An unusual diagnosis for chest pain – Takotsubo cardiomyopathy: A case report

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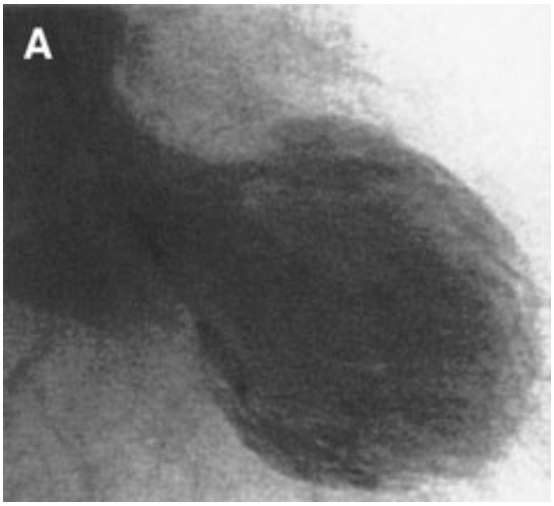
ABSTRACT

Takotsubo Cardiomyopathy or Stress Cardiomyopathy is a non-ischaemic cardiomyopathy which mimics acute coronary syndrome (ACS). It is becoming an increasingly reported condition, accounting for approximately 2% of all suspected cases of ACS. It is a well-recognized cause of acute heart failure, lethal ventricular arrhythmias, and ventricular rupture. Herein, we describe a patient who exhibited this syndrome and we review the existing literature on this condition and its associations. The case is about a 35 year old female who developed severe chest pain one hour following a normal vaginal delivery of twin babies. In view of her electrocardiogram and cardiac biomarkers, she was immediately diagnosed and managed as a case of ACS. But her subsequent investigations revealed a diagnosis of Takotsubo Cardiomyopathy. Her coronary angiography was normal and her echocardiogram which initially showed significant LV dysfunction improved drastically within a few weeks.

Key words: catecholamine, stress, echocardiography, takotsubo cardiomyopathy, ventricular dysfunction

INTRODUCTION

Takotsubo Cardiomyopathy is a transient cardiac syndrome that involves left ventricular apical akinesis. In Japanese, "takotsubo" means "fishing pot for trapping octopus," and the left ventricle (LV) of a patient diagnosed with this condition resembles that shape (Figure 1). Takotsubo Cardiomyopathy, which is typically precipitated by acute emotional stress, is also known as "Stress Cardiomyopathy" or "Broken-Heart Syndrome".



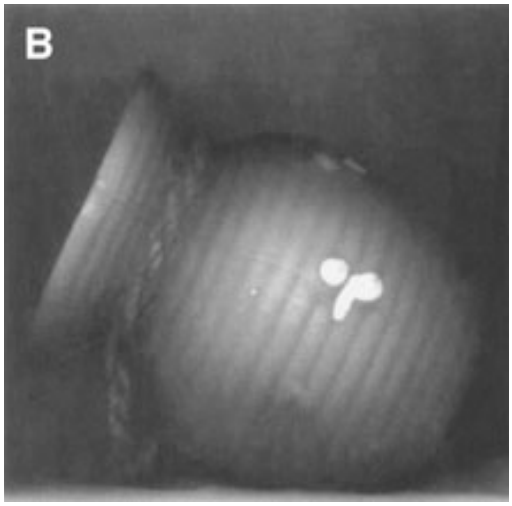


Figure 1. The shape of the LV (A) resembles the fishing pot for trapping octopus (B).

CASE REPORT

A 35-year old gravida five para four live four full term twin pregnant female presented with labor pain and subsequently had a normal vaginal delivery of twin babies. One hour following the delivery she developed severe typical substernal chest pain radiating to neck, jaw, back and left shoulder, associated with

vomiting, shortness of breath and profuse sweating. Her past medical history was insignificant.

