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Medicine

SUDDEN CARDIAC ARREST WITH NORMAL LABORATORY PARAMETERS: AN UNCOMMON PRESENTATION OF TAKOTSUBO CARDIOMYOPATHY IN A 72-YEAR-OLD SOUTH INDIAN MALE

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ABSTRACT

Background: Takotsubo cardiomyopathy (TTC), synonymously known as stress-induced cardiomyopathy, is a reversible form of stress-associated left ventricular systolic dysfunction that frequently mimics acute coronary syndrome. It represents approximately 1–2% of suspected acute coronary presentations[1,7] and predominantly affects postmenopausal women. Although chest pain and biomarker elevation are typical, presentation as sudden cardiac arrest in elderly males with entirely normal laboratory findings is distinctly uncommon. **Case Presentation:** A 72-year-old South Indian male with no prior cardiovascular disease suffered ventricular fibrillation cardiac arrest shortly after facing acute emotional distress related to the bereavement of a close family member. He was successfully resuscitated with prompt defibrillation. Post-resuscitation clinical evaluation revealed stable hemodynamics and normal laboratory parameters, including cardiac troponin and NT-proBNP levels. Electrocardiography showed mild nonspecific ST changes. Transthoracic echocardiography demonstrated apical akinesis with compensatory basal hypercontractility and reduced left ventricular ejection fraction (40%), consistent with classical TTC. Coronary angiography revealed no significant coronary artery obstruction. Conservative medical management resulted in full recovery of ventricular function. **Conclusion:** This case illustrates a rare and potentially misleading presentation of TTC manifesting as ventricular fibrillation cardiac arrest without biochemical evidence of myocardial injury. Early echocardiographic assessment in unexplained cardiac arrest is critical for diagnosis, particularly when emotional stress is implicated. Awareness of this reversible condition helps guide appropriate management and avoid unnecessary invasive interventions.

KEYWORDS :

INTRODUCTION

Takotsubo cardiomyopathy was initially described in Japan in 1990 by Dote et al.[9] and is defined by transient regional left ventricular systolic dysfunction in the absence of obstructive coronary artery disease[1,7]. The condition derives its name from the resemblance of the left ventricular silhouette to a traditional Japanese octopus trap during systole[1] on echocardiography.

Current data suggest TTC accounts for approximately 1–2% of patients evaluated for suspected acute coronary syndrome[1,7]. Around 80–90% of affected individuals are postmenopausal women; however, male patients, though less frequently affected, tend to experience more severe complications, including cardiogenic shock and malignant arrhythmias[7]. Emotional triggers such as bereavement are well-recognised precipitants and are believed to provoke intense sympathetic activation leading to transient myocardial dysfunction or stunning[1,5,7].

We report an atypical case of emotionally triggered TTC in an elderly male presenting primarily with ventricular fibrillation, cardiac arrest and normal biochemical parameters.

Case Presentation: A 72-year-old man with no history of hyper-tension, diabetes, coronary artery disease, pheochromocytoma, Cushing's disease or structural heart abnormalities experienced sudden collapse at home shortly after receiving news of his younger sibling's death.

Bystanders observed immediate loss of consciousness and absence of pulse. Emergency medical personnel initiated cardiopulmonary resuscitation. Cardiac rhythm analysis revealed ventricular fibrillation. A single 200 J biphasic defibrillation shock restored spontaneous circulation. The patient was transported to the emergency department for further evaluation.

Initial Clinical Evaluation

Upon arrival in the intensive care unit:

- Blood pressure: 110/68 mmHg
- Heart rate: 98 beats/min
- Respiratory rate: 22/min
- Oxygen saturation: 98% on room air
- Temperature: 36.8°C

He was alert, oriented, and neurologically intact.

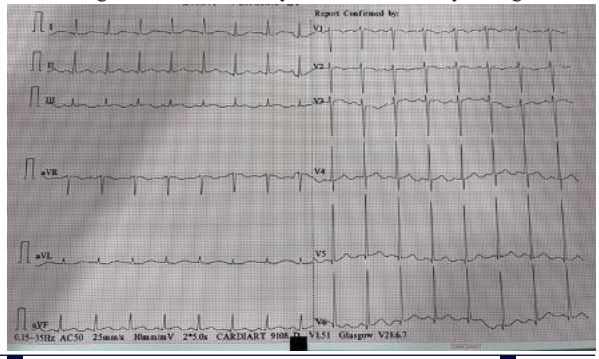
Laboratory Findings: Comprehensive laboratory assessment demonstrated values within normal reference ranges (Table 1). Importantly, cardiac troponin I and NT-proBNP levels were not elevated. Although TTC frequently presents with modest biomarker elevation disproportionate to ventricular dysfunction[1,7], completely normal cardiac markers have been documented in select cases[1].

