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Case Report

Case Report on Takotsubo Syndrome; A Life-Threatening Storm

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Abstract

Takotsubo syndrome (TTS) or stress cardiomyopathy, mimics acute coronary syndrome with normal coronary vasculature on angiography. International literature has described Takotsubo Syndrome in association with thyrotoxicosis. The authors report a case, the first of its kind in Pakistan, illustrating the presentation of Takotsubo Syndrome in a young man with thyrotoxicosis that resulted in a fatal outcome. Thus, emphasizing the need for prompt recognition of TTS as a preliminary diagnosis in the Emergency Department (ED).

Keywords: Takotsubo Syndrome, Stress cardiomyopathy, Thyrotoxicosis, STEMI, Cardiogenic Shock.

Introduction

TTS is a rare malady of transient, acute systolic, and diastolic left ventricular (LV) dysfunction; often triggered by physical, stressful, or emotional trauma. The clinical presentation is synonymous with acute coronary syndrome (ACS), with an estimated prevalence of 1- 2% of patients with suspected ACS, carrying an in-hospital mortality rate of 5% [1,2]. Several cases linking TTS with thyroid diseases have been reported in international literature. However, to the author's best knowledge, this case is Pakistan's first of its kind. The authors report a case that turned up in the Emergency Department (ED) of The Indus Hospital and Health Network (IHNN), Karachi, Pakistan.

Case Report

A 32-years old gentleman, New York Heart Association Class 1 [11], known hyperthyroid, (uncontrolled) for two years, presented on 6th December 2021, at the Emergency Department of The Indus Hospital and Health Network (IHHN), Karachi, with chest pain accompanied by acute shortness of breath for 1 day; fatigue for 4 days. The patient reported typical chest pain, sudden onset, and exertional exacerbation 24 hours back. He reported the development of paroxysmal nocturnal dyspnea (PND), orthopnea, and non-productive cough at the same time. On further inquiry, episodic bilateral tremors of hands were reported for the past year along with paroxysmal cold sweats and palpitations. There were no associated symptoms of fever, flu-like illness, nausea, vomiting, diarrhea, vertigo, neurological deficits, or obvious deformity. The rest of the systemic review was unremarkable.

Upon ED presentation, his vitals were a blood pressure of 122/85 mmHg, heart rate 100/min, temperature 97.8F, respiratory rate 20/min, oxygen saturation of 87%. On general physical examination, he was well oriented but restless and agitated. Chest auscultation revealed decreased air-entry on the right side with crepitation and occasional wheezes on the left. The rest of the physical examination was within normal limits.

His ECG revealed ST-segment elevation involving the anterior chest leads, most notably in V1-V4 (Figure 1). As per ACS protocol, oral aspirin and clopidogrel were administered along with supportive oxygen therapy.

