

Coronary Spasm and Polymorphic Ventricular Tachycardia One Year After Takotsubo

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Abstract

Stress cardiomyopathy is typically considered to be a disease with a favorable long-term prognosis, with malignant arrhythmias accompanying only the acute phase. We describe a 51-year-old female who presented with palpitations one year after stress cardiomyopathy and complete recovery of apical left ventricular wall motion. Coronary spasm was strongly suspected based on transient ST-segment elevations followed by sustained polymorphic ventricular tachycardia captured on ambulatory Holter. Contrast injection during coronary angiography reproduced spasm and ventricular arrhythmia that resolved with intracoronary nitroglycerine. The patient was intolerant to nitrates therefore discharged on 2 calcium channel blockers. Shared decision was made to implant cardioverter defibrillator.

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Stress cardiomyopathy, or takotsubo syndrome, continues to fascinate clinicians, researchers, and patients. Etiology has been difficult to elucidate in spite of the fact that the disease was described 27 years ago. Suggested culprits have included catecholamine excess, stress-induced complex neurocardiac interplay, coronary microvascular dysfunction, and coronary spasm leading to ischemic stunning.¹⁻³ Coronary spasm was one of the first etiologies proposed by Sato et al.⁴ However, coronary spasm can be found during angiography in only 18% of acute stress cardiomyopathy cases and often does not correlate with the affected areas of the myocardium.⁵

Stress cardiomyopathy is typically thought of as a disease with a favorable long-term prognosis with malignant arrhythmias accompanying mostly the acute phase. However, recent evidence suggests that stress cardiomyopathy carries an unfavorable long-term prognosis, with an estimated 20% to 30% increased risk of death.^{1,6,7}

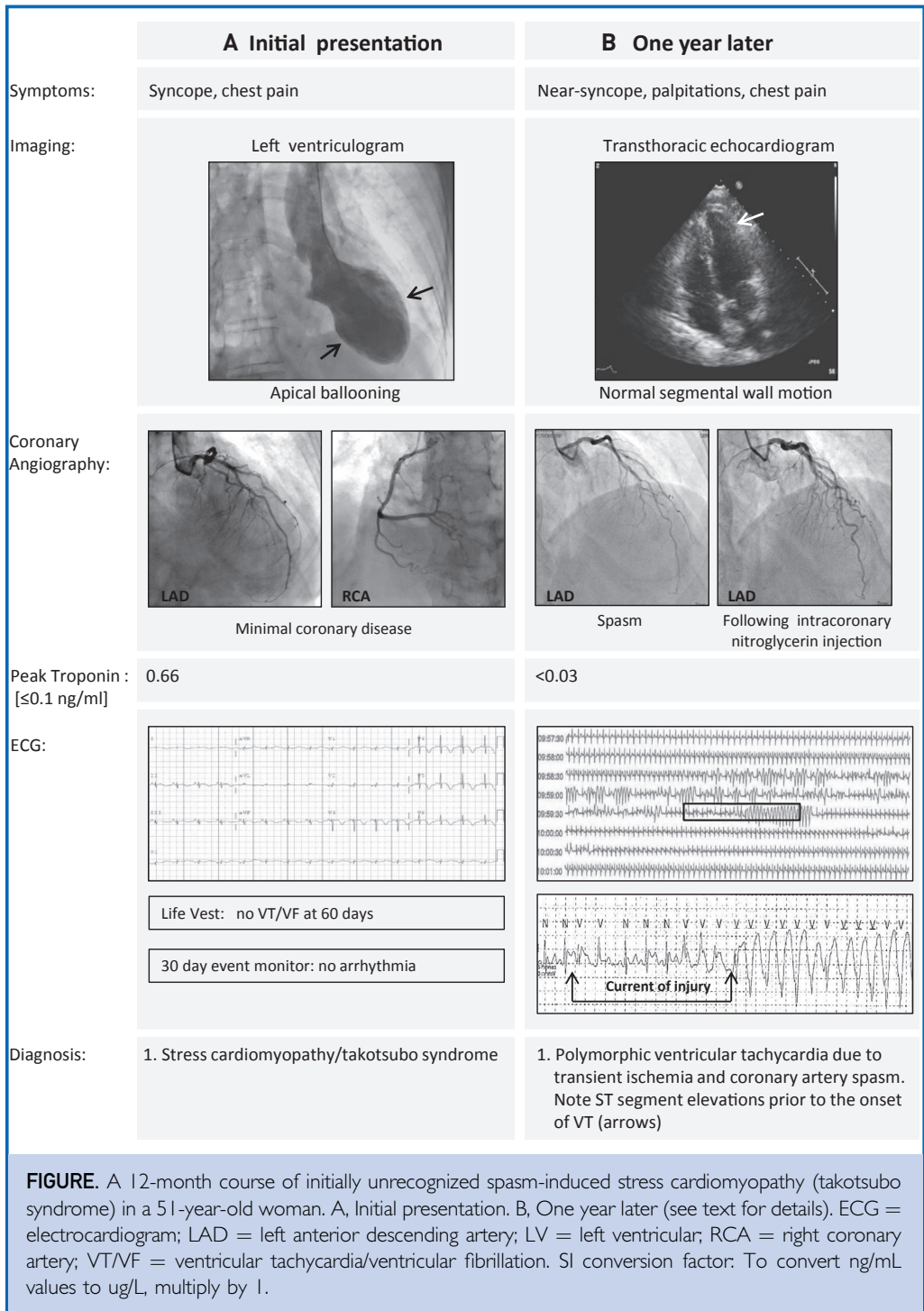
We describe an unusual case of transient ischemia-induced polymorphic ventricular tachycardia (VT) in a female patient one year after diagnosis of stress cardiomyopathy. To

