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Case Report

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Takotsubo Cardiomyopathy in Postpartum of a Uterine Rupture

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Introduction

Takotsubo cardiomyopathy is a heart condition associated with the acute onset of chest pain, abnormalities in heart enzymes and the EKG, and a distinct pattern of left ventricular dysfunction on echocardiography. We are reporting the case of a parturient diagnosed with Takotsubo cardiomyopathy or Broken Heart Syndrome at D2 postpartum of a cesarean delivery in a context of Uterine Rupture.

Case report

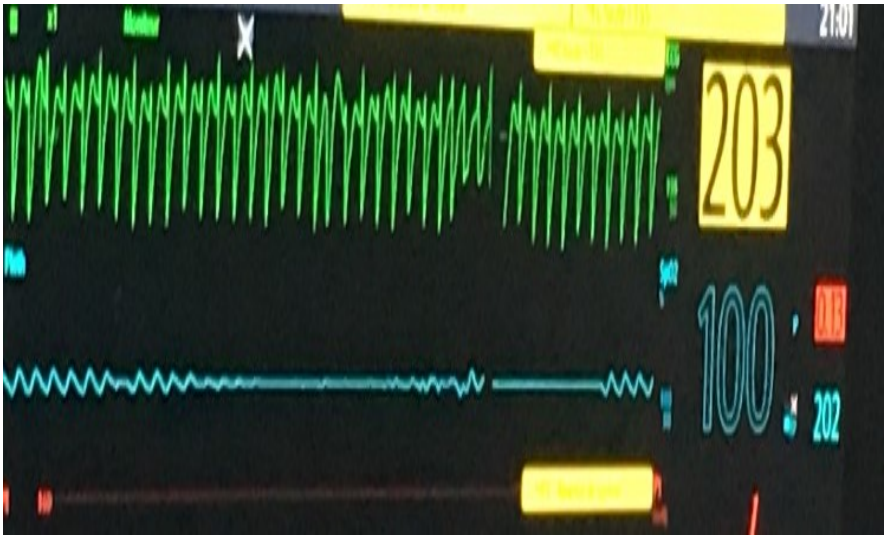
A 46-year-old patient, 6G6P [6 infants living vaginally], without cardiovascular risk factors, with no particular pathological history other than myomectomy in 2020.

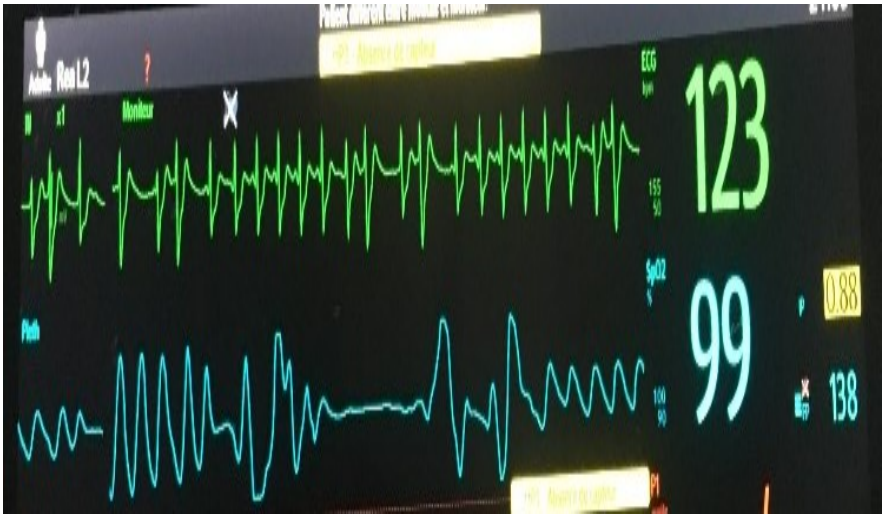
Admitted to the maternity ward, address of a CHP for childbirth in a non-reassuring fetal state associated with severe anemia Hb: 6g / dl.

The patient on admission was conscious 15/15, tachycardium at 120 bpm, BP: 100/70, Polypnea at 25cpm. Gynecologic obstetric examination uterus was relaxed, no bleeding, cervix dilated to 1cm, Fetal cardiac activity present with severe fetal bradycardia at 42 bpm. The patient was rushed to the operating theater for fetal extraction by cesarean section under spinal anesthesia, giving birth to a newborn male, apgar 0/10.

