# A Ruptured Basilar Aneurysm Resulting in Takotsubo Cardiomyopathy.

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# A Ruptured Basilar Aneurysm Resulting in Takotsubo Cardiomyopathy

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## INTRODUCTION

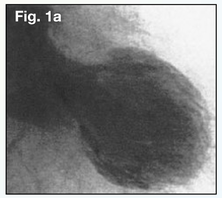
Subarachnoid hemorrhage (SAH) can often be a devastating clinical diagnosis. The most common cause is a ruptured saccular cerebral aneurysm, with a reported 30-day fatality as high as 40% and permanent disability in 50% of the survivors [1]. This high morbidity and mortality can be partly explained by the high catecholamine surge resulting from SAH and its systemic effects, particularly to cardiac function [2]. The heart's ability to maintain adequate hemodynamics and volume status is imperative for patient recovery. Reversible cardiac dysfunction is a well-known complication of subarachnoid hemorrhage. Arrhythmias, myocardial infarction, and cardiac arrest are well known complications in patients with SAH and can result in death and delayed cerebral ischemia [3-5]. Rarely, SAH can also be associated with Takotsubo cardiomyopathy, which has a prevalence of 0.8% among all patients with aneurysmal SAH [6].

The development of Takotsubo cardiomyopathy (TCM), a transient dysfunction of the left ventricle which often mimics myocardial infarction, is increasingly recognized as a complication of SAH. TCM was first reported in Japan, when the peculiar ventricular morphology was noted to be quite similar in shape to the takotsubo, a vase with a rounded bottom and narrow neck used by Japanese fishermen as an octopus trap (Fig. 1). Patients with the syndrome show evidence of exacerbated sympathetic activation. TCM, while generally considered a self-limiting process, is important to recognize because it is associated with increased mortality rates resulting from the development of left ventricular thrombus, congestive heart failure (CHF), and arrhythmias [2]. It is a complication that is sometimes described in case reports and small case series. It is generally agreed upon that conservative medical management is the mainstay of therapy for TCM, however no clinical trials to date exist to help guide treatment. This case report serves to introduce an example of SAH-induced TCM in order to raise awareness of this cardiac complication and the need for optimum clinical management.

## CASE

A 75-year-old female with hyperlipidemia and no known coronary artery disease presented to the Emergency Department (ED) after suddenly grasping her head and collapsing onto the floor unconscious.

On physical exam, she was afebrile, hypertensive with a blood pressure of 162/96 mmHg, pulse of 114 bpm and a respiration rate of 24. Cardiopulmonary exam was unremarkable. Neurologically she was unresponsive with Glascow Coma Score of 8, Hunt and Hess grade 4, Fisher 4, and intubated for airway protection. Computer Tomography (CT) of her head revealed diffuse subarachnoid hemorrhage with intraventricular hemorrhage (Fig. 2). CT Angiography (CTA) demonstrated a 6.5 mm basilar tip aneurysm, and the patient underwent endovascular coil embolization (Fig. 3). After an initial screening troponin I of <0.02 ng/mL obtained during ED assessment, a second troponin I elevated to 1.43 ng/mL at the eleven hour mark and later down-trended to 0.90 ng/mL. An echocardiogram showed mild to moderate reduction in left ventricular systolic function with severe hypokinesis of the apical segments. ( Fig. 4). Cardiology consultation was obtained. Given echocardiographic findings and clinical scenario, there was a suspicion for stress-induced (Takotsubo) cardiomyopathy. A recommendation was made to wean off the norepinephrine as this was likely exacerbating the catecholamine effect onto the heart. Repeat echocardiogram was performed 15 days later which showed complete resolution of prior left ventricular dysfunction and apical hypokinesis (Fig. 5). As there was a clear improvement in cardiac function, an ischemic workup with a left heart catheterization was not warranted. Neurologically, she made gradual improvements throughout her hospital stay. By the time of discharge to inpatient rehabilitation, she was alert, with improving speech, following commands, and moving all 4 limbs against gravity well.



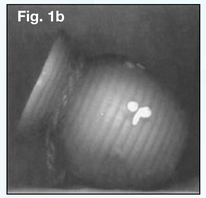




**Figure 1a:** Ventriculography demonstrating takotsubo shape
**Figure 1b:** A Japanese takotsubo jar, for which this syndrome derives its name.

