

Stress during MRI Causes Cardiomyopathy! Repeatedly! - A Case Report and Review of Pathogenesis

A. Singh^{1*}, A. Murthy², A. Schussheim³, G. Lancaster⁴

¹Arti Singh MD, Resident, Department of Internal Medicine, Bridgeport Hospital, Bridgeport, CT, USA

²Avinash Murthy MD, Fellow, Division of Cardiology, Department of Internal medicine, Bridgeport Hospital, Bridgeport, CT, USA

³Adam Schussheim MD, Clinical Cardiologist, Cardiac Specialists PC, Fairfield, CT, USA

⁴Gilead Lancaster MD, Director of Non-Invasive Cardiology, Division of Cardiology, Department of Internal Medicine, Bridgeport Hospital, Bridgeport, CT, USA

*Corresponding author: artisingh2060@yahoo.com

Received May 08, 2014; Revised May 27, 2014; Accepted May 28, 2014

Abstract Stress cardiomyopathy is characterized by reversible wall motion abnormalities and transient systolic dysfunction in the absence of obstructive CAD or acute plaque rupture. Majority of cases involve intense emotional or physical stress triggers in post-menopausal women. Catecholamine surge, one postulated mechanism, causes acute but completely reversible myocardial dysfunction. MRI can be associated with significant stress, particularly in claustrophobic people. Our patient, with no cardiac or psychiatric history, demonstrated a stressful response to MRI developing Takotsubo cardiomyopathy (TCM) both times after the MRI in 2009 and 2013. Her anxiety likely provoked the TCM. Coronary angiography showed no obstructive disease and echocardiography showed wall motion abnormalities and systolic dysfunction, with full recovery each time confirmed on repeat echo. Patient was managed with conservative medical therapy both times. A thorough literature search failed to reveal any previously reported cases of recurrent cardiomyopathy precipitated by anxiety from MRI scan. Judicious use of anxiolytics may have averted a stressful response in our patient, perhaps preventing the development of TCM.

Keywords: cardiovascular medicine, cardiomyopathy, echocardiography, coronary angiography, takotsubo

Cite This Article: A. Singh, A. Murthy, A. Schussheim, and G. Lancaster, "Stress during MRI Causes Cardiomyopathy! Repeatedly! - A Case Report and Review of Pathogenesis." *American Journal of Medical Case Reports*, vol. 2, no. 5 (2014): 97-101. doi: 10.12691/ajmcr-2-5-2.

1. Manuscript

An eighty two year old female underwent outpatient magnetic resonance imaging (MRI) scan for shoulder pain. Her past medical history was notable for chronic atrial fibrillation, valvular mitral regurgitation, morbid obesity, osteoarthritis, and chronic low back pain. She was anxious going into the MRI scanner, and while in the scanner she complained of acute shortness of breath. Emergency medical services noted agonal respirations, hypoxia, tachycardia, and elevated blood pressure; she was intubated on the scene, and transferred to our hospital for further management. Her electrocardiogram (ECG) was notable for atrial flutter with rapid ventricular response (RVR) at 140 beats per minute (bpm) with antero-septal ST segment elevations (Figure 1). She underwent emergent cardiac catheterization, the angiograms did not reveal any obstructive coronary artery disease (CAD) (Figure 2, Figure 3). Chest radiography was notable for cardiomegaly and bilateral pulmonary edema. 2D echocardiography (Echo) revealed a normal size left ventricle (LV), severe hypokinesia with an estimated ejection fraction of less than 25% of the left ventricle with preserved function only at the base (Figure 4). She was

diagnosed with Takotsubo cardiomyopathy (TCM) and was managed conservatively. She responded to the treatment well, and repeat echocardiography done 4 days later showed good LV function with an estimated EF at 50-55%. No regional wall motion abnormalities were present (Figure 5).

