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Case report

Takotsubo cardiomyopathy in the post-anesthetic care unit☆

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ABSTRACT

Takotsubo cardiomyopathy (TC) is a transient left ventricular dysfunction that may mimic an acute coronary syndrome. Characteristically, it presents with chest pain of acute onset, electrocardiographic alterations suggestive of myocardial ischemia, and transient akinesis of the apex and the distal portion of the left ventricular anterior, lateral and inferior walls, with compensatory hyperkinesis of the basal walls. These changes in myocardial contractility make the ventricle acquire the typical appearance of a "takotsubo", a Japanese word that refers to a vessel used as an octopus trap. It is frequently triggered by psychological or physical stress in postmenopausal women. Ventricular function recovers quickly and prognosis is excellent.

There are several case reports of associations between anesthesia and surgery with TC. The adrenergic discharge during or after anesthesia may affect the myocardium and gives rise to transient ventricular dysfunction in some patients. We describe a case of TC in a patient undergoing eye surgery.

The case is of a 72-year-old patient scheduled for cataract surgery under general anesthesia. Functional class was normal and there were no special events during the procedure. In the post-anesthetic care unit, the patient had an episode of respiratory failure associated with an inverted T-wave. The transthoracic echocardiogram revealed ventricular dysfunction, and the coronary arteriogram showed normal coronary arteries. The patient was diagnosed with TC and she recovered normal ventricular function within 4 weeks.

Conclusions: TC is a reversible ventricular dysfunction, and there is a growing number of cases described in the literature in relation to anesthesia. Anesthetists must be aware of this condition every time there is a cardiac dysfunction or hemodynamic collapse in the perioperative setting.

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Cardiomiopatía Takotsubo en la unidad de cuidado postanestésico

Palabras clave:
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Disfunción ventricular
Síndrome coronario agudo
Isquemia miocárdica

RESUMEN

La cardiomiopatía Takotsubo es una disfunción transitoria del ventrículo izquierdo que puede simular un síndrome coronario agudo. Se presenta de manera característica con dolor torácico de inicio agudo, alteraciones electrocardiográficas sugestivas de isquemia miocárdica y acinesia transitoria del ápex y de la parte distal de las paredes anterior, lateral e inferior del ventrículo izquierdo con hipercinesia compensatoria de las paredes basales. Estos cambios de la contractilidad miocárdica hacen que el ventrículo tome la típica apariencia de un «takotsubo», palabra japonesa que hace referencia a una vasija usada como trampa para cazar pulpos en Japón. Es frecuentemente desencadenada por estrés psicológico o físico en mujeres posmenopáusicas; la función ventricular se recupera rápidamente con excelente pronóstico.

La anestesia y el ámbito perioperatorio han sido relacionados a varios reportes de casos de cardiomiopatía Takotsubo. La descarga adrenérgica durante o después de la anestesia puede afectar el miocardio y desarrollar una disfunción ventricular transitoria en algunos pacientes. Nosotros describimos un caso de cardiomiopatía Takotsubo en una paciente que fue sometida a una cirugía ocular.

Una paciente de 72 años de edad fue programa para cirugía ocular por cataratas bajo anestesia general. Su clase funcional previa era normal y no ocurrieron eventos especiales durante el procedimiento. En la unidad de cuidados postanestésicos la paciente presentó un episodio de falla respiratoria asociada a inversión de la onda T. El ecocardiograma transtorácico reveló una disfunción ventricular y la arteriografía coronaria evidenció arterias coronarias normales. Se diagnosticó cardiomiopatía Takotsubo, y 4 semanas después del episodio la función ventricular retornó a la normalidad.

Conclusiones: La cardiomiopatía Takotsubo es una disfunción ventricular reversible y cada día se describen más casos relacionados con la anestesia. Los anestesiólogos debemos estar alerta a esta condición cada vez que se presente una disfunción cardíaca o un colapso hemodinámico durante el ámbito perioperatorio.