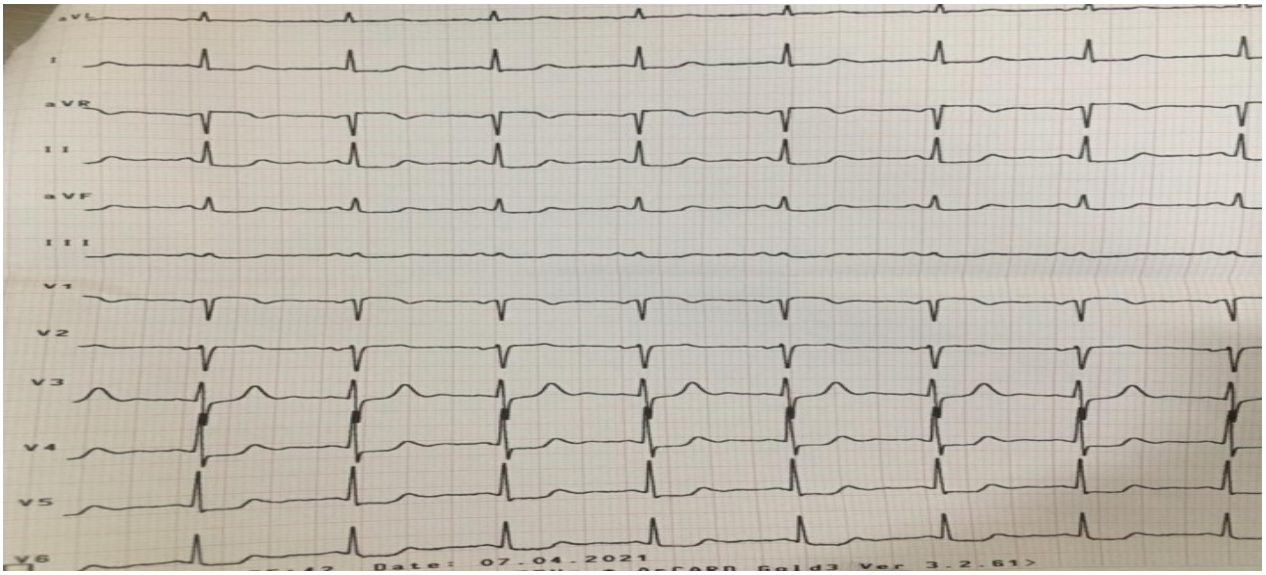
On exploration, a posterior fundic rupture of 4cm was found associated with a hemoperitoneum of great abundance. In addition, the patient presented intraoperative hemodynamic instability necessitating recourse to vasopressors [Noradrenaline at 0.5 mg / h], Intraoperative transfusion of 2 corpuscles [HB at 9.9 g / dl Post transfusion], then the patient was admitted to intensive care for additional support.

On Postpartum D2, the patient presented with sudden onset chest pain radiating to the neck and left upper limb associated with arrhythmias caused by bigeminal extrasystole, and an attack of ventricular tachycardia.

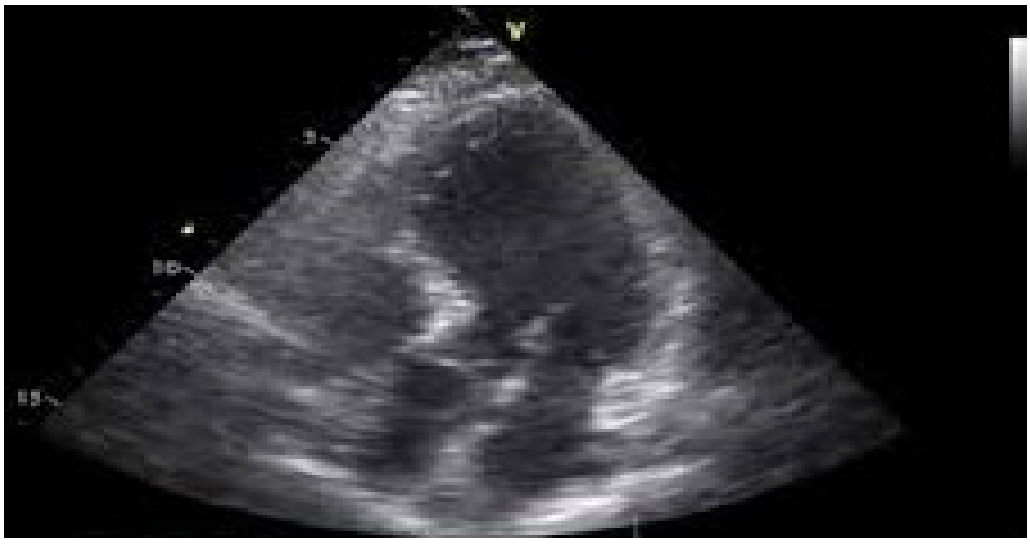




An ECG was carried out objectifying:
*ST-segment elevation in AVR
*NSTEMI in antero-septo-apical and inferior



