

Takotsubo cardiomyopathy in the patient with hemorrhagic stroke

Abstract

Takotsubo cardiomyopathy refers to the number of rare diseases, however, it is an increasingly reported syndrome characterized by transient regional left ventricular dysfunction in the absence of significant coronary artery disease. Takotsubo cardiomyopathy is typically triggered by an acute medical illness or by intense emotional or physical stress, although a triggering event is not always present. Represent a case of Takotsubo cardiomyopathy in the patient with hemorrhagic stroke.

Keywords: takotsubo cardiomyopathy, acute coronary syndrome, hemorrhagic stroke, stress, left ventricle, electrocardiography, echocardiography

Volume 3 Issue 5 - 2015

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Received: May 15, 2015 | **Published:** October 12, 2015

Abbreviations: TC, takotsubo cardiomyopathy; ECG, electrocardiogram; EF, ejection fraction; ACS, acute coronary syndrome; ICA, internal carotid artery; MCA, middle cerebral artery; LV, left ventricle; HR, heart rate; CT, computed tomography; PLAX, parasternal long axis position; 4AC, 4-chamber apical position; 5AC, 5-chamber apical position; 2AC, 2-chamber apical position; AHF, acute heart failure; AMI, acute myocardial infarction.

Introduction

Stress cardiomyopathy, also called apical ballooning syndrome, broken heart syndrome, takotsubo cardiomyopathy, and stress-induced cardiomyopathy, is an increasingly reported syndrome generally characterized by transient systolic dysfunction of the apical and/or mid segments of the left ventricle that mimics myocardial infarction, but in the absence of obstructive coronary artery disease. The clinical presentation of stress cardiomyopathy is similar to that of an acute myocardial infarction. Takotsubo cardiomyopathy or stress-cardiomyopathy related to the category of rare cardiomyopathies, gradually becoming one of the often diagnosable heart diseases. Stress cardiomyopathy was first described in Japan^{1,2} and was subsequently reported in non-Asian populations, including the United States^{3,4} and Europe.⁵ Currently TC recognized as an independent no so logical form and has a code in the International Classification of Diseases.

Clinical presentation and background

May 6, 2013 a woman of 52years with a symptom of stroke admitted in neurological department of the Tver regional clinical hospital. The patient's condition was extremely grav. From the words delivered medical staff and the relatives she fell ill acutely 06.05.2013, around noon have any sudden severe headache, weakness in the right limbs, loss of consciousness. According to relatives, the patient had no chronic diseases. Sometimes when measuring the arterial pressure was recorded mild hypertension, the appearance of which is associated with psycho-emotional factor (the patient worked in a managerial position). On admission the patient contact was unavailable. Level of consciousness was assessed as coma 2 on the scale of Glasgow. Somatic status without features. Respiratory rate 18 breaths per minute. SpO2 94%, it oxygen support (through a mask 3 l/min) SpO2 99%. Blood pressure 185/110mm Hg, HR 40min.

Diagnosis and management

CT brain revealed saccular aneurysm at the bifurcation of the left internal carotid artery, complicated by intracerebral medial

hemorrhage. Ultrasonic triplex scanning of the brachiocephalic arteries revealed backbone-modified type of blood flow in the pool of the internal carotid artery on the left side (peripheral type), the initial manifestation of dyslipidemia, hypoplasia of the right vertebral artery. Transcranial triplex scanning of the arteries of the circle of Willis revealed signs of perfusion steal in the MCA on the right, the extension III of the ventricles to 12mm. Registered ECG: sinus tachycardia 98 per minute, the impairment of repolarization in the lateral wall (Figure 1).



Figure 1 ECG 06.05.2013 13:23: sinus tachycardia 98 per minute.