Table 1: Laboratory Parameters on Admission

Parameter	Patient Value	Reference Range
Hemoglobin	13.8 g/dL	13.0–17.0 g/dL
Total Leukocyte Count	7,200 /mm³	4,000–11,000 /mm³
Platelet Count	2.4 ×10⁵ /mm³	1.5–4.5 ×10⁵ /mm³
Serum Sodium	139 mEq/L	135–145 mEq/L
Serum Potassium	4.2 mEq/L	3.5–5.0 mEq/L
Serum Creatinine	0.9 mg/dL	0.6–1.3 mg/dL
Blood Urea Nitrogen	18 mg/dL	7–20 mg/dL
AST	28 U/L	10–40 U/L
ALT	30 U/L	7–56 U/L
Random Blood Glucose	108 mg/dL	70–140 mg/dL
Troponin I	0.01 ng/mL	<0.04 ng/mL
NT-proBNP	92 pg/mL	<125 pg/mL (<75 yrs)
TSH	2.4 µIU/mL	0.4–4.0 µIU/mL
Arterial pH	7.4	7.35–7.45
Serum Lactate	1.2 mmol/L	0.5–2.2 mmol/L

Arterial blood gas analysis and thyroid profile were normal.

Electrocardiography: The initial ECG demonstrated mild nonspecific ST-segment abnormalities in the precordial leads without dynamic evolution. Typical ECG manifestations of TTC may include ST-segment elevation, T-wave inversion, and QT interval prolongation, the latter contributing to electrical instability and ventricular arrhythmo-genesis[8].



Echocardiography

Transthoracic echocardiography revealed:

- Left ventricular ejection fraction of 40%
- Apical akinesis
- Basal hyperkinesis
- No left ventricular outflow tract obstruction
- No structural valvular abnormalities

This pattern corresponds to the classical apical ballooning phenotype, the most prevalent morphological variant of TTC[1,7].

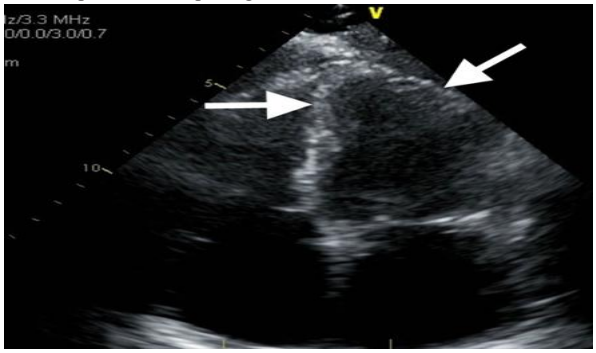


Figure 1: Two dimensional Echocardiographic Apical four-chamber view demonstrating apical ballooning with basal hypercontractility.

The image demonstrates left ventricular apical akinesis(arrow) with compensatory basal hyperkinesis(arrowheads), consistent with Takotsubo cardiomyopathy.

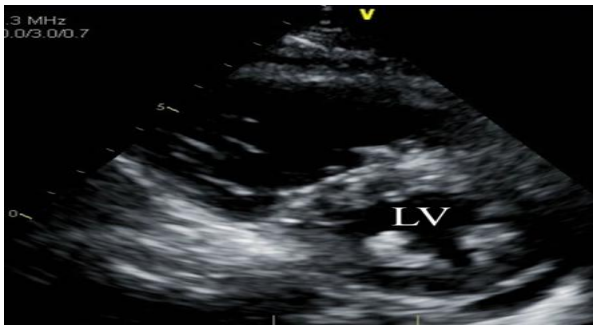


Figure 2: Parasternal short-axis view confirming apical hypokinesis.

Cardiac Magnetic Resonance Imaging

Cardiac Magnetic Resonance Imaging (CMR) was performed to further characterize the myocardial abnormality and exclude alternative causes of transient left ventricular dysfunction. The study demonstrated no evidence of myocardial edema, fibrosis, scar formation, infiltrative cardiomyopathy, or late gadolinium enhancement suggestive of myocardial infarction or myocarditis. No structural cardiac abnormalities were identified. These findings were consistent with the diagnosis of Takotsubo cardiomyopathy.

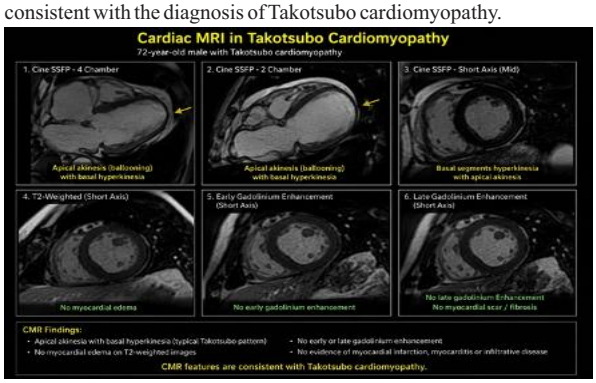


Figure 3 : Cardiac MRI confirming features consistent with takotsubo cardiomyopathy

Coronary Angiography

Urgent coronary angiography demonstrated minimal non-obstructive coronary atherosclerosis with less than 30% luminal stenosis in both the right coronary artery (RCA) and left coronary artery (LCA), without evidence of significant obstructive coronary artery disease. Cardiac magnetic resonance imaging further excluded myocardial infarction, myocarditis, infiltrative cardiomyopathy, or other structural myocardial disease. Together with the characteristic echocardiographic findings, these investigations strongly supported the diagnosis of Takotsubo cardiomyopathy.

