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SHORT COMMUNICATION

Stress cardiomyopathy: Same pathology caused by variety of triggers.

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Abstract

Stress cardiomyopathy (SCM), also known as Takatsubo cardiomyopathy or broken heart syndrome has been well documented in literature since its first description. With more awareness of the condition and increasing evidence base, the reported incidence of SCM has steadily increased over years and currently estimated at 1-2 % of the patients presenting with acute coronary syndrome.

There are different criteria proposed for diagnosis of stress cardiomyopathy and this address more or less the same clinical questions while diagnosing this not so uncommon condition.

Despite extensive research , there has been a lack of understanding on the exact mechanism and causative factors leading to SCM. We report a variety of cases where a different trigger appears to have been responsible for causation of SCM. It would seem that irrespective of the original trigger, the final clinical picture and course remains identical. These further strengths the idea that the condition is mediated by a common neurocardiovacular pathway in susceptible individuals. Further exploration of these mechanisms would be crucial to improve our strategies to try to prevent and effectively manage this potentially disabling condition.

Keywords

Stress, Cardiomyopathy, Takatsubo cardiomyopathy, acute coronary syndrome, broken heart syndrome, Stress cardiomyopathy.

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Introduction

Since its initial description in the 1990s, stress cardiomyopathy (SCM), also known as Takatsubo cardiomyopathy or broken heart syndrome, has been extensively described in literature[1]. With more awareness of the condition and increasing evidence base, the reported incidence of SCM has steadily increased over years and currently estimated at 1-2 % of the patients presenting with acute coronary syndrome[2,3].

Criteria for diagnosis of stress cardiomyopathy

Traditionally SCM has been diagnosed using Mayo criteria[4] that later on gave way to European heart failure association criteria[5] and additionally, the InterTAK Diagnostic Criteria (International Takotsubo Diagnostic Criteria) have just lately been proposed[6].

Mayo Clinic Standards

i) A stressful trigger is frequently present, but not always, and the left ventricular mid segments may briefly show hypokinesis, akinesis, or dyskinesis, with or without apical involvement.
ii) Absence of obstructive coronary disease or acute plaque rupture seen on angiography.
iii) New electrocardiographic anomalies (ST-segment elevation or T-wave inversion) or a little increase in cardiac troponin.
iv) Absence of myocarditis or pheochromocytoma.

Criteria of the European Society of Cardiology's Heart Failure Association

i) A stressful trigger is typically, but not always, present before motion abnormalities in transient regional wall of the left or right ventricle myocardium.
ii) The regional wall motion anomalies frequently result in circumferential dysfunction of the affected ventricular segments and typically go beyond a single epicardial vascular distribution.
iii) Absence of any clinical abnormalities or culprit atherosclerotic coronary artery disease to account for the pattern of transient LV dysfunction seen, including acute plaque rupture, thrombus development, and coronary dissection (e.g., hypertrophic cardiomyopathy, viral myocarditis).
iv) ST-segment elevation and depression, LBBB, T-wave inversion, and/or QTc prolongation during the acute phase (3 months) are new and reversible electrocardiography abnormalities.
v) BNP or NT-proBNP levels in the serum were noticeably raised throughout the acute phase.
vi) Positive but only mildly elevated cardiac troponin as determined by traditional testing (i.e., disparity between the troponin level and the amount of dysfunctional myocardium present).
vii) Improvement in ventricular systolic function on follow-up cardiac imaging (3 to 6 months).

International Takotsubo Diagnostic Standards

i) Apical ballooning or aberrant midventricular, basal, or localized wall motion are symptoms of patients who have temporary left ventricular dysfunction (akinesia, hypokinesia, or dyskinesia). There may be right ventricular involvement. Transitions between any type may occur in addition to these regional wall motion patterns. It is uncommon for the regional wall motion anomaly to only affect the subtended myocardial territory of a single coronary artery; instead, it typically affects more than one epicardial vascular distribution (focal Takotsubo syndrome).
ii) Takotsubo syndrome events can be preceded by an emotional, physical, or combination of triggers, but this is not required.
iii) Takotsubo syndrome may be brought on by pheochromocytoma as well as neurologic conditions (such as Seizures, transient ischemic attack/stroke, or subarachnoid hemorrhage).
iv) ST-segment elevation and depression, ATc prolongation, and T-wave inversion are the most recent ECG abnormalities to appear; nonetheless, rare cases exist without any ECG changes.
v) In majority of cases cardiac biomarkers (such as troponin and creatine kinase) are only modestly raised; nevertheless, large elevations of brain natriuretic peptide are frequent.

