CASE REPORT

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Takotsubo cardiomyopathy in aneurysmal subarachnoid hemorrhage – a case report

Takotsubo kardiomiopatija kao posledica aneurizmatskog subarahnoidalnog krvarenja

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Abstract

Introduction. Subarachnoid haemorrhage (SAH) can be followed by cardiac abnormalities. We describe a patient with Takotsubo cardiomyopathy and neurogenic pulmonary edema (NPE) after aneurysmal SAH. **Case report.** A previously healthy, postmenopausal woman, suffered from aneurysmal SAH with consequent hydrocephalus. After external ventricular drainage, craniotomy and clipping of the posterior inferior cerebellar artery aneurysm, the patient developed acute heart failure and NPE. Transthoracic echocardiogram showed the left ventricular apical ballooning and hypercontractile basal segments. On chest radiography, bilateral pulmonary infiltrates were seen. Seventeen days after the SAH attack, the patient was discharged from hospital. Postponed coronary angiography revealed no signs of coronary artery disease. **Conclusion.** This case and review of the relevant literature suggest that Takotsubo cardiomyopathy and neurogenic pulmonary edema are not uncommon after aneurysmal SAH.

Key words:
coronary angiography; diagnosis; echocardiography; pulmonary edema; subarachnoid hemorrhage; takotsubo cardiomiopathy; ventricular function, left.

Apstrakt

Uvod. Subarahnoidalno krvarenje (SAH) može biti praćeno srčanim poremećajima. Prikazali smo bolesnicu kod koje se razvila slika Takotsubo kardiomiopatije i neurogenog plućnog edema, posle ataka SAH. **Prikaz bolesnika.** Prethodno zdravoj osobi ženskog pola, u postmenopauzalnom životnom dobu, dogodilo se akutno SAH je izazvano pucanjem aneutizmatskog proširenja intrakranijalnog, arterijskog krvnog suda praćeno razvojem hidrocefalusa. Posle izvođenja spoljašnje ventrikularne drenaže, kraniotomije i klipsovanja aneurizme na zadnjoj, donjoj malomoždanoj arteriji, kod bolesnice se razvila klinička slika akutne slabosti srčanog mišića i neurogenog edema pluća. Transtorakalni ehokardiogram ukazao je na naduvavanje vršnog dela leve srčane komore i hiperkontraktilnost njenih bazalnih segmenata. Na nativnoj radiografiji pluća viđeni su obostrani, oblačasti infiltrati plućnog parenhima. Posle 17 dana od ataka SAH bolesnica je otpuštana iz bolnice. Naknadna koronarna angiografija nije pokazala znake oboljenja koronarnih arterija. **Zaključak.** Na osnovu kliničke slike bolesnice i uvidom u referentnu literaturu, zaključujemo da se Takotsubo kardiomiopatija i neurogeni edem pluća mogu očekivati sa značajnom verovatnoćom kod bolesnika sa aneurizmatskom SAH.

Ključne reči:
angiografija koronarnih arterija; dijagnoza; ehokardiografija; pluća, edem; krvarenje, subarahnoidno; kardiomiopatija, takotsubo; srce, funkcija leve komore.

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Introduction

Many reports in recent relevant literature emphasize that subarachnoid hemorrhage (SAH) can be followed by cardiac abnormalities [1–7]. The prevalence of SAH-induced neurogenic stunned myocardium varies between 10% and 28% [8, 9]. ECG changes, serum cardiac necrosis markers and wall motion abnormalities have been supposed to be the most common.

Nonetheless, Takotsubo cardiomyopathy (TCM) was casually detected in these patients. The pathophysiology of TCM after SAH is uncertain, but catecholamine release is thought to be the underlying cause in most cases [10–13].

Neurogenic pulmonary edema (NPE) is a clinical syndrome characterized by the acute onset of pulmonary edema following a significant central nervous system insult. In the patients with SAH, reports of NPE incidence range from 2% to 42.9% [14–16].

We described a patient with TCM and NPE after aneurysmal SAH.

