

# Stress Induced Cardiomyopathy

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# Case Presentation

- TE 50 yo woman presented to SLH 2/16/11
- Was called home from work 2/15 PM by neighbor who told her apartment complex was on fire
- Developed chest tightness
  - Radiated to left arm, neck
  - Dyspnea and near syncope also
- In ED NTG relieved symptoms

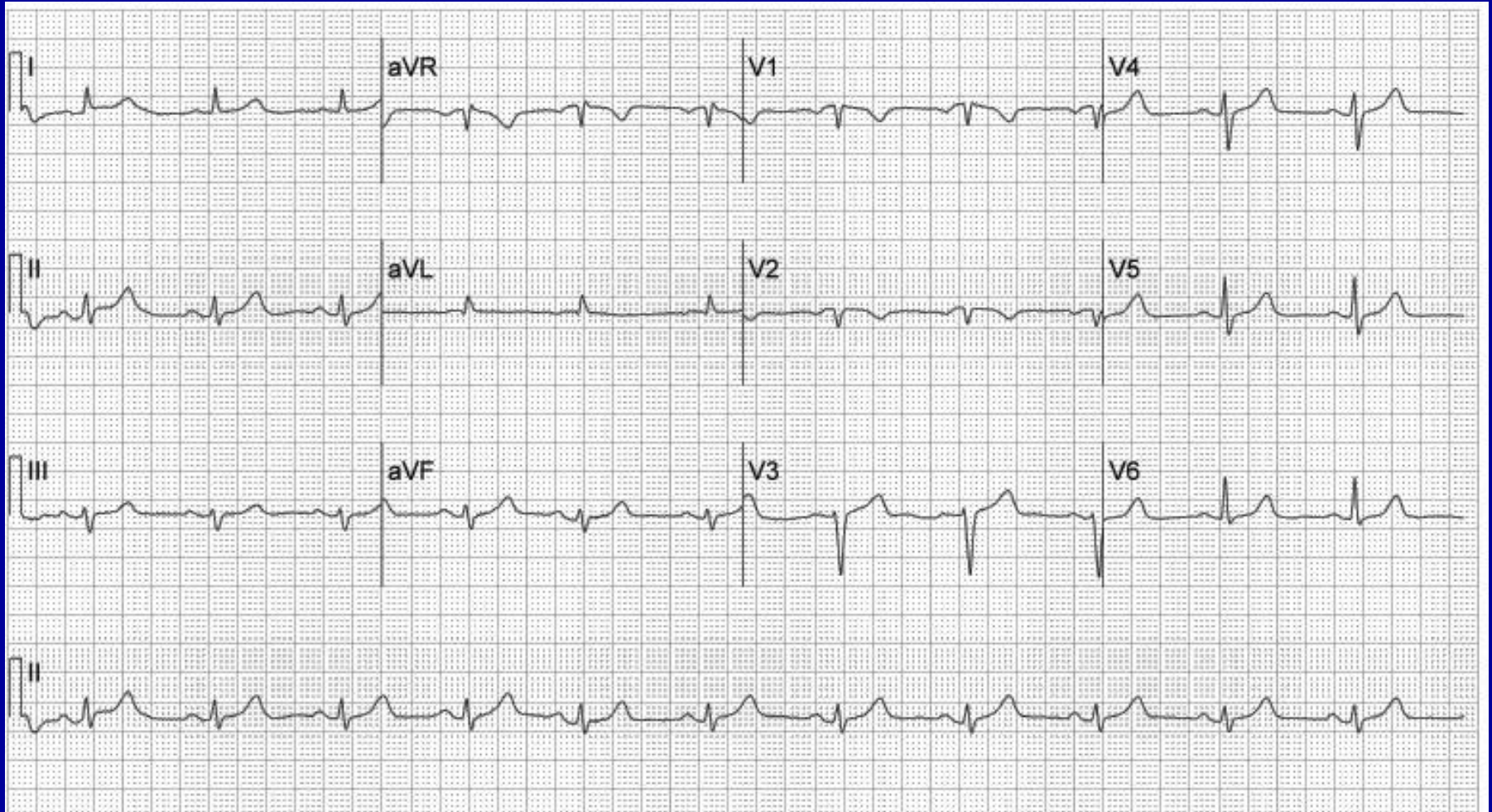
# Case Presentation

- PMH: HTN, POTS and ?dyslipidemia
- FH: Father had CAD in his 50's
- SH: Lifelong nonsmoker, works as RN

