A Case Report on Cerebrogenic Fatal Cardiac Arrhythmia in a Patient with Acute Ischemic Stroke

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Abstract

Background: Patients with acute ischemic stroke are susceptible to cardiac arrhythmias however, fatal arrhythmias are rare in the absence of cardiac disease. Cardiac arrhythmias can develop in lesions at the right side of the brain specifically the insular, frontal and parietal area. Data that show the direct relationship of ischemic stroke and arrhythmia are scarce but they are indirectly attributed to an imbalance in the autonomic nervous system. This paper aims to present a rare case of an association between a fatal arrhythmia and right thalamic infarct.

Case: Presenting a case of a 39-year-old admitted as a survivor of sudden cardiac death from ventricular fibrillation. She presented with a history of left sided weakness a week prior but no work-up was done. Baseline serum electrolytes and cardiac markers were all normal. Electrocardiogram (ECG) post-cardioversion showed sinus tachycardia. Echocardiogram and cardiac computed tomography (CT) angiography were normal. Magnetic resonance imaging (MRI) and angiography (MRA) of the brain showed an acute infarct at the right thalamus and an absent left internal carotid artery (ICA). Electroencephalogram (EEG) was negative. Bisoprolol was given and an Automatic Implantable Cardioverter Defibrillator (AICD) was subsequently placed. No recurrence of cardiac arrhythmia was noted on continuous cardiac telemetry monitoring during her hospitalization and on six months of follow-up.

Conclusion: Fatal cardiac arrhythmias, can occur in patients with acute thalamic infarct even beyond 24 hours in the presence of other confounding factors despite the absence of cardiac pathology. This case showed the association of heightened autonomic imbalance caused by an acute stroke, decreased cerebral flow, and fatal arrhythmia. This elucidates the importance of cardiac monitoring in acute ischemic stroke. With the paucity of information on serious cardiac arrhythmia and ischemic stroke, a future study on this correlation will be useful.

Keywords: Cerebrogenic cardiac arrhythmia, post-stroke arrhythmia, acute ischemic stroke

Introduction

Serious cardiac arrhythmia occurring after an acute stroke is an under-recognized threat. These may include atrial fibrillation, ventricular tachyarrhythmia, supraventricular tachycardia, and atrioventricular blocks. Fatal cardiac arrhythmias such as ventricular fibrillation may occur but are rarely documented. Cardiac arrhythmias are noted to be more common in the first 24 hours after an acute ischemic stroke and would show a decreasing trend after 72 hours.[1] Arrhythmias may still be seen within three weeks post stroke.[1,2] These are commonly seen in lesions at the right side of the cerebrum particularly in the insular cortex, frontal, parietal cortex, amygdala, basal ganglia and thalamus.[3,4] The mechanism of arrhythmia that is proposed in several studies is the increase in catecholamines brought about by the disruption in the balance of the autonomic nervous system (ANS). The sympathetic nerve terminals that are connected to the heart's subendocardium release catecholamines directly into the heart and this affect the cardiac conduction system and cause the arrhythmia.[5]

A congenitally absent left internal carotid artery is rare and only occurs in less than 0.01% of the population. They are often asymptomatic due to a well-compensated collateral circulation. Symptoms only occur when atherosclerotic disease sets in or in conditions causing vascular blood flow disruption. Often times, the symptoms are neurogenic like a transient ischemic attack but there was no documentation of sudden cardiac death nor cardiac arrhythmia.[5,6]

We are presenting a case of a possible cerebrogenic fatal cardiac arrhythmia in a patient with an acute right thalamic infarct. This case validates the importance of cardiac monitoring in patients with acute stroke.

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Case

A 39-year-old female was brought in the emergency room (ER) after surviving a sudden cardiac death from ventricular fibrillation. Seven days prior to admission, she developed persistent left-sided mild hemiparesis and hemisensory loss on the face, arm, and leg. Forty minutes prior to admission, the patient had sudden loss of consciousness preceded by an emotional outburst. As related by the relatives, the emergency medical services (EMS) was called and they documented her to be unresponsive and with ventricular fibrillation (Vfib) on their monitor. Chest compressions were done followed by defibrillation which resulted to a return of spontaneous circulation after three to four minutes of resuscitation. The post arrest ECG according to the EMS responders was then normal sinus rhythm and she was brought to the ER.

The patient had a history of episodic syncopal attacks since her teenage years, this was investigated extensively two years prior to admission and was determined to occur due to a brain blood circulation etiology. Tests revealed a congenitally absent left internal carotid artery that was well compensated by collateral blood flow from the anterior and posterior communicating artery. However, Transcranial Doppler revealed that hyperventilation maneuver (causing cerebral artery vasoconstriction) dropped cerebral blood flow significantly to 43% and caused dizziness that she experiences prior to subsequent syncopal attacks. Brain MRI revealed an old lacunar infarct in the right internal capsule where she was asymptomatic. Extensive work up for stroke in the young yielded unremarkable findings. Examinations to rule-out a hypercoagulable syndrome also were unremarkable. She has no relatives who had a sudden cardiac death nor any familial heart disease that can cause syncope.