Case report

A previously healthy and normotensive 48-year-old female developed progressive loss of consciousness. She was firstly admitted to a regional hospital, with Glasgow Coma Score (GCS) 7 and Hunt & Hess grade 4, and was sedated with midazolam for endotracheal intubation.

Because of suspected cerebrovascular insult, she was transferred to the Emergency Department of University Hospital. On admission, she was unconscious, but sedated, endotracheally intubated, with spontaneous respirations and narrow, symmetric and light-reactive pupils. She had bilateral flexion on rough stimuli.

She was transferred to the computed axial tomography (CAT) scan cabinet. The head CAT scan showed diffuse SAH with blood in the fourth ventrides and ambient cistern, and lateral ventricles, as well as diffuse edema and hydrocephalus – Fisher grade 4 Figures 1A and B, respectively.

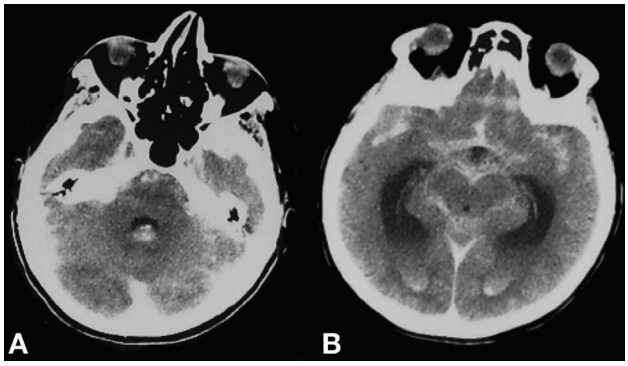

Fig. 1 – The head computed axial tomography scan shows: A) diffuse subarachnoid hemorrhage with blood in the fourth ventricles; B) ambient cistern and lateral ventricles with concomitant hydrocephalus.

She was immediately brought to the operating room for external ventricular drainage (EVD). Ventricular drainage was derived from the frontal horn of the right lateral ventricle. After intervention, the patient was still unconscious and was transferred to the intensive care unit (ICU) for the mechanical ventilation (CPAP, FiO2 40%).

In the ICU, the noninvasive blood pressure (NIBP) measurement was initiated and a central line was inserted for measuring the central venous pressure (CVP). She was hemodynamically stable with NIBP 120 – 130/ 80–85 mmHg, heart rate 72–100 beat per minute (bpm) and CVP 4 cm H_2O.

On the ECG monitor: negative T waves were seen and troponin I was elevated to 2.9 ng mL^{-1} (normal < 0.04 ng mL^{-1}), but the findings were considered as neurogenic stunned myocardium.

Creatine kinase was 146 IU/L and creatin kinase muscle and brain (CK – MB) isoenzyme was slightly elevated to 40 IU/L (normal range: 5–25 IU/L). Intravenous infusion of nimodipine was initiated as well as the routine antibiotic treatment. The patient also got carbamazepine, 2 x 200 mg, per sondam, and 2,500 mL of intravenous crystalloids.

The next day, the patient was awake, successfully disconnected from the mechanical ventilation and extubated. Two days after admission, the digital subtraction angiography of brain blood vessels (Figure 2) and multislice computed tomography (MSCT) angiography with 3-dimensional reconstruction of blood vessels (Figure 3) were done. The diagnosis of the aneurysm of the left posterior inferior cerebellar artery was confirmed.

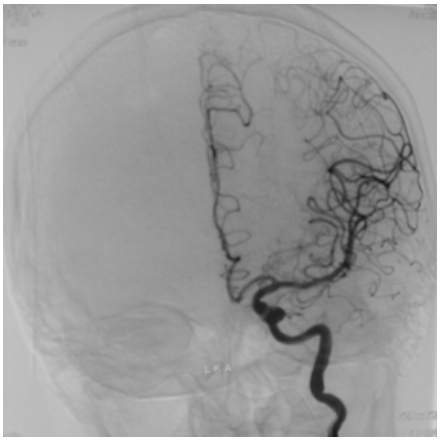

Fig. 2 –Digital subtraction angiography of brain blood vessels reveals an aneurysm of the left posterior inferior cerebellar artery.

