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Case Report

Role of echocardiography in stress cardiomyopathy diagnosis after tracheal extubation

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ABSTRACT

Stress cardiomyopathy or Takotsubo cardiomyopathy is a new syndrome and still insufficiently recognized among emergency patients, hospitalized patients. Many different physical and emotional stressors are triggers, but individual susceptibility to Takotsubo cardiomyopathy can not be predicted. Takotsubo cardiomyopathy in surgical and critical care population is a huge diagnostic challenge. Apart that these patients are treated in stressful environment and conditions. Postoperatively due to change of consciousness and inability to speak we can not rely on verbal symptoms to make differential diagnosis. Although essential sometimes they can not be submitted to coronary angiography to exclude obstructive coronary disease due many risk factors. So, then we follow clinical course, electrocardiographic, radiologic and echocardiographic dynamic changes, laboratory findings and consiliar opinion to make diagnosis. We represent a case of Takotsubo cardiomyopathy in a 59-years old postmenopausal Caucasian woman after tracheal extubation. She was submitted to surgery of intraabdominal collection evacuation in short general endotracheal anesthesia.

Keywords: Anesthesia, Critical care, Echocardiography, Heart failure, Takotsubo cardiomyopathy

INTRODUCTION

Takotsubo cardiomyopathy (TCM) or broken heart syndrome is an acute, reversible, transient myocardial systolic dysfunction. It is frequent among postmenopausal women. Clinical picture usually presents as acute coronary syndrome with chest pain and shortness of breath as the main symptoms.[1,2] This is the case of TCM in a woman manifested as acute respiratory failure due to acute cardiac decompensation after tracheal extubation. She was submitted to the surgery of intraabdominal collection evacuation in short general endotracheal anesthesia.

CASE REPORT

A 59-years old Caucasian woman was admitted to the intensive care unit (ICU) due to respiratory insufficiency after tracheal extubation. She was submitted to the surgery of intraabdominal collection evacuation. After

general endotracheal anesthesia induction, she was mild hypotensive and heart rate decreased to 53 bpm during surgery.

Afterwards she was during the whole procedure hemodynamic stable due to optimalisation of anesthetic's dosing and intravascular volume. Upon the end of surgery, she was decurarized using neostigmine and atropine. Subsequently after recovery of satisfactory spontaneous respirations was extubated. Immediately after she complained of dyspnea, became respiratory insufficient and hypoxic. Pulse oximeter value dropped to 89%. At the same time, her heart rate increased to 160 bpm and blood pressure to 180/127 mmHg. Promptly she was intubated and mechanical ventilated. After bolus of amiodarone her heart rate decreased to 102 bpm. Her blood pressure was 125/70mmHg. She was analgosedated and transferred to the ICU.

She suffered from arterial hypertension, chronic obstructive pulmonary disease, epilepsio and hyperlipidemia. Two years ago, she had malignant hypertension complicated with non-ST-segment myocardial infarction (NSTEMI) and pulmonary edema. At the same time due to acute Leriche syndrome she was submitted to urgent aortobifemoral bypass graft surgery.

Two months later postponed coronarography excluded coronary artery obstruction and demonstrated ejection fraction of 56%, normal left ventricle contractility and systolic function with first degree diastolic dysfunction. One year ago, she underwent postoperative ventral hernia repair and adhesiolysis due to ileus. Her drug therapy included bisoprolol, antihypertensive combination of felodipine and ramipril, normabel, acetylsalicylic acid, clopidogrel, frusemide, levetiracetam and rosuvastatin.

