Neurogenic Myocardial Stunning Caused by Glioblastoma: A Case Report

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Neurogenic myocardial stunning or stress-induced cardiomyopathy is a reversible form of cardiomyopathy accompanying a variety of neurological conditions, occurring more often in elderly women. Even though the underlying pathophysioloy is not yet fully understood, increased sympathetic activity secondary to emotional or physical stress is believed to be the trigger of myocardial injury. This case report presents an 80-year-old woman with an undiagnosed primary brain tumor, manifesting as heart failure due to transient left ventricle dysfunction. Angiography excluded coronary artery obstruction. The extent of myocardium involved did not correspond to a particular coronary artery distribution. She was treated in accordance with heart failure guidelines and made a full recovery within a week.

Key words: cardiomyopathy, glioblastoma, ventriculography, echocardiography.

Neurogenní omráčení myokardu způsobené glioblastomem – kazuistika

Neurogenní omráčení myokardu neboli stresová kardiomyopatie je reverzibilní forma kardiomyopatie doprovázející celou řadu neurologických stavů, přičemž častěji se vyskytuje u starších žen. Ačkoliv patofyziologie, která je podkladem, není stále plně objasněna, má se za to, že spouštěčem poškození myokardu je zvýšená aktivita sympatiku v důsledku emočního nebo fyzického stresu. Kazuistika popisuje 80letou ženu s nediagnostikovaným primárním nádorem mozku, manifestovaným jako srdeční selhání v důsledku tranzitorní dysfunkce levé komory. Angiografie vyloučila obstrukci koronárních tepen. Rozsah postiženého myokardu neodpovídal konkrétní distribuci koronárních tepen. Pacientka byla léčena v souladu s pokyny pro léčbu srdečního selhání a během jednoho týdne se plně uzdravila.

Klíčová slova: kardiomyopatie, glioblastom, ventrikulografie, echokardiografie.

Introduction

Neurogenic stunning of the myocardium is a relatively rare form of cardiomyopathy, with a good prognosis. Up to 71 % of the affected population are women (1). This reversible disease commonly presents as heart failure with pulmonary edema, and electrocardiogram (ECG) abnormalities mimicking myocardial infarction. Life-threatening arrhythmias can occur. In severe cases cardiogenic shock may be present. Emotional or physical stress leading to an increase in sympathetic stimulation of the myocardium is considered to be the cause of cardiac injury. The precise mechanism underlying the association between the sympathetic surge and myocardial stunning is unknown, however three hypotheses have been suggested. One possibility is multi-vessel coronary artery spasm leading to ischemia with increased sympathetic tone causing vasoconstriction in the absence of coronary disease. Another option is sympathetically mediated microcirculatory dysfunction and microvascular spasm leading to abnormal coronary flow. The third possible mechanism is direct injury of cardiomyocytes. In the later high catecholamine levels encourage influx of extracellular calcium by increasing intracellular cyclic adenosine monophosphate (c-AMP), with the calcium overload decreasing myocyte viability. In animal models catecholamines can cause calcium overload and myocardial injury by interfering with sodium and calcium transporters through oxygen-derived free radicals. At the histological level, a distinct pattern of catecholamine induced myocardial damage is observed. There is contraction-band necrosis, characterized by hypercontracted sarcomeres and dense eosinophilic transverse bands, with an interstitial mononuclear inflammatory response in contrast to the polymorphonuclear inflammation seen with myocardial infarction (2).

A variety of central nervous system (CNS) events have been associated with this syndrome

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Figure 1. *Ventriculography illustrating the left ventricle during systole (A), and diastole (B)*

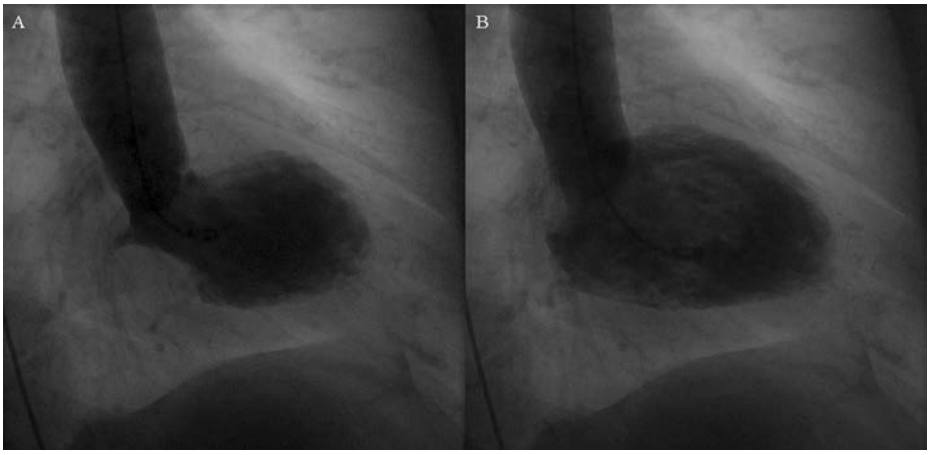


Figure 2. *Coronarografie illustrating the left (A) and right (B) coronary arteries*