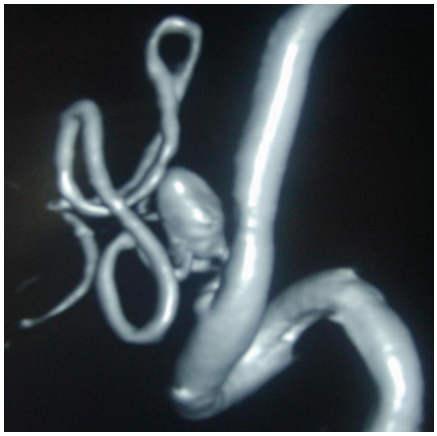

Fig. 3 – Multislice computed tomography angiography with 3-dimensional reconstruction cofirms the aneurysm of the left posterior inferior cerebellar artery.

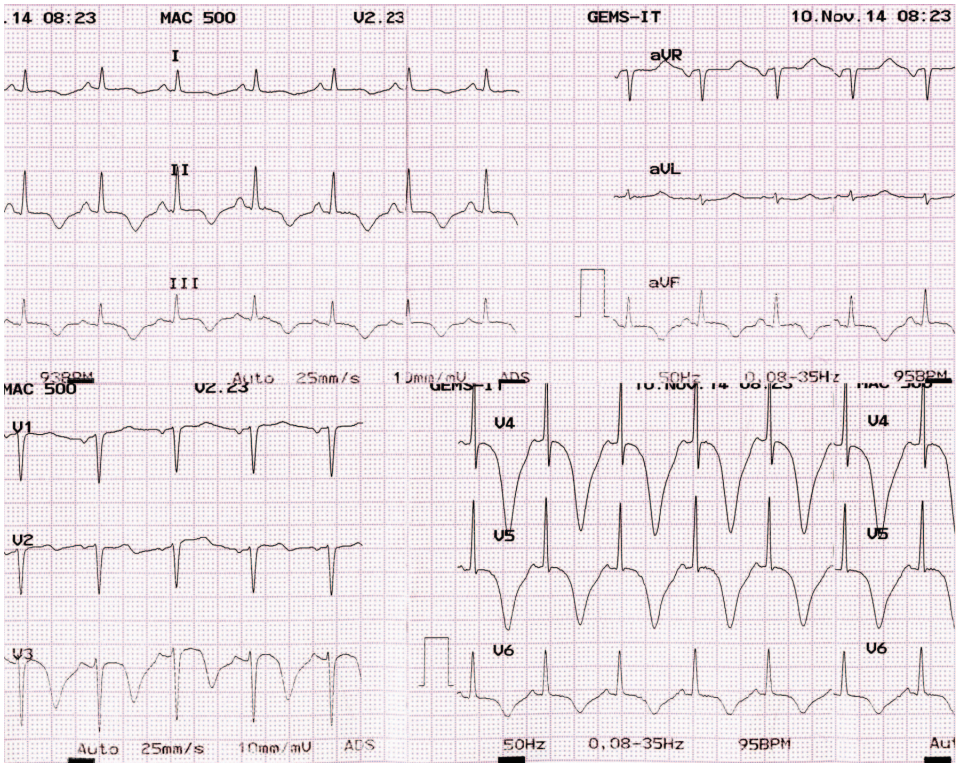

Fig. 4 – The 12-lead electrogardiogram recorded in the patient two days after aneurysmal subarachnoid hemorrhage attack showing deep and negative T-waves in D2, D3, AVF from V3 to V6 leads, as well as a prolonged QTc interval.

The patient was prepared for craniotomy. On chest radiography, the normal findings were described. Deep and negative T-waves were found in: D2, D3, AVF, from V3 to V6 ECG leads as well as prolonged QTc interval (Figure 4).

Troponin I level was lowered to 0.522 ng mL^{-1}. A cardiologist introduced bisoprolol to the therapy. After cardiologist consultation, craniotomy and clipping of aneurism were done, in the general endotracheal anesthesia. It was administered to the patient in a standard manner, and maintained with: remifentanil, sevoflurane and rocuronium in continuous infusion. The patient was extubated on the operating table, eupneic and with no neurological deficit.