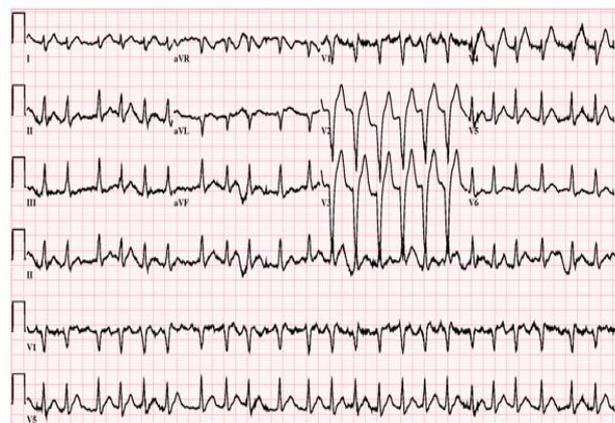


Figure 1. ELECTROCARDIOGRAM, SHOWING ATRIAL FLUTTER WITH RAPID VENTRICULAR RESPONSE AND ANTERO-SEPTAL ST SEGMENT ELEVATIONS, 2009 TCM EVENT

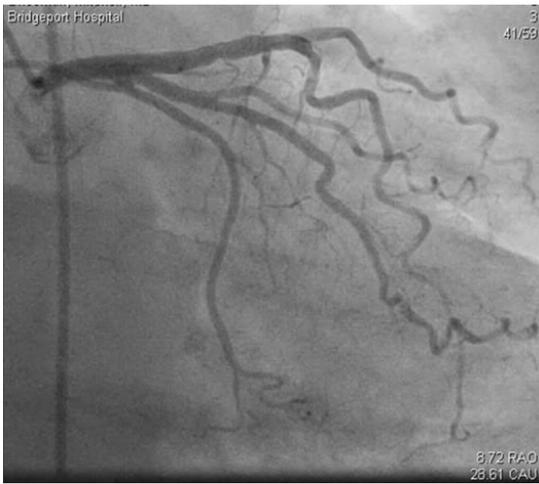


Figure 2. CORONARY ANGIOGRAPHY, RAO VIEW, SHOWING LEFT CORONARY ARTERY SYSTEM FREE OF OBSTRUCTIVE CORONARY ARTERY DISEASE

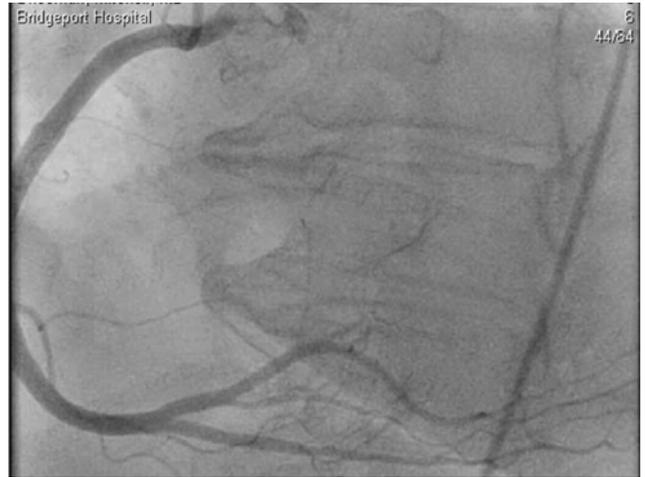


Figure 3. CORONARY ANGIOGRAPHY, LAO VIEW, SHOWING RIGHT CORONARY ARTERY SYSTEM FREE OF OBSTRUCTIVE CORONARY ARTERY DISEASE

