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Stress-Induced Cardiomyopathy: A Case of Takotsubo Cardiomyopathy and Review of Literature

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Abstract:- Takotsubo syndrome is an acute cardiomyopathy with a clinical presentation similar to acute coronary syndrome, characterized by reversible alterations in ventricular function, leading to complications of acute heart failure in the absence of underlying coronary artery disease. Its triggering pathophysiology is unknown and there is no consensus regarding its treatment. A clinical case of unusual characteristics is presented, which debuted in cardiorespiratory arrest secondary to polymorphic ventricular tachycardia, with rapid return to spontaneous circulation, and good response to medical management.

Keywords:- *Takotsubo, Broken Heart Syndrome, Stress-Induced Cardiomyopathy, Cardiomyopathy.*

I. INTRODUCCIÓN

Takotsubo syndrome is an acute cardiomyopathy with a clinical presentation similar to acute coronary syndrome [1]. This heart disease was first described in Japan in the 1990s [2]. it is a rare condition, accounting for approximately 1% of all patients with chest pain and suspected acute coronary syndrome [3]. it mainly affects postmenopausal women, with an associated episode of severe emotional or physical stress. Its presentation as chest pain with anginal features, electrocardiographic changes and elevation of cardiac enzymes make early diagnosis difficult [1,3], until the absence of coronary obstruction is found on angiography and the characteristic alterations in myocardial contractility of the left ventricle, generally reversible [2]. The etiology of this syndrome is not completely defined; it has been associated with an exaggerated discharge of sympathetic activity (catecholamine cardiotoxicity), leading to coronary spasm and microvascular dysfunction [4]. It has typically been considered a benign pathology; its management is based on the control of peripheral sympathetic activity with beta-blockers [16].

II. CASE PRESENTATION

A 66-year-old female patient with a history of dyslipidemia under management with atorvastatin and apparently episodes of nonsustained ventricular tachycardia for 30 years that is not being managed at the moment, with no other cardiovascular risk factors, who comes to the emergency department for an episode of oppressive chest pain of approximately 30 minutes duration, which occurs during the funeral of her sister. The patient was admitted to the emergency department for an episode of oppressive chest pain lasting approximately 30 minutes, which occurred during her sister's funeral. On admission to our institution, she presented cardiovascular collapse with witnessed cardiorespiratory arrest, arrest rhythm monomorphic ventricular fibrillation, high quality resuscitation was initiated and defibrillation with 200J was administered on 2 occasions, and she returned to spontaneous circulation with resuscitation maneuvers.

