Takotsubo Cardiomyopathy Caused By Acute Ischemic Stroke.

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Abstract

Takotsubo cardiomyopathy, also known as stress cardiomyopathy, usually occurs secondary to extreme physical and emotional stress. Subarachnoid hemorrhage has been strongly related to stress cardiomyopathy due to catecholamine surge in such patients; however, ischemic stroke has been rarely associated with takotsubo cardiomyopathy. Whether ischemic stroke is the cause or the result of takotsubo cardiomyopathy is difficult to ascertain in some cases. We present a case of a 78-year-old female who came with stroke symptoms and developed cardiogenic shock secondary to takotsubo cardiomyopathy.

Keywords:

Takotsubu cardiomyopathy, Ischemic stroke.

Introduction:

Takotsubo cardiomyopathy involves transient left ventricular regional wall dysfunction associated with apical ballooning. The onset of cardiomyopathy is often triggered by extreme physical or emotional stress [1]. The physical trigger can be any illness like neurologic disorders (such as SAH and acute stroke), post-surgical, and acute respiratory failure. Some patients even don't have any trigger. It is much more common in older adults and in women [2]. Takotsubo cardiomyopathy has a strong association with the neurological diseases like subarachnoid hemorrhage (SAH) but it can rarely be associated with acute ischemic stroke, mostly occurs soon after the stroke onset.

Case Discussion:

A 78-year-old female with the past medical history of hypertension only was brought to hospital with abrupt onset left-sided weakness, slurred speech and left facial droop. Patient was brought to the emergency department (ED) where CT scan of head showed acute right MCA infarct (Fig. 1). On neurological examination, power was 0/5 in left upper and lower extremity. She had left sided facial droop and slurred speech. Reflexes were 1+ bilaterally and cerebellar function testing were normal. She was not deemed a t-PA candidate as her symptoms started 6 hours before hospital admission. She was given per rectal Aspirin as she failed swallowing examination. In addition to the deficit, she also complained of severe chest pain, dyspnea and diaphoresis. EKG showed acute anterolateral ST elevation MI and troponins were elevated to 6.28 ng/ml. Rest of the labs including cbc, chem 7, LFTs and TSH were normal. She developed worsening respiratory symptoms secondary to cardiogenic shock for which she was intubated in ED. Emergent cardiac catheterization was done for acute MI, which showed takotsubo CM with EF 20% and normal coronary arteries (Fig. 2). Echocardiogram showed mild to moderate diffuse hypokinesis more prominent in the distal left ventricle, again representing Takotsubo cardiomyopathy (Fig. 3). Repeat CT scan head after 24 hours later showed nonhemorrhagic, evolving, right MCA territory infarct (Fig. 4). MRI of brain demonstrated stable extension of acute infarction involving multiple zones of the right cerebral hemisphere (Fig. 5). Gradient echo images showed acute hemorrhages (deoxyhemoglobin) within the right lentiform nucleus and corona radiata (Fig 6). MRA head showed severe narrowing at the right middle cerebral artery at the sylvian bifurcation (Fig 7). She was started on Coreg 6.25 mg p.o. b.i.d. as well as Entresto 24/26 mg p.o. b.i.d for her heart failure. Her condition significantly improved and she was discharge to acute rehabilitation center

Discussion:

Takotsubo cardiomyopathy (also called Apical ballooning syndrome or stress cardiomyopathy) is a syndrome which has transient left ventricular (LV) regional wall motion abnormality and mimics acute coronary syndrome [3]. It resembles acute coronary syndrome clinically and also on electrocardiographic, biochemical and echocardiographic findings, but no obstructing lesion is seen in epicardial coronary arteries on coronary angiography. About 1 to 2 percent of patients who present with the troponin positive acute coronary syndrome (ACS) have takotsubo

cardiomyopathy [4]. However, the area of wall motion abnormality usually extends beyond the single coronary artery territory.

The clinical manifestations are also similar to that of acute coronary syndrome [5]. The most common symptoms are acute chest pain, dyspnea, and syncope [3]. Some patients develop signs and symptoms of cardiac failure, sudden cardiac arrest, mitral regurgitation, arrhythmias [1, 5], or cardiogenic shock [3]. Possible mechanisms of this cardiomyopathy include catecholamine-induced microvascular dysfunction or spasm leading to stunning of the myocardium [6].

Takotsubo cardiomyopathy can be associated with many neurological diseases like acute ischemic stroke and subarachnoid hemorrhage (SAH). The predominant area of infarct at or close to the insular cortex in most patients suggest that insular area ischemia in acute stroke is strongly associated with takotsubo cardiomyopathy. The insular cortex appears to play a major role in the cardiac autonomic control as patients having an insular infarct (especially right sided) have been reported to show decreased variability in heart rate, complex arrhythmias, and sudden death [7]. The features of stunning seen after ischemic stroke are different from that of seen after SAH. The biggest difference is the distribution of regional LV wall motion abnormality. The basal and mid-ventricular segments are usually involved after SAH-induced cardiomyopathy, while after ischemic stroke apical segment involvement is predominant [8].

