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A PROSPECTIVE OBSERVATIONAL STUDY ON ELECTROCARDIOGRAM AND TROPONIN T CHANGES IN STROKE

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| ARTICLE INFO | ABSTRACT |
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| Article History: Received 9 th October, 2017 Received in revised form 10 th November, 2017 Accepted 26 th December, 2017 Published online 28 th January, 2018 | Background: The mechanisms explaining morphological electrocardiogram (ECG) changes and increased troponin T (TnT) in acute stroke are unclear. The aims of the present study were to assess the prevalence of ECG and TnT changes in acute ischemic stroke, to investigate whether ischemiclike ECG changes correlate to a rise in TnT and to examine whether ECG changes and elevated TnT predict a poor short-time outcome. Methods: From 2015 to 2017 over a period of two years a Prospective observational study on total of 200 patients suffering from acute ischemic stroke were included prospectively in |
| Key words: | the present study. ECG and TnT was carried out and analyzed at admission in all patients. |
| ECG, Stroke, Ischemia, Troponin T, MRS | MRS was compared on admission day and 90 th day with NIHS and Tnt. The troponin T values was correlated to prognosis after the evaluation of modified Rankin scale. Regression Statistics and Annova was performed and data was statistically analyzed at end of the study using SPSS ver. 21 Results: In total of 200 patients 0.5% subjects were from 0-30 yrs. of age. 45% from 31-60 yrs., majority 52.5% from 61-90 yr. and 2% above 90 yrs. 61% were male and 39% were female. Systemic illness included 78% hypertensive subjects, 89.5% diabetes mellitus, 27% had lacunar and 73/5 had large vessel stroke. NIHS had a significant value of 0.024 as 43.5 & had bad prognosis and 56.5 had good prognosis. 2% had Left ACA+Right ACA, majority of 74.5 % had Left MCA+Right MCA, 1.5% had Left MCA+ACA & Right MCA+ACA 20.5% had Left PCA+Right PCA and 1.5% had Left MCA+ACA & Right MCA+ACA 20.5% had Left PCA+Right PCA and 1.5% had Right MCA+PCA watershed. 43.5% had Normal Troponin, 38% had Borderline Troponin, and 18.5% had High Troponin. Aspect was 54.50% for 6-7, 44% for 8-9, 1.5% for more than 9. In Electrocardiogram 1% had AF, left ventricular hypertrophy, 0.5% had Deep Q wave in inferior, 1% had Hyperacute waves, 5% had Left ventricular hypertrophy, 0.5% had ST depression, T wave inversion, 0.5% had ST elevation, 0.5% had ST upsloping septal leads, 2.5 % had T wave inversion, 0.5% had ST elevation, 0.5% had ST upsloping septal leads, 2.5 % had T wave inversion, 0.5% had ST elevation, 0.5% had ST depression and Q waves are related to an increase in TnT, suggesting that these ECG changes may indicate coexisting ischemic stroke should be offered adequate treatment with secondary prevention and preferably a follow-up with focus on cardiologic as well as neurological aspects. |

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INTRODUCTION

The brain-heart connection was described early in the 20th century when Levy showed that changes in central nervous system (CNS) metabolism influenced cardiac function [1]. Later, several reports have been published regarding the role of the hypothalamus in controlling cardiac rhythm, especially the function of the sinus node [2, 3].

*Corresponding author: **Dinesh Kumar** Department of Neurology, Jubilee Mission Medical College Hospital and Research Institute, Thrissur, Kerala Morphological ECG changes of repolarization type occur when the hypothalamus and other parts of the brain are stimulated experimentally [4, 5]. In clinical practice, physicians regularly encounter patients with ECG changes related to CNS lesions. In 1947, the first clinical study of ECG changes in encephalopathy was published [6]. During the 1950s and 60s, several reports regarding ECG changes in subarachnoid hemorrhage, intracerebral hemorrhage and ischemic stroke followed up these early findings [7, 8]. Repolarization disturbances and dysrhythmias occurring in acute stroke may be due to release of catecholamine's into the patients' general circulation [9–11], direct neuronal effects mediated from the CNS via neurons ending on the heart [11] or coexisting ischemic heart disease [12]. Whereas hormonal and neuronal effects on cardiac function at present are of uncertain clinical relevance, coexisting ischemic heart disease represents a major issue. During the last decade, it has become evident that acute stroke in many cases is accompanied by a rise in the concentration of troponins in serum, indicating concomitant coronary disease [13, 14]. For the clinician, it is important to know whether ECG abnormalities encountered in stroke patients are caused by a coexisting acute coronary syndrome, as this would call for cardiologic intervention. Thus far, little is known about the clinical consequences of ECG and troponin changes in acute cerebrovascular disease.

