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TAKOTSUBO CARDIOMYOPATHY- A DISEASE IN DISGUISE

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Contribution

NK conceived the idea. AGI, AWK and AAR helped in final draft of case report and acquisition of figures. All authors approved the final version for publication and ensured the accuracy and integrity of the case report.

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ABSTRACT

We are reporting a case of 75-year-old woman, who presented in E.R of Tabba Heart Institute, with complains of acute chest pain and shortness of breath. Initially patient was diagnosed and treated as acute coronary syndrome. Coronary angiography showed minimal atherosclerosis in epicardial arteries. Left ventriculogram showed apical ballooning with hyper contractile basal segments. Cardiac enzymes were elevated. Echocardiography revealed similar findings suggestive of Takotsubo cardiomyopathy (TCMP). She was managed medically and was discharged home in stable condition. At four months follow-up she was asymptomatic with normalization of ECG and ECHO findings. Takotsubo Cardiomyopathy should be considered in the differential diagnosis along with acute coronary syndrome in postmenopausal patients presenting with angina with or without stress or ECG changes and positive cardiac enzymes.

Key Words: Anterior wall myocardial infarction, Takotsubo cardiomyopathy, Postmenopausal women, Left ventriculogram.

INTRODUCTION

Takotsubo Cardiomyopathy (TCMP) or broken heart syndrome was first described by Sato et al in Japan in 1990. [1] In Japanese 'tako tsubo' translates to 'octopus pot' which depicts the morphological appearance of heart on left ventriculography in patients with TCMP. [2] It is a non-ischemic acute condition that is triggered by emotional or physical stress, however in rare cases it can be idiopathic. [3] It is characterized by transient LV dysfunction, electrocardiographic changes that can mimic acute anterior wall myocardial infarction (AWMI) with minimal elevation of cardiac enzymes in the absence of obstructive coronary artery disease (CAD) on coronary angiography. [2] TCMP is usually misdiagnosed in emergency department as acute coronary syndrome until angiography depicts normal or minimally diseased coronary arteries and typical appearance of left ventricle on left ventriculography as happened in our case.

CASE REPORT

A 75 year old female with cardiac risk factors of obesity, hypertension and old age, presented in Emergency Department with acute onset of chest pain for 7

hours radiating to back associated with shortness of breath. Her heart rate was 100 beats per minute, regular and blood pressure was 135/79 mmHg. Initial ECG revealed deep T wave inversions in leads I, II, III, AVF and V2-V6 (Figure 1).

Troponin I was elevated at 8.87 ng/ml. The patient was misdiagnosed as ST segment elevation myocardial infarction (STEMI) and was started on Streptokinase infusion however it was stopped due to significant hypotension. She was urgently transferred to cardiac catheterization laboratory. Coronary angiography revealed non-obstructive coronary artery disease. Left ventricular end diastolic pressure was significantly elevated at 45 mmHg. Left Ventriculography showed decreased ejection fraction of 30-35 % with "apical ballooning" and "hyper contractility at the base" (Figure 2).

Figure 1: Presenting ECG of Patient

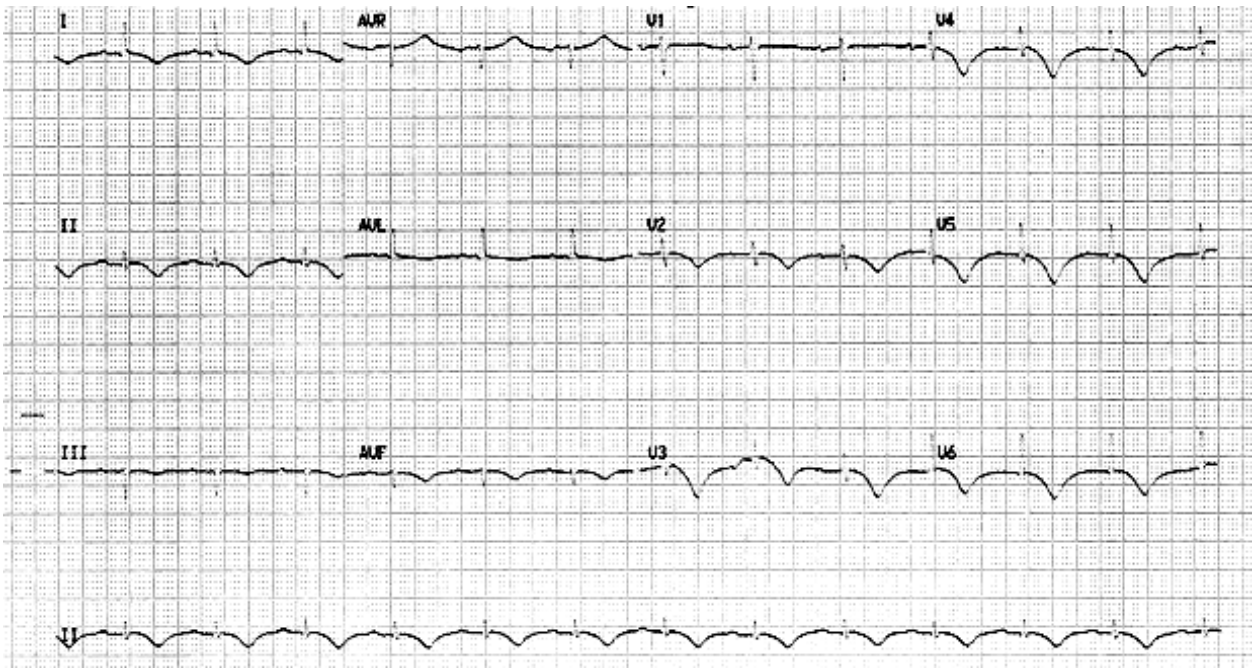
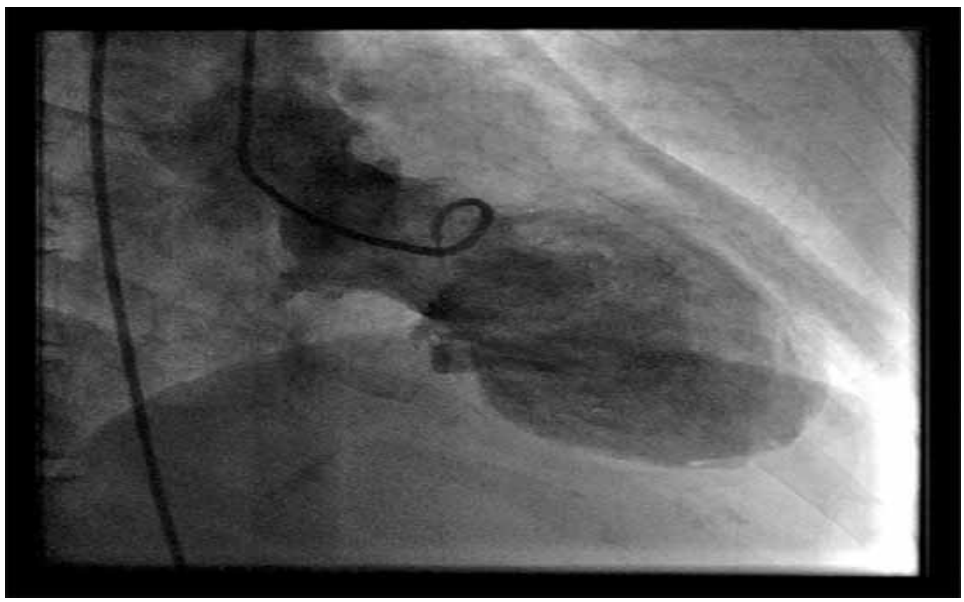


