

A rare case of atypical mid-variant takotsubo cardiomyopathy during dobutamine stress echocardiography (RCD code: III-5B)

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Abstract

Dobutamine stress echo (DSE) allows for a relatively safe and accurate assessment of ischaemia in patients, who are unable to exercise. Side effects of DSE include nausea, vomiting, and in certain cases, excessive rise and fall in blood pressures. However, in the setting of an intrinsic catecholamine surge, dobutamine stress testing can lead to transient ventricular ballooning. Here, we report a case of a 66-year-old female who developed chest pain with ST-segment elevation on electrocardiogram (ECG) along with wall motion abnormalities seen on transthoracic echocardiogram (TTE) during dobutamine stress testing. Her urgent coronary angiogram showed non-obstructive coronaries with systolic mid-anterolateral and antero-septal wall ballooning on left ventriculogram with basal and apical sparing. The ECG changes resolved prior to discharge and cardiac function returned to normal on follow-up at 3 months. The absence of obstructive coronary lesions with wall motion abnormalities on TTE and left ventriculogram, followed by the return of cardiac function to normal on follow-up TTE highlights this case as a unique occurrence of a dobutamine-induced, mid-ventricular variant, atypical takotsubo cardiomyopathy. To the best of our knowledge, this is the first case of mid variant atypical takotsubo cardiomyopathy occurring secondary to dobutamine administration reported from the Indian subcontinent. The occurrence of this phenomenon points towards an extremely rare effect that may occur as a consequence of stress testing with dobutamine. JRCD 2018; 3 (6): 213–217

Key words: rare disease, stress testing, non-obstructive coronary disease, left ventricular ballooning

Background

Dobutamine stress echocardiogram (DSE) is a useful and safe imaging modality for assessing coronary ischaemia. Dobutamine induces myocardial ischaemia which manifests as ST-segment changes on electrocardiogram (ECG) and left ventricular (LV) wall motion abnormalities (WMA) on transthoracic echocardiogram (TTE). Side effects of DSE include nausea, vomiting, and in certain cases, excessive rise and fall in blood pressures. However, in the setting of an intrinsic catecholamine surge, dobutamine stress testing can provoke transient ventricular ballooning. Here, we report a case of an elderly female who developed mid-ventricular transient cardiomyopathy during DSE.

Case presentation

A 66-year-old single female, active smoker with no other cardiac risk factors, arrived at the cardiac clinic with symptoms of chest heaviness, sweating, and dyspepsia at rest lasting for a few months. She had no exertional symptoms and led a sedentary lifestyle. She was vitally stable and her examination was within normal limits. Her resting ECG showed normal sinus rhythm (Figure 1) and baseline TTE showed normal LV function with no WMA (Video 1). Stress echocardiography was recommended to rule out coronary ischaemia.

Due to the patient's exercise intolerance, DSE was performed. Dobutamine was infused via peripheral intravenous cannula at incremental doses, starting at 10 mcg/kg and increased to 20 mcg/kg to achieve the target heart rate response. In view of a slow heart

