



A CASE OF REVERSIBLE STRESS CARDIOMYOPATHY

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ABSTRACT

Various physical and psychological insults can cause acute and rapidly reversible left ventricular dysfunction. This is called stress cardiomyopathy or Takotsubo cardiomyopathy. The act of suicidal hanging involves intense physical stress and distressing emotional outburst. We report a case of rapidly reversible left ventricular dysfunction following attempted suicide by hanging. The pathogenesis of left ventricular dysfunction in hanging is multifactorial but the main mechanism is due to catecholamine surge causing myocardial stunning.

KEY WORDS: Stress cardiomyopathy, hanging, reversible LV dysfunction, and catecholamine surge.



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INTRODUCTION

Stress cardiomyopathy or Takoabito cardiomyopathy is characterized by transient, reversible hypokinesia of the distal interventricular septum and the apex of the left ventricle LV, with normal coronary arteries and was first reported in the Japanese population.¹ Various psychological and physical insults can cause this acute and rapidly reversible left ventricular dysfunction.² Physical stress like trauma, surgery, sepsis, envenomation due to scorpion sting and especially emotional distress can cause reversible left ventricular systolic dysfunction. This is a temporary effect which lasts a few days and is generally followed by complete recovery. Over recent years, increasing numbers of cases, arising in various circumstances of physical or mental stress, have been described in the literature. Nonetheless, only two reported cases brought on following a suicide attempt have been documented, one following the consumption of glufosinate ammonium herbicide intoxication³ and the other by hanging⁴. Suicidal attempt by hanging involves extreme physical stress and intense emotional outburst. We report a case of rapidly reversible left ventricular dysfunction following attempted suicide by hanging.

CASE REPORT

A 23 year old young married male was brought to the emergency room with history of attempted hanging from a ceiling fan using saree after an intense argument with his wife and family members. He had no other comorbidities. On examination he was restless and gasping for breath. He was drowsy and did not respond to verbal commands. His pupils were 3 mm each and reacting to light. Pulse was feeble and rapid at 134 bpm and his systolic blood pressure was 80/60 mmHg. His

oxygen saturation was 85% at room air. There was mark of hanging around his neck and no injury to cervical spines could be detected. On auscultation his heart sounds were feeble with no murmur with bilateral crepitations over his chest. His electrocardiogram ECG showed sinus tachycardia and hyper acute t wave changes at the time of admission. Chest X-ray was normal. His hemogram was within normal limits. His renal parameters and electrolytes were normal. Arterial blood gas analysis revealed a pH of 7.35, PCO₂ of 35 mmHg and paO₂ of 71 mmHg and bicarbonate of 19.6meq/L. His oxygen saturation improved with noninvasive ventilation and his blood pressure could be maintained with Dopamine infusion at 10µg/kg/min. Bedside echocardiogram revealed global hypokinesia of basal, mid and inferior segment of left ventricle with mild systolic dysfunction with an ejection fraction of 45%. The right ventricle was normal in size and function. Qualitative estimation of Troponin T was positive as indicated in the report. His Total creatine phosphokinase and Creatine phosphokinase – MB fraction were 312 and 21 Units/ Liter respectively. He was given Dobutamine infusion in addition to Dopamine. MRI brain was normal. Steroids and Mannitol were also given to decrease cerebral edema. Suspecting acute coronary syndrome, he underwent a coronary angiogram which normal epicardial coronaries. His hemodynamics and neurologic status improved rapidly and he recovered completely on 3rd day without any neurologic sequelae. After complete weaning of inotropic support, repeat ECG showed normal sinus rhythm with resolution of ST-T changes. Echocardiogram revealed no wall motion defect and normal left ventricular systolic function with an ejection fraction of. He received psychiatric counselling before discharge.

