

# A Case of Intracranial Hemorrhage Causing Stress-Induced Cardiomyopathy

LESLIE RUSSELL, MD; PHILIP STOCKWELL, MD

## ABSTRACT

The classic finding of Takotsubo's cardiomyopathy is left ventricular systolic dysfunction with echocardiographic evidence of apical ballooning in the absence of significant coronary disease. Intracranial hemorrhage is a known cause for stress-induced cardiomyopathy with a similar echocardiographic presentation. This diagnostic finding suggests a similar pathophysiologic mechanism between neurogenic cardiac damage and the wide array of medical and psychosocial disorders that are known to cause stress-induced cardiomyopathy (Takotsubo's syndrome). The neurogenic-cardiac variant of stress-induced cardiomyopathy is associated with good cardiovascular prognosis; the hallmark feature of the disorder is complete echocardiographic resolution of systolic dysfunction within a short period of time. While malignant presentations are rare, the disorder can present as severe heart failure or ventricular tachyarrhythmias. We report a case of a near life-threatening episode of polymorphic ventricular tachycardia due to a subarachnoid hemorrhage (SAH)-induced stress-cardiomyopathy.

**KEYWORDS:** Takotsubo's cardiomyopathy, intracranial hemorrhage, arrhythmia, echocardiogram

## CASE

A 58-year-old woman presented to the emergency department with acute-onset confusion and headache. CT scan of the brain showed diffuse intracranial hemorrhage (Figure 1). Her mental status improved until day four when she was found unresponsive. Cardiac telemetry showed polymorphic ventricular tachycardia (Figure 2), which degenerated to ventricular fibrillation. Cardiopulmonary resuscitation was performed with successful return of spontaneous circulation.

ECG obtained after the cardiac arrest showed T wave inversions in leads I, II, aVL, and V2-V6 (Figure 3). Echocardiogram



Figure 1. CT scan of brain demonstrating right intraparenchymal hemorrhage.

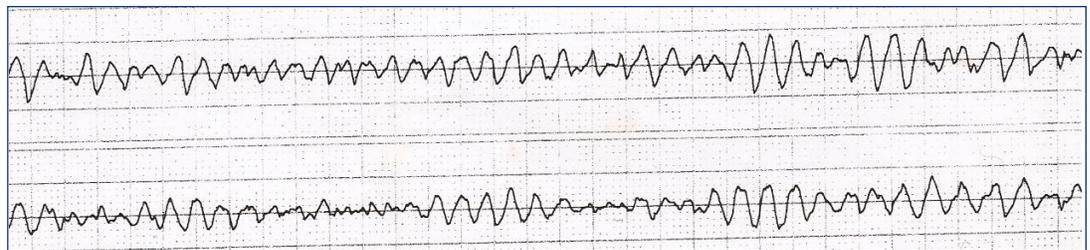


Figure 2. Cardiac telemetry strip demonstrating polymorphic ventricular tachycardia

showed a left ventricular ejection fraction of less than 30% with apical and lateral wall akinesis. Serum troponin level was 4.8 [reference range 0.00-0.15]. Due to the SAH, cardiac catheterization was not performed. The patient improved and had no further arrhythmias. Repeat echocardiogram one week after the arrest showed normal left ventricular systolic function with resolution of lateral and apical akinesis.

