Cardiac electrical and biochemical abnormalities in acute stroke: A case report

**M. Umair Bakhsh, Hassan Alkhawam, Feras Zaiem,
Mohammed El-hunjul, Jaspirt Takher, Anirudh Pareek,
Robert Sogomonian**

ABSTRACT

Introduction: Cardiac troponin T (cTnT) is a highly sensitive and specific marker of myocardial necrosis that aid in diagnosis of myocardial infarction. The cTnT elevations along with ECG changes noticed in patients with acute Stroke without major cardiac events.

Case Report: We report a case of 87-year-old female presented with cough, lethargy, decreased oral intake, shortness of breath, chest pain and altered mental status. Head computed tomography (CT) scan did not reveal any acute intracranial pathology. Laboratory work showed cardiac troponin T elevation with ECG changes were concerning for cardiac ischemia. Careful examination was significant for right facial droop and right sided weakness. A repeat head CT scan showed acute stroke.

Conclusion: Patient with ischemic stroke may have shown to have an association with elevated cTnT. They are at an increased risk of mortality from renal and/or cardiac failure. Though ECG changes and cTnT levels can be attributed to stroke, given the common risk factors shared by coronary artery disease and stroke, a cardiac work up is warranted to rule out any acute myocardial event or look up for the source of stroke which most likely to be cardiac.

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CASE REPORT

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attributed to stroke, given the common risk factors shared by coronary artery disease and stroke, a cardiac work up is warranted to rule out any acute myocardial event or look up for the source of stroke which most likely to be cardiac.

Keywords: Acute stroke, Cardiac, Coronary artery disease, Myocardial infarction, Stroke, Troponin

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INTRODUCTION

Cardiac troponin T (cTnT) is a highly sensitive and specific marker of myocardial necrosis that aid in diagnosis of myocardial infarction. The cTnT elevations along with ECG changes have been reported previously in patients with acute Stroke (ischemic or hemorrhagic [1]. Literature search demonstrated that Electrocardiographic (ECG) changes might be seen with acute stroke. Various mechanisms have been set forth to validate the relation between acute stroke and troponin elevation with ECG changes which are imputed to the increase in catecholamine, which in turn, lead to myocyte injury [2–4]. Elevated troponin is seen after stroke and is associated with increased mortality [5]. We reported a case and then reviews different mechanisms involved in troponin elevation in the setting of acute stroke.

M. Umair Bakhsh¹, Hassan Alkhawam¹, Feras Zaiem², Mohammed El-hunjul³, Jaspirt Takher¹, Anirudh Pareek¹, Robert Sogomonian¹

Affiliations: ¹Department of Medicine, Elmhurst Hospital Center-Icahn School of Medicine at Mount Sinai, Elmhurst, NY, USA; ²Mayo Clinic, Mayo Evidence Based Practice Center, Rochester, MN; ³Department of Medicine, Saint Joseph Hospital, Chicago, IL, USA.

Corresponding Author: Hassan Alkhawam, MD, Department of Medicine, Elmhurst Hospital Center, Icahn School of Medicine at Mount Sinai, 7901 Broadway, Elmhurst, New York 11373, USA; Email: hassan.alkhawam@mssm.edu

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CASE REPORT

We report a case of a 87-year-old female with past medical history significant for hypertension, lymphoma status post neck radiation, aortic stenosis, dementia and glaucoma. This patient presented with cough for 3 days, lethargy and decreased oral intake. The patient reports no fever, chills, nausea, vomiting, diarrhea or palpitations. About four hours prior to admission, patient experienced shortness of breath and increased cough and chest pain for which she was taken to the emergency department where she was found to have altered mental status. Laboratory work showed cardiac troponin T elevation, cTnT was found to be 1.6 ng/mL (normal range 0.0 – 0.2 ng/mL). ECG changes were concerning for cardiac ischemia - T wave inversions in lead V1-V3, with no prior ECG for comparison (Figure 1). Head CT scan did not reveal any acute intracranial pathology. Given her negative head CT scan and ECG changes she was given heparin bolus, clopidogrel 600 mg, aspirin 325 mg and cardiologist consulted for possible catheterization.

Upon arrival patient was alert, afebrile with stable vital signs. Initial physical examination was significant for altered mental status but later on, patient was noticed has right facial droop and right sided weakness. A repeat head computed tomography (CT) scan showed right cerebellar hypodensity consistent with acute stroke. Thereafter, she became lethargic and the arterial blood gas showed severe acidosis with hypercarbia due to which she had to be intubated. Neurosurgery was consulted and she was started on hypertonic saline and dexamethasone for concern of cerebral edema and risk of possible midline shift of the brain.

