Stress cardiomyopathy induced during dobutamine stress echocardiography

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ABSTRACT

Background: Catecholamines play a central role in pathogenesis of stress cardiomyopathy (SC). We aimed to review the clinical characteristics, procedural details and outcomes of patients with SC during dobutamine stress echocardiography (DSE).

Methods/Results: A total of 20 adults [age 64±15 years, 80% women, 67% hypertension, 20% diabetes, 33% hypercholesterolemia, 19% chronic kidney disease, 13% known anxiety disorder] with SC during DSE were identified from local digital archives of our laboratory (n=3) or reports in English literature (n=17). Indication for DSE was suspected coronary artery disease (CAD) in all patients. Left ventricular (LV) ejection fraction was normal at baseline. SC developed at a blood pressure of 154±47/86±24 mmHg, heart rate of 130±17 bpm (88±10% predicted maximum) and peak rate-pressure product of 20559±3898 mmHg*bpm. ST segment elevation was seen in 65%. SC occurred at peak dobutamine infusion rate of 38±6 μg/kg/min in 85% and during recovery in 15%. Atropine [0.7±0.6 (0.25‑2) mg] was given to 7 patients. LV ejection fraction dropped to 30±6% with apical (40%), apical and mid (45%) or basal and mid (10%) circumferential LV ballooning. One patient (5%) had a mixed pattern of wall motion abnormality. LV outflow tract obstruction developed in 15%. Major adverse cardiac events occurred in 7 (35%) and included death (n=1), congestive heart failure (n=2), hypotension (n=3) and atrial fibrillation with heart failure (n=1). At a mean follow up duration of 19±19 days, complete or partial recovery of LV wall motion abnormality was seen in 18 and 1 patient, respectively.

Conclusion: SC uncommonly occurs during DSE. However, death and other adverse events (hypotension, heart failure and atrial fibrillation) may occur and require urgent attention. Once managed, complete recovery is expected in most patients.

Key Words: Complications, dobutamine stress echocardiography, dobutamine, outcomes, stress cardiomyopathy, takotsubo

INTRODUCTION

Stress cardiomyopathy (SC), also known as transient left ventricular regional ballooning syndrome, was first fully described by the unifying term “takotsubo” by Dote et al.[1] It is mostly characterized by circumferential wall motion abnormalities in the apical, mid, or basal portions of the left ventricle (LV) singly or in various combinations. It is often associated with electrocardiographic (ECG) abnormalities including ST-segment elevation, T-wave inversion, and Q-waves, suggestive of acute coronary syndrome in addition to mild elevation of cardiac enzymes.[1-3] The pathophysiology of SC is not well understood. However, there is compelling evidence...
for a central role of endogenous catecholamines in its pathogenesis.\(^3,4\) In addition, exogenously administered catecholamines such as epinephrine, dobutamine, isoproterenol, terbutaline, phenylephrine, p-synephrine, albuterol, and salmeterol have been associated with the development of SC.\(^5\)

Table 1: Demographic and clinical characteristics of twenty adults with stress cardiomyopathy induced by dobutamine stress echocardiography