The EKG abnormalities most frequently seen in leads V3 and V4 are immediate ST elevation followed by negative T waves [9]. Cardiac markers in patients with takotsubo cardiomyopathy, including troponin-T and CK-MB, are generally within normal limits or modestly elevated. While BNP levels tend to increase 10 times than the normal limit [10]. BNP is known to be a marker of left ventricular dysfunction (systolic and diastolic). Treatment options for takotsubo cardiomyopathy are largely supportive. Treatment is the same as in systolic heart failure with Beta-blocker and ACE Inhibitors as the mainline therapy. Serial cardiac imaging (echocardiograms) may be done to see the progression [2].

Our patient presented with acute ischemic stroke and was found to have cardiogenic shock secondary to stress cardiomyopathy. We believe that underlying cause of stress cardiomyopathy was ischemic stroke. Patient did not have symptoms of heart failure before, so it was less likely that stroke occurred due to embolism from intracardiac thrombus. In addition, narrowing of MCA on MRA and absent thrombus on TTE ruled out embolic cause of stroke. We believe takotsubo cardiomyopathy occurred after not before ischemic stroke. Our patient responded well to convention heart failure treatment.

Conclusion:

1. The diagnosis of takotsubo cardiomyopathy should be considered in older adults (especially females) who present with suspected acute coronary syndrome but the clinical presentations and EKG abnormalities are out of proportion to the degree of cardiac enzymes elevation. An emotional or physical trigger is often present.

2: Acute ischemic stroke is a rare cause of takotsubo cardiomyopathy. Stress cardiomyopathy should be included in differential diagnosis if stroke patients develop acute coronary syndrome symptoms after onset of stroke.

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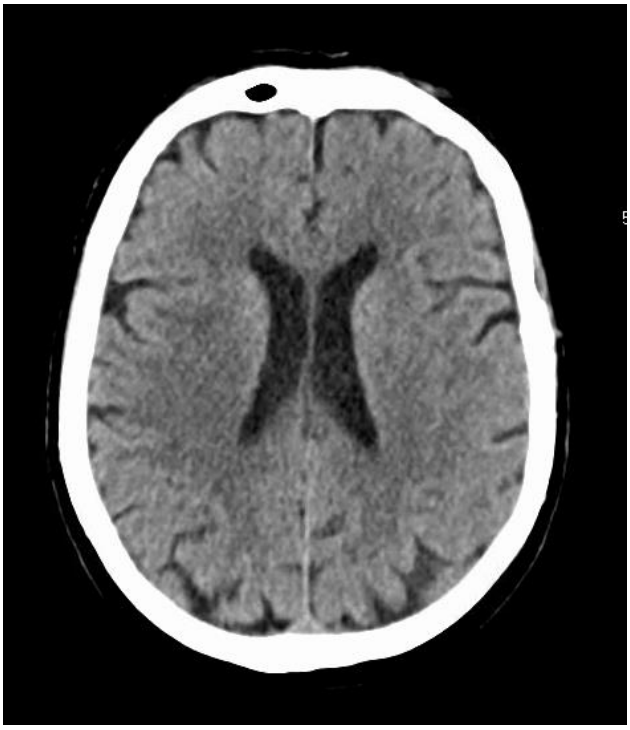

Fig 1: CT scan of head showed acute right MCA infarct.

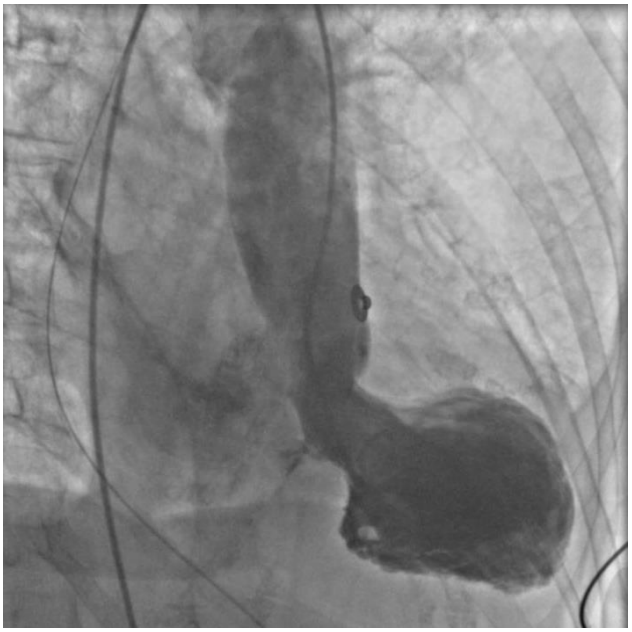

Fig 2: Cardiac Catheterization showed Takotsubo cardiomyopathy with normal coronary arteries.

