

**STRESS INDUCED CARDIOMYOPATHY**



*“La Belle Affligée”, La Gazette Du Bon Ton, no.8 Paris 1922*

*“...there are quiet victories and struggles, great sacrifices of self, and noble acts of heroism, in it - even in many of its apparent lightnesses and contradictions - not the less difficult to achieve, because they have no earthly chronicle or audience - done every day in nooks and corners, and in little households, and in men’s and women’s hearts - any one of which might reconcile the sternest man to such a world, and fill him with belief and hope in it . . .”*

*Charles Dickens, “The Battle of Life”, 1846*

*There is no pain so great as the memory of joy in present grief.*

*Aeschylus, (525 BC – 456 BC)*

*“Grief can induce the steadiest minds to waver”*

*Sophocles, (496 BC – 406 BC), Antigone.*

*“There is no grief which time does not lesson and soften”*

*Marcus Tullius Cicero, (106 BC – 43 BC) Epistles, IV, 5.*

*“Suppressed grief suffocates, it rages within the breast and is forced to multiply its strength”*

*Ovid, (43 BC – 17 AD) Tristium, V, I, 63*

*Give sorrow words; the grief that does not speak whispers the o'er-fraught heart and bids it break.*

*William Shakespeare, (1564-1616)*

*“Tears are the silent language of grief”*

*Voltaire, (1694-1778)*

*“Grief is the agony of an instant, the indulgence of grief the blunder of a life”*

*Benjamin Disraeli (1804-1881)*

*“There is no grief like the grief that does not speak”*

*Henry Wadsworth Longfellow, (1807-1882) Hyperion Bk II, Ch 2.*

*“But there is one thing worse than an absolutely loveless marriage. A marriage in which there is love, but on one side only; faith, but on one side only; devotion, but on one side only, and in which of the two hearts one is sure to be broken.”*

*Oscar Wilde (1854-1900)*

*Severe emotional distress is a part of the every day “battle of life”. It manifests in many strange ways. It may cause even the strongest mind to waiver. For some it remains a silent tearful struggle, without any “earthly chronicle or audience”, in others the heart is sure to be “broken”. In all however there is “no grief which time does not lesson and soften”.*

*In the medical field the syndrome of the “broken heart” is the “agony of an instant”, but mercifully in most cases it will, like many other forms of grief, heal with time.*

## **STRESS INDUCED CARDIOMYOPATHY**

### **Introduction**

**Stress-induced cardiomyopathy** is a recently recognized and increasingly reported syndrome of **transient systolic dysfunction** of the **apical and/ or mid segments** of the **left ventricle** that *mimics* myocardial infarction in the **absence** of significant coronary artery disease.

**Patients may present with chest pain, indistinguishable from myocardial infarction. They may also have ST segment elevation on their ECGs, indistinguishable from myocardial infarction.**

Terminology for the condition appears to be evolving. In the literature besides **stress induced cardiomyopathy** the following terms have also been used:

- Apical ballooning syndrome.
- Broken heart syndrome.
- Tako tsubo cardiomyopathy.

The condition was first described in Japan where it is known as Tako tsubo cardiomyopathy.<sup>2</sup> The appearance on ventriculography of the apical and mid-ventricular ballooning on systole together with compensatory hyperkinesis of the basal myocardial walls, was said to have the appearance of a traditional Japanese octopus trap – the “tako tsubo”.

**It is an emerging important differential diagnosis of acute occlusive coronary artery infarction.**

It is thought that the condition is being recognized more frequently now due to the more widespread early and aggressive intervention with coronary angiograms in patients who present with acute coronary syndromes.

There may be significant cardiac compromise during the acute episode; however the condition is usually transient with resolution of symptoms within one to four weeks, with a good longer term prognosis as compared to those who suffer from a myocardial infarction.

### **Epidemiology**

The condition is most commonly seen in post-menopausal women, though it is not exclusive to this demographic and both younger women and males can present with the condition.

It may be seen in as much as 2.5 % of cases of chest pain with ST segment elevation.

### **Pathology**

The exact pathophysiology of stress induced cardiomyopathy is uncertain, however, the evidence currently available suggests a catecholamine-mediated mechanism via cardiac sympathetic nerves.