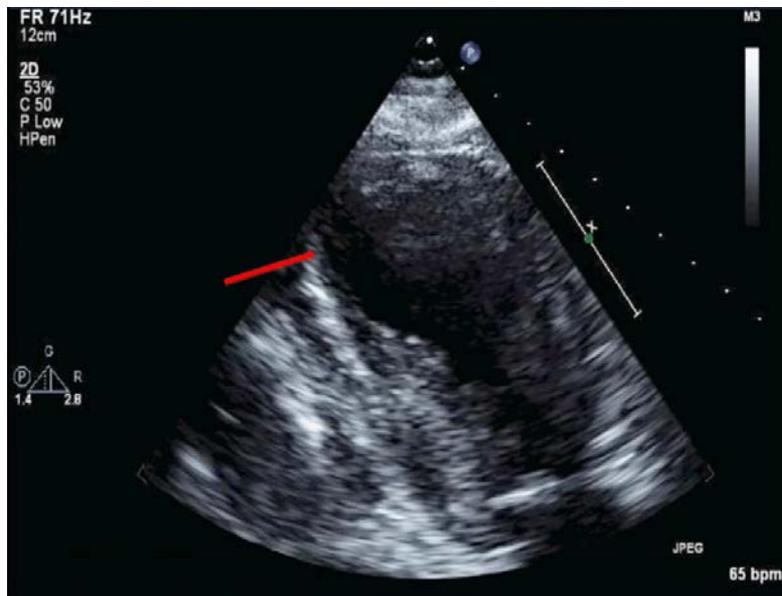


Figure 4. 2D ECHOCARDIOGRAPHY, 2 CHAMBER VIEW, SHOWING APICAL BALLOONING AT END-SYSTOLE, 2009 TCM EVENT



Figure 5. 2D ECHOCARDIOGRAPHY, 2 CHAMBER VIEW, SHOWING RESOLUTION OF APICAL BALLOONING, AND A CONTRACTILE APEX AT END-SYSTOLE, 2009 TCM RECOVERY

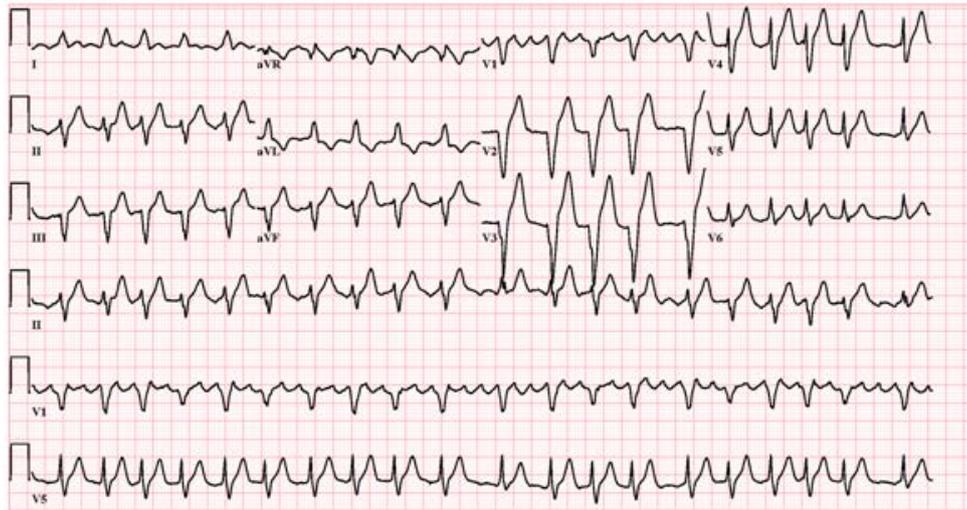


Figure 6. ELECTROCARDIOGRAM, SHOWING ATRIAL FLUTTER WITH RAPID VENTRICULAR RESPONSE AND ANTERO-SEPTAL ST SEGMENT ELEVATIONS, 2013 TCM EVENT



Figure 7. 2D ECHOCARDIOGRAPHY, 4 CHAMBER VIEW, SHOWING APICAL BALLOONING AT END-SYSTOLE, 2013 TCM EVENT

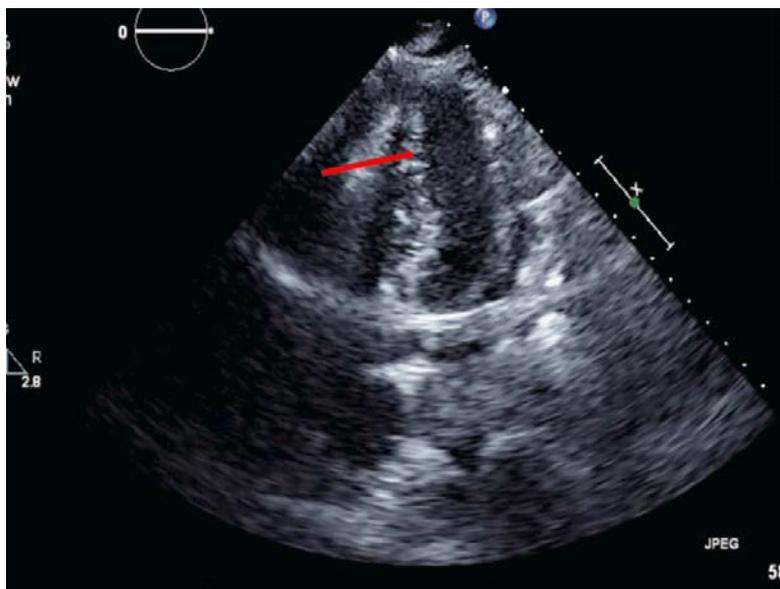


Figure 8. 2D ECHOCARDIOGRAPHY, 4 CHAMBER VIEW, SHOWING RESOLUTION OF APICAL BALLOONING, AND A CONTRACTILE APEX AT END-SYSTOLE, 2013 TCM RECOVERY