In addition, on admission the patient underwent ultrasound. Echocardiography: violations of local contractility alone is not revealed, EF 61%. The seal of the aorta. Mitral regurgitation 1, tricuspid regurgitation 1. According to compression ultra sonography deep and superficial veins of legs were passable, the data for venous thrombosis has not been received. According to laboratory tests on admission revealed significant deviations was not. Only a dynamic study in the hemogram on the second day had leukocytosis, usual for the hemorrhagic stroke. The patient examined by a cardiologist, at the time of the cardiac examination revealed no pathology. On the basis of data sets was diagnosed with hemorrhagic stroke with the formation of the medial hematoma in the left hemisphere with the breakthrough of blood into the ventricular system on the background of rupture of saccular aneurysm of the bifurcation of the left ICA. Coma (06.05.13) The patient received multi component therapy in intensive care unit: mannitol 400,0 intravenously, emoxipin 1% 10ml intravenously, an intravenous infusion of magnesium sulfate 25%, enap 2.5mg intravenously once, etamzilat sodium 12,5% 4,0 intravenous *3 times a day, gordox 100000 intravenously *2 times a day, ascorbic acid 5% 10ml intravenous, glycine 1000mg/day sublingually, semax 1% intranasal omeprazole 20mg/day, nutritional support via nasogastric tube. From the evening of may 06, the patient's condition deteriorated: depression of consciousness up to coma III, a tendency to hypotension. Registered ECG: sinus tachycardia which 101 per minute, with QS V1-V3, ST elevation in these leads, the voltage reduction R from V4-V6 and in the first standard lead. Thus, there were no discordant to changing contrast, in the inferior leads also slight ST elevation (Figure

2). On the ECG, registered in 9 hours from a previous similar changes, QS appeared in V4 (Figure 3).

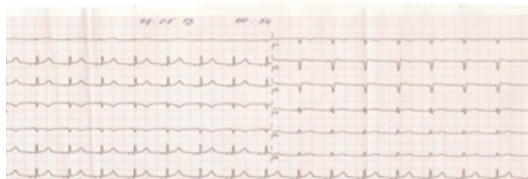


Figure 2 ECG 07.05.2013 00:54: sinus tachycardia which 101 per minute, with QSV1-V3, ST elevation in these leads, the voltage reduction R from V4-V6 and in the first standard lead.



Figure 3 ECG 07.05.2013 9:27: QS appeared in V4.

Repeated echocardiography revealed the zone of violation of local contractility (akinesis and pronounced hypokinesis) all segments on the medial and apical level with the formation of acute left ventricular aneurysm, a marked reduction of EF (23%), hyperkinesis all segments at the basal level (Figure 4-8). Troponin test was strongly positive (Figure 9). The patient is re-examined by a cardiologist, on the basis of ECG, Echo pattern, and the presence of markers of myocardial damage diagnosed with acute myocardial infarction in type 2 complicated by development of acute left ventricular aneurysm, acute heart failure Killip class IV. We carried out differential diagnosis between stress cardiomyopathy and myocarditis.

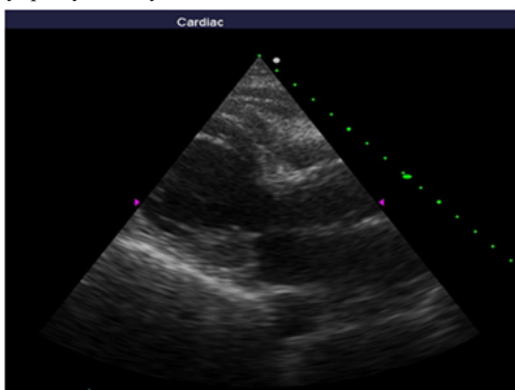


Figure 4 Echocardiography: PLAX, hyperkinesis of the basal segments with apical ballooning.

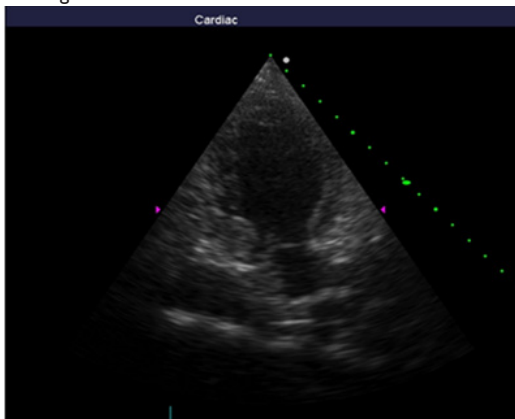


Figure 5 Echocardiography: 2AC, hyperkinesis of the basal segments with apical ballooning.

