

A 23-Year-Old Woman with Variant Neurogenic Stunned Myocardium

Elizabeth Mahanna, MD; Azra Bihorac, MD, MS, FASN

Department of Anesthesiology, University of Florida College of Medicine, Gainesville, FL

Introduction

Transient left ventricular dysfunction occurring following subarachnoid hemorrhage (SAH) is a well-documented phenomenon. It is a form of stress cardiomyopathy and has been called numerous terms over the years, including Tako-Tsubo cardiomyopathy, apical ballooning syndrome, neurogenic stressed cardiomyopathy and neurogenic stunned myocardium (NSM). Traditionally, it is characterized by transient, dyskinesis of the left ventricular (LV) apex in a distribution of myocardium covered by more than one coronary artery. A variant or inverse form in which the mid or basal LV segments are dysfunctional and apical LV segment contraction is preserved was first described in 2005 by Ennezat et al. and has since been increasingly reported.

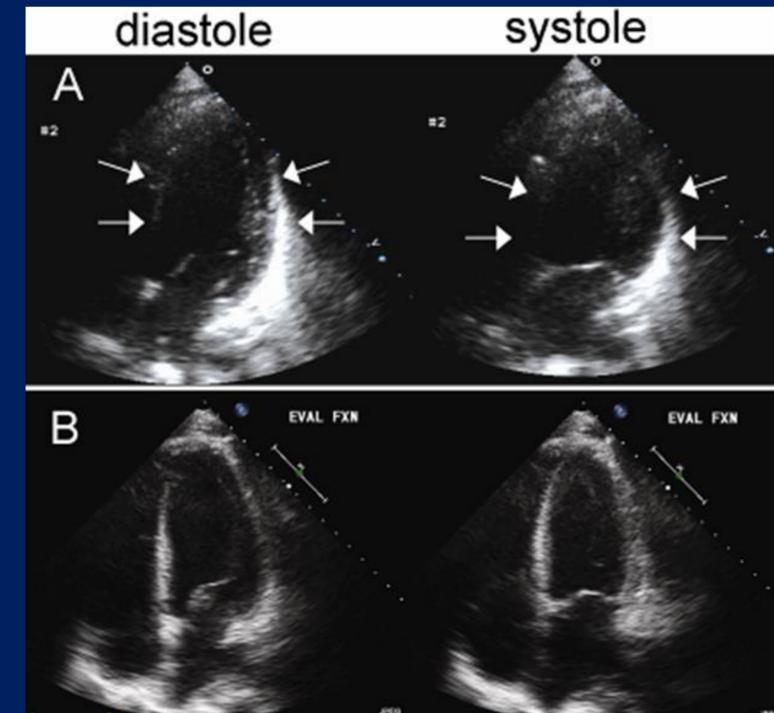
Case Presentation

A 23-year-old woman presented with sudden onset of severe headache, left facial droop, and left extremity weakness. On admission, her cardiovascular examination, electrocardiogram (ECG), and chest x-ray were all within normal limits. A CT of the head showed SAH, right temporoparietal hemorrhage, and subfalcine herniation. A subsequent cerebral angiogram showed a complex AVM along the right lateral ventricle fed by the right anterior and posterior choroidal arteries, with an associated anterior choroidal aneurysm.

The patient underwent angiogram with onyx embolization of the aneurysm, which was complicated by occlusion of the distal right internal carotid artery, causing ischemia of the proximal middle cerebral artery distribution. Over the following 24 h, she developed further hemorrhage and herniation and underwent a decompressive craniectomy and evacuation of the hematoma. She went into shock, requiring large volume resuscitation, and vasopressor and inotrope support. She quickly developed pulmonary edema, necessitating tracheal intubation. Invasive hemodynamic monitoring demonstrated depressed cardiac output. Her cardiac biomarkers were mildly elevated (troponin-T 0.57, creatinine kinase 43, CK-MB 17.9, and CK-MB index of 3.7). A twelve-lead ECG showed sinus tachycardia with no ST-T wave changes. Echocardiogram showed moderate LV dysfunction with severe circumferential basal hypokinesis and hyperkinetic apical segments. The ejection fraction (EF) was 25% to 30%.

The patient was diagnosed with variant NSM and supportive therapy was initiated. Over the subsequent 9 days, the patient's cardiac status gradually improved and she was weaned off inotropic support. Six months later, she returned for cranioplasty with no residual cardiopulmonary abnormalities and only mild left-sided weakness. At 1 year follow-up, her echocardiogram showed normal left and right ventricular systolic function.

Echocardiogram of the patient's inverted neurogenic stunned myocardium



A. An apical view of the left ventricle shows basal ballooning during systole in the acute period.

B. An apical view 13 days later shows recovery with uniform contraction of the left ventricle.

References

1. Lee VH, et al. J Neurosurg. 2006;105:264-70.
2. Shoukat S, et al. Neurocrit Care. 2013;18:257-60.
3. Fujita K, et al. Surg. Neurol. 2007;68:556-61.

Discussion

NSM is an important complication of SAH with wall motion abnormalities associated with worse outcome. There are 66 cases of SAH-induced NSM reported. Like classic SCM, SAH-induced forms predominate in women (85%) with a mean age of 57 years. Among these, all but six showed changes on ECG and mildly elevated cardiac biomarkers.

Even among the small number of reported cases, our patient is an outlier. Her heart failure occurred after embolization when she developed re-bleeding and elevated ICP. She represents one of four cases exhibiting no changes on ECG; her diagnosis was made after developing pulmonary edema. NSM was unexpected given her young age. Our patient is the only reported case of AVM-associated NSM and the only woman in her 20s where SAH-induced NSM was present.

Conclusion

In conclusion, to aid in better recognition of NSM in the neurosurgical ICU, we present a unique case of a young woman with variant NSM precipitated by SAH from rupture of a complex AVM and associated aneurysm. Although rare, this case represents the variability in neurogenic stunned myocardium.