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TAKOTSUBO CARDIOMYOPATHY: A CASE-REPORT

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ABSTRACT

A 55 year-old-woman with no medical history presented to the emergency department complaining of chest pain and dyspnea. An ECG was performed, which showed ST-segment elevation in precordial leads from V1 to V5; elevated values of troponin I serum levels were found. Cardiac catheterization did not show obstructive coronary lesions, however hypokinesis of the apex and left ventricular segments were observed, with sparing of the basal segments. Afterwards a stressor precipitant was identified. The patient was diagnosed of takotsubo cardiomyopathy. Takotsubo cardiomyopathy probably accounts for 1–2% of all cases of suspected acute myocardial infarction, its true prevalence is unknown because of under diagnosis. The pathophysiological aspects of the disease are still a matter of debate and a standard treatment consensus is lacking.

INTRODUCTION

In the early 1990s, Japanese authors reported a reversible cardiomyopathy that seemed to be precipitated by acute emotional stress. Patients were usually postmenopausal women and developed signs and symptoms of an acute coronary syndrome associated with a transient apical and mid-ventricular wall motion abnormality despite the lack of obstructive coronary artery disease at the time of emergent coronary angiography, which usually recovers from weeks to months.

The shape of the ventricle at end systole resembled the Japanese fisherman's octopus pot – the takotsubo – from which the syndrome derives its original name.

It is now recognized that takotsubo cardiomyopathy (TC) stressor is not just an acute emotional stress, but also acute intracranial events, including intracranial bleeding, head trauma and ischemic stroke; acute medical illness, including sepsis and surgical procedures; overproduction of endogenous catecholamines (pheochromocytoma); and administration of exogenous catecholaminergic agents can be precipitant factors for the disease. TC has been acknowledged by the ACC/AHA as a unique form of reversible cardiomyopathy.

Clinical case

A 55 year-old-woman with no medical history, presented to the cardiac department complaining of oppressive chest pain of moderate intensity accompanied by dyspnea 6-h lasting.

An electrocardiogram (ECG) was performed on admission, which showed ST-segment elevation in precordial leads from V1 to V5, and T-wave inversion from V4 to V6. Initial troponin I determination showed a value of 3.6ng/ml which was above the 99th percentile upper reference limit.

ST-segment elevation acute coronary syndrome was first considered and an invasive approaching was implemented. Cardiac catheterization showed no evidence of obstructive lesions in the coronary arteries. The ventriculography showed an atypical pattern of myocardial contraction, with hypokinesis affecting the mid ventricular and apical segments; whereas the motion in the basal segments was spared.

The following troponin serum levels were 2.6ng/ml and 1.2ng/ml, which were lower than those expected for the magnitude of ST segment elevation. No other relevant data was gathered. Stress takotsubo cardiomyopathy was suspected and further research was warranted.

A cardiac magnetic resonance imaging (MRI) was performed to evaluate the presence of myocardial edema as a marker of an inflammatory process, no abnormalities suggestive of this entity were found. Normal urinary test with a negative determination of metanephrines and the absence of suprarenal masses on MRI helped us to reject the presence of pheochromocytoma. Takotsubo cardiomyopathy was diagnosed and evidence-based medical therapy implemented.

DISCUSSION

Patients with TC are most commonly postmenopausal women. In a systematic review, women accounted for 82–100% of patients with an average age of 62–75 years, although cases have been described in individuals aged 10–91 years. Accordingly to recent reports, this condition probably accounts for 1–2% of all cases of suspected acute myocardial infarction. However its true incidence is unknown, since out of Japan there have been relatively few reported cases. Till 2001 there had been reported just 2 TC cases in non-Japanese people. The first case reported in Mexico was in 2004 and then in 2011 two more cases were reported in just one year. Therefore it is suggested that a lack of awareness about the disease is one of the reasons of underdiagnosis.

TC is considered to be among stress-related cardiomyopathies, which occur during times of enhanced sympathetic tone and can be precipitated by excessive endogenous or exogenous catecholamine stimulation of the myocardium, a condition that is also seen in cases of intracranial hemorrhage, ischemic stroke, head trauma, pheochromocytoma, and in critically ill patients. Several mechanisms have been implicated in its pathophysiology: multivessel epicardial coronary artery spasm, coronary microvascular impairment, direct catecholamine cardiotoxicity and neurogenic stunned myocardium, though none of them can wholly explain the wide expectrum of the disease. A different hypothesis is that high levels of circulating epinephrine trigger a switch in intracellular signal trafficking, from Gs (stimulating) protein to Gi (inhibitory) protein signaling through the β2 adrenoreceptor, producing a negatively inotropic effect that is greatest at the apical myocardium, in which the density of β-adrenoceptors is the highest. Overall, a catecholamine-implicated mechanism seem to be the one with the best background.

The most frequent clinical symptoms of TC on admission are chest pain and dyspnea resembling acute myocardial infarction. Moreover, the ECG findings on admission often include ST elevation in precordial leads. Subsequent T-wave inversion and Q-wave formation also are frequently found. However coronary angiography, which is the best single tool to diagnose this condition, shows no flow-limiting lesions.

There have been identified different morphological patterns of left ventricular (LV) affection, which includes: apical and mid-ventricular dysfunction, isolated mid-ventricular and basal dysfunction or isolated mid-ventricular dysfunction (apical-sparing), isolated basal

dysfunction, global hypokinesis, and other non-coronary distribution wall motion abnormalities.

Researchers at the Mayo Clinic proposed diagnostic criteria in 2004, which included: (1) transient akinesis or dyskinesis of the left 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 electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) and (4) absence of all of the following: recent significant head trauma, intracranial bleeding, pheochromocytoma, obstructive epicardial coronary artery disease, myocarditis, and hypertrophic cardiomyopathy.

The best imaging study to make the diagnosis of TC is cardiac catheterization, however cardiac MRI allows complete anatomical information and may help to distinguish TC from other acute cardiac syndromes. Many recent studies have reported that the number of patients with mid-ventricular dysfunction is increased compared with the initial description of the disease. The atypical presentation with apical sparing and affection only in the basal segments is not rare.

Cardiac MRI imaging helps in excluding the differential diagnoses of myocarditis or aborted myocardial infarction, and defining the ventricular abnormalities in patients with poor echogenicity, while nuclear cardiac imaging is useful for the detection of viable myocardium and the functional abnormalities.

There are no specific treatments for the left ventricular failure of TC because cardiac function is normalized within a few weeks. The use of long-term adrenoceptor blocker therapy seems rational to prevent recurrence, although its use in the acute phase of TC is still a matter of debate. Treatment with a combined alpha and beta blocker seems rational, so as we do not leave alfa-activity with an unopposed effect, favoring vasoconstriction. However beta-blocking drugs not always have provided absolute protection against recurrent event.

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