Takotsubo Cardiomyopathy in the setting of Urosepsis

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Cite this article as: Saraf A, Hirachan A, Sharma R. Takotsubo Cardiomyopathy in the setting of Urosepsis. Nepalese Heart J 2023; Vol 20(2), 51-53

Submission date: September 11, 2023
Accepted date: September 29, 2023

Abstract

Takotsubo cardiomyopathy is a rare cardiac condition associated with transient left ventricular apical wall motion abnormalities in association with stress but without any significant coronary artery disease. We present a case of takotsubo cardiomyopathy presenting as acute pulmonary edema in the background of urosepsis.

Keywords: Echocardiography, Stress induced cardiomyopathy, Takotsubo cardiomyopathy

DOI: https://doi.org/10.3126/nhj.v20i2.59446

Introduction

Takotsubo cardiomyopathy (TCM) is an acute onset heart syndrome characterized by transient wall-motion abnormalities involving the left ventricular apex and mid-ventricle in the absence of obstructive epicardial coronary disease¹ which is usually seen in association with stress. This may mimic acute myocardial infarction but there is no stenosis on coronary angiogram.²,³ First described by Satoh and Dote, it occurs more commonly in women.¹ It has been named “Takotsubo”-shaped cardiomyopathy because of its unique “short neck round-flask”-like LV apical ballooning which resembles the takotsubo (japanese for octopus pot or trap) of japan.³ This transient left ventricular apical ballooning syndrome has been seen to resolve within a few weeks.⁵

Case Report

A 70-year-old diabetic and hypertensive elderly lady presented to another center with history of high grade fever, burning micturition and dysuria and was admitted in line of urinary tract infection with intravenous antibiotic (injection meropenem 1 gram 8 hourly) and other supportive measures. Her renal function test was normal with complete blood count showing mild leukocytosis. Her liver function test showed mild elevation of transaminases (aspartate aminotransferase - 72 U/L and alanine aminotransferase- 50 U/L). C- Reactive Protein was raised at 162 mg/L (< 10). Urine routine examination showed plenty of leucocytes, no RBCs or any casts.

On day 2 of admission, she developed sudden onset of shortness of breath with central chest pain associated with diaphoresis. On initial evaluation she was in respiratory distress with impending respiratory failure hence was intubated, sedated and paralyzed and put on mechanical ventilation on CMV mode . Her blood gas analysis showed normal pH (7.42), hypoxemia -PaO₂ (66 mmHg) and PaCO₂ level of 32 mmHg. For further multidisciplinary and intensive care, patient was shifted to our center. Her 12 lead electrocardiogram (ECG) showed diffuse T wave inversion and ST depression in anterolateral leads. (Fig 1) In the ICU, patient was started on low dose adrenaline and noradrenaline for systemic hypotension and septic shock. An urgent bedside echocardiography was done which showed hypokinetic mid and apical anterior septum and anterolateral wall and LV apex with moderate left ventricular systolic dysfunction with Ejection Fraction (EF) ~ 35%, mild mitral regurgitation with dilated left ventricle . Other significant lab parameters included high sensitive troponin I levels elevated at 3326 ng/L (<9 ng/L) and CK-MB levels were 20 U/L (0-16 U/L) with normal d-dimer levels. An urgent coronary angiogram was done which showed mid to distal minor plaque in left anterior descending artery, proximal plaque in left circumflex coronary artery and normal origin and caliber right coronary artery. (Fig 2) Findings were suggestive of minor coronary artery disease and hence the diagnosis of Myocardial Infarction in Non Obstructive Coronary Artery (MINOCA) and Takotsubo Cardiomyopathy in Acute LV failure was made.She was further treated with inj meropenem, iv diuretics (torsemide), inj low molecular heparin, antiplatelets (aspirin), statin, beta blockers (bisoprolol), spironolactone and other supportive measures. She had a progressive improvement in blood gas parameters and hemodynamics and hence was extubated on day 4 of admission. A repeat echocardiogram was done on day 7 of admission which showed persistent hypokinesia in mid antero-
septal segment, apical septal/ apical inferior segment and apical cap with slight improvement of ejection fraction to 40-45%. She was later discharged on day 10 with single antiplatelet, statin, beta blocker and low dose diuretics and asked to follow up with a planned cardiac MRI scan on a later date.

A follow up cardiac MRI done 4 weeks later showed non dilated LV with improvement of ejection fraction to 54% with no evidence of regional wall motion abnormality, infarction or fibrosis.

**Discussion**

TCM is a rare life threatening event usually seen in relation with stress, hence its name stress-related cardiomyopathy or broken heart syndrome. These patients show loss of motion or hypokinesia at the apex and an apical balloon-like dilation pattern associated with preservation of the base. In most patients, there is slight elevation in the cardiac enzyme level on admission. However, the enzyme levels decrease rapidly and do not seem to have prognostic significance. According to a retrospective review, patients with this condition accounted for approximately 2% of all the patients with suspected acute coronary syndrome. Further 90% of these patients were postmenopausal women. The average age of patients was found to be 68 years, although children or young adults have also been shown to be affected.

Various hypotheses have been suggested regarding the pathogenesis of TCM: including coronary microvascular dysfunction, coronary artery spasm, catecholamine-induced myocardial stunning, reperfusion injury following acute coronary syndrome, myocardial microinfarction and abnormalities in cardiac fatty acid metabolism. Currently, catecholamine-induced cardio toxicity and microvasculature dysfunction are the most supported theories. Sex hormones may also exert important influences on the sympathetic neurohormonal axis and on coronary vasoreactivity. Women appear to be more vulnerable to sympathetically mediated myocardial stunning and post-menopausal alteration of endothelial function in response to reduced estrogen levels which explains why it is more common in women. Researchers at the Mayo Clinic proposed diagnostic criteria in 2004, which included: (1) transient akinesis or dyskinesia of the left apical and mid-ventricular segments with regional wall-motion abnormalities extending beyond a single epicardial vascular distribution, (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) and (4) absence of all of the following: recent significant head trauma, intracranial bleeding, pheochromocytoma, obstructive epicardial coronary artery disease, myocarditis, and hypertrophic cardiomyopathy.

In the acute phase, supportive and symptomatic treatment should be given. Acute heart failure in TCM is managed the same way as heart failure from any other illness; by oxygen, respiratory support as needed, and preload and afterload reduction. This includes diuretics, ACE inhibitors or ARBs. Hemodynamically unstable patients may need cardiopulmonary support, continuous venovenous hemofiltration and even intra-aortic balloon pump support. In those patients who required isotropic support, use of levosimendan has been shown to be associated with a reduced length of hospital stay and a rapid recovery time. The recommended duration of treatment is not well known but in general, treatment with HF medication is four weeks until systolic function improves.

The in-hospital mortality rate may vary from 0-8% with recurrence rate range from 0-15%. Patients with TCM generally have good prognosis, the recovery rate being almost 96%.9

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**Figure 1: 12 lead ECG showing diffuse T wave inversion in anterior leads**

**Figure 2: Normal Right Coronary Artery, Minor disease in Left Coronary Artery on angiogram**

**Figure 3: Follow up cardiac MRI showing non dilated LV with normal ejection fraction and no residual fibrosis**

**Conclusion**

This case highlights the occurrence of TCM with acute left ventricular failure in the setting of sepsis secondary to UTI. Although the pathogenesis of this condition is still not widely accepted, it is important to bear in mind TCM's resemblance to acute coronary syndrome and the potentially lethal complications of this clinical syndrome.

**References**