vi) Takotsubo syndrome does not contradict severe coronary artery disease.
vii) There is no evidence of infectious myocarditis in the patients.
viii) Most affected are postmenopausal women.

Aetiology for Stress Cardiomyopathy

Stress-related physical or mental situations might result in stress cardiomyopathy. The most frequent emotional stresses mentioned are loss of a loved one, financial loss, natural disaster, and violence, with the majority involving a sense of impending doom, peril, or desperation[7,8]. However, episodes of stress cardiomyopathy can also occur after positive surprises, or "happy heart syndrome"[9].

Acute critical illness, surgery, excruciating pain, sepsis, and flare-ups of asthma or chronic obstructive pulmonary disease are only a few of the documented physical stressors. Central nervous system issues have been linked to seizures, ischemic or hemorrhagic stroke, encephalitis/meningitis, head trauma, posterior reversible encephalopathy syndrome, and severe acute lateral sclerosis. Although these illnesses have been given the name "Takotsubo phenocopies" to distinguish them from "classic" stress cardiomyopathy and conditions of neurogenic stunning myocardium, they all have a pathophysiology characterized by high CANS activation[7,10,11–13].

Clearly, not every individual has the cardiomyopathy after a stressful incident, and in 30% to 35% of the cohort series, stress cardiomyopathy episodes without any discernible stressful trigger have been reported[14,15]. This outcome could be partially explained by the protective (or unfavorable) effects of psychosocial factors, such as coping mechanisms, social networks, and coexisting mental health conditions at the time of the occurrence.

We report a series of cases all associated with relatively uncommon trigger factors for the disorder.

1) Classical CM:

A 68 year old female presented with chest pain after argument at home. ECG consistent with acute anterior STEMI. An urgent coronary angiography revealed healthy, unblocked coronary arteries (Figure 1, 2). Left ventriculogram showed typical Takatsubo cardiomyopathy (Figure 3) Echocardiogram showed severe LVSD with apical akinesia. She went on to have cardiac MRI scan that was consistent with stress CM showing typical systolic apical ballooning. (Figure 4).