Four days after the SAH attack, the patient became tachydyspneic, tachycardic (heart rate 110 bpm) and hypertensive (NIBP 150–160/90–95 mmHg). CVP was 12 cm H_2O. The arterial blood gas analysis showed the following results: pO_2 was 7.9 kPa (normal range 11–14 kPa), pCO_2 4.3 kPa (normal range 4.5–6 kPa), pH 7.48 (normal range 7.35–7.45), SaO_2 92% (normal range 94%–98%), pO2/ FiO2 169 (on the rebreathing mask, FiO2 35%). On the chest auscultation, bilateral rales were heard. During suction through the endotracheal tube, bloody, foamy aspirate was obtained.

She was immediately intubated and assisted with mechanical ventilation (BiLevel mode: with FiO_2 40%, PEEP 4 cm H_2O, peak inspiratory pressure 18 cmH20, pressure support 12 cmH2O and 12 respirations per minute) and midazolam infusion were initiated.

In the repeated arterial blood gas analysis pO2 was 17.3 kPa, pCO2 4.8 kPa, pH 7.51, SaO2 99%, pO2/ FiO2 324. On the chest radiography, bilateral pulmonary infiltrates were seen (Figure 5).

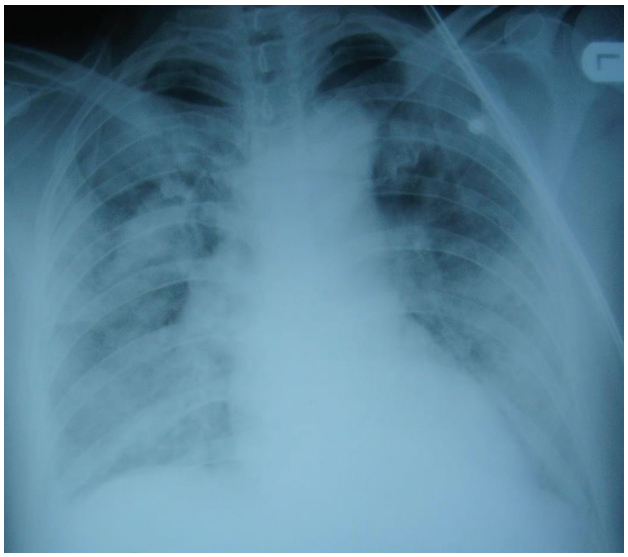

Fig. 5 – A chest x-ray of the patient four days after aneurysmal subarachnoid hemorrhage attack showing bilateral pulmonary infiltrates (neurogenic pulmonary oedema).

The N-terminal pro B-type antriuretic peptide (NT – proBNP) level was 5,829 pg mL^{-1} and troponin I level was 0.59 ng mL^{-1}. In the intensive care unit, transthoracic echocardiogram was made, showing the ballooning of left ventricular (LV) apex and midventricle and hypercontractile basal segments (Figure 6).

The LV diameters in systole and diastole were normal, the ejection fraction (EF) was about 50% and there were no

foreign masses in the apex of LV. Loop diuretic (furosemide 2 x 20 mg, iv.) was initiated as well as low molecular weight heparin – nadroparin 0.4 mL, subcutaneous, x 1, with the permission of a neurosurgeon. Because of high level of C-reactive protein 197/μg/mL, (the normal finding was less than 5 μg/mL) and because of the presence of previously inserted EVD, we decided to start a wide-spectrum antibiotic therapy with meropenem, vancomycin and metronidazole.

At the same time, we took samples of cerebrospinal fluid (CSF) for cytological and biochemical analysis, as well as CSF, blood, urine and tracheal aspirates for microbiological analyses. The analysis of CSF showed no cellular elements with: proteins 1.5 mg mL^{-1} and glucose 3.8 mmol L^{-1}. The CSF culture was sterile as well as the urine culture and blood culture. In the tracheal aspirate, there were 10^3 colonies forming units (CFU) per mL of coagulase negative *Staphylococcus*, sensitive on vancomycin.

Mechanical ventilation was continued till the 8th day after the SAH attack, when weaning from it was done successfully, because of the progressive resolution of pulmonary infiltrates, but without extubation.

