Case Report

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Diagnostic Dilemma in a Case of Ischaemic CVA

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Abstract

Background: A 72 years aged female patient presented with history of chest pain which was resolved with medication. Later she developed slurring of speech, weakness of left side of body, hoarseness of voice, deviation of mouth to right. *Case presentation:* Initial ECG had ST depressions and T inversion, bedside qualitative Trop-T was positive with a unremarkable CT brain plain even after 24 hours of onset of her symptoms suggestive of stroke, arouse a diagnostic dilemma for the emergency physician of coexisting NSTEMI with ischemic stroke in her case and patient was initially admitted under cardiology. Further work up ruled out NSTEMI and confirmed MCA territory acute ischaemic stroke. *Conclusion:* Acute stroke patients may have ischaemic changes and / or QT prolongation in ECG and elevated cardiac troponins, probably as a result of Neurogenic cardiac damage, which may arise diagnostic dilemma for the emergency physician as in this case and it shows prognostically poor outcome in acute stoke stroke patients.

Keywords: Stroke; Troponin; Coronary Syndrome.

Background

Heart disease and stroke show intense connections as indicated by the high proportion of strokes with cardioembolic origin whereas, different types cardiac derangement like myocardial dysfunction, arrhythmia, ECG alterations and rise of cardiac troponins [1,2] commonly occurs in the context of acute neurological disorders also.

Several studies have reported an increased cTnT in 5-34% of patients with acute ischemic stroke [8] which was associated with stroke severity on hospital admission, short- long-term clinical outcome and increased risk of mortality [9,10,11,12], indicating prognostic significance of increased cTnT in acute ischemic stroke.

The cause of rise of serum troponin level is unknown but possibly related to the uncertain. Coincidence of acute coronary syndrome (ACS) leading to ischemic myocardial necrosis because stroke and myocardial infarction share common risk factors and there is a high prevalence of coronary artery disease (CAD) in stroke patients [13].

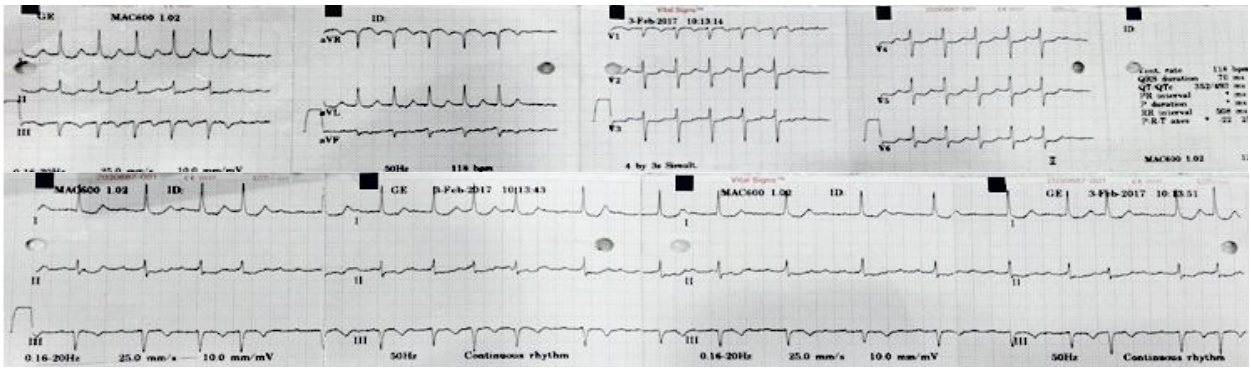
Another possible cause is Neurogenic cardiac damage where cardiac injury might result from autonomic imbalance after stroke [14].

Due to the uncertainty regarding the underlying etiology of cTnT elevation in ischemic stroke patients the extent and time point of cardiologic work-up in this clinical situation is still under debate. Therefore, there is uncertainty about how acute ischemic stroke patients with increased cTnT levels should be managed regarding diagnostic and therapeutic workup.

Case History

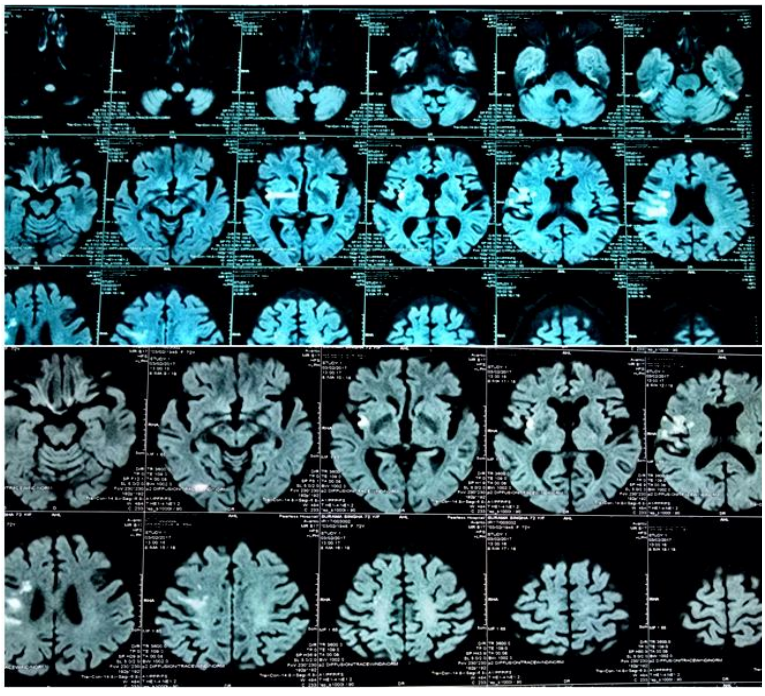
A 72 years old female patient who was a known hypertensive for last 8-10 years, presented in the emergency department with a history of sudden onset pain upper central abdomen and chest, radiating to back and left arm, associated with mild respiratory distress, lasted for more than 2 hours, gradually