Takotsubo cardiomyopathy (TC)

TC is a transient condition that mimics acute myocardial infarction (AMI). TC, apical ballooning or broken heart syndrome are terms that have been used to define a reversible cardiomyopathy often caused by a stressful situation, with a presentation indistinguishable from myocardial infarction. It has been reported almost exclusively in postmenopausal women, and emotional or physical stress may be the triggering event. Echocardiographic changes return to normal within weeks or months. Anesthesia and the perioperative setting have been associated with this condition.

Case report

The patient gave her informed consent.

A 72-year-old woman was scheduled for cataract surgery. There was a significant clinical history of hypertension, diabetes mellitus and chronic obstructive pulmonary disease. She was receiving losartan, glibenclamide, metformin, furosemide and theophylline. She had no history of cigarette smoking and her functional class was normal (NYHA 1). There was no history of obesity (BMI was 27) or obstructive sleep apnea, and there were no parameters suggesting a difficult airway. Laboratory tests and the electrocardiogram done before surgery were normal.

On the day of surgery, the patient was premedicated with 2 mg of midazolam IV and her blood sugar reading was 109 mg/dl. Basic monitoring was instituted (NIBP, pulse oxymetry, capnography and lead-II EKG). Blood pressure was 160/90 and heart rate was 90 bpm. The patient was asymptomatic and did not report chest pain. General anesthesia was induced with fentanyl 100 μg, lidocaine 60 mg, propofol 60 mg, and vecuronium 2 mg. A number 3 laryngeal mask was used for airway management, and maintenance was achieved with 3% sevoflurane and oxygen at a rate of 1 l/m. Half way into surgery, an additional dose of 2 mg of vecuronium was required. The surgery lasted 45 min and was uneventful, and the muscle relaxant was reverted at the end of the procedure using 2 mg of neostigmine and 1 mg of atropine, due to evidence of residual muscle relaxation. No peripheral nerve stimulator was available. The laryngeal mask was removed after confirmation of adequate breathing and consciousness.

The patient was taken to the Post Anesthesia Care Unit where she was monitored and received 50% oxygen through a Venturi mask. After 20 minutes, she presented desaturation, agitation and her blood pressure increased above 200/110, with no improvement when 100% oxygen was used. She was intubated using fentanyl 100 μg, lidocaine 60 mg, propofol 60 mg and vecuronium 2 mg. A 7.5 orotracheal tube was used and

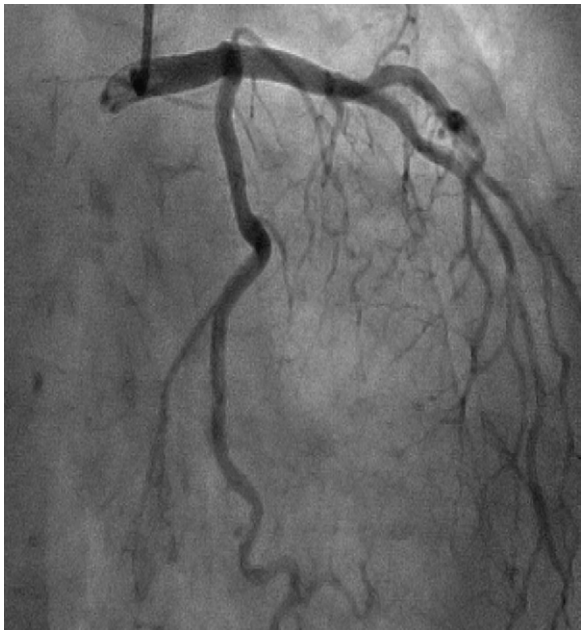

Fig. 1 – Left coronary artery arteriogram showing absence of significant lesions.

the patient was put on mechanical ventilation. Capnography showed severe hypercapnia (ETCO2 80 mmHg) and the electrocardiogram showed an inverted T wave on the anterior wall.

The patient was admitted to the Intensive Care Unit. Cardiac enzymes became slightly elevated (troponin I 0.05 ng/ml) and transthoracic echocardiography revealed akinesis of the entire apex with anterior hypokinesis and a 35% ejection fraction. Coronary arteriography revealed normal coronary arteries (Figs. 1 and 2). Ventriculography showed apical akinesis and basal hyperkinesis. Pulmonary embolism was ruled out by CT angiography, and dilatation of the left cardiac cavities was reported. During hospitalization, the patient was treated with aspirin, metoprolol, enalapril, atorvastatin and low molecular weight heparin. Weaning from mechanical ventilation was achieved after 24 hours and the patient was discharged 7 days later with a prescription of metoprolol, aspirin, enalapril and furosemide. On follow-up four weeks later, the patient was found to be clinically stable with no chest pain or dyspnea. The follow-up echocardiogram revealed recovered motility of the ventricular wall and an ejection fraction of 55%, with no segmental abnormalities.