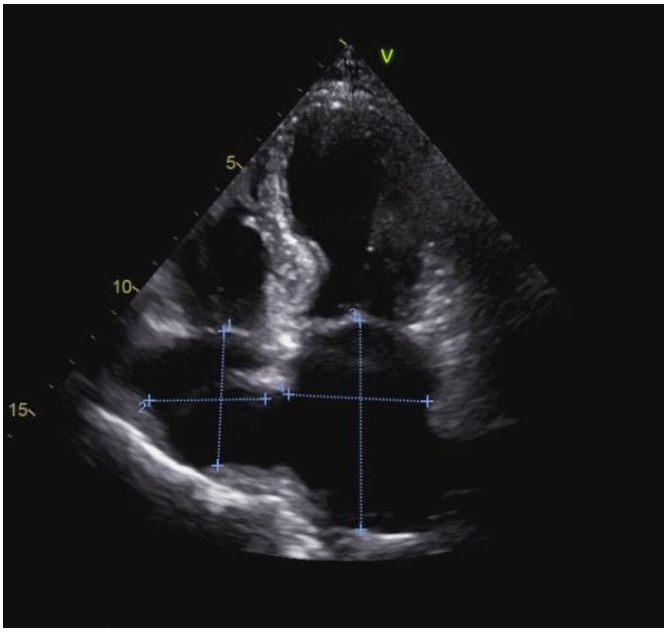

Fig 3: Echocardiogram showed mild to moderate diffuse hypokinesis more prominent in the distal left ventricle, representing Takotsubo cardiomyopathy

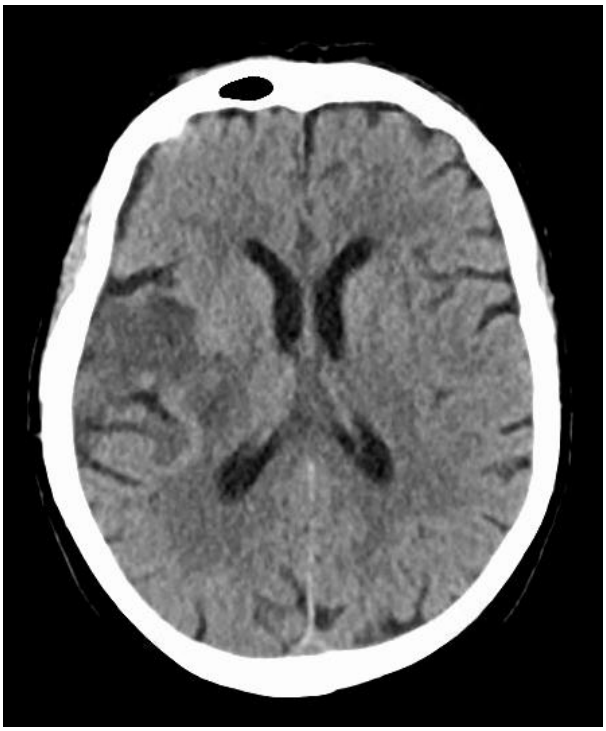

Fig 4: Repeat CT scan head after 24 hours later showed nonhemorrhagic, evolving, right MCA territory infarct.

