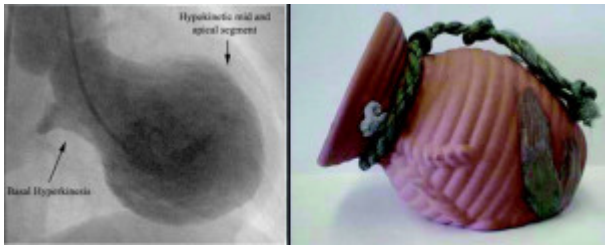
Definition:

Stress cardiomyopathy is a condition in which intense emotional or physical stress can cause rapid and severe heart muscle weakness.[1] It mimics myocardial infarction with changes in the electrocardiogram and echocardiogram, but without any obstructive coronary artery disease.[3] Stress CMP and **Tako-tsubo CMP** are used synonymously but **Takutsubo** is the most common and typical form of this disorder. A midventricular type, basal type, focal type and global type have also been described.[2]



History:

The pattern of left ventricular dysfunction was first described in Japan in 1991and has been referred to as "**tako-tsubo cardiomyopathy**,"named after the fishing pot with a narrow neck and wide base that is used to trap octopus. "**Tako-tsubo cardiomyopathy**", also known as "apical ballooning syndrome", "ampulla cardiomyopathy", "stress cardiomyopathy" or "broken-heart syndrome" is now increasingly recognised in other countries as well.[3] "Transient left ventricular apical ballooning" has also been used to describe similar cardiac contractile function in patients after physical or emotional stress.

Stress Cardiomyopathy occurs in approximately 1-2% of patients presenting with troponin-positive suspected ACS or Suspected STEMI. A prevalence of 1.2% was reported from a registry of 3265 patients with troponin-positive ACS. Similarly, Stress CMP accounted for 1.7-2.2% of cases presenting with suspected ACS or STEMI in a systematic review.[2]

Etiology: The exact cause of this condition is unclear. But it is often preceded by an intense physical or emotional event in 85% of cases. Some potential triggers are- unexpected death of a loved one, a frightening medical diagnosis, sudden financial loss, strong arguments, job loss anddivorce. Physical stressors include acute asthma, surgery, chemotherapy and stroke.[4]

It's also possible that some drugs, rarely, may cause broken heart syndrome by causing a

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surge of stress hormones. Drugs that may contribute to **broken heart syndrome** include:Epinephrine, Duloxetine,Venlafaxine,Levothyroxine.[4]

Pathophysiology: The exact pathogenesis of Stress Cardiomyopathy is unclear but there are few theories that have been suggested:

1. Catecholamine Induced-In some studies it was found that serum catecholamine concentration was 2-3 times higher in SCMP than in MI. It was also seen that exogenously administered catecholamines and pheochromocytoma produce similar picture. Catecholamine triggers α1-mediated coronary vasospasm and β1-mediated hyperdynamic basal contraction, as basal contraction has higher density of sympathetic nerve endings and higher content of norepinephrine.
2. Microvascular Dysfunction- the characteristic findings of microvascular dysfunction found were endothelium-dependent vasodilatation, excessive vasoconstriction and impairment of myocardial perfusion. Afonso et al demonstrated that circulatory disturbance, indicating coronary microvascular dysfunction was found on a myocardial contrast echocardiography and epicardial coronary arteries were normal.
3. Cytokine Induced-Frencesco et al.[5] found higher levels of circulating cytokines, viz.IL-2, IL-4,IL-10, TNFα, IFNγ and EGF on admission whereas IL-2 and EGF were higher even at 120hrs. Ahmad Abdin presented a case of Takotsubo Cardiomyopathy in a patient of Hemophagocytic Lymphohistiocytosis.[6]
4. Dynamic mid-cavity or LV outflow tract obstruction due to any cause

Clinical Features: Stress CMP occurs most commonly in females(90%).Patients typically present with chest pain, shortness of breath, sweating, dizziness, nausea, vomiting, palpitation, etc.

Investigations and findings:

1. ECG: ST-segment elevation seen in half of all cases. St segment depression, QT prolongation, T-wave inversion, abnormal Q-waves are also seen but rarely.
2. Troponin- Serum troponin are raised in most of the cases(median initial troponin 7.7 times the upper limit of normal).
3. BNP and NT proBNP- are elevated in most patients. BNP levels were elevated in 82.9% of patients with StressCMP in the International Takotsubo Registry study.
4. ECHO: RWMA with moderate to severe LV systolic dysfunction.
5. Coronary Angiograpthy: Normal coronaries in angiography documentation of which is must for diagnosis of this condition.

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Management:

- First line
  - Sedation/anxiolytics/analgesics is therapeutic
  - If possible, treat with combined α/β-blockers to reverse "catecholamine toxicity"; avoid unopposed β-blockade
- If hypotensive / cardiogenic shock
  - No LVOT obstruction:
    - standard therapy- ↓ preload/afterload; judicious inotropes
  - LVOT obstruction: (due to compensatory hyperdynamic contractions of basal segment of LV)
    - Peripheral vasoconstrictors; potential worsening of obstruction with catecholamines
    - Gentle volume resuscitation to decrease LVOT gradient
    - Early consideration of MCS- IABP or preferably pVAD to decompress LV and bypass LVOT
- If in pulmonary edema
  - Diuretics and fluid management
  - PA catheter helpful to optimize filling pressures

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