# Case Presentation

- Vitals: BP 114/75 HR 74, RR 16
- Exam otherwise normal
- Labs
  - Troponin
    - 2/16 0300: 0.11
    - 2/16 0855: 1.53

# Case Presentation



# Case Presentation

- Cath
- Echo

# Case Presentation

- Cath
  - Normal coronary arteries
  - EF 25% with severe apical hypokinesis
- Echo
  - EF 25-30%, apical hypokinesis

# Case Presentation

- Diagnosed with stress cardiomyopathy and discharged
  - Placed on following meds:
    - Lisinopril/HCTZ 10/12.5 mg daily
    - Carvedilol 3.125 mg twice daily
    - Aldactone 25 mg daily

# Stress Cardiomyopathy

- Also known as:
  - Takotsubo syndrome
  - Broken heart syndrome
  - Apical ballooning syndrome

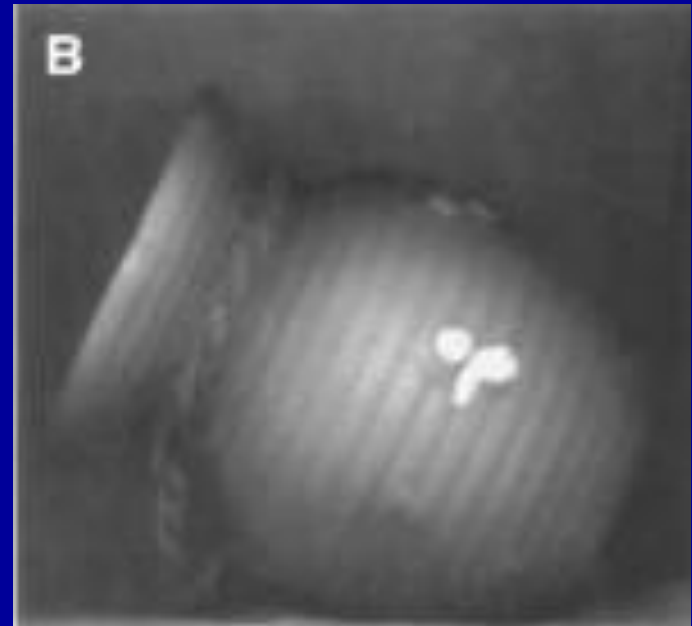
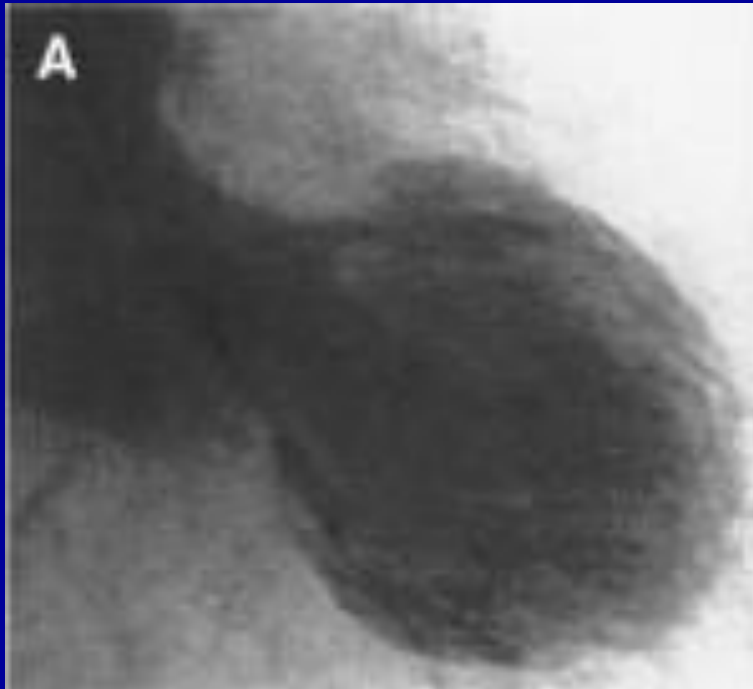
# Stress Cardiomyopathy

## Takotsubo Syndrome

- 1<sup>st</sup> described in Japan in 1991
- Named after the tako-tsubo, which is an octopus trap
  - Shape of the trap is similar to the appearance of LV apical ballooning noted in patients with this form of cardiomyopathy
- Was later described elsewhere as well and is being increasingly recognized.