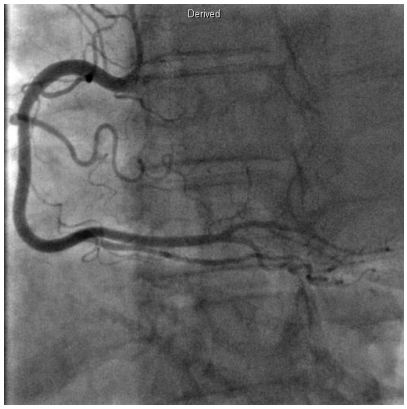

Figure 1: Coronary angiogram showing unobstructed RCA

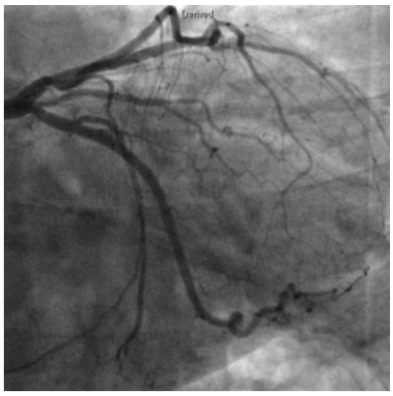

Figure 2: Coronary angiogram showing unobstructed Left coronary system

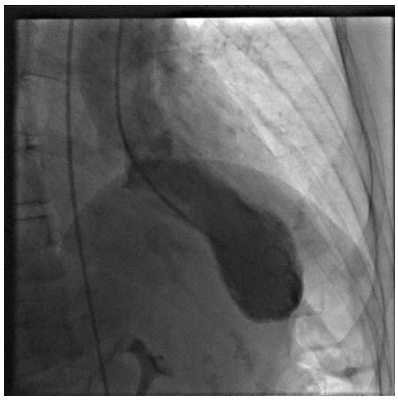

Figure 3: Left ventriculogram

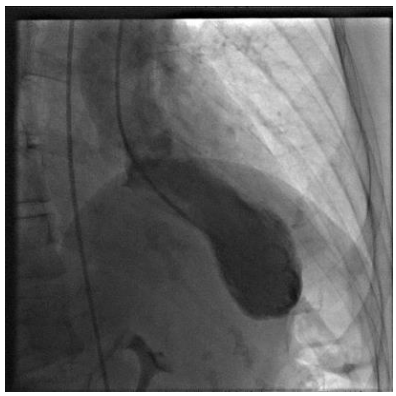

Figure 4: Ventriculogram

2) **CM associated with anaphylactoid reaction:** A 25-year-old lady who had been treated with Flucloxicillin for infection at the site of flu vaccine. After 5 doses became acutely unwell with difficulty breathing and swallowing. Presented to ED where treated with IM adrenaline. She became acutely unwell; IV adrenaline was administered in error at high doses. She went into circulatory shock and had to be transferred to ITU. CMR was consistent with reverse (basal) Takatsubo cardiomyopathy (Figure 5, 6).

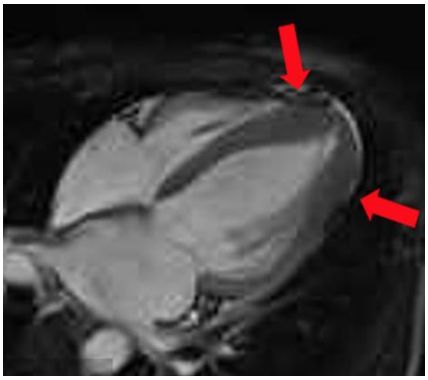

Figure 5: Cardiac MRI scan showing reverse Takatsubo cardiomyopathy

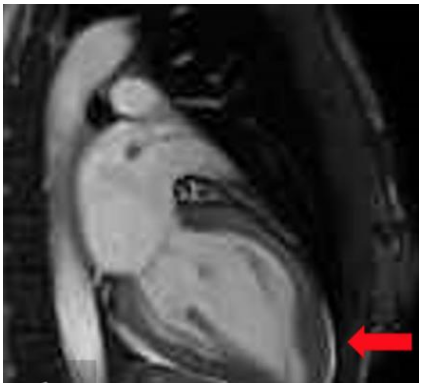

Figure 6: Cardiac MRI scan

3) CM secondary to ACE i:

60 years old female who was recently started on ACE I for hypertension, presented with chest pain and ECG suggestive of anterolateral STEMI, TNI 921. Coronary Angio showed normal coronary (Figure 7, 8) arteries. Left ventriculogram showed typical Takatsubo cardiomyopathy (Figure 9).