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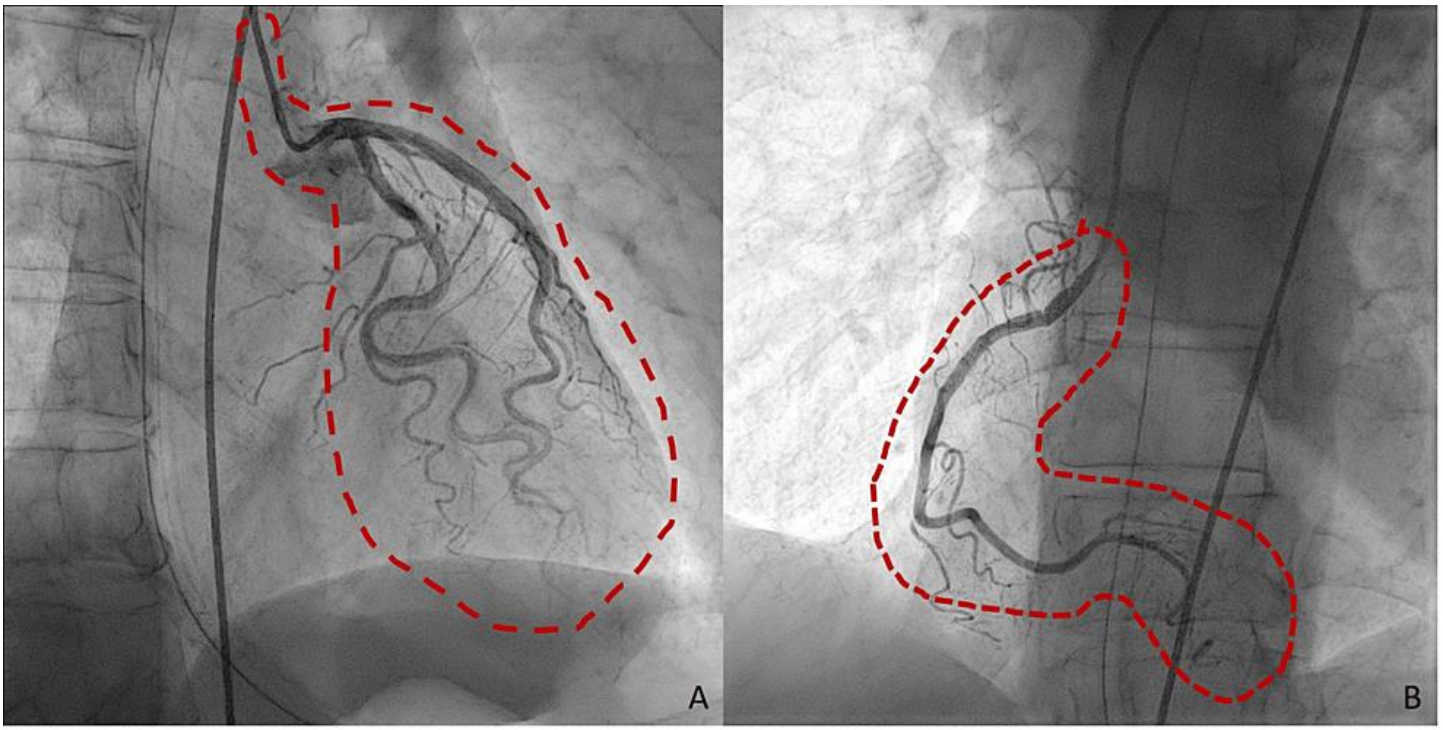

Fig 1: Angiographic evaluation A. Left Circulation B. Right Circulation

➢ *Coronary arteries without evidence of occlusion*

The post-resuscitation electrocardiogram shows alterations in the ST segment and J point given by low level in anterior and lateral face, associated with positive cardiac enzymes (troponin i (HS): 42.9ng / l range: 0.0 - 11. 6), is immediately taken to coronary angiography, ruling out angiographically significant coronary artery lesions (Figure 1); Transthoracic echocardiogram shows severe compromise of systolic function, with severely decreased ejection fraction (LVEF: 26%), also with alteration in segmental contractility given by akinesia of the middle and distal segment of the interventricular septum, akinesia of the middle and distal segment of the inferior, posterior, lateral and distal segment of the anterior wall with left ventricular ballooning, findings suggestive of Takotsubo's heart disease (Figure 2).

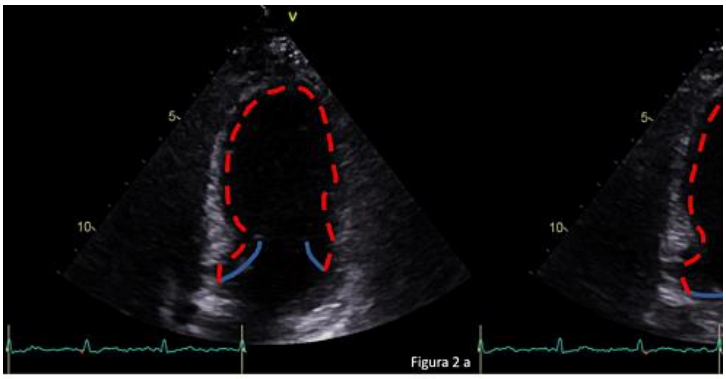

Fig 2: Echocardiographic evaluation of ventricular function on admission transthoracic echocardiogram (apical 4-chamber window) shows evidence of left ventricular ballooning.

