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Challenges of Management of Stunned Myocardium Associated with Acute Subarachnoid Hemorrhage: A Case Report

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Abstract

A 40 year old lady presented with headache and vomiting having no past history of hyper-tension, diabetes, smoking, alcohol or drug abuse. Computed Tomography (CT) scan of brain revealed sub-arachnoid haemorrhage in parasaggital frontal and lt. Sylvian fissure. Digital Subtraction Angiogram (DSA) was performed and revealed a small aneurysm (4x 2.5 x 2) mm in anterior communicating artery. After 12 hours of DSA patient complaints of sudden severe headache followed by unconsciousness. Repeat CT performed and revealed new onset rt. fronto-basal intra-cranial hematoma consistent with rebleed. On admission the patient was with normal Glasgow Coma Scale (GCS-15), Blood pressure (BP125/80mm/Hg), ECG & Echocardiogram. After rebleed patient developed low BP (50/35mmHg), GCS down gread (05), ECG showed sinus tachycardia with poor progression of R(V1-V3) wave, elevated cardiac Troponin –I(-4919.6 Pg). Then patient was given a regimen of ionotropic agent noradrenalin at dose 5mcg/kg/h. From day 3th of rebleed the patient was clinically improving, BP(120/84mmHg), GCS(8), ECG normal, Cardiac Troponin-I- (790.8 Pg/dl). Then emergency endovascular ACOM coil embolization was done. *[Journal of National Institute of Neurosciences Bangladesh, 2018;4(2): 150-153]*

Keywords: Sub-arachnoid hemorrhage; Stunned myocardium; aneurysm; coil embolization

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Contribution to Authors: SSI, KMR, SUD & DME were involved from the diagnosis of the patient upto management of the patient in the cath lab. AH had performed the general anesthesia. UKM has involved in the pre- and post-management of coiling in ICU.

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Introduction

Cardiac complications are not uncommon after aneurysmal subarachnoid hemorrhage. Electro-cardiographic (ECG) changes occur in 50% to 100% of such cases[1]. The ECG changes associated with (SAH) are prolongation of QT interval, depression or elevation of ST segments and T wave inversion are the commonly encountered. Left ventricular dysfunction and frank myocardial infarction are also infrequently observed. The reversible cardiac dysfunction associated with subarachnoid hemorrhage may be due to the 'neurogenic stunned myocardium'. Frequent symptomatic vasospasm also occurs, possibly related to poor cardiac output and the inability to optimize triple-H (hypertnsion, hypervolemia, hemodilution) therapy. Reduced cardiac output in severely affected patients might increase the risk of delayed cerebral ischemia from vasospasm. However, differentiation of myocardial infarction from neurogenic stunned myocardium and successful aggressive endovascular

management of aneurysm can prevent future risk of haemorrhage and lower morbidity in comparison to surgical approach.

Case Presentation

A 40 years old house wife admitted with severe headache and vomiting (Hunt and Hess grade II- SAH) with no past history of hypertension, diabetes, smoking, alcohol drug abuse. Computed tomography scan (CT-scan) of brain revealed thin sub-arachnoid haemorrhage (Fisher grade-II) in para-sagittal frontal and left Sylvian fissure (Figure A). Digital Subtraction Angiogram (DSA) of brain was performed on 20th day of SAH with full aseptic precaution and revealed a small aneurysm measured 4mm length x 2.5mm breadth x 2mm neck in anterior communicating artery (Figure B).

After 12 hours of DSA the patient experienced sudden onset of severe headache followed by unconsciousness. Repeat CT-scan was performed and revealed new onset right frontobasal intracranial hematoma (Figure C) suggestive of re-bleed. On admission, patient's Glasgow Coma Scale (GCS) was normal (15). Blood pressure was normal (125/80 mmHg). ECG and Echocardiogram were normal. After rebleed, patient developed low blood pressure (50/35 mmHg) and low GCS (5). ECG showed sinus tachycardia with poor progression of R wave in V1, V2, V3 with elevated Cardiac troponin–I (4919.6 pg/dL). Echocardiography was not performed. Patient was shifted in intensive care unit and was given a regimen of inotropic agent noradrenaline dose (5mcg/kg/h) by infusion pump. The clinical status of the patient was improving with intensive care management. On day 3rd from rebleed patient's GCS was eight with normal BP (110/80 mmHg), ECG; however, the cardiac Troponin-I reduced (790.8 pg/dL). A board of neuro-interventionist, Cardiologist and neurosurgeon had finally recommended for emergency endovascular coil embolization. TransendEX 0.014inch micro-guidewire and placed at the neck of the aneurysm. Two detachable coils (3mmx4cm & 2mmx3cm, Target 360 ULTRA, inc Boston scientific Corp, USA) were deployed and the aneurysm was completely packed (Figure D). On the day 6th of rebleed the patient developed mild (muscle power 4/5) weakness in left side of the body. TCD was performed and revealed high mean systolic velocity in right anterior cerebral artery (>97 cm/s) and in right middle cerebral artery (>100 cm/s), suggestive of vasospasm. Injection Nimodipin 2mg/hour by infusion pump was given for 3 hours for the management of vasospasm,

