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Case Report

Takotsubo Syndrome During Prophylactic Cesarean Section

Nassima Ouguerzi[1]*, Moad Belouad[1], Samia Bennauceur[1], Abdeladem Ayadine[1], Ahmed Fakri[2], Saad Benali[1], Moulay Mahdi Elhassani[1] and Jaouad Kouach[1]

[1]Department of Gynecology and Obstetrics, Military Hospital of Instruction Mohamed V Rabat, Rabat, Morocco

[2]Department of Anesthesia, Military Hospital of Instruction Mohamed V Rabat, Rabat, Morocco

***Corresponding Author:** Nassima Ouguerzi, Department of Gynecology and Obstetrics, Military Hospital of Instruction Mohamed V Rabat, Rabat, Morocco.

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Abstract

Takotsubo cardiomyopathy, often referred to as broke heart syndrome, apical balloon syndrome, or stress cardiomyopathy, occurs when a stressful emotional or physical event causes the left ventricle of the heart to enlarge, resulting in sudden heart failure. The condition was initially reported in Japan in 1990. Catecholamine drive is critical in the development and pathophysiology of Takotsubo cardiomyopathy.

Supportive and symptomatic medicine remains the basis of therapy, with a focus on increasing left ventricular function for several days and complete recovery in 3 - 4 weeks. Takotsubo cardiomyopathy, which is comparable to myocardial infarction, need a rigorous diagnosis and therapy to provide the best possible outcome.

This abstract highlights the importance of recognizing Takotsubo cardiomyopathy in clinical practice, particularly in high-stress scenarios such as surgical procedures, to ensure timely and effective management. Further research is warranted to elucidate the underlying mechanisms and improve therapeutic approaches for this intriguing cardiac phenomenon.

Keywords: *Takotsubo; Broken Heart; Cardiomyopathy; Caesarean Section; Reversible*

Introduction

Hikaru Sato, MD, PhD first reported Takotsubo cardiomyopathy in Japan in 1990 [1].

Takotsubo cardiomyopathy (TC) is a brief and reversible systolic anomaly of the left ventricle's apical portion that mimics myocardial infarction (MI) in the absence of coronary artery disease (CAD) [2].

The frequency is 1.0 - 2.5%, with most instances to occur in post-menopausal women [3]. In clinical terms, individuals present with chest pain accompanied by electrocardiographic changes, elevated troponin and left ventricular dysfunction mimicking acute myocardial infarction, but without obstructive coronary disease or ruptured plaques [4].

We report the case of this syndrome during a prophylactic cesarean section to consolidate our current knowledge and describe the pathophysiology, diagnostic features, current therapeutic strategies and clinical results of this interesting pathology.

Case Report

31-year-old patient, no medical history, caesarean section 6 years ago for pelvis with limits, under spinal anaesthesia without incident.

On assessment, the patient was classified as ASA 1 and was a candidate for a prophylactic caesarean section for a borderline pelvis.

After spinal anaesthesia, the patient initially presented with tachycardia and headache, with secondary worsening of the respiratory tract; desaturation under high concentration mask at 50% and crepitus on auscultation, with recurrence to orotracheal intubation with rapid Sequence on respiratory and hemodynamic criteria.

The newborn was in a state of apparent death with an APGAR score of 2/10 and was transferred to neonatal intensive care after intubation.

The patient was placed on positive pressure ventilation, with Furosemide 80mg iv, and a right central jugular venous line and a 16 left peripheral venous line were inserted in the upper limb with the introduction of adrenaline using a self-pulsing syringe.

After conditioning, the patient was intubated, ventilated and sedated with Midazolam and Fentanyl. Hemodynamically, she was given adrenaline 10 mg/50cc with an Auto-Pulse Syringe at speed 12, the patient recovered cardiac function after 20 days in intensive care and then in cardiology. The newborn recovered respiratory function in neonatal intensive care without neurological sequelae.

Discussion

Takotsubo syndrome was first described in Japan over 30 years ago and has been reported in around 1 to 2% of all ACS cases suspected of being "troponin-positive" [5]. More to the point, the contribution of societal stress was highlighted during the Coronavirus (COVID)-19 pandemic, where social isolation, financial stress and psychological anxiety led to a significant increase (3-4x) in the incidence of TTS compared with the pre-pandemic incidence [6].

