Array Publishers
Case Report
Open Journal of Case Reports

Neurogenic Stunned Myocardium with Aneurysmal Subarachnod Hemorrhage Treated by Endovascular Coiling of ICA Aneurysm: A Case Report

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Received Date: 20 July, 2020; **Accepted Date:** 27 July, 2020; **Published Date:** 3 August, 2020

Abstract

Introduction: Stunned myocardium with reduced cardiac output in aneurysmal subarachnoid hemorrhage patients might increase the risk of delayed cerebral ischemia from vasospasm. Due to compromised cardiac status endovascular coiling may be preferred over surgical clipping.

Case presentation: 68 years old female was brought to Emergency department with altered sensorium. She was diagnosed with Fisher Grade -II SAH and right ICA aneurysm. She developed acute heart failure. Cardiac troponin level was 2.6 ng/ml. On ECG there was prolonged QT interval. Transthoracic echocardiogram showed global hyperkinesias with an ejection fraction of 20%. Patient underwent endovascular coiling of the aneurysm. The patient improved after 15 days of vigorous medical management with reversal of cardiac functions.

Conclusion: Our case highlights the importance of differentiating stunned myocardium from myocardial infarct. Endovascular management is ideal option in this setting. Excluded aneurysm will facilitate aggressive medical management to treat this reversible condition.

Keywords: Stunned myocardium; Aneurysmal subarachnoid hemorrhage; Endovascular coiling

Introduction

Cardiac abnormalities are frequently seen in aneurysmal subarachnoid hemorrhage with a significant impact on disease course. Neurogenic Stunned Myocardium (NSM) in aneurysmal Subarachnoid Hemorrhage (SAH) is associated with a wide spectrum of reversible left ventricular wall motion abnormalities [1,2]. This is case report of neurogenic stunned myocardium in acutely ruptured subarachnoid aneurysm. Role of early diagnosis of this condition and differentiating it from myocardial infarctions, early treatment of ruptured aneurysm to prevent rebleeding and aggressive medical management of pulmonary edema and Cerebral vasospasm will be discussed along with relevant review of literature.

Case presentation

68 years old female was brought to Emergency department with altered sensorium. On Clinical evaluation she had Left hemiplegia (motor power on right side 5/5 and Left side 2/5) with Glasgow Coma Scale (GCS) was 10/15(E3M3V4), her Hunt and Hess scale was 3. World Federation of Neurosurgical Societies (WFNS) grade was 4. She was diagnosed with Fisher grade –II SAH predominantly in suprasellar cistern and bilateral sylvian fissures on CT scan (Figure 1A). Her radiograph of Chest showed pulmonary edema in bilateral upper zones suggestive of neurogenic pulmonary edema (Figure 1B). Digital Subtraction Angiography (DSA) showed multilobed aneurysm of communicating segment right ICA (Figure 1C). She had tachycardia and tachypnea. BP was 100/70. Her saturation was dropping on mask ventilation. Hence she was intubated. On Electrocardiogram (ECG) there was prolonged QT interval and new T wave inversion seen. Cardiac troponin level was 2.6 ng/ml. transthoracic echocardiogram showed severe systolic dysfunction with an ejection fraction of 20 % with global hypokinasia. Team of Intensivists suggested aggressive medical management of cardiac dysfunction. Need to secure the aneurysm was discussed with interdisciplinary team and family members. It was decided to do endovascular coiling of the aneurysm. The Procedure was done in cathlab (Artis Zee, biplane o DSA, Siemens, Munich, Germany) under general anesthesia. Balloon assisted Coiling with complete exclusion of aneurysm from circulation with angiographic cure was achieved uneventfully (Figure 1D). No significant vasospasm seen. After the endovascular coiling general anesthesia was reversed however it was decide to keep patient intubated and ventilate with pressure support. Her blood pressure was maintained above 120/80 with inotropic support. She had episodes of clinical blooming and glooming for 15 days. Her cardiac function returned normal with gradual tapering of iontropic agents. Cardiac enzymes were reversed to normal levels. Echcardiography at this stage showed EF of 60%. Her motor power on left side improved to 4/5. She showed significant clinical improvement with her Modified Rankin Scale (MRS) was zero at the time of discharge. After the discharge she was lost to follow up.

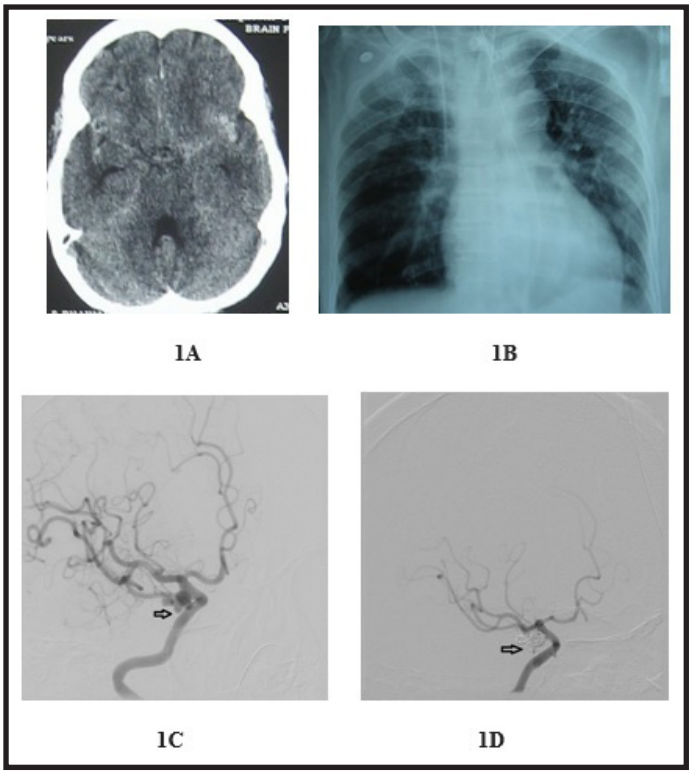

Figure 1. 1A. Showed diffuse SAH with extension into bilateral sylvian fissures. 1B. Showed radiograph of chest depicting pulmonary edema in predominantly upper zones, DSA. 1C. Showed multilobed aneurysm (Arrow) of communicating segment of right ICA DSA. 1D. Showed complete exclusion of the aneurysm from circulation by endovascular coiling (Arrow showed coil mass). Please note there is no vasospasm seen.

