

CASE REPORT



## Heart broken. An unusual case of biventricular takotsubo cardiomyopathy complicated by STEMI and Left ventricular thrombi. A case report

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### ABSTRACT

Takotsubo cardiomyopathy (TCM) is a rare occurrence in patients with troponin-positive acute coronary syndrome (ACS). It usually manifests as transient apical ballooning of the left ventricle with concomitant occurrence of right ventricular involvement in only one-third of cases. Biventricular TCM is associated with more hemodynamic instability as compared to left sided alone. Depressed ventricular systolic function and localized ventricular dyskinesia can facilitate clot formation in ventricular cavity. We present a case of 80-year-old man who presented to the ED for evaluation of hypotension. An electrocardiogram suggested acute anterior wall myocardial infarction. He underwent emergent coronary angiography and was found to have mid to apical akinesis and basal hyperkinesis with normal left coronaries and chronic total right coronary artery occlusion with excellent collaterals from left. A transthoracic echocardiography (TTE) revealed left ventricular ejection fraction 25–30% and akinesis of left and right ventricle except in the basal region. TTE with definitely showed sessile thrombus. In our patient, sepsis was the most important triggering factor given initial presentation of hypotension with leukocytosis. Broad spectrum antibiotics including vancomycin and Zosyn were started considering a combination of septic and cardiogenic shock. Repeat EKG showed resolution of ST-T segment elevation but our patient remained hemodynamically unstable even with two pressure support and, ultimately, died 72 hours after admission. Herein, we emphasize on the importance right ventricular involvement and its relation to hemodynamic instability. This case highlights the importance of anticipating hemodynamic instability and clot formation in patients with biventricular Takotsubo cardiomyopathy.

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## 1. Introduction/Background

Takotsubo cardiomyopathy (TCM), also called stress cardiomyopathy, is characterized by regional systolic dysfunction of the ventricle(s), mimicking myocardial infarction (MI), but in the absence of angiographic evidence of obstructive coronary artery disease or acute plaque rupture. First described in Japan in 1990 [1], its incidence is reported in 1–2% of patients presenting with troponin-positive acute coronary syndrome (ACS). It usually manifests as transient apical ballooning of the left ventricle with concomitant occurrence of right ventricular involvement in only one-third of cases. The association with physical (e.g., acute respiratory failure, sepsis, etc.) and emotional (e.g., grief, anger, panic, financial loss, etc.) triggers is well defined [2]. Though the exact pathophysiology is not well established, catecholamine excess [3], microvascular dysfunction and coronary artery spasms [4] are postulated mechanisms.

We present a case of 80-year-old man with hemodynamic instability and ST-segment elevation MI

(STEMI) who was found to have echocardiographic evidence of biventricular takotsubo cardiomyopathy with LV thrombus.

## 2. Case presentation

### 2.1. History

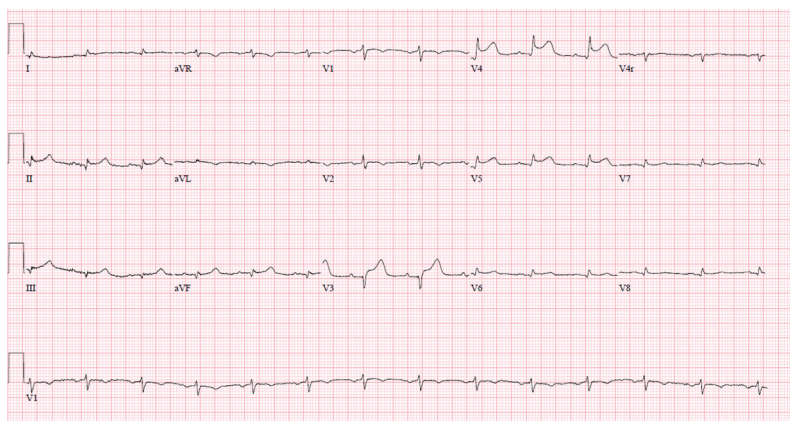
An 80-year-old man with past medical history of peripheral artery disease, hypertension and end stage renal disease (on peritoneal dialysis) was sent to the emergency department from a nursing home for evaluation of hypotension. He denied any chest pain, shortness of breath, fever, cough, palpitation, nausea, vomiting, abdominal pain and changes in urinary or bowel habits. On arrival, patient's blood pressure was 63/29, pulse was 72/min and temperature 97.2 F. Laboratory studies showed the following values: WBC count 16.5 K/UL, Hgb 14.3 GM/DL, PLT 115 K, Creatinine 12.59 MG/DL, BUN 56 MG/DL, Sodium 133 MMOL/L, Potassium 5.4 MMOL/L, BNP