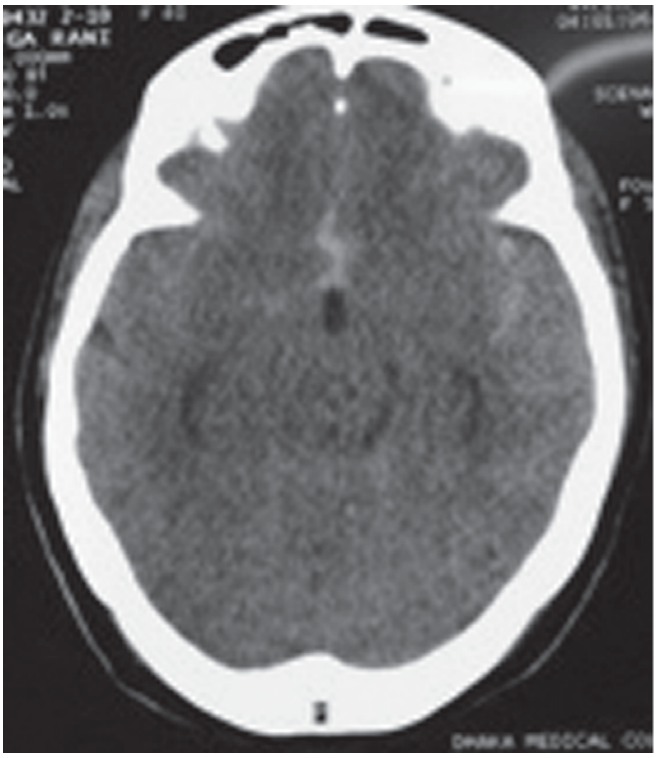

Figure (A): CT scan showed hemorrhage in frontal inter-hemispheric and both Sylvian fissure