Aims and Objectives

The aims of the present study were (i) To assess the prevalence of electrocardiogram and troponin T changes in acute stroke, and (ii) To assess the prognosis of troponin T changes with outcome in stroke. The objectives were to assess the MRS on admission day and 90^{th} day with comparison of NIHS and Tent.

MATERIALS AND METHODS

Prospective observational study was carried out on two hundred patients of acute stroke in Jubilee Mission Medical College and Research, Thrissur from 2015 to 2017 after taking consent from patient and after approval from hospital ethical committee. All the patients coming to Jubilee Mission Hospital within 48 hours of acute stroke were included in the study. Patients with previous history of coronary artery disease, patients on drugs which can cause electrocardiogram changes, all the cases of head injury, all the patients with conditions like renal failure, acute pulmonary embolism rhabdomyolysis and post defibrillation causing increase in troponin levels were excluded. The diagnosis of stroke was made mainly on history of patient, clinical examination and radiological evaluation by computerized axial tomography/ magnetic resonance imaging. Troponin T levels will be estimated in all the patients of acute stroke coming to hospital within forty eight hours of attack and on day 90th via telephonic conversation. Electrocardiogram was done in all above mentioned patients using a twelve lead Electrocardiogram machine. Patients will be classified as with elevated troponin T and without troponin elevation. National Institute of Health Stroke Scale (NIHS), Alberta Stroke Programme Early Computerized Tomography Scale (ASPECTS) was done in all patients mentioned in above study. Modified Rankin scale was done on all patients of stroke at ninetieth day. The patients with score of less than three were classified as having favorable outcome and with more than three as unfavorable outcome. The troponin T values was correlated to prognosis after the evaluation of modified Rankin scale. Regression Statistics and Annova was performed and data was statistically analyzed at end of the study using SPSS ver. 21

Table No 1 MRS DAY 1 V/S NIHS

| SUMMARY | MRS DAY 1 V/S | | | | | | | |
|-------------------|---------------|-------------------|---------|-------------|----------------|--------------|----------------|----------------|
| OUTPUT | NIHS | | | | | | | |
| Regression | n Statistics | | | | | | | |
| Multiple R | 0.418 | | | | | | | |
| R Square | 0.175 | | | | | | | |
| Adjusted R Square | 0.171 | | | | | | | |
| Standard Error | 4.923 | | | | | | | |
| Observations | 201 | | | | | | | |
| ANOVA | | | | | | | | |
| | df | SS | MS | F | Significance F | | | |
| Regression | 1 | 1023.14 | 1023.14 | 42.21 | 0.00 | | | |
| Residual | 199 | 4823.05 | 24.24 | | | | | |
| Total | 200 | 5846.19 | | | | | | |
| | Coefficients | Standard Error | t Stat | P- value | Lower 95% | Upper 95% | Lower 95.0% | Upper 95.0% |
| Intercept | 3.21 | 1.93 | 1.67 | 0.10 | -0.58 | 7.01 | -0.58 | 7.01 |
| MRS DAY 1 | 2.89 | 0.44 | 6.50 | 0.00 | 2.01 | 3.77 | 2.01 | 3.77 |

| SUMMARY OUTPUT | MRS DAY 90 V/S NIHS | | | | | | | |
|-------------------|------------------------|-------------------|--------|-------------|----------------|-----------|----------------|-----------------------|
| Regressio | on Statistics | | | | | | | |
| Multiple R | 0.28 | | | | | | | |
| R Square | 0.08 | | | | | | | |
| Adjusted R Square | 0.07 | | | | | | | |
| Standard Error | 5.21 | | | | | | | |
| Observations | 201 | | | | | | | |
| ANOVA | | | | | | | | |
| | df | SS | MS | F | Significance F | | | |
| Regression | 1 | 452.26 | 452.26 | 16.69 | 0.00 | | | |
| Residual | 199 | 5393.93 | 27.11 | | | | | |
| Total | 200 | 5846.189 | | | | | | |
| | Coefficients | Standard Error | t Stat | P- value | Lower 95% | Upper 95% | Lower 95.0% | <i>Upper</i> 95.0% |
| Intercept | 9.90 | 1.42 | 6.96 | 0.00 | 7.10 | 12.71 | 7.10 | 12.71 |
| MRS at day 90 | 1.71 | 0.42 | 4.08 | 0.00 | 0.88 | 2.53 | 0.88 | 2.53 |