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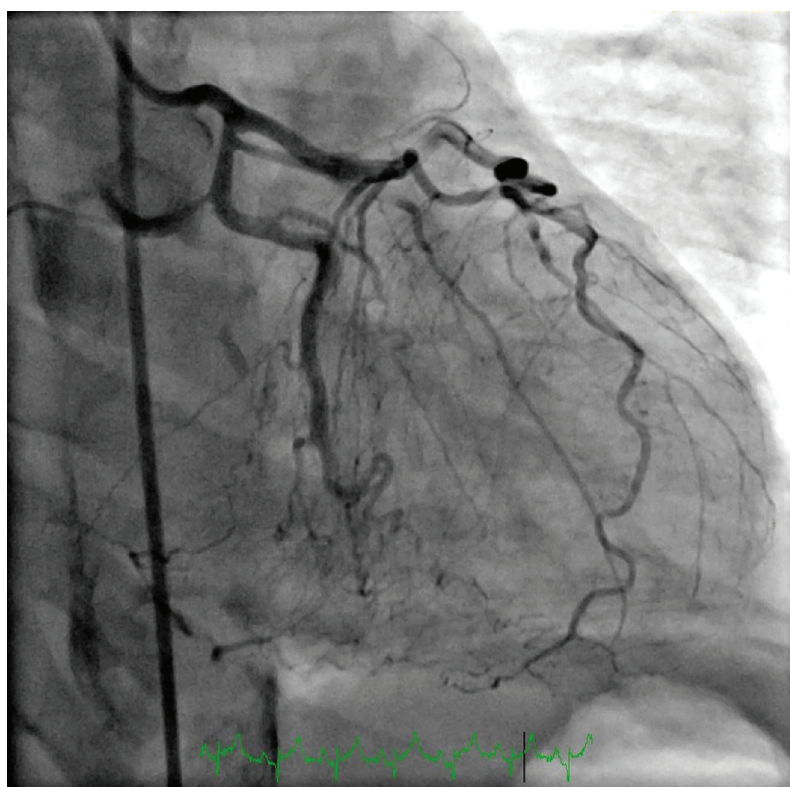
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143 PG/ML, Troponin 0.05 NG/ML. An hour in the ED, EKG showed significant ST segment elevation in lead V3, V4 and V5 and Q wave in lead II, III and AVF [Figure 1]. A diagnosis of acute ST elevation MI was made and oral aspirin 325 mg, clopidogrel 300 mg, atorvastatin 80 mg and 3500 units/kg of heparin were administered. Emergent cardiac catheterization was performed. Coronary angiography showed chronic total right coronary artery occlusion with collaterals from left and patent left coronary arteries [Figures 2 and 3]. Left ventriculography revealed wall motion abnormalities with mid to apical akinesis and basal hyperkinesis [Figure 4]. Post catheterization, the patient