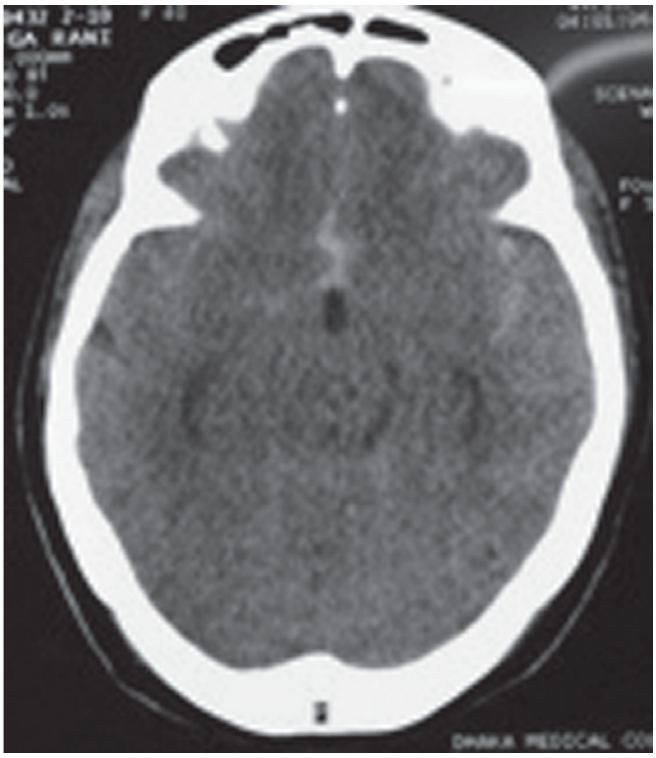

Figure (C): Right fronto-basal intra-cranial hematoma

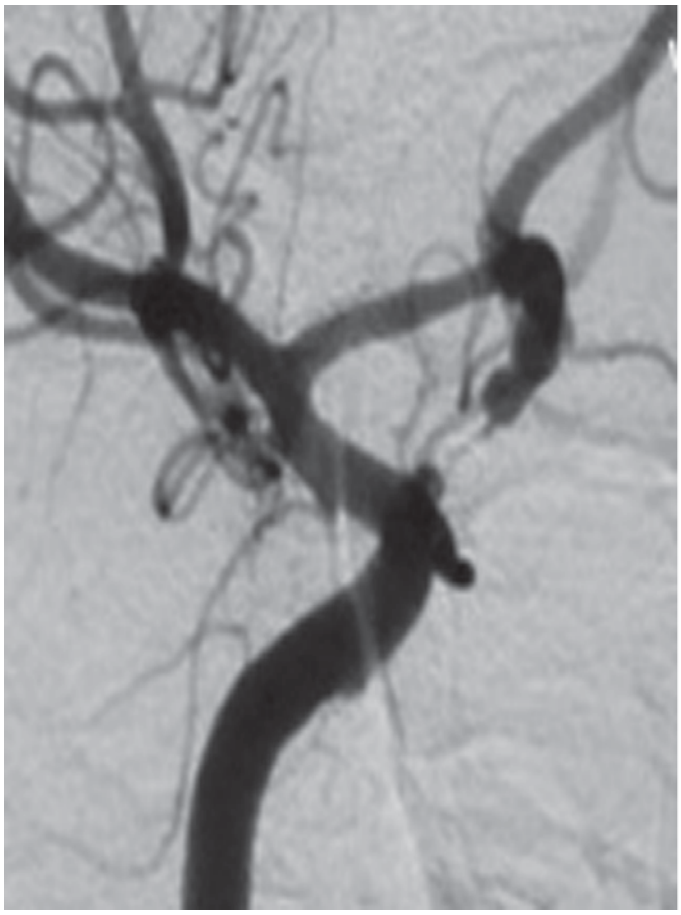

Figure (B) : Anterior Communicating artery aneurysm

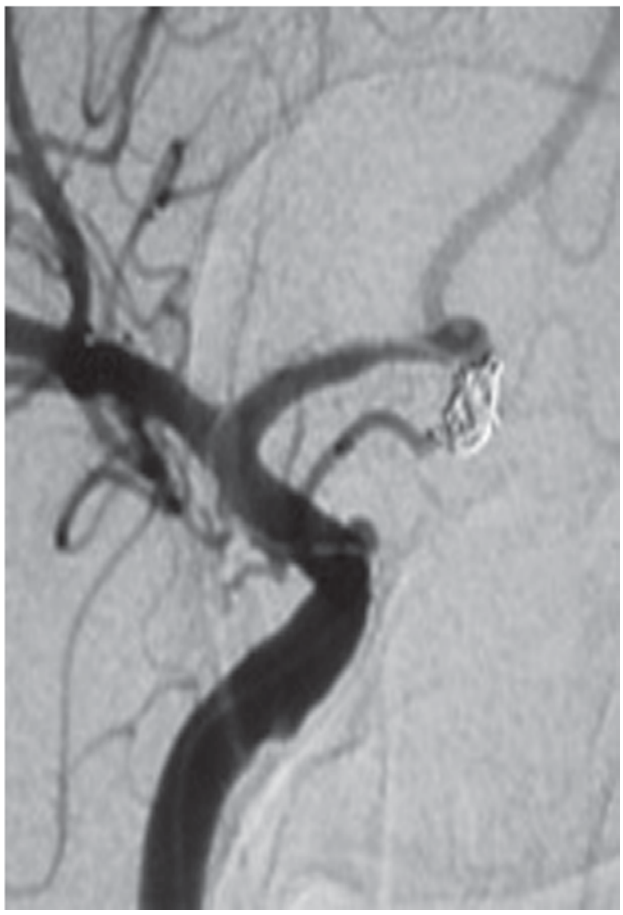

Figure (D): Aneurysm was embolized

instead of triple- H (hypertension, by infusion pump, for the management of vasospasm was given for 3 hours, instead of triple-H (hypertension, hypervolumea, hemodilution) therapy to avoid cardiac fluid over load. Post coiling echocardiogram was normal. The patient was discharged home and was doing well with good conversation.

