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Case Report

Perioperative Takotsubo Cardiomyopathy in Young Adult: A Case Report and Review of the Literature

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Abstract

Background: Tako-tsubo syndrome is an increasingly common cause of perioperative left ventricular dysfunction. It's often transient and reversible. Until then, it mainly affect elderly patients but recently it begins to spread to a younger population. ***Case report:*** we report the case of a young female patient with takotsubo cardiomyopathy following a simple Laparoscopic abdominal surgery.

Keywords: Tako-Tsubo syndrome; cardiomyopathy of stress; perioperative; apical ballooning.

INTRODUCTION
BACKGROUND

Tako-tsubo syndrome, often referred to as "cardiomyopathy of stress", is an increasingly common cause of left ventricular dysfunction, often transient and reversible [1].

It is characterized by an apical ballooning of the left ventricle with a hyperkinesis of the base, without any obstruction of the supra-epicardial coronary arteries [2].

We report the case of a TAKO-TSUBO Syndrome occurring in immediate postoperative period in a 31-year-old ASA-I patient undergoing laparoscopic cholecystectomy.

Case Presentation

A 31-year-old female patient with body mass index at 21.3 kg / m2, was operated for uncomplicated laparoscopic cholecystectomy. her medical past history was normal.

During the pre-anesthetic consultation, heart rate was 55 b/min, noninvasive blood pressure (NIBP) at 110/60 mmHg and pulsed saturation at 99%. Cardiopulmonary auscultation was normal and the rest of the clinical examination found no abnormality. Airway evaluation was normal and no difficult airway access sign was found. The patient was considered ASA I and no biological or radiological assessment was requested.

On the surgical table, monitoring was performed by noninvasive blood pressure (NIBP), an electrocardio scope and pulse oximetry.

After pre-oxygenation and vascular filling, general anesthesia was performed with fentanyl, propofol and rocuronium. Orotracheal intubation was performed without difficulty. Capnography showed respiratory variations of the CO_2 curve and respiratory pressures were within the norms. Anesthesia was maintained by sevoflurane at 1 MAC (minimum alveolar concentration).

The total duration of the procedure was 45min, the blood loss was negligible and the intakes limited to 2L of saline serum 0.9%.

Throughout the procedure, the patient maintained a stable BP between 100-110 / 55-60 mmHg with a HR between 70 and 80 B/min. SpO2 remained stable at 100% and EtCO2 at 35 mmhg. Analgesia was provided by nefopam with paracetamol.

In the post-interventional monitoring room, after warming up and complete waking, the patient was extubated.

After 10 min of extubation, an arterial hypotension at 75/40 mmHg was associated with a tachycardia at 130 b/min with pulsed arterial saturation at 93%. The patient did not report chest pain or dyspnea. Rapid and immediate clinical examination did

not show mottling or cyanosis or signs of respiratory distress and neurological examination was normal with equal and reactive pupils without any sensory or motor deficit. Cardio-pulmonary auscultation was normal. However, the patient was somnolent, but with an adapted response to verbal stimuli. The bandage was clean, the pain was absente (analogue visual scale evaluated was less than 3), the temperature was at 37.2C. The conjunctiva were normally stained, the hemoglobin was at 11.9 g / dl and abdominal ultrasound was normal.

An immediate symptomatic treatment, based on ephedrine associated with vascular filling with saline (1.5L), was ineffective. Norepinephrine was initially introduced at the dosage of 400 µg / kg / min and the patient was transferred to intensive care unit for supplemental management

The electrocardiogram found repolarization disorders in anteroseptoapical territory (Figure-1). Chest x-ray showed a cardiomegaly with alveolo-interstitial syndrome and apical vascular redistribution.

Transthoracic echocardiography found apical hypo-kinesia with a left ventricular ejection fraction (LVEF) at 40% and left ventricular balloonization (on the 4-cavity mammary section), in favor of the diagnosis of the tako- Tsubo syndrome (Figure 2 & 3).