Four years later in April 2013, she was scheduled for another MRI as part of a pelvic mass workup during which she again was anxious going into the scanner and became dyspneic during the examination. She was intubated, and transferred to our hospital for further care. She was again found to be in atrial flutter with RVR (128 bpm), with elevated blood pressure (Figure 6). She did not undergo cardiac catheterization as her presentation was similar to her previous TCM. An echocardiogram done on the day of admission showed severely reduced LV function with an estimated ejection fraction of 20-25%, with severe hypokinesis in the anterior wall (Figure 7). Given the similarities of this presentation to the TCM episode, she was again diagnosed with TCM, managed conservatively, and responded well to medical management alone. Repeat echocardiogram 10 days later showed low normal systolic function (Figure 8). We present a rare case of recurrent stress cardiomyopathy precipitated by MRI.

2. Discussion

Takotsubo cardiomyopathy (TCM) is known by various names like stress cardiomyopathy, transient left ventricular ballooning syndrome, “broken heart syndrome”, or neurogenic myocardial stunning. It is characterized by reversible wall motion abnormalities and transient systolic dysfunction of the apical and/or mid segments of the left ventricle in the absence of obstructive CAD or acute plaque rupture. This pattern of wall motion abnormality represents the “typical” form of stress-induced cardiomyopathy where the contractile functions of the mid and apical segments of the LV are depressed, and there is hyperkinesis of the basal walls, producing a balloon-like appearance of the distal ventricle with systole. In the “atypical” or “apical sparing” variant, the ventricle hypokinesis is restricted to the mid-ventricle or basal segments, with apical sparing.

Most TCM cases are triggered by either emotional or physical stressors. Women account for the vast majority of the cases, with mean age of greater than 60 years. Emotional stressors cited in various case reports include death or critical illness of loved ones, arguments, natural disasters, and devastating financial losses. Physical stressors include extreme exercise, alcohol/opiate withdrawal, cocaine or illicit drug abuse, surgeries, sepsis, trauma, CVAs or a stress test. However, we were not able to find any reported cases in the literature where MRI precipitated TCM.

Multivessel coronary artery spasm as a mechanism of TCM is no longer strongly supported in literature. [1] The most supported postulated mechanism for TCM is that of catecholamine excess from severe emotional or physical stress that causes transient contractile dysfunction from microvascular dysfunction and myocardial stunning. Catecholamine excess is also associated with direct myocardial toxicity; this is supported by endomyocardial biopsies in TCM patients demonstrating contraction band necrosis and mononuclear inflammatory infiltrates indicative of catecholamine-mediated cardiotoxicity. [2] Catecholamine surge may also lead to the development of transient LV Outflow Tract (LVOT) obstruction, apical, midventricular ischemia and corresponding wall motion

abnormalities. Studies have proposed that certain genetic profiles may predispose or make people more susceptible to TCM [3].

