

CASE REPORT

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Takotsubo cardiomyopathy developing during recovery from general anesthesia: A case report

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Abstract

The case is here presented of a 37-year old female with obstetric complaints who underwent surgery for dermoid cysts and developed sudden shock during recovery from general anesthesia. The aim of this case presentation was to share the importance of premedication when it was thought to be related to stress in the etiopathogenesis and the management of this Takotsubo (TC) case, which started with clinical sudden onset of a shock table during recovery from general anesthesia following surgery. In cases of suddenly developing shock, TC must be considered in the differential diagnosis.

Keywords: Takotsubo cardiomyopathy, general anesthesia, stress

Introduction

Takotsubo cardiomyopathy (TC) is also known as stress-related cardiomyopathy, apical ballooning syndrome and broken-heart syndrome. This is a sudden onset syndrome that manifests with chest pain and is characterized by an increase in cardiac enzymes but aneurysmal normal coronary vessels and akinetic ballooning of the left ventricular apex on ventriculography[1]. This syndrome, which is triggered by stress, is seen more in elderly, postmenopausal females and may develop after any type of surgical procedure [1]. The term stress cardiomyopathy was first introduced by Cebelin and Hirsch in 1980 [2], and was defined as a disease by Dote et al in 1991 [3]. It has been reported that TC is found in 1.7%-2.2% of patients who present with acute coronary syndrome (ACS) [4]. The TC case here presented is of a 37-year old female who underwent surgery by the Obstetrics and Gynecology Dept. for dermoid cysts and developed sudden shock clinically during recovery from general anesthesia.

Case Report

AIn the preoperative evaluation of a 37-year old, 70 kg female, who was planned to undergo surgery by the Obstetrics and Gynecology Dept. for dermoid cysts, other than stress-related

urticaria, the examination findings were normal. Information about the procedure to be applied was given to the patient and an informed consent form was obtained. To reduce stress before the operation, pre-medication of 2mg midazolam was administered approximately 1 hour preoperatively. On the operating table, standard monetarization was applied with non-invasive arterial blood pressure, ECG and pulse oximetry. Anesthesia induction was made with 3mg/kg propofol and 1mcg /kg fentanyl, then curarization was obtained with 0.6mg /kg rocuronium bromide and orotracheal intubation was performed without any problems. Anesthesia maintenance was provided with 50% nitrogen, 50% O₂ 3L/min and 6% desflurane. In addition, a 0.02 mcg/kg/min remifentanyl infusion was started.

As the patient had stress urticaria, 45.5mg mg pheniramine, 8 mg dexamethasone and 50 mg ranitidine was administered as prophylaxis. Throughout the surgery, mechanical ventilation was applied at 8 ml/kg tidal volume, in VCV mode, 4 PEEP pressure at 12/min frequency. During the operation, 1500 ml crystalloid was given and 110 cc urine output was observed. Near the end of the operation, analgesia of 1gr paracetamol, 20mg tenoxicam and 50 mg meperidine was administered. Postoperatively, reversal was made with 200mg sugammadex and the patient with a sufficient level of spontaneous respiration was extubated without any problems. At approximately 1 min after extubation, for no foreseen reason, anurticaria-like color change of the skin occurred on the upper chest, shoulders, head, neck and face regions of the body and immediately afterwards, the patient had insufficient

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respiration and hypotension developed, and then after a short period of tachycardia, bradycardia (42/min) developed. The patient was rapidly desaturated and with mask ventilation, aeration was strengthened and orotracheal intubation was re-applied and mechanical ventilation started at 8 ml/kg tidal volume, at 15/min frequency with 100% O₂ in VCV mode. In this process, the patient had resistant hypotension and bradycardia, so intermittent IV push 10mg ephedrine, 0.5mg 2x1 IV push adrenalin and 0.5 mg IV push atropine were administered.

A central venous catheter was opened in the right femoral vein and left radial artery catheterization was applied. The patient was evaluated as in a shock table, so 1000 cc colloid and 1000 cc crystalloid was applied by opening 2 large peripheral venous routes. An infusion of 0.5 mcg/kg/min noradrenalin and 15 mcg/kg/dk dopamine was started. Despite 100% O₂ ventilation, the patient had hypoxemia and with vasopressor support and IV hydration, vital signs were sinus rhythm 98/min, arterial blood pressure 75/35 mmHg, and SPO₂ 80%-85%. Immediately, a serohemorrhagic dense secretion started to come from the intubation tube. Widespread crepitation rales were present in both lungs at rest. The patient was aspirated at frequent intervals and with the consideration of anaphylactic shock, acute cardiogenic pulmonary edema, acute coronary syndrome and pulmonary emboli as a preliminary diagnosis, 20 mg furosemide IV, methylprednisolone 1 gr IV, ranitidine 50 mg IV and clexane 0.6 SC were administered. As there was metabolic acidosis in the blood gas, 5 ampoules of bicarbonate were administered IV. Calcium and potassium replacement was made because of hypocalcemia and hypokalemia. As hypotension persisted, a 0.5 mcg/kg/min adrenalin infusion was started. The Cardiology Clinic was informed and during transfer to the Intensive Care Unit (ICU), preparations were completed for bedside echocardiography. The patient was transferred to ICU when the vital signs were most suitable for transfer (BP, 125/85 mmHg, heart rate 122 bpm, SPO₂ approximately 85%). Akinetic ballooning of the left ventricular apex was seen and % 15-20 ejection fraction was measured in bedside echocardiography and also there was no thrombus in heart cavities. Despite cardiac enzymes were increased the coronary vessels were normal at the angiography. After 6 hours at the patient with under vasoactive agent and mechanical ventilation support; heart cavities were almost within normal limits and % 50-60 ejection fraction was measured in echocardiography. After these measurements patient's support was cut in a controlled manner and patient was extubated on the 12th hour. At the end of 24 hours patient's all vital signs, cardiac enzyme levels were in normal limits, ECG with sinus rhythms and heart cavities were within normal limits and % 60-65 ejection fraction was measured in echocardiography. After 48 hours patient was transferred to Obstetrics and Gynecology Department. Medical care was responded and the patient was discharged on the 10th day of the hospital.