The biology exams found hemoglobin at 11.9 g / dL, leukocytes at 4700 / ml, platelets at 280,000 / ml, troponin at 4250ng / ml (110-fold normal), BNP at more than 6000 pg /ml (100 fold normal) with urea at 1.2 g / l and serum creatinine at 27 mg / l associated with oligo-anuria. Arterial gasometry showed pH = 7.22 - PaO_2 = 49 mmHg- $PaCO_2$ = 51 mmHg HCO3- = 15mmol / l).

The immediate evolution was marked by the installation of acute respiratory distress with cyanosis and desaturation (SpO2 less than 75%), requiring the use of mechanical ventilation after failure of non-invasive methods (high concentration mask and CPAP) With adrenaline in high doses.

The progression after 48h was favorable with successive extubation, adrenaline withdrawal and decreased troponin (2000ng / ml) and BNP (3500pg / ml) with recovery of diuresis and normalization of renal function (urea = 0, 4 g / l and serum creatinine = 13 mg / l).

The thoracic radiograph showed a healthy parenchyma. The ECG showed a regression of the ST sub-shift and the ETT performed after 72h was normal with a 66% LVEF. Coronary angiography was without anomalies.

The patient was transferred to the cardiology department for further care.

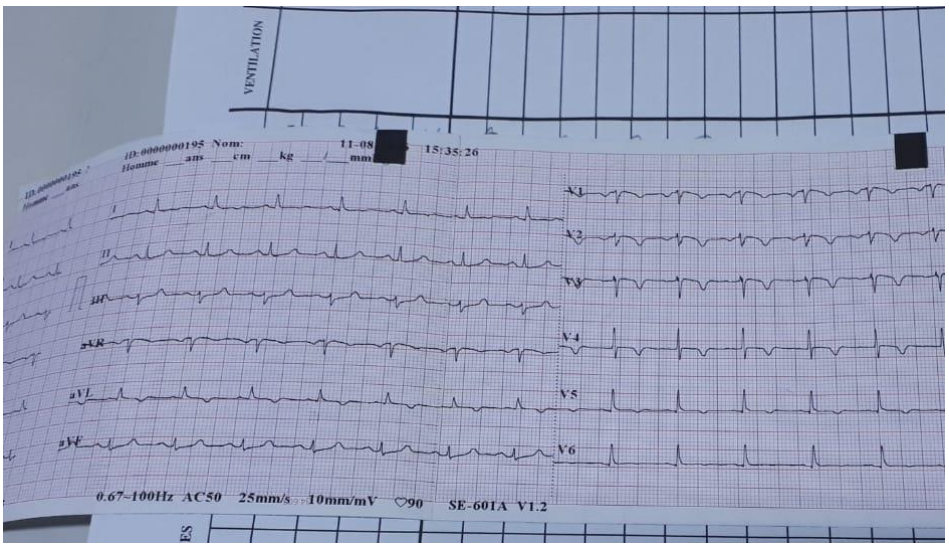

Fig-1: Electrocardiogram with repolarization disorders in anteroseptoapical

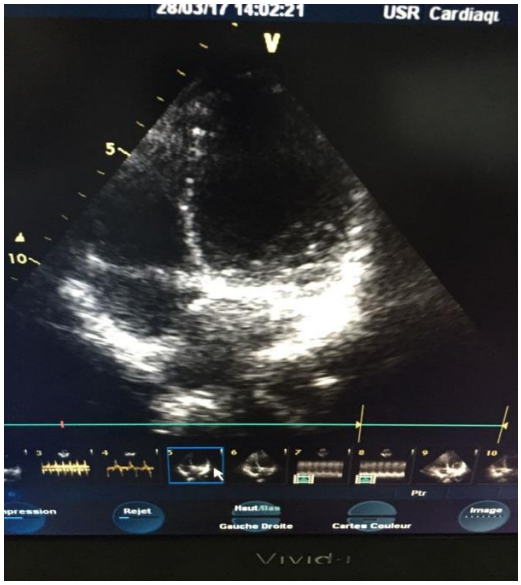

Fig-2: Echocardiography 4 cavities cut showing base dilation of the left ventricle