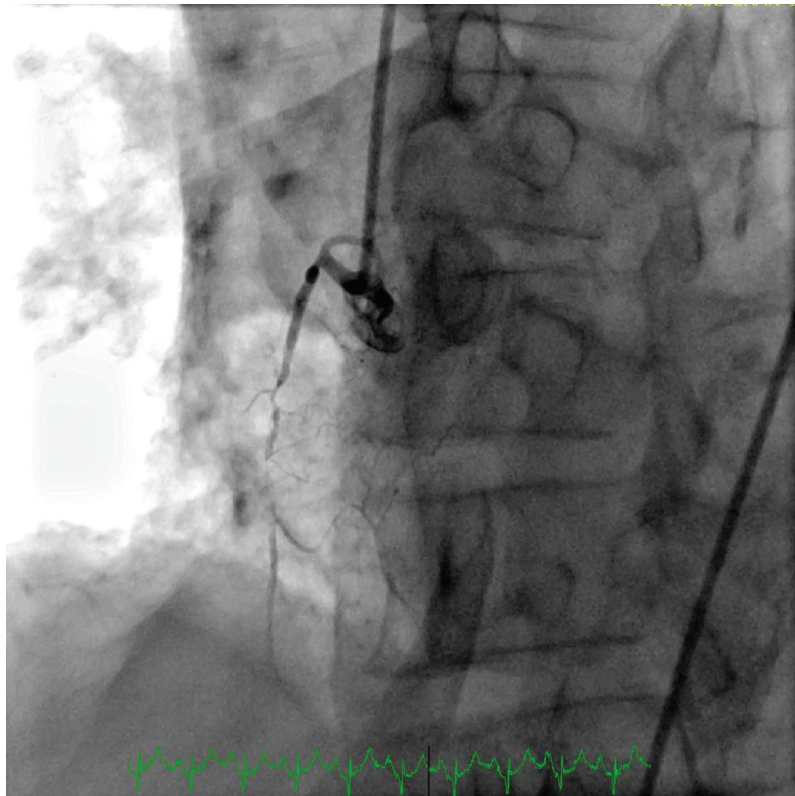
was transferred to ICU for further management. In the ICU, the patient was started on levophed pressure support as the patient was hypotensive. Four hours after the admission, CK-MB and Troponin reached a maximum value of 11.6 ng/ml and 2.10, respectively. Six hours after the admission, EKG showed resolution of ST elevation in anterior leads. Given the patient had leukocytosis in the presence of hypotension, broad spectrum antibiotics including vancomycin and Zosyn were started considering a combination of septic and cardiogenic shock. Approximately 12 hours later, transthoracic echocardiography revealed left ventricular ejection fraction 25–30%, elevated left ventricular



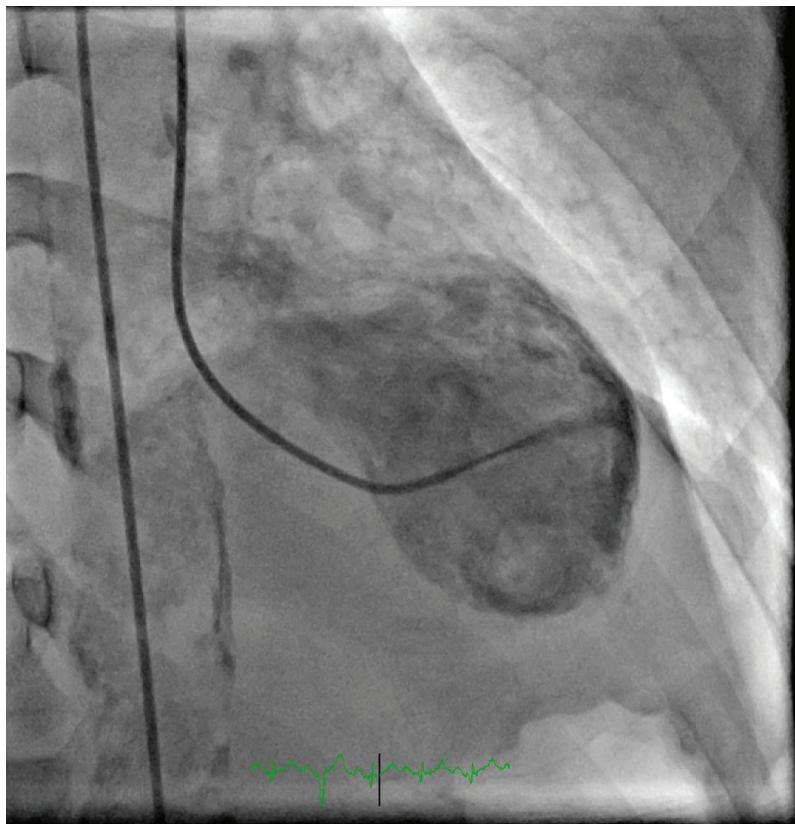
**Figure 1.** Electrocardiogram showing ST segment elevation in lead V3, V4 and V5 with Q waves in II, III and AVF.



