CASE REPORT

Takotsubo cardiomyopathy: A case report

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Abstract

Takotsubo Cardiomyopathy is a cardiac syndrome of rare type involving apical akinesis of the left ventricle. Symptoms of acute myocardial infarction, i.e. chest pain, S-T changes, and positive cardiac enzymes, are observed in patients with Takotsubo Cardiomyopathy. Cardiac angiography of Takotsubo Cardiomyopathy patients manifest left ventricular apical ballooning with no remarkable coronary artery stenosis. Mostly the cases are managed on the lines of treatment for Acute Coronary Syndrome. We present a case of Takotsubo Cardiomyopathy in a teenage girl from Karachi, Pakistan. The prevalence of Takotsubo Cardiomyopathy in Pakistan is yet to be mapped due to its rarity.

Keywords: Takotsubo Cardiomyopathy, Rare Cardiac Syndrome, Teenage Girl, Karachi, Pakistan.

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Introduction

Takotsubo Cardiomyopathy (TC), first discussed in Japan in 1990s, is a severe cardiac condition involving left ventricular apical ballooning.[1] It presents as acute myocardial infarction (MI). The reported cause of this syndrome is associated to an episode of emotional or physical stress.[2] The prevalence of Takotsubo Syndrome is estimated to be 2% of all reported cases of ACS, and as high as up to 5% if considered only in women.[3] It is very pertinent to establish this diagnosis, as patients with conditions similar to an uncomplicated ACS may present to the emergency department (ED). Here, we discuss the case of a 15-year-old girl who presented to the emergency department with complaint of chest pain. Upon investigations, ECG changes, with raised cardiac markers was observed, and was subsequently diagnosed with Takotsubo Cardiomyopathy.

Case Report

A 15-year-old girl, resident of Karachi, Pakistan, presented to the Emergency Department of PNS Shifa Hospital, Karachi, on November 16, 2021. She had no known medical history, and reported with the complaint of typical chest pain of moderate nature associated with dyspnoea for the past four hours. An electro cardiogram (ECG) was performed which showed ST Segment elevation in pre-cordial leads from V1 to V5 and inversions of T wave in II, III & aVF (Figure-1). Initial Trop-I value was 8.83 ng/ml (< 0.3 ng/ml). Her vitals were recorded to be: heart rate of 118/min, BP 90/60mm of Hg, respiratory rate 24/min, and O2 saturation of 97% on air.

ST segment Elevation Myocardial Infarction (STEMI) was first considered and accordingly an invasive approach was used. The patient was shifted to catheterisation laboratory where no evidence of obstructive lesions was observed in coronary arteries during the procedure (Figure-2).

The subsequent Serum Troponin levels of the patient were recorded to be 8.830 ng/ml (< 0.3 ng/ml) and 1.04 ng/ml (< 0.3 ng/ml). Her NT-pro BNP levels were 3262 pg/ml (< 125 pg/ml). Echocardiography showed an ejection fraction of 30% with severe generalised LV dysfunction along with trace Tricuspid Regurgitation and minimal pericardial effusion. During detailed anamnesis, the patient revealed

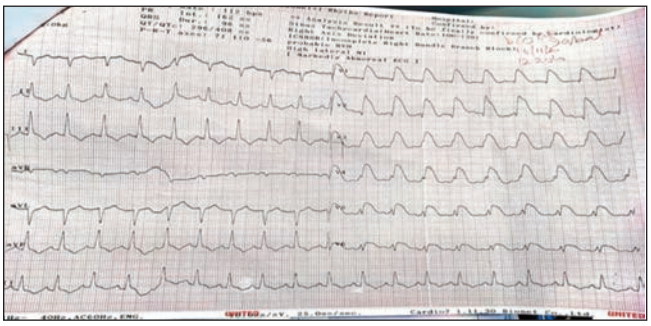

Figure-1: ECG recording at presentation indicating ST Segment elevation and T wave inversions.

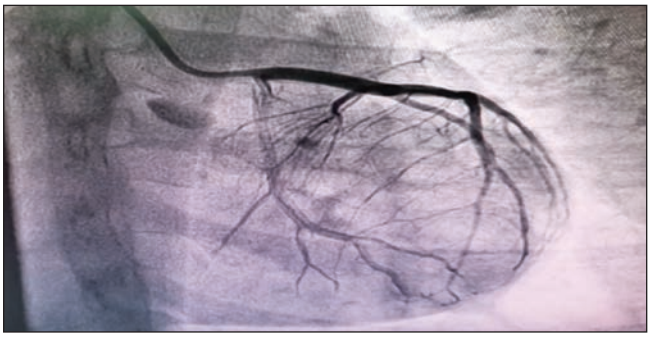

Figure-2: Coronary angiography shows no blockage in arteries.

