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A JOYOUS OCCASION INDUCED CARDIOMYOPATHY MANIFESTING AS NON ST SEGMENT ELEVATION MYOCARDIAL INFARCTION

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Contribution
RA conceived the idea of the case report. Data collection and manuscript writing was done by RA and AJK. All the authors contributed equally to the submitted manuscript.

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ABSTRACT

Takotsubo cardiomyopathy is a potentially reversible cardiomyopathy presenting as acute coronary syndrome. It presents with normal coronary and classical echo features. We are presenting a case that had an unusual trigger factor and unusual ECG presentation.

Keywords: ECG changes, reversible cardiomyopathy, Takotsubo cardiomyopathy

INTRODUCTION

Takotsubo Cardiomyopathy (TTC) has a transient left ventricular apical ballooning appearance, which remains undiagnosed until a coronary angiography is performed in the absence of coronary obstruction. It has a similar presentation as acute myocardial infarction and represents 1-2% of acute coronary syndrome (ACS) cases.[1,2] The highest prevalence is found in postmenopausal women, and it requires a specific trigger that causes an alteration in the catecholamine hormonal receptor sensitivity due to insufficient estrogen production.[2,3] However, the exact pathophysiology remains unknown, even the theory of catecholamine induce cardiomyopathy is still not well established as the main mechanism. It could indirectly cause myocardial toxicity and spasm rather than be the cause in cardiomyopathy processes. In a population with normal cardiac function with no underlining comorbidities or other high risk features, the majority (90%) has a good prognosis. A worse prognosis is expected in males, a physically stressed status or concomitant arterial fibrillation (AF).[1,4]

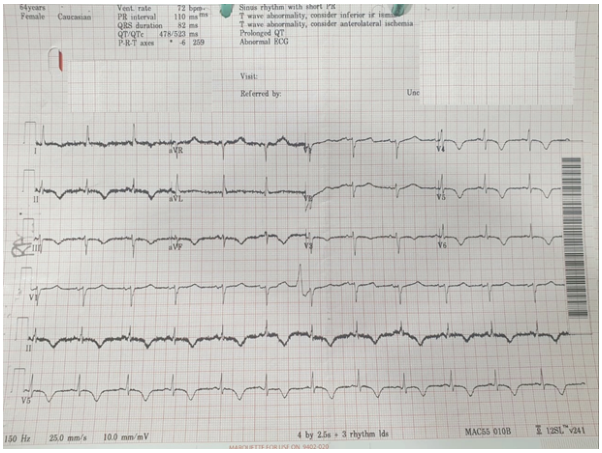
CASE REPORT

A 64-year-old female patient presented at the Emergency Department (ED) complaining of progressive typical chest pain for 3 days, which were becoming more frequent and severe. A day before the presentation she was dancing at a wedding. The patient has a medical history of hypertension and is prescribed on telmisartan and bronchial asthma, no history of diabetic mellitus, dyslipidemia and nor smoking. With each attack there is diaphoresis, palpitation and severe shortness of breath. She reported that she has a stressful life with a low income and many responsibilities.

She reported a previous attack in 2017, mimicking the current event but milder. At that time, she presented to our clinic with an unremarkable EKG and troponin I (TnI) level, the Echocardiogram (ECHO) showed normal left ventricular wall thickness, with an Ejection Fraction of 54%, normal filling pressure, and no regional wall motion abnormalities was noted. A cardiac CT was done which indicated normal coronary arteries, no evidence of mural plaques or luminal stenosis and the rest of examination were unremarkable.

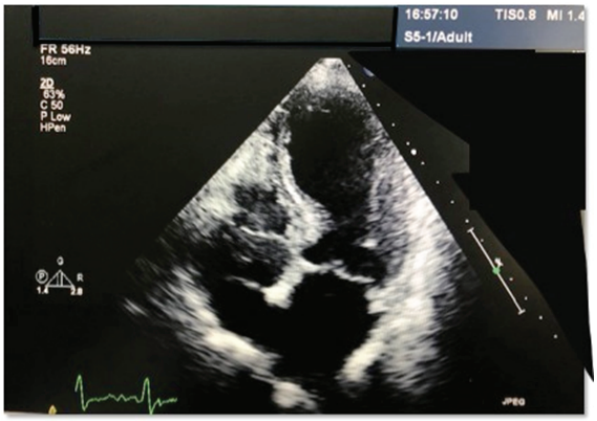
With the current admission, the clinical examination was unremarkable, she was alert and oriented, with stable vital signs, the cardiovascular examination was unremarkable and a chest examination indicated equal bilateral vesicular breathing. The EKG showed normal sinus rhythm with Diffuse T Wave Inversion (Figure 1) and the troponin I (TnI) level on presentation was 7410.3 (normal< 34.20 pg/mL)