A repeated echocardiographic study, 5 days afterwards, showed TCM in regression, with better contractility of the apical LV segment and EF of approximately 60% (Figure 7).

NT-proBNP was 384 pg mL^{-1} and troponin I level was 0.059 ng mL^{-1}. CRP level was 50.2 μg mL^{-1}. Nine days after the SAH attack, the pulmonary infiltrates were completely resolved, so the patient was extubated.

After extubation, the patient was conscious, eupneic, with heart rate 88 bpm, NIBP 130/85 mmHg and CVP 4 cm H_2O. In the arterial blood gas analysis on room air, it was found that pO_2 was 11.6 kPa, pCO_2 5.6 kPa, SaO_2 98%. Two days after, her previously installed EVD was removed in the ICU. The next day, she was discharged from the ICU, and on the 17th day after the insult, she was discharged from hospital. At the time of discharge, she was conscious, eupneic, without the neurological deficit. Postponed coronary angiography revealed no signs of coronary artery disease.

Discussion

The pathophysiology of cardiac dysfunction after SAH is not always clear. The three main theories explaining the pathogenesis of SAH-induced cardiac injury include: multivessel coronary artery spasm causing ischemia, microvascular dysfunction and catecholamine hypothesis.

There is a lack of convincing clinical, or animal data supporting the theory of SAH-induced multivessel coronary artery vasospasm [17]. The clinical data limited to single case reports have failed to demonstrate a decreased perfusion in SAH myocardium [18].

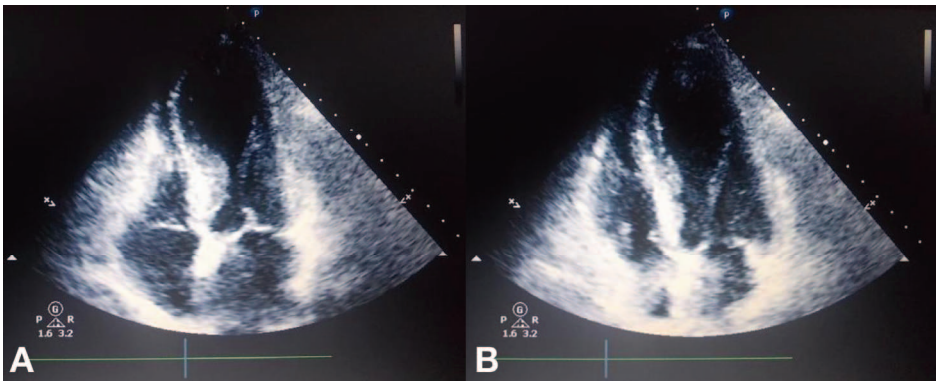

Fig. 6 – Transthoracic echocardiogram (apical 4-chamber view) 5 days after subarachnoid hemorrhage attack showing apical hypokinesia and basal sparing of left ventricle in systole (A) and in diastole (B).

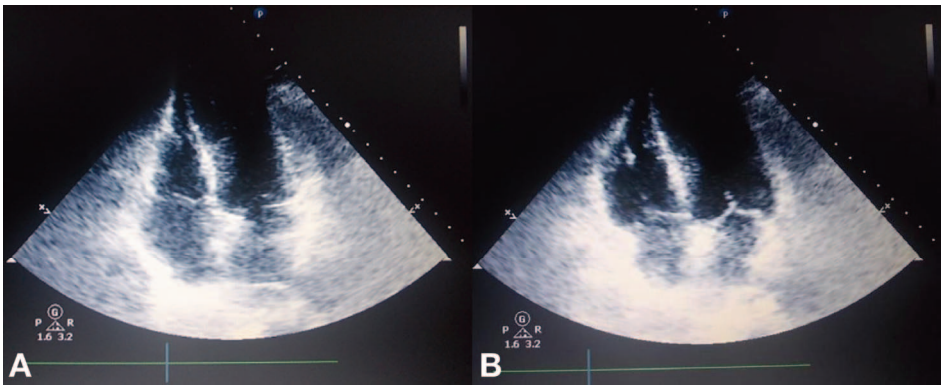

Fig. 7 – Transthoracic echocardiogram (apical 4-chamber view) 9 days after subarachnoid hemorrhage attack showing better apical contractility of left ventricle in systole (A) and in diastole (B).