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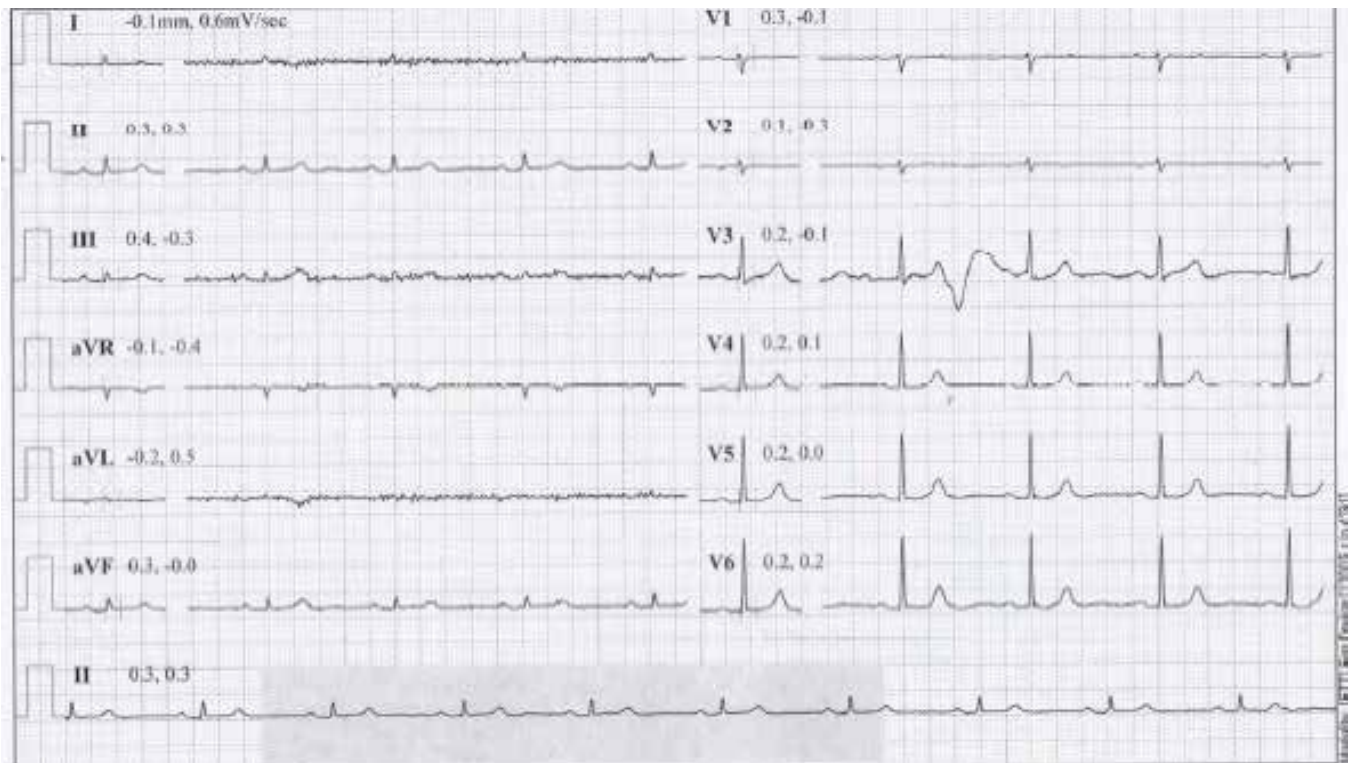