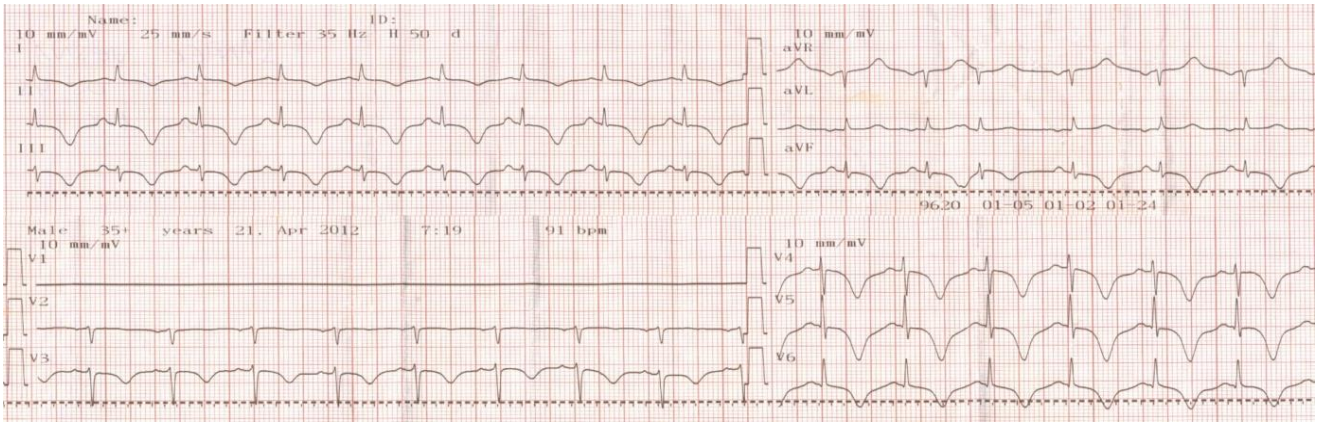

Figure 1: Admission electrocardiogram.

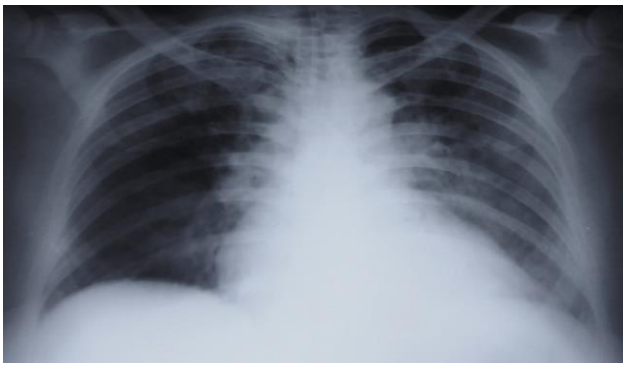

Figure 2: Admission chest x-ray which demonstrated left ventricle enlargement with apical shadow reaching left lateral thoracic wall and bilateral perihilar vascular redistribution with alveolar infiltrates.

At admission to the ICU she was analgosedated, intubated on mechanical ventilation, hemodynamic stable. Blood pressure was 130/80 mmHg, heart rate 102 bpm. On auscultation, inspiratory crepitation was heard over the left and right lung basal area to the level of scapula matching pulmonary edema. As well her heart auscultatory was tachycardia, no murmurs were heard. Further, her electrocardiogram demonstrated sinus rhythm 83 bpm, negative T wave in I, II, III, AVF and all precordial leads with mild ST-segment denivelation, and QT prolongation (QTc 528 msec) (Figure 1).

Emergency chest x-ray showed acute cardiac demo-pensation manifested with bilateral perihilar vascular redistribution and alveolar infiltrates. Left ventricle was enlarged and the apical shadow reached left lateral thoracic wall in comparison to the preoperative chest X-ray (Figure 2). Laboratory results revealed leukocytosis (L 16.8 x 109/L), CRP 11 mg/L, elevated D-dimers (7.59 mg/L), troponin T (cTnT 0.096 µg/L) and creatinine phosphokinase (CK 86 U/L) were normal. Her central venous pressure was 16 mmHg. After few hours, she became slightly hemodynamic unstable. Transthoracic echocardiogram revealed left ventricle heart failure with left ventricle ejection fraction of 25%, symmetrical

regional wall akinesias extending equally into the apical inferior and one third of lateral walls. Also, perfusion lung scan excluded acute pulmonary embolism, and multislice computed tomography angiography of the brain demonstrated no signs of acute ischemia, intracranial bleeding or expansive focal lesions. Control laboratory results demonstrated peak elevation of troponin T (O.114 µg/L).