The most widely accepted theory for the SAH-induced neurogenic myocardial stunning is the "catecholamine hypothesis". This theory suggests that the catecholamine-induced cardiac injury is the underlying cause of cardiac damage in the patients with SAH. Compared with the controls (healthy patients and those with headache), the patients with SAH have an increase in plasma noradrenalin within 48 h after the insult that persists during the first week and normalizes within 6 months [10].

The SAH animal studies were in agreement with the clinical studies [12, 13]. The experimental SAH animal studies not only demonstrate immediate excess sympathetic nervous activation with higher circulating catecholamine concentrations but the heart also appears to be more sensitive to the sympathetic stimulation as well [12, 13, 19]. The local noradrenalin production in the myocardium may surpass the systemic elevation of catecholamines and precipitate global, or regional LV systolic dysfunction [11, 20–22]. An explosive rise in the intracranial pressure (ICP) may cause the sympathetic activation via hypothalamic damage, and therefore an initial transient loss of consciousness at ictus, may represent a risk factor for possible cardiac damage.

Based on previously available data, TCM and neurogenic stunned myocardium appear to be both a marker of the severity of SAH and an independent predictor of symptomatic cerebral vasospasm – both elements associated with worse outcome [9].

NPE is pulmonary edema after the acute neurological insult without underlying lung or heart disease. There are some mechanisms of NPE after SAH.

First, at high pressure, a disruption of the capillary endothelium and alveolar epithelium will occur due to the raised capillary pressure with the development of a high-permeability of blood-lung barrier. A hydrostatic form of NPE develops.

Secondary, a severe depression of the left myocardial function occurring after SAH was regarded as another mechanism involved in NPE pathogenesis, as demonstrated in the retrospective study of 20 patients with NPE [22]. This is evident with most NPE patients demonstrating the increased pulmonary wedge pressure and the reduced cardiac output, or the reduced LV function [6].

Thirdly, some molecules, such as S100B and caspase-1, can be the link between the brain and the lungs that determines the development of NPE after SAH [23, 24].

TCM is a form of neurogenic stunned myocardium which is characterized by the reversible LV regional wall motion abnormalities with a pattern of apical akinesia and concomitant sparing of basal segments. TCM has been reported all over the world and was acknowledged by the American Heart Association as a form of reversible cardiomyopathy.

Four Mayo Clinic diagnostic criteria are required for the diagnosis of TCM: 1) transient left ventricular wall motion abnormalities involving the apical and/or midventricular myocardial segments with wall motion abnormalities extending beyond a single epicardial coronary artery distribution; 2) absence of obstructive epicardial coronary artery disease that could be responsible for the observed wall motion abnormality; 3) ECG abnormalities, such as transient ST-segment elevation and/or diffuse T wave inversion associated with a slight troponin elevation; and 4) the lack of proven pheochromocytoma and myocarditis.

To our knowledge, there are three series of the SAH patients with TCM and few case reports [25–30]. The incidence of TCM in SAH is 0.6%–0.8% [26, 27]. According to Guglin and Novotorova [31] literature reviews in 2011, there were 61 cases of TCM in SAH from 1990.

The first signs of cardiac dysfunction (negative T wave in II lead) in our patient were noticed on admission by ECG monitoring and elevated troponin I. Two days after hemorrhage, the deep negative T-waves in inferior and anterolateral ECG leads and prolonged QTc interval were seen. The patient had no signs and symptoms of acute cardiac disease. Troponin I was lowered, but still elevated as well as CK-MB.

The cardiac abnormalities can be seen with SAH. The ECG changes are present in 50% to 100% of patients, and include the deep T-wave inversion and QTc prolongation. The troponin elevation is seen in 20% to 40% of patients [4, 32, 33]. Elevated troponin I level occurs more frequently in severe SAH, as measured by Hunt and Hess grade, and the peak on the day of ictus with a decay thereafter 5.