increasing, aprox 36 hours back from presentation in emergency, when she was taken to local general practitioner who treated her with injectable Dicyclomine hydrochloride and pain subsided after approximately 15-20 minutes thereafter, after which she returned to home. Then almost 8 hours after that she developed slurring of speech, deviation of mouth to right side, weakness of left side of body and hoarseness of voice. She had no history of convulsion / fall / head injury / vomiting / loss of consciousness / incontinence / amnesia / fever. On presentation in emergency her vitals were stable. She was conscious, alert and oriented with GCS: E4 V5(hoarseness of voice and slurring of speech were present) M6. Pupils were 2.5 mm bilaterally equally reactive to light. Planter reflex: right- Flexor, left-equivocal. Power: right-5 / 5, left-4 / 5. Deviation of mouth to right side. A CT scan was done which was unremarkable. ECG-12 leads were showing atrial fibrillation with fast ventricular rate with 'ST' depression in V4, V5, V6 and 'T' inversion in V3. Bedside Trop-T was also positive.



Patient was admitted under cardiology with provisional diagnosis of NSTEMI with ischaemic CVA and started on oral statin, dual antiplatelet, s / c LMWH and other supportive medications. Later her 2D-Echocardiography was done: no RWMA, grade1 diastolic dysfunction with LVEF=65%. Lab reports were, normal WBC count, creatinin-0.53, normal coagulation profile platelet count-2lakh / cumm, CKMB=8, normal liver and thyroid function test. MRI brain was suggestive of acute infarction of right MCA territory. Carotid Doppler: mild diffuse increase in intimal thickening, no stenosis or soft plaque. Next day his GCS decreased to E2V3M6, ECG-12 leads returned to normal sinus rhythm with resolved ST depressions and T inversion. NSTEMI was ruled out and the patient was transferred under Neuromedicine.

MRI Brain Plain



Discussion

Cardiac abnormalities in patients with acute stroke were first noticed in 1947 in 4 patients which often cause diagnostic as well as therapeutic dilemma for clinicians. Khechinashvili et al. in a systematic review showed abnormalities like ischaemic changes in ECGs and / or QT prolongation in patients with acute stroke, which were mostly prevalent in patients with pre-existing coronary artery disease. They found ECG changes are non specific to diagnose acute MI in settings of acute stroke. Dogan et al. showed ECG abnormalities are frequently seen in stroke patients without any pre-existing heart disease and atrial fibrillation is more common in ischaemic stroke than hemorrhagic stroke. Balaney B, et al. has done a literature review of elevated cardiac troponin level in acute ischaemic stroke where he found that It is not uncommon to see elevated troponin in the setting of acute ischaemic stroke. In TRELAS study only approximately 24% of patients with acute ischemic stroke and troponin elevation have angiographic evidence of a "culprit lesion" and the rate of percutaneous vascularization was only 21% in this cohort. Nevertheless, elevated troponin has worse prognosis in stroke patients and should prompt closer evaluation for ischemic heart disease. A meta analysis by Touze´ et al concluded that Patients with TIA or stroke have a relatively high risk of MI and nonstroke vascular death, risk of MI at 10 years after acute stroke was approximately 20%. Though the cause of these ischaemic ECG abnormalities, arrhythmias and elevated cardiac troponin level remains unclear. G.V. Dous et al. in his literature review addressed it as a neurogenic heart syndrome (NHS). It is a neurally mediated process due to increase in catecholamine release as a result of hypoperfusion of the posterior hypothalamus causing autonomic nervous system imbalance and increased sympathetic output. Therefore, activation of the sympathoadrenal system could be an important contributor to myocardial damage in stroke patients causing elevation of cardiac troponins and ECG abnormalities.

Conclusion

Acute stroke patients may have ischaemic changes and / or QT prolongation in ECG and elevated cardiac troponins with or without any pre-existing cardiac disease, probably as a result of neurogenic cardiac damage, which may arise diagnostic dilemma for the emergency physician as in this case. Though it is uncommon to have an acute MI in these group of stroke patients still they should undergo thorough cardiac evaluation for coronary artery disease to exclude their coexistence in acute stroke settings. And even if coronary artery disease is excluded in them, elevated cardiac troponin and ischaemic ECG abnormalities are bad prognostic indicators in acute stroke patients with higher incidence of MI in future.

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