our knowledge, no coronary spasm-induced ventricular arrhythmia has been reported in takotsubo survivors beyond the acute phase.

CASE REPORT

A 51-year-old woman with a significant smoking history and anxiety presented to the emergency department after an episode of syncope while sitting, followed by 12 hours of typical chest pain radiating to her left shoulder with associated dyspnea. One sublingual nitroglycerin tablet completely relieved her symptoms; however, initial troponin T level was detectable at 0.66 ng/mL (reference, 0-0.1 ng/mL; to convert ng/mL values to ug/L, multiply by 1) and prompted admission for acute coronary syndrome. T-wave inversions in leads V₄ through V₆ were noted on 12-lead electrocardiogram. Transthoracic echocardiogram (TTE) revealed severely decreased left ventricular ejection fraction (<30%), with a large area of apical akinesis extending to mid-segments. Cardiac catheterization confirmed minimal diffuse coronary artery disease and elevated left ventricular end-diastolic pressure at 28 mm Hg (reference, ≤14 mm Hg). The patient was diagnosed with stress cardiomyopathy with a typical apical ballooning pattern

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(Figure A). However, no specific stressor could be identified other than the underlying anxiety. The patient was discharged with metoprolol and lisinopril and wearing a life vest, which recorded no arrhythmic events at 60 days. Repeat TTE 2 months later revealed complete

left ventricular segmental wall motion recovery with ejection fraction 60%.

Six months after the initial presentation, the patient had recurrent episodes of shortness of breath and palpitations. An ambulatory 30-day event monitor was placed and captured

53 episodes of dyspnea, none of which correlated with any significant arrhythmia. Her symptoms were attributed to anxiety and lead to an increase in anti-anxiety therapy. The patient continued to smoke tobacco despite extensive counseling.