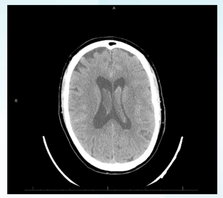


**Figure 2:** Intraventricular hemorrhage appreciated within the lateral ventricles bilaterally. Patient also had evidence of blood within the third and fourth ventricles.

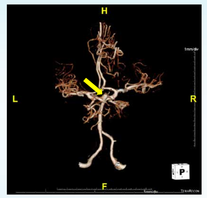



**Figure 3:** CTA with white arrow indicating 6.5 mm basilar tip aneurysm

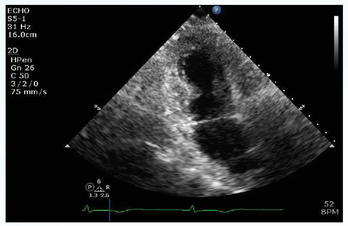



**Figure 4:** Transthoracic echocardiogram two-chamber apical (mid-systole) demonstrating hypokinesis and "ballooning" of the left ventricular apex characteristic of takotstubo cardiomyopathy.

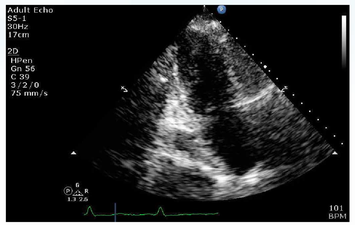



**Figure 5:** Two-chamber apical view taken mid-systole demonstrating resolution of prior hypokinesis.

## DISCUSSION

The prevalence of TCM is estimated to be 0.8% among all patients of aneurysmal SAH [6]. Many other TCM prevalence studies and meta-analyses in SAH patients do not distinguish between aneurysmal and non-aneurysmal causes. In a meta-analysis of 10 studies, 3581 patients were admitted with SAH, of which 157 patients were found to have developed TCM symptoms, representing 4.4% of the total patient population [9]. In another meta-analysis of over 53 papers, the incidence of TCM varied drastically from 0.8% to 33% [10].

Patients who develop SAH-induced TCM are shown to be at increased risk of worse outcomes. Patients with TCM were more likely to have moderate to severe subarachnoid bleeds (such as our patient) as measured by the Hunt and Hess classification [11]. There are significant increases of in-hospital mortality in patients that develop TCM [2, 9]. Patients with SAH-induced TCM are more likely to have low Glasgow coma scale scores [10]. SAH alone carries a significantly high mortality. Although TCM carries a favorable prognosis in the general population, this pattern of cardiac dysfunction in patients with SAH may be associated with pulmonary edema, prolonged intubation, and cerebral vasospasm, potentially further escalating risk of mortality [7]. The pathophysiology behind the development of TCM in the setting of aneurysmal SAH still remains unclear, however high levels of catecholamine release is the most widely accepted theory. It is believe that this catecholamine surge can cause direct coronary vasospasm, which during a hypersympathetic state can lead to poor cardiac perfusion and dysfunction. There is also evidence for cathecholamines causing direct toxicity to the myocardium [2]. This superimposes additional challenges on the hemodynamic management of aneurysmal SAH patients in terms of optimal blood pressure and cerebral perfusion [7]. The use of vasopressors and inotropes remains controversial, as permissive hypertension is a key therapeutic goal in patients with SAH. While there is existing evidence that vasopressor agents can exacerbate TCM [8], more recent research suggests that vasopressors can be used safely [2].

In this case, the patient's TCM was not discovered until after the patient underwent interventional therapy. This raises the question as to whether or not early cardiac screening with serial myocardial enzyme testing, EKGs and an echocardiogram should be standard studies for all SAH patients. Current American Heart Association Guidelines for the management of aneurysmal subarachnoid hemorrhage do not recommend routinely obtaining these studies during emergency evaluation and preoperative care, nor indicate to obtain these studies post-procedure [12]. Given that TCM may contribute to mortality in this population, potentially this change in protocol should be considered. However, further research is necessary, as there is a paucity of literature detailing whether early recognition of will lead to better outcomes. More evidence is also needed to help guide clinicians with therapeutic decision making in aneurysmal SAH patients with TCM, particularly with hemodynamic management, which remains a complicated endeavor as the incidence of this condition in the overall population is low.

