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Review Article

Stress cardiomyopathy: yet another type of neurocardiogenic injury 'Stress cardiomyopathy'

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ABSTRACT

Tako-tsubo syndrome pertains to rare acquired cardiomyopathies, characterized by left ventricular dyskinesia and symptomatology typical for acute myocardial infarction (AMI). Despite its low incidence and relatively benign course, stress cardiomyopathy should be thoroughly differentiated from AMI. The importance of tako-tsubo consists of the fact that its manifestation initially resembles AMI. Despite seemingly low incidence of tako-tsubo, acute coronary syndromes globally constitute a major epidemiological issue and both clinical entities should be accurately differentiated. Many patients present with only mild troponin release, certain extent of regional wall motion abnormalities (RWMA) and absence of hemodynamically significant coronary artery stenosis. In such instances, a careful interview aimed at preceding emotional or physical traumatic event should be undertaken. The subsequent verification of the diagnosis is based upon prompt recovery of contractile function. Although precise diagnostic criteria were formulated, symptomatology of tako-tsubo might be clinically misleading due to the possibility of concomitant coronary vasospasm, atypical pattern of RWMA and presence of non-significant coronary disease. For this reason, its exact rate might be underestimated. Stress cardiomyopathy reflects merely a single aspect of a much wider range of neurocardiogenic injury, which encompasses cardiac dysfunction associated with subarachnoid hemorrhage, intracranial hypertension and cerebral ischemia. Both psychological and physical insult to central nervous system may trigger a disastrous response of sympathetic nervous system, eventually leading to end-organ catecholamine-mediated damage. This review sought to delineate the phenomenon of tako-tsubo cardiomyopathy and deliver evidence for common pathophysiology of the broad spectrum of neurocardiogenic injury.

1. Introduction

Discovery of tako-tsubo syndrome (stress-induced cardiomyopathy; transient left ventricular apical ballooning; apical ballooning syndrome, ABS; broken heart syndrome) underscored the importance of patient's psychological condition, which is a frequently neglected factor in daily clinical practice. The startling possibility of cardiac dysfunction secondary to psychological distress has caught the attention of researchers, who keep searching for a reliable explanation of this phenomenon. It has been known for decades that brain–heart interactions occur in the event of acute neurological catastrophe, e.g., subarachnoid hemorrhage (SAH) and intracerebral hemorrhage [1–4], various forms of intracranial hypertension [5] or cerebral ischemia [6]. From the very beginning, it was postulated that the dysfunction of autonomic nervous system might serve as a plausible explanation to concurrent cerebral and myocardial lesions, and both sympathetic and parasympathetic nervous system were deemed responsible for the clinical presentation [7,8]. However, little was known about extensive interactions between brain and heart function on a psychological level until 1990 when Hikaru Sato described an acute form of cardiomyopathy characterized by transient left ventricle apical dyskinesia (tako-tsubo cardiomyopathy) triggered by severe emotional stress [9].

Despite a great deal of publicity, paucity of data makes it difficult to create a comprehensive definition of tako-tsubo syndrome. Contemporary knowledge is based intrinsically upon several case series and few cohorts of roughly 100 patients. Recently, a multicenter International Tako-tsubo Registry (InterTAK Registry) was established to explore natural history, clinical phenotype and prognosis of stress cardiomyopathy. The study completion date is planned for the year 2020 [10], and it should provide reliable high-volume data on this rare clinical entity.

In a classic form of tako-tsubo, a powerful traumatic life event (20–47% of all patients) precipitates angina-like chest pain [11,12], with concomitant ST-segment elevation and rise of cardiac troponin values, along with transient regional wall motion abnormalities (RWMA) concerning predominantly apical segments of left ventricle (Fig. 1). The mysterious term tako-tsubo derives from the characteristic image of apical dyskinesia on ventriculography resembling Japanese fishing pot

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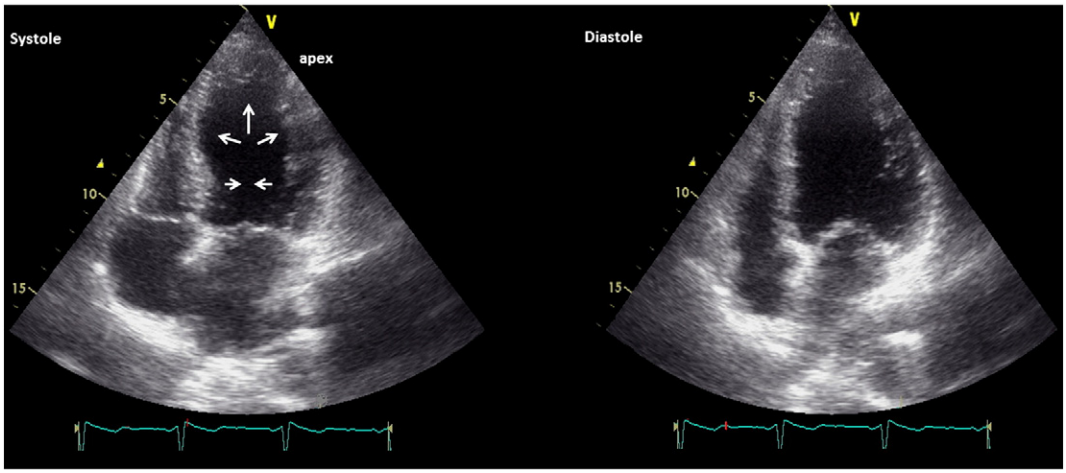