Figure 1: ECG showing deep T inversion in pericardial leads



The cardiac angiography showed normal coronaries with distal apical ballooning noticed at the LV angiogram, highly suggestive of a stress-induced CM. The TnI level, after the angiography, reduced to 510 on the day of discharge. A repeated Echo indicated grossly, a normal left ventricle in size with normal wall thickness, with a mildly reduced left ventricular systolic function ejection fraction (45%), and with severe apical wall hypokinesis (Figure 2).

Figure 2: Echocardiogram showing apical ballooning



The diagnosis of Takotsubo was contemplated, with the differential diagnoses of myocarditis and pheochromocytoma considered to ensure the case complied with the Mayo Clinic's diagnostic criteria. She was discharged with ACEL and BB.

DISCUSSION

Our patient was diagnosed with TTC after the angiography. The interesting finding in this case is the nature of the provoking factors that were dancing and joyful emotions. Although she has a history of two risk factors, a chronically

stressful life and a depressed mood, it has been reported that 55.8% of patients diagnosed with TTC have a history of neurological or psychiatry disorders compared with 25.7% of patients diagnosed with ACS.[1] However, the triggers were found to be a combination of both physical and joyful emotions as potent stimuli, and due to the patient's response, she subsequently developed TTC. The underlying factor is an increased level of catecholamine, which can have a direct toxic effect on the myocardial and microvasculature and result in vasospasm.[1] Unfortunately these phenomena not well established and its association with cardiomyopathy is unclear. Other interesting finding in this case was the electrophysiological manifestations indicating T-Wave inversion (TWI), which occurs infrequently in the cases reported over a decade. T-Wave inversion can be observed in TTC as the only manifestation to identify the underlining myocardia dysfunction or sometimes, it presents as dynamic changes. This sign can present from the beginning and peak on 2.3+/-2.0 days. It can also be persistent for months and even after the discharge from the hospital for 7.6 days.[5] According to literature, being observed in the pericardial leads is preferable. Our patient's EKG indicated TWI in V3 to V6 and the inferior limb leads, which is suggestive that the most affected part is the apical side. The sign disappeared at Day 3, as expected, when she was discharged.[1] Another observed sign was a prolonged QT (523), frequently seen in TTC patients, rather than a patient with AMI. However, both signs could reflect the presence of a myocardial repolarization abnormality.[1]

In clinical practice, a useful way to diagnose TTC is to use the Revised Mayo Clinic Criteria, which include four criteria that should be satisfied. The presence of a transient left ventricular systolic dysfunction as well as the absence of coronary obstruction or plague rupture on an angiogram. The EKG findings include ST elevation, with or without TWI or a moderate elevation in troponin. The forth is to exclude other differential diagnoses that mimic the same condition such as pheochromocytoma and myocarditis.[1] It should be noted that the presence of coronary disease does not exclude the diagnosis if the region of the wall abnormality is not related to the affected coronary.[2] There is some parameters reported, that if observed, are suggestive of a TTC diagnosis rather than an AMI. These parameters include ST segment depression in the AVR with a specificity of 95%, or a high peak level of troponin and a high level of BNP/pro-BNP. Practically, cardiac catheterization is performed in all cases.[1]

TTC is considered as a benign condition in a patient with no underlining comorbidities, high risk factors or an independent factor for mortality that may cause major adverse events.[1] A mortality rate of 5% has been reported. The International Takotsubo Registry reported ventricular tachycardia (3.0%), ventricular thrombus (1.3%), and ventricular rupture (0.2%) as causes of mortality. The highest mortality rate was reported in patients who developed TTC secondary to neurological diseases, physical activities, medical conditions or procedures.[1] In terms of therapy, ACEI/ARBs have showed a survival benefit superior to BB.[2] Recurrence time varies between 25 days and 9.2 years with an annual rate of 1.5 %.[3]

Takotsubo cardiomyopathy should be considered in patients with ACS, normal coronary and classical echo features. Triggers, either emotional or physical, may also provide a clue, especially in post-menopausal women.

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