A CASE OF ACUTE HEART FAILURE IN COVID-19

Arezoo Mohamadifar, MD
Fellowship of Heart Failure and Transplantation
Masih Daneshvari Medical and Research Center
• A 72-year-old woman, fever and dyspnea

• RR: 25/min, O2 sat : 92%, Lungs: bilateral inspiratory crackles and expiratory rhonchi.

• HR: 70/min, BP: 150/70 mmHg.

• Electrocardiography (ECG) sinus rhythm with normal repolarization and a normal QTc interval (440 ms)
SEVEN DAYS AFTER ICU ADMISSION

Troponin-I: 0.4 ng/ml
• Troponin levels were normalized 7 days after the diagnosis of Takotsubo cardiomyopathy. At that time, the ECG showed sinus rhythm and normalization of the T-waves.

• Follow-up echocardiography showed important improvement of left ventricular systolic function to an LVEF of 45%, however with persistent hypokinesia of the apical segments.

• Three months after the diagnosis TC, the patient visited our outpatient clinic. Echocardiography at that time showed normal contractility of the apical myocardial segments, with normalization of the left ventricular systolic function (LVEF 55%), as expected in TC.
TAKOTSUBO SYNDROME
BROKEN HEART SYNDROME, ‘STRESS CARDIOMYOPATHY
APICAL BALLOONING SYNDROME
EPIDEMIOLOGY

• 1–3% of all and 5–6% of female patients presenting with suspected STEMI
• About 90% of TTS patients are women
• 4% to 13% of are men, in men, physical stress rather than emotional stress
PATHOPHYSIOLOGY

- **Sympathetic stimulation**
  - Plaque rupture
  - Multi-vessel epicardial spasm
  - Microcirculatory dysfunction
  - Catecholamine toxicity on cardiomyocytes
  - Activation of myocardial survival pathways
Predisposition and risk factors
- Hormonal factors
- Genetic factors
- Psychiatric and neurologic disorders

Triggers
- Emotional stressors
- Physical stressors
Types of takotsubo syndrome
**InterTAK Diagnostic Criteria**

1. Patients show transient* left ventricular dysfunction (hypokinesia, akinesia, or dyskinesia) presenting as apical ballooning or midventricular, basal, or focal wall motion abnormalities. Right ventricular involvement can be present. Besides these regional wall motion patterns, transitions between all types can exist. The regional wall motion abnormality usually extends beyond a single epicardial vascular distribution; however, rare cases can exist where the regional wall motion abnormality is present in the subtended myocardial territory of a single coronary artery (focal TTS).b

2. An emotional, physical, or combined trigger can precede the takotsubo syndrome event, but this is not obligatory.

3. Neurologic disorders (e.g. subarachnoid haemorrhage, stroke/transient ischaemic attack, or seizures) as well as pheochromocytoma may serve as triggers for takotsubo syndrome.

4. New ECG abnormalities are present (ST-segment elevation, ST-segment depression, T-wave inversion, and QTc prolongation); however, rare cases exist without any ECG changes.

5. Levels of cardiac biomarkers (troponin and creatine kinase) are moderately elevated in most cases; significant elevation of brain natriuretic peptide is common.

6. Significant coronary artery disease is not a contradiction in takotsubo syndrome.

7. Patients have no evidence of infectious myocarditis.b

8. Postmenopausal women are predominantly affected.

---

- Pheochromocytoma
- Concomitant CAD
IN-HOSPITAL COMPLICATIONS

Frequent
- Acute heart failure (12-45%)
- LVOTO (10-25%)
- Mitral regurgitation (14-25%)
- Cardiogenic shock (6-20%)

Moderate
- Atrial fibrillation (5-15%)
- LV-thrombus (2-8%)
- Cardiac arrest (4-6%)
- AV-block ~5%

Rare
- Tachyarrhythmia (2-5%)
- Bradyarrhythmia (2-5%)
- Torsades-de-pointes (2-5%)
- Death (1-4.5%)
- Ventricular tachycardia/fibrillation ~3%
- Acute ventricular septal defect <1%
MANAGEMENT OF ACUTE COMPLICATIONS

- Congestive Heart Failure
- Cardiogenic Shock
- Apical Thrombosis
- Arrhythmias
- Ventricular Rupture
**Acute Heart Failure Treatment**

**Mild TTS w/o signs of HF**
- Cardiology unit with telemetry monitoring for at least 48 hrs
- Consider:
  - ACE inhibitor or ARB
  - Beta-blocker

**Heart Failure/Pulmonary Edema**
- Intermediate Care Unit (preferentially)
- Consider:
  - ACE inhibitor or ARB
  - Beta-blocker
  - Diuretics (if no LVOTO)
  - Nitroglycerin (if no LVOTO)

**Hypotension/Cardiogenic Shock**
- Intensive Care Unit (preferentially)
- LVOTO
- Consider:
  - IV fluid (if no HF)
  - Short acting Beta-blocker
  - LVAD (Impella)
- Avoid:
  - Diuretics
  - Nitroglycerin
  - IABP

**Primary pump failure**
- Consider:
  - Levosimendan
  - LVAD (Impella)
  - VA-ECMO

**Arrhythmias**
- (e.g. VT, VF, Torsades de pointes, AV-Block, Long QTc)
- Consider:
  - Beta-blocker
  - Temporary RV pacing if AV block
  - Life Vest
- Avoid:
  - QT interval prolonging drugs
  - Beta-blockade in bradycardia and QTc >500 ms
  - Permanent devices

**Thrombo- &/or Embolism**
- (e.g. LV-thrombus, Embolization)
- Heparin/Vit.K Antagonists/NOAC (until first follow-up)
- Consider anticoagulation:
  - if LVEF ≤30% &/or a large LVD involving the apex is present

**Treatment of Complications**

**Three months or until RWMA recovery**
- Consider:
  - ACE inhibitor or ARB

**Treatment of other underlying disorders, e.g.**
- Coronary artery disease:
  - Aspirin
  - Statin
- Depression/Anxiety:
  - Combined psycho-cardial rehabilitation

**Recurrence Prevention**
- Consider:
  - Hormone replacement
  - ACE inhibitor or ARB
COURSE AND PROGNOSIS

• In most cases, RWMA resolves within days to a few weeks.
• The in-hospital death 0% to 8%. Prognosis is generally favorable.
• ECG or echocardiographic findings may be useful for predicting short-term prognosis.
• Recurrence rate ranges from 0% to 15%, despite treatment with calcium-channel blockers, nitrates, β-blockers, statins or aspirin.
THANKS