Case Report

Transesophageal echo diagnosis of perioperative unusual transient left ventricular apical ballooning syndrome

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ABSTRACT

Stress cardiomyopathy, or Takotsubo syndrome, is a widely recognized cardiac pathology with a clinical presentation similar to acute coronary syndrome and related to physical or emotional stress. Perioperatively, it is challenging to identify it given the variety of forms and scenarios in which it can present. We describe a 22-year-old patient with an atypical presentation of Takotsubo syndrome during anesthesia induction, which highlights the usefulness of transesophageal echocardiography for the initial diagnosis.

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Key words: Perioperative care; Takotsubo stress cardiomyopathy; Transesophageal echocardiography

INTRODUCTION

The introduction is a brief background statement of the problem, identifying the specific disease about which the case report is written or a device about which a complication occurred.

Stress cardiomyopathy, or Takotsubo syndrome, was first described in 1990.[1] The widespread recognition it has gained, since then has allowed the definition of its clinical characteristics and its strong association with physical or emotional stress. From an anesthesiology point of view, there are several reports of perioperative presentations, most of which were initially interpreted as a perioperative coronary event.[2,3] The growing use of intraoperative transesophageal echocardiography (TEE) has become an indispensable tool for the early diagnosis and subsequent management.

CASE REPORT

A 22-year-old, 48-kg female patient with a 6-month history of temporomandibular joint pain was scheduled for bilateral temporomandibular meniscopexy. The patient underwent an uneventful general anesthesia for an appendectomy at age 10.

The patient was admitted to surgery with a heart rate of 92 bpm, respiratory rate of 16 breaths per minute, blood pressure of 128/78 mmHg, and oxygen saturation on room air at 91%. Preoxygenation was performed, and induction was begun with midazolam 2 mg, intravenous (IV), remifentanil 0.14 μg/kg/min IV, propofol 140 mg IV, and rocuronium 35 mg IV. Following intranasal application of oxymetazoline HCl 0.025%, five drops, and topical lidocaine gel, the nasotracheal tube was inserted, with confirmation of proper placement

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through symmetrical auscultation and capnography. As the nasotracheal tube was being fixed, the patient presented sinus bradycardia associated with ST changes, which rapidly progressed to supraventricular tachycardia with a heart rate of 168 bpm, hypotension, desaturation, and piloerection with persistence of ST changes. Increased airway pressure associated with rhonchi was noted, and therefore, bronchospasm was considered to be a possible etiology. After four puffs of salbutamol and 100 mg hydrocortisone IV, bloody material was seen coming from the nasotracheal tube, compatible with pulmonary edema. A chest X-ray was ordered, which showed severe bilateral pulmonary edema [Figure 1]. In the light of these findings, an emergency TEE was performed.

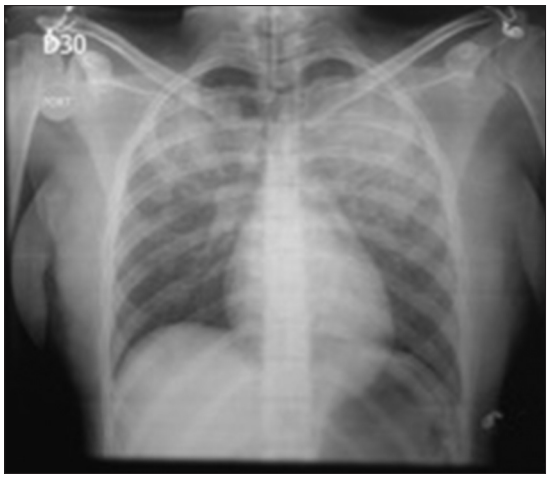

Figure 1: Chest X-ray with bilateral pulmonary edema signs

The TEE showed severe left ventricular dysfunction with akinesia of the apical and mid segments of the anterolateral and septal walls, hypokinesia of the anterior and anteroseptal walls, with spared of the basal segments of the septal and inferior septal and inferior walls. The right ventricle was moderate dilated with a mild dysfunction and moderate tricuspid regurgitation. The cardiac output was calculated at 1.8 l/min with Left ventricular outflow tract velocity time integral method [Figure 2 and Supplementary Video 1]. Vasopressor support was begun with noradrenaline at 0.1 μg/kg/min IV, with which the patient improved slowly, reaching a heart rate of 103 bpm, blood pressure of 94/66 mmHg, and oxygen saturation at 92 at 100% FiO_2%. Vasopressor support was continued, surgery was canceled, and the patient was transferred to the Intensive Care Unit (ICU) for stabilization.

