

## IMAGES AND VIDEOS

# Iatrogenic reverse takotsubo cardiomyopathy

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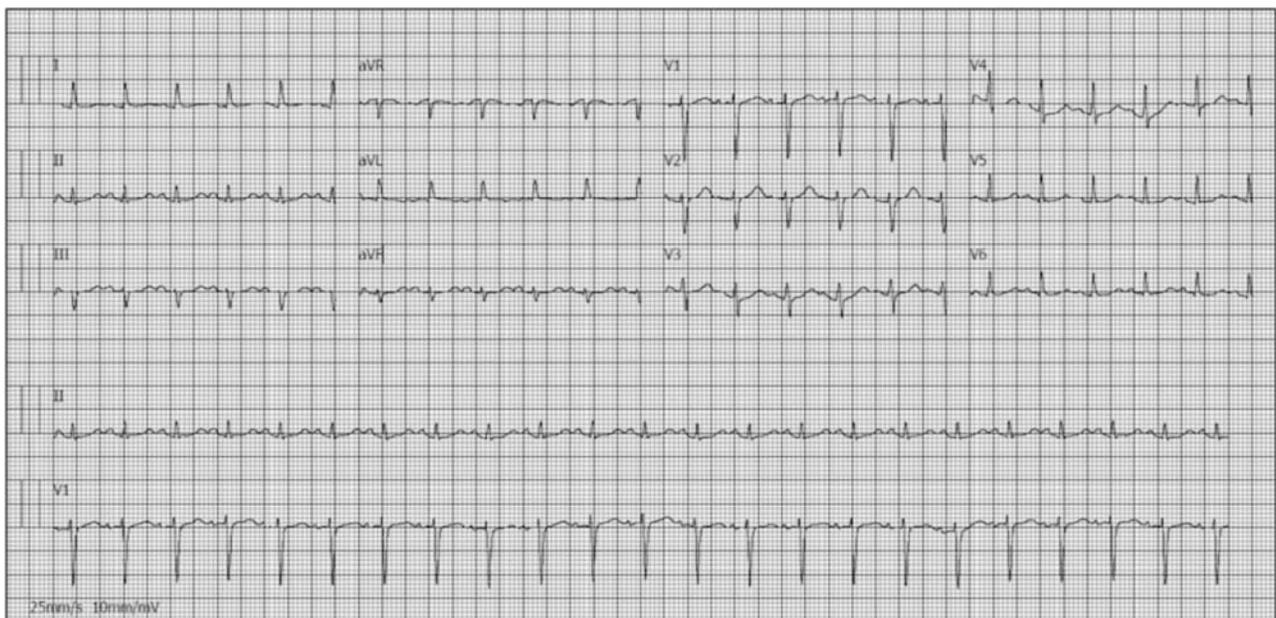
A 47-year-old woman with a history of ischemic stroke and dyslipidemia presented with severe gastrointestinal (GI) symptoms that lasted for a week. The abdominal CT was normal, but due to persistent complaints, she was admitted for clinical monitoring.

Intravenous  $\beta$ -blockers were administered for a sinus tachycardia. Soon after esmolol, acute cardiac failure ensued with prompt progression to cardiogenic shock. Owing to acute respiratory failure, the patient was mechanically ventilated and inotropes were started. Electrocardiogram showed no signs of myocardial ischemia (Fig. 1), with a slight prolongation of the QTc interval

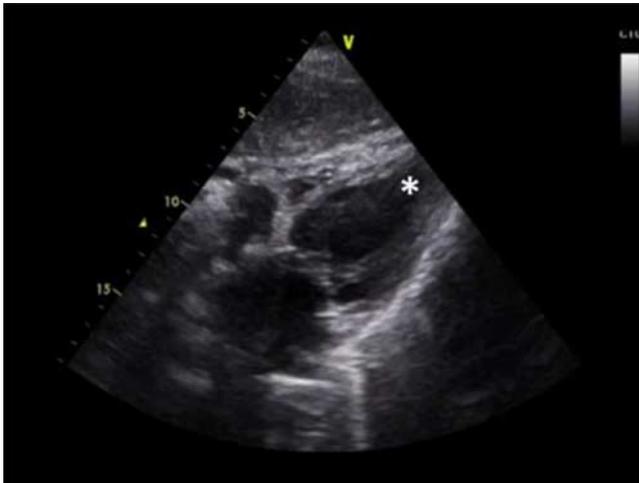
(479 ms). A mild troponin I elevation was detected (8.2  $\mu\text{g/l}$ ) and the nt-pro BNP levels were 12 706 pg/ml.

