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Case Report Open Access

Takotsubo Cardiomyopathy Shortly Emerged after Falling through the Ice - A Case Report

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Abstract

This case is dedicated to a 71-year-old woman with stress induced cardiomyopathy emerged shortly after falling through the ice. She suffered from acute heart failure and fever. Electrocardiogram showed a small-scale ST elevation and echocardiography revealed typical dyskinesia of the apical segments, while coronary angiography demonstrated minimal changes in coronary arteries. The diagnosis of Takotsubo cardiomyopathy was suggested. The treatment of the patient included beta -blockers, angiotensin-converting enzyme inhibitors, aspirin, diuretics and antibiotics. In a while, the left ventricular function had fully recovered.

Keywords: Takotsubo cardiomyopathy; Ventricular dysfunction; Apical ballooning syndrome

Introduction

Takotsubo cardiomyopathy (TCM), also known as stress-induced cardiomyopathy, apical ballooning syndrome, and broken heart syndrome, is condition characterized by left ventricular apical ballooning, electrocardiographic changes with ST elevation or depression, formation of abnormal Q-wave, and elevation of cardiac enzymes mimics acute myocardial infarction, but without evidence of coronary artery disease [1,2].

Initially TCM was described by Satoh et al. [3]. In Japanese, there is a fisherman's octopus pot, the takotsubo, with rounded body and narrow neck [4]. The similar appearance we can see on echocardiogram, cardiac MRI, or ventriculography, at the end of systole the heart looks like a bulb with short neck and round bottom. The syndrome was renamed stress cardiomyopathy in 2006 [5].

There are a lot of theories but the etiology of the syndrome isn't clear yet. These theories include multivessel epicardial spasm, microvascular coronary dysfunction, catecholamine-induced injury and neurohumoral-related myocardial stunning [6].

The true incidence of stress cardiomyopathy is higher than estimated, as it remains unrecognized in many cases. TCM predominantly occurs in elderly postmenopausal women (over 90%) with the median age from 63 to 76 years of age. Mortality is only 1.1% [7,8].

Case Report

71-year-old female with history of hypertension, hyperlipidemia was skiing and suddenly fell through the ice. It took her about 10-20 min to get out from the cold water, and 1 hour to reach home. She was hospitalized in 4 hours after an accident with weakness, shortness of breath, tachycardia. The initial examination revealed a light drop of blood pressure (105/70 mm Hg), sinus tachycardia (130 beats/min), bubbling wheezes in medium and basal parts of the lungs, mostly on the right side.

A twelve-lead standard electrocardiogram (ECG) showed sinus tachycardia (129 beats/min) with small-scale ST-segment elevation in V_4-V_6 leads. Gradually negative T waves appeared in I, avL, V_4-V_6 leads. Previous ECG was not presented.

Routine laboratory tests showed increased levels of white blood cells 18.2*10*9/L (NR-9*10*9/L), neutrophils 81.3% (NR-75%), troponin T level was 1.10 ng/ml (NR<0.03 ng/ml), SaO2 80%.

X-ray examination and the chest computer-aided tomography showed dimming of the all lung fields and pleural effusion about 150 ml. Echocardiography revealed left ventricular systolic dysfunction (hyperkinesis of basal segments and akinesis of apical segments of the left ventricular). Left ventricular ejection fraction (LVEF) was reduced to 42%. Obstructive coronary heart disease was rejected during the coronarography (Figure 1).

The rest myocardial perfusion scintigraphy by gated single photon emission computed tomography was performed. It revealed a characteristic shape of the left ventricle, perfusion defects in apical and average part of anterior wall (Figure 2).

The patient was treated symptomatically (with β-blockers, angiotensin-converting enzyme inhibitors, aspirin, antibiotics and diuretics). The condition of the patient improved steadily, weakness and shortness of breath disappeared. The treatment in hospital took 7 days.

At the follow-up visit, echocardiography showed significant improvement in the left ventricular systolic function with a normal ejection fraction (about 63%) and normal wall motion. In two months myocardial perfusion scintigraphy revealed no relevant perfusion defects (Figure 3).

Discussion

The described case of such a rapid progress of the symptoms is not solitary. In the literature, there are many situations with acute appearance of cardiomyopathy just after information about somebody's death or defeat of the favorite soccer team [3,9].

Takotsubo cardiomyopathy is a rare, but an increasingly recognized type of cardiomyopathy occurring after a recent emotional or physical

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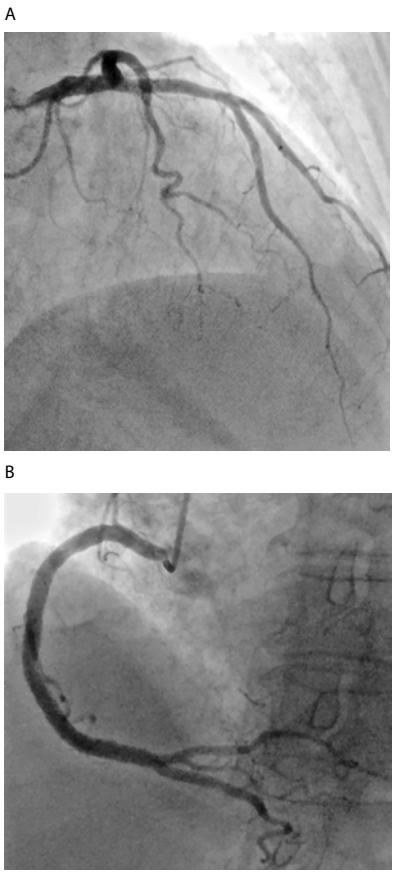

Figure 1: Coronarography. (A) Left main coronary artery. (B) Right coronary artery.