- End diastole
- Isovolumetric contraction
- End systole

During his stay in the Intensive Care Unit, complete anticoagulation was started with low molecular weight heparin (1mg/kg/BID), statins in intensive doses, angiotensin converting enzyme inhibitor + beta-blocker in low doses (enalapril 2.5mg/BID + metoprolol 12.5 mg/day). However, with the onset of hemodynamic instability, ACE inhibitors and beta-blockers were suspended, requiring vasopressor support with low-dose noradrenaline and inotropy with levosimendan; with early withdrawal less than 24 hours later, also meeting clinical and gasometric criteria for controlled awakening, with adequate tolerance to weaning from mechanical ventilation, so she was extubated early. She was considered a candidate for implanted cardiodesfibrillator, as a strategy for secondary prevention of sudden death; with good evolution and adequate clinical status, she was discharged after four days.

Stress cardiomyopathy, also called takotsubo cardiomyopathy/syndrome, apical balloon syndrome or broken heart syndrome, is a syndrome characterized by transient and reversible regional systolic dysfunction, mainly affecting the apical area of the left ventricle, causing the ventricle to dilate, leading to transient and completely reversible heart failure in most cases (<21 days). This contractile compromise, which is not explained by an ischemic lesion, as it characteristically affects more than one coronary vascular territory (epicardial coronary arteries) [1,2], was first described by Hikaru Sato in 1990 in Japan, with the name "takotsubo" comes from the

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Japanese term for an octopus fishing trap, which has a shape similar to the apical systolic balloon of the dilated left ventricle, which is the most frequent and typical form of presentation of this disorder [5]. It accounts for 1 - 2% of patients with chest pain [1,4,6], affects mainly women, with an approximate female: male ratio of 9:1, usually postmenopausal women between 55 and 65 years of age, and in up to 70% of cases a stressful event (physical or emotional) is identified [2,3].

Takotsubo cardiomyopathy is classified into four main variants according to the anatomical distribution of the contractility alterations: apical, basal, mid-ventricular and focal [7,8,9].

- Apical type: This is the classic form that represents 80% of cases, characterized by basal hyperkinesia, apical ballooning and hypokinesia of the apical segments, which may extend to the midventricular segments. It is associated with further complications, mainly mechanical, such as left ventricular outflow tract (LVOT) obstruction and mitral valve insufficiency [8,10].
- Basal type: Characterized by basal hypokinesia and apical hypercontractility, it has been found to be associated with subarachnoid hemorrhage and pheochromocytoma [11].
- Midventricular type: characterized by hyperkinesia of the apical segment and dyskinesia of the bulging midventricular region, giving a hawk beak appearance on the left ventriculogram, this variant was first described by Roncalli et al. in 2007 [8,12,13].
- Focal type: characterized by isolated dysfunction of the anterolateral segment of the left ventricle [8].

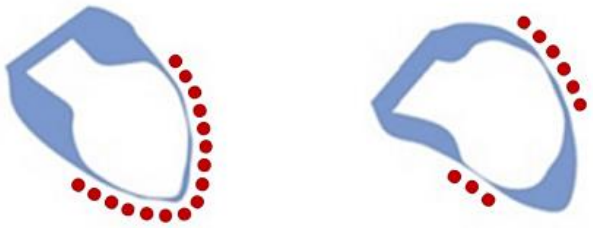

Fig 3: Graphical representation Anatomical Variants

The apical variant is considered as the typical Takotsubo syndrome, and the basal, midventricular and focal variants as atypical forms, mainly related to young patients, less compromised ejection fraction, electrocardiographic changes mainly ST depression and lower mortality [8,9], however, there seems to be no difference in terms of short and long-term mortality between the different anatomical patterns [9].

