Stress-induced cardiomyopathy in aneurysmal subarachnoid hemorrhage

Tijana Nastasović1, Branko Milaković1,2, Mila Stošić1, Miloš Kaluđerović1, Olga Petrović3, Dimitra Kalimanovska Oštirić2, Danica Grujičić2,4
1Center for Anesthesiology and Reanimatology, Clinical Center of Serbia, Belgrade
2Medical Faculty, University in Belgrade
3Clinic for Cardiology, Clinical Center of Serbia, Belgrade
4Clinic for Neurosurgery, Clinical Center of Serbia, Belgrade

INTRODUCTION

The prevalence of neurogenic stunned myocardium in patients with SAH has varied between 10 and 28%1. Stress-induced cardiomyopathy (takotsubo cardiomyopathy, TCM) is a form of neurogenic stunned myocardium which is not common in these patients. The pathophysiology of TCM after SAH is uncertain, but catecholamine release is thought to be the underlying cause in the most cases2,3. We describe a case report of TCM after aneurysmal SAH.

CASE REPORT

A previously healthy 48-year-old female had progressive loss of consciousness. She was transferred to the emergency department of university hospital. On admission, her Glasgow Coma Score (GCS) was 7, with spontaneously respirations and narrow, symmetric and reactive pupils to light. She was immediately endotracheally intubated and was taken to the head computed tomography (CT) scan. The head CT scan showed diffuse SAH with blood in cisterna ambiens, the fourth and lateral ventricles, diffuse edema and hydrocephalus- Fisher grade 4. Her Hunt and Hess score was 4.

She was immediately brought to the operating room for external ventricular drainage (EVD). After intervention, the patient wasn’t awake and she was brought to intensive care unit (ICU) for mechanical ventilation (continuous positive airway pressure mode, FiO2 40%). The next day patient was awake, than she was weaned from mechanical ventilation and extubated. On control echocardiogram, the signs of apical ballooning syndrome resolved.

Conclusions: This case and review of the literature suggest stress-induced cardiomyopathy can mimic acute coronary syndrome after aneurysmal subarachnoid hemorrhage.

Key words: subarachnoid hemorrhage, stress-induced cardiomyopathy, takotsubo cardiomyopathy, left ventricle

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were done. Patient was extubated on the operating table, eupneic and with no neurological deficits.

Four days after SAH attack, patient became tachydyspneic, tachycardic (heart rate 110bpm) and hypertensive (BP 150-160/90-95mmHg). CVP was 12cmH2O. In arterial gas exchange analysis pO2 was 7.9kPa, pCO2 4.3kPa, pH 7.48, SaO2 92%, pO2/FiO2 169 (on the rebreathing mask, FiO2 35%). On chest auscultation bilateral rales were sounded. On suction through endotracheal tube bloody, foamy aspirate was obtained. She was immediately intubated and assisted mechanical ventilation (Bi level mode with FiO2 40%, PEEP 4cmH2O) as well as Midazolam infusion (0.5mg/kg/h) as well as as an effective intervention.

In chest radiography, bilateral pulmonary infiltrates were seen. NT-proBNP level was 5829pg/ml and troponin I was 0.59ng/ml. Transthoracic echocardiography in the ICU showed TCM- ballooning left ventricular (LV) apex and midventricle and hypercontractile basal segments (Figure 1). LV diameters in systole and diastole were normal, ejection fraction was about 50%. Loop diuretic was initiated as well as low molecular weight heparin - Nadroparin.

A repeated study 5 days after showed TCM in regression with better contractility of the apical segment of the LV and ejection fraction of 60%. NT-proBNP was 384pg/ml and troponin I was 0.059ng/ml. Pulmonary infiltrates also resolved, so the patient was extubated. After extubation, arterial blood gas analysis on room was normal.

Two days after, EVD of the patient was removed in the ICU. At the time of discharge, the 17th day after insult, she was conscious, eupneic, without neurological deficit.

