DOI: 10.14260/jemds/2014/3272

CASE REPORT

TAKOTSUBO CARDIOMYOPATHY: A CASE REPORT

Krishna M. Baradol[1], Vijaykumar C. Zalaki[2], Mohammed Ghouse Khonkhoni [3], Dharmesh A. Ladhad[4]

HOW TO CITE THIS ARTICLE:
Krishna M. Baradol, Vijaykumar C. Zalaki, Mohammed Ghouse Khonkhoni, Dharmesh A. Ladhad. "Takotsubo Cardiomyopathy: A Case Report". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 38, August 25; Page: 9824-9828, DOI: 10.14260/jemds/2014/3272

ABSTRACT: Takotsubo cardiomyopathy is a condition caused by intense emotional or physical stress leading to rapid and severe reversible cardiac dysfunction. A 44 year old labourer presented with three days old bilateral traumatic fracture of femur and severe respiratory distress; he was ventilated for one day. Echocardiography ruled out pulmonary embolism. Patient remained stable for the next three days. On the fifth day, he appeared fearful, presented with sudden chest pain, tachycardia and hypotension. Echocardiography revealed ejection fraction of 34%, global hypokinesia of left ventricle with apical ballooning and no regional wall motion abnormalities. Coronary angiography was done which revealed no vascular abnormalities. Diagnosis of Takotsubo cardiomyopathy was made and vasopressors were started. Psychiatric treatment of physical and emotional stress was done. Patient gradually improved with ongoing treatment and on eighth day his cardiac function reverted back to normal. Takotsubo cardiomyopathy can be efficiently managed by early recognition, proper supportive treatment and meticulous management of physical and emotional stress.

KEYWORDS: Takotsubo cardiomyopathy, stress cardiomyopathy, ampulla cardiomyopathy, broken heart syndrome

INTRODUCTION: Takotsubo cardiomyopathy or stress-induced cardiomyopathy is a condition caused by intense emotional or physical stress leading to rapid and severe reversible cardiac dysfunction.[1] It was first described in Japan and was subsequently reported in United States and Europe.[2-8] Takotsubo means octopus trap in Japanese.[2] It is also known as left ventricular apical ballooning, ampulla cardiomyopathy and broken heart syndrome.[9] It is generally characterized by transient systolic dysfunction of the apical and/or mid segments of the left ventricle that mimics myocardial infarction (MI), but in the absence of obstructive coronary artery disease.[2-7,10]

CASE HISTORY: (Table 1) First day: A 44 year old labourer presented with three days old bilateral (B/L) traumatic fracture of femur and severe dyspnoea. On examination the patient was restless and in severe respiratory distress. On chest auscultation B/L coarse crepitations were heard all over the lung fields. He was immediately intubated, put on mechanical ventilatory support.

All investigations were normal except total leucocyte count: 12, 700/mm.^{3} Chest X-ray showed B/L basal haziness suggestive of B/L pneumonia. Two dimensional echocardiography (2D ECHO) revealed: pulmonary artery pressure-55 mm Hg; pulmonary embolism was ruled out.

He was put on empirical antibiotics. Supportive measures like fluid therapy, feeding, propped up position, position change, suctioning, physiotherapy, thromboprophylaxis, stress ulcer prophylaxis and glycemic control were taken care of throughout. Second day: Patient was extubated. Patient remained stable on third and fourth days.

Fifth day: (Table 1) Patient appeared lethargic, depressed and fearful. He presented with sudden onset chest pain associated with tachycardia and hypotension. 2D ECHO revealed: left

DOI: 10.14260/jemds/2014/3272

ventricle ejection fraction (LVEF) of 34%; gross global hypokinesia of left ventricle (LV) with apical ballooning and no regional wall motion abnormalities. Cardiac enzymes were elevated.

Coronary angiography was done which revealed no vascular abnormalities. Probable diagnosis of Takotsubo cardiomyopathy was made. Patient was started on vasopressors (dobutamine, dopamine and noradrenaline infusions). Psychiatric treatment was initiated to counter emotional stress.

Eighth day: (Table 1) General condition was better; stable vitals, no vasopressors. Chest was clear. Chest X-ray showed resolved basal haziness. 2D ECHO revealed normal cardiac function. Both fractures were fixed surgically. Patient continued to remain stable with ongoing supportive therapy and was discharged from hospital on 35th day.

DISCUSSION: A number of features of stress-induced cardiomyopathy, including its association with physical or emotional stress, suggest that this disorder may be caused by diffuse catecholamine induced microvascular spasm or dysfunction, resulting in myocardial stunning, or by direct catecholamine associated myocardial toxicity. [10]

Unlike in present case (44 years), it is more common in the age group of 61-76 years.[10] Triggering factors preceding this syndrome are emotional and physical stress. A wide variety of emotional stressors have been reported, including panic, fear, anxiety, grief and anger.[9] In present case, the patient, being from poor socioeconomic status and lone bread earner of his family, was severely traumatized mentally by the sudden accident, fracture and fear of future.