The transthoracic echocardiogram (TTE) was performed after the clinical diagnosis of acute heart failure, which revealed a non-dilated left ventricle (LV) with severe systolic dysfunction. Apical segments had a preserved contractility, contrary to the mid-basal ones, which was not supportive of a coronary event. Moderate functional mitral regurgitation (MR) was identified (Figs 2, 3 and Videos 1, 2).

A coronary angiogram revealed no significant disease and a LV angiogram confirmed severe systolic



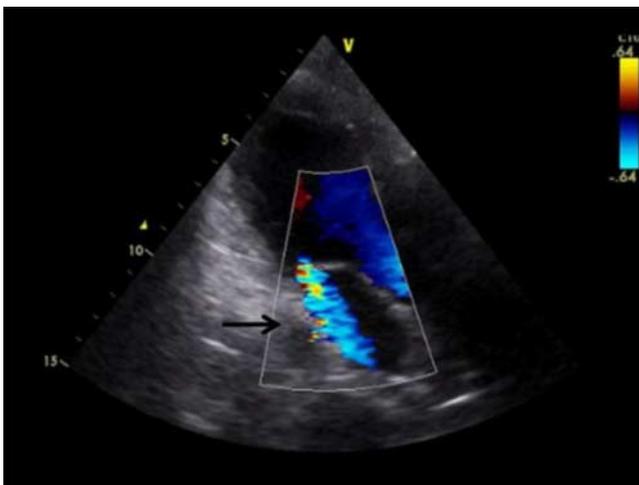
**Figure 1**  
ECG after acute heart failure onset. Sinus tachycardia. No signs of myocardial ischemia.



**Figure 2**  
Initial TTE sub-costal view, end-systolic frame. Non-dilated LV and severe LV systolic dysfunction are observed. Preserved contractility of the apical segments can be noticed (asterisk).

dysfunction, with MR. Similar to the TTE, a wall motion pattern of preserved apical contractility was recognized (Fig. 4 and Video 3).

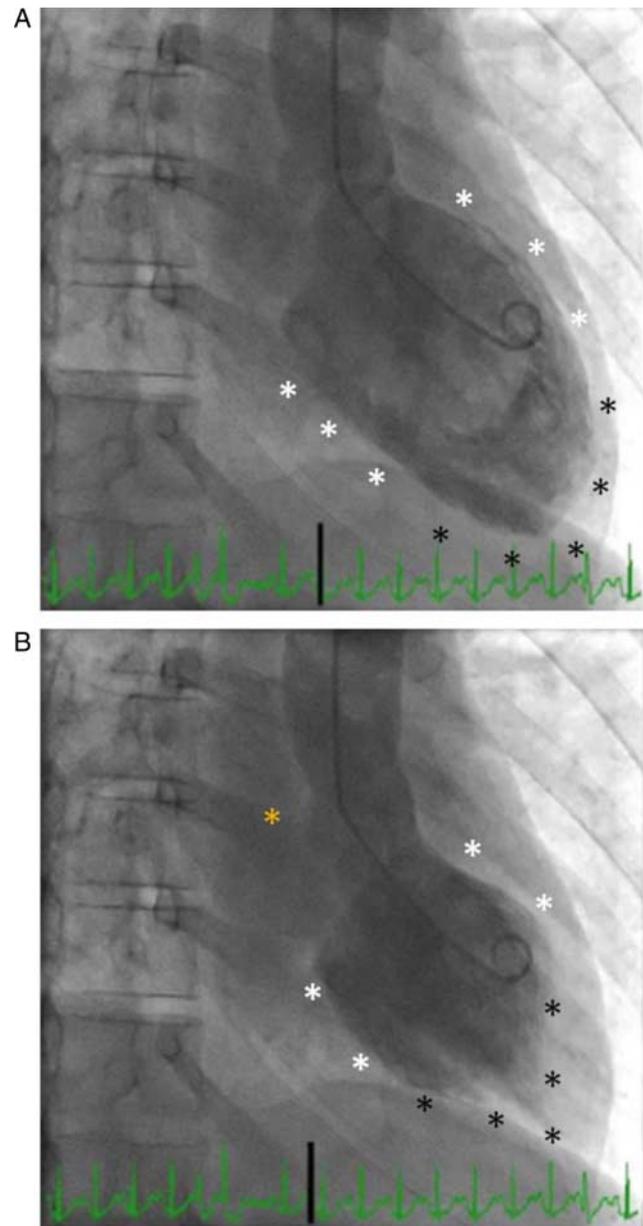
The subsequent clinical evolution was excellent. The TTE, performed 72 h after admission, showed a normal LV systolic function with no wall motion abnormalities and no MR (Videos 4 and 5). The nt-pro BNP decreased to 6917 pg/ml. Improvements were also registered two weeks later through cardiac MRI evaluation, and no myocardial edema, fibrosis, or infarction was identified (Fig. 5 and Video 6).