# Stress Cardiomyopathy Takotsubo Syndrome



Kurusu, S., et al. 2002. *American Heart Journal*. 143: 448-455.

# Stress Cardiomyopathy

- May account for up to 2% of suspected ACS
- In-hospital mortality ranges 0-8%
- Much more common in women (~90%), especially postmenopausal women (>80% of cases)
- Mean age 58-75 years
- Triggers: death of loved one, other catastrophic news, devastating financial losses, natural disasters, physical illness/ICU, etc.

# Stress Cardiomyopathy

## Diagnostic Criteria

1. Transient a/dyskinesis of apical and midventricular segments in association with regional wall motion abnormalities that extend beyond the distribution of a single epicardial vessel
2. Absence on angiography of obstructive coronary artery disease or evidence of acute plaque rupture
3. New ST segment elevation or T wave inversions on ECG
4. Absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, myocarditis, or hypertrophic cardiomyopathy

# Stress Cardiomyopathy

## Pathophysiology

- Catecholamine excess
  - Norepinephrine levels are elevated in ~75% in some studies
  - Plasma catecholamines are significantly higher than in cases of MI
  - May induce microvascular spasm or dysfunction → myocardial stunning or direct myocardial toxicity
  - Limited endomyocardial biopsy data c/w histologic signs of catecholamine toxicity
- Coronary artery spasm or microvascular spasm
- Myocarditis

# Stress Cardiomyopathy

## Clinical Presentation

- Chest discomfort
- Dyspnea
- ECG abnormalities
- Elevated cardiac biomarkers
  - Typical rise and fall pattern
- Shock
  - Rare

# Stress Cardiomyopathy

## Complications

- Tachyarrhythmias, bradyarrhythmias
- Pulmonary edema
- Cardiogenic shock
- Transient LV outflow tract obstruction
- Mitral valve dysfunction
- Acute thrombus formation and stroke
- Death

# Stress Cardiomyopathy

## Evaluation

- Cardiac catheterization
  - Documents lack of CAD
  - Ventriculography reveals EF and typical wall motion pattern
    - Average LV EF range 20-49%
    - Wall motion abnormalities typically involve the distribution of more than one coronary artery
- Echocardiography
  - Also reveals EF and wall motion

# Stress Cardiomyopathy Management

- Supportive, conservative therapy
  - Hydrate, remove stress (if possible)
- Treat LV dysfunction with standard heart failure regimen
  - ACE-Inhibitor/ARB
  - Beta blocker
  - Diuretics as needed
  - *Usually treated for at least 6 months*

# Stress Cardiomyopathy Management

- For pts who are hypotensive with shock, perform echo to evaluate for LVOT obstruction.
  - No LVOT obstruction → inotropes, IABP if needed
  - +LVOT obstruction → NO inotropes (can worsen obstruction), use beta blockers (+/-  $\alpha$  agonist Phenylephrine), IABP if needed
  - +/- fluid resuscitation (evaluate pulmonary status)

# Stress Cardiomyopathy

## Prognosis

- Overall, good prognosis.
  - If patient survives the acute phase, long-term prognosis is excellent
- 0-8% in-hospital mortality, likely closer to 1-2%
- Recovery of LV function, typically in 1-4 weeks
- Late sudden death (rare) and recurrent disease (<10%) have been reported

# Stress Cardiomyopathy

## Summary

- Syndrome of transient dysfunction of apical/midventricular LV with compensatory hyperkinesis of basal segment resulting in apical ballooning.
- It is triggered by significant emotional or physical stress.
- It is more common in post-menopausal women.