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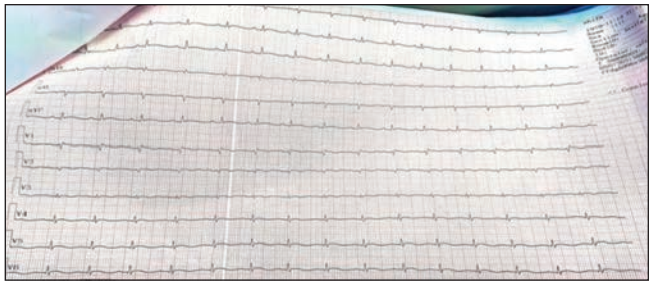

Figure-3: ECG recording on the eighth day of hospitalisation showing resolution of ST segment elevation to inverted T waves in precordial leads.

that watching her elder brother's ill health owing to dengue fever and the traumatic experience of seeing him having febrile seizures, she started to experience chest discomfort. Based upon ECG, echocardiography and normal coronary arteries, initial diagnosis of Takotsubo Cardiomyopathy (TC) was made. The patient was treated with potassium sparing diuretics (Aldactone), Angiotensin Converting Enzyme Inhibitors (Captopril), and Asprin. In the follow-up Echocardiogram, LV function showed improvement with an ejection fraction of 45%. Upon repeat ECG, ST Segment elevations were noted to have resolved with absence of Q wave formation over a period of eight days (Figure-3).

Discussion

Takotsubo Cardiomyopathy was first diagnosed in Japan in 1990. It is considered to be stress-related cardiomyopathy syndrome. It is a rare type of transient LV dysfunction. Patients with Takotsubo Cardiomyopathy are observed to have typical symptoms of acute myocardial infarction (MI).[4] It usually occurs as a result of stimulation of catecholamine from adrenal glands. Higher levels of catecholamine have been observed in patients of Takotsubo Cardiomyopathy as compared to that of Myocardial Infarction and Heart failure.[5] The exact cause of Takotsubo Cardiomyopathy still remains unclear, however, it is postulated that macrovascular and microvascular dysfunction occurs as a result of catecholamine release.[6] During coronary angiography, obstructive coronary artery disease evidence is not noted. Moreover, repeat echocardiography in Takotsubo Cardiomyopathy patients exhibit normal readings of LV function, with no abnormalities in wall motion and normal ejection fraction in a few weeks. No ECG criterion has yet been established that enables to distinguish among ST-segment elevation observed in Takotsubo Cardiomyopathy and MI.[7] As regards ECG changes, they tend to resolve in most of cases over a period of few weeks to months. During an episode of Takotsubo Cardiomyopathy, beside temporary nature of ECG changes slight increase in Trop-I or creatinine kinase–MB (CK-MB) is common.[2] The distinguishing feature among Takotsubo Cardiomyopathy and MI is that in MI myocardial necrosis takes place, while in Takotsubo Cardiomyopathy cardiac insult results into myocardial stunning. The following changes in ECG indicate myocardial stunning:

a. Symmetric T-wave inversion in all leads.

b. QT interval prolongation

c. R wave progression is absent

d. PR interval is prolonged

Allied complications of Takotsubo Cardiomyopathy are not that common; however, it may include unstable BP, heart failure, and arrhythmias of atria and ventricle. An active blockade in the LV outflow tract is caused due to the wall motion abnormalities in apical and mid ventricular region of the heart.[8]

Patients with Takotsubo Cardiomyopathy are initially managed on the lines of MI. Urgent coronary angiography is done to look for arterial occlusion. All CAD patients are kept on aspirin, heparin, and ACE inhibitors. If need be, β-blockers and diuretics can also be administered. Stress Cardiomyopathy is mostly managed empirically besides adopting a standard treatment protocol for congestive heart failure.[9] Sympathetic stimulation is assumed to be one of the triggers in Takotsubo Cardiomyopathy. Routine usage of β-blocker after recovery from Takotsubo Cardiomyopathy is not recommended unless indicated for any other reason.[10] The patients who do not respond to intravenous fluid and/or β-blockers and experience an active LV outflow tract issue are recommended to be maintained on phenylephrine. Patients with hypotension can be kept on inotropic support and intra-aortic balloon pump counter pulsation is also considered useful. Haemodynamic instability is considered to be linked to transient right ventricular dysfunction. One third of the reported Takotsubo Cardiomyopathy patients suffer from transient right ventricular dysfunction. Transient right ventricular dysfunction is more severe cardiac condition and involves prolonged hospitalisation.[11] The rate of recurrence of Takotsubo Cardiomyopathy is 1.5-2% per annum.[12] Besides the probability of relapse, Takotsubo Cardiomyopathy may have an impending mortality risk.[13,14]

Conclusion

Takotsubo Cardiomyopathy is a cardiac condition which is increasingly being reported world over due to better awareness. The pathogenesis of the disease has not yet been fully comprehended, however, one of the triggering mechanisms is considered to be catecholamine release. The patients with Takotsubo Cardiomyopathy present with similar symptoms as that of ACS and clinicians need to establish correct differential diagnosis for appropriate

treatment regimen.

Consent: Written consent of the patient was obtained for publishing the case ananomously for promotion of science.

Disclaimer: None.

Conflict of Interest: The co-author is member of Institutional Ethical Committee, PNS Shifa Hospital, Karachi.

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