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EMERGENCY STAY TRIGGERED TAKO-TSUBO SYNDROME A CASE REPORT AND REVIEW OF THE LITERATURE

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ABSTRACT: Tako-tsubo cardiomyopathy is an increasingly recognized as clinical syndrome characterized by transient apical left ventricular dysfunction. An episode of intense emotional or physical stress is reported in many cases prior to presentation. The pathophysiology remains unknown, but high levels of circulating catecholamine are presumed to be the triggering factor and the most favored explanation for this primary acquired cardiomyopathy. We describe the case of a 48-year-old woman presenting Takotsubo cardiomyopathy (CTM), triggered 4 hours later during her stay in the emergency department to manage a hypertensive emergency.

Keywords: Apical ballooning syndrome; Diagnostic criteria; Myocardial infarction; Myocarditis; Tako-tsubo cardiomyopathy.

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1.INTRODUCTION

Transient apical ballooning of the left ventricle Tako-tsubo [TTS] syndrome or broken heart syndrome[1][2], is an acute cardiomyopathy associated with reversible kinetic disorders. These disorders mainly affect the middle and apical segments of the left ventricle in the absence of significant lesions of the coronary arteries. The clinical appearance is similar myocardial infarction with a small increase in cardiac enzymes, which discord with the extent of left ventricular kinetic disorders, a crucial diagnostic criterion. This syndrome affects the menopausal woman often as a result of intense physical-emotional stress[3][2].

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2.PATIENT AND OBSERVATION:

Mrs. K, 48 years old, with known history of high blood pressure for a year under Angiotensin II Receptor Antagonists (ARA-II), initially admitted to the emergency for the management of a pressure blood peak (blood pressure at 250/130 mmHg), requiring a stay in the waste room of the department, Four hours later, the patient accused acute chest pain and an electrocardiogram (ECG) revealed repolarization disorders, the patient was conscious, eupneic, the blood pressure was 150/80 mmhg on the right arm and 110 / 90 mmHg on the left arm. The ECG shows sinus rhythm at 91 bpm with a suspended ST segment in the anterior and T waves loose, sharp in inferior, and negative in lateral high. In front of the asymmetry of blood pressure, a thoracic angioscan have been realized, eliminating an aortic dissection. Blood tests showed elevated concentrations of troponin I (1404 ng/l ; normal < 26ng/l). Transthoracic echocardiography (TTE) revealed extensive apical akinesis, and apical segments of all walls, with moderate LV dysfunction EF = 43% [figure 2]. Emergency coronary angiography showed an angiographically normal coronary arteries, cardiac MRI [figure 1] was performed, five days later, established the diagnosis of Tako-tsubo syndrome, with no contrast enhancement on late enhancement sequences, and no sequelae of myocarditis and myocardial necrosis. Medical treatment of heart failure was initiated. The patient was seen in control consultation, one month after, the cardiac check was quite reassuring, so it was asymptomatic with normalization of the electrocardiogram and the trans echocardiographic[Figure 3] showed an absence of segmental hypo/ akinesia and recovery of left ventricular ejection fraction (LVEF).

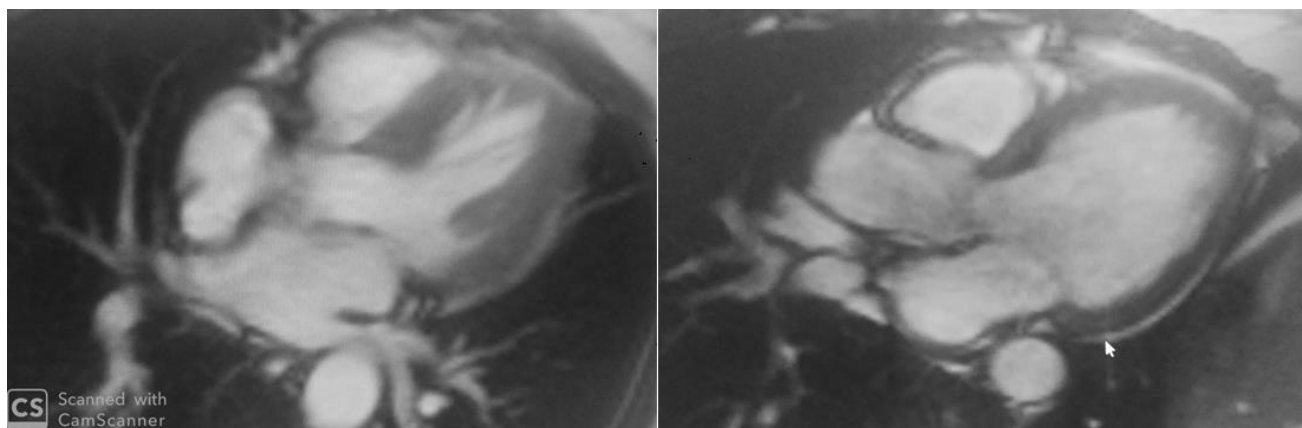


Figure 1: Cardiac MRI showing absence of late enhancement after gadolinium injection