The mechanism of neurally mediated adrenoceptor trafficking in the setting of catecholamine excess is becoming widely supported. Any stressor that increases circulating catecholamines to supra-physiologic levels can induce TCM in susceptible patients. Of the catecholamines, norepinephrine exerts its action mainly through beta1 receptors which only couples to Gs (s for stimulatory) protein subunit, with overall inotropic effect especially during a catecholamine surge. In the human heart, the basal region has the highest density of sympathetic neurons and thus the greatest effect of norepinephrine is in the base. Circulating epinephrine, however, has higher affinity for beta 2 receptors, and has a predominant effect in the mid-ventricular and apical segments. At supra-physiologic levels of epinephrine (as in during a TCM inciting stressor), the beta 2 receptor switches from Gs to Gi (i for inhibitory) protein. This phenomenon is known as signal trafficking, and has an overall inhibitory effect on myocardial contractility during a catecholamine surge. [4] During a TCM inciting stressor, which leads to epinephrine and norepinephrine surge, there will be basal hyperkinesis (mainly due to norepinephrine effects) with mid-ventricular and apical dyskinesis or akinesis (mainly due to epinephrine effects). This theory of catecholamine surge with signal trafficking is the leading pathophysiologic explanation accounting for the “typical” wall motion pattern in TCM found on echocardiography. This neural mechanism is believed to play a protective role by allowing a portion of the myocardium to “hibernate”. In the setting of extreme stress, if the heart is prevented from maximally contracting indefinitely, myocyte necrosis can be potentially avoided. It is believed to be an evolutionary survival mechanism ensuring a functional myocardium if the organism survives the inciting stressor. [5] In our case, claustrophobia was the likely potent stressor causing intense surge in catecholamines resulting in TCM.

The clinical presentation of TCM is similar to that of Acute Myocardial Infarction (AMI) with common presenting signs and symptoms of acute sub-sternal chest pain, dyspnea, syncope, shock, ECG abnormalities, and positive cardiac biomarkers. Chest pain is the most common presenting symptom in 70-90%, with dyspnea present in only 20% of TCM cases. Syncope, pulmonary edema, cardiac arrest or shock, and life-threatening ventricular arrhythmias secondary to high circulating catecholamine are rare presentations. [1,6] The troponin elevations are mild despite the severe hemodynamic compromise, and extent of wall motion abnormalities noted on echocardiography. The prevalence of TCM ranges from 0.7-1% in the population. [7] Recurrence rate of TCM, which was once thought to be rare (1-2%), is now believed to be as high as 5-10% due to increasing awareness and thus recognition of this syndrome [1,8].

TCM, therefore, is an important differential to consider in post-menopausal females presenting with chest pain and have a clear history of emotional or physical inciting stressor. Differential diagnosis for chest pain are broad, and not limited to ACS, myocarditis, coronary artery dissection, coronary vasospasm. Cardiac MRI should be considered an important complementary tool to

echocardiography and coronary angiography, especially when the diagnosis remains unclear. Cardiac MRI is a valuable tool for detecting inflammation, edema, necrosis, fibrosis, and is the best tool for diagnosing right ventricular involvement in TCM. [9] It can also help distinguish between myocarditis, myocardial infarction and stress cardiomyopathy. In Cardiac MRI TCM has minimal to no late gadolinium enhancement (LGE), which can aid in making the correct diagnosis and guide appropriate management without delay.

TCM resolves spontaneously when the catecholamine storm abates and thus stress reduction should be part of both acute and ongoing management strategy, especially if TCM is complicated by continued stress or if the patient suffers a repeat experience from the same stressful event as noted in our patient. TCM is a transient condition, usually fully reversible, however cases of TCM with persistent apical dysfunction have been reported. [10,11] TCM is mainly treated with supportive care and conservative medical management.

Given the LV systolic dysfunction, it is reasonable to treat these patients with Beta Blockers (BB) and Angiotensin-Converting-Enzyme Inhibitors (ACE-I). Given that TCM may recur (as it did in our patient), one can consider maintaining "at risk" patients on either a BB or combined Alpha and Beta blocker indefinitely. However, Beta blockers have not proven effective in preventing either the initial or recurrent TCM events. [12] The role of mood stabilizers and anti-depressants in TCM have not been well studied [7].

TCM, though generally fully reversible, is not entirely benign and patients can develop complications similar to those of patients with coronary artery disease. The overall, long-term survival of TCM patients is comparable to that of the general age-matched population. [3] However, the prognosis in the acute setting is similar to that of patients presenting with an AMI, and thus TCM patients should be closely monitored for development of heart failure, cardiogenic shock, malignant arrhythmias, LVOT obstruction, LV mural thrombi and death. CHF occurs in approximately 20% of TCM cases, and is more likely if right ventricle is also involved. [3] Echocardiography is used in TCM for monitoring the patient and to document recovery. Provided the patient survives the acute complications of TCM, nearly all patients experience complete recovery within days to weeks from onset of the event.