Figure 1. Electrocardiogram at baseline

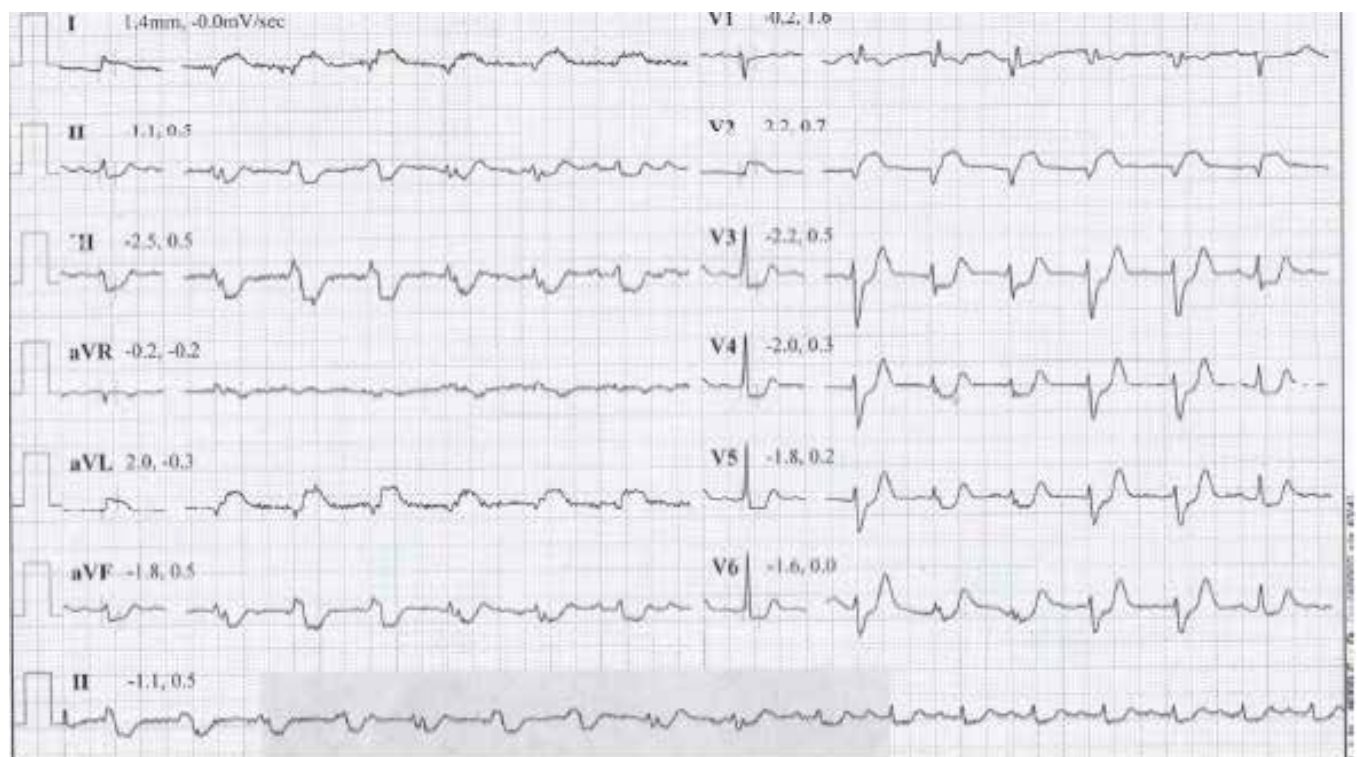


Figure 2. Electrocardiogram during dobutamine stress echo showing ST segment elevations (4mm) in leads I, aVL, V1-V2 with ST segment depressions in leads II, III, aVF, V3-V6

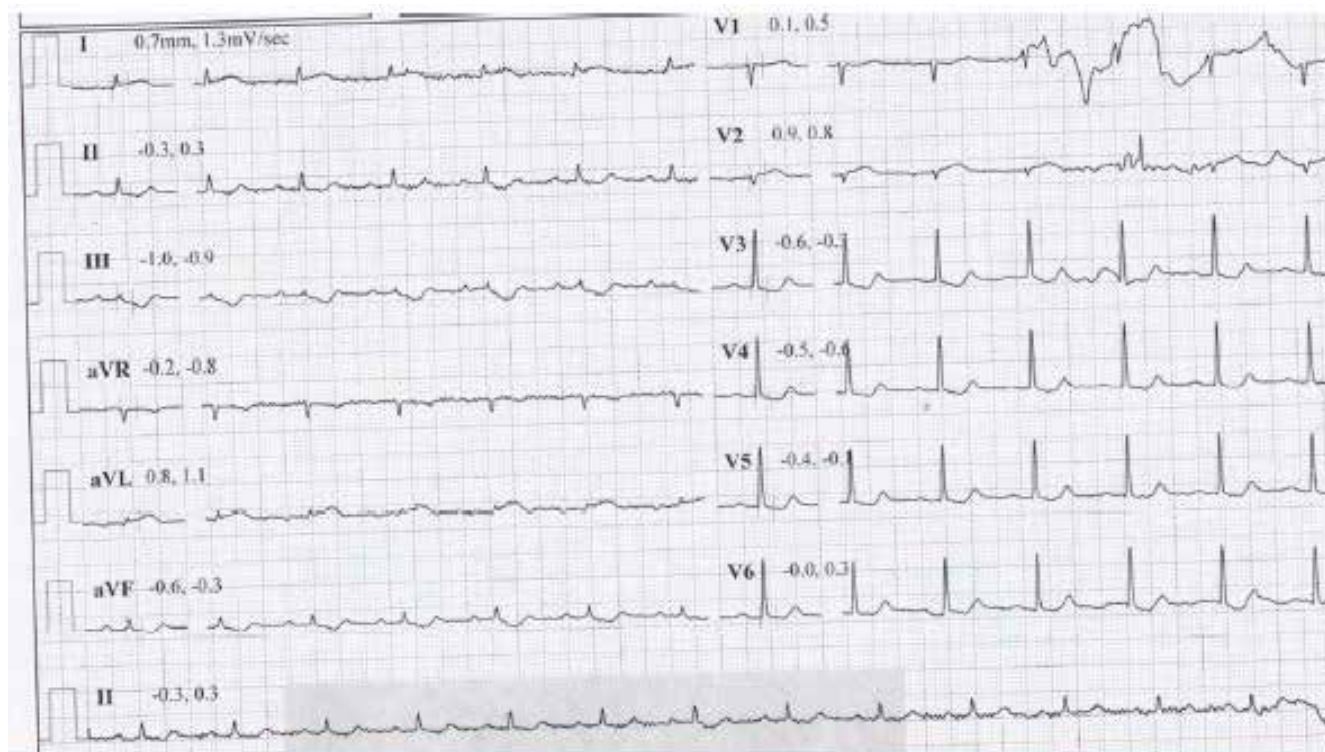


Figure 3. Electrocardiogram after termination of test and administration of intravenous metoprolol showing settling of ST elevation

rate response to dobutamine, 0.4 mg intravenous (IV) atropine was administered.