Her cardiac troponin T (cTnT) on admission was 1.37 ng/mL, which peaked to 1.5 ng/ml and then finally trended down to 0.2 ng/ml. Patient's creatine kinase (CK) MB isoenzyme level on admission was 6.3 ng/ml which trended down to 2.4 ng/ml (CK-MB normal range 0–3 ng/mL). Her initial 12 lead ECG on admission showed T wave inversion in leads V1-V2, with mild elevations in V4-V6, however, serial ECGs showed resolution of T wave inversion and ST elevations (Figure 1).

Echocardiography (echo) showed normal left ventricular size with hyperdynamic left ventricular systolic function and moderate to severe aortic stenosis. Carotid Doppler showed small atherosclerotic plaques at both carotid bifurcations however there was no evidence of obstruction to flow.

The serial head CT scan of the patient showed acute right cerebellar infarction with interval increase from 2.5 to 3.3 cm. Magnetic resonance imaging (MRI) scan showed multifocal infarcts with largest involvement within the right superior cerebellum, also additional infarcts were seen in the left cerebellum and left parietal lobe and areas of petechial hemorrhages in the right superior cerebellar infarct. Serial ECGs were stable with down trending troponin, therefore cardiology team at

our center considered not to perform any cardiology intervention.

DISCUSSION

Stroke is the fourth leading cause of death [6] and the leading cause of long-term disability in United States. Stroke is defined as an acute neurological condition that occurs either due to brain ischemia (thrombosis, embolism, systemic hypoperfusion) or brain hemorrhage (intracerebral hemorrhage or subarachnoid hemorrhage). 80% of the stroke is ischemic in nature while 20% is hemorrhagic. The cTnT is a highly sensitive and specific marker for myocardial necrosis that is used in risk stratification of patients with acute coronary syndrome and is used in the diagnosis of acute myocardial infarction. There is a complex overlap between cardiovascular and cerebrovascular diseases. Interestingly, elevations of cardiac troponin levels and ischemic ECG changes have been described previously in the setting of acute ischemic stroke [1]. Patients with elevated troponin levels were more likely to have myocardial ischemia, as seen on the ECG [7].

Elevated cTnT have been reported in 5–34% of the patients with acute ischemic stroke. This was shown by another study, which enrolled 222 subjects, which showed that 20% of the patient had troponin > 0.2 µg/L. It was seen that these patient also had higher catecholamine

Figure 1: ECG changes were concerning for cardiac ischemia - T wave inversions in lead V1–V3.

levels when measured and ECG changes were suspicious of acute myocardial infarction [8]. Patients with elevated cTnT had evidence of myocardial damage in 10–34% of patients with acute ischemic stroke[9–13].

The mechanism associated with acute ischemic stroke causing cTnT elevation and ECG changes is not fully understood. A few possible explanation have been set forth describing this association. One possible explanation is associated with loss of the inhibitory effect of sympathetic stimulation that is exerted by the insular cortex. Right insular cortex maintains its inhibitory effect by suppressing the sympathetic system. Once this area is affected there is loss of sympathetic inhibition leading to catecholamine surge in the body which can lead to myocytolysis [14]. This subsequent catecholamine surge may also induce LV dysfunction with LV apical ballooning [2–4]. Imaging showed that patient with raised troponin have stroke that more commonly involved the right posterior, superior, and medial insula and the right inferior parietal lobe exerting loss of inhibition of sympathetic pathway by insular cortex [13].

Another possible explanation of the elevated cTnT in stroke patient can be attributed to the common risk factors that are shared by stroke and myocardial infarction and increased prevalence of coronary artery disease in stroke patient [15]. Also, any stressful condition such as stroke would trigger the body's natural response to release glucocorticoids and catecholamine that might be an additive factor contributing to elevated cTnT in the setting of stroke.

Patients who were admitted with an acute ischemic stroke, serum cTnT level at the time of admission was a powerful predictor of mortality [5]. Furthermore, patient with elevated cTnT had a higher frequency of heart and/or renal failure as well as increased risk of mortality within the following two years [16].

CONCLUSION

Patient with ischemic stroke have shown to have an association with elevated cardiac troponin T (cTnT). They are at an increased risk of mortality from renal and/or cardiac failure. Multiple explanations have been set forth to define the association. Though ECG changes and cTnT levels can be attributed to stroke, given the common risk factors shared by coronary artery disease and stroke, a cardiac workup is warranted to rule out any acute myocardial event.

Author Contributions

M. Umair Bakhsh – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Hassan Alkhawam – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Feras Zaiem – Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published

Mohammed El-Hunjul – Substantial contributions to conception and design, Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published

Jaspirt Takher– Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Anirudh Pareek– Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Rober Sogomonian– Substantial contributions to conception and design, Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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