| Number | Years | 1st author | Age (years) | Sex | Diabetes | Hypertension | Hyperlipidemia | Psychological factors | DSE indication | Peak dobutamine infusion* (µg/kg/min) | Atropine (mg) | SC phenotype | Complete recovery |
|--------|-------|------------|-------------|-----|----------|--------------|----------------|---------------------|---------------|--------------------------------------|--------------|----------------|-----------------|
| 1      | 2008  | Silberbauer\(^9\) | 75          | Female | − | − | + | + | Chest pain | 30 | 0.5 | Apical | + |
| 2      | 2008  | Cherian\(^10\) | 85          | Female | − | − | + | − | Preoperative | 40 | NA | Apical | − |
| 3      | 2009  | Vasconcelos Filho\(^11\) | 76          | Female | − | + | − | − | Suspected CAD | NA | NA | Apical and mid | + |
| 4      | 2009  | Margery\(^12\) | 61          | Female | − | + | − | − | Exertional dyspnea | 40 | NA | Apical and mid | + |
| 5      | 2009  | Abraham\(^13\) | 46          | Male | NA | NA | NA | NA | Preoperative | 40 | NA | Apical | + |
| 6      | 2009  | Vasconcelos Filho\(^11\) | 46          | Female | NA | NA | NA | NA | Atypical chest pain | 40 | NA | Apical and mid | + |
| 7      | 2010  | Mosley\(^14\) | 50          | Female | − | − | − | + | Chest pain | 30 | 0.5 | Apical and mid | − |
| 8      | 2010  | Silberbauer\(^9\) | 74          | Female | − | + | − | + | Chest pain | 40 | 0.25 | Apical | + |
| 9      | 2011  | Shah\(^15\) | 85          | Female | − | + | − | − | Exertional dyspnea | 30 | NA | Apical | + |
| 10     | 2011  | Cadeddu\(^16\) | 48          | Female | − | − | − | − | Atypical chest pain | 40 | 0.5 | Mid and basal | + |
| 11     | 2011  | Arias\(^17\) | 77          | Female | − | + | − | − | NA | 40 | NA | Apical | + |
| 12     | 2012  | D’Alloia\(^18\) | 56          | Female | NA | NA | NA | NA | Suspected CAD | 30 | NA | Apical and mid | + |
| 13     | 2012  | Chia\(^19\) | 53          | Male | − | − | − | + | Chest pain | 40 | NA | Apical and mid | + |
| 14     | 2012  | Ho\(^20\) | 83          | Female | − | + | − | − | Suspected CAD | 40 | 0.5 | Apical | + |
| 15     | 2012  | Shah\(^21\) | 71          | Female | − | + | − | − | Exertional dyspnea | 40 | NA | Apical | + |
| 16     | 2013  | Fineschi\(^22\) | 73          | Male | + | + | + | − | Suspected CAD | 40 | NA | Apical | + |
| 17     | 2019  | Present study | 74          | Female | − | + | − | − | Chest pain | 50 | 2.0 | Mixed | + |
| 18     | 2019  | Present study | 35          | Male | + | + | + | − | Preoperative | 50 | 0.3 | Apical and mid | + |
| 19     | 2019  | Present study | 61          | Female | − | + | − | − | Preoperative | 30 | NA | Apical and mid | + |

*µg/kg/min, Died, Partial recovery of wall motion was noted 2 days after the event, NA: (information) Not available. CAD: Coronary artery disease, DSE: Dobutamine stress echocardiography, SC: Stress cardiomyopathy, +: present, −: Absent

Figure 1: Flow diagram demonstrating the literature search process in selecting included cases in this study
Dobutamine stress echocardiography (DSE) is a well-established and safe pharmacologic alternative to exercise stress testing for diagnosis and risk stratification of obstructive coronary artery disease (CAD). In properly selected patients, DSE is uncommonly associated with serious and life-threatening complications. SC has been uncommonly reported in association with DSE. This study aimed to review the patient characteristics and procedural details of DSE-associated SC in reported cases as well as additional patients encountered in our own laboratory.

**METHODS**

### Study patients

An exhaustive literature search on PubMed using the search phrases “dobutamine stress echocardiography and takotsubo,” “dobutamine stress test induced takotsubo,” “stress test induced takotsubo,” and “DSE induced takotsubo” yielded 14 publications (17 cases) between the years 2008 and 2013 [Figure 1 and Table 1]. Studies were excluded if they were not strictly case reports of SC during DSE. In addition, a cluster of three patients from our institution were included. The demographic, clinical, and procedural information in the latter three patients is detailed in Table 2. In total, twenty patients with DSE-induced SC were identified. SC was defined as LV wall motion abnormality not confined to an epicardial coronary artery blood supply distribution in a patient with normal baseline wall motion and no obstructive CAD on invasive angiography.

### Statistical analysis

Continuous variables were reported using mean and standard deviation, whereas discrete variables were reported as percentages. Segmental LV wall motion abnormalities were identified based on the 16-segment model and were analyzed separately for each patient.

### RESULTS

#### Study patients

The mean age of the patients was 64 ± 15 years, and 80% were women. Major comorbidities reported in those in whom baseline characteristics were known included hypertension (69%), hyperlipidemia, (38%), diabetes (13%), and chronic kidney disease (19%) [Table 3]. Only 13% of the cases had known anxiety disorder and 25% of cases were reported to have been in significant emotional distress at or around the time of their DSE.