# Stress Cardiomyopathy

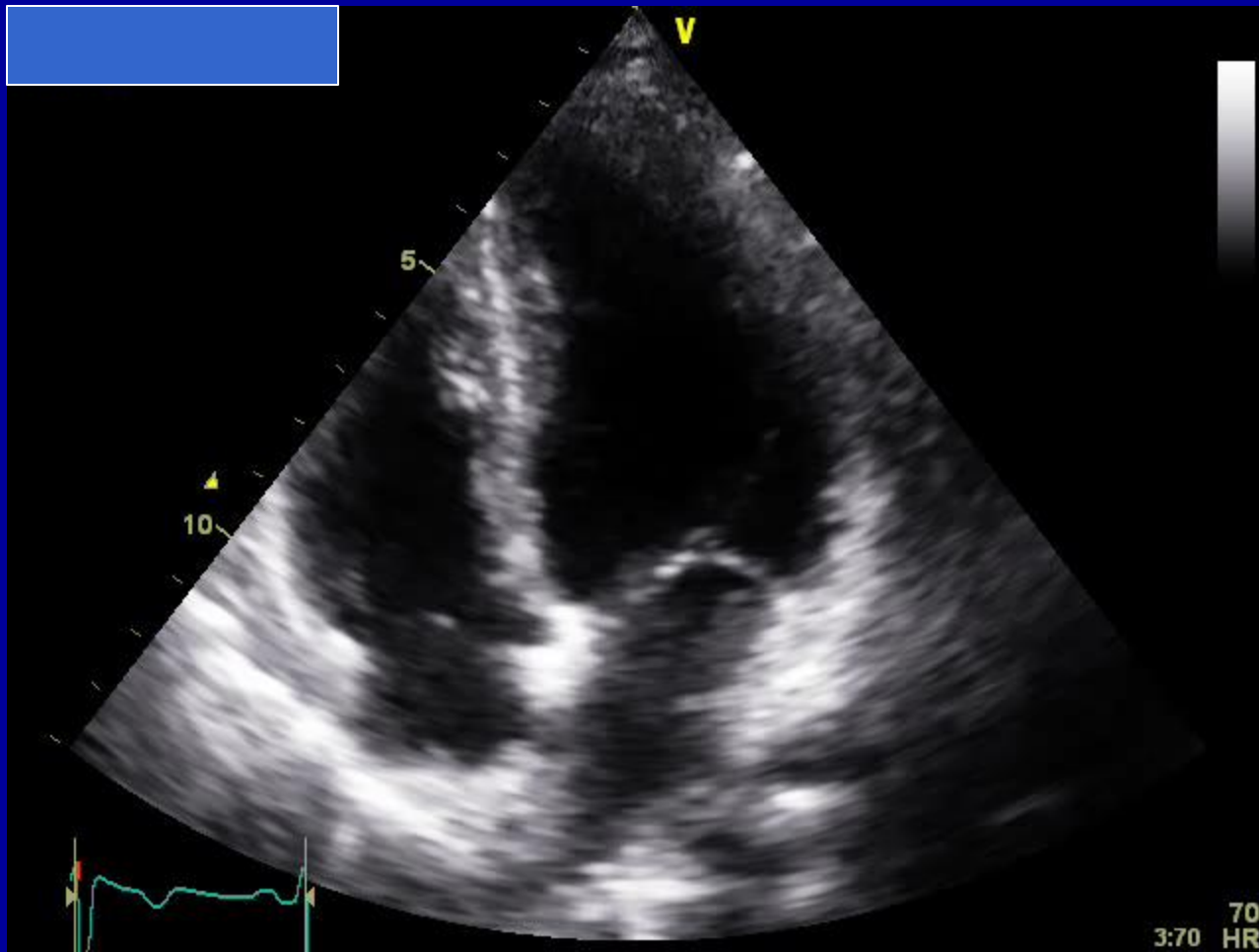
## Summary

- Presentation is similar to MI (symptoms, ECG changes, and biomarker elevations).
- Accounts for ~1-2% of suspected ACS cases.
- No significant coronary artery disease or evidence of plaque rupture can be identified.
- LV function recovers, typically within 4 weeks.