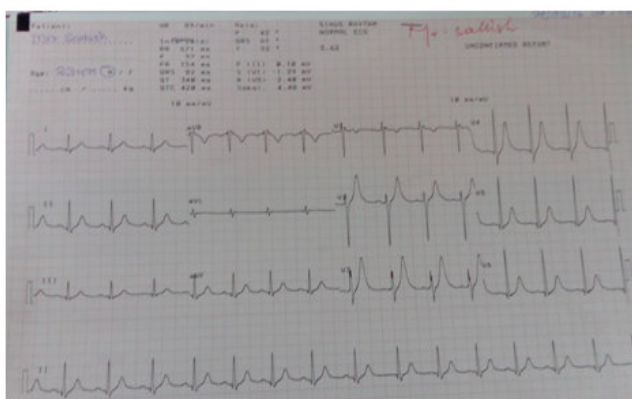
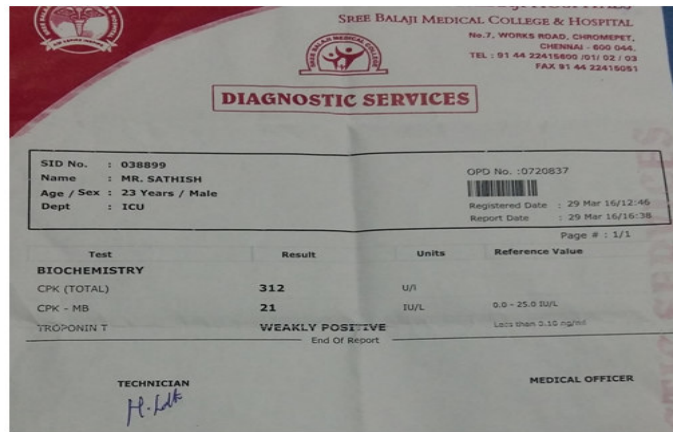