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**Table 2: Demographic, clinical, and procedural details of three patients with dobutamine stress echocardiography-induced stress cardiomyopathy from our institution**

| Patient | 1 | 2 | 3 |
|---------|---|---|---|
| Age (years) | 74 | 35 | 61 |
| Sex | Female | Male | Female |
| CAD risk factors | | | |
| Hypertension | + | + | + |
| Diabetes | – | + | – |
| Hypercholesterolemia | – | + | + |
| Smoking | – | + | – |
| Family history of premature CAD | – | – | – |
| Known atherosclerosis | – | – | +* |
| CAD | – | – | +* |
| Stroke | – | – | – |
| Peripheral vascular disease | – | – | – |
| Chronic renal failure (mg/dl) | + | + | + |
| Blood urea nitrogen | 46 | 37 | 50 |
| Serum creatinine | 2.7 | 13.1 | 6.1 |
| Hemoglobin (g/dl) | 10.2 | 11.6 | 13.7 |
| Hematocrit (%) | 30.2 | 34.0 | 40.0 |
| Resting echocardiogram | Normal | LV hypertrophy | Normal |
| Resting electrocardiogram | Normal | LV hypertrophy | Normal |
| Peak dobutamine dose (µg/kg/min) | 50 | 50 | 30 |
| Total atropine dose (mg) | 2 | 0.3 | 0 |
| Peak heart rate (beats/min) | 113 | 157 | 135 |
| Percentage maximum predicted heart rate achieved | 77 | 85 | 85 |
| Peak systolic/diastolic blood pressure (mmHg) | 210/100 | 109/60 | 140/80 |
| Symptoms at peak dobutamine infusion | None | None | Belching |
| ECG changes at peak stress | None | ST elevation (inferior and apical) | ST elevation (inferior, anterior and apex) |
| LV wall motion abnormality at peak stress | Mixed | Apical and mid | Apical and mid |
| Coronary angiography following DSE | Mild luminal irregularities | Mild luminal irregularities | Mild luminal irregularities |

*Nonobstructive. CAD: Coronary artery disease, DSE: Dobutamine stress echocardiography, ECG: Electrocardiogram, LV: Left ventricle (ventricular), ST: Segment, +: present, –: Absent
Table 3: Summary of demographic and clinical characteristics of twenty adults with dobutamine stress echocardiography-induced stress cardiomyopathy

| Variable | n (%) or range (mean±SD) |
|----------|--------------------------|
| Demographic information (n=20) |  |
| Age (years)* | 35-85 (64 ± 15) |
| Male:female | 4 (20):16 (80) |
| Cardiac risk factors (n=16) |  |
| Diabetes | 2 (13) |
| Hypertension | 11 (69) |
| Hyperlipidemia | 6 (38) |
| Current smoking | 3 (19) |
| Past smoking | 1 (6.3) |
| Family history of premature CAD | 1 (6.3) |
| Family history of CAD | 1 (6.3) |
| Known CAD | 1 (6.3) |
| Obesity | 0 |
| Other comorbid conditions (n=16) |  |
| Chronic obstructive pulmonary disease | 1 (6.3) |
| Chronic kidney disease | 3 (19) |
| Peripheral or carotid arterial disease | 1 (6.3) |
| Psychological factors (n=16) |  |
| Anxiety disorder | 2 (13) |
| Depression | 3 (19) |
| Significant emotional distress | 4 (25) |
| History of atypical chest pain | 6 (38) |

*Numbers in parentheses indicate available data in the cohort. CAD: Coronary artery disease, SD: Standard deviation

Baseline electrocardiogram
Baseline ECG was described as normal in 69%, while ST-segment/T wave abnormalities or LV hypertrophy were seen in 20% and 13%, respectively. Right bundle branch block was present in one patient and none had left bundle branch block.

Baseline echocardiography
LV wall motion and qualitative systolic function (ejection fraction 55–66 [58 ± 5] %) was normal in all patients. There was no report of significant pericardial, valvular, or myocardial abnormality other than LV hypertrophy that was present in 24% of the patients.

Development of stress cardiomyopathy during dobutamine stress echocardiography
The peak dobutamine infusion rate was 30–50 (38 ± 6) µg/kg/min, and seven (35%) patients had received 0.25–2 (0.70 ± 0.60) mg of atropine at the time of SC development. Symptoms of chest discomfort (80%), shortness of breath (17%), back pain (17%), or belching (8%) were noted at the time of developing SC and one patient had paradoxical sinus deceleration. Systolic and diastolic blood pressures at the development of SC were 59–235 (154 ± 48)/44–135 (86 ± 24) mmHg, respectively, at a heart rate of 110–157 (130 ± 17) beats/min (70–105 [88 ± 10] % predicted maximum heart rate). SC developed at the peak stage of DSE in 17 (85%), while 3 (15%) patients developed SC during the recovery phase. ST-segment elevation was reported in 13 (65%) patients, while three (15%) patients had isolated ST-segment depression. T wave abnormality alone was reported in one patient. Ventricular arrhythmias were reported in three patients (15%), and no patient was reported to have had significant supraventricular rhythm abnormality at the time of SC development. Echocardiography and/or invasive ventriculography at the time of SC development revealed LV apical ballooning in eight, LV apical and mid ballooning in nine, LV mid and basal ballooning in two patients, and a mixed pattern (apical ballooning in addition to inferior and inferolateral wall motion abnormality). Basal LV hypercontractility was seen in seven patients, and three patients had evidence of outflow tract obstruction (gradients of 60–120 [88 ± 30] mmHg). Fourteen patients had elevated cardiac troponin levels with a maximum reported value of 5.65 ng/mL.