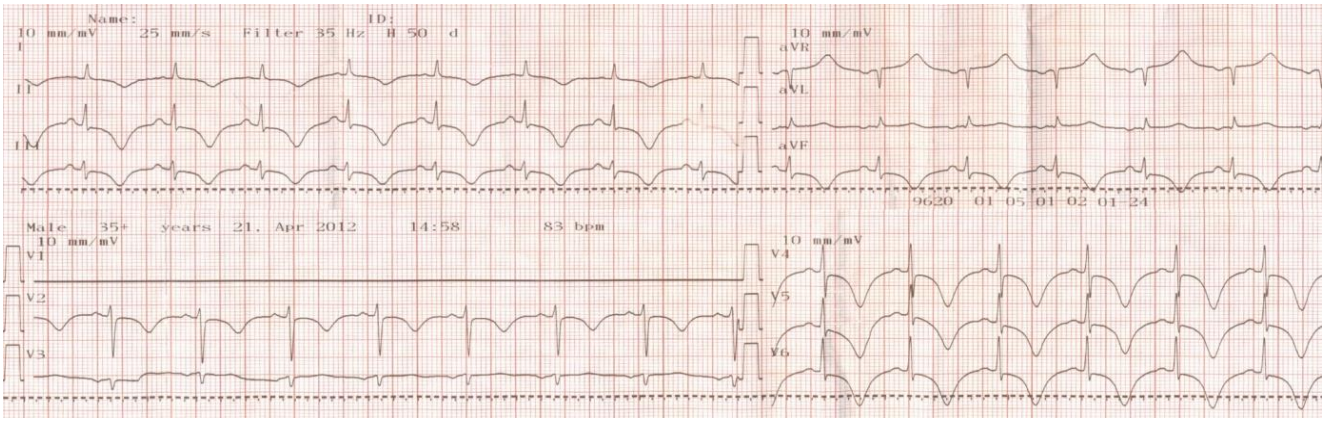

Figure 3: Electrocardiogram 24 hours after admission showing progression of T waves in almost all leads.

Follow-up electrocardiogram revealed progression of large and deep negative T waves with ST segment denivelation in almost all leads (Figure 3). Because of clinical picture of myocardial ischemia coronarography was indicated to exclude the acute coronary incident or TCM. Consiliar cardiology decision was made that because of recent operation, hemodynamic instability of patient and nonspecific elevated troponin T coronarography intervention will be deferred. The patient was closely monitored. Diuretic (frusemide) therapy, pharmacological inotropic (dobutamine) and vasopressor (noradrenaline) support together with antiaggregating (acetylsalicylic acid and clopidogrel) and anticoagulation (low molecular weight heparin) therapy were immediately started. Volume intake was also carefully restricted due to cardiac decompensation.

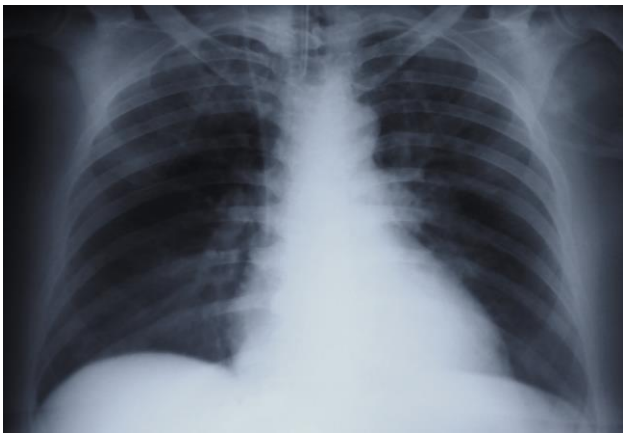

Figure 4: Follow-up chest X-ray after two days demonstrated regression of perihilar bilateral infiltrates and almost normal heart shadow.

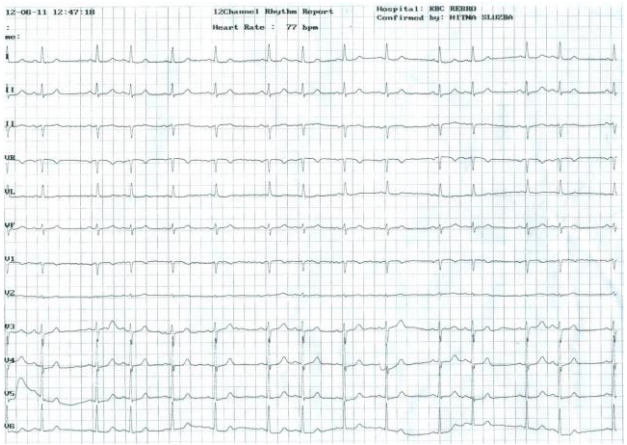

Figure 5: Follow-up normal electrocardiogram two months later after Takotsubo cardiomyopathy.