**Figure 3**  
Initial TTE apical three-chamber view. Functional MR can be noticed.

### Video 1

Initial TTE sub-costal view. Non-dilated LV and severe LV dysfunction, normal LV wall thickness, preserved LV apical contractility, and mid- to base akinesis of the LV are indicated. Download Video 1 via <http://dx.doi.org/10.1530/ERP-14-0009-v1>



**Figure 4**  
LV angiogram end-diastolic (A) and end-systolic (B) frames. Akinesis of the mid- and base segments of the LV anterior and inferior walls (white asterisks) is indicated. Preserved apical contractility (black asterisks) and MR (yellow asterisks) can also be observed.

### Video 2

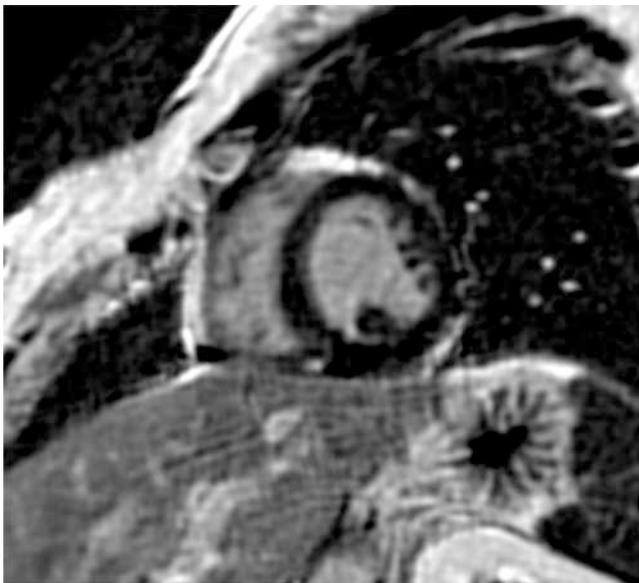
TTE apical three-chamber view, showing functional MR. Download Video 2 via <http://dx.doi.org/10.1530/ERP-14-0009-v2>

### Video 3

LV angiogram. There is a preserved LV apical contractility, in contrast to the mid- to base akinesis. Functional MR can also be noticed. Download Video 3 via <http://dx.doi.org/10.1530/ERP-14-0009-v3>

### Video 4

TTE apical four-chamber view 48 h after heart failure onset, with complete recovery of LV dysfunction and wall motion abnormalities. Download Video 4 via <http://dx.doi.org/10.1530/ERP-14-0009-v4>



### Figure 5

Two weeks after acute heart failure onset, no significant changes were noted in the cardiac late gadolinium enhancement imaging.

### Video 5

TTE apical three-chamber view, 48 h after heart failure onset. Complete resolution of the MR can be noticed. Download Video 5 via <http://dx.doi.org/10.1530/ERP-14-0009-v5>

### Video 6

Cardiac MRI after 2 weeks. True fast imaging with steady-state free precession four-chamber view. Normal LV systolic function and no wall motion abnormalities could be observed. Download Video 6 via <http://dx.doi.org/10.1530/ERP-14-0009-v6>

We hypothesize that the cause of the non-apical ballooning syndrome was a combination of esmolol and a preceding stress, due to a GI illness with sympathetic stimulation.

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#### Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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