RESULTS

CUMMADY

Data from the patients records regarding blood pressure, hyperlipidemia, diabetes mellitus, cigarette smoking, previous stroke and coronary heart disease were recorded. Neurological outcome was assessed by the modified Rankin scale (MRS) [16] at 90th day via telephonic conversation. The MRS is a global stroke scale comprising the following subgroups: 0 1/4 no symptoms, 1 ¼ no significant disability despite symptoms (able to carry out all usual activities), 2 ¹/₄ slight disability (unable to carry out all previous activities but able to look after own affairs without assistance), 3 ¹/₄ moderate disability (requiring some help but able to walk without assistance), $4\frac{1}{4}$ moderately severe disability (unable to walk without assistance and unable to attend to bodily needs without assistance), $5\frac{1}{4}$ severe disability (bedridden, incontinent, requiring constant nursing care), 6 ¹/₄ death. In statistical analyses, the MRS scores were dichotomized into favorable outcome (0-3) versus unfavorable outcome (4-6). In total of 200 patients 0.5% subjects were from 0-30 yrs. of age. 45% from 31-60 yrs., majority 52.5% from 61-90 yr. and 2% above 90 yrs. 61% were male and 39% were female. Systemic illness included 78% hypertensive subjects, 89.5% diabetes mellitus, 27% had lacunar and 73/5 had large vessel stroke. NIHS had a significant value of 0.024 as 43.5 & had bad prognosis and 56.5 had good prognosis.

According to Troponin T level 43.5% had bad prognosis and 56.5% had a good prognosis. 2% had Left ACA+Right ACA, majority of 74.5 % had Left MCA+Right MCA, 1.5% had Left MCA+ACA & Right MCA+ACA 20.5% had Left PCA+Right PCA and 1.5% had Right MCA+PCA watershed. 43.5% had Normal Troponin, 38% had Borderline Troponin, and 18.5% had High Troponin. Aspect was 54.50% for 6-7, 44% for 8-9, 1.5% for more than 9. In Electrocardiogram 1% had AF, left ventricular hypertrophy, 0.5% had Deep Q wave in inferior, 1% had Hyperacute waves, 5% had Left ventricular hypertrophy, 0.5% had LBBB, majority of 58.5% were normal, 2% had prolonged QT, 6% had RBBB, 22% had ST depression, T wave inversion, 0.5% had ST elevation, 0.5% had ST upsloping septal leads, 2.5 % had T wave inversion, 0.5% had U wave. MRS day 1 v/s NIHS and MRS day 90 v/s NIHS were significant in ANOVA(Table 1 & 2) also MRS day 1 v/s Trop T and MRS day 90 v/s Trop T had significant value. (Table 3 & 4)

DISCUSSION

We found a high prevalence of ECG abnormalities in the acute stage of ischemic stroke. The most frequent changes were prolonged QTc, atrial fibrillation and ECG changes consistent with ischemic heart disease, i.e. Q waves, ST depression and Twave inversion.