Discussion

TC is known to be triggered by stress, and in 30%-40% of patients emotional stress has been identified as the trigger [4]. In a study by Maekawa et al, psychological or physical stress was determined in more than 50% of patients [5]. Deniz et al operated on a 92-year old patient under spinal anesthesia for transurethral resection-

bladder tumor (TUR-BT) and reported that TC developed at 1 hour postoperatively [6]. Demir et al reported that ventricular fibrillation developed in a 27-year old female during recovery from general anesthesia following surgery for chronic sinusitis and nasal polyps and as a result of examinations, TC was diagnosed [7].

There are studies that have suggested that the disease, which is seen more in the winter months, is of vasospasm or viral origin [9]. The current patient was compatible with this view. The disease is seen in post-menopausal females in 90% of cases [8-10].

The emergence of TC is closely related to catecholamine's [11]. At the moment of stress, the contraction of the myocardial muscle is affected associated with the stimulation of the sympathetic nerve system. The distribution of the sympathetic nerves within the myocardium explains the impairment in the segmental wall movement at the moment of stress. In addition, sympathetic stimulations related to microvascular dysfunction of the coronary vessels [12]. The rate of coronary flow reserve is a functional marker of the small coronary vessels and is reduced in TC-like conditions that impair left ventricle function [13].

Prasad [14] published the Mayo Clinical Diagnosis Criteria to guide differential diagnosis in this disease. The Mayo Clinical Diagnosis Criteria are:

1. Recurrent left ventricular dysfunction accompanied by segmentally independent SHDB associated with any coronary artery (but some atypical cases can show SDHB consistent with the myocardial segment fed by any coronary artery; see the other conditions below),
2. The presence of obstructive coronary artery disease or torn plaque is discounted with angiography,
3. Newly-emerging ECG changes and/or an increase in tropine,
4. Absence of head trauma, intracranial bleeding, pheochromocytoma, myocarditis, hypertrophic cardiomyopathy.

Some specific ECG findings have been reported which are helpful in the diagnosis of TC. The most commonly seen ECG finding is the elevation of the SB segment in precordial derivation. This elevation is not seen to be as great as acute MI and ST segment depressions cannot be determined in corresponding depressions [15]. In a retrospective study, it was reported that greater ST elevation in V4-V6 compared to V1-V3, pathological Q-wave not observed and no corresponding changes in inferior derivations could be findings of TC rather than acute myocardial infarctus [16].

ECG and ventriculography are extremely important in the diagnosis of TC. Shimizu et al published a classification according to the type of wall movements [17]. The Shimizu classification of TC is as follows:

1. Takotsubo type: apical akinesis and basal hyperkinesis,
2. Reverse Takotsubo: basal hyperkinesis and apical akinesis,
3. Mid-ventricular type: mid-ventricular ballooning accompanied by basal and apical akinesis,
4. Localized type: Takotsubo-type clinical findings of left ventricle dysfunction with no concomitant ballooning in any part of the left ventricle.

As the current case presented with a shock table while efforts were being made to maintain the vital status, diagnosis was made from transthoracic ECG, which was the first test, applied.

Interventions directed to heart failure and to correct left ventricle function, as the basic target of treatment, must be started without delay because the progressive process can result in cardiogenic shock, acute pulmonary edema and sudden cardiac death. The early clinical tables of cases reported in literature have shown extreme shortness of breath, tachycardia, elevated ST segment on the ECG and elevated troponine. As the current case started with clinically sudden onset of a shock table, hemodynamic stability could be provided with high-dose vasopressor and inotropic agents. With the exception of cardiogenic shock at the time of first diagnosis in these patients in literature, short, medium and long-term prognoses have been reported to be close to excellent [8,18]. Even if left ventricle function is severely impaired on first presentation when diagnosis is made, recovery starts within a few days and with return to a normal state within a few months, normal functions have been reported to be maintained [8,19]. In the current case, cardiac functions started to improve within 12 hours and the supportive treatments were terminated. Cardiac functions showed a rapid improvement in the following days and approached normal within approximately 2 weeks.

The aim of this case presentation was to share the importance of premedication when it was thought to be related to stress in the etiopathogenesis and the management of this TC case, which started with clinical sudden onset of a shock table during recovery from general anesthesia following surgery. In cases of suddenly developing shock, TC must be considered in the differential diagnosis.

Competing interests

The authors declare that they have no competing interest.

Financial Disclosure

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