Discussion

Neurogenic Stunned Myocardium (NSM) is a devastating complication of Subarachnoid Hemorrhage (SAH). The most widely accepted mechanism in the pathogenesis of NSM is catecholamine-mediated direct myocardial injury. Proposed hypotheses include multivessel coronary vasospasm, abnormalities in coronary microvascular function, and catecholamine mediated cardiotoxicity. NSM shows global hypokinesia where as its variant called Takotsubo cardiomyopathy usually presents with a regional wall motion abnormality typically with left ventricular apical akinesia or apical ballooning. They both mimic myocardial infarction and have transient left ventricular hypokinesis. Due to their catecholamine-mediated natures, both are thought to have the same pathophysiology and follow the same course of illness. In terms of treatment, both are potentially reversible and can be managed by intensive medical care. Some believe that they are the same entity and that they should both fall under stress-induced cardiomyopathy. Apart from SAH, other neurogenic conditions like ischemic stroke, subdural hematoma, head injury and epilepsy are known to cause NSM and Takotsubo cardiomyopathy [3-5]. It is of prime importance to differentiate this from MI. Findings that favor diagnosis of NSM are (1) no history of heart disease, (2) new onset of cardiac dysfunction (ejection fraction < 40%), (3) wall motion abnormalities that do not correspond to ischemic ECG changes, and (4) troponin values less than 2.8 ng/ml in patients with EF < 40% [6,7].

Left Ventricular dysfunction after SAH increases the risk of cerebral infarction from vasospasm, hypotension, and pulmonary edema, but with aggressive ICU support it is possible to avoid adverse short-term survival or functional outcome. This condition can be cured by medical management alone. Medical management involves ACE inhibitors, Beta Blockers, Diuresis and Vasopressors. Judicious management of fluid with aim prevention of cardiac overload is advocated. Antihypertensive medication may confer cardio-protection and reduce the risk of catecholamine-mediated injury after SAH. Post-menopausal women are relatively more prone to develop neurogenic stress cardiomyopathy [8].

The insula plays major role in cardiovascular regulation, specifically in limbic-autonomic regulation. The insular cortex contains baroreceptive units of sympatho-excitatory and sympatho-inhibitory neurons that regulate blood pressure and heart rate. Stimulation or injury of the insula gives rise to changes in these cardiovascular parameters. Lesions in this part of the brain have been found to result in an increase in norepinephrine levels in the blood which cause left ventricular dysfunction. Stimulation of the right insula elicited sympathetic effects like tachycardia and hypertension, while stimulation of the left insula resulted in parasympathetic responses such as bradycardia and vasodepressor effects [9]. Genetic polymorphisms of the adrenoceptors are associated with an increased risk of cardiac abnormalities after SAH. Cardiac dysfunction after SAH is a form of epigenetic mediated neurocardiogenic injury [10].

Subarachnoid Hemorrhage (SAH) is a devastating form of hemorrhagic stroke. It carries high mortality rate, with 12% of patients dying before reaching the hospital. Many of those who reach hospital are in bad clinical grade. Rebleeding rates is 1% per day up to 21 days. Mortality increases by 5 times after rebleeding. Neurogenic Stunned Myocardium may lead to adverse outcome. Hence earlier the treatment of aneurysm better is the outcome. There is an absolute risk reduction of 7·4% in endovascular coiling than surgical clipping as per ISAT trial [11]. Hence we decided to treat the aneurysm with endovascular coiling so that cardiac abnormalities can be managed aggressively. Endovascular management of vasospasm with intra-arterial nimodipin infusion should be carefully carried out as it can lead to hypotension further complicating the already compromised cardiac and neuroal tissues. Titration with ionotropes along with intravenous volume expanders is advisable while treating the vasospasm.

It is considered highly important for Endovascular interventional radiologists, neurosurgeons and intensivists to be familiar with this clinical entity, since this transient cardiac function disturbance can be diagnosed at the initial presentation with unique wall motion abnormalities on echocardiography [12]. Early diagnosis of this condition, early treatment of ruptured aneurysm preferably with endovascular coiling, meticulous medical management of cardiac dysfunction and masterly patience results in better patient outcome and short-term survival. This case report gives insight into diligent management of this condition. However further large prospective studies will throw more light on how to improve clinical outcome in patients presented with NSM associated with aneurysm SAH.

Conclusion

Our case highlights the importance of early diagnosis and differentiating neurogenic stunned myocardium from myocardial infarction associated with aneurysmal subarachnoid hemorrhage. Endovascular management is ideal option for treatment of aneurysm in this setting. Excluded aneurysm will facilitate aggressive medical management to treat this reversible but potentially devastating condition.

Consent

Written informed consent could not be obtained. All reasonable attempts to obtain consent from the patient were made but the patient stays in remote village who after discharge lost to follow up. Every effort to preserve the patient's anonymity was made. There is no reason to believe that the patient would object to publication.

Conflict of Interest

All authors do not have any conflict of interest to be declared.

Acknowledgement

We would to like to sincerely thankful to all the residents doctors of the respective department for the helping us in retrieving patient's clinical data and images.

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