**DISCUSSION**

We have described a case report with TCM after SAH. The pathophysiology of cardiac dysfunction after SAH is not always clear. The most widely accepted theory for SAH-induced neurogenic myocardial stunning is the “catecholamine hypothesis” This theory suggests that catecholamine-induced cardiac injury is the underlying cause of cardiac damage in patients with SAH. An explosive rise in intracranial pressure may cause sympathetic activation via hypothalamic damage. SAH animal studies have been in agreement with the clinical studies. Experimental SAH animal studies not only demonstrate immediate excess sympathetic nervous activation with higher circulating catecholamines concentrations, but the heart also appears to be more sensitive to sympathetic stimulation as well. Local norepinephrine production in the myocardium may surpass the systemic elevation of catecholamines and precipitate global or regional LV systolic dysfunction. TCM, also known as apical ballooning syndrome, is a form of neurogenic stunned myocardium, characterized by reversible LV regional wall motion abnormalities (RWMA) with a pattern of apical and midventricular a/hypo/dyskinesia and concomitant sparing of basal segments, ECG changes and release of myocardial enzymes that mimic myocardial infarction in the absence of obstructive coronary artery disease, attributed to a surge in catecholamine levels precipitated by an acute physical or emotional stressor.

To our knowledge, there are tree series of SAH patients with TCM and few case reports. The incidence of TCM in SAH is 0.8-6%. According to Guglin et al, literature reviews in 2011 showed that there were 61 cases of TCM in SAH from 1990.

Cardiac abnormalities can be seen with SAH. ECG changes are present in 50 to 100% of patients, and include deep T-wave inversion and QTc prolongation. Troponin elevation is seen in 20 to 40% of patients. Elevated troponin I level occurs more frequently in severe SAH, as measured by Hunt and Hess grade, and peak on the day of ictus with a decay thereafter. Elevated plasma BNP and NT-proBNP is significantly associated with RWMA, reduced ejection fraction, diastolic dysfunction, pulmonary edema, troponin I elevation, as well as early in-hospital mortality. Systolic dysfunction usually develops within the first 2 days after a neurologic event and then recovers. Overall, 10-28% of patients with SAH have global or regional LV systolic dysfunction.

There is no consensus about treatment of TCM. As the number of cases of TCMP increases, medications for its prevention continue to be investigated. In animal experiments, α - and β-blockade may be able to prevent TCMP.

In conclusion, our case report reminds us that cardiac dysfunction is fairly common after aneurismal subarachnoid hemorrhage. Currently, our prevailing practice is to measure cardiac biomarkers levels in all SAH patients and, so far, to reveal the patients with the risk of RWMA. Routine transthoracic echocardiography may be necessary these patients.

**SUMMARY**

**STRESOM-INDUKOVANA KARDIOMIOPATIJA KOD ANEURIZMATSKOG SUBARAHNOIDALNOG KRVA-
RENJA**

Uvod: Neurogeni ošamučeni miokard je čest posle aneurizmatskog subarachnoidalnog krvenja. Stresom-indukovana kardiomiopatija (takotubo kardiomiopatija) je oblik neurogenog ošamučenog miokarda koji se retko javlja posle subarachnoidalnog krvenja. U ovom radu prikazuju se slučaj stresom indukovan kardiomiopatije posle aneurizmatskog subarachnoidalnog krvenja

pet dana, pacijentkinja je dekonektovana sa mehaničke ventilacije, a potom i ekstubirana. Kontrolna ehokardiografija je pokazala znake bolje pokretljivosti apikalnih segmenata leve komore.

Zaključak: Ovaj prikaz slučaja i pregled literature pokazuju da stresom-indukovana kardiomiopatija može izgledati kao akutni koronarni sindrom posle aneurizmatskog subarhnoidealnog krvenjena.

Klučne reči: subarahnoidalno krvenjeno, stresom-indukovana kardiomiopatija, takotsubo kardiomiopatija, leva komora

REFERENCES


Abbreviations:
Subarachnoid hemorrhage (SAH), Takotsubo cardiomyopathy (TCM), Glasgow Coma Score (GCS), computed tomography (CT), external ventricular drainage (EVD), N-terminal pro-brain natriuretic peptide (NT-proBNP), intensive care unit (ICU), blood pressure (BP), central venous pressure (CVP), electrocardiography (ECG), regional wall motion abnormality (RWMA), left ventricle (LV), brain natriuretic peptide (BNP)