Figure 1
ECG of the patient showing tall t waves at the time of admission



weakly positive

Figure 2
Cardiac enzymes of the patient being positive

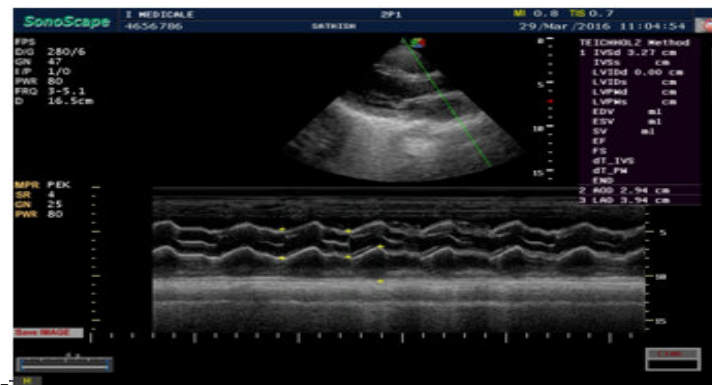


Figure 3
ECHO of the patient showing inferior wall hypokinesia

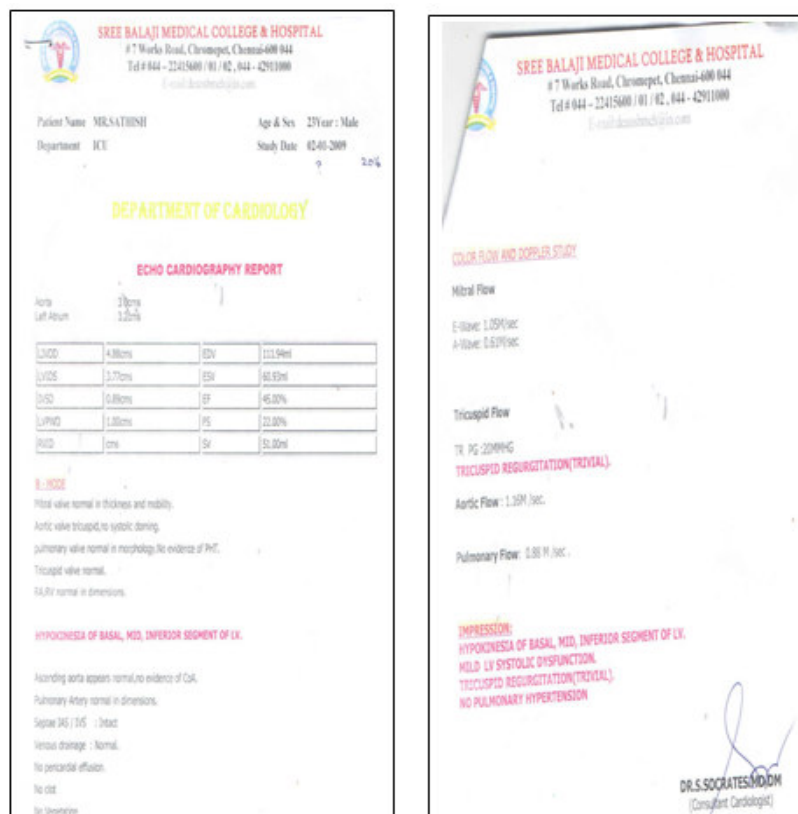


Figure 4,5
ECHO report of the patient with inferior wall hypokinesia

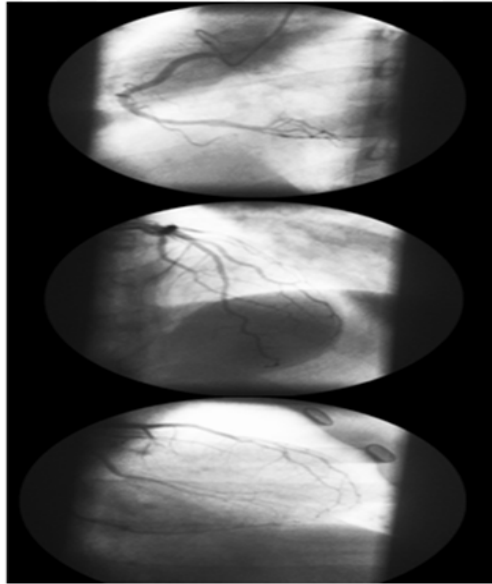



Figure 6
Angiogram of the patient showing normal coronaries

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TECHNICIAN: C. PRIYANKA		SCUB NURSE: RAJAYAGUMALAR	
Patient ID	729837	CATH NO	2249
Patient Name	MR. SATYAN	Study Date	06/04/2016
Age / Sex	23 / Male	REF. DOCTOR	CARDIOLOGY
CORONARY ANGIOGRAM REPORT			
HB	15.3 mg/dl	WEIGHT	KG
UREA	20 mg/dl	BP	110/80 mmHg
CREATINE	1.2 mg/dl	HEARTRATE	82/M
BLOOD GROUP	* positive	BMI	
VIRAL MARKERS	NEGATIVE	RBS	W/dl
CONTRAST	NON IONIC (DININ PAQUE 300)		
ACCESS SITE	RIGHT RADIAL		
CATHETER	1.0 (S)		
HEPARIN DOSE	2500 UNITS		
OTHER MEDICATION:			
LMTA	- NORMAL		
LAD	TYPE 3 HESSEL, NORMAL		
D1	- NORMAL		
L1X	NON DOMINANT, NORMAL		
	D12 - NORMAL		
BCA	- DOMINANT, NORMAL		
	PCA - NORMAL		
	PLV - NORMAL		

LV FUNCTION	- MILD LV SYSTOLIC DYSFUNCTION
IMPRESSION	- NORMAL EPICARDIAL CORONARIES
DIAGNOSIS	- STRESS CARDIOMYOPATHY MILD LV SYSTOLIC DYSFUNCTION NORMAL EPICARDIAL CORONARIES
ADVICE	- MEDICAL MANAGEMENT.