Fig. 1. Typical echocardiographic presentation of the acute phase of tako-tsubo cardiomyopathy. A 61-year-old female presenting with acute retrosternal chest pain, mild troponin elevation and dyskinesia of the apical segments of left ventricle following deep brain stimulation for Parkinson's disease.

used as an octopus trap. The whole clinical picture imitates ST-segment elevation acute myocardial infarction (AMI); however, emergent coronary angiography reveals no hemodynamically significant lesions in major coronary arteries [13] (Table 1). According to hitherto studies, the onset of cardiac dysfunction can be associated with anger, frustration, financial and employment problems, grief and loss of a close person and interpersonal conflict, as well as panic, fear and anxiety [11,13]. A rise in the incidence of tako-tsubo was also reported after the Central Niigata Prefecture earthquake in Japan in 2004 [14]. Surprisingly, cases of tako-tsubo can also be preceded by physical and neurological distress (in about 42–43% of cases [11,12]), such as acute respiratory failure, malignancy, infection, injury, perioperative period and last but not least pathology of central nervous system, most frequently associated with SAH [11]. A considerable extent of physical triggers was related with iatrogenic exposure to catecholamine or β-agonist agents in the course of treatment, such as inhaled β-agonists for acute respiratory failure or iv dobutamine during stress echocardiography or the use of phenylephrine [11].

In the beginning, stress cardiomyopathy was thought to be indigenous for Far East (predominantly Japan), where the syndrome was first described [9]. However, it turned out that its prevalence is worldwide and numerous case series from USA and Europe were published in recent years. Although the exact incidence is unknown, it concerns approximately 0.7–2% of all patients with initial suspicion of acute coronary syndrome [15–19]. This syndrome more frequently affects postmenopausal women (96%) and the cause of this predisposition is unknown [11]. Contrary to females, tako-tsubo cardiomyopathy in males is frequently precipitated by physical stress [20]. Depending on the report, the mean age at presentation is 65–68 years [11,12,17,21]. Only 10% of patients are below the age of 50 years [11].

In line with the systematic review conducted by Gianni et al., patients with tako-tsubo cardiomyopathy did not distinguish themselves with an increased prevalence of standard cardiovascular risk factors, barring slightly elevated extent of hypertensive individuals (arterial hypertension, 43%; diabetes, 11%; dyslipidemia, 25%; smoking, 23%) [15], which may potentially be linked to increased sympathetic tone or chronic serum catecholamine elevation.

Table 1
Revised criteria for the diagnosis of tako-tsubo cardiomyopathy proposed by Mayo Clinic [13]

Each and every criterion should be met for a liable diagnosis of ABS:
1. Transient hypokinesia, akinesia or dyskinesia of the left ventricular mid-segments with or without apical involvement RWMA extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture
3. New ECG abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin
4. Absence of Pheochromocytoma Myocarditis

It is possible that a patient with obstructive coronary atherosclerosis may also develop 'tako-tsubo' cardiomyopathy, but these cases are often misdiagnosed as acute coronary syndrome and such instance are rare in literature.

2. Signs and symptoms

The primary presentation involves predominantly angina-like chest pain (63% of patients) [11], but occasionally, dyspnea or syncope is the primary manifestation. The incidence of various symptoms is highlighted in Table 2. In addition, some cases of pre-admission cardiac arrest were reported [15,22]. Interestingly, the presentation was shown to vary depending on the etiology. Chest pain was the predominant symptom in case of tako-tsubo triggered by psychological stimulus, whereas dyspnea was more frequently observed in cardiomyopathy precipitated by physical insult or medical illness [23]. Up to 22% of patients exhibit various extent of pulmonary edema [12] and mild to moderate congestive heart failure with retention of fluids [11]. Hypotension and syncope can develop due to diminished left ventricular ejection fraction and reduced stroke volume or the presence of dynamic left ventricular outflow tract (LVOT) obstruction [24,25].

3. Electrocardiographic abnormalities

In a classic scenario, stress cardiomyopathy is characterized by electrocardiographic (ECG) abnormalities resembling AMI; however, these findings are also strikingly similar to those seen in cardiac dysfunction associated with cerebral pathology, most importantly

Table 2
Incidence of tako-tsubo symptoms [11,12,21]

Symptoms	Incidence (%)
Chest pain/discomfort	63–90
Dyspnea	18–32
Nausea and vomiting	16
Syncope	3–4
Cardiac arrest	1–2
Transitory global amnesia	2
Asymptomatic	8–15