| SUMMARY OUTPUT | MRS DAY 1 V/S TROP T | | | | | | | |
|---|-------------------------------------|---------------------------|---------------|-------------|-------------------|--------------|----------------|----------------|
| Regres | ssion Statistics | | | | | | | |
| Multiple R R Square Adjusted R Square Standard Error Observations | 0.40 0.16 0.16 0.72 201 | | | | | | | |
| ANOVA | | | | | | | | |
| | df | SS | MS | F | Significance F | | | |
| Regression Residual Total | 1 199 200 | 19.57 102.97 122.55 | 19.57 0.52 | 37.82 | 0.00 | | | |
| | Coefficients | Standard Error | t Stat | P- value | Lower 95% | Upper 95% | Lower 95.0% | Upper 95.0% |
| Intercept | 3.87 | 0.08 | 47.89 | 0.00 | 3.71 | 4.03 | 3.71 | 4.03 |
| TROP T | 12.29 | 2.00 | 6.15 | 0.00 | 8.35 | 16.23 | 8.35 | 16.23 |
| | Tab | ole No 4 MR | RS DAY | 90 V/S | TROP T | | | |
| SUMMARY OUTPUT | MRS DAY 90 V/S TROP T | | | | | | | |
| Regre | ession Statistics | | | | | | | |
| Multiple R | 0.49 | | | | | | | |
| R Square | 0.24 | | | | | | | |
| Adjusted R Square Standard Error | 0.24 0.77 | | | | | | | |
| Observations | 201 | | | | | | | |
| ANOVA | 201 | | | | | | | |
| ANOVA | df | SS | MS | F | Significance F | | | |
| Regression | 1 | 37.82 | 37.82 | 64.08 | 0.00 | | | |
| Residual | 199 | 117.45 | 0.59 | | | | | |
| Total | 200 | 155.2637 | | | | | | |
| | Coefficients | Standard Error | t Stat | P- value | Lower 95% | Upper 95% | Lower 95.0% | Upper 95.0% |
| Intercept | 2.75 | 0.09 | 31.86 | 0.00 | 2.58 | 2.92 | 2.58 | 2.92 |
| TROP T | 17.09 | 2.13 | 8.00 | 0.00 | 12.88 | 21.29 | 12.88 | 21.29 |

Table No 3 MRS DAY 1 V/S TROP T

However, ischemicECG changes are known to be prevalent in elderly patients [17, 18], and, accordingly, as prestroke ECG records were not evaluated, pre-existing ECG changes cannot be differentiated from changes that occurred after onset of stroke symptoms. Needless to say, a study based on ECG registrations recorded within a few days before onset of stroke symptoms, would be impossible to carry out. The present study concentrates on the acute phase of ischemic stroke, and data regarding the long-term outcome of the included patients, are not at hand. Consequently, the results must be interpreted with caution. Yet, the study is based on a strictly nonselected stroke sample, in the sense that all included patients were admitted directly to the stroke without regard to his or her clinical condition. A follow-up study clearly would be useful in order to assess the prognostic value of TnT and ECG changes in the longer term, not only regarding neurological sequels but also regarding cardiologic complications such as recurrent ischemia, myocardial infarction and congestive heart failure amongst these patients. The prevalence of elevated TnT is lower in the present sample than in previous studies [13, 14]. We have no obvious explanation for this. TnT concentrations were measured both at admission and on day 1, and, consequently, we have no reason to suspect that the blood samples were drawn too early to detect a rise in troponins. TnT was chosen as the gold standard for myocardial injury. TnT is found to be highly specific for myocardial necrosis [19] although the exact mechanism explaining a rise in TnT in stroke patients is not fully understood. It can be debated whether myocardial necrosis, expressed as a rise in TnT, is due to ischemic or non-ischemic heart disease [19]. Nevertheless, TnT plays a major role in diagnosing ischemic heart disease in cardiac care units worldwide. Q waves and ST depressions are ECG changes typically associated with ischemic heart disease. Q waves indicate a previous or recent myocardial infarction [20] whereas ST depressions may represent a current acute coronary syndrome [21]. In patients presenting with symptoms of an acute coronary syndrome, e.g. chest pain or dyspnoea, the significance of such ECG changes are obvious. However, in stroke patients without clinical symptoms consistent with coronary disease, less is known about the implications of ischemic-like ECG changes. The relationship between these ECG changes and elevated TnT found in the present study may indicate that such ECG abnormalities are caused by concomitant ischemic heart disease rather than by hormonal or neuronal effects of the cerebrovascular lesion. In these patients, medical or interventional cardiologic therapies may have beneficial effects. Every cerebrovascular accident is likely to exert a considerable stress on the patient's heart [22]. Patients with acknowledged coronary disease, probably have a reduced prestroke cardiac function. Elevated TnT, therefore, may represent a lower cardiac tolerance to stress caused by the cerebrovascular event. This may be one explanation for the relationship between elevated TnT and a poor short-term prognosis found in the present study. The clinical relevance of a prolonged QTc is under debate [23]. It has been suggested that prolonged QTc is associated with sudden death in some medical conditions, e.g. myocardial infarction [23], epilepsy [24] and subarachnoid hemorrhage [25]. In the present study, prolonged QTc was related to an unfavorable short-term neurological outcome but not to death. One reason may be that prolonged QTc occurs in patients with large cerebral lesions, and thus reflects a critical medical condition caused by a severe ischemic stroke. Validation of a prolonged QTc and a