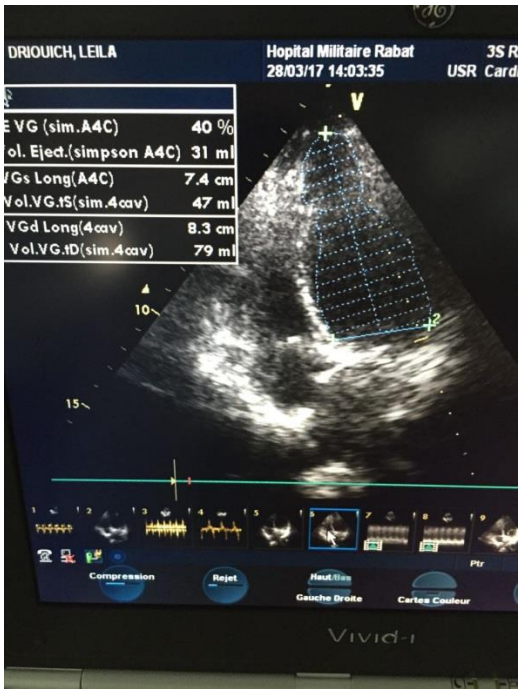

Fig-3: Echocardiography: 4 cavities cut showing an apical balloonization of the left ventricle

DISCUSSION

Tako-tsubo cardiomyopathy is a form of stress cardiomyopathy. It was originally described in the Japanese literature in 1990 [3, 4] and was only reported in the English literature until 2001 [5]. It is characterized by an apical ballooning of the left ventricle with a hyper kinesis of the base, in the absence of supra-epicardial coronary obstruction [1].

Tako-tsubo cardiomyopathy has at least 75 names [6]. The name tako-tsubo, which refer to the jar which in Japanese fishermen trap octopuses, has been validated by the European Society of Cardiology (ESC) and remains the most widely used in the literature [7].

Tako-tsubo syndrome occurs every year in approximately 2-9 / 100,000 people in the general population. It can also occur up to 1 case out of 6700 during perioperative period [8].

In the first publications, the tako-tsubo syndrome affected women in 90% of cases. Patients' age ranged from 58 to 77 years, with rare endpoints of 10 and 89 years. Recent studies have shown an expanded clinical profile to a significant minority of younger patients (age less than 50 years) and men [1].

In our case, the patient was young, confirming the reach of an increasingly young population.

The mechanism of the tako-tsubo syndrome remains hypothetical until now. Previous studies and observations have established that the akinetic territory does not correspond to a coronary arterial territory. The tako-tsubo syndrome is not explained by the obstruction of the supra-epicardial coronary arteries, nor by a coronary spasm. [10-12].

The current assumption is that excessive catecholaminergic stimulation alters cardiomyocytes, their metabolism and microcirculation.

Many circumstances support this hypothesis, since emotional or physical stress preceded the occurrence of tako-tsubo syndrome in the majority of published cases. Plasma levels of noradrenaline and adrenaline were 2 to 3 times higher [1].

Other factors have been implicated in the genesis of tako-tsubo syndrome, including disorders of calcium metabolism, depression and the use of antidepressants, estrogens and their preventive role [13]. Genetic factors, including certain alpha and beta-adrenergic receptor polymorphisms, were suspected. But no study in this direction has been carried out to assert this hypothesis [14].

In our case, surgical stress preceded the occurrence of tako-tsubo syndrome, which is in favor of the catecholaminergic theory.

The diagnosis of tako-tsubo syndrome is based on the Mayo-clinic criteria published in 2008 [15] (Transient hypokinesis,akinesis, or dyskinesis of the left ventricular mid segments, with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often but nor always present - Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture - New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin - Absence of pheochromocytoma and myocarditis).

Recently, European Society of Cardiology (ESC) has also included others criteria in addition to those of Mayo-clinic: the presence of left limb block or QT prolongation on ECG, elevation of troponin, increased natriuretic peptid brain levels, and the notion of complete reversibility At the end of 3 to 6 months [7].

Tako-tsubo syndrome is completely reversible, with no complications or sequelae in 50% of cases, whereas in half the complications can occur: congestive heart failure (12-45%), pulmonary edema (8-20%) And cardiogenic shock (4 to 20%) [7].