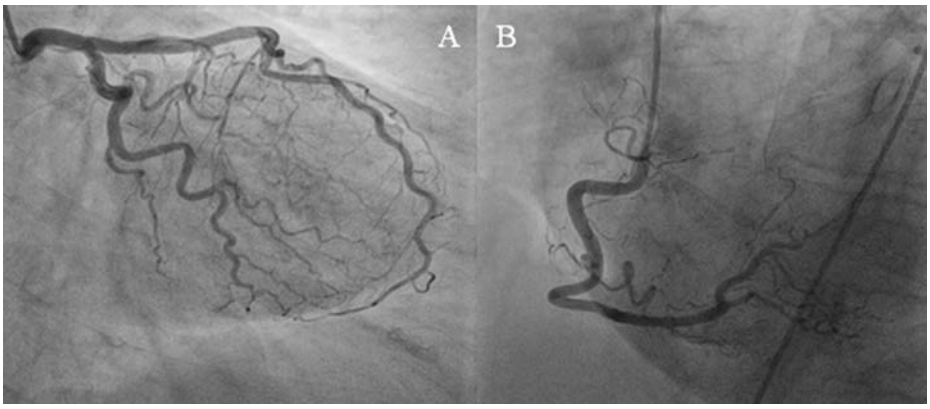
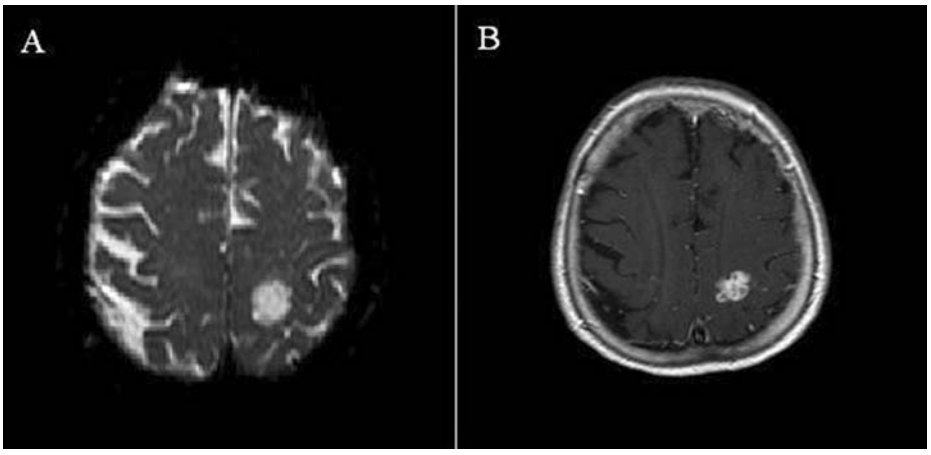


Figure 3. *CT scan (A), MRI (B) of the brain*



including subdural, subarachnoidal and intracranial hemorrhage as well as spinal cord and brain injuries, status epilepticus, etc. (3). The left ventricular systolic dysfunction which usually develops within the first 2 days after a neurologic event (1), does not correspond to a specific coronary artery distribution and in general resolves within a few days to weeks (4). Diagnosis is based on left ventricle dysfunction in the absence of coronary artery obstruction, very often diagnosed during cardiac catheterization for suspected myocardial infarction. Treatment is consistent with that of heart failure, including beta blockers, angiotensin converting enzyme inhibitors and diuretics. Although there is little evidence on long-term therapy, treatment with beta blockers can be continued to help prevent recurrence by reducing the effects of adrenaline and other stress hormones (4, 5).