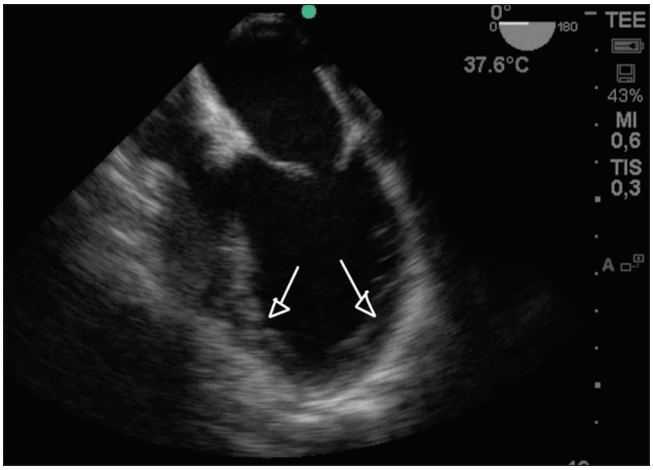

Figure 2: Mid-esophageal, four chambers view transesophageal echocardiography. Arrows showing left ventricular dilatation

In the ICU, a pulmonary artery catheter was placed, showing a cardiac index of 1.63 l/min/m^2, and a wedge pressure of 20 mmHg, with normal systemic and pulmonary resistance. A transthoracic echocardiogram reported severe biventricular dysfunction with hypokinesia of the distal third of the anterior and inferior septum, and therefore, treatment was started with milrinone at 0.5 μg/kg/min IV [Supplementary Video 2]. troponin I on the day one was 3.7 ng/mL, and the troponin on the second day was 0.7 ng/mL (normal value <0.1 ng/mL). The patient continued a gradually favorable course with a progressive increase in the cardiac index to 3.1 l/min/m^2, and an ejection fraction up to 60% [Figure 3 and Supplementary Video 3]. She was weaned from mechanical ventilation on the second day and discharged from the ICU on the 6th day postoperative. The following day, cardiac catheterization was performed, in which no coronary disease was found [Figure 4].

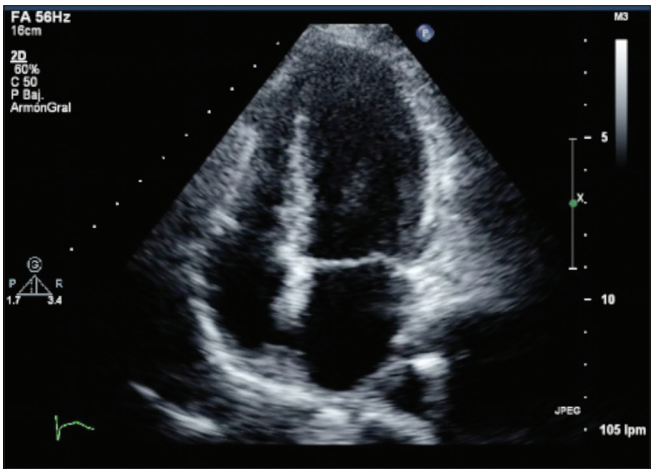

Figure 3: Four-chamber apical view from a transthoracic echocardiogram. The right heart is normal. The left ventricle is slightly dilated without atrial alteration