Hospital Course and Follow-Up

The patient remained hemodynamically stable during 48 hours of intensive care monitoring. No recurrent arrhythmias were observed. Ventricular arrhythmias are reported in approximately 5–9% of TTC cases and are associated with worse short-term outcomes7.

Medical therapy included low-dose beta-blocker and ACE inhibitor. Antiplatelet therapy initiated empirically was discontinued following normal angiographic findings[14].

He was discharged on day four in stable condition. At six-week follow-up, repeat echocardiography demonstrated normalization of left ventricular function (LVEF 60%) with complete resolution of apical wall motion abnormalities. Functional recovery within 4–8 weeks is characteristic of TTC7.

DISCUSSION

Takotsubo cardiomyopathy is increasingly recognized as a stress-related cardiomyopathy characterized by transient myocardial dysfunction. Excessive catecholamine release during emotional or physical stress is considered a central mechanism leading to transient myocardial stunning [5,7]. Plasma catecholamine levels in TTC have been reported to exceed those seen in acute myocardial infarction [5].

Several mechanisms have been proposed, including coronary microvascular dysfunction, transient coronary vasospasm, intracellular calcium overload, and direct catecholamine-mediated myocardial toxicity [5,7]. The apical myocardium appears particularly susceptible to catecholamine-mediated injury due to a higher density of β-adrenergic receptors.

Large observational data from the International Takotsubo Registry involving over 1,700 patients demonstrated that TTC predominantly affects postmenopausal women and frequently presents with clinical features similar to acute coronary syndrome. However, the registry also highlighted that male patients tend to have worse in-hospital outcomes and higher complication rates, including arrhythmias and cardiogenic shock [10].

Similarly, the position statement from the European Society of Cardiology Taskforce emphasized that TTC should be considered in patients presenting with acute cardiac symptoms, transient left ventricular dysfunction, and absence of obstructive coronary artery disease. [13] The statement also highlighted the role of emotional stressors and neuro-cardiac interactions in the pathophysiology of the syndrome [11].

Although TTC was previously considered a benign condition, contemporary evidence indicates complication rates comparable to acute coronary syndrome, including cardiogenic shock, ventricular arrhythmias, and in-hospital mortality [7]. Ventricular arrhythmias occur in approximately 5–9% of cases and may lead to sudden cardiac arrest.

Our patient presented with several unusual features:

- Male gender
- Presentation as ventricular fibrillation cardiac arrest
- Normal cardiac biomarkers
- Completely normal routine laboratory parameters
- Rapid recovery of ventricular function
- Cardiac MRI showing no significant deviations from the normal for the patient given age, gender and past medical history

The absence of biomarker elevation in this case underscores that normal laboratory parameters do not exclude significant transient myocardial dysfunction, particularly when a clear emotional trigger is present[12]

Cardiac magnetic resonance imaging serves as a valuable adjunct in the evaluation of Takotsubo cardiomyopathy by excluding alternative diagnoses such as acute myocarditis, myocardial infarction with non-

obstructive coronary arteries (MINOCA), infiltrative cardiomyopathies, and other structural myocardial disorders. In the present case, CMR demonstrated no evidence of myocardial edema, fibrosis, scar formation, or late gadolinium enhancement, further supporting the diagnosis of Takotsubo cardiomyopathy in conjunction with the characteristic echocardiographic pattern, absence of significant coronary artery disease on angiography, and complete recovery of ventricular function during follow-up

CONCLUSIONS

This report describes an uncommon manifestation of Takotsubo cardiomyopathy presenting as ventricular fibrillation cardiac arrest in an elderly male with entirely normal laboratory findings. Emotional stress was the likely precipitating factor. The diagnosis was supported by characteristic echocardiographic findings, coronary angiography demonstrating less than 30% stenosis of the RCA and LCA without obstructive coronary artery disease, and a normal cardiac magnetic resonance study showing no evidence of myocardial infarction, myocarditis, fibrosis, or infiltrative cardiomyopathy. The absence of a history of pheochromocytoma or Cushing's syndrome further excluded important differential diagnoses. Clinicians should maintain a high index of suspicion for Takotsubo cardiomyopathy in cases of unexplained cardiac arrest, particularly when multimodality imaging excludes obstructive coronary and structural myocardial disease. Prompt recognition and supportive management can result in complete recovery of ventricular function.

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