Clinical examination revealed a regular pulse rate of 94 beats per minute, a blood pressure 170/100 mmHg, respiratory rate 20 breaths per minute and SpO2 97%. She was restless with presence of mild lower limb edema. Chest examination was normal. Cardiovascular system examination revealed S3 gallop rhythm. Abdominal examination revealed well-contracted and retracted uterus. Complete blood count, coagulation profile, liver function test, renal function test, fasting blood sugar were all within normal ranges. Electrocardiogram (Figures 2, 2a, 3, 3a, 4, 4a and 4b) revealed ST depression in leads II, III, aVF, and V3-V6 (down sloping). Cardiac markers were elevated (CKMb 50 U/L and Troponin I 2.58 mcg/L). Echocardiography revealed dilated left ventricle (LV), with multiple segmental wall motion abnormalities (SWMA) in the territories of the left anterior descending (LAD) and the right coronary arteries (RCA) and depressed LV function, with an estimated LV ejection fraction (EF) of 40% (Fig. 3). There was moderate to severe mitral regurgitation (MR). Lipid profile revealed mixed dyslipidemia (Total Cholestrol: 246 mg/dL, Tryglycerides: 251mg/dL, LDL 169 mg/dL, and HDL 26 mg/dL).

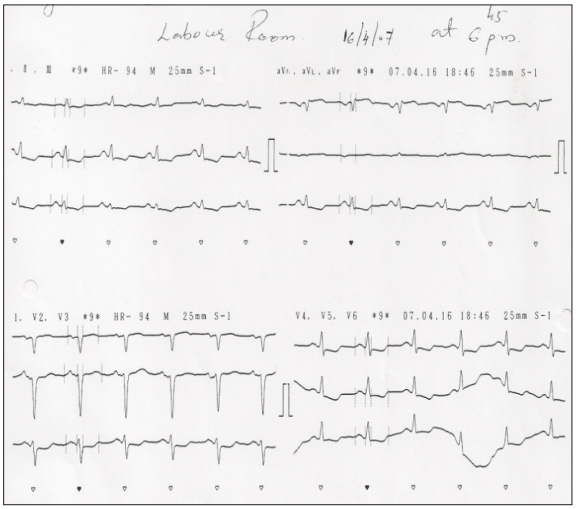


Figure 2. The 1st ECG of the patient taken inside the labour room one hour after delivery. It shows clear ST segment depression and T wave inversion in inferior, anterior and lateral leads.

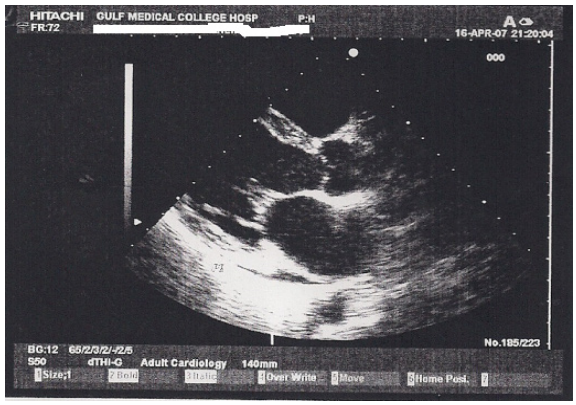
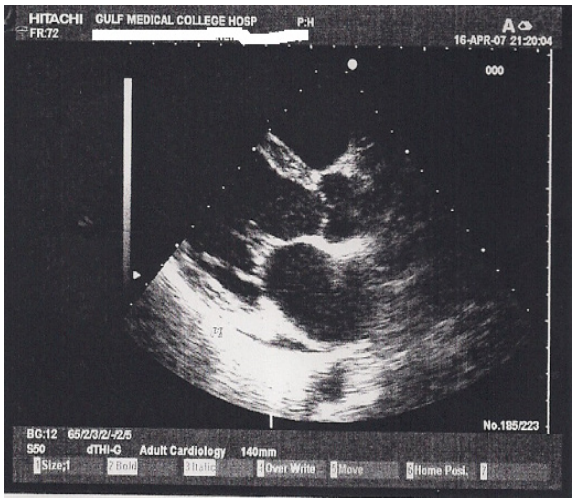
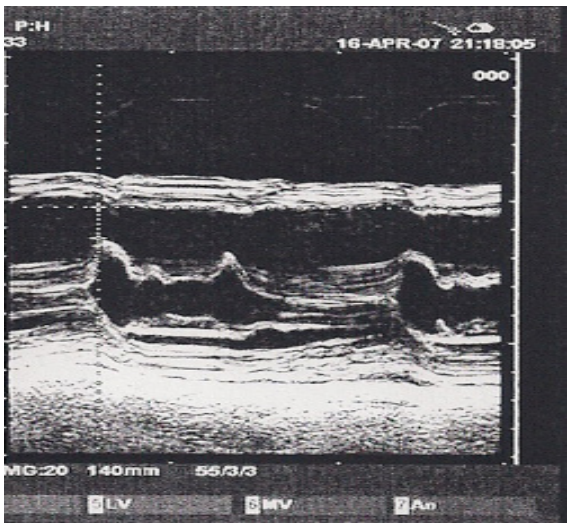
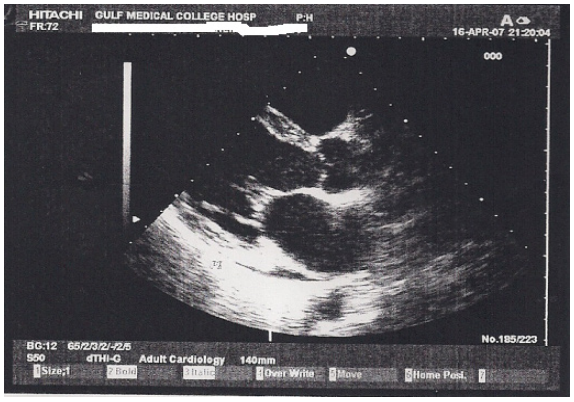