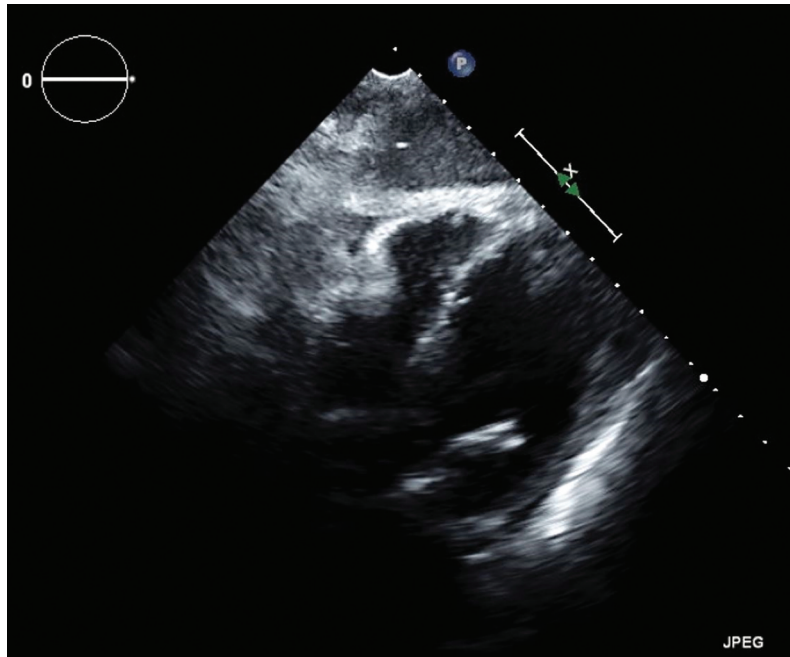
**Figure 2.** Left heart cath. showing patent left coronaries.



**Figure 3.** Left heart cath. showing occlusion of RCA with collaterals from left.



**Figure 4.** Left ventriculography shows wall motion abnormalities with mid to apical akinesis and basal hyperkinesis.



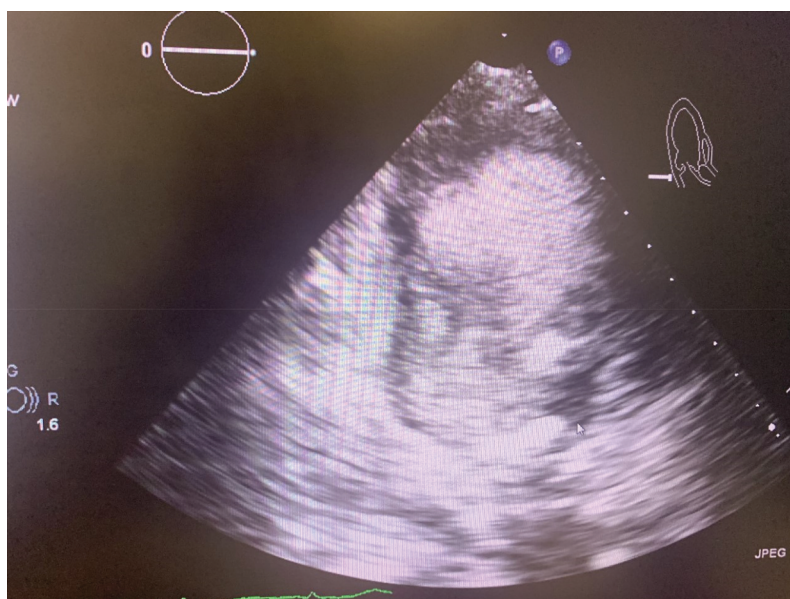
**Figure 5.** Two-dimensional echocardiogram shows mid-to-apical ballooning of the right and left ventricle.