Troponin I is 100% sensitive in detecting the LV dysfunction in SAH, compared to CK-MB which is much less sensitive at 29%–60% [4]. The superiority of troponin I over CK-MB as a marker of myocardial injury is consistent with the cardiac literature [34].

BNP and NT-proBNP are another noteworthy serum markers associated with neurogenic stunned myocardium [35]. Elevated plasma BNP is significantly associated with the regional wall motion abnormalities (RWMA), reduced ejection fraction, diastolic dysfunction, pulmonary edema, troponin I elevation, as well as early in-hospital mortality [35, 36].

Because these analyses were no significantly elevated for the diagnosis of acute myocardial infarction, we considered ECG changes as the SAH-induced cardiac injury with no contraindications for craniotomy. After craniotomy, our patient was hemodynamically stable, but the ECG changes persisted. Systolic dysfunction usually develops within the first 2 days after a neurologic event and then recovers [37].

Overall, 10%–28% of patients with SAH had a global or regional LV systolic dysfunction [8]. The development of NPE most frequently occurs within the first week from the beginning of SAH with a peak around day 3. The incidence of NPE decreased with time after SAH. NPE displayed biphasic in the SAH patients, the first peak with cardiogenic NPE caused by a cardiac dysfunction immediately after SAH, and hydrostatic NPE resulted from hypervolemia and low cardiac contractility 7 days after SAH [38].

Four days after hemorrhage, our patient became tachydyspneic, was found hypoxemic, and had to be intubated. Repeated ECG showed decreasing of negative T-waves, but troponin I was almost the same. Furthermore, the physical examination had shown auscultatory bilateral rales and chest radiography showed bilateral pulmonary infiltrates. Our pa-

tient fulfilled the criteria for NPE (physical auscultatory findings, need for oxygenation or mechanical ventilation and bilateral pulmonary infiltrates) [39].

A very high level of NT-proBNP level in blood confirmed cardiac origin of NPE, but our patient had no previous cardiac disease (cardiomyopathy, valvular disease or coronary artery disease). Transthoracic echocardiography clarified our case.

Apical and midventricular hypokinesia of the LV with a basal hypercontractility is a pattern seen in TCM. TCM with the reduced LV function led to congestive heart failure (20%) and pulmonary edema (10%) [15, 40]. The clinical presentation of TCM often resembles acute myocardial infarction, induced by the emotional or physical stress and predominantly occurs in postmenopausal women [41].

The reason behind the striking female predominance (more than 90%) is unclear [42, 43]. The diagnostic features of TCM include the reversible regional wall motion abnormalities beyond a single coronary artery distribution (typically involving the LV apex and midventricle with relative sparing of the basal segment), ECG abnormalities, minor elevation in cardiac biomarkers, and absence of significant coronary artery disease [42–46].

The Mayo Clinic criteria are different in involving midventricle with or without apex, absence of myocarditis and pheochromocytoma and not important role of stress. The cardiac catheterization in the SAH patient is a rare occurrence and should be reserved for the patients with SAH and features incompatible with neurogenic stunned myocardium.

There is no consensus about treatment of TCM. It includes a supportive therapy (intubation and mechanical ventilation, inotropic support, antihypertensives), β-blockers, diuretics, aspirin (if there is coexistent coronary artery disease), low-molecular-weight heparins (if the aneurysm is "solved"), ACE inhibitors [47].

However, the clinicians should be vigilant about potential difficulties that may arise, as the combination of reduced LV function in the setting of cerebral vasospasm window may amplify the deleterious effect of both. This subset of patients may be better treated with inotropic medications during cerebral vasospasm.

As the number of cases of TCM increases, medications for its prevention continue to be investigated. In the animal experiments, α- and β-blockade may be able to prevent TCM [48]. Some clinical data are encouraging. Further prospective studies are warranted to better understand and prevent complications of SAH.

Conclusion

Our case report reminds us that cardiac dysfunction is fairly common after aneurismal SAH and can mimic acute coronary syndrome. Currently, our prevailing practice is to measure the cardiac biomarkers levels in all SAH patients and, so far, to reveal the patients with the risk of regional wall motion abnormalities. Routine transthoracic echocardiography may be necessary in the patients with aneurismal SAH.

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