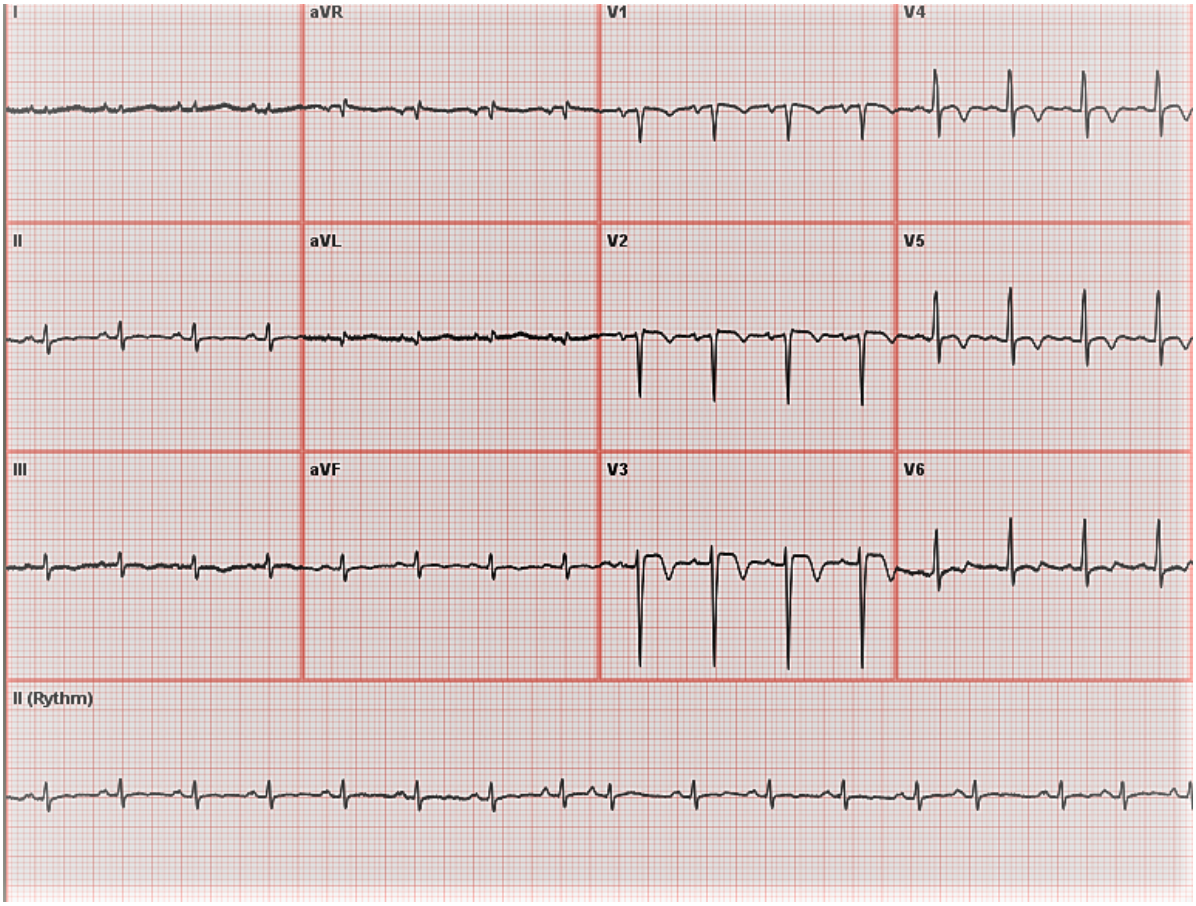

Figure 1: ECG showing normal sinus tachycardia Q waves in V1-V2 leads suggestive of septal infarct with ST-segment elevation V2, V3, V4, and deep T wave inversion in V3, V4, V5, V6).

Blood workup revealed a high sensitivity cardiac Troponin I (hsCTrop-I): 117ng/L (normal range: males 34.2ng/L; females 15.6ng/L). Total bilirubin: 2.85mg/dl (normal range: 0.3-1.2mg/dl), Direct bilirubin: 0.77mg/dl (normal range: 0-0.5mg/dl), Serum Sodium: 127mEq/L (normal range: 135-145mEq/L), Serum Potassium: 5.4mEq/L (normal range: 3.5-5.1mEq/L), and Thyroid-stimulating hormone (TSH) level: <0.0083mIU/L (normal range 0.4-4.4mIU/L). A complete blood picture, urea, and creatinine were normal.

The patient was rushed to the angiography suite after a preliminary diagnosis of STEMI was established. The coronary angiogram denoted normal coronary vasculature. (Figure 2a). Post Catheterization, a portable echocardiogram revealed severe left ventricular dysfunction with global involvement and an ejection fraction of 25%, segmental wall motion abnormalities, and dilated minimally pulsatile Inferior vena cava (Figure 2b). A plain chest radiograph revealed cardiomegaly with bilateral bulky hila and pulmonary edema with right-sided pleural effusion.

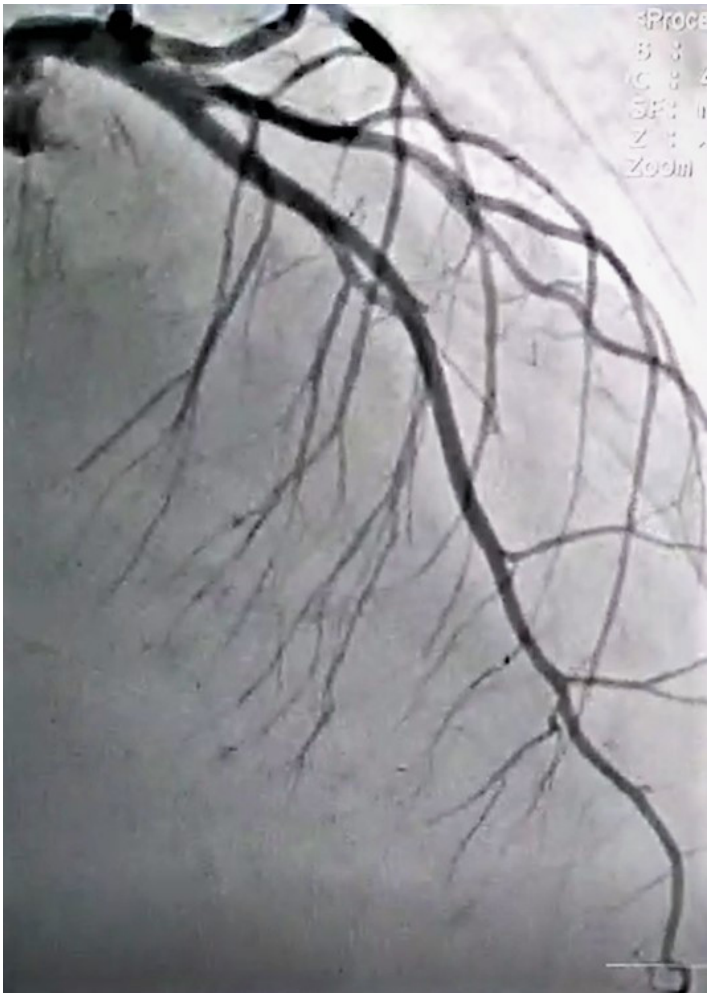

Figure 2a: Left heart catheterization showing normal coronary vessels.

