Takotsubo Cardiomyopathy
Broken Heart Syndrome

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Takotsubo Cardiomyopathy

• Cardiomyopathy characterized by transient apical and midventricular LV dysfunction in the absence of significant coronary artery disease, triggered by emotional or physical stress
  • In setting of depressed/abnormal function of distal and apical LV segments there is compensatory hyperkinesis of basal walls → “ballooning” of apex during systole
• Typically recover normal LV function in 1-4 weeks
Takotsubo Cardiomyopathy

- 1st 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
Takotsubo Cardiomyopathy
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Aliases

- Takotsubo cardiomyopathy
- Stress-induced cardiomyopathy
- Transient left ventricular apical ballooning syndrome
- Apical ballooning syndrome
- Broken heart syndrome
- Ampulla cardiomyopathy
Takotsubo Cardiomyopathy

- May account for up to 2% of suspected ACS
- In-hospital mortality ranges from 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.
Psychiatric Factors

Triggering Event

- Stressful trigger is positive predictor of TCM
- Emotional type better prognosis
- Physical type more often in males

High prevalence of psychiatric disorders

- Anxiety, depression (including family history)
- Chronic psychological stress
Mayo Clinic Criteria (2008)

- **Transient dyskinesis of LV mid segments**
  - Regions of wall motion abnormality extend beyond single epicardial vascular distribution
  - Stressful trigger often, but not always, present

- **Absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture**

- **New ECG abnormalities**
  - ST elevation and/or T wave inversions, or modest elevation in cardiac troponin

- **Absence of other precipitants (e.g., Pheochromocytoma, myocarditis)**
(Postulated) Pathogenesis

• 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
Pathophysiology

- Supraphysiologic catecholamine levels
  - Increased Beta-2 receptor binding
    - Hypercontraction
    - Apoptosis
    - Necrosis
    - Rising troponin levels
- "Stimulus Trafficking"
  - Via PI3K/AKT pathway
    - Switch from Gs to Gi
    - Declining contractile function
    - Hypokinesis
    - Apical ballooning
    - Rapid recovery
Presentation...
(similar to acute MI)

- Substernal chest pain
- ECG abnormalities
  - ST elevation (usually anterior precordial leads)- 82%
  - ST depression
  - T wave inversion
  - QT prolongation
  - Abnormal Q waves
- Elevated cardiac biomarkers
- Dyspnea
- Shock
- Syncope
Acute Complications

- Tachydysrhythmias, bradydysrhythmias
- Pulmonary edema
- Cardiogenic shock
- Transient LV outflow tract obstruction
- Mitral valve dysfunction
- Acute thrombus formation and stroke
- Death
Because presentation is similar to ACS, proceed to cardiac catheterization/PCI, if available, or fibrinolysis.

LV ventriculogram and/or echocardiography can both be used to visualize apical ballooning with a/dyskinesis of apical ½ to 2/3 of the LV

- Average LV EF range is 20-49%
- Can have “atypical” ballooning of the middle or basal portions of the LV (much less common)
- Wall motion abnormalities typically involve the distribution of more than one coronary artery

Ventriculography and echocardiography also allow evaluation for LV outflow tract obstruction (~16%)

Cardiac catheterization reveals lack of flow limiting coronary lesions or evidence of plaque rupture.
Management

• Supportive, conservative therapy
  • Hydrate, remove stress if possible

• Treat LV dysfunction with standard heart failure regimen- including beta blocker, ACE inhibitor, diuretics (if volume overloaded), aspirin
  • Usually treated for ~6 month

• 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 (+/- α agonist Phenylephrine), IABP if needed
  • +/- fluid resuscitation (evaluate pulmonary status)
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.
Take Home Points

• Takotsubo cardiomyopathy is a 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 postmenopausal women

• 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
References

