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Case report

Uncommon type of tako-tsubo cardiomyopathy – Case report and current view

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ABSTRACT

Tako-tsubo cardiomyopathy is a heart disease that imitates acute myocardial infarction. Classical findings include apical and mid segment hypokinesia. However, it may have different appearance than was originally described.

In our case report we describe a case of woman with tako-tsubo cardiomyopathy (TTC), who was admitted to hospital after a stressful event because of chest pain, with normal coronary angiogram and with mild elevation of Troponin-I level.

Electrocardiogram corresponded with non-Q myocardial infarction of inferior wall.

Following left ventriculography, echocardiography and magnetic resonance, impaired contractility of the basal part of inferior wall was noticed, together with good global ejection fraction. Full recovery was attained in five weeks after the onset.

This impairment of left ventricle is not typical for TTC. In our patient, the basal part of left ventricle was affected, and not the apex as it is usually seen in TTC. This finding corresponds to rare ''inverted'' form of TTC. Another atypical feature is segmental involvement that, moreover, covered the inferior wall. This morphological pattern, according to our best knowledge, has not yet been described in literature.

Pathophysiology, epidemiology and clinical significance are shortly reviewed in the paper.

Introduction

Tako-tsubo cardiomyopathy (TTC) is relatively recently described disease, which imitates clinical, laboratory and electrocardiographical (ECG) findings in acute myocardial infarction (AMI). The disease was first described by Japanese authors in 1991 [1,2]. The term ''tako-tsubo'' corresponds to the typical shape of left ventricle, which resembles to traditional Japanese octopus trap (''tako''=octopus, ''tsubo''=pot) [2]. Typically, it is characterized by ball-shaped ballooning of anterior wall and the apex. Low contractility in this area is

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accompanied by hyperkinesia of the basal part of left ventricle [3–5]. In approximately one quarter of the patients, there is also similar impairment of the right ventricle [6,7].

An emotional or physical stress is often initiating factor of TTC [8], however, in many cases, immediate cause is not apparent. Most of the patients are women in post-menopausal age [3,8,9]. Potentially life-threatening complications include cardiogenic shock, arrhythmias or pulmonary oedema [4], in-hospital mortality is about 2% [10]. Long-term prognosis is mostly favourable with complete resolution [3,11].

The diagnostic criteria according to Mayo Clinic are as follows:

1. New abnormalities on ECG (ST-segment elevation and/or T-wave inversion) and/or cardiac troponin elevation.
2. Transient (reversible) akinesia or dyskinesia of the left ventricular mid segments, eventually also with involvement of the apex, independently of vascular distribution.
3. Absence of obstructive coronary artery disease at coronary angiography.
4. Absence of myocarditis or pheochromocytoma.

Original criteria also included an absence of head injury or intracranial haemorrhage. However, the wall motion abnormalities in these cases, which are also known as neurogenic stunned myocardium (NSM), likely share the same pathogenetic mechanisms with the classical form of TTC [8,12].

The condition of no coronary atherosclerosis has also been put into question. The etiopathogenic mechanisms leading to TTC can be basically present regardless the presence or absence of coronary atherosclerosis [13]. From the diagnostic point of view, this can be compared for example to coincidence of ischaemic heart disease and dilated cardiomyopathy: one disease, in principle, does not exclude the other.

The terms ''tako-tsubo cardiomyopathy'', as well as alternative name ''apical ballooning syndrome'' are being still in use, in spite of many patients having abnormalities of different parts of heart wall than the apex [11,14]. Other synonymous terms are ''stress cardiomyopathy'' or ''broken-heart syndrome''. The disease is responsible for 1–2% of cases diagnosed as acute coronary syndrome [15], it is thus necessary to bear it in mind in differential diagnostics of acute chest pain with negative findings on coronary arteriogram and impaired ejection fraction.

Case report

We present a case of 56 year old female with positive family history of ischaemic heart disease, post hysterectomy and adnexectomy due to cervical carcinoma at 34 years; she had migraine with occasional attacks, but otherwise no disease. She was working as a secretary in public services, smoking 5–6 cigarettes daily and drinking 2–3 cups of coffee per day, she denied drinking alcohol.

The patient presented to the Emergency with chest pain and hypertension after a car accident when she was annoyed due to indecency of the second participant in this accident. Paramedics were called by her husband, who measured the blood pressure of 180/120 mmHg, regular heart rate of 80 beat per minute, oxygen saturation of 97%. She was given captopril 12.5 mg sublingually and diazepam 5 mg intravenously and she was immediately transferred to the nearest hospital for further management.

Laboratory tests on admission showed slight elevation of Troponin-I level of 0.48 micrograms per litre (μg/L) and reached the maximum of 0.72 μg/L within 24 h, while normal values do not exceed 0.014 μg/L. Neither creatine kinase (CK), nor its isoenzyme CK MB was elevated, as there were no other laboratory abnormalities noticed on admission.

Transthoracic echocardiography (TTE) was performed on admission (Fig. 1a and b) with the finding of posterobasal hypokinesis of the inferior wall and slightly decreased left ventricular systolic function with left ventricular ejection fraction (LVEF) of 48%. Electrocardiogram on admission showed Q wave in lead III, aVF with negative T wave in lead I, III, aVF and in lead V5–V6 (Fig. 2a and b) with normal AV conduction. The patient was given clopidogrel, acetyl salicylic acid (ASA) and low molecular weight heparin and she was transferred to the cath lab of St Anne's University Hospital in Brno.

Coronary angiography was performed and no stenosis or other impairment of coronary arteries was detected (Fig. 3a and b). On ventriculography, posterobasal hypokinesis of inferior wall was demonstrated (Fig. 3c and d), decreased left ventricular systolic function with LVEF of 45%. A suspicion for an atypical pattern of tako-tsubo cardiomyopathy was expressed. In differential diagnosis, thrombembolic AMI or AMI following coronary vasospasm could have been hypothesized.

Two days later the patient was discharged with following medication: metoprolol succinas 25 mg once daily and ASA 100 mg once daily. ECG and TTE on discharge showed the same finding as was seen on admission. Patient was followed up for next 2 months.

Second TTE (Fig. 1c and d) was performed 2 weeks after discharge and it showed only slight posterobasal hypokinesis of inferior wall. In the same time, a cardiac magnetic resonance imaging (Fig. 4a–d) was performed in order to get more information about the myocardial muscle. The examination confirmed the posterobasal hypokinesis of inferior wall. No scar in the myocardium, as a sign of AMI, was found in the late gadolinium enhancement.

Unfortunately the patient has not been agreeable to undergo the second MRI scan in order to assess if there are any new changes in the myocardium.

We assumed that if the late gadolinium enhancement had not shown any scar within the myocardium it is fairly convincing that there was no myocardial infarction.

The third TTE (Fig. 1e and f) was performed by the same echocardiographer 5 weeks after discharge.

Normal segmental and global left ventricular function was found. ECG curve showed no Q wave or T wave inversion in inferior leads (Fig. 2c).

The findings in cardiac MRI, as well as the complete resolution of ventricular wall motility and rearrangement of the ECG curve, were thus consistent with the first proposed diagnosis, i.e. TTC.