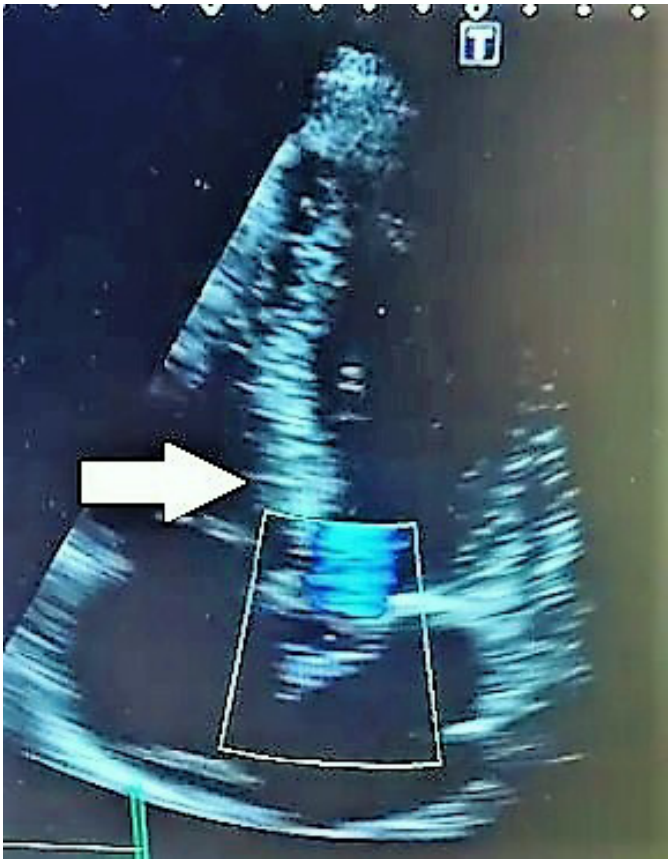

Figure 2b: A 2D image from transthoracic echocardiography showing septal movement with apical ballooning of the LV.

Post-procedure, the patient was hemodynamically unstable and was taken on ionotropic support and subsequently intubated. Soon after, the patient became pulseless and CPR was performed according to advanced cardiac life support (ACLS) guidelines. Unfortunately, ROSC was not attained Thirty minutes into the CPR.

Consent was obtained from the patient's father for the purpose of publishing this case report with pictures whilst maintaining confidentiality.

Discussion

Takotsubo syndrome is an entity with acute, reversible, and transient left ventricular wall motion dysfunction along with chest pain, ECG changes, mild rise in cardiac biomarkers, and normal coronaries on angiography [1] as observed in this case report. We used the following Modified Mayo's criteria for diagnosis [3].

1. Short-lived akinesis, dyskinesis, or hypokinesis of the midsegments of the left ventricle, with or without the involvement of apex; the wall motion abnormalities should extend beyond a region supplied by a single epicardial vessel; a stressful trigger is usual but not always.
2. Lack of obstructive coronary disease or any evidence of plaque rupture on angiography.
3. New ECG findings like ST-Elevations and/or T-wave inversions with a moderate rise in cardiac troponin.
4. Without pheochromocytoma or myocarditis.

On the grounds of absence of a history suggestive of viral illness along with clinical features of normal ECG findings and a frequent rise in troponin, myocarditis was ruled out in our patient; such has also been observed by Taghavi et al [4]. Similarly pheochromocytoma was ruled out based on an international consensus, stating pheochromocytoma to be a physical trigger for Takotsubo syndrome[5]. Hence a diagnosis of Takotsubo syndrome was made in our patient [6].

The exact etiology for Takotsubo syndrome remains dubious, with elevated levels of catecholamines in the plasma, microvascular dysfunction, inflammation, coronary spasm, myocardial infarction, and estrogen deficiency being some of the suggestive mechanisms [1].

Thyrotoxicosis, mimicking a state of adrenergic excess was found to be an instigating trigger for TTS in our case and many other cases found in international literature [7-8]. TTS is typically found in postmenopausal women [1] but the International Takotsubo Registry concluded that 10% of the patients with TTS were less than 50 years old, of which most were males [8].

Literary evidence has shown TTS to be associated with complications and mortality similar to the ACS [1]. Chanavuth et al. reported increased mortality in males with TTS, with a high incidence of cardiogenic shock and major arrhythmic events [9]. Our patient was a young male who developed secondary takotsubo syndrome complicated by severe pump failure from the stress of thyroid storm, culminating in death. Many cases of Takotsubo syndrome have been reported in Pakistan but, none has depicted an association between TTS and hyperthyroidism in young males [10].

This case report brings forth the skeptical etiology and presentation of TTS in our population linked to hyperthyroidism. It highlights the importance of timely identification and management of this disease to reduce morbidity and mortality.

Conclusion

Takotsubo syndrome, mimicking acute coronary syndrome precipitated by thyroid storm in young men is rare and should be considered at the time of presentation to improve patient outcomes and mitigate catastrophes.

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