III. PATHOPHYSIOLOGY

The causal mechanism is not clearly defined; it has been associated with catecholamine excess (catecholamine toxicity), microvascular dysfunction, and coronary spasm [2].

Catecholamines released in large quantities (catecholamine toxicity) induced by physical or emotional stress can cause diffuse and transient microvascular spasm directly compromising coronary flow [1,8], in addition to generating direct cardiotoxicity on cardiomyocytes. Excess catecholamines induce intracellular calcium overload, inhibiting the expression of SERCA2a (sarcoplasmic-Ca2+-adenosinetriphosphatase), which generates a rapid increase in the phospholambam/SERCA2a ratio, causing a decrease in calcium affinity and resulting in alterations of myocardial contractility, reaching a state similar to post-ischemic myocardial stunning [1,5,6] [1,5,6].

IV. DIAGNOSIS

The clinical picture is similar to that of an acute coronary syndrome, the cardinal symptom is usually chest pain with anginal features, preceded by an episode of intense emotional or physical stress, which is identified in more than 70% of patients [1,2] . In a systematic review of 19 studies and 1109 of 2015 Pelliccia F., et al, found that in approximately 13% of patients it is not possible to determine the triggering event [3], and that patients with psychiatric or neurological disorders, mainly intracranial hemorrhage, are more predisposed to develop stress cardiomyopathy [2,3].

The most common presenting symptom is chest pain, but it may present with other anginal equivalents such as dyspnea or syncope [1,10], Pelliccia F., et al, found that more than half of the patients presented chest pain as the cardinal/initial symptom (55%), followed by dyspnea (26%), as well as more than half of the patients presented ST alterations (53%) mainly in the anterior leads [3], other less frequent electrocardiographic alterations have also been described, such as QT interval prolongation, T wave inversion, ventricular tachycardia (VT), ventricular fibrillation (VF) and torsade de pointes [2]. Gili S, et al, performed a subgroup analysis of the InterTAK Registry, finding that of the 2098 patients, 84 presented as cardiac arrest, of which 44% were found as ventricular fibrillation in arrest rhythm (37 cases), 42, 9% pulseless electrical activity (36 cases) and ventricular tachycardia in only 13.1% of cases (11 patients) [11,14], these life-threatening arrhythmias occur early in patients with Takotsubo syndrome (first 24 hours), are associated with a significant worsening prognosis in the short and long term. Jesel, et al. in a cohort of 214 patients collected over 8 years, identified decreased left ventricular ejection fraction (LVEF) and conduction disturbances (QRS >105ms) as independent predictors for the development of life-threatening arrhythmias, with a significant increase in both factors. in-hospital and 1-year mortality (39.1 % vs 8.9 %; $P < 0.001$ and 47.8 % vs 14.1 %; $P < 0.001$ respectively) [12].

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Elevation of troponin is also characteristic, as are other markers of myocardial injury such as natriuretic peptides (BNP and Pro-BNP) (Table 1) [1,3]; Wittstein et al. found that plasma levels of catecholamines were two to three times higher in patients with Takotsubo cardiomyopathy than in patients with myocardial infarction [4,10].

Table 1: Biomarkers in Takotsubo syndrome

Biomarker	Troponin	BNP	NT-proBNP
Takotsubo Cardiomyopathy	↑	↑↑	↑↑
Acute Myocardial Infarction	↑↑	↑	↑

The diagnosis of this entity is to rule out an acute coronary syndrome of the myocardial infarction type, for which the Mayo Clinic Diagnostic Criteria were proposed to facilitate the approach [3,7,14].

- Transient left ventricular dysfunction: temporary hypokinesia, dyskinesia or akinesia in LV segments with or without apical involvement, involving more than a single vascular distribution.
- Absence of significant obstructive coronary artery disease on arteriography.
- Recent changes detected on electrocardiogram (ECG) (ST-segment elevation and/or T-wave inversion) or significant elevation of serum cardiac troponins.
- Absence of pheochromocytoma or myocarditis.

