

Electrocardiographic Changes and Serum Troponin Levels in Patients with Acute Stroke, A Prospective Descriptive Analytical Study from Sudan

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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 diseases 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 descriptive analytical study was 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 sampling, with total coverage during study period was considered. 50 patients were included in the study. Data were analyzed by using (SPSS) version 25. 12 standards 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 suggests that ECG abnormalities in patients with acute stroke are very common, especially tachycardia. The site of lesion appears 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 wide. 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 deaths 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 evidenced by impairment of physiological regulation of heart rate and blood pressure, namely decreased heart rate variability (HRV), and impaired baroreceptors (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 month after stroke.

Autonomic dysfunction causing increased surge of catecholamine and cortisol. Catecholamine excess over activate the B adrenergic receptors, over activation of these receptors leads to tonic opening of Ca channels causing impairment in sequestration of intracellular Ca, which is necessary for relaxation of cardiac muscles. Sequestration of intracellular Ca 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 but not necessarily indicate that myocardial injury occurred. 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. 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 the cerebral cortex located within the lateral sulcus of the brain. Stimulation of the insular cortex results in cardiovascular effect with some lateralization. Stimulation of the right insular cortex more frequently produces a sympathetic effect. And stimulation of left cortical insula produces-parasympathetic (13).Small case reports of isolated insular lesions have suggested that right insular hemorrhagic or ischemic stroke resulted in bradycardia and sometimes asystole. 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 of 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

This is prospective descriptive analytical study. It 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 sampling, with total coverage during study period. 50 patients were included in the study.

Inclusion criteria:

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

Exclusion criteria:

Patients below 16 years 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 includes demographic data, past medical history, examination findings, imaging study, electrocardiographic records and 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 were taken from patient/co patient after explanation of the study, its nature, the confidential keeping of data and their right to quiet themselves at any time during study. Ethical clearance was obtained from the national Centre 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 during 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 consumed 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 were ischemic in nature. 46%(23) patients were hemorrhagic in nature. Subarachnoid hemorrhage found in 2 patients (4%).

Distribution of areas affected was as the following; The temporoparietal region was found to be the most affected area, 20% of these patients suffered from hemorrhagic stroke, while 18% suffered from ischemic stroke. Basal ganglia were the second most affected area, (8%) of these patients suffered from hemorrhagic stroke and (8%) suffered from ischemic stroke. Third most affected area was the occipitoparietal area, (8%) of these patients suffered from hemorrhagic stroke, and (6%) suffered from ischemic stroke.

Other sites such as hypothalamic, 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 ECG traces of this study showed variant abnormalities. The highest frequency were observed for tachycardia found in 27 patients

54 % (13 of them with temporoparietal lesion). The second most common ECG finding was LVH seen in 24 patients (48%).

Bradycardia gave 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 temporoparietal 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

We found that ECG abnormalities in patients with acute stroke are very common, especially tachycardia. The site of lesion appears 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.

Mean age in this study was 70.3+/-6.4sd. 60% were in age ranging from 50 to 75. 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 like hypertension affects the majority of this group(68%). While 22% were diabetic and 26% were smokers or tobacco users. Hyperlipidemia was found in 10% of these patients identified by lab results or clinically by presence of exanthemata or exanthema. Also 10% of patients had a history of TIA. 6% of them had a 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 the Rosier scale. As diagnosis of stroke is unlikely if the score below zero, strokes 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 strokes were ischemic in nature, 46% were hemorrhagic in 25, 23 patients respectively. 2 patients identified with subarachnoid hemorrhage. Localizing stroke identifies the following: Both ischemic and hemorrhagic stroke tends to favor the temporoparietal 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 temporoparietal and basal ganglia are supplied by the middle cerebral artery (anterior circulation). This is followed by the occipital parietal area as 6% suffered hemorrhagic stroke at this area while 8% developed ischemic stroke. 37 patients of this study (74%) suffers from a 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 the most frequent changes, same goes for another study done by Kokoschka Ibrahim et al.

Our observation was similar to what was achieved by Sullvin Lavy who found that both disturbances 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 is 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, occipetoparietal lesions, and was 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 temporoparietal stroke; this may explain the dominant parasympathetic tone of left insula. But lateralization showed no statistical significance in this study.

Troponin 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 temporoparietal stroke. The other two patients presented with basal ganglia stroke. All of them showed ECG changes. Increased heart rate, supraventricular arrhythmias were recognized. And fatal ventricular fibrillation was identified in one patient. The Turkish study state that 5 patients with RMCA-insular lesions died suddenly compared with two patients of LMCA –insular lesions during hospitalization which suggests 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 is more seen with the right temporoparietal lesions more than the left lesions.

Strength and Limitations:

To our knowledge this is the first study to be done in Sudan concerning this topic. We emphasized on being strict with the inclusion and exclusion criteria of selecting proper candidate to enter the study to ensure highest possible accuracy.

As any study, we had some limitations. First of all, the sample size wasn't big because in such situations, as for patients with stroke, the condition of the patients is usually critical, so it wasn't easy to collect more samples. Also the number of the doctors wasn't enough to collect samples, as many of the doctors were busy with the events of the Sudanese evolution at that time. Also we couldn't do many investigations nor investigated all the patients due to the lack of proper facilities.

Recommendation:

ECG changes which are 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.

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,

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.

Consent for publication

Consent for publication has been taken from both patients and authors.

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