The syndrome occurs at a time of surging catecholamine levels, which can be precipitated by emotional or physical stress but may also occur without an identifiable precipitant.

Catecholamine levels are characteristically far higher than in matched patients with acute anterior myocardial infarction and similar degrees of left ventricular dysfunction and heart failure.

Although there is no dispute that the syndrome is due to a sudden surge in catecholamines, there is considerable controversy as to how this results in the reversible wall motion abnormality and different theories exist relating to either effects on the microvasculature or to direct effects on the myocardial cells.

### Types

Stress induced cardiomyopathy has been subdivided by some into:

#### Typical cases:

In the more commonly described “typical” type of stress-induced cardiomyopathy, the contractile function of the **mid and apical** segments of the LV are depressed.

There is compensatory **hyperkinesis** of the **basal walls**, producing a ballooning of the apical segments with systole.

#### Atypical cases:

In a minority of cases the ventricular hypokinesis is restricted to the mid-ventricle region with relative sparing of the apical regions. This subtype has been termed “atypical”

### Clinical Features

**The presenting illness most closely mimics a STEMI**, and indeed the diagnosis of stress induced cardiomyopathy cannot be made until angiography has been performed.

#### Precipitating factors:

The syndrome is thought to be precipitated in the pre-disposed by an acute hyper-adrenergic stimulus.

This may be in the form of:

- Severe emotional distress
- Severe physical distress

It should be noted however that in a number of cases, no specific precipitating factor can be ascertained.

### Important points of history:

These include:

1. Chest pain:
  - This is the commonest presenting symptom and is seen in up to 90 % of cases
  - It may be indistinguishable in nature from ischaemic cardiac chest pain caused by occlusive coronary artery disease.

*Less commonly the following may be the predominant presenting symptoms:*

2. Dyspnoea
3. Palpitations
4. Syncope

### Important points of examination

As with acute myocardial infarction, features of high circulating adrenaline levels are also common:

1. Anxiety
2. Tachycardia
3. Hypertension or hypotension if systolic function is significantly compromised
4. Diaphoresis
5. Peripheral shutdown.

### Acute complications:

These can be severe and may include

1. Arrhythmias
  - These can be recurrent and intractable, they can be both tachyarrhythmias (including VT or VF) and bradyarrhythmias
2. Hypotension with cardiogenic shock:

The can be due to

- Systolic dysfunction.
- Left ventricular outflow tract (LVOT) obstruction. This is due to a physiological hyperkinesis of the basal sections of the heart

3. Acute pulmonary oedema
4. Acute mitral regurgitation.
5. Apical thrombus formation and subsequent stroke have also been described

## Investigations

### Blood tests

1. FBE
2. U&Es/ glucose
3. Troponin levels:
  - Levels do rise, but usually only to a modest degree compared to STEMI, or to the degree of wall motion abnormality observed on echocardiography.
  - In some cases there will be no troponin rise at all, but this will **not** exclude a diagnosis of stress induced cardiomyopathy.

### EKG

1. ST segment elevation in the anterior precordial leads:
  - This can be indistinguishable from STEMI
  - Some attempts have been made to outline ST segment morphological criteria to help distinguish ST segment elevation due to STEMI from that of stress induced cardiomyopathy, however these are not useful as they cannot make a definitive diagnosis, nor rule out the need for angiography.
2. Non-specific ST-T wave changes

### CXR

This is primarily to rule out other diagnoses for chest pain or to look for secondary complication such as acute pulmonary oedema

### Coronary angiography

The diagnosis of stress induced cardiomyopathy depends on coronary angiography.

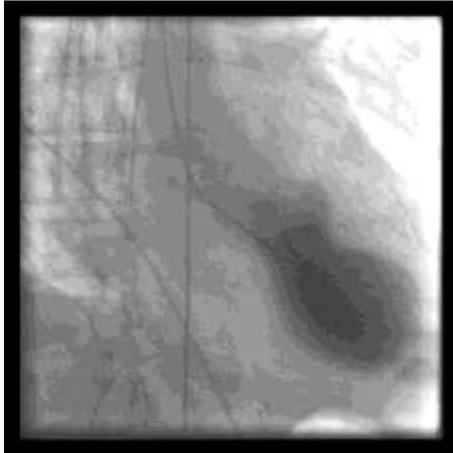
Coronary angiography is essentially normal, there being no significant occlusive lesion, or readily identifiable site of probable recent occlusion, a “culprit” lesion.