Troponins very removed at 4585.90 ng / l
Transthoracic Heart Echo:
VG non-dilates, non-hypertrophy, site of hypokinesia of the Antero-septal and Infero-Septal walls. FE at 52%
Minimal IM
No aortic valve disease
VCI at 12mm



The Patient underwent coronary angiography for the suspicion of NSTEMI.

Coronary angiography returned to normal:

Common trunk is of normal length, without stenosis.

The anterior interventricular artery is medium in size and free from significant lesions in all three segments.

The circumflex artery is of normal size dominant without stenosis.

The right coronary artery is dominated small in size and free from any significant damage.

About two weeks later, the ECG returned to normal and the echocardiogram showed complete recovery of LV systolic and diastolic function.

Discussion

Takotsubo syndrome is an acquired cardiomyopathy with transient and reversible left ventricular dysfunction that can mimic acute coronary syndrome [1].

The term takotsubo (tako = octopus, tsubo = a pot) was introduced by Sato and Dote in 1990 and 1991 to describe the left ventricular silhouette during systole [2].

The etiology is linked to excessive exposure to plasma catecholamines mediated by exaggerated sympathetic stimulation during emotional (14-46%) or intense physical (17-70%) stress. Finally, the etiology remains unknown in some cases [3].

Its etiology and pathophysiology remain uncertain. The sympathetic system appears to play a central role: its exaggerated response to emotional or physical stress triggers can induce microvascular dysfunction and catecholamine-induced cardiotoxicity due to cyclic calcium overload mediated by AMP [1].

The diagnosis of TCM is based on the Mayo Clinic diagnostic criteria. This includes the presence of transient akinesia, hypokinesia or dyskinesia of the left ventricle with or without apical involvement; this regional defect in ventricular wall movement extends beyond a single epicardial vascular perfusion territory. Additionally, there is no obstructive coronary artery disease or agiographic evidence of acute plaque rupture. The patient may also have ECG abnormalities (such as an ST segment elevation with or without T wave inversion) or moderately elevated cardiac troponin levels [4]. Our patient had transient ECG abnormalities and no agiographic evidence of acute plaque rupture or vessel occlusion meeting the Mayo criteria for TCM.

Women in the postpartum period, especially after a cesarean delivery, may represent another new vulnerable group at increased risk for TTS. TTS in the postpartum period should be considered as a different clinical entity from peripartum cardiomyopathy with specific clinical, therapeutic and prognostic implications [5].

Regarding our patient, she presents several factors explaining the occurrence of Tako-tsubo, first she had presented a hemorrhagic shock following the uterine rupture causing hemodynamic instability and the massive involvement of the Adrenergic system, the second factor is the vulnerability of the patient in the absence of emotional expression t especially the difficulty of adapting to the neonatal death that she had just learned which generated depression in the parturient as well as a state of emotional stress.

The presence of depression in this type of pathology has been reported in several studies: Vidi et al. (34 patients with tako-tsubo): 21% of patients present with a depressive or anxiety disorder, this factor being more present than other cardiovascular risk factors such as smoking or diabetes; Mudd et al .: found a prevalence of depressive or anxiety disorder of 40% in 110 patients with tako-tsubo; And several other studies have found elevated levels of catecholamines, especially norepinephrine, in depressed patients [6].

Therapeutically, Treatment during the acute phase a characteristic feature of TS is spontaneous resolution in hours or weeks. Therefore, treatment in the acute stage should be supportive and focus on appropriate treatment of complications. There are no controlled studies or guidelines on how to treat TS. In mild cases, in addition to supportive therapy with beta blockers and aspirin may be considered [2].

Conclusion

The prevalence of stress cardiomyopathy is rare, and the pathophysiology of this disease remains to be defined. The case that we have presented highlights Hypovolemia as à complication of any childbirth as well as the emotional state of a patient in per partum may predispose to this type of pathology. This implies that the risk of an event may depend not only on the person's cardiovascular vulnerability following exposure to stress, but also on their coping mechanisms.

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