end diastolic pressure and akinesis of left and right ventricle except in the basal region [Figure 5]. Although the patient remained chest pain free, but continued to remain on levophed and neosynephrine for pressure support. Workup for sepsis was unremarkable. A day later, a repeat echocardiogram showed sessile LV thrombus and akinesis of both left and right ventricles except in the basal region [Figure 6]. Anticoagulation with heparin was started. Plan was to repeat the echo after 3–4 days to monitor the response to therapy. Patient remained hemodynamically unstable and, ultimately, died 72 hours after admission. In

accordance with his family wishes, an autopsy was not performed.

### 3. Discussion

TCM is a rare occurrence in patients with troponin-positive acute coronary syndrome (ACS). Given the association of left ventricle (LV) apical ballooning with sepsis, it is not an uncommon observation in the medical ICU population. Though more common in the female population (female-to-male ratio, 9:1) [2], our patient was an 80-year-old male. With regard to pathophysiology, although most patients with



**Figure 6.** Two-dimensional echocardiography with definity showing LV thrombus.

TCM typically show no obstructive lesions on coronary arteriography [4], one intravascular ultrasound study found evidence of mid left anterior descending (LAD) coronary artery plaque rupture in five of five patients diagnosed with stress cardiomyopathy [5]. In contrast, our patient had chronic total right coronary artery occlusion with collaterals from left and normal left coronary arteries. With regard to predisposing factors, as per data, the prevalence of physical triggers exceeds that of emotional triggers [2]; our case most likely had the physical trigger in the form of septic shock, given the fact that patient was living in a nursing home and had leukocytosis with hemodynamic instability. It is in line with the findings of a small prospective study of 92 patients in a medical intensive care unit, where it was found that sepsis was the only variable associated with the development of left ventricular apical ballooning (LVAB) [6].

The most common presenting symptom is acute substernal chest pain, but some patients present with dyspnea or syncope [2]. Our patient presented with hypotension and was found to have leukocytosis. Moreover, it is known that troponin levels and electrocardiographic changes on admission are not sufficient to differentiate between TCM and ACS. Moreover, few of the cases of TCM are associated with ACS (as our patient) [7], hence early coronary angiography is necessary to rule it out.

There are data to support that patients with physical triggers are more at risk of severe complications, including death, than patients with emotional triggers. The false impression that was created by certain studies with low-risk patients that this is a universally benign disease is now dismissed after close analysis of patients with physical triggers, with a rate of death per patient-year of 5.6% and a rate of stroke or transient ischemic attack of 1.7% per patient-year [2]. Daoko et al. reported that biventricular takotsubo cardiomyopathy (similar to our patient) is associated with more hemodynamic instability as compared to left ventricular alone [8]. The clinical course of our patient was not promising, as he continued to remain on levophed and neosynephrine for pressure support, with repeat echocardiographic findings of sessile LV thrombus and akinesis of both ventricles except in the basal region. Although there are some data that supports the use of intravenous beta blockers in such patients [9], several avenues are needed to be unveiled regarding prevention and treatment of TCM.

## 4. Conclusion

The case we present is of biventricular takotsubo cardiomyopathy associated with LV thrombus which is rare. This case highlights the importance of anticipating hemodynamic instability and clot formation in patients with biventricular Takotsubo cardiomyopathy.

## Disclosure statement

No potential conflict of interest was reported by the authors.

## Informed consent

Consent was taken from son as patient expired.

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