At a 20 mcg/kg dose of dobutamine and a heart rate of 130 beats/min (81% target heart rate), the patient experienced central chest heaviness with profuse sweating. The ECG showed a run of ventricular bigeminy followed by ST-segment elevations (4 mm) in leads I, aVL, and V2 with ST-segment depressions in leads II, III, aVF, and V3-V6 (Figure 2).

The peak stress echocardiographic images showed akinesia of the mid to distal septum and anterolateral wall with basal wall hypercontractility (Videos 2–3).

Patient management and follow up

The test was terminated and the patient was given intravenous metoprolol 5 mg IV and sublingual nitrate 0.5 mg. The chest pain resolved as the heart rate decreased to 82 beats/min and the ECG ST-segment elevation resolved (Figure 3). She was immediately fast tracked to the coronary care unit and loaded with acetylsalicylic acid 300 mg per os, clopidogrel 300 mg per os, and IV Heparin 5000 units. Her repeat echocardiogram immediately after termination of the test showed persistent WMA (Video 4).

Urgent coronary angiogram was performed which showed mild plaquing in the left main, mild plaquing in the proximal to mid left anterior descending artery (Video 5), and a non-dominant right coronary artery (Figures 4–5).

A left ventriculogram in right anterior oblique view showed mid-anteroseptal wall systolic ballooning with apical and basal sparing. Left anterior oblique showed mid-anteroseptal and antero-

lateral wall systolic ballooning with apical and basal sparing with an estimated ejection fraction (EF) of 45% (Video 6).

The maximum level of high sensitivity Troponin I was 8.03 ng/dl (cut off >0.04 ng/dl).

Repeat TTE after 24 hours showed an estimated EF of 50% with severe hypokinesia of mid-anterolateral and mid-anterior septal segments.

The patient was transferred back to the coronary care unit where lifestyle modifications and smoking cessation were advised.

She remained stable and was discharged on antiplatelets, statin, and angiotensin converting enzyme inhibitors. The patient was reviewed in the clinic 2 weeks after discharge and subsequently had a follow-up TTE. She remained asymptomatic and felt well during. Her repeat TTE after 3 months showed no WMA (Video 7).

Review of literature

The differential diagnosis that was considered in the setting of chest pain with ST-segment elevation on ECG and WMA on TTE was plaque rupture induced by the stress testing. However, the segmental WMA in the mid-septal and anterolateral segments with the sparing of basal and apical segments could not be attributed to any single coronary artery distribution in the presence of a non-occlusive angiogram. Intravascular ultrasound (IVUS), however, was not performed. A more probable mechanism was multivessel coronary artery spasm secondary to catecholamine surge.

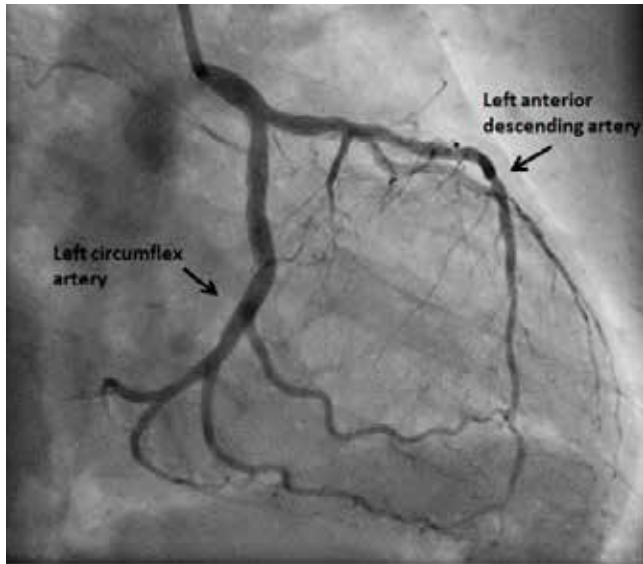


Figure 4. Right anterior oblique view of coronary angiogram showing non-obstructive disease in left anterior descending and left circumflex arteries

The reversion of WMA to near normal on follow-up TTE pointed towards a diagnosis of dobutamine-induced, atypical mid variant takotsubo cardiomyopathy.

Takotsubo syndrome (TTS), also known as broken heart syndrome or stress-induced cardiomyopathy, was first described in the 1990's as transient apical ballooning of the LV in the absence of obstructive coronary artery disease, predominantly present in female patients [1]. Takotsubo cardiomyopathy (TCMP) is known to be triggered by numerous emotional and physical stressors [2].

The apical ballooning pattern or 'typical TCMP pattern' is the most commonly described pattern of TCMP. However, other patterns of myocardial ballooning have been described and have been termed as atypical variants of TCMP. These include basal ballooning, mid ventricular ballooning, and focal ballooning patterns [3].