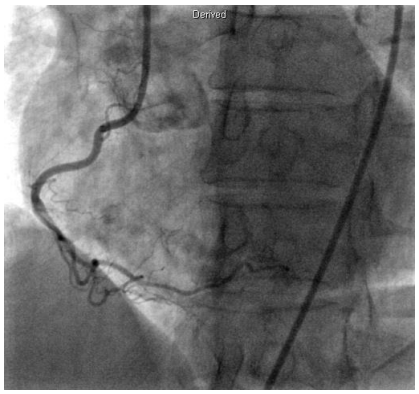

Figure 7: Coronary angiogram showing unobstructed RCA

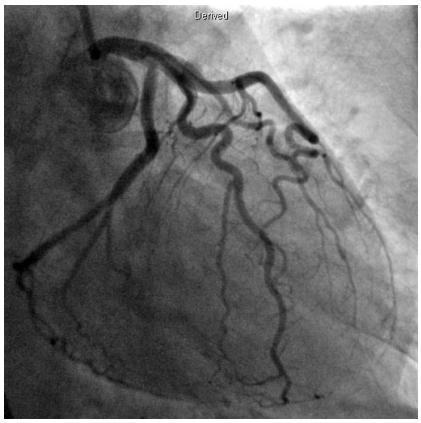

Figure 8: Coronary angiogram showing unobstructed Left coronary system

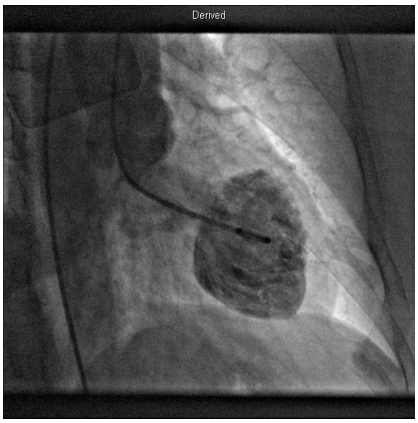

Figure 9: Left ventriculogram confirming typical apical balooning

4) CM associated with SAH:

74 years old lady presented through Primary PCI pathway. She had earlier developed chest pain with associated headache that was attributed to use of GTN. Her ECG was suggestive of anterior STEMI. Coronary angiogram showed normal unobstructed coronary arteries. (Figure 10 &11). LV gram showed regional stress cardiomyopathy affecting the anterior wall (Figure 12). An urgent CT head showed extensive subarachnoid haemorrhage (Figure 13).

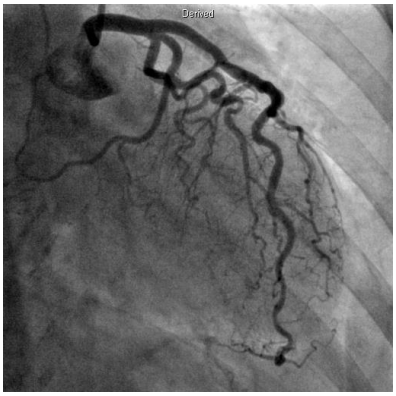

Figure 10: Coronary angiogram showing unobstructed Left coronary system

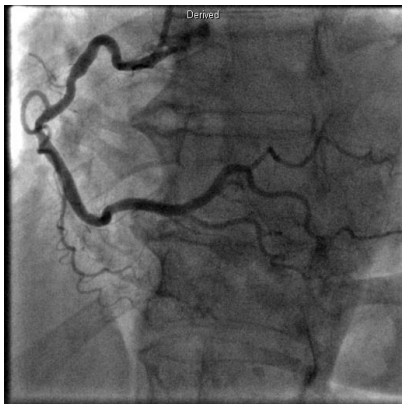

Figure 11: Coronary angiogram showing unobstructed Right coronary artery

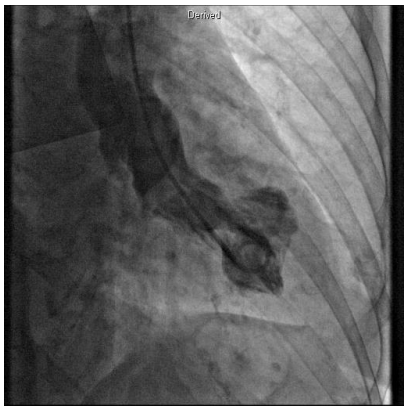

Figure 12: Left ventriculogram, showing apical cardiomyopathy

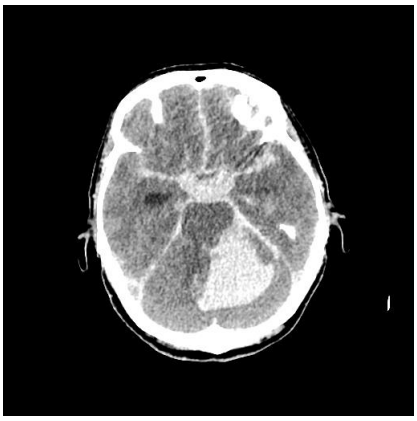

Figure 13: CT Brain showing large intracranial haemorrhage

5) **CM associated with ACS:** 78 years old lady with NSTEMI, LV gram showed stress cardiomyopathy (Figure 14), but severe proximal LAD lesion (Figure 16) confirmed on OCT that showed ruptured plaque (Figure 17). OCT guided PCI to LAD was performed. This was a case of stress cardiomyopathy induced by stress of acute myocardial infarction.