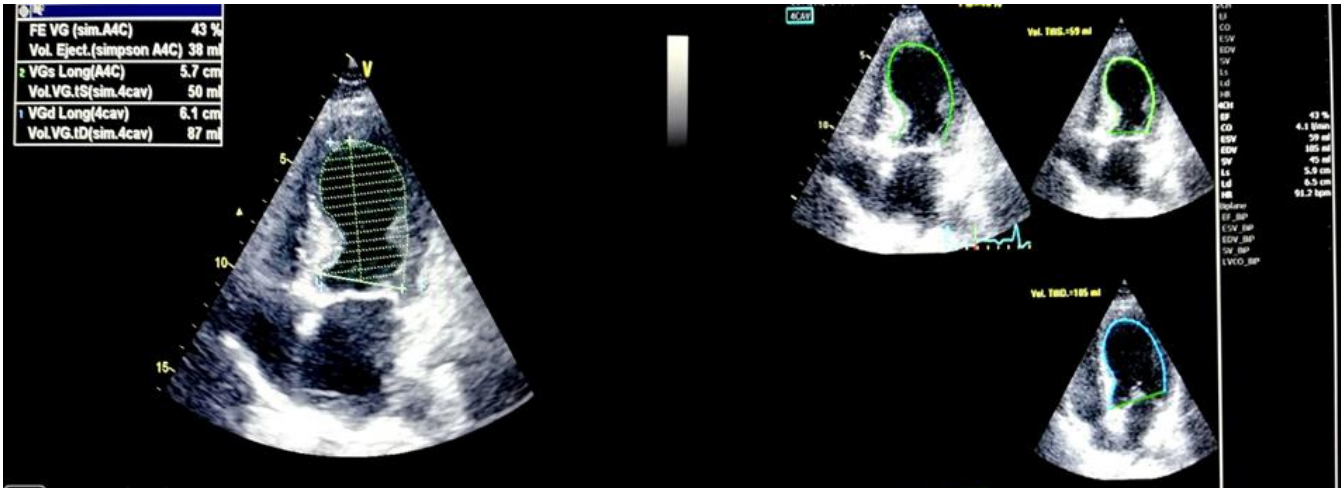


Figure 2: VG dysfunction in TTE

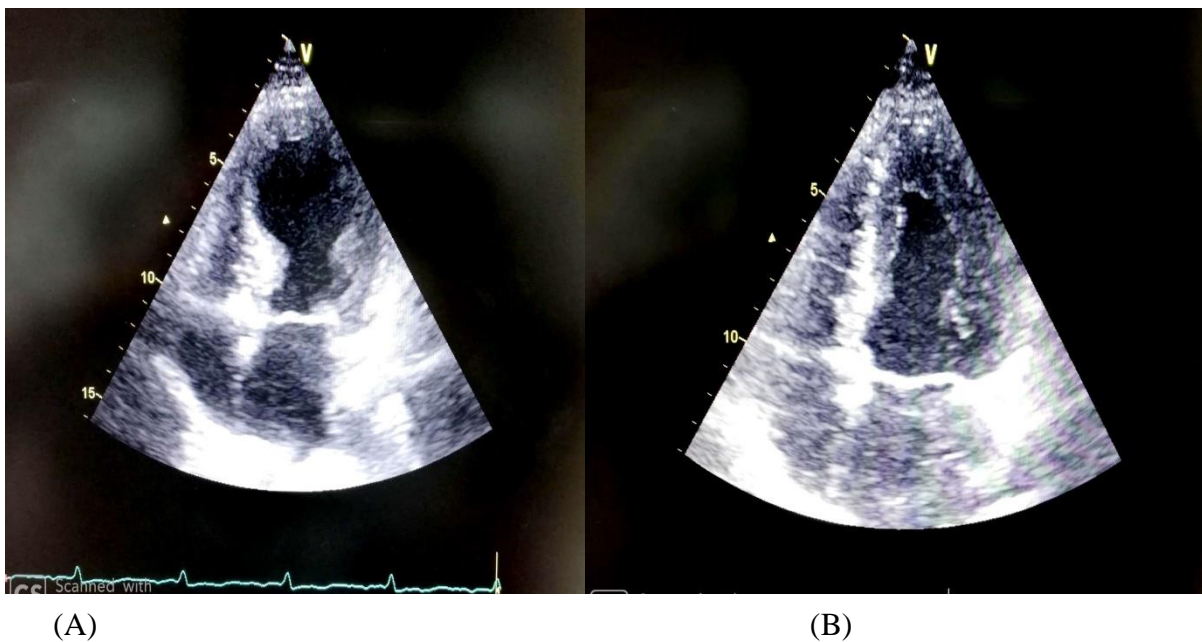


Figure 3: (A): apical ballooning syndrome visible in 2D echocardiography of our patient (B): recuperation after one month: apical ballooning disappearance

3.DISCUSSION

Tako-tsubo syndrome (TTS) is a pathology that has been defined by the Mayo Clinic criteria in 2004, modified in 2007[4][5]. Thus, the diagnosis of Tako-tsubo syndrome can only be made in the presence of specific angiographic criteria:

- Transient appearance of hypokinesia, dyskinesia or apical akinesia of the left ventricle with or without medial segmentation at ventriculography.
- Regional abnormalities of the left ventricular wall not systematized to a coronal territory.
- Lack of obstructive coronary disease or angiographic signs of acute rupture of athermanous plaque.

They will have to be supplemented by anamnestic criteria (triggers linked to the stress sometimes

absent), electrocardiographic (recent onset of ST segment elevation and / or T wave inversion) and biological (absence or moderate elevation of troponin). Finally, it is necessary to eliminate coronary heart disease, pheochromocytoma, acute viral myocarditis or acute cerebrovascular disease.

It was described in Japan in 1990(3) and named "takotsubo" because of the angiographic shape taken by the left ventricle resembling an octopus trap used by Japanese fishermen (tako / octopus, tsubo / pot).

Since then, numerous series have been published both in Japan [6][7] and in Western countries [8][9] and found under several names: tako-tsubo, transient apical ballooning syndrome of the left ventricle, stress heart disease . The ground is that of the menopausal woman. In all series, there is a clear female predominance that varies between 81 [6] and 100%[8]. The average age is high. He was 66 ± 15 years old in the Liepiecki et al. [8] and 71 ± 11 years in the Bybee et al. [4]. We often find stress triggering this cardiomyopathy. In the series of Tsuchihashi et al. [6] (n = 88), a possible trigger factor is found in 65% of cases, most commonly surgery. In Western series, the share of emotional stress seems more important. Thus, the triggering factors of the Wittstein et al. [10]and Sharkey et al. [11] are exclusively of this order (Table 1).

Table 1: Triggers of takotsubo syndrome

Emotion	physical stress
Death or serious illness of a relative	Extra cardiaq surgery
Financial loss	Cholecystectouemy
Divorced	Hysterectomy
Dispute	Pose pacemaker
Anger	Asthma attack, pneumothorax
Opiate withdrawal	Public speaking intense pain : fracture, renal
Jobb loss	colic
Move	Taking cocain
Flight	Stress ultrasound with dobutamine
Car accident	