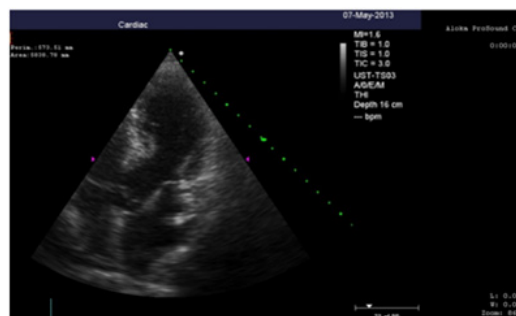


Figure 6 Echocardiography: 5AC, hyperkinesis of the basal segments with apical ballooning.

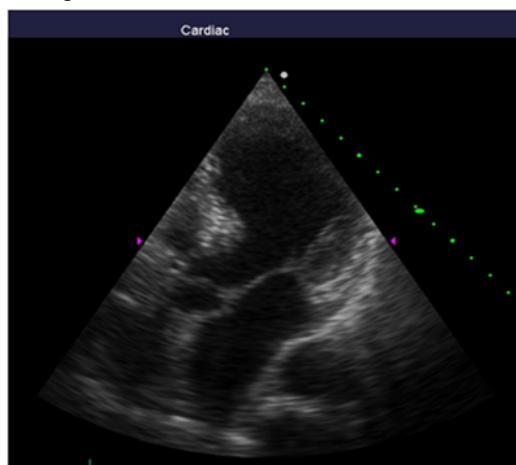


Figure 7 Echocardiography: 5AC, hyperkinesis of the basal segments with apical ballooning.

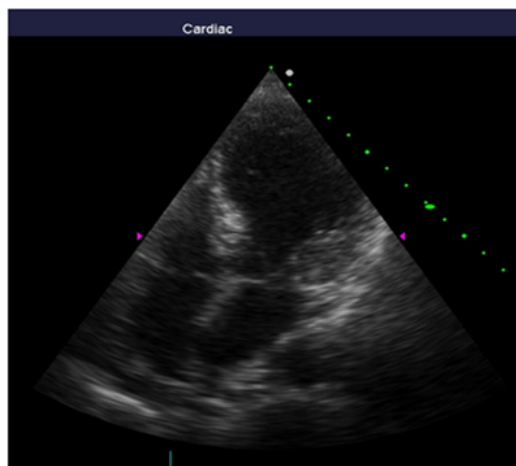


Figure 8 Echocardiography: 4AC, hyperkinesis of the basal segments with apical ballooning.

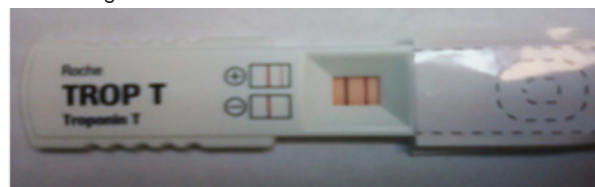


Figure 9 Troponin test was strongly positive.

Due to progression of arterial hypotension is connected vasopressor support (phenylephrine 0, 1%-1, 33mcg/kg/min, dopamine 0, 5%-5, 56mcg/kg/min). During the following days the patient's condition

progressively worsened: increased oppression consciousness, increased hypotension despite vasopressor and inotropic support. 08/05/13 04:55 biological death was pronounced (Figure 10). The patient was sent for post-mortem examination. The clinical diagnosis was the following: Hemorrhagic stroke with the formation of the medial hematoma in the left hemisphere with the breakthrough of blood into the ventricular system on the background of rupture of saccular aneurysm of the bifurcation of the left ICA. Edema and dislocation of the brain. Coma (06.05.13).

Acute circular Q-wave myocardial infarction type II.

a. Acute aneurysm of the left ventricle. AHF Killip class IV.

The results of post-mortem examination following.