At the ER, she was drowsy but oriented to three spheres. Vital signs then showed, heart rate of 106/min with regular rhythm, blood pressure 120/70mmHg, respiratory rate 19 breaths per min and temperature of 36.8°C. She had an absent left carotid pulse but had a strong right carotid pulse. Cardiovascular and pulmonary exams were normal. Pertinent neurologic exam showed left sided hemiparesis with a shallow left nasolabial fold and motor strength of 3/5 on the left upper and lower extremities, and an 80% intact sensation on both the left upper and lower extremities. The right upper and lower extremities had motor strength of 5/5 with 100% intact sensory perception.

Cardiac diagnostics done to detect a structural abnormality were all unremarkable. Brain natriuretic peptide (BNP), D-Dimer, Troponin-I, CK-MB, Sodium, Potassium, Magnesium, Calcium, Creatinine, thyroid function tests and lipid profile were all normal. ECG done on admission showed sinus tachycardia (Figure 3). Chest radiograph was

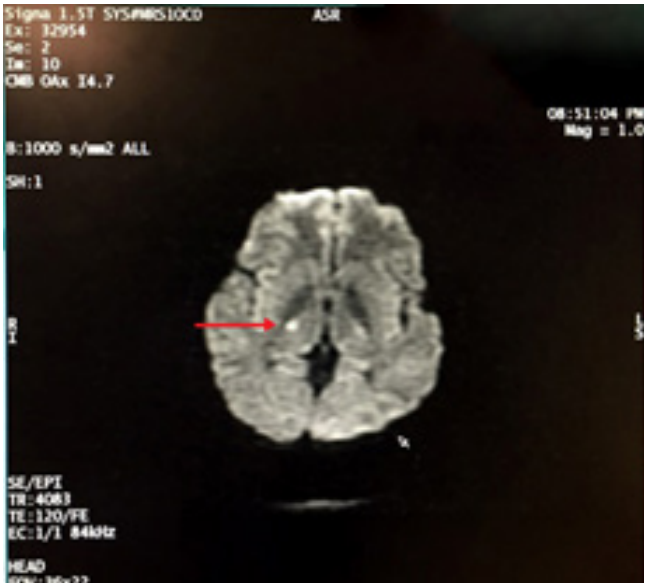

Figure 1. Plain Brain MRI (DWI) of patient case with Acute Right Thalamic Infarct (red arrow)

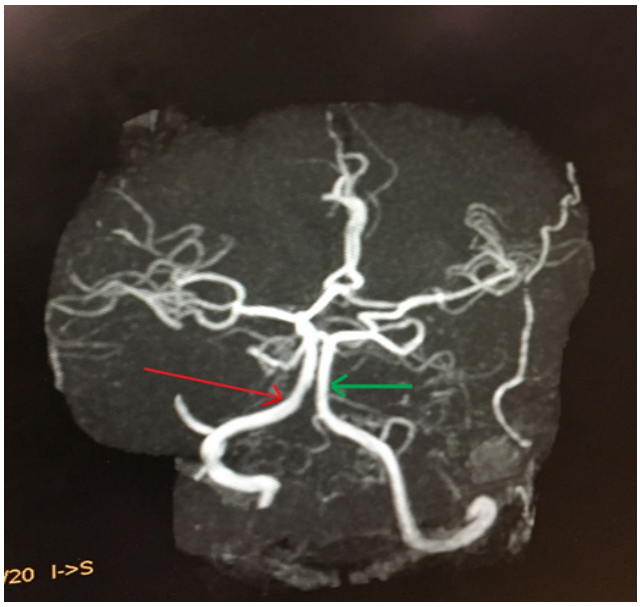

Figure 2. MRA of patient case with congenitally absent left internal carotid artery. Right internal carotid artery (red arrow) has normal course and caliber; basilar artery (green arrow) has normal course and caliber.

unremarkable. Echocardiogram showed an ejection fraction of 73.2% with normal size and adequate contractility. CT Angiography revealed unremarkable coronary arteries, no aortic aneurysm and normal pulmonary vessels.