In our case, the triggering factor was probably the stress of stay in the emergency department responsible for an elevation of catecholamines which is incriminated in the occurrence of this pathology. Several physiopathological hypotheses have been proposed in recent years

[6][10][12][13]. currently the preferred pathophysiological hypothesis is the catecholergic discharge following acute stress, [10][14][13]. It has been shown that emotional stress from increased catecholamine levels can sometimes result in impaired left ventricular ejection fraction [15]. Pheochromocytoma and subarachnoid hemorrhage may also cause abnormalities of left ventricular contractility similar to the disorders found in TakoTsubo cardiomyopathy (TTC), and the pathophysiological mechanism of this left ventricular dysfunction is clearly the major elevation of catecholamines or intense sympathetic stimulation [16]. In Tako-Tsubo cardiomyopathy, catecholamine levels are also significantly higher in the acute phase compared to those found in patients with myocardial infarction[10] and endomyocardial biopsies were found in patients with cardiomyopathy. Stress stigmas of toxicity of catecholamines. Coronary spasms, including coronary microcirculation, have also been suggested. The spastic hypothesis of large coronary trunks was quickly ruled out, where as the first Japanese studies initially favored this hypothesis (spasm caused only in 14% of cases) [6]. Anomalies of microcirculation have also been reported during the acute phase of Tako-tsubo cardiomyopathy (TTC)[17]. However, these perfusion abnormalities are probably a consequence of myocardial related to catecholergic discharges. Finally, an infectious origin (myocarditis) has been mentioned because left ventricular dysfunction does not correspond to a systemized territory of a coronary artery. However, the anatomopathological and magnetic resonance study of Takotsubo cardiomyopathy (TTC) did not find any signs of myocarditis, making it possible to eliminate this physiopathological hypothesis[18] [12][19]. Electrical appearance of the tako-tsubo cardiomyopathy is that of acute coronary syndrome, ECG objective an elevation of ST segment typically in the precordial, a wave inversion Sometimes a long QT can occur. Transthoracic echocardiography [figure 2] is also important, especially in the case of an unresolved intensive care patient in cardiology to assess left ventricular systolic function and to look for a possible complication [table 2]. The use of new techniques such as longitudinal myocardial deformation tools (strain) and contrast agents to opacify left cardiac cavities improves endocardial contour analysis segmental kinetics analysis. Ventriculography also finds kinetic disorders with apical balloon appearance and calculates the ejection fraction. Finally, echocardiography allows monitoring of the left ventricular ejection fraction and thus ensure the recovery of this cardiomyopathy.