The occurrence of ST-segment elevation during DSE is rare and largely reported as case reports [4–8], however, dobutamine has also been implicated in the aetiology of takotsubo cardiomyopathy [9–14]. Atypical variants of TCMP including the basal [15] and mid ventricular variant [16] with DSE have also been previously described.

Chandraprakasam et al. presented a robust review on dobutamine-induced TCMP and concluded that patients developing dobutamine-induced TCMP were largely females whose cardiovascular risk factor profile was similar to those patients who developed TCMP due to other causes, with 90% of the study population having hypertension, hyperlipidaemia, and smoking as predisposing risk factors [17].

The mechanism of dobutamine-induced TCMP remains unclear and numerous theories have been put forward to explain the aetiology including the occurrence of multi-vessel coronary vasospasm due to catecholamine surge, a transient LV outflow tract obstruction, or mid cavity obstruction due to systolic anterior motion of the mitral leaflet and drug-induced hypertension [15–17].

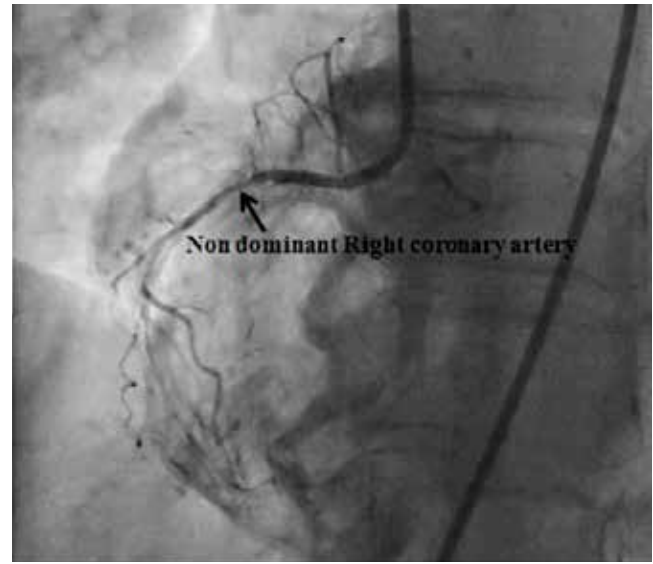


Figure 5. Right anterior oblique view of coronary angiogram showing non dominant right coronary artery

The aetiology of atypical TCMP can be explained by the strong correlation between the location of the WMA and myocardial metabolism. It is postulated that differences in the distribution of myocardial receptors (ie. β -adrenergic receptors) and the difference in metabolism in various areas of the myocardium contribute to the differential location of WMA [18].

In our case, the occurrence of ST-elevations during DSE with a systolic ventricular ballooning abnormality on echocardiography and focal systolic mid-anterolateral and anteroseptal ballooning on left ventriculography, along with a non-obstructive coronary angiogram followed by the reversion of ECG and echocardiographic changes at rest are typical enough to label the case as an occurrence of dobutamine-induced atypical TCMP.

Plaque rupture resulting in WMA was considered as a possible cause of the changes, however, a review of the literature in cases of TCMP suggest that in patients who had TCMP, IVUS did not show evidence of plaque rupture [19]. In our case, the WMA were not confined to a single artery distribution and a non-obstructive left-sided coronary system argued against plaque rupture or disruption and therefore, IVUS was not performed.

Reviewing our case in light of the documented literature [20], we find that our patient had a predisposition to develop TCMP in response to dobutamine due to her age and gender. She was an active smoker and data suggests that smoking predisposes patients to developing TCMP because of endothelial injury induced by smoking. We used atropine to reach the target heart rate, and its use is known to disturb the equilibrium between the parasympathetic and sympathetic autonomic drive leading to enhanced sympathetic tone [21] and thus, contributing to dobutamine-related transient CMP.

To the best of our knowledge, this is the first case report of dobutamine-induced focal mid-ventricular variant of stress cardiomyopathy originating from the Indian subcontinent.

Conclusion

1. Dobutamine stress testing is a relatively safe investigation for patients who are unable to exercise and has mild side effects. However, rarely, dobutamine stress testing can induce a transient cardiomyopathy, particularly if atropine is used.
2. Dobutamine-induced cardiomyopathy can present as both a typical pattern of wall motion abnormalities or as an atypical pattern with focal wall motion abnormalities.
3. Dobutamine-induced takotsubo cardiomyopathy predominantly involves female patients, who are smokers and have risk factor profiles similar to those patients who developed TCMP due to other causes.

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