The InterTAK Diagnostic Scale, is a score composed of 7 variables (Table 2) all parameters are easily obtained in the emergency department and no diagnostic imaging is needed to complete it, which estimates the probability of presenting Takotsubo syndrome, to try to differentiate it from acute coronary syndrome, its main differential diagnosis. In 2016 Jelena R, et al validated the score with a derivation cohort (Takotsubo, n = 218; acute coronary syndrome, n = 436), recruited from the International Takotsubo Registry (www.takotsubo-registry.com) and from a leading hospital in Zurich [15]. An optimal value for the diagnosis of Takotsubo syndrome of ≥70 points was established, with a sensitivity of 89 % and a specificity of 91 %; with an area under the curve (AUC) of 0.971 [95 % confidence interval (CI): 0.96-0.98] [15].

Table 2: InterTAK Diagnostic Score [15]

Criteria	Points
Female Sex	25
Emotional Trigger	24
Physical Trigger	13
Absence of ST-segment Depression	12
Psychiatric Disorders	11
Neurologic Disorders	9
QTc Prolongation	6
	100

Clinical predictors takotsubo syndrome
*Absence of ST-segment depression, Except in lead aVR

However, significant coronary artery disease may coexist in a high proportion in patients with stress cardiomyopathy, Napp LC, et al in a study that collected a total of 1016 patients with Takotsubo syndrome, defined according to the InterTAK Diagnostic Criteria, found that 23.0 % had concomitant coronary artery disease with significant obstruction, of which 47 patients required percutaneous coronary intervention, an additional 41.2% had non-obstructive coronary artery disease and only 35.7% had angiographically normal coronary arteries [6], so the diagnosis of stress cardiomyopathy requires serial evaluation of ventricular systolic function to define the reversibility of the changes presented.

V. TREATMENT

It has been considered a benign pathology, so treatment is generally conservative, with supportive therapy, seeking to treat symptoms, resolve physical or emotional stress, prevent the development of complications and reduce recurrence. However, no guidelines have been established for its management and therapeutic strategies are based on clinical experience and expert consensus.

➢ *Beta-blockers*

Beta-blockers such as carvedilol [14,16] have been proposed to control the myocardial response to catecholamine excess and also to reduce the incidence of arrhythmias; However, their use should be cautious, since their use is contraindicated in patients with suspected coronary spasm, very low LVEF, hypotension, bradycardia or QTc > 500 ms, because of the possibility of worsening LVOT and leading to cardiogenic shock, so they should be started with caution and gradually increased according to clinical response [17]. It is not possible to give a clear recommendation on the use of beta-blockers; trials are needed to support this hypothesis.

Angiotensin-converting enzyme inhibitors (ACEi) or angiotensin II receptor blockers (ARB-II). Segmental contractility disorders usually resolve spontaneously, ACEi/AR-II have been proposed to counteract persistent activation of the renin-angiotensin-aldosterone system, decreasing afterload, limiting cardiac remodeling and limiting recurrence [10,16,17].

The most frequent complication of Takotsubo cardiomyopathy is acute heart failure and cardiogenic shock, which occurs in approximately 20% of patients. In general, management is according to standard guidelines for heart failure with reduced ejection fraction (HFrEF), controlling preload and reducing afterload [8], careful water resuscitation should be initiated, early initiation of non-adrenergic inotropic drugs such as levosimendan should be started. considered [18,19], limiting the use of beta-blockers [14,18].

Left ventricular outflow tract (LVOT) obstruction occurs when the base of the heart contracts forcefully, making blood flow in the aortic outflow tract very turbulent, causing mitral insufficiency due to involvement mainly of the anterior leaflet. This phenomenon occurs in approximately 10% of patients, most frequently in the classic (apical) variant of Takotsubo cardiomyopathy [20].

Early recognition of OTVI is important, as these patients respond differently to treatments [20]:

- afterload reduction and diuretics may worsen OVTI
- Inotropic agents may exacerbate OVTI.