Table 2: The different ultrasound aspects

<p>Characteristic Form (70%):</p> <p>Achievement of the more average apical portions of all the walls with normo or hyperkinesia of the basal segments.</p> <p>Appearance of apical ballooning</p>
<p>Median form (25%):</p> <p>Achievement of the medial segments with respect to the apex and the basal collar.</p>
<p>Inverted form:</p> <p>Akinesia of the basal segments.</p> <p>Difficult ultrasound diagnosis</p>

Magnetic resonance imaging (MRI)[figure 1] is becoming increasingly important in the exploration of cardiomyopathies and thus very useful in Tako-Tsubo cardiomyopathy. MRI perfectly characterizes left ventricular systolic dysfunction and ensure the absence of late enhancement after gadolinium injection. This makes it possible to differentiate between Takotsubo cardiomyopathy (TTC) and ischemic heart disease, or even myocarditis. The true limit of magnetic resonance imaging is its poor availability, with most often the completion of the examination several days after the acute phase. Serious complications can occur in the acute phase and are mainly: cardiogenic shock, heart failure, mitral insufficiency, left intraventricular gradient, associated right ventricular dysfunction, left intraventricular thrombus formation (with its complications thromboembolic) and exceptionally left ventricular arrhythmias [18][9]. In the acute phase, the management of Tako-tsubo cardiomyopathy (TTC) should be from acute coronary syndrome to removal of coronary lesions from coronary angiography. Once the diagnosis of Tako-tsubo cardiomyopathy is made, there is currently no formal recommendation. However, because of this left ventricular dysfunction, the prescription of the converting enzyme inhibitor and beta-blocker is advocated, at least until the recovery of the LVEF. In cases of cardiogenic shock, the intra-aortic counter-pulse balloon may be used, preferentially than pressurized amines, which may further aggravate a left intraventricular gradient, If he is present. The prescription of long-term beta-blockers is not currently justified, as no study has demonstrated its usefulness, in order to reduce the risk of recurrence. Finally, it is important to make the diagnosis of Tako-Tsubo cardiomyopathy, because the similar clinical presentation of an acute coronary syndrome, the diagnosis is not easy made, it may delay diagnosis and must be known in order to provide the best chance at early detection and avoid a long-term treatment of ischemic heart disease. Thus, the patient will be exposed to a risk of unjustified haemorrhage, given the prescription of dual antiplatelet therapy for one year.

4. CONCLUSION

Tako-Tsubo cardiomyopathy is a new cardiomyopathy initially described in 2001. It is defined by reversible characteristic left ventricular dysfunction and usually presents as an acute coronary syndrome. The diagnosis is based on a set of arguments (clinical, electrocardiogram, biomarkers, echocardiography, coronarography, and ventriculography MRI). This diagnosis is most often retrospective and exclusion. Differential diagnosis with angiographically healthy coronary infarction and acute segmental myocarditis is sometimes difficult. It is also essential to ensure the recovery of contractile function to confirm this diagnosis. Initial management should be that of an acute coronary syndrome. Our case shows that, in addition to the precipitating factors cited in the literature, long stay stress in emergencies can be responsible for this condition, that's why it's so important to Reduce Wait Time in the emergency as much as possible.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

HUMAN AND ANIMAL RIGHTS

No Animals/Humans were used for studies that are base of this research.

CONSENT FOR PUBLICATION

Not applicable.

AVAILABILITY OF DATA AND MATERIALS

The authors confirm that the data supporting the findings of this research are available within the article.

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CONFLICT OF INTEREST

The authors report no relationships that could be construed as a conflict of interest.

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