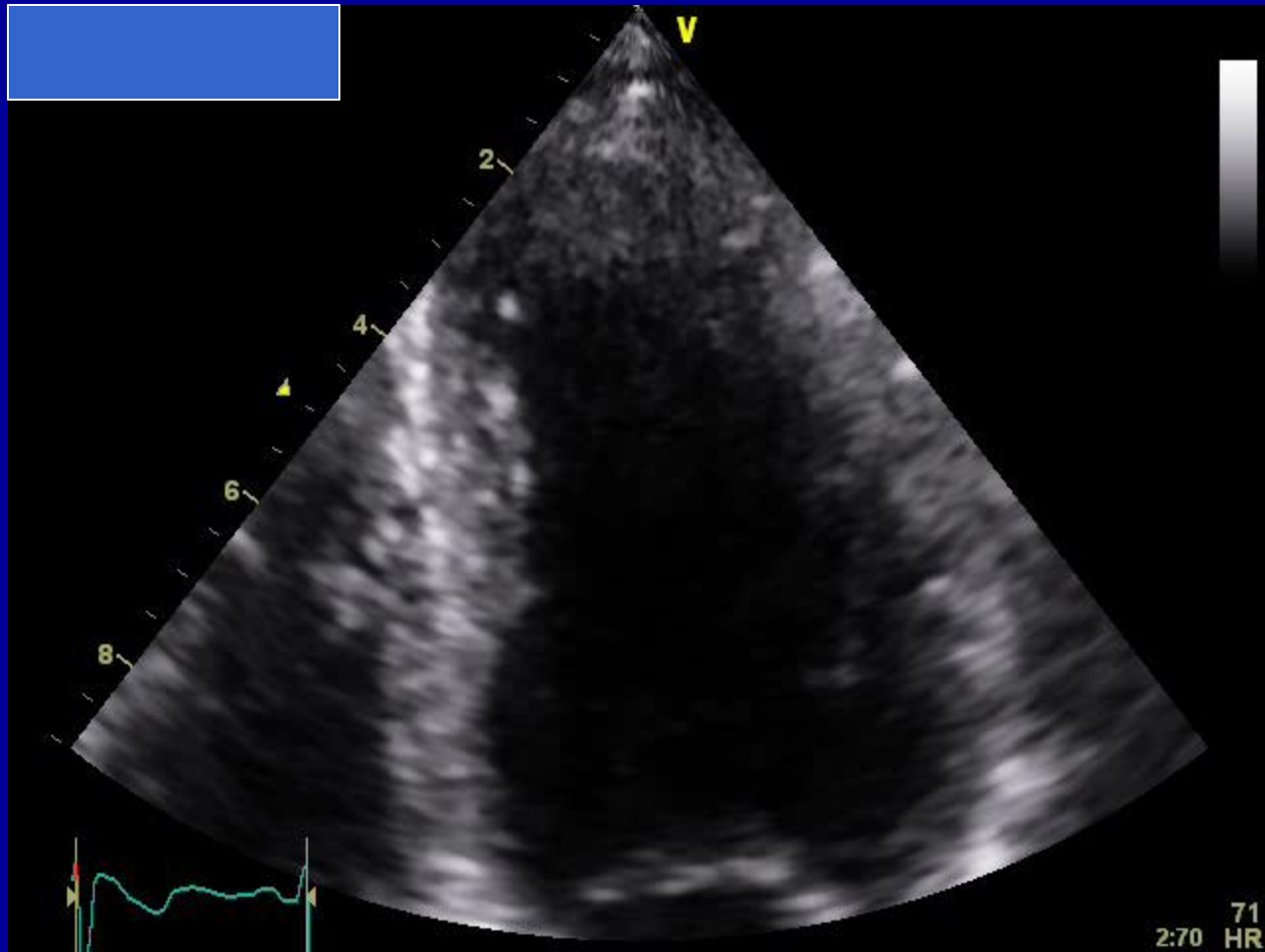
# Follow-Up

- 3/1/11 office visit
  - Doing well
  - No chest pain or dyspnea
  - Limited echo repeated

# Follow-Up



# Follow-Up



# Follow-Up

- 3/1/11 office visit
  - Doing well
  - No chest pain or dyspnea
  - Limited echo repeated
    - EF normal with normal wall motion
- Has done well since