3. Conclusion

In our case, claustrophobia was the likely stressor, as our patient demonstrated a stressful response to MRI both times developing TCM. It is plausible the patient's chronic atrial fibrillation and flutter with rapid ventricular response and high blood pressure could have resulted in the heart failure or perhaps the emergent intubation was responsible for the TCM. However, in our patient what came first was her anxiety, as she was clearly noted to be anxious going into the MRI scanner both times. This anxiety likely caused a surge in the "fight or flight" catecholamine hormones which induced both stress cardiomyopathy and further arrhythmogenesis. The ensuing atrial fibrillation with RVR likely caused flash

pulmonary edema, worsened by the patient's systolic dysfunction from the concomitant stress cardiomyopathy triggered by her severe emotional anxiety from the MRI scan.

This case increases awareness that stress should not be taken lightly, especially in the elderly. Thorough education and maybe the use of upfront anxiolytics could have averted a stressful response from our patient, probably avoiding both of the events. It is crucial to consider TCM in the differential of a post-menopausal female presenting with dyspnea in the setting of acute emotional or physical stress. We report a rare case of recurrent TCM caused by MRI.

Funding Source(s)

None.

Conflicts of Interest

None. All authors of this work state that there are no conflicts of interest. All authors meet the criteria for authorship and attest to its authorship. The work represented is original, and accept responsibility for the scientific content of the manuscript. Again, there are no conflicts of interest, and agree to release the copyright should the manuscript be accepted for publication.

References

- [1] Hurst, R.T., Prasad, A., Askew, J.W 3rd. et al. Takotsubo Cardiomyopathy: A Unique Cardiomyopathy With Variable Ventricular Morphology. *JACC Cardiovasc Imaging*. 2010 Jun;3(6):641-9.
- [2] Bybee, K.A., Prasad, A. Stress-related cardiomyopathy syndromes. *Circulation*. 2008 Jul 22;118(4):397-409.
- [3] Kotla, S.K., Nathaniel, C. Recurrent Stress-Induced Cardiomyopathy: A Case Report and Review Article. *Case Rep Med*. 2011;2011:160802.
- [4] Jain S., Souqiyeh A., David S., Takotsubo cardiomyopathy: reversible stress-induced cardiac insult - a stress protective mechanism. *Am J Cardiovasc Dis* 2013;3(1):53-59.
- [5] Amruthlal, Jain S.K., Larsen, T.R., Souqiyeh, A. et al. Takotsubo cardiomyopathy: reversible stress-induced cardiac insult - a stress protective mechanism. *Am J Cardiovasc Dis*. 2013;3(1):53-9.
- [6] Hasdemir, C., Vuran, O., Yuksel, A., Yavuzgil, O. Stress cardiomyopathy (Tako-Tsubo) associated with sustained polymorphic ventricular tachycardia. *Pacing Clin Electrophysiol*. 2013 Apr;36(4):111-4.
- [7] Wahab, A., Wahab, S., Panwar, R. et al. Tako-tsubo Cardiomyopathy or Broken Heart Syndrome. *Iranian Cardiovascular Research Journal*. 2010;4(1):33-34.
- [8] Sardar, M.R., Kuntz, C., Mazurek, J.A. et al. Recurrent takotsubo cardiomyopathy in the setting of transient neurological symptoms: a case report. *J Med Case Rep*. 2011 Aug 24;5:412.
- [9] Koulouris, S., Pastromas, S., Sakellariou, D., et al. Takotsubo cardiomyopathy: the "broken heart" syndrome. *Hellenic J Cardiol*. 2010 Sep-Oct;51(5):451-7.
- [10] Barcin, C., Kursaklioglu, H., Kose, S., Amasyali, B., Isik, E. Takotsubo cardiomyopathy in a patient with Addison disease: is apical ballooning always reversible? *Int J Cardiol*. 2010 Jan 7;138(1):15-7.
- [11] Shim, I.K., Kim, B.J., Kim, H., et al. A case of persistent apical ballooning complicated by apical thrombus in takotsubo cardiomyopathy of systemic lupus erythematosus patient. *J Cardiovasc Ultrasound*. 2013 Sep;21(3):137-9.
- [12] Sharkey, S.W., Windenburg, D.C., Lesser, J.R. et al. Natural History and expansive clinical profile of stress (tako-tsubo) cardiomyopathy. *J Am Coll Cardiol*. 2010 Jan 26;55(4):333-41.