Around the world, TTS is more common in older persons and in women, especially those who have gone through menopause. It's interesting to note that in western countries, women are nine times more likely to be diagnosed with TTS than in Japan, where men are more likely to be diagnosed with TTS [7].

Significant stress in Takotsubo cardiomyopathy produces swelling of the left ventricle. In 75 - 80% of patients, this phenomenon occurs at the apex of the left ventricle, producing the classic appearance of Takotsubo cardiomyopathy. In 10 - 20% of patients, mid-ventricular ballooning is observed. In rare cases, basal, biventricular or focal ballooning has been seen. Ventricular ballooning causes transient ST-segment elevation on the ECG, elevated troponin and B-type natriuretic protein (BNP) levels, and decreased ejection fraction, leading to acute systolic heart failure [8].

Many pathogenic processes have been hypothesized for Takotsubo cardiomyopathy, while the exact pathophysiology remains unknown. Takotsubo cardiomyopathy has been linked to stressful triggers, and as a result, it is thought that the adrenergic system is important to the pathophysiology of the condition. More precisely, it is believed that catecholamines released during stressful times, such as norepinephrine and adrenaline, cause apical ballooning by directly damaging the heart and/or by disrupting the heart's microvasculature. In individuals with Takotsubo cardiomyopathy, elevated serum catecholamine levels have been reported, as well as localized catecholamine release in cardiac nerve terminals [9].

Since Takotsubo cardiomyopathy is more prevalent in women who have gone through menopause, oestrogen may be involved in the pathogenesis of the illness. Nevertheless, there isn't much proof in the literature to back up this theory. In animal research, hormone replacement treatment has already demonstrated protection against Takotsubo cardiomyopathy [10]. Moreover, none of the sixteen postmenopausal women with Takotsubo cardiomyopathy in a short single-center case series from 2010 were on hormone replacement treatment. Researchers have concluded that there are other mechanisms besides oestrogen deficiency that contribute to the development

of Takotsubo cardiomyopathy, as evidenced by more recent small retrospective studies that did not show hormone replacement therapy to prevent the development of the syndrome in a small subset of postmenopausal study subjects [11].

Takotsubo cardiomyopathy presents very similarly to myocardial infarction. In fact, most patients diagnosed with Takotsubo cardiomyopathy were initially suspected of having acute coronary syndrome. Approximately 2% of people initially suspected of having ACS are eventually diagnosed with Takotsubo cardiomyopathy. More than 75% of patients report having chest discomfort, 50% report having dyspnea, and more than 25% have dizziness as their most prevalent symptom. Additionally, patients may occasionally develop syncope and generalized weakness. Patients with Takotsubo cardiomyopathy typically present with crackles, tachycardia, hypotension, low blood pressure, and chest discomfort on physical examination; these symptoms are consistent with abrupt systolic heart failure [8].

Since the clinical presentation of TTS frequently resembles that of an acute myocardial infarction, it can be challenging to diagnose. Therefore, in any patient presenting with chest discomfort and suspected ACS, TTS should be investigated as a differential diagnosis, especially if there has been a history of severe mental or physical stress or sickness.

Many diagnostic standards have been created over time, including the revised Mayo Clinic criteria which are widely used to diagnose Takotsubo cardiomyopathy (Table 1 below).

1. Temporary hypokinesis, dyskinesis, or akinesis in LV segments with or without apical involvement; aberration in regional wall motion exceeding past a single vascular distribution; the existence of stress elicitation.
2. No presence of significant coronary artery disease.
3. Recent changes in electrocardiography (ECG) (ST segment elevation and/or T-wave inversion) or significant elevation of cardiac troponin serum levels.
4. Non-existence of pheochromocytoma or myocarditis.

Table 1: *Summary of TC diagnosis criteria [12].*

The most recent are the InterTAK diagnostic criteria (Table 2 below) developed by the Takotsubo International Registry. The most significant changes from the previous modified Mayo Clinic criteria recognise that significant coronary artery disease can co-exist with TTS and is not mutually exclusive, and that while the absence of pheochromocytoma was previously required, the InterTAK diagnostic criteria recognise that pheochromocytoma can function as a trigger for TTS.