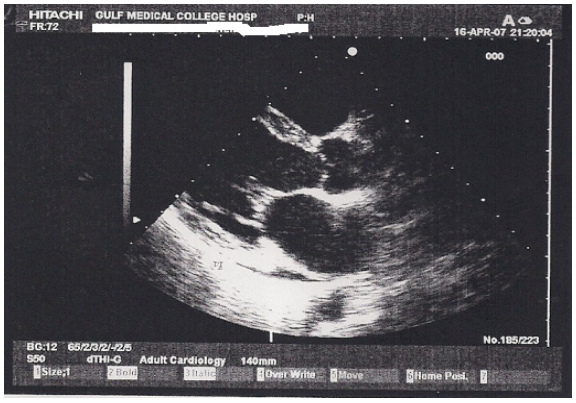
Figure 2a. Echocardiographic 2-D image obtained from the patient on the same day of admission. Note the dilated LV.

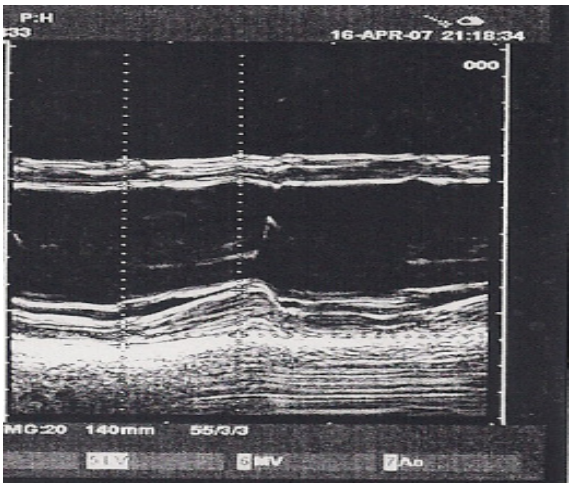




Figures 3 and 3a. M-mode echocardiographic image at the level of the mitral valve of the same patient. Note the decresed excursion of the anterior MV leaflet, a sign of Lv dilatation & dysfunction.







Figures 4, 4a & 4b. M-mode echocardiographic image at the level of LV. Note the dilated LV cavity and the akinesia at the septal region.

The patient was diagnosed as a case of ACS – Non ST segment elevation myocardial infarction (NSTEMI) and was started on anti-ischemic, antihypertensive and lipid lowering medications. At the time of discharge coronary angiography revealed normal anatomy of the coronary arteries. At subsequent visits, there was significant improvement of her symptoms. Two weeks later her echocardiography became normal with no SWMA, normal LV dimensions and an EF of 60%. She was advised lifestyle modification and kept only on antiplatelet drugs. ACE inhibitors and beta blockers were discontinued as she was lactating.

