

Heart broken twice: a case of recurrent Takatsubo cardiomyopathy

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Accepted 5 December 2019

DESCRIPTION

A 53-year-old woman with a past medical history of diabetes, hypertension, persistent atrial fibrillation on anticoagulation and resolved takotsubo cardiomyopathy (TTC) 2 years ago presented with 2 days of dyspnoea, fever and dysuria. Her vitals were blood pressure—90/60 mm Hg, pulse rate—110/min, irregular rhythm, respiratory rate—16/min and SPO₂—90% on room air. Examination was positive for use of accessory muscles of respiration, bilateral coarse crackles over the lung bases and pedal oedema 2+ up to the knees. Laboratory work showed leucocyte count of $12.3 \times 10^9/L$, urine culture grew *Escherichia coli* and chest X-ray showed bilateral pleural effusion. Hence, diagnosis of sepsis due to urinary tract infection, congestive heart failure and atrial fibrillation with fast ventricular rate was made. She was started on intravenous ceftriaxone. Her fever and tachycardia responded within next 24 hours, but she continued to have hypotension and hypoxia. A transthoracic echocardiography showed a low ejection fraction of 15%, mild mitral regurgitation, with systolic apical ballooning of the left ventricle and hyperkinesis of the basal walls ([figure 1](#)). She suffered a stress-induced cardiomyopathy 2 years back with stressor being sudden death of both the parents. At that time, she was initiated on beta-blockers, following which the ejection fraction improved. Based on combined history of TTC, the classical echo findings and physical stress of urinary tract infection, the diagnosis of recurrent TTC was made at this admission. She was initiated on metoprolol succinate and lisinopril at tolerable doses. For *E. coli*-related urinary tract infection, she finished 7 days of antibiotics. A follow-up two-dimensional echo showed an improved ejection fraction to 40%.

Takotsubo syndrome is a rare acute cardiac crisis event that frequently is headed by a stressful trigger.

In our case, patient had both emotional (first event) and physical trauma (second event). Classically, emotional or physical stress triggers an excessive sympathetic nervous system stimulation responsible for TTC. Nyman *et al* recently did a systematic review on trigger factors in TTC and found that physical factors are more common than emotional factors.¹ Also, they found that most vulnerable group to have evident trigger factors leading to TTC are women ≤ 50 years of age and men. The review consisted of 1330 TTC cases, out of which 1112 cases (83.6%) had at least one trigger factor with 67.3% physical and 32.7% emotional factor. Most common physical factor was illness (60%) followed by procedure/surgery-related stress (40%). Similarly, anxiety/panic attack/fear (31.6%) constituted the most common emotional trigger followed by grief/loss (23.1%).¹ Some of the commonly reported physical triggers are chronic obstructive pulmonary disease, subarachnoid haemorrhage, thyrotoxicosis, Addisonian crisis, sepsis and so on.¹ In addition, recently Amariles *et al* reviewed literature and reported at least 20 drugs that were recognised as possible cause for drug induced TTC.² Recently, Auger *et al* also found TTC to have an association with autoimmune disease, pneumonia, renal disease, mental disorders as compared with population controls.³ Although, no well-established literature is available to date, but recently electrolyte imbalances especially hypomagnesaemia and hypocalcaemia have been linked to TTC.⁴ Role of electrolyte imbalances as a trigger factor needs to be confirmed through well-designed trials. Despite vigilant search no known trigger factor is found in approximately 30% of the TTC cases.⁵

It is characterised by transient ventricular wall dysfunction in the absence of coronary artery disease.⁶ This relatively rare condition (<2% of ACSs in women and just 0.2% in men) is not

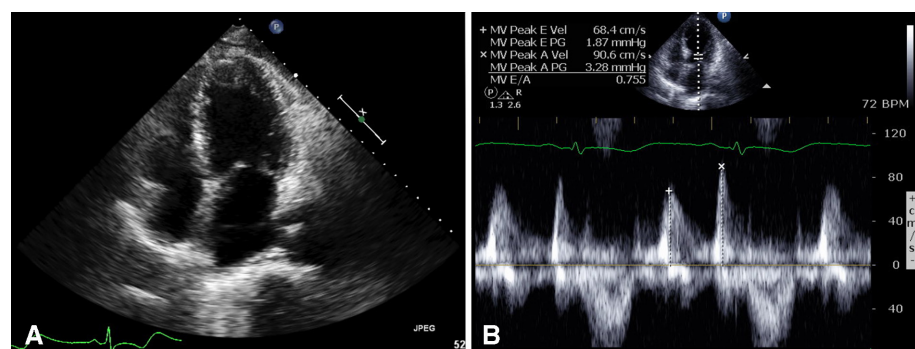


Figure 1 (A) Apical four chamber view showing ballooning of the apical segment of the left ventricle and (B) mitral regurgitation.



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To cite: Sahu KK, Mishra AK, Doshi A, *et al*. *BMJ Case Rep* 2020;**13**:e232253. doi:10.1136/bcr-2019-232253

often recognised initially because either symptoms are too mild and very non-specific.⁷ Recurrence is even further uncommon, reported only in 5%–6% of TTC cases. Interestingly, the risk of recurrence is maximum during initial years and gradually declines thereafter. Elesber *et al* reported the highest 2.9% in the first 4 years and 1.3% thereafter.⁸ The most widely accepted proposition for TTC is due to catecholamine surges secondary to stress.⁶ Varying beta receptor densities lead to differential effect on myocardium with resultant apical ballooning. However, the role of hormonal influences and innate susceptibility remain elusive. Clinical significance of recurrence of TTC is not clear due to sparse literature and probable under reporting. But there is increasing evidence that TTC is not a benign disease as various complications like ventricular arrhythmias, cardiogenic shock has been reported. Knowledge about rare cardiac entities and their complications is of utmost importance for timely diagnosis and management.^{9,10}

Learning points

- ▶ Takotsubo cardiomyopathy (TTC) is a rare entity and has classical echo findings.
- ▶ Recurrence of TTC is rare and recurrence rate decreases with time.
- ▶ TTC is not a benign entity and conduction abnormalities like ventricular arrhythmias can occur.

Contributors KKS: case writing, review and proofread. AM: case writing. AD: literature review. KM: management and critical review.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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