The clinical presentation of stress-induced cardiomyopathy is similar to that of an acute MI. [5, 10] The most common presenting symptom is acute substernal chest pain, but some patients present with dyspnoea, syncope, shock, or electrocardiographic abnormalities. [10]

Our patient presented with chest pain, shock and electrocardiographic changes. Acute complications of stress-induced cardiomyopathy can include heart failure, tachyarrhythmias, bradyarrhythmias, mitral regurgitation and cardiogenic shock.[5, 6, 10] Acute systolic heart failure is the most common complication of stress-induced cardiomyopathy, as in present case, and occurs in ~45% of patients.[9]

Electrocardiographic abnormalities are the most common finding. ST segment elevation was present in 34-56% of patients. [10] Electrocardiography of our patient showed T inversion and nonspecific ST-T changes. Cardiac biomarker levels are usually elevated as in present case. [10]

Echocardiography usually shows the characteristic apical ballooning of the LV. [5, 6, 10] Overall systolic function is reduced, and the reported average LVEF has ranged from 20-49%. [5, 10] These all findings correlate with present case.

All four of the proposed Mayo Clinic diagnostic criteria, are required for the diagnosis: [10] 1) Transient hypokinesis, akinesis or dyskinesis of the left ventricular mid segments with or without apical involvement. The regional wall motion abnormalities typically extend beyond a single epicardial coronary 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. Our case met with all four criteria. Pheochromocytoma and myocarditis were ruled out clinically.

Patients who are in shock should undergo urgent echocardiography to determine if left

ventricular outflow tract (LVOT) obstruction is present, which has been described in 13-18% of cases. [10]

Patients without significant LVOT obstruction who are hypotensive due to pump dysfunction as in present case can be treated cautiously with inotropes such as dobutamine and dopamine. Since the condition is potentially caused by catecholamine excess, the impact of sympathomimetics remains to be established. Intra-aortic balloon counterpulsation (IABP) is the preferred therapy when there is marked LV dysfunction associated with severe hypotension or shock. [5, 10] Since IABP was not available at our centre, we managed with inotropes.

In present case, physical stressor was fracture which was fixed. His emotional stressors including panic, fear, anxiety and grief were well tackled by the psychiatrist (psychological and emotional support and counselling).

For patients without intraventricular thrombus but with severe left ventricular dysfunction, anticoagulation is suggested until akinesis or dyskinesis has resolved or for three months, whichever is shorter. [10] In present case we continued anticoagulation till patient mobilization.

Patients who survive the acute episode typically recover normal ventricular function within one to four weeks. [5, 6, 10] Our patient recovered in three days with normal cardiac function.

CONCLUSION: Takotsubo cardiomyopathy is a critical condition presenting with rapid and severe reversible cardiac dysfunction. In the absence of IABP also, severe shock can be managed with cautious use of vasopressors. The success lies in early recognition, proper supportive treatment and meticulous management of physical and emotional stress.

REFERENCES:

1. Ramaraj R. Stress cardiomyopathy: aetiology and management. Postgrad Med J 2007; 83: 543-6.
2. Sato H, Taiteishi H, Uchida T. Takotsubo-type cardiomyopathy due to multivessel spasm. In: Kodama K, Haze K, Hon M, editors. Clinical aspect of myocardial injury: From ischemia to heart failure. Tokyo: Kagakuhyouronsha; 1990. p. 56.
3. Dote K, Sato H, Tateishi H. Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases. J Cardiol 1991; 21: 203.
4. Hachamovitch R, Chang JD, Kuntz RE. Recurrent reversible cardiogenic shock triggered by emotional distress with no obstructive coronary disease. Am Heart J 1995; 129: 1026.
5. Sharkey SW, Lesser JR, Zenovich AG. Acute and reversible cardiomyopathy provoked by stress in women from the United States. Circulation 2005; 111: 472.
6. Desmet WJ, Adriaenssens BF, Dens JA. Apical ballooning of the left ventricle: first series in white patients. Heart 2003; 89: 1027.
7. Bybee KA, Prasad A, Barsness GW. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. Am J Cardiol 2004; 94: 343.
8. Kurowski V, Kaiser A, von Hof K. Apical and midventricular transient left ventricular dysfunction syndrome (tako-tsubo cardiomyopathy): frequency, mechanisms, and prognosis. Chest 2007; 132: 809.
9. Lee JW, Lim HK, Kim JY, Ryoo S. Stress-Induced Cardiomyopathy: Clinical Observations. In: Veselka J, editor. Cardiomyopathies - From Basic Research to Clinical Management. Croatia: In

DOI: 10.14260/jemds/2014/3272

Tech; 2012. p. 665-82.