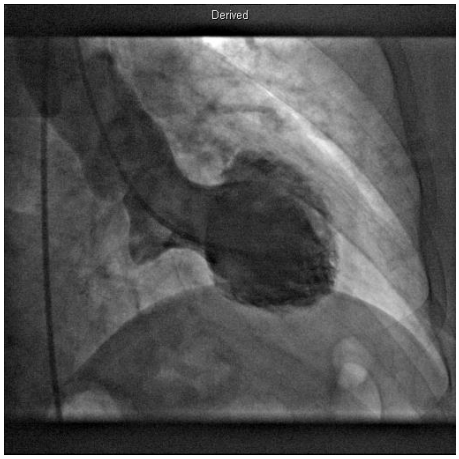

Figure 14: Left ventriculogram showing apical cardiomyopathy

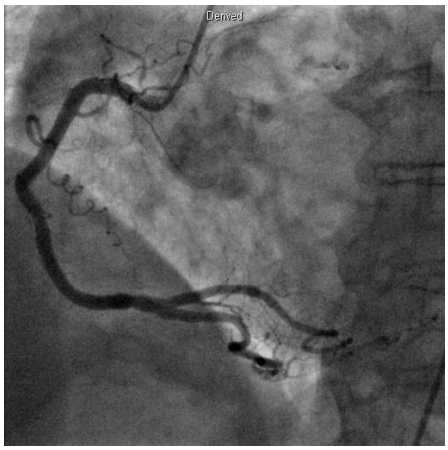

Figure 15: Coronary angiogram showing unobstructed Right coronary artery

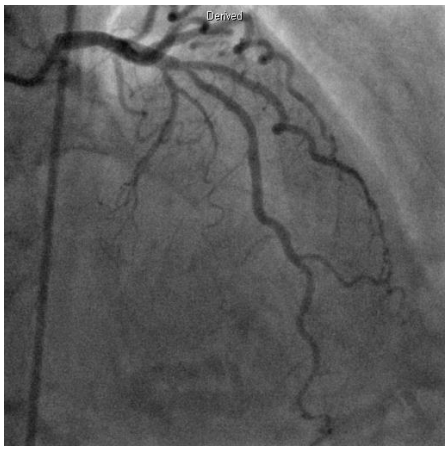

Figure 16: Coronary angiogram showing Severe proximal lesion

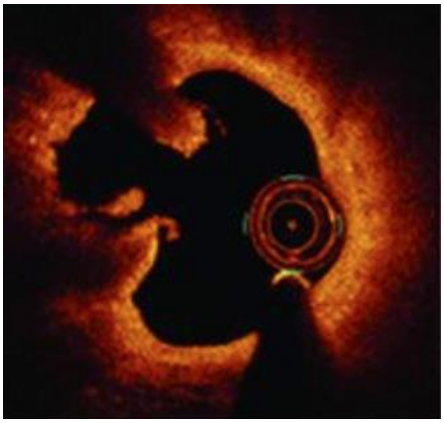

Figure 17: OCT of LAD

Discussion

What causes the characteristic changes of stress cardiomyopathy still remains to be fully explored. Using neuroimaging techniques[16,17], researchers have been able to substantiate the long-standing hypothesis of causative connections between heart and brain[15-19]. The fact that psychological and/or physical trauma causes myocardial dysfunction in one subset of people while others appear to be unaffected by them also remains areas of uncertainty.

In our reported series of cases, we have shown a wide variety of trigger factors leading to characteristic features of Takatsubo cardiomyopathy. This on one hand does highlight the vast spectrum of clinical settings that this condition can present while on the other hand it provides more evidence to the hypothesis that Taktsubo cardiomyopathy most likely is mediated by a final common neuro-cardio-vascular pathway in a group of susceptible individuals[20]. Additionally, as this series shows, stress cardiomyopathy appears to be much more common than previously thought and should be considered in the differential diagnosis in the setting of acute patient presentations with clinical signs and symptoms.

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