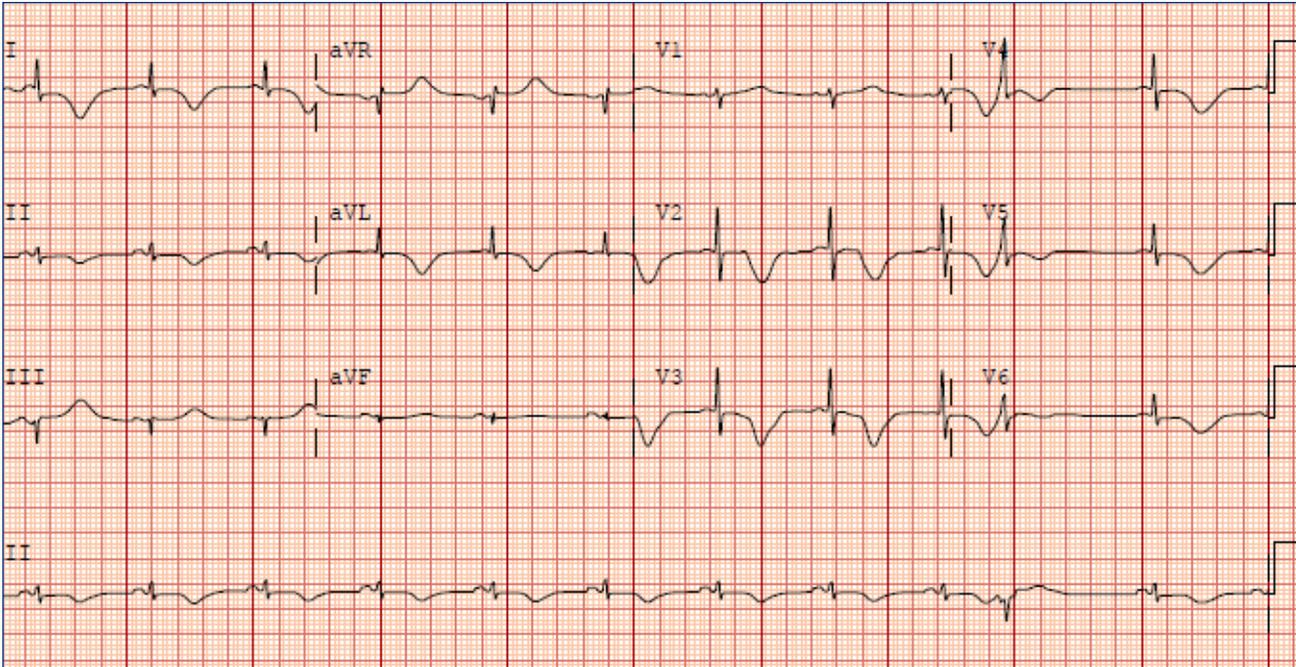


Figure 3. ECG after cardiac arrest illustrating T wave inversion in leads I, II, aVL, and V2-V6

## DISCUSSION

Stress-induced cardiomyopathy is characterized by transient left ventricular systolic dysfunction. It is classically referred to as Takotsubo's cardiomyopathy as echocardiogram findings of the left ventricle demonstrate apical ballooning which resembles the historic Japanese octopus catcher or "tako-tsubo." It is known that intracranial hemorrhage can lead to a variant of stress-induced cardiomyopathy. The similarities between the traditional stress-induced cardiomyopathy and the neurologic related Takotsubo-like variant suggest these two disorders are on a spectrum of a single disease. The hallmark feature of both disorders is complete systolic recovery on echocardiogram within a short period of time.

Takotsubo's cardiomyopathy results from severe physiologic or psychological stress. Presenting features include chest pain, elevation in serum troponin levels, and ECG changes suggestive of ischemic heart disease. The neurogenic variant of Takotsubo-like cardiomyopathy is associated with specific physical stressors such as subarachnoid hemorrhage. Mild troponin elevation is seen in 20-30% of patients.<sup>1</sup> Echocardiogram in both entities usually demonstrates left ventricular apical ballooning with akinesis and basal hyperkinesis, though numerous variants have been documented.<sup>2</sup> Cardiac catheterization typically reveals no significant coronary obstruction in the distribution of these wall motion abnormalities.

While there are numerous etiologies to stress-induced cardiomyopathy, the pathophysiologic mechanism remains elusive. Studies suggest catecholamine excess as the most likely cause of the disorder, but other hypotheses include

microvascular damage and coronary vasospasm.<sup>3-5</sup> Intracranial hemorrhage has been shown to cause catecholamine surge that persists for 7 to 10 days.<sup>6</sup> As it is known that catecholamines result in cardiac toxicity, it is believed that this surge contributes to the left ventricular dysfunction.<sup>2</sup> Evidence implicating catecholamine excess comes from animal models, in which rats with induced subarachnoid hemorrhage were noted to have resilience against development of Takotsubo's-like ventricular dysfunction after undergoing pharmaceutical or surgical sympathectomy.<sup>7</sup> Furthermore, another study illustrated that rats subject to immobilization stress had attenuation of subsequent development of left ventricular apical ballooning if receiving adrenoceptor blockade.<sup>4,5,8</sup>

Greater than 75% of stress-induced cardiomyopathy occurs in postmenopausal women.<sup>4</sup> This suggests estrogen depletion has a contributing role.<sup>3</sup> It has been shown that female rats that had undergone ovariectomy were less likely to have stress-related left ventricular dysfunction if receiving supplemental estrogen.<sup>2,3,5</sup> Though more research needs to be done, these findings suggest a possible role for estrogen in primary prevention.