Follow-up chest x-ray after two days demonstrated regression of alveolar infiltrates and almost normal left ventricle shadow (Figure 4). As well follow-up transthoracic echocardiography done after three days confirmed enhancement of the left ventricle systolic function with ejection fraction of 50% and residual mild hypo contractility of the apical inferior and one third of left ventricle walls. Troponin T decreased to 0.088 µg/L. Electrocardiogram showed larger and deeper T waves with more expressed ST segment denivelation in the same leads as previous. Clinical progress, nonspecific low cardiac biomarkers, electrocardiographic, radiologic and transthoracic echocardiographic dynamics referred TCM as the cause of cardiorespiratory failure. Coronary angiography was postponed until she felt better. The patient was extubated after six days and sent to the

surgical ward after seven days. During her stay in the ICU she was afebrile. Inflammatory parameters were steady. Swab wound and tracheal aspirate were sterile. She was discharged from hospital after nine days uneventfully. Two months later her follow-up electrocardiogram was normal and she had no cardiac symptoms (Figure 5). Moreover, her postponed coronarography excluded detectable coronary artery obstruction.

DISCUSSION

Differential diagnosis of TCM in critical ill patient is still a great challenge. Anesthesia and surgery are stressful triggers for patient. This stress response begins with anesthesia induction and usually lasts 3 or 4 days after.[3] Although, our patient was slightly hypotensive after induction probably because of preoperative hypovolemia and anaesthetic hemodynamic depression, tracheal extubation was the main stressor for the TCM development. Interestingly, our patient had a history of NSTEMI two years ago with normal coronary angiography although she had generalized atherosclerosis, arterial hypertension, hyperlipidaemia and smoking as risk factor. We presume that she could had subcritical eccentric lesions of coronary arteries which can not be seen on coronary angiogram.[4,5] Yet, this facilitated coronary vasospasm or maybe transient plaque rupture in our patient secondary to catecholamine surge and led to NSTEMI as a consequence.[6] Endogenous catecholamine-induced myocardial stunning, ischemia-mediated stunning due to microvascular or multi-vessel epicardial spasm or dysfunction, myocardial metabolic dysfunction and microinfarction are proposed mechanisms for the TCM development.[7] These all mechanisms associated with loss of estrogen protective role on vascular system in postmenopausal women lead to endothelial dysfunction resulting in the myocardial regional wall motion abnormalities as it was in our case.[7,8] Moreover, women are more prone to microvascular disease which also contributes the TCM development.[9] Apical form is the most common TCM to greater density of the adrenergic receptors and its vulnerable structure.[8,10] Therefore the heart is very sensitive to sympathetic stimulation, particularly there is strong apical reaction.[6] Thus, it is not enough to emphasize that TCM is hard to recognize especially in surgical and critical care medical population. First, it is difficult to communicate with them because of changed state of consciousness. So, we usually rely on laboratory results, imaging tests, vital parameters and conciliar opinion to make definitive diagnosis. Secondarily, though Mayo criteria for the diagnosis of TCM have been proposed, for the hospitalized patient in the surgical ICU conditions are little different.[11] Apical ballooning syndrome should be differentially suspected in every patient with acute reduction of left ventricle systolic function in association with at least one of the following complications such as pulmonary edema, hemodynamic compromise, ventricular arrhythmias, troponin elevation and electrocardiographic evidence of ischemia, especially if there is no history of coronary artery disease.[7] Our patient developed acute left ventricle systolic reduction with bilateral pulmonary edema, ventricular tachyarrhythmia and mild hemodynamic compromise. Also, electrocardiographic ischemic changes were found. Despite coronary angiography is a standard method for coronary artery disease exclusion, our patient due to hemodynamic instability, low cardiac biomarkers and recent operation was followed-up and evaluated with chest x-ray and transthoracic echocardiography. The required coronary angiography was postponed and excluded significant coronary artery obstruction. Nowadays, echocardiography in TCM diagnosis is becoming first diagnostic imaging technique in clinical practice due its accessibility and early diagnosis.[12]

Treatment is supportive. Stressor must be immediately removed and appropriate treatment must be engaged.

CONCLUSION

To conclude, TCM should be considered as differential diagnosis in critical care patients who acutely develop left ventricle systolic dysfunction especially in association with pulmonary edema. Sometimes, chest x-ray and transthoracic echocardiography are the first imaging methods for making the definitive diagnosis of TCM in these patients in accordance to clinical course, exclusion of other differential diagnosis and medical history.

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