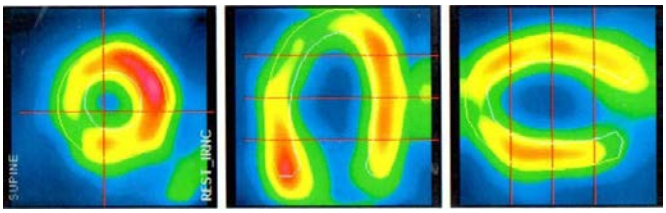

Figure 2: The rest myocardial perfusion scintigraphy made on the second day after incident, revealing perfusion defects in apical and average part of anterior wall.

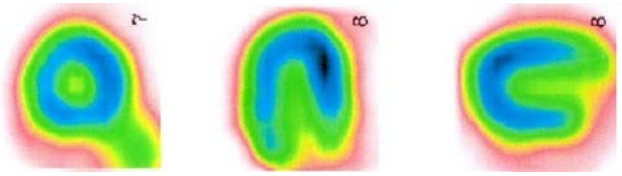

Figure 3: The rest myocardial perfusion scintigraphy made in two months after incident, without any significant perfusion defects.

stress or neurohumoral disorder. The prevalence of Takotsubo cardiomyopathy in the general population is estimated to be 1.2% to 2.2% [10]. Most patients are postmenopausal women.

Since the first description in Japanese studies, several case series have been published. Despite this the etiology and pathophysiology is not fully understood. There are a lot of mechanisms underlying the development of this transient ventricular dysfunction.

Researchers have implicated gender-related differences in this syndrome. Akashi et al. have hypothesized that the reduced estrogen levels after menopause explain the predisposition of elderly women to this cardiomyopathy. Estrogen is thought to promote vasodilatation through endothelial nitric oxide synthase. Postmenopausal women lose this protective effect, which may predispose them to coronary spasm and myocardial stunning in the setting of elevated catecholamine levels. But this does not explain the occurrence of the disease in men [11].

So a central role in the pathogenesis of Takotsubo cardiomyopathy has been identified for the level of catecholamines. Kume et al. have described elevated levels of catecholamines as the cause of cardiomyopathy. There are a lot of studies confirming the increased level of catecholamine in Takotsubo cardiomyopathy in compared with myocardial infarction [12,13].

Under usual catecholamine release, epinephrine and norepinephrine contact to myocardial β1 and β2 adrenoreseptors and increase heart contractility and rate (the positive inotropic effect). It is worth noting, that there is an increased density of beta adrenoreceptors in the apex. During the emotional stress excess release of catecholamine initiates a cascade of intracellular events that leads to a decrease inotropy. In connection with the pathophysiological features of the location of the β-receptors we can see characteristic apical ballooning and hyperkinesis of basal segments [14].

Another way of transient dysfunction of the left ventricular is microvascular dysfunction or a multivessel acute epicardial coronary spasm that caused by increased level norepinephrine. It is interesting to suggest, that that cold water also contributed to the development of TCM. It is known that cooling causes reflexive vasoconstriction, due to increased affinity of alpha-adrenergic receptors for norepinephrine in the vascular walls [15].

Some studies indicate that ultrastructural damage of cardiomyocytes can be connected with direct action of high level catecholamine, that to cyclic adenosine mono-phosphate (AMP)-mediated calcium overload of the cell, with the resultant decrease in synthetic activity and viability, or oxygen-derived free radicals [10].

Among the evidence suggesting an important role of catecholamines in the pathophysiology of cardiomyopathy is the observation that cardiomyopathy is common in patients with pheochromocytoma [16].

To assist in the diagnosis of stress-induced cardiomyopathy, the proposed Mayo criteria suggested that all of the following 4 conditions be met [16,17]:

1. Transient akinesis or dyskinensis of the left ventricular apical and mid-ventricular segments with regional wall-motion abnormalities extending beyond a single epicardial vascular distribution.
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
3. New ECG abnormalities (either ST-segment elevation or T-wave inversion).
4. Absence of: pheochromocytoma, myocarditis, intracranial bleeding.

There are no specific treatments for Takotsubo cardiomyopathy, but supportive measures are needed in many cases.

The prognosis is usually good. The average time to recovery is about 2-3 weeks. The risk of recurrence ranges from 2-10%, and the long-term prognosis is usually excellent.

Conclusion

There is the description of many reasons connected with the development of Takotsubo cardiomyopathy including emotional stress, pheochromocytoma, physical distress, surgery, but catecholamines are believed to play a central role. Such a rapid increase of symptoms, similar with an acute coronary syndrome (chest pain, dyspnea, ischemic changes on electrocardiogram (ST changes, or T-wave inversion) and cardiac enzyme elevation, which leads to the need of supplementary methods (coronary angiography) to establish the diagnosis. It may sparing patients the risks of unnecessary thrombolytic therapy.

Takotsubo cardiomyopathy should be considered in all patients presenting with acute onset chest pain and elevated cardiac biomarkers.

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