Figure 2: Left Ventriculography Showing Apical Ballooning



Echocardiography correlated with finding on left ventriculography. The patient was transferred to the coronary care unit for further management. She was started on beta blockers, angiotensin converting enzyme inhibitors (ACE-I), spironolactone, dual antiplatelets and anticoagulants. She remained stable and was discharged home after 72 hours. Repeat echocardiogram after 4 months showed normalization of her left ventricular function.

DISCUSSION

TCMP has been reported in up to 2% of acute coronary syndrome admissions in Japan, USA and Europe with 90 percent of the reported cases being post-menopausal women. Its initial principal symptom is chest pain like angina, followed by shortness of breath, making it difficult to make a distinction from acute coronary syndrome on the basis of symptoms.[3] One of the most characteristic features of TCMP is its association with wide variety of emotional or physical stressors ranging from quarrels, accidents, natural calamities, seizures, anaphylaxis, exercise stress test, electrical cardioversion, radiofrequency ablation, and sexual intercourse. Approximately one-third of patients do not have identifiable stress triggers.[3] The pathogenesis of TCMP is still unclear but studies suggest that increased catecholamine level in the blood of these patients is thought to be pathognomic for this disease.[4] Catecholamine increased levels might cause vasospasm of an epicardial coronary artery or microvasculature.[4] LV wall-motion abnormalities seen in these patients are most likely due to neurogenetically mediated myocardial stunning as reported by Akashi et al.[5] ST segment elevation in the precordial leads and dynamic and diffuse T wave inversions in most leads are the universal ECG findings as observed in our case as well.[6] The distinctive finding on echocardiography is left ventricular apical ballooning which is characterized by akinesis of the apical one-half to two thirds of the left ventricle, ensuing in low ejection fraction.[7] In order to rule out the ischemic origin of the apical ballooning coronary angiography is performed which mostly reveals normal or non-obstructive disease in epicardial coronary arteries. This when coupled with echocardiographic or left ventriculographic findings, suggests the diagnosis of TCMP. Diagnosis of TCMP can be made if all four conditions of modified Mayo Clinic criteria are met:

(1) Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present.

(2) Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.

(3) New electrocardiographic abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin.

(4) Absence of pheochromocytoma or myocarditis. TCMP has favorable prognosis.[3,8]

Heart failure, pulmonary edema and cardiogenic shock are found to be the most fearsome complications.[3] In order to ensure a complete resurgence of left ventricular dysfunction a repeat echocardiogram is advised 4 to 8 weeks after discharge. Without any consensus treatment guidelines, the management plan of TCMP is solely supportive in nature and dependent on case outcomes. Most experts recommend beta blockers and ACE-I in the acute phase for the postulated sympathetic over activity and left ventricular dysfunction respectively. To avoid recurrence beta blockers are indicated for long-term use.[3] This however is challenged by recent data which revealed inadequate efficacy of beta blockers, calcium channel blockers and nitrates in recurrence prevention.[9] Our patient was treated as per these guidelines and she recovered fully. Recurrence occurs in < 10% of patients.[10] There is no evidence to support use of long-term medical therapy to reduce the risk of recurrence.[11]

CONCLUSION

TCMP is a relatively less common but not a rare entity. It remains an important differential diagnosis of acute coronary syndrome, especially in post menopausal women. The diagnosis remains eluded until the coronary angiogram shows normal or minimally diseased epicardial coronary arteries along with the typical apical ballooning appearance of the left ventricle on imaging. This disease has rarely been reported in Pakistan probably because of under diagnosis due to lack of awareness, which is pivotal for the diagnosis of this disease. Numerous variables have been identified as a stress factor in majority of cases but many of the cases remain idiopathic like our case. There is no recommended duration and strategy of treatment, however supportive and symptomatic measures including medications for left ventricular dysfunction have been used, which have shown successful recovery of left ventricular function.

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