Discussion

It is thought that the cardiac changes that occur in association with SAH are the occurred from increased central sympathetic activity, which typically results in a hyper dynamic cardiovascular state[1]. Small myocardial enzyme elevations reflect relative cardiac decompensation and failure of the left ventricle to meet these inotropic demands. Massive peripheral vasoconstriction may also further aggravate the left ventricular decompensation by increasing the cardiac afterload[2]. Transient increase in sympathetic nervous activity induces myocardial damage, referred to as neurogenic stunned myocardium, which is hypothesized to be caused by oxygen-derived free radicals or transient calcium overload. Both free radical and calcium overload is associated with decreased responsiveness of contractile filaments to calcium, which is secondary to selective troponin-I proteolysis[3]. Neurogenic stunned myocardium associated with SAH is characterized by a transient nature like post ischemic dysfunction is fully reversible), the dysfunction's not being caused by a primary defect in myocardial perfusion like normal coronary arteries, this probably causes the myocardial oxygen demands to exceed oxygen supply. Neurogenic stunned myocardium associated with SAH is characterized by a transient nature (ie, post ischemic dysfunction is fully reversible), the dysfunction's not being caused by a primary defect in myocardial perfusion (ie, normal coronary arteries), this probably causes the myocardial oxygen demands to exceed oxygen supply like neurogenic stunned myocardium from that caused by coronary artery disease in patients with SAH[4].

Elevation of cardiac enzymes, particularly cardiac troponin-I, has been found to be a highly sensitive and specific indicator of myocardial dysfunction in cases of

aneurysmal SAH[5]. In the present case, cardiac troponin-I level at the time of initial presentation was high (4919.6 pg/dL) and returned to normal (<1.9 Pg/dl) as the cardiac status improved. However, in some cases, an invasive procedure such as coronary angiography may become necessary to rule out myocardial dysfunction due to coronary artery disease. The ECG abnormalities have been attributed to increase circulating and local myocardial tissue catecholamine in conjunction with low myocardial intracellular potassium. Hypothalamic stimulation in animals can induce cardiac changes similar to those observed after SAH, and most patients dying as a result of SAH have been shown to have both hypothalamic and myocardial lesions at autopsy[6-7]. Impaired myocardial contractility related to contraction band necrosis occurs in animals after excessive cardiac sympathetic stimulation[8] and this reversible form of cardiac pathologic abnormality is found in 50% of patients with fatal SAH at autopsy[6,9-10]. Neurogenic stunned myocardium due to non-traumatic SAH is probably associated with more incidence of symptomatic vasospasm because of reduced cardiac output and hypotension due to impaired myocardial contractility.

The prevention and management of vasospasm is dependent on maintaining the fine balance of cerebral blood flow and perfusion pressure. This requires close monitoring of intracranial pressure and blood pressure. The patients are managed in intensive care units with continuous invasive hemodynamic monitoring and support. Calcium channel blockers have been considered to be useful in the management of patients before vasospasm occurs and in the management of patients with symptomatic vasospasm. Transcranial Doppler sonography is used frequently to monitor and detect vasospasm before the patient suffers ischemic neurologic deficit or infarct. Elevated Transcranial Doppler velocities often initiate the use of Triple-H therapy and subsequently guide it. Recently, perfusion CT has shown some promise in detecting early vasospasm and may become a valuable and easily accessible imaging technique, particularly for monitoring vasospasm.

However, impaired myocardial function due to stunning can significantly limit the effectiveness of Triple-H (hypervolumia, hyperdilusion, and hypertension) therapy because such therapy depends on optimal cardiac function. Continuous monitoring of central hemodynamic variables, such as pulmonary artery occlusion pressure and cardiac index, allow aggressive but watchful fluid management while monitoring the cardiac status. Positive inotropic agents and pressors have been used with significant success in these patients. In the present case, patient achieved good outcome related to vasospasm. This case highlights the need for a meticulous, multidisciplinary management strategy for each aneurysm, taking into consideration the urgency of treatment and suitability to different therapeutic techniques.

Conclusion

Reversible cardiac dysfunction associated with SAH may be due to neurogenic stunned myocardium. Associated high incidence and severity of delayed cerebral vasospasm is related to poor cardiac output and the inability to optimize hyperdynamic hypervolemic therapy. Most of these patients are not considered to be suitable surgical candidates and can be best treated with endovascular therapy. With aggressive management, patients can recover from these reversible cardiac complications that happened in our case.

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