ) respectively for hemorrhage and ischemia. other sites such as, hypothalamus parietal occipital and temporal strokes percentages ranging from 4%to 2%. 37 patients of this study (%74) were suffering from a stroke that involved the anterior circulation. Posterior circulation stroke identified in 9 patients (18%).Subarachnoid hemorrhage was found in 2 patients (4%), in addition, 2 patients had bilateral / multiple stroke (4%).All EVG traces of this study showed variant abnormalities .The highest frequency were observed for

tachycardia found in 27 patients 54 % (13 of them with temporo parietal lesion). The second most common ECG finding was LVH seen in 24 patients (48%).

Bradycardia gives a percentage of (20%) identified in 10 patients. Sharing same percentage ST-depression found in 10 patients

Other Ischemic changes observed are T depression in 5 patients (10%), Significant ST elevation identified once. Arrhythmias: supra ventricular are recorded, AF (12%) Atrial flutter (2%) other SVD and PCA. ventricular fibrillation identified in one patient.

Other abnormal presentations were RBBB, LBBB, abnormal Q Wave and prolonged QT wave.

Troponin status: troponin I was positive in 7 patients (14%) whereas 43 patients (86%) were negative twice.

In the positive troponin patients, the anterior circulation stroke was identified (5 patients with temporo-prenatal stroke, 2 patients with basal ganglia stroke).

ECG changes identified in all positive troponin patients supra ventricular arrhythmias were the most frequent. ventricular fibrillation was recorded once. fibrillation was recorded once.

## Discussion

Mean age in this study was 70.3+/-6.4sd. 60% were in age ranged from 50 to 75. Evaluating Of all 50 patients included in this study 26 were males (52%), 48 % were females. the majority of them reside in Khartoum State. Risk factors hypertension affects the majority of this group, 68%. 22% were diabetic. 26% were smokers or tobacco users. Hyper lipedeamia found in 10% of these patients identified by lab results or clinically by presence of exanthemata or exthanthema. 10% of patients had a history of TIA. 6% of them with history of alcohol consumption. Regarding cardiac disease 6% with history of chronic AF, 4% known to have ischemic heart disease, 4% diagnosed with valvular lesions. 12% show no obvious risk factors. Unlike the Turkish study, there was no advanced cardiac evaluation as echocardiogram is not performed at the level of casualty. The suspected stroke patients were 48 using Rosier scale. As diagnosis of stroke is not likely if the score below zero, stroke in 2 patients scoring zero were confirmed by imaging. Stroke confirmed by CT scan in 41 patients (82%), while 9 patients (18%) underwent MRI imaging. In this study 50 % of stroke were ischemic in nature, 46% were hemorrhagic identified in 25, 23 patients respectively. 2 patients identified with sub arachanoid hemorrhage. Localizing stroke identifying the following: Both ischemic and hemorrhagic stroke tends to favor the temporo parietal regions (20% of hemorrhagic stroke/18% of ischemic).

Basal ganglia was the second affected area (8% of patients developed ischemia at this area, 8% have hemorrhage at this site). both temporo parietal and basal ganglia are supplied by the middle cerebral artery (anterior circulation.). This followed by occipital parietal area 6% suffered hemorrhagic stroke at this area while 8% developed ischemic stroke. 37 patients of this study (74%) suffering stroke involving the anterior

circulation (anterior and middle cerebral arteries). Sadber mentioned autonomic control were decreased in patients with stroke and more pronounced decrease is found in territory middle cerebral artery insular cortex.(14) All traces in this study showed ECG abnormalities, the most frequent changes was tachycardia present in 27 patients( 54%).LVH features found in 24/34 of hypertensive patients. bradycardia and ST depression ,share the same percentages of 20%. T wave inversion observed in 10%.in contrast to Goalmerza et.al who mentioned ST depression, T inversion as most frequent changes, same goes for other study done by Kokoschka Ibrahim et al.

Our observation was similar to what achieved by Sullvin Lavy who found that both disturbance in rhythm ,conduction and ischemic-T alteration were detected but frequency of the former exceeded that of later. Supra ventricular arrhythmias were caught, AF was the most frequent 12%, although half of them are known cases of chronic At .this followed by atrial flutter SVT ,PCA, also those findings are similar to findings of the Iranian study. In this study a fatal arrhythmia VF was seen once. Other ECG changes were observed in small proportions are RBBB, LBBB, abnormal Q waves, prolonged QT interval. Regarding rate changes and location tachycardia was more frequently identified in the temporoparietal lesions. 14% affect the right temporoparietal lobe .12% affect the left. Bradycardia was a closed finding to occipital, occipeto parietal lesions it documented in subarachnoid hemorrhage. The relation of rhythm changes to stroke location has no statistical significance (  $p= 0.9$ ) more than 0.05. A rare finding was presence of bradycardia in left temporo-parietal stroke this may explain the dominant parasympathetic tone of left insula. But lateralization showed no statistical significance in this study.

Troponine I is a sensitive marker of cardiac alteration ,elevated in myocardial infarction ,myocarditis ,pericarditis ,atrial fibrillation and heart failure ,elevated troponin I also has been found in patients with chronic renal failure ,sepsis ,critical illness, pulmonary embolism and COPD.

Elevated levels of troponin have been reported in 10-34% of patients with acute stroke (Kerr et al)(15). In this study 14 % have positive readings matching what have been reported. Trying to localize stroke in those with positive troponin (7 patients).it was clear that all of them have an anterior circulation stroke, (5/7) suffered temporo-parietal stroke the other two patients presented with basal ganglia stroke. All of them showed ECG changes. Increase heart rate, supraventricular arrhythmias were recognized. And fatal ventricular fibrillation was identified in one patient. The Turkish study state that a 5 patients with RMCA-insular lesions died suddenly compared with two patients of LMCA –insular lesions during hospitalization which suggest that cardiac autonomic tone may be regulated by insula, and that these patients are more prone to cardiac complications such as arrhythmias. In this study it is found that ST segment /T wave inversion more seen with the right temporo-parietal lesions more than the left lesions.

## Conclusion

The mechanism explaining morphological electrocardiogram (ECG) changes and increase in troponin in acute stroke is not clear.

The observation of this study suggests ECG abnormalities in patients with acute stroke are very common. Concomitant cardiac diseases may present.

The site of lesion appears as a factor of the genesis of arrhythmia.

Serum troponin elevation may play role in diagnosis of neurogenic injury plus ECG which appears to be non-costive,

### **Recommendation:**

ECG changes which are seriously justifying intensive monitoring.

Locating the stroke may reflect future cardiac dysfunction.

Identifying preexisting cardiac disease is important. Advanced facilities such as echocardiography are needed at the level of causality/Emergency room.

Improving stroke care capabilities may improve stroke outcomes.

## **Declarations**

### **Availability of data and materials**

The materials datasets used and/or analyzed during this study are available from the corresponding author on reasonable request.

### **Ethical Considerations**

Ethical approval was obtained from Sudan specialization board, ethical committee.

### **Competing interests**

The authors declare that they have no competing interests.

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### **Authors' contributions**

All authors participated in planning the study, data collection, results and discussion sections.

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