rise in TnT concentration as predictors for an unfavorable outcome showed that these variables had low sensitivities. One reason may be that other factors, such as the size of the cerebral lesion, have a considerable effect on the patients' outcome. However, in clinical practice, the predictive value of a positive test may be just as relevant as the sensitivity. In the present study. We conclude that prolonged QTc and ECG changes of coronary disease type are prevalent in the acute stage of ischemic stroke. Regardless of whether these ECG changes arose before or after the onset of stroke symptoms, the relationship between elevated TnT and ischemic-like ECG changes suggests that Q waves and ST depressions in many cases are caused by co-existing ischemic heart disease. MRS day 1 v/s NIHS and MRS day 90 v/s NIHS were significant in ANOVA(Table 1 & 2) also MRS day 1 v/s Trop T and MRS day 90 v/s Trop T had significant value. (Table 3 & 4). Stroke patients with elevated TnT have a less favorable short-term outcome than other stroke patients. Doctors in charge of the stroke unit should be extra observant when dealing with these patients, and take care to offer an adequate secondary prevention. Patients should be ensured anti-platelet therapy or, when cardio-embolic disease is present, anti-coagulation therapy. In addition, a proper intervention regarding risk factors, especially hypertension, hyperlipidemia, diabetes and smoking should be offered. A clinical follow-up of these patients a few weeks after onset of stroke would help doctors assure that appropriate treatment has been given.

Clinical Importance

Stroke is one of the most disabling diseases of nervous system. The relationship between acute stroke and coronary artery disease is complex and they are related to each other in multiple ways (26). Acute stroke confers significant increase in adverse cardiac outcomes during short term and longtime follow up. A subset of patients with stroke might be at higher risk for long term adverse cardiovascular outcomes. Identifying these patients, ideally with a simple test or biomarker can help to reduce their long term risk of adverse events. Troponin is a highly sensitive and specific marker for myocardial necrosis and prognosis that is used in diagnosis and prognosis of patients with acute coronary syndrome (26). However troponin elevation has been documented in multiple clinical settings in absence of acute coronary syndrome (27, 28).Increase in troponin has been documented in all types of stroke including ischemic and hemorrhagic stroke (29). Several studies have confirmed the elevation of troponin T in high catecholamine states such as ischemic stroke. The high concentration of catecholamine's in the myocardium can lead to impairment of cardiac function due to perfusion disturbances at level of capillaries caused by enhanced platelet aggregation (30). The prognostic value of elevated troponin in acute cerebrovascular accident remains a controversial topic. Increased mortality has been predicted by elevated troponin studies (31). In most of situations it remains unclear if a patient had acute myocardial infarction that leads to stroke or other way around. In absence of evidence for an acute coronary syndrome one should consider the long term significance of elevated troponin. Electrocardiogram changes were observed in patients with acute ischemic stroke and may cause diagnosis and management dilemmas. The most frequent abnormalities noted in various studies were prolonged QT interval, q waves, ST -T changes, bundle branch block, premature complexes and atrial fibrillation with ischemic stroke and intracerebral

hemorrhage (32). The changes like atrial fibrillation were more common in ischemic stroke than hemorrhagic stroke. Electrocardiogram changes noted in various studies in ischemic stroke patients include pathological q waves, atrioventricular block, and atrial fibrillation, left ventricular hypertrophy, ST segment elevation / depression and bundle branch block. It has been found in literature that augmentation of intracardiac sympathetic nerve activity occurs producing cardiac myocyte damage and depolarizing ionic shifts, resulting in electrocardiogram repolarization changes and arrhythmogenesis (32).In Kerala large scale studies are not yet conducted to assess the prevalence of electrocardiogram and troponin T changes in acute stroke

CONCLUSION

ECG changes are prevalent in acute ischemic stroke. ST depression and Q waves are related to an increase in TnT, suggesting that these ECG changes may indicate coexisting ischemic heart disease. A rise in TnT predicts a poor outcome. Patients with acute ischemic stroke should be offered adequate treatment with secondary prevention and preferably a follow-up with focus on cardiologic as well as neurological aspects.

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