Other complications can also occur such as mitral regurgitation, wall thrombosis, arrhythmias and syncope.

ESC has compiled a list of risk factors for complications or deaths during tako-tsubo syndrome [7]. (Major risk factors are: Age ≥ 75yr, Systolic blood pressure < 110mmhg, Clinical pulmonary edema, Unexplained syncope, ventricular tachycardia or fibrillation, LVEF < 35%, Left ventricular outflow tract obstruction ≥ 40mmHg, Mitral regurgitation, Apical thrombosis, New ventricular septal defect or contained left ventricular wall rupture).

Thus, patients with high risk of complications should be hospitalized in an intensive care unit with monitoring and armed surveillance for at least 72 hours [7].

Treatment remains empirical [1,16], as there is no clinical trial to guide management. The administration of a beta-blocker is considered reasonable by most authors to prevent recurrence if one considers that a sympathetic hyperreactivity contributed to the pathogenesis and in case of obstacle to the ejection of the left ventricle . Nevertheless, the real interest of beta-blockers is not established.

An ACE inhibitor or diuretic must be administered to prevent or treat left ventricular failure. Antiplatelet therapy associated with anticoagulant therapy should be used to treat and prevent thromboembolic complications [1, 16].

In cardiogenic shock, most authors prefer inotropic positive noncathecolaminic agents (millrinone, vasopressin, levosimendan) [17, 18].

A general review of the literature found 131 cases of tako-tsubo syndrome occurring during the perioperative period [19,20]. It is not yet clear whether this syndrome is the result of surgical conditions, surgery itself or anesthesia. 66% had general anesthesia, 12% had locoregional anesthesia, 4% had combined anesthesia and 5% had no anesthesia [19, 20].

Most patients underwent visceral (28%) and cardiothoracic surgery (16%). The average age was 54 years with 40% of patients under 50 years. There is also a female predominance (80% are women) [19,20].

Our patient was a young woman (31 years old), who underwent visceral surgery (This is consistent with data from the literature).

In 58% of the cases the clinical manifestations of tako-tsubo syndrome occurred in the postoperative period. 15% of cases were attributed to pain and anxiety, while 5% were related to neuromuscular blockage and adrenaline use [19, 20].

In our patient the onset of symptoms was secondary to anxiety. Clinical presentation varied with signs of heart failure in 66% of cases (hypotension, dyspnea, pulmonary edema), while in 30% of cases the symptoms of acute coronary syndrome were in the foreground (chest pain, electrical changes And increased cardiac enzymes) [20].

In our case the clinical presentation was mixed with the signs of heart failure and ST- acute coronary syndrome.

The diagnosis of tako-tsubo syndrome was suspected by cardiac ultrasound in 68% of cases (apical ballooning) with a left ventricular ejection fraction (LVEF) between 31 and 40% [19].

In our patient the diagnosis was also suspected by cardiac ultrasound in the presence of the typical ballooning with a LVEF at 40%.

Initial management required the use of vasoactive drugs and mechanical ventilation in 42% of cases. That was also the case with our patient.

The progression was favorable with recovery of normal cardiac function in the following week in 82% of the cases and the hospital mortality rate was 6.1%.

The evolution in our patient was also favorable in the first week following the onset of symptoms.

Several authors emphasize the importance of the prevention of an initial episode by the preoperative psychopharmacological approach, deep intraoperative anesthesia and optimal analgesia with postoperative sedation.

CONCLUSION

Tako-tsubo cardiomyopathy results from the functional and transient inhibition of myocardial cells associated with the release of catecholamines. The circumstances surrounding the anesthetic act and intensive care (pain, general or loco regional anesthesia or even sedation for a gesture called benign) have been associated with the occurrence of a tako-tsubo syndrome.

The anesthetist must be informed of this syndrome, discuss it with any compatible sign and initiate the diagnostic procedure, including early echocardiography, coronary angiography and / or ventriculography and, if necessary, cardiac MRI.

Serious complications may occur in the acute phase that warrant surveillance in an intensive care unit.

Conflicts of Interest

The authors do not declare any conflicts of interest

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