Cranial MRI (Figure 1) revealed an acute infarct over the right thalamus and the MRA (Figure 2) showed an absent left internal carotid artery, with normal caliber of the right internal carotid artery and its branches. These findings were also noted in the Carotid Duplex scan. Electroencephalogram

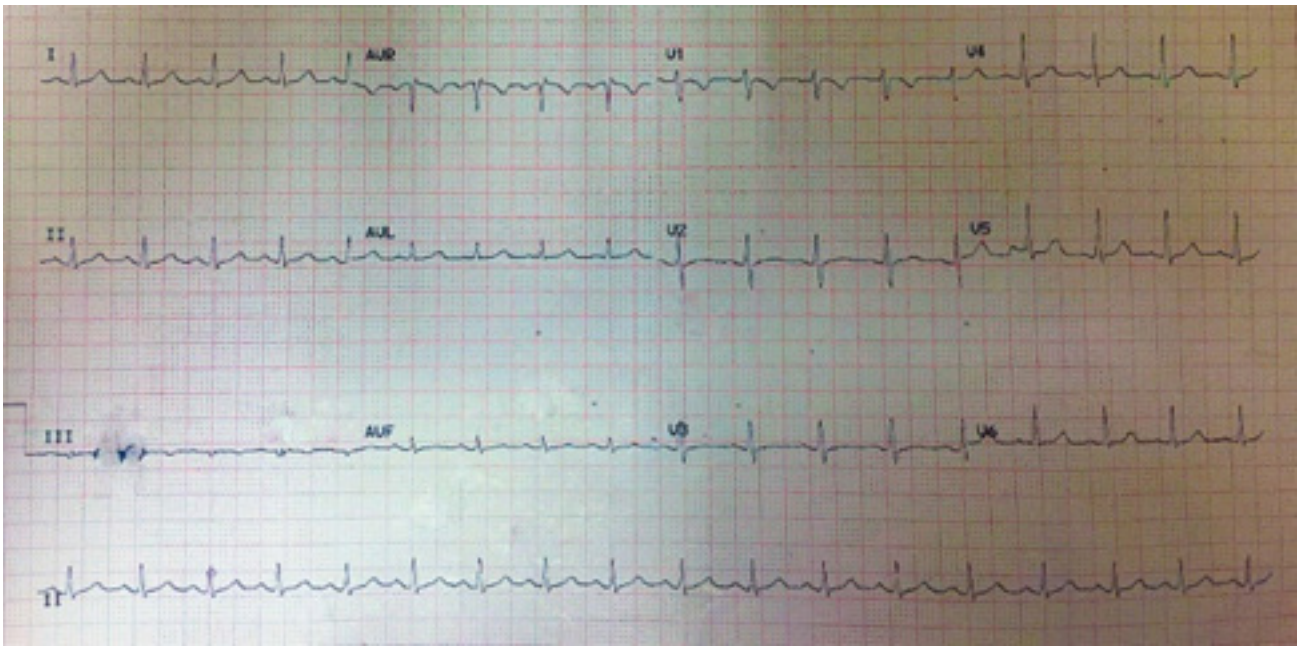

Figure 3. 12-Lead EKG of patient case at ER post Cardioversion (Sinus Tachycardia, Normal Axis)