Case description

An 80-year-old woman, who had undergone right shoulder disarticulation for chondrosarcoma, was admitted to the Coronary Care Unit shortly after midnight for evaluation of chest pain and dyspnoe. She had experienced sudden weakness and shortness of breath accompanied by crushing pain in the precordium as well as paresthesia and tremor in the absent limb prior to admission. On physical examination she was pale in appearance, orthopnoic and tachypnoic with a respiratory rate of 24 breaths per minute, pulse oximetry revealed an oxygen saturation of 90 %, fine insipratory crackles were present on the bases of the lungs. Her blood pressure was 90/60 mmHg; she had a fast and regular heart without murmurs. Apart from the missing right upper limb there were no other abnormalities. There was no jugular vein distention or ankle edema. ECG showed sinus tachycardia of 120/min., slight ST-segment elevation could be seen in leads V2–4 and Q waves in leads V1–4, but no contralateral ST depression was present. Mild cardiomegaly without significant pulmonary congestion was observed on her chest X-ray. Lab results showed a high-sensitivity troponin T of 109 ng/l on admission, with a peak of 421 ng/l the following day (normal range 0–14 ng/l), and a white blood cell count of 22.8x10^9/l. NT-proBNP level at admission was low (162 ng/l) despite obvious heart failure, however it was assessed early after the onset of symptoms and we believe that the peak level would have been much higher if another assessment was obtained with enough delay from the onset (in our facility limited to twice annually per patient). Serum electrolytes were normal. Heart failure treatment was started with intravenous furosemide, to which the patient responded well, with rapid regression of dyspnoe over night. The following morning cardiac catheterization was performed. Left ventriculography revealed akinesia of the apical two thirds of the left ventricle (Figure 1).

However, coronary angiogram showed no organic stenosis or vasospasm of the epicardial coronary arteries (Figure 2). On echocardiography, in accordance with ventriculography, there was severe hypokinesia of the left ventricle except for the basal segments, no other major abnormalities were found. Magnetic resonance imaging was planed initially, but was considered unnecessary as the diagnosis became clear during her stay.

Later that evening a convulsive state was observed, starting in the right lower extremity with deviation of the head and the eyes to the left, later on extending to the left-sided extremi-

ties. Diazepam was administered intravenously which lead to relieve. The patient was examined by a neurologist, during which the convulsions reoccurred. She admitted experiencing two similar attacks prior to admission. The neurologist confirmed a focal right-sided epileptical fit with secondary generalization. Computed tomography (CT) revealed an abnormal mass in the left parietal lobe suspicious of metastasis or primary brain tumor (Figure 3A). The following day magnetic resonance imaging (MRI) confirmed the presence of a glioblastoma (Figure 3B). The patient was transferred to the neurological ward and consequently to a neurosurgical facility for surgery. Echocardiography performed prior to surgery (a week after the initial assessment) documented normal ventricular motion and systolic function.

Conclusions

Considering the facts that glioblastoma is a rare primary brain tumor and that neurogenic stunning of the myocardium itself is not so common, makes the combination of both a distinct clinical entity.

Acute heart failure accompanying various neurologic events has been known for over a century; in 1908 Shanahan described neurogenic pulmonary edema (6). Later on, ECG abnormalities, including ST-T-segment changes, QT prolongation, U waves, and various arrhythmias were discovered virtually in all patients with subarachnoidal hemorrhage (SAH) and in many patients with other neurologic events (7).

In an analysis published in 2011, Maya Guglin MD and Irina Novotorova MD comparred neurogenic stunned myocardium with Tako-Tsubo cardiomyopathy and suggested expanding the definition of „stress-induced cardiomyopathy" to be used instead, as they share a same mechanism and clinical course and probabaly represent the same sydrome (1). Perhaps the diference between them is the presence of an organic CNS lesion in the prior.

The patient in this case study was treated according to heart failure guidelines and made a full recovery within a week from the cardiological point of view, with her overall prognosis remaining poor because of malignancy.

Despite modern and advanced medical technology, obtaining detailed personal anamnesis remains the main key to an early and correct diagnosis.

LITERATURE

1. Gulin M, Novotorova I. Neurogenic Stunned Myocardium and Takotsubo Cardiomyopathy Are the Same Syndrome: A Pooled Analysis. Congestive Heart Failure, 2011; 17(3): 127–132.
2. Wittstein I, Thiemann D, Lima J, Baughaman K, Schulman S, Gerstenblith G, Wu K, Rade J, Bivalacqua T, Champion H. Neurohumoral Features of Myocardial Stunning Due to Sudden Emotional Stress. The New England Journal of Medicine, 2005; 352: 539–548.
3. Davidson D, Terek M, Cchaw L. Neurogenic pulmonary edema. Annual Update in Intensive Care and Emergency Medicine 2012; 16: 212.
4. Harrison T. Harrison's Principles of Internal Medicine (18th ed., Vol. I). New York: Mac Graw Hill Medical, 2012: ISBN: 978–0-07–174890–2.
5. Reeder GS, Prasad A. (2014, APRIL 8). Stress (takotsubo) cardiomyopathy. Retrieved JULY 19, 2014, from UpToDate: http://www.uptodate.com/contents/stress-takotsubo-cardiomyopathy.
6. Shanahan W. Acute pulmonary edema as a complication of epileptic seizures. NY Medical Journal 1908; 37: 54–56.
7. Brouwers PJ, Wijdicks EF, Hasan D, Vermeulen M, Wever E, Frericks H, et al. Serial electrocardiographic recording in aneurysmal subarachnoid hemorrhage. Stroke 1989; 20: 1162–1167.

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