- The diagnosis of stroke is confirmed, but no cardiac pathology was not identified in the myocardium was completely intact, no signs of atherosclerotic lesions of coronary arteries.
- According to the postmortem epicrisis, death occurred from rupture of a large aneurysm of artery of the brain, complicated by penetration of blood into the cerebral ventricle, edema and dislocation of his trunk, which was also the direct cause of death. Cardiac pathology is not revealed (Figure 11).
- When it became clear that the symptoms of heart disease was caused by TC, representing the nosological form of acquired cardiomyopathy characterized by transient left ventricular dysfunction in response to physical or mental stress.

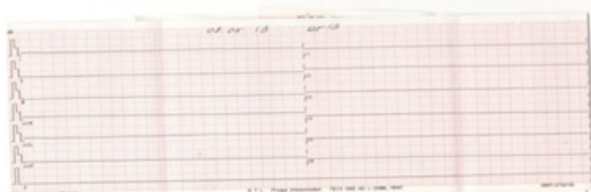


Figure 10 ECG 08.05.2015 05:13: asystole.

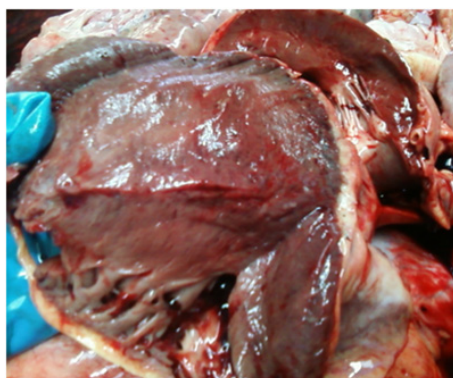


Figure 11 Post-mortem examination: cardiac pathology is not revealed.

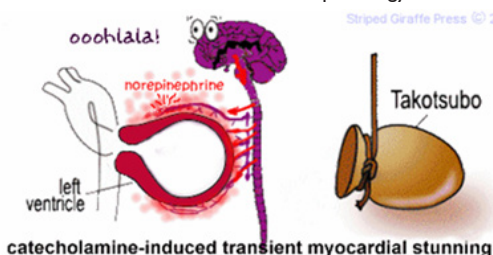


Figure 12 Humorous scheme of pathogenesis TC (Stripped Giraffe Press, 2005).

Discussion

Takotsubo cardiomyopathy clinical and ECG criteria resembles ACS, develops predominantly in postmenopausal women without signs of ischemic heart disease and has a relatively favourable prognosis. The term “takotsubo” is taken from the Japanese name for an octopus trap, which has a shape that is similar to the apical ballooning configuration of the LV in systole in the typical form of this disorder. Due to the fact that transient LV dysfunction is often caused severe emotional stress, such as death of a loved one, this state is also called “broken heart syndrome” (broken heart syndrome). To date, most researchers prefer the term “transient left ventricular dysfunction syndrome”. About a third of cases involve both right and left ventricles. To various stress circumstances preceding the development of CT include acute neurological diseases, urgent condition surgical procedure. Described cases of TC in patients with endocrine disorders: Addison’s disease, pheochromocytoma, thyrotoxicosis/ hyperthyroidism, as well as in diseases such as asthma, cancer of the esophagus. Menopause is an important risk factor TC in women.

The true incidence of TC is unknown. Probably syndrome not enough has been diagnosed has not yet been described in detail in Japanese literature. There is an assumption that in 0.7-2.5% of cases, with TC scans correctly diagnosed acute myocardial infarction. In Japan, the TC is detected in 1.7 to 2.2% of patients with ACS. In European studies, the prevalence of TC among all patients with ACS, subjected to angiography, reaches 2-3%. All these trials found a significant prevalence of older women. Etiopathogenesis TC is not exactly known. As already noted, the disease occurs mainly in females of older age. Among discuss possible etiological factors in the occurrence of TC the dominant is the emotional and physical stress, accompanied by acute release of catecholamines, increased sensitivity of adrenergic receptors, impaired sympathetic innervation of the heart and autonomic dysfunction that develops in response to stress.⁶ Currently being considered the following possible theories of the pathogenesis of TC: the increase in sympathoadrenal activity while reducing parasympathetic tone, catecholamines-induced multiple coronarospasm, coronary microvascular dysfunction, direct cardio toxicity of catecholamines, resulting in the development of the so-called “catecholamine stunning” of the myocardium (Figure 12). This is confirmed by the fact that in the blood of patients with TC show elevated catecholamines, the introduction of exogenous catecholamines could to contribute to its development.