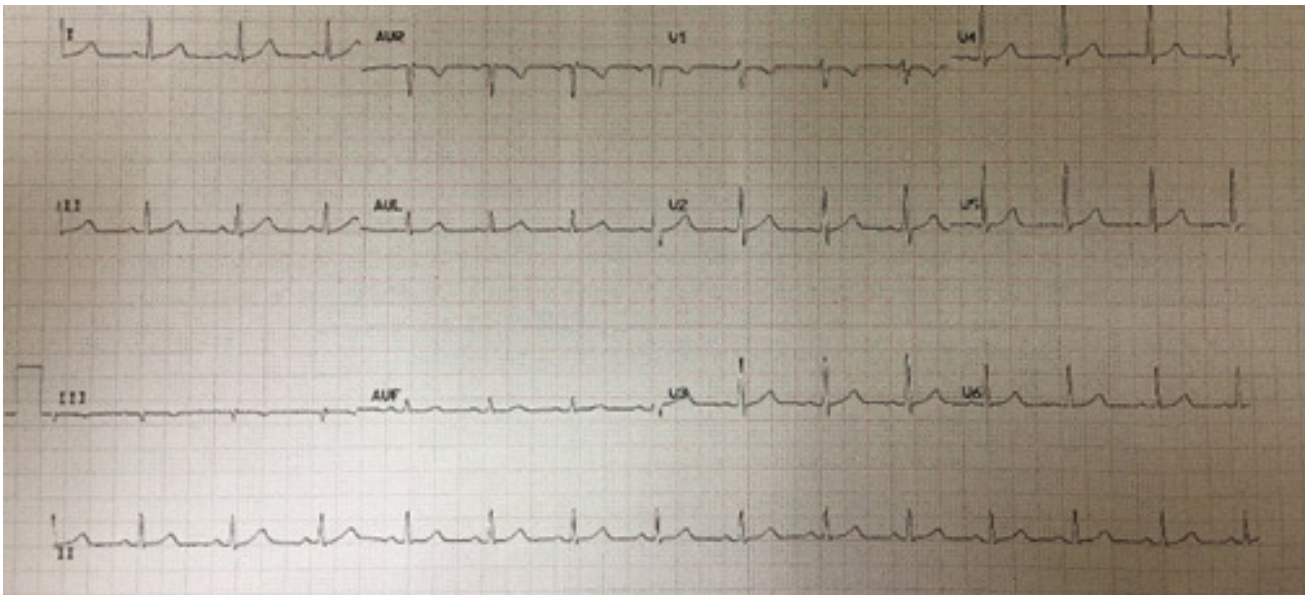

Figure 4. 12-Lead EKG of patient case seven days post Ventricular Fibrillation Normal Sinus Rhythm, Normal Axis)

was normal. Investigation for cause of stroke in the young with a hypercoagulability panel (anticardiolipin antibody, antithrombin III, factor V leiden gene mutation, factor VIII activity, homocysteine, protein C activity, protein S activity, prothrombin gene mutation and aPTT-LA) was normal.

She was treated with bisoprolol 2.5mg tablet once a day and clopidogrel 75mg tablet once a day. Continuous cardiac telemetry monitoring did not record occurrence of cardiac arrhythmia. The 12-Lead ECG taken seven days post ventricular fibrillation (Figure 4) shows normal sinus rhythm with normal axis. Despite the unremarkable course in the hospital, the patient and family decided to have an implantation of an AICD. The patient was discharged and maintained on bisoprolol and clopidogrel. Six months post discharge, review of the AICD record did not show recurrence of any cardiac arrhythmia and the patient remained asymptomatic.

Discussion

There is correlation between acute ischemic stroke and cardiac arrhythmia. However, the paucity of cases precludes predicting patients who will develop serious cardiac arrhythmia.

Studies have shown that most fatal cardiac arrhythmias in those with acute ischemic stroke are seen in those with inherent structural heart disease[1] which our patient did not have. Fatal cardiac arrhythmia in those with acute ischemic stroke were also noted to be associated with myocardial ischemia, ventricular dysfunction, and electrolyte abnormalities. In our patient, test results were all unremarkable negating a possible cardiac and electrolyte cause of the ventricular fibrillation. The question then arises, what caused the ventricular fibrillation in the absence of a cardiac pathology?

The interplay between the congenitally absent ICA, hyperventilation from an emotional outburst, and acute thalamic infarct may therefore be the key contributors to the patient's fatal cardiac arrhythmia. Despite the documentation of a good collateral circulation, it is a possibility that intimal changes within the vessels of both the anterior and posterior circulation maybe present. These changes coupled with vasoconstriction from hyperventilation can significantly decrease cerebral blood flow and predisposed this patient to arrhythmia from catecholamine surge.[8,9] The acute ischemic stroke in the right thalamus may have also been a result of the diminished cerebral flow despite this area being supplied by four arterial systems.[10] Maria del Mar Salez de Ocariz, et al noted in their study that thalamic stroke commonly occurs in young adults and that its mechanism is often undetermined of which majority are ischemic in nature. Smoking was found to be the main risk factor associated with thalamic infarct, however, in isolated thalamic infarct, single lesion as in our case, most of the patients have no predominant risk factors.[10]

Among the proposed pathophysiology of arrhythmogenesis after stroke, autonomic dysfunction is the most probable for our patient. Despite paucity of data on fatal arrhythmia in thalamic infarcts, the effect of excess catecholamine caused by decrease cerebral flow and infarct can account for the cerebrogenic fatal arrhythmia.

Conclusion

Fatal cardiac arrhythmias can occur in patients with acute thalamic infarct even beyond 24 hours, especially in the presence of other confounding factors despite the absence of cardiac pathology. This paper presented an unusual case of a patient with a congenitally absent left ICA with an acute ischemic stroke at the right thalamus who survived a sudden cardiac death from ventricular fibrillation occurring within seven days from the onset of her neurologic symptoms. The proposed association of fatal arrhythmia and acute thalamic infarct was shown to be possible in our patient. The congenitally absent left ICA and hyperventilation further contributed to arrhythmogenesis because of the resultant diminished cerebral blood flow. It is very important to advise the patient to avoid activities that can trigger hyperventilation such as strenuous physical and heightened emotional stressors. This paper validates the need for cardiac monitoring in patients with acute ischemic stroke. We recommend future studies in acute ischemic stroke patients to look into the frequency of occurrence of serious and fatal cardiac arrhythmias and to correlate it with the location of the lesion.

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