1. Patients show transient left ventricular dysfunction (hypokinesia, akinesia or dyskinesia) presenting as apical ballooning or midventricular, basal or focal wall motion abnormalities. Right ventricular involvement can be present. Besides these regional wall motion patterns, transitions between all types can exist. The regional wall motion abnormality usually extends beyond a single epicardial vascular distribution; however, rare cases can exist where the regional wall motion abnormality is present in the subtended myocardial territory of a single coronary artery (focal TTS).
2. An emotional, physical or combined trigger can precede the takotsubo syndrome event, but this is not obligatory.
3. Neurologic disorders (e.g. subarachnoid haemorrhage, stroke/transient ischaemic attack or seizures) as well as pheochromocytoma may serve as triggers for takotsubo syndrome.
4. New ECG abnormalities are present (ST-segment elevation, ST-segment depression, T-wave inversion and QTc prolongation); however, rare cases exist without any ECG changes.
5. Levels of cardiac biomarkers (troponin and creatine kinase) are moderately elevated in most cases; significant elevation of brain natriuretic peptide is common.
6. Significant coronary artery disease is not a contradiction in takotsubo syndrome.
7. Patients have no evidence of infectious myocarditis.
8. Postmenopausal women are predominantly affected.

Table 2: *International Takotsubo (InterTAK) diagnostic criteria [13].*

Hospitalisation in cardiology departments is necessary for Takotsubo cardiomyopathy. The course of treatment is mostly supportive and lasts until the left ventricle spontaneously returns, which typically happens 21 days after the disease first appears. On the other hand, individuals with severe instances can need intensive medical care and, if the illness is resistant, left ventricular mechanical support [14].

The first step is to determine the patient's haemodynamic stability. Haemodynamically stable patients are managed for systolic heart failure. In the event of pulmonary congestion, diuretics and vasodilators (such as nitroglycerin, nitroprusside or nesiritide) may be used. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs) and beta-blockers are often used to reduce cardiac workload and control hypertension when it exists. In some cases, aldosterone receptor antagonists, such as spironolactone, may also be beneficial. As aldosterone might potentiate the effects of catecholamines on the cardiovascular system by further raising systemic BP, the use of an aldosterone antagonist may be particularly cardioprotective in patients with Takotsubo cardiomyopathy [15].

Patients with hypotension and low cardiac output (cardiogenic shock) can be divided into those with left ventricular outflow tract obstruction (LVOTO) and those without. In patients without LVOTO, inotropes such as milrinone, dobutamine and dopamine can be used. Vasopressors and left ventricular assist devices may be necessary in refractory cases [14].

Inotropes should not be administered to patients with LVOT, as they increase basal hypercontractility and may worsen obstruction. Beta-blockers, which reduce basal contractility, and IV fluids must be used instead. Vasopressors can be applied in patients with LVOTO. During the first few weeks after the onset of Takotsubo cardiomyopathy, patients should be seen frequently by a cardiologist using serial echocardiography to assess their recovery.

Patients with significant left ventricular systolic failure should be evaluated for the possibility of intraventricular thrombus development and systemic embolization. While 1-2% of individuals with TTS may experience complications due to left ventricular thrombus, routine preventive anticoagulation is not advised [16].

Overall, the prognosis for Takotsubo cardiomyopathy is good. Around 95% of patients recover full cardiac function within a few weeks. Current studies estimate in-hospital mortality at around 5%, which is higher than previously thought, and most deaths occur in patients with haemodynamic instability [17].

Conclusion

In conclusion, Takotsubo cardiomyopathy, often triggered by emotional or physical stress, presents a unique challenge in clinical diagnosis due to its similarity to acute myocardial infarction. The condition predominantly affects postmenopausal women, suggesting a potential link to hormonal changes, although other mechanisms are also implicated. The prognosis for patients is generally favorable, with a significant majority recovering full cardiac function within weeks. However, awareness of the condition is crucial, especially in high-stress medical situations such as cesarean sections, where the risk of stress-induced cardiomyopathy may be heightened. Continued research and clinical vigilance are essential to improve diagnostic accuracy and treatment strategies, ensuring optimal outcomes for affected patients.

Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

All authors contributed to the conduct of this work. All authors also declare that they have read and approved the final version of the manuscript.

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