Additionally only a minority of patients display coronary spasm with acetylcholine provocation testing.

There is the characteristic akinesis of the distal half of the anterior and inferior walls and apex on left ventricular angiography which cannot be explained by the occlusion of a single vascular territory (ie, the left anterior descending artery does not extend beyond the apex to supply the distal half of the inferior wall).

In the apical sparing variant, this also cannot be explained on the basis of occlusion of a single coronary vessel.

### Left ventriculography



*Left: Left ventriculography during systole showing apical ballooning akinesis with basal hyperkinesis in a characteristic takotsubo ventricle. Right, a traditional Japanese Tako tsubo, octopus catching pot.*

### Echocardiography

This is useful for assessing the nature and degree of cardiac wall motion abnormalities, as well as the degree of functional impairment.

It may also show secondary complications such as acute mitral valve regurgitation or apical thrombus formation.

The wall motion abnormality returns to normal in many patients within days, and certainly within the first month. **Unless at least echocardiography and preferably coronary angiography are performed within the first 48 hours, the diagnosis may be missed.**

### Mayo Clinic criteria

The published Mayo Clinic criteria <sup>3</sup> for diagnosing stress induced cardiomyopathy have been fairly well accepted.

Criteria are based on:

- ECG findings
- Angiography findings
- The absence of other possible causes for the observed pathology.

The full criteria are:

Transient, reversible akinesis or dyskinesis of the left ventricular apical and mid-ventricular segments with regional wall motion abnormalities extending beyond a single vascular territory on left ventriculography.

1. Absence of obstructive coronary artery stenosis (> 50% of the luminal diameter or angiographic evidence of acute plaque rupture).
2. New electrocardiographic abnormalities consisting of ST-segment elevation or T-wave inversion.
3. Absence of:
  - Recent head trauma
  - Intracranial bleeding
  - Pheochromocytoma
  - Obstructive epicardial coronary artery disease
  - Myocarditis
  - Hypertrophic cardiomyopathy.

### Management

Management consists of:

1. Immediate attention to any ABC issues.
  - Oxygenation is the first priority. Some patients may develop acute pulmonary oedema, and require CPAP
2. Pain control:
  - Titrate IV opioid analgesia as required.
3. Maintain continuous ECG monitoring.
4. **Hypotension:**

Some patients with stress-induced cardiomyopathy will develop hypotension and shock, which could be due either to **severe systolic dysfunction** or to **LVOT obstruction**.

Because it will influence the choice of treatment, patients who develop severe hypotension should undergo urgent echocardiography to determine if LVOT obstruction is present.

**Management in these cases is complex and not well defined and will require close consultation with Cardiology and ICU.**

In general terms:

**In patients with hypotension who do not have significant outflow obstruction, intravenous inotropes, such as adrenaline or dopamine should be used**

**In patients with hypotension and moderate-to-severe LVOT obstruction, however inotropic agents may worsen the degree of obstruction and beta blockers may in fact be required.**

**In patients with hypotension, whether with or without significant LVOT obstruction, who do not respond to initial medical therapy resuscitation, an IABP will be required.**

5. Follow-up treatment:

Outside of critical hypoxic or hypotensive illness, there is no specific or proven treatment for stress induced cardiomyopathy.

During the period of recovery standard systolic heart failure medication (ACE inhibitors and angiotensin receptor blocker, and diuretics) and beta blockers should be given, particularly in cases of LVOT obstruction.

The optimal duration of treatment has not been established.

Prospective trials of long-term adrenergic receptor blockade for prevention of recurrence may provide evidence to support its use as a longer term management strategy.

Prognosis

- The wall motion abnormality in stress induced cardiomyopathy returns to normal in many patients within days, and certainly within the first month. This means that patients who do not succumb to acute haemodynamic compromise have an excellent longer term prognosis with no long-term sequelae.
- Recurrence rates are *low*, with estimates ranging between 5%–10%

References

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3. Bybee, KA, Kara, T, Prasad, A, et al. Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. Ann Intern Med 2004; 141:858.

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