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Takotsubo Cardiomyopathy: The Pathophysiology of Broken Heart Syndrome

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Introduction

Takotsubo cardiomyopathy (TC) is a syndrome that was identified in the early 1990s in Japan to explain an acute coronary syndrome (ACS) presentation without the typical coronary artery occlusion that accompanies ACS. Although rare, Takotsubo cardiomyopathy or *broken heart syndrome*, has become a topic of interest in the world of cardiology care. It has stumped the medical world with its identical presentation to a ST-elevated myocardial infarction (STEMI), with symptoms of angina, ST-segment elevation on 12-lead EKG, elevated myocardial enzyme markers, and moderate to severe left ventricular dysfunction without coronary artery occlusion

Signs and Symptoms

Classic signs and symptoms of myocardial infarction (MI)

- crushing sub-sternal chest pain
  - can include typical radiation to left arm, back, and jaw seen in MI
- elevated troponin level
- ST-segment elevation on EKG (See Figure 1)
- marked systolic dysfunction with decreased ejection fraction (EF) via echocardiography
  - left ventricular apical ballooning with hypokinesia of the myocardium
- absence of coronary artery occlusion during cardiac catheterization
- concurrent acute physical or emotional stress event
  - death of a loved one is typical
- absence of coronary artery occlusion during cardiac catheterization
- myocardial dysfunction restored without intervention (i.e. balloon angioplasty or cardiac stent)

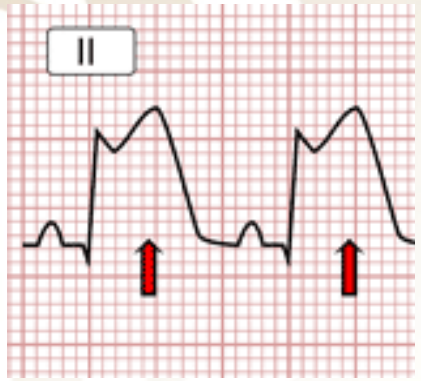

Figure 1: Classic "fireman's hat" ST-segment elevation seen in STEMI and also in Takotsubo Cardiomyopathy.

Underlying Pathophysiology

Massive Catecholamine Release

During great stress, the sympathetic nervous system is in over-drive, producing an excess of endogenous catecholamines like epinephrine and norepinephrine. These endogenous chemicals stimulate the Beta-adrenergic cells located in the heart that increase heart rate and contractility and induce hypertrophy on the cells. This is seen in the *ballooning* of the left ventricular apex most common in TC.

Left Ventricular Apical Ballooning

The term tako-tsubo, a Japanese term, "describes a narrow neck fishing pot used to catch octopus" (see Figure 2). This narrow neck and bowed out base of the pot are characteristic of the left ventricle ballooning out during TC. The apex is saturated with Beta-adrenergic receptors and during episodes of catecholamine excess, is believed to be in hyper-drive and attributes to the exaggerated shape, left ventricular dysfunction, and leaking of troponin, which is a manifestation of myocardial damage. This hypertrophy is believed to induce increased filling pressures and cause a significant decrease in the EF.

Coronary Artery Vasospasms

Surges of epinephrine during intense periods of physical or emotional stress also collect in the coronary arteries and believe to cause vasospasms of the coronary vasculature, causing myocardial ischemia and the ST-segment elevation classically identified during STEMI (ST elevated myocardial infarction) and TC.

Significance of Pathophysiology

The significance of the pathophysiology lies in its exact mimicking of acute coronary syndrome. Patients must be treated per ACS protocol and only once coronary angiography confirms there is an absence of coronary occlusion and criterion is met can a patient be diagnosed and treated with TC. The underlying pathophysiology has not been clearly defined so further research on the condition is highly warranted. TC is significant in its causative factor being endogenous – although risk factors such as hyperlipidemia, smoking, and alcohol use has been identified, the intense catecholamine surge is not a modifiable factor.

Parasympathetic Dysfunction

Norcliffe-Kaufmann et al., (2016) introduce their findings that participants in their study had a parasympathetic dysfunction that led to prolonged periods of sympathetic stimulation after stress that contributed to their cardiomyopathy. Without the typical "rest and digest" function of the parasympathetic nervous system, the sympathetic nervous system continues to stimulate catecholamine production and induces the left ventricular apical ballooning typical in TC.