VI. PROGNOSIS

In general, the prognosis of Takotsubo cardiomyopathy is good. About 95 % of patients fully recover LV function, may begin to recover within several days, and recover completely within 3 to 4 weeks [8], seems to depend, at least in part, on the triggering factor.

Patients with Takotsubo syndrome are at high risk for cerebrovascular events mainly during the first 30 days, so it is preferred to initiate prophylactic anticoagulation in high-risk patients, such as the presence of a large area of myocardial hypokinesia mainly with apical involvement [8].

VII. DISCUSSION

The reported case presented clinical features that are considered atypical in Takotsubo syndrome. The electrocardiographic, kinetic and myocardial conduction alterations found in the patient of the case represent a low incidence in the literature reports, and its rapid temporal evolution led to a near-fatal complication. the debut of the pathology that led to cardiorespiratory arrest was of rapid evolution, with approximately 30 minutes of symptomatology prior to admission to the emergency department, with a more than favorable response to resuscitation maneuvers and electrical therapy, the early presence of a malignant arrhythmia that led to cardiorespiratory arrest is unusual, El-Battrawy et al. found that more than half of the patients with Takotsubo syndrome who presented arrhythmias presented cardiac arrest [13].

The electrocardiographic findings in the acute phase, mainly persistent ST-segment inversion in the inferior aspect of the heart, associated with troponin elevation, suggested a coronary occlusive compromise, which was ruled out by coronary angiography. In addition, the patient's echocardiography showed akinesia of the middle and distal segment of all left ventricular walls, with apical ventricular ballooning, configuring a typical Takotsubo syndrome; which is not concordant with InterTak score of 49 points (woman + emotional stress), the patient had an intermediate/low probability of presenting this syndrome.

The presence of ventricular arrhythmias in patients with Takotsubo syndrome represents a negative prognostic factor that increases in-hospital morbidity and the risk of sudden cardiac death [20], there is no clear recommendation regarding the treatment of electrical complications in patients with Takotsubo syndrome, since it is considered a reversible pathology, ideally drugs that prolong QTc should be avoided, since it has been documented that a corrected QT interval >500 ms or a QRS >105ms [12,20], are an arrhythmogenic risk factor for developing ventricular tachycardia; in our patient neither of these 2 characteristics was documented during observation. In addition, strict monitoring of beta-blockers and renin-angiotensin-aldosterone system inhibitors is necessary to avoid bradycardia and hypotension [16,18].

The implantation of permanent electrical devices in patients with Takotsubo syndrome is a controversial issue, without finding a clear recommendation in ventricular arrhythmias, in general, the possibility of recurrence should be considered as the determining factor for the implantation of these devices. In the case presented, the debut with monomorphic ventricular tachycardia leading to cardiac arrest, the severity of the arrhythmia, the good functionality and high life expectancy of the patient led the treating group to consider implanting a cardio defibrillator.

VIII. CONCLUSIONS

Takotsubo syndrome is a transient and reversible cardiomyopathy, generally with a good prognosis. It is characterized by apical ballooning of the left ventricle, associated with a recent episode of physical or psychological stress, which is not identifiable in all cases. Catecholamine levels have been shown to play a vital role in the pathogenesis and pathophysiology, inducing diffuse vasospasm and microvascular dysfunction secondary to the adrenergic storm generated. Due to its clinical features, EKG changes and increased cardiac biomarkers, its presentation is very similar to acute myocardial infarction, so this should be its main

differential diagnosis and a careful diagnostic approach should be performed, characteristically finding healthy coronary arteries on angiographic evaluation. The main therapy is supportive treatment and is reported to be effective as ventricular function usually begins to be restored within several days and recovers completely in 3 to 4 weeks; the search for acute complications such as left ventricular outflow tract obstruction or valvular compromise, mainly mitral, is essential before initiating treatment, especially in patients with hemodynamic instability.

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