Outcomes
SC was associated with major adverse cardiac event in seven patients (35%). These included death (n = 1), congestive heart failure (n = 4), hypotension (n = 3), and atrial fibrillation (n = 1). One patient with heart failure required insertion of an intra-aortic balloon pump for hemodynamic support. Overall, 19 of the survived SC and 18 experienced complete recovery of LV function over a follow-up period of 1–90 (19 ± 19) days. The remaining one patient experienced near-complete recovery of LV systolic function 2 days following SC with no reported long-term follow-up.

**DISCUSSION**

The present case series indicates that SC is a rare and distinct complication of DSE. It occurs primarily in older hypertensive women referred for the evaluation of suspected CAD. SC during DSE occurred during dobutamine infusion rates of 30–50 µg/kg/min and an average atropine dose of 0.7 mg and manifested itself mainly at the peak hemodynamic response. One patient developed paradoxical sinus deceleration. SC was characterized as circumferential LV wall motion abnormality involving the apical or a combination of apical and mid-ventricular segments in the majority of patients. One patient, however, had a mixed pattern of LV wall motion abnormality involving the apical segments circumferentially, whereas the mid-inferior and inferolateral segments were involved focally. Atypical forms have been described previously in patients with SC and have been variously defined as any LV wall motion abnormality sparing the apex or mixed patterns of wall motion abnormality as seen in our patient. In general, there have not been significant differences found in clinical presentation or outcomes of patients with typical and atypical SC. In this series, there was no report of right ventricular involvement by SC. Serious adverse cardiac events occurred in 35% of patients in this series and included death, congestive heart failure, hypotension, and atrial fibrillation. The reported death occurred in an 85-year-old woman who...
developed severe chest tightness at dobutamine infusion rate of 40 µg/kg/min.\[10\] She had received no atropine, and her heart rate and blood pressure at the time of chest tightness were recorded as 123 beats/min and 124/62 mmHg, respectively.\[10\] She was found to have circumferential apical LV wall motion abnormality with basal LV hypercontractility as well as severe dynamic outflow tract obstruction.\[10\] Severe heart failure and sudden development of pulseless electrical activity were mentioned as the final reasons for death.\[10\] The latter may have occurred due to a mechanical complication of SC such as LV free wall, septal, or papillary muscle rupture as reported previously.\[26‑28\] In a large international registry of SC, death was also reported to have occurred in ~ 5% of the patients as an in-hospital complication.\[29\]

The pathophysiology of hypotension and heart failure in patients with SC is complex and multifactorial and often involves LV systolic dysfunction, outflow tract obstruction, paradoxical sinus deceleration, inappropriate vasodilation, or a combination of these factors.\[30\] Finally, atrial fibrillation has been reported in patients undergoing DSE and along with SC point to the central role of endogenous and exogenous catecholamine in the pathogenesis of both conditions.\[31\]

Limitations
The current report relies on compiled cases of DSE-induced SC from published reports in English language literature. The findings thus may have been affected by certain bias in underreporting poor outcomes or isolated rare events. In addition, the study is primarily a retrospective analysis of a small number of reported cases with limitations inherent to such an approach. We hope that this report would encourage development of a registry that will help in the understanding of the scope and significance of this unique observation. Controversies continue to exist regarding the uniformity of the pathophysiological mechanisms responsible for the development of SC phenotypes.\[32\] However, at this point, it does appear that catecholamines continue to be central to the process.

CONCLUSIONS
SC is an infrequent distinct complication of DSE. Although it generally has a relatively benign course with favorable overall outcomes, serious adverse events may occur in some individuals. Knowledge of this association should improve timely detection of this type of complication and lead to better preparation for appropriate therapeutic interventions.

Research quality and ethics statement
The authors of this manuscript declare that this scientific work complies with reporting quality, formatting, and reproducibility guidelines set forth by the EQUATOR Network. The authors also attest that this clinical investigation was determined to not require institutional review board/ethics committee review.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

Ethical conduct of research
This study was approved by the Institutional Review Board / Ethics Committee. The authors followed applicable EQUATOR Network (http://www.equator-network.org/) guidelines during the conduct of this research project.

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