

Cardiac Troponin Levels in Cerebrovascular Accidents

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ABSTRACT

Background: It has been observed that in patients of acute cerebrovascular accidents (CVA), cardiac troponin levels are frequently raised in absence of acute cardiac insult and even in the absence of significant past history of ischemic heart disease.

Aim: To find out relation and prognostic significance of raised levels of cardiac troponin in acute CVA, an observational study was conducted in patients of acute CVA in which their cardiac troponin levels were measured.

Materials: 100 patients of acute cerebrovascular accident were enrolled in the study. Only patients of acute ischemic stroke, presenting within 24 hours were selected for the study. A detailed history regarding ischemic heart disease was taken and patients who were having cardiac problem in the past were excluded from the study. Other causes of similar symptoms were also ruled out by carrying out a detailed metabolic profile and CT scan brain was kept as a diagnostic tool to confirm the CVA.

Results: Out of 100 patients 51 were having raised troponin I. Furthermore, their ECG was also showing acute ischemic changes although past history of ischemic heart disease was not present. During their average hospital stay of 7 days 2 died due to various complications. Rest were discharged and followed up every month, apart from symptoms and risk factors of CVA also for any cardiac symptoms on monthly basis for 6 months. Their ECG was also done regularly and echocardiography was also performed on monthly basis.

Conclusion: It can be correlated that cardiac troponin levels can also get raised during brain injury especially the massive one even in the absence of acute or previous cardiac injury and it is perhaps a poor prognostic factor regarding future cardiovascular events. So, such patients should be also carefully monitored for development of any cardiac event in the next few months in order to prevent major cardiovascular mortalities in these patients.

Keywords: Cardiac troponin levels, CVA, IHD

INTRODUCTION

It has been observed that acute CVAs and coronary heart disease share many risk factors. Furthermore, it is also noticed that patients with acute CVAs are somehow more vulnerable to acute coronary events in the short or long term. (1) If we can identify those patients of stroke, who have specifically increased short- or long-term risk of coronary heart disease, we can more vigilantly monitor and hence treat them. This can be done by doing simple test like cardiac troponin levels. Apart from cardiac troponin levels some patients of stroke also show acute ischemic changes in ECG. (2-3). Cardiac troponins T and I are quite specific for myocardial injury^{4,5}. Elevation of these cardiac troponins in acute ischemic CVAs show that some sort of myocardial insult also occurs in CVAs in the absence of acute coronary syndrome or even in the absence of significant past history of IHD. It has been observed that in almost 5 to 34% patients of acute ischemic CVAs, cardiac troponin T and I levels are also elevated⁶. In several studies the raised cardiac troponin level was associated with long- and short-term outcomes and increased risk of mortality. (7,8,9,10). Various mechanisms have been proposed for this cardiac insult in acute ischemic CVAs. One mechanism of this neurocardiogenic damage is autonomic imbalance after stroke and increased surge of catecholamines, which may cause global left ventricular dysfunction^{11,12}. So, our purpose of study would be to identify those patients of acute ischemic CVAs, which have high levels of cardiac

troponin, and intensive monitoring of these patients regarding cardiac symptoms and signs, echocardiography and hence preventing adverse cardiac outcome in these high-risk patients.

METHODS & MATERIALS

First 100 patients of Ischemic CVAs, diagnosed on CT scan, presenting in emergency were enrolled in study after taking proper consent. Their proper history and all routine metabolic profile were done, in order to diagnose CVA and rule out other similar metabolic conditions. An ECG was also done to see any ischemic changes. Past history was also thoroughly enquired to rule out any ischemic heart disease. Cardiac troponin I and T were done by ELISA. Depending on raised cardiac troponin levels and ECG changes CVA patients were categorized in 2 groups, those with raised cardiac troponin levels and those CVA patients without raised troponin. Then the first group was followed up during their stay in the hospital and then on monthly basis for 6 months. After discharge by filling a questionnaire asking ischemic symptoms and getting done their ECG and echocardiography. Percentage of patients developing ischemic symptoms, developing ECG changes or echocardiographic changes and those developing major ischemic events were recorded. Then these results were compared with international studies.

RESULTS

Male and female patients of age group 41±22 years were in our study. 73 patients were male and 27 were females. 52 patients show a definite infarct while 48 were showing focal

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neurological deficits and ruled out for any metabolic cause but their CT was normal, probably done earlier in window period. 51 patients show increased troponin T or I levels with or without ECG changes of ischemic nature. 15 out of these 51 patients (29%) showed ECG changes, while 36(71%) showed raised cardiac enzymes without any ECG changes. Out of these 51 patients, those who were showing ischemic changes too i.e., 29%. 2(3.9%) patients died during their hospital stay averaging almost 7 days. Then during their monthly follow up 18 (35%) patients develop either ischemic symptoms or showed ECG changes in first 2 months. While 4(8%) patients develop major MI in first 3 months.

DISCUSSION

Increased troponin levels in CVAs predicts increased mortality as evident by many studies¹³⁻¹⁶. Elevated levels of cardiac troponin in 10 to 34% patients in a study conducted by Kerr et al in comparison to 51% patients of our study in which trop I was raised¹⁷. Kerr et al measured levels of trop I in 7 days of symptoms. Our study indicated that 29% patients with raised troponin levels developed ischemic symptoms in first 2 months. 4 developed major MI attack. This finding is in accordance with the studies conducted by Jensen et al, Faiz et al^{18,19} which also showed increased rate of ischemic symptoms in patients who show raised troponin levels after acute CVAs. That is why American heart association routinely recommends measuring levels of these biomarkers²⁰. A big prospective trial TRELAS (Troponin elevation in acute ischemic stroke) is a prospective study which analyzed that how many patients with acute ischemic stroke develop raised cardiac troponin in first 72 hours and it showed that median cTn on presentation did not differ between patients with acute ischemic stroke and non ST elevation MI. This study also supported our study which also showed that considerable percentage of patients with acute ischemic CVA were with raised troponin I levels. However coronary angiography in TRELAS showed that coronary lesions were less in patients of acute CVA with raised cardiac troponin but without history of IHD than the patients with acute coronary syndrome with raised troponin²¹. But one thing is clear that patients with acute ischemic CVAs may have raised trop I or T levels and might have significant coronary vessel lesions which can decrease their survival if not identified in time. Subendocardial hemorrhage or swollen myocytes surrounding epicardial nerves during early stroke has been suggested to be the cause of cardiac damage⁽²²⁾. Another retrospective study showed that 16.8 % patients with acute ischemic stroke show elevated troponin levels although this percentage is very less than our percentage of patients i.e., 51% but it clearly indicated that there is some degree of neurogenic cardiac damage during acute ischemic stroke²³. Barber et al²⁴ found that the cause of raised cardiac troponin levels is increased surge of catecholamine in patients of acute ischemic stroke. But whatever the cause of this cardiac damage is, it must be identified and should be corrected in order to prevent long term morbidity and mortality.

CONCLUSION

Although our study was on small scale enrolling a small number of patients. It clearly indicated that a significant number of patients develop cardiac damage too in ischemic CVA. These patients develop cardiac symptoms in few months after CVA and few of them also develop major MI and these patients also have positive ECG changes and structural echocardiographic changes if these patients are properly identified these untoward cardiac events can be prevented.

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