Clinical manifestations of TC are nonspecific. Patients may appear anxious to mention increased sweating, possible tachycardia, or, conversely, bradycardia. Most often TC clinical manifestations are similar to those of AMI: acute chest pain (50-68%), shortness of breath for several days. In the case of acute LV failure may occur in cardiogenic shock. Although the symptoms TC scan are similar to the symptoms of AMI, mortality as a result 20% of patients with TC are received in the office of the emergency aid in a critical condition, but almost all of them survive. The main problem is that being a rare and little known disease, TC is often not diagnosed. The following are the proposed Mayo Clinic diagnostic criteria, all four of which are required for the diagnosis⁶:

- Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement. The regional wall motion abnormalities typically extend beyond a single epicardial coronary distribution. A stressful trigger is often, but not always, present.
- Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.

- c. New ECG abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin.
- d. Absence of pheochromocytoma or myocarditis.

Some researchers believe ECG method is sufficient for diagnosis of TC, because unlike AMI for the TC characteristic concordance of changes in T waves and ST segment (elevation of the ST segment, inversion of the T wave or QT interval). For the ECG the differential diagnosis of anterior AMI and TC use lower diversion: lack of ST-segment depression in the inferior leads, especially if the ST-segment elevation in lead II is greater than in lead III, speaks in favor of TC.

As the CT simulates an attack of acute coronary syndrome, it is first necessary to exclude the latter. CT is a common differential diagnosis of AMI, irrespective of presence of ST elevation. Differentiation is important because treatment is also different: while patients presenting with S-T elevation need to undergo cardiac catheterisation as the first diagnostic step, in practice this functions in parallel with clinical assessment, detection of elevated BNP/NT-proBNP levels, ECG performance and echocardiography as initial diagnostic tests and should be followed by the demonstration of extensive myocardial oedema on CMR.⁷ It is also necessary to differentiate between cardiac (pericarditis, aortic dissecting, cerebrovascular insufficiency) and extracardiac diseases (heart lung, pulmonary embolism, pneumonia, pneumothorax, esophageal spasm, cholecystitis, pathology of the spine), which may experience such symptoms.

Suggested clinical case met the criteria for diagnosis the Mayo clinic. In addition, there is evidence of relationship with acute cerebral infarction: it is a well-recognised complication of both cerebral infarction and haemorrhage.⁸

In the absence of clinical trial data, the appropriate duration of therapy is not known. β -blockers can be useful with the onset of the disease and in the long term. The need to apply other standard ambulatoriale means, such as statins, aspirin and calcium blockers questionable. Because of sympathoadrenal activation is central to the pathogenesis of TC, for the treatment and prevention of recurrence preferred long-term therapy with β - blockers with α -adrenoceptor blocking activity (e.g., carvedilol). Given the risk of parietal thrombus formation (5-8%) which is quite threatening complication in certain situations may be prescribed anticoagulants. After the treatment patients are advised to reduce the load and, if possible, to protect yourself from stress. Despite formidable initial clinical manifestations and data of instrumental methods of examination the prognosis of this disease is favorable. In 95% of patients have full recovery after transient LV dysfunction within 4-8weeks, occurring in one third of them by the end of the first week, still in the hospital. Various complications occur in about 20% of patients. Mortality from TC as a result of cardiogenic shock, heart failure, pulmonary edema, thinning and rupture of the myocardium is from 1 to 3.2%. Relapses occur in 2-10% of cases and is probably triggered by an associated trigger that should be considered when collecting history in each case.

Conclusion

The main problem is that being a rare and little known disease, TC is often not diagnosed. For suspected acute coronary syndrome in patients with various disorders, especially in women of menopausal period, you need to be vigilant in terms of TC.

Acknowledgments

None.

Conflicts of interest

Author declares there are no conflicts of interest.

Funding

None.

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