 DR. SOORATES, MD (CARDIOLOGY)
 ASSOCIATE PROFESSOR

Figure 7,8
Angiogram of the patient showing normal coronaries



Normal parameters.

Figure 9
Post recovery echo of the patient with normal parameters

DISCUSSION

Emotional stress like anger, intense grief and acute physical stress like surgery, trauma, intense physical exertion and can lead to cardiovascular events⁵ After an identifiable trigger Reversible left ventricular dysfunction is not uncommon. The triggering event could be an emotionally traumatic event, acute medical illness or strenuous physical activity⁶. Reversible left ventricular dysfunction occurs in such situations in the absence of underlying coronary lesion. This entity has been recently well recognized as Stress cardiomyopathy or Takaosubo Cardiomyopathy. Transient LV dysfunction characterized by segmental hypokinesia of the apex and distal septum, with normal coronary arteries was first described in the Japanese population by Sato et al. who called it the Takaosubo syndrome for the typical appearance on left ventriculogram resembling an octopus trap with a balloon like bottom and a narrow neck¹. The pathophysiological mechanisms behind Takaosubo syndrome are still the object of discussion, although a multifactorial origin is the most popular theory. Alterations in the coronary microcirculation, vasospasm, neurogenic stunning caused by acute autonomic dysfunction, and catecholaminergic cardiotoxicity have all been implicated. Suicidal hanging involves a very intense emotional and physical stress. In our case, the patient had left ventricular dysfunction leading to hypotension. All abnormalities resolved over time. This is likely a form of stress cardiomyopathy. Usually cardiac enzymes are elevated only slightly as in our case. Our patient had hypokinesia of basal, mid and inferior segment of left ventricle instead of apical ballooning as described originally. Generalized wall motion abnormalities are also described in stress cardiomyopathy although segmental wall motion abnormalities are more commonly seen.⁷ We believe myocardial stunning is the mechanism causing reversible left ventricular dysfunction⁸. Preceding the act of hanging catecholamine surge is known to occur. Catecholamine excess⁹ and increased cortisol secretion causes Myocardial stunning. Excessive catecholamine stimulation may result in direct cardiac myocyte toxicity

and also lead to microvascular endothelial dysfunction or spasm.¹⁰ Myocardial perfusion studies using single photon emission computed tomography SPECT and technetium-99 tetrofosmin tomographic myocardial imaging indicate reversible myocardial ischemia in stress cardiomyopathy, in the absence of coronary artery occlusion.¹¹ Another postulated mechanism for left ventricular dysfunction is hypoxemia but as left ventricular dysfunction was documented even after his oxygen saturation improved to near normal this is unlikely in our patient. We reviewed four other reports of transient LV dysfunction associated with attempted suicidal hanging or accidental strangulation. In three of these cases, the echocardiogram revealed global hypokinesia just like our case rather than the apical and distal septal involvement that is characteristic of ABS.¹²⁻¹⁴ Sivanandan et al. reported a case of apical akinesia and globally impaired left ventricular function in an 8-year-old boy following accidental strangulation.¹⁵ Pulmonary edema is well known to occur after attempted hanging.¹⁶ Airway obstruction can cause this sudden onset pulmonary edema. It is possible that at least some of the reports of presumed post-obstructive pulmonary edema may in fact be due to transient global or segmental left ventricular hypokinesia. The optimal treatment of stress cardiomyopathy is unclear. With intravenous propranolol a reduction in the gradient between the apex of the left ventricle and outflow has been observed¹⁷ our patient went on to make an uneventful recovery and had complete reversal of his ECG and echocardiographic findings on follow-up a week after the event. Favorable outcomes have been reported overall with supportive treatment in stress cardiomyopathy with an in hospital mortality of 0–8%¹⁸ We present this case in view of its rarity as this is one of the few cases of reversible left ventricular dysfunction reported in South Asia, that too in the setting of attempted suicidal hanging.

CONFLICT OF INTEREST

Conflict of interest declared None.

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