One year after the initial presentation, the patient reported “not feeling well” and having palpitations, so ambulatory Holter monitor was placed. Next day, she developed left arm numbness, dizziness, and near-syncope while driving. On arrival to the emergency department, she was asymptomatic, 12-lead electrocardiogram was unremarkable, electrolytes within the reference range, and serial troponin levels remained undetectable. Holter interrogation revealed transient sinus tachycardia with prominent ST-segment elevations, followed by a large burden of polymorphic VT that correlated with the patient’s symptoms (Figure B). Based on these findings, transient ischemia due to coronary spasm was strongly suspected. Contrast injection during coronary angiography precipitated diffuse left anterior descending coronary spasm and polymorphic VT that resolved with intracoronary nitroglycerine administration; again, only minimal epicardial coronary artery disease was found. Cardiac magnetic resonance imaging and TTE were completely normal.

At that point, β -blocker was discontinued and replaced with 2 calcium channel blockers (amlodipine and diltiazem) as she was intolerant to long-acting nitrates. Extensive counseling to promote tobacco cessation was provided. Before discharge, the patient underwent exercise treadmill stress test that ruled out catecholamine-induced polymorphic VT. There was a mutual agreement to proceed with implantable cardioverter-defibrillator (ICD). At 4-month follow-up, ICD interrogation did not reveal any recurrent ventricular arrhythmia.

DISCUSSION

Stress cardiomyopathy is diagnosed on the basis of clinical presentation, typical cardiac imaging pattern of transient myocardial wall motion abnormalities extending beyond the territory of one coronary vessel, and normal coronary arteries on angiography.^{1,8}

Stress cardiomyopathy is typically thought of as a disease with a relatively favorable long-term prognosis, with malignant arrhythmias almost always accompanying the acute phase. Our patient presented with near-syncope and a high burden of polymorphic VT proceeded by transient ST-segment elevations one year after she was diagnosed with stress cardiomyopathy. This case suggests that selected patients may be at the long-term risk of sudden death due to recurrent spasm-induced ventricular arrhythmia. A few recent reports also support that stress cardiomyopathy survivors may be at increased risk of both atrial and ventricular arrhythmias 6 weeks to 8 years from left ventricular segmental wall motion recovery.⁹⁻¹¹

On the basis of the presented case, we believe that recurrent coronary spasm may be one of the mechanisms responsible for electrical instability in takotsubo survivors. Therefore, in patients with a history of stress cardiomyopathy presenting with unexplained syncope/near-syncope, coronary spasm-induced ventricular arrhythmia should be considered as a potential underlying mechanism. Young women with risk factors, such as severe anxiety and tobacco use, are at particularly high risk of developing coronary spasm-induced myocardial stunning and arrhythmia, even in the absence of documented spasm during routine coronary angiography.

Once diagnosed, the long-term management of stress cardiomyopathy is generally supportive, primarily consisting of treating associated heart failure with angiotensin-converting enzyme inhibitors, β -blockers, and aspirin as well as individualized anticoagulation and diuretics.¹² We would like to remark that β -blockers alone are ineffective in preventing recurrent coronary spasms or related electrical instability. Calcium channel blockers alone, or in combination with nitrates, would be more appropriate under such circumstances; however, they are currently not recommended by 2014 American College of Cardiology/American Heart Association guidelines. Finally, patients with a history of spasm-induced ventricular arrhythmia are at a lifelong risk of sudden death and may benefit from ICD implantation.¹³

CONCLUSION

This case provides insight into the natural history of stress cardiomyopathy, suggesting that it may be associated with the long-term risk of sudden cardiac death. Sympathetic alterations may persist beyond the acute phase, leading to recurrent coronary spasm and life-threatening ventricular arrhythmias. In patients with history of stress cardiomyopathy presenting with unexplained syncope/presyncope, coronary spasm-induced ventricular arrhythmia should be considered as a potential underlying mechanism. The need for a defibrillator in stress cardiomyopathy survivors raises a clinical dilemma.

Abbreviations and Acronyms: **ICD** = implantable cardioverter-defibrillator; **TTE** = transthoracic echocardiogram; **VT** = ventricular tachycardia

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