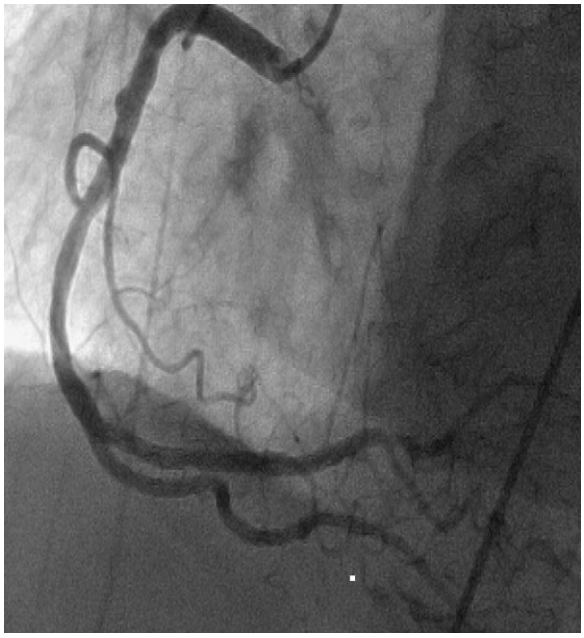

Fig. 2 – Right coronary artery arteriogram with absence of significant lesions.

Discussion

The first report on TC in the literature was published in 1990 by Hikaru Sato. The name Takotsubo came from the classical shape of the left ventricle in systole, similar to the shape of a vessel used by the Japanese for capturing octopuses.[1] The prevalence of the disease is unknown. In Japan, it is estimated to account for 1–2% of all patients admitted to the hospital with chest pain and acute ST changes.[2] In the United States, 2–2.2% of patients presenting with a clinical picture of acute MI with ST elevation or unstable angina are diagnosed with TC.[3] It is predominantly found in older women, and postmenopausal women account for 90% of cases.[4] The reason for female predominance is basically unknown, although estrogen deficiency after menopause appears to play a role in the pathogenesis. Emotional stress or a physical event appears to be the trigger in close to two-thirds of the events reported.

Several theories have been proposed. There is no doubt that catecholamines are involved, considering that many patients have a prior history of emotional stress, although the way they affect myocardial contraction is not clear. Very high catecholamine levels have been reported, and structural and ultrastructural changes in the myocardium suggest direct adrenergic toxicity.[5] Multivessel spasm must be present in order to explain wall abnormalities that go beyond the territory of a single epicardic coronary artery. Martin et al. demonstrated an increase in vascular reactivity and reduced endothelial function in response to acute mental stress in patients with a prior episode of TC. Vasomotor dysfunction plays a role as a potential factor in the pathogenesis.[6] An increase in the density of B-adrenoreceptors in the cardiac apex has been observed in experimental animals.[7] A transient obstruction of the left ventricular outflow tract has also been proposed as another mechanism in the pathogenesis. The fact that elderly women have a sigmoid septum might give rise to a severe obstruction in the case of a catecholamine discharge, leading to apical ischemia as a result of wall stress.[8] Another theory has to do with a neurologically mediated mechanism. A similar pattern of ventricular dysfunction has been observed in patients with subarachnoid hemorrhage. Interestingly, this has been associated with ruptured aneurysms in the anterior half of the circle of Willis. These aneurysms lie on the amygdala and the right insular cortex, which control sympathetic flow to the heart.[9] Transient thrombotic occlusion of an unstable plaque in the transapical anterior descending artery is another new theory.[10] Regardless of this range of hypotheses,