## CONCLUSION

Since first being described in the literature, Takotsubo cardiomyopathy has become an increasingly recognized complication of SAH. It is an important diagnosis to consider as it can mimic an acute coronary syndrome and it can lead to more complications with treatment of SAH, particularly in the sub-population of SAH patients with aneurysms. Cardiac screening may be beneficial, however no clear guidelines are present to help guide therapeutic management. While outcomes are favorable with conservative therapy alone, there are still questions to be answered, particularly of the role vasopressors play in management. Awareness of this syndrome and reporting of it in the literature only serves to the betterment of medical therapies and patient outcomes.

## References:

1. Dority, J. S., & Oldham, J. S. (2016). Subarachnoid Hemorrhage An Update. Anesthesiology Clinics, 34(3), 577-600. Retrieved April 9, 2017, from https://www.elsevier.com/.
2. Talahma M, Akkhachroum AM, Alyahya M, . Takotsubo cardiomyopathy in aneurysmal subarachnoid hemorrhage: Institutional experience and literature review. Clin Neurol Neurosurg 2016; 141: 65–70. Retrieved April 9, 2017, from https://www.elsevier.com/.
3. Ahmadian A, Mizzi A, Banasiak M, et al. Cardiac manifestations of subarachnoid hemorrhage. Heart Lung Vessel. 2013;5:168–78. Retrieved April 9, 2017, from https://www.ncbi.nlm.nih.gov/pubmed/.
4. van der Bilt IA, Hasan D, Vandertop WP, Wilde AA, Algra A, Visser FC, Rinkel GJ. Impact of cardiac complications on outcome after aneurysmal subarachnoid hemorrhage: A meta-analysis. Neurology. 2009;72:635-42. Retrieved April 9, 2017, from http://www.neurology.org/content/72/7/635.long.
5. Hravnak M, Frangiskakis JM, Crago EA, Chang Y, Tanabe M, Gorcsan J, 3rd, Horowitz MB. Elevated cardiac troponin I and relationship to persistence of electrocardiographic and echocardiographic abnormalities after aneurysmal subarachnoid hemorrhage. Stroke. 2009;40(11):3478–84. Retrieved April 9, 2017, from http://stroke.ahajournals.org/content/40/11/3478.
6. Deshmukh A, Kumar G, Pant S, Rihal C, Murugiah K, Mehta JL. Prevalence of Takotsubo cardiomyopathy in the United States. *Am Heart J*. 2012;164:66–711.
7. Bhatnagar V, Manikandan S. Takotsubo cardiomyopathy in aneurysmal subarachnoid hemorrhage. *Indian J Anesth*. 2014; 58 (2):233-235.
8. Elgendy AY, Elgendy IY, Mansoor H, Mahmoud AN. Clinical presentations and outcomes of Takotsubo syndrome in the setting of subarachnoid hemorrhage: a systematic review and meta-analysis. Eur Heart J Acute Cardiovasc Care. 2016. Retrieved April 9, 2017, from https://www.ncbi.nlm.nih.gov/pubmed/.
9. Abd TT, Hayek S, Cheng J, et al. Incidence and clinical characteristics of takotsubo cardiomyopathy post-aneurysmal subarachnoid hemorrhage. Int J Cardiol. 2014;176(3):1362–4. Retrieved April 9, 2017, from https://www.elsevier.com/.
10. J. Finsterer, A. Bersano. Subarachnoid bleeding triggering Takotsubo syndrome. Int. J. Cardiol., 197 (2015), pp. 107–109. Retrieved April 9, 2017, from https://www.elsevier.com/.
11. Skolnick AH, Michelis K, Nayar A, Fisher D, Korenzon I. Transient Apical Ballooning Syndrome Precipitated by Dobutamine Stress Testing. *Ann Intern Med*. 2009;150:501-502. doi: 10.7326/0003-4819-150-7-200904070-00015.
12. Bederson JB, Connolly ES, Batjer HH et al. American Heart Association Guidelines for the management of aneurysmal subarachnoid hemorrhage. *Stroke*. 2009: 40: 994-1025.