The treatment of stress-induced cardiomyopathy is supportive. Complete myocardial recovery is typically seen within several weeks. Complications are rare, but include left ventricular wall rupture, atrial and ventricular arrhythmias, and apical thrombus formation.<sup>2</sup> In the case documented above, the patient experienced ventricular tachyarrhythmia. The arrhythmia likely occurred from severely reduced left ventricular function resulting in increased arrhythmogenic potential. In one retrospective study it was noted that close

to 1% of patients with stressed induced cardiomyopathy suffered from a ventricular arrhythmia.<sup>9</sup> As these arrhythmias can be fatal, more research needs to be done to ascertain which patients are at higher risk for these events. By determining the exact mechanism of the cardiomyopathy, treatment can be established to prevent or attenuate the left-ventricular dysfunction and thus circumvent complications.

## References

1. Chen M. Transient stress cardiomyopathies in the elderly: clinical & pathophysiologic considerations. *The Journal of Geriatric Cardiology*. 2012 March; 9(1):38-48.
2. Ako, J, Sudhir K, et al. Transient left ventricular dysfunction under severe stress: brain-heart relationship revisited. *The American Journal of Medicine*. 2006;119(1):10-17.
3. Lee VH, Oh JK, et al. Mechanisms in neurogenic stress cardiomyopathy after aneurysmal subarachnoid hemorrhage. *The Journal of Neurocritical Care*. 2006;5(3):243-249.
4. Ueyama T, Kasamatsu K, Hano T, et al. Emotional stress induces transient left ventricular hypocontraction in the rat via activation of cardiac adrenoceptors: a possible animal model of "tako-tsubo" cardiomyopathy. *Circulation Journal*. 2002;66(7):712-713.
5. Ueyama T, Hano T, Kasamatsu K, Yamamoto K, et al. Estrogen attenuates the emotional stress-induced cardiac responses in the animal model of Tako-tsubo (Ampulla) cardiomyopathy. *J Cardiovasc Pharmacology*. 2003;42(1):117-119.
6. Naredi S, Lambert G, Eden E, Zall S, Runnerstam M, Rydenhag B, Friberg P. Increased sympathetic nervous activity in patients with nontraumatic subarachnoid hemorrhage. *Stroke*. 2000;31(4): 901-906.
7. Shao Y, Redfors B, et al. Novel rat model reveals important roles of B-adrenoreceptors in stress-linked cardiomyopathy. *The International Journal of Cardiology*. 2013;0167-5273 1874-1754.
8. Y-Hassan S. Insights into the pathogenesis of Takotsubo syndrome, which with persuasive reasons should be regarded as an acute cardiac sympathetic disease entity. *ISRN Cardiol*. 2012; 2012:593735.
9. Tsuchihashi K, Ueshima K, et al. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. *The Journal of the American College of Cardiology*. 2001;38(1):11-18.
10. Naidech AM, Kreiter KT, et al. Cardiac troponin elevation, cardiovascular morbidity, and outcome after subarachnoid hemorrhage. *Circulation*. 2005;112(18):2851-2856.
11. Trio O, de Gregorio C, et al. Myocardial dysfunction after subarachnoid haemorrhage and tako-tsubo cardiomyopathy: a differential diagnosis? *Therapeutic Advances in Cardiovascular Disease*. 2010;4(2):105-107.

## Authors

Leslie Russell, MD, is an Internal medicine resident at The Warren Alpert Medical School at Brown University.

Philip Stockwell, MD, is a Cardiologist affiliated with Rhode Island Hospital and is an assistant professor of medicine (clinical) at The Warren Alpert Medical School at Brown University.

## Correspondence

LRUSSELL2@lifespan.org