



A Case Report of Heart Block due to Intracranial Hemorrhage

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Abstract

Brain and heart have many connections both in disease and physiology. Most of the literature refers to neurological complication of underlying cardiac disease. In this case report we present a cardiac complication due to acute stroke, which is rather frequent in clinical practice. Here we present a patient with second degree heart block followed by neurological deficit, due to acute intracranial hemorrhage. The heart block regressed along with neurological stabilization a few days later.

Introduction

Acute stroke has been associated with a variety of medical complications including cardiac abnormalities like myocardial infarction, stress cardiomyopathy and arrhythmias. Cardiac arrhythmias in acute stroke leads to hemodynamic instability causing cerebral hypoperfusion to critical areas, which has a negative impact on short term prognosis. It also increases risk of sudden cardiac death and recurrent thromboembolism particularly in patients with intracranial hemorrhage. The greatest risk of arrhythmias is within first 24hrs of stroke and with marked decline in time.

Case Report

A 24 year old female, gave birth to a male baby by normal vaginal delivery with episiotomy. She had an uneventful antenatal history. Few hours after delivery patient complained of headache with palpitation and sweating. Examination vitals

were stable with systemic examination normal. An hour later her heart rate was around 45 per minute, ECG showing second degree heart block. 10 minutes later patient complained of left sided sensory loss from head to toe. Examination showed loss of pain, touch and vibration sensation over the left side of body, with 0/5 power on the left side, with exaggerated deep tendon reflexes and extensor plantar. CT Brain showed hemorrhage in the right gangliocapsular and thalamic region of size 3x3 cm. Her complete haemogram, renal function tests and liver function tests were normal. PT, INR & aPTT were normal and test for APLA syndrome & ANA were negative.

MRI Brain taken on the next day showed hyperintensity involving the right side midbrain, pons, thalamus, basal ganglia, internal capsule and insular cortex suggestive of acute Intracranial Hemorrhage with evidence of midline shift. She was treated with Anti Edema drugs, anti seizure

drugs and supportive medication.

Echocardiography was normal. Patient was put on continuous cardiac monitoring and inj. Atropine 0.6 mg slow IV given if heart rate was 40 beats. Patient cardiac status improved and returned to normal sinus rhythm a day later. Her neurological deficit gradually improved in the next four weeks with no further episodes of cardiac arrhythmias. At the time of discharge sensory loss over left side has improved and her motor power was 4/5 on left side.

Discussion

The incidence and type of cardiac complications in acute stroke depends on the nature of neurological events (SAH or intracerebral hemorrhage) and in large vessel ischemic infarction. Acute stroke causing cardiac complication are due to 1) direct effect on autonomic system and the subsequent neurohormonal factors; 2) a direct result of stroke like atrial fibrillation and 3) coincidental circumstances.

Cardiac arrhythmias are frequently observed within first 24hrs of acute stroke, even in the absence of structural heart disease. An increase in the sympathetic activity has been proposed as a causative factor in the genesis of cardiac abnormalities following acute stroke⁴. ECG changes (ST and T wave changes) and arrhythmia is commonly seen in SAH and stroke involving right hemisphere, which leads to disinhibition of the insular cortex and increase of sympathetic tone⁵.

The insula is one of the most important areas of cortex, involved in the autonomic function control. It has interconnection with thalamus, limbic system, and other cerebral areas associated with sympathetic function. Involvement of left insular decreases basal sympathetic tone and result in parasympathetic hyperactivity, right insular lesions decreases parasympathetic activity and augment cardiac sympathetic tone. In our case, brad -arrhythmia has occurred even before the manifestation of neurological deficit. An

increased in sympathetic activity due to insular cortex involvement is the causative factor for cardiac arrhythmia in stroke patient, is evident in this case also.

The increase in sympathetic tone causes increase in serum catecholamine, leading to myocardial changes in the form of myocytolysis. Autopsy of stroke patient revealed petechial subendocardial hemorrhage and focal myofibrillar degeneration, which was reproducible with intravenous administration of catecholamines⁶.

Structural brain damage (Right insular cortex & ipsilateral amygdale) leads to central autonomic stimulation Local catecholamine excess calcium channel activation leading to increased cytosolic and intramitochondrial calcium level with free radical release. This results in peroxidation of membrane lipids with ECG changes and arrhythmia.

A recent study shows the involvement of insular cortex in cerebral hemorrhage or infarct was associated with high risk of cardiac arrhythmias and sudden cardiac death⁵. Evaluation and follow up of 493 patients by MRI concluded that acute right insular cortex involvement was associated with increased vascular mortality.

From this case, we conclude that the cardiac abnormalities seen in stroke patients may actually be caused by the cerebral event and do not have underlying cardiac disease. All patients of acute CVA needs continuous cardiac monitoring for initial 24hrs. High degree of suspicion of acute CVA is needed while ruling out the causes of cardiac arrhythmias.

Reference

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