The patient has transient apical ballooning syndrome (TABS) or Takotsubo Cardiomyopathy which mimics ACS. It is associated with only moderate elevation of myocardial enzymes and within a few weeks, full recovery of left ventricular function.

DISCUSSION

Takotsubo Cardiomyopathy, also known as Transient Apical Ballooning Syndrome[1], Apical Ballooning Cardiomyopathy[2], Stress-induced Cardiomyopathy and Gebrochenes-Herz-Syndrome[3], mimics ACS. It is a type of non-ischemic cardiomyopathy in which there is a sudden temporary weakening of the myocardium. Since this weakening can be triggered by emotional stress, such as the death of a loved one, a break-up, or constant anxiety, the condition is also known as Broken Heart Syndrome[4].

Dote et al.[5] first described this syndrome in Japanese patients in 1990; the name relates to the peculiar shape of the left ventricle during systole on imaging studies, which resembles an octopus-fishing pot called a Takotsubo. It is becoming increasingly recognized around the world[2,4]. Several cases of this interesting cardiomyopathy have been reported in Japan[5-12], and more recently in the United States[13-17] and Belgium[18]. Studies reported that 1.7 - 2.2% of patients who had suspected acute coronary syndrome were subsequently diagnosed with Takotsubo cardiomyopathy (TCM)[14,19]. Literature review showed 57.2% were Asians, 40% were Caucasians, and 2.8% were of other races[20-23]. It also reported a mean patient age of 67 years, although cases of TCM have occurred in children and young adults[24-25] . Nearly 90% of reported cases involve postmenopausal women[26].

Although the exact etiology is still unknown, the syndrome appears to be triggered by a significant emotional or physical stressor. This is thought to be caused by excessive exposure to catecholamine mediated by exaggerated

sympathetic stimulation[16]. It is a well-recognized cause of acute heart failure, ventricular arrhythmias, and ventricular rupture[27]. Most complications of TCM occur during the acute phase of illness. The reported complication rate is about 19%[20]. Heart failure, cardiogenic shock and pulmonary edema occur in 3% to 46% of patients[15, 28-29].

The modified Mayo Clinic criteria for diagnosis of TCM can be applied to a patient at the time of presentation and must contain all four aspects:

- Transient hypokinesis, dyskinesis, or akinesis of the left ventricular midsegments, with or without apical involvement; the regional wall-motion abnormalities extend beyond a single epicardial vascular distribution, and a stressful trigger is often, but not always, present.
- New electrocardiographic abnormalities (either ST-segment elevation and / or T-wave inversion) or modest elevation in cardiac troponin level.
- Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
- Absence of pheochromocytoma or myocarditis[25].

Since TCM is initially indistinguishable from the classic acute ACS, immediate treatment should include management of coronary ischemia and pulmonary edema. This management includes antiplatelet, anticoagulant and antihypertensive agents. Once TCM is diagnosed, treatment is primarily supportive. Aspirin can be discontinued unless coronary disease or peripheral vascular disease is concomitant. β-Blockers may be continued long-term to protect against catecholamine sensitivity, which may predispose to this syndrome. Heparin and Coumadin should be used if apical thrombus is present, or a severe apical defect makes thrombus formation likely[15]. Close follow-up care with a cardiologist, usually with repeated clinical assessments and serial echocardiograms, is needed in the weeks after diagnosis to ensure resolution of the cardiomyopathy. Thereafter, annual clinical follow-up is advised, because the long-term effects and natural history of TCM are unknown[14,27,30].

The prognosis is excellent, with nearly 95% of patients experiencing complete recovery within 4-8 weeks. Even when ventricular systolic function is heavily compromised at presentation, it typically improves within the first few days and normalizes within the first few months[1,12,18]. The recurrence rate varies but is estimated at 3%[31]. Estimates of mortality rates have ranged from 1-3.2%[20,26].

CONCLUSION

The patient reported here is a case of presentation of Takotsubo Cardio-myopathy mimicking acute coronary syndrome. This condition needs further research as to clarify its pathophysiology, especially in relationship to women. Also important is distinguishing any potentially modifiable risk factors. Additionally, future research should focus on techniques for differentiating Takotsubo cardiomyopathy from classic acute coronary syndrome and on establishing standardized criteria for diagnosis and clinical guidelines for treatment and follow-up

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