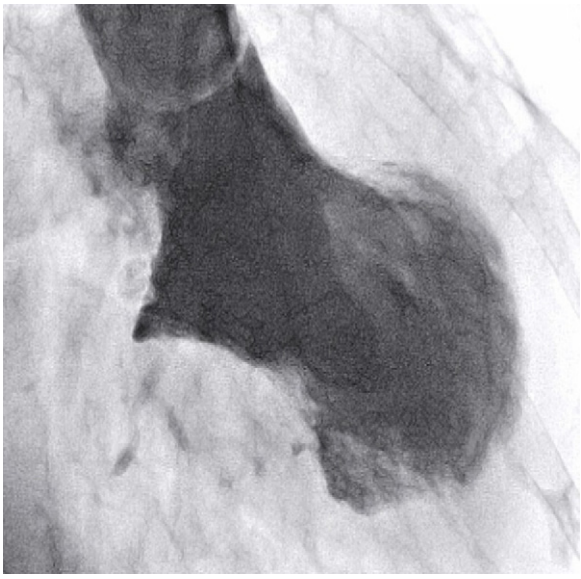

Fig. 3 – Ventriculography during ventricular contraction. "Takotsubo image".

there are many questions still unanswered about the etiology and pathogenesis of TC.

Signs and symptoms may be indistinguishable from acute MI. In TC there may be certain changes that can help differentiate it from acute MI. In a retrospective series, it was demonstrated that a higher voltage in the ST elevation comparing V4–V6 with V1–V3, together with an absent pathological Q wave and reciprocal changes in the lower leads, could have a higher sensitivity and specificity in differentiating TC from acute MI.[11,12] Another finding that may help is that the mid ventricular circumference is completely affected, while the compromise in acute MI is more segmental. These differences confirm the idea that TC affect the myocardium beyond the territory of a single coronary artery and may help differentiate between the two entities non-invasively.[13,14] The typical left ventricular image in Takotsubo may be seen on echocardiography, contrast (Fig. 3) ventriculography or nuclear magnetic resonance. When available, MRI may be particularly useful, since it can demonstrate absence of myocardial necrosis in gadolinium-enhanced images. As with most patients with a clinical picture of acute MI, TC patients are usually taken to emergency catheterization, and coronary arteriography is one of the pillars for diagnosis.

There are numerous reports in the literature about the association between TC and anesthesia. There are critical instances in anesthesia and surgery when catecholamine discharge may affect the heart. Airway manipulation, pain, extubation, anesthesia awareness, residual muscle relaxation and other situations may trigger an adrenergic response. Several cases of TC have been described before and during anesthesia induction.[15–17] Other reports have described an association with awakening and the postoperative setting.[18,19] There are some reports of associations with succinylcholine, atropine and antibiotics given intraoperatively.[20–22]

The management of TC has not been established. Since most cases are initially considered to be acute coronary syndrome, once the diagnosis of TC is confirmed, the recommendation is to discontinue anti-platelet aggregation and nitrate therapy. Beta-blockers and angiotensin converting enzyme inhibitors are considered useful, considering that this is a catecholamine-induced syndrome. Diuretics and anticoagulation should be considered in the event of congestive heart failure and severe systolic dysfunction. In the event of cardiogenic shock, some authors recommend the use of aortic counterpulsation balloon over inotropes, because of the etiology of the cardiomyopathy. They even suggest that it is more advisable to use levosimendan instead of dopamine or dobutamine.[23]

The complications described in TC are infrequent and include cardiogenic shock, ventricular tachycardia, left ventricular rupture and death. However, this is considered a syndrome with a benign prognosis since in most cases, ventricular function comes back to normal (and that is part of the diagnosis) within two to four weeks after the onset of the event.

We present the case of a patient with many factors that may have triggered TC. The atropine dose, the residual relaxation, hypercapnia and the post-anesthetic respiratory failure might have been the triggers, giving rise to an adrenergic discharge in a post-menopausal woman. Reports in the literature have shown that TC is not uncommon in the perioperative setting and, as anesthetists, we must be aware of any situation that may trigger a sympathetic response in these kinds of patients. We also need to keep in mind the intra-operative use of diagnostic tools such as trans-esophageal and transthoracic echocardiography, in particular at this time when anesthetists are better trained in the use of perioperative ultrasound.

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Conflict of interest

None.

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