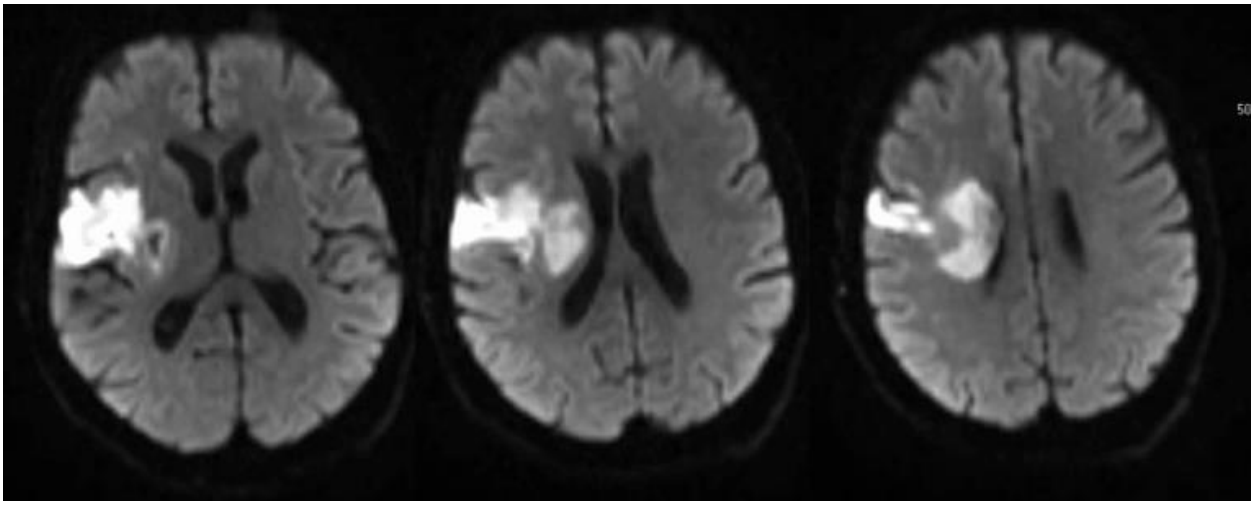

Fig 5: MRI of brain demonstrated stable extension of acute infarction involving multiple zones of the right cerebral hemisphere.

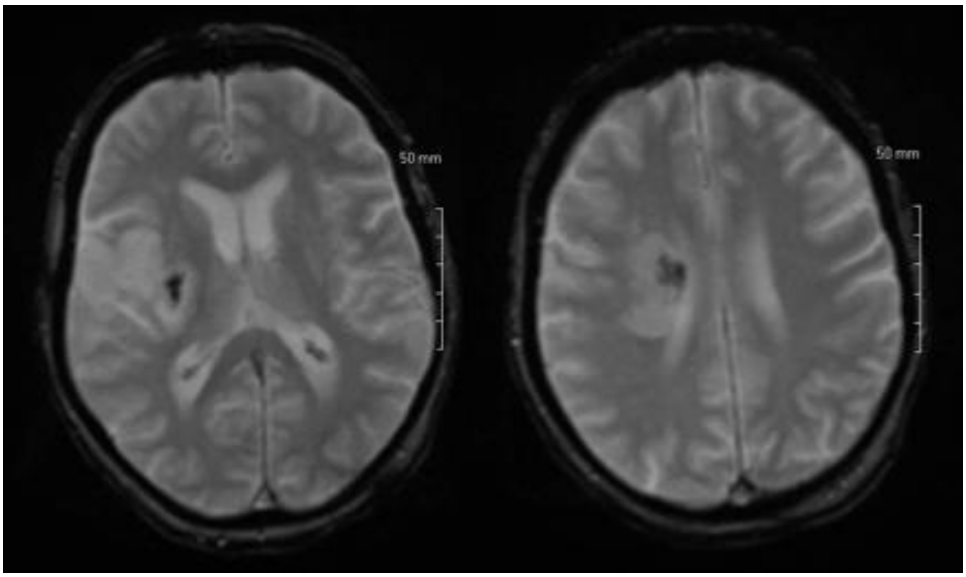

Fig 6: MRI brain (Gradient echo images) showed acute hemorrhages (deoxyhemoglobin) within the right lentiform nucleus and corona radiata.

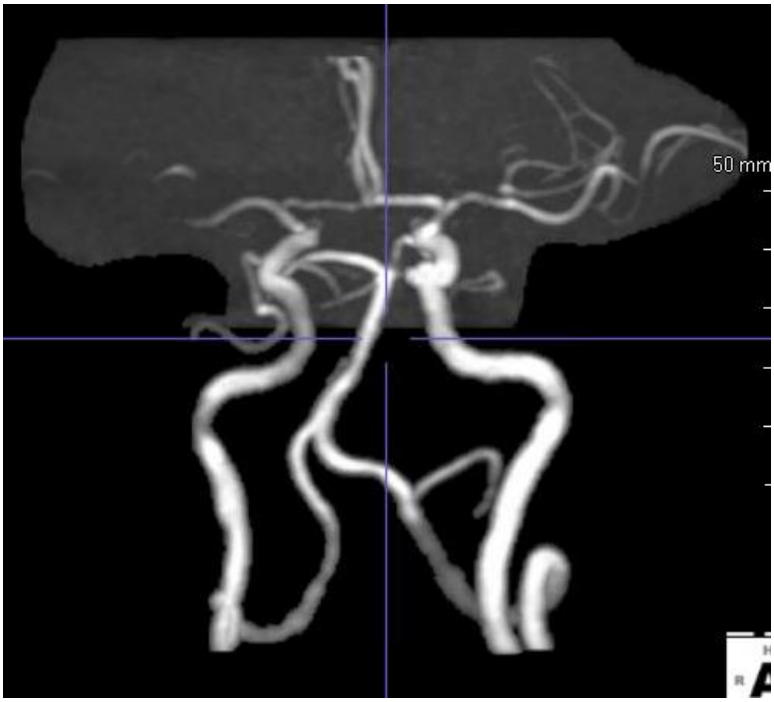

Fig 7: MRA of head showed severe narrowing at the right middle cerebral artery at the sylvian bifurcation