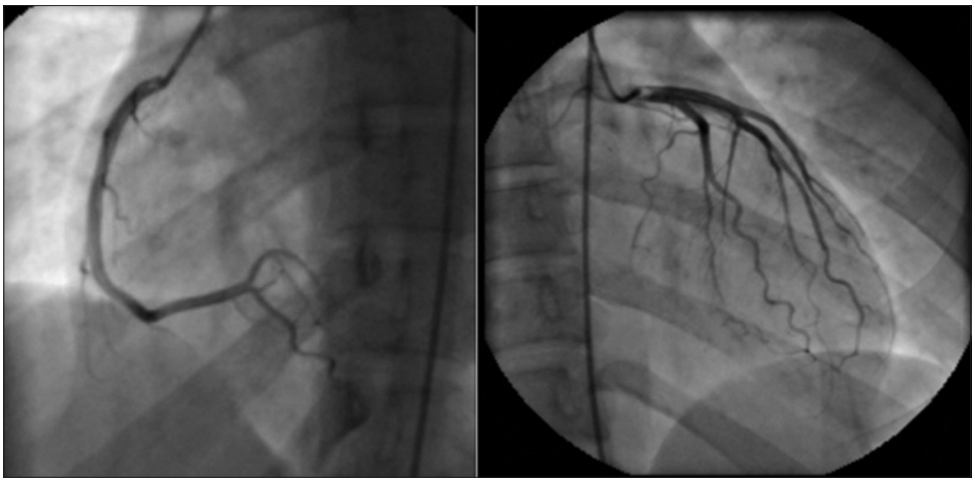

Figure 4: Coronary arteriography showing no significant lesions

DISCUSSION

Stress-induced cardiomyopathy, also known as Takotsubo cardiomyopathy, was first described in Japan in 1990.[1] It is a sudden onset cardiopathy with signs and symptoms of heart failure and/or acute coronary syndrome (ACS), such as electrocardiographic changes, elevated cardiac enzymes, and signs of low output. Its main characteristic is the absence of an evident coronary lesion and the complete recovery of ventricular function to premorbid levels in a relatively short period.[4,5]

Since its initial description, there has been a gradual increase in the number of publications related to the disease, finding prevalence close to 1% (ranges from 0.7% to 2.5%) in patients with suspected ACS.[6,7] Because this pathology is not fully understood, there is a growing interest in elucidating the triggering factors, be they primary or secondary, as well as the underlying pathophysiology.[8] The predominant classic presentation is chest pain in the context of a middle-aged woman undergoing emotional stress.[5,8] However, young patients may have an atypical presentation in the context of surgical or anesthetic stress, such as that shown in our case.[9]

The diagnostic criteria require both ruling out active coronary disease as well as confirming ventricular dysfunction. The first is typically evaluated through cardiac catheterization, which can be performed immediately, or after stabilizing the patient while ventricular dysfunction is easily characterized with echocardiographic assistance. Intraoperative TEE becomes an invaluable diagnostic aid when the syndrome is suspected in patients undergoing a surgical intervention, and probably represents the diagnostic method of choice in this context.[10]

Treatment depends on the clinical presentation and is aimed at symptomatic treatment and vasopressor support until the condition reverses. Symptomatic recovery may take 1 week, but the process of normalization of ventricular function alterations is achieved, on average, at 6 weeks.[4,5] Patients with severe clinical presentations may have acute complications such as pulmonary edema, arrhythmias, cardiogenic shock, ventricular rupture, and death. These last three outcomes present in 7.1% of cases within the first 30 days.[5]

Cases that appear during the intraoperative period represent a diagnostic challenge for the anesthesiologist.[11,12] Among the differential diagnoses of our case are ACS, a reaction to medications and pulmonary edema due to negative pressure. The literature contains multiple case reports related to the administration of medications, with the most prevalent being the alpha-adrenergic drugs, fundamentally adrenaline.[13,14] The administration of oxymetazoline has been associated with this pathology but in doses much greater than those used in the present case.[15] Negative pressure edema was ruled out by the clinical presentation, and ACS presents with a more pronounced elevation of troponins and was later ruled out with coronary angiography, which was normal.

There are multiple reports of stress cardiomyopathy in the literature, whether triggered by physical or emotional stress, with a progressive increase in the report of cases in the intraoperative setting.[8,10,13] Echocardiographic diagnosis is generally used in the typical presentation,

but there are few reports on its use for diagnosis under anesthesia.[14] In this case report, we show an atypical presentation with its echocardiographic diagnosis and acute intraoperative management, in addition to its progress and subsequent resolution in the ICU.

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Conflicts of interest

There are no conflicts of interest.

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