Takotsubo cardiomyopathy: Presentation, management and outcome

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Introduction

- Takotsubo cardiomyopathy was first described in Japan in 1990 by Sato et al..
- American Heart Association (AHA) classification of cardiomyopathy (2006)
- Also known as broken heart syndrome, apical ballooning syndrome, and stress induced cardiomyopathy
- It is generally characterized by transient systolic dysfunction of the apical and mid segments of the LV that mimics myocardial infarction in absence of obstructive CAD
The term “takotsubo” was taken from the Japanese name for an octopus trap pot.

Pathophysiology

- The exact mechanism of this disorder is **not well understood**.

- Postulated mechanisms:
  1. Excessive sympathetic stimulation
  2. Metabolic abnormalities
  3. Microvascular dysfunction
Pathophysiology (contd.)
Excessive sympathetic stimulation


Pathophysiology (contd.)
Metabolism abnormalities

Myocardial perfusion images with Tc-99 m tetrofosmin SPECT (A), F-18 FDG PET images (B), and I-123 MIBG images (C) in a patient with TC. The metabolic and sympathetic images are abnormal, but the perfusion images are normal. Cimarelli et, Nucl Cardiol 2008
Pathophysiology (contd.)
Microvascular dysfunction

- Reduction in coronary flow reserve

- Decrease in time of diastolic velocity deceleration

Types of Takotsubo
Incidence

- 2.2% of patients admitted with diagnosis of acute MI
- 2% of patients admitted with decompensated heart failure
- 0.02% of all hospitalization

Clinical presentation
Demographics

- Sex: *Female predominance (80-100%)
- Age: *Postmenopausal (62-76 years)
  *5-11% under age of 50 years
- Triggers: *Physical stress (36%)
  *Emotional stress (27.7%)
  *1/3 of cases no apparent trigger.
Clinical presentation (contd.)
Symptoms and Signs

- Chest pain (76%)
- Dyspnea (47%)
- Syncope (7.7%)
- Arrhythmia
- Cardiac arrest

Investigations

- **ECG:**
  - Diffuse T wave inversion (97%)
  - ST segment elevation (68%)
  - Q waves (27%)
  - ST segment depression (10%)
  - Conduction abnormalities
  - QT prolongation
Phases of ECG changes:

- Phase 1: ST segment elevation (acute phase)
- Phase 2: T wave inversion (day 1-3)
- Phase 3: transient improvement of T wave inversion (day 2-6)
- Phase 4: Giant T wave inversion and QT prolongation (tell recovery)
Investigations (contd.)

- **Biomarkers:**
  - Cardiac Troponin (86%)
  - Creatine kinase-MB (74%)
  - Plasma B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP)

Investigations (contd.)

- **Imaging:**
  - Typical wall motion abnormalities
  - Assessment of LV systolic function
  - Detection of RV involvement (30%)
  - Detection of LVOT obstruction
  - Complications (LV thrombus, LV free wall rupture)
  - Absence of late gadolinium enhancement (LGE) by cardiac MRI
Investigations (contd.)

- **Coronary angiography**:
  - Non obstructive coronary artery disease (<50% stenosis)
  - Obstructive coronary artery disease (10%)

Investigations (contd.)

- **Left ventriculography**: [Images of ventriculography]
Diagnosis

- Mayo Clinic diagnostic criteria:

1. Transient hypokinesis, akinesis or dyskinesis of the left ventricular mid segments with or without apical involvement. The regional wall motion abnormalities typically extend beyond a single epicardial coronary distribution. A stressful trigger is often, but not always present.

2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.

3. New electrocardiographic abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin.

4. Absence of pheochromocytoma or myocarditis.

Complications

- Congestive heart failure (20%)

- Cardiogenic shock

- Apical thrombus (5.3%)

- Ventricular rupture

- Arrhythmias:
  1. Life threatening ventricular arrhythmia (8.6%)
  2. AV block
Treatment

General principles of therapy:
1. Initial management should be directed towards myocardial ischemia (Dual anti-platelets, anti-thrombotics, nitrates, statins, β blockers).
2. IV diuretics in patients with heart failure and volume overload.
3. Once the diagnosis of Takotsubo cardiomyopathy has been made, standard medications for left ventricular systolic dysfunction (ACEI/ARBs, β blockers, diuretics).
4. The appropriate duration of therapy is not known.
5. There is no consensus regarding long-term management (ACEI/ARBs and β blockers).

Treatment (contd.)

Cardiogenic shock:
• First step is to rule out left ventricular outflow tract (LVOT) obstruction.
1. No LVOT obstruction:
   • IV inotropic agents and vasopressors (dopamine, dobutrex)
   • Mechanical circulatory support (Intra-aortic balloon counter pulsation)
   • Inotropic agents and IABP may cause LVOT obstruction. Echocardiography to rule out such adverse effects if there is hemodynamic deterioration.
Treatment (contd.)

- Cardiogenic shock (contd.):
  2. **LVOT obstruction:**
     • Discontinue IV inotropic agents and nitroglycerine immediately
     • β blockers (in the absence of severe heart failure)
     • IV fluids (in absence of significant pulmonary congestion)
     • Phenylephrine (IV fluids and β blockers intolerance)

**Treatment (contd.)**

- **Thromboembolism:**
  • It is recommended approximately three months of **anticoagulation if intraventricular thrombus is detected.**
    The duration of anticoagulation may be modified based on the rate of recovery of cardiac function and resolution of the thrombus.
  • For patients **without thrombus** but with severe left ventricular dysfunction, It is suggested that anticoagulation until akinesis or dyskinesis has resolved or for three months, whichever is shorter.
Treatment (contd.)

- **Arrhythmias**
  - Should be managed on case by case bases
  - Devices implantation is not justified given the reversible nature of the disease
  - Correct risk factors of QTc prolongation (hypokalemia, hypomagnesemia, bradycardia, anti-arrhythmic drugs)
  - Werabale cardioverter-defibrillators for life threatening ventricular arrhythmia tell recovery.
  - Temporary pacemaker implantation if AV block associated with hemodynamic instability or marked QTc prolongation

Prognosis

- In-hospital mortality rates (0-8%).
- Recurrence rate (0-15%)
- 30 days MACCE (7.1%).
- Long term outcome:
  - Death rate from any cause (5.6% per patient-year)
  - MACCE (9.9% per patient-year)
  - Men have worse outcome than women
Take Home Messages

- Takotsubo cardiomyopathy is currently a diagnosis of exclusion.

- Full understanding of the pathophysiology of takotsubo cardiomyopathy is limited.

- There is no specific therapy for takotsubo cardiomyopathy.

- Prognosis is generally favorable with complete recovery of LV function but early detection of complications and prompt management are crucial.

- Data from randomized trials is needed.

Don’t go breaking your heart
Thank you