10. Reeder GS, Prasad A. Stress-induced (takotsubo) cardiomyopathy [Internet]. 2013 Nov [cited 2013 Nov 21]; Available from: http://www.uptodate.com/2013.

	Day 1	Day 2	Day 5	Day 6	Day 7	Day 8
General condition	Critical	Better. Productive cough.	Dull, depressed, fearful	Sick, fearful	Fair, anxious, fearful	Better
Temperature	Afebrile	Afebrile	Afebrile	Febrile	Febrile	Afebrile
HR	120/min	96/min	124/min	110/min	100/min	86/min
BP	90/60 mm Hg	120/70 mm Hg	82/60 mm Hg	90/60 mm Hg	100/60 mm Hg	110/70 mm Hg
RR	34/min	24/min	26/min	24/min	22/min	20/min
SpO_2	60% on room air, 80% with O_2	96% with O_2	99% with O_2	100 % with O_2	96% on room air	98% on room air
Ventilator On/Off	On. CMV mode. Sedated, paralyzed.	Off	–	–	–	–
ECG	WNL	–	ST-T changes, T-wave inversion	–	–	WNL
2D ECHO		–		–	–	
LVEF	60%		34%			55%
LVIDd	37 mm		49 mm			38 mm
LVIDs	21 mm		38 mm			22 mm
PE	No e/o PE		No e/o PE			No e/o PE
DD	Absent		Present			Absent
PAP	55 mm Hg		25 mm Hg			20 mm Hg
RWMA	No		No. Global LV hypokinesia with apical ballooning.			No
CVP	12 cm H_2O	14 cm H_2O	18 cm H_2O	14 cm H_2O	12 cm H_2O	13 cm H_2O
Vasopressors	–	–	Started	Continued	Tapering down	Stopped
Fracture Management	–	B/L skeletal traction	–	–	–	Closed reduction and internal fixation under EA+SA

Table 1*: Case History

*HR-Heart rate, BP-Blood pressure, RR-Respiratory rate, SpO_2-Oxygen saturation, ECG-Electrocardiography, 2D ECHO-Two dimensional echocardiography, LVEF-Left ventricle ejection fraction, LVIDd-Left ventricular internal dimension (diastolic), LVIDs-Left ventricular internal dimension (systolic), PE-Pulmonary embolism, DD-Diastolic dysfunction, PAP-Pulmonary artery pressure, RWMA-Regional wall motion abnormalities, CVP-Central venous pressure, CMV-Controlled mandatory ventilation, WNL-Within normal limits, e/o-evidence of, B/L-Bilateral, LV-Left ventricle,

DOI: 10.14260/jemds/2014/3272

EA+SA-Epidural plus spinal anaesthesia.

List of Abbreviations:

MI-Myocardial infarction.
B/L-Bilateral.
2D ECHO-Two dimensional echocardiography.
LV-Left ventricle.
LVEF-Left ventricle ejection fraction.
~-Approximately.
LVOT-Left ventricular outflow tract.
IABP-Intra-aortic balloon counterpulsation.

AUTHORS:

1. Krishna M. Baradol
2. Vijaykumar C. Zalaki
3. Mohammed Ghouse Khonkhoni
4. Dharmesh A. Ladhad

PARTICULARS OF CONTRIBUTORS:

1. Consultant Intensivist, Department of Critical Care Medicine, Vaatsalya Hospital, IMZ Plaza, Dollars Colony, Gokul Road, Hubli, Karnataka.
2. Consultant Intensivist, Department of Critical Care Medicine, Vaatsalya Hospital, IMZ Plaza, Dollars Colony, Gokul Road, Hubli, Karnataka.
3. Consultant Intensivist, Department of Critical Care Medicine, Vaatsalya Hospital, IMZ Plaza, Dollars Colony, Gokul Road, Hubli, Karnataka.
4. Consultant Intensivist, Department of Critical Care Medicine, Vaatsalya Hospital, IMZ Plaza, Dollars Colony, Gokul Road, Hubli, Karnataka.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Krishna Baradol,
12A, Laxmi Park, Sanman Colony,
Near Shree Mata Ashram,
Hubli-580024, Karnataka.
Email: drkrishbm@gmail.com

Date of Submission: 09/08/2014.
Date of Peer Review: 11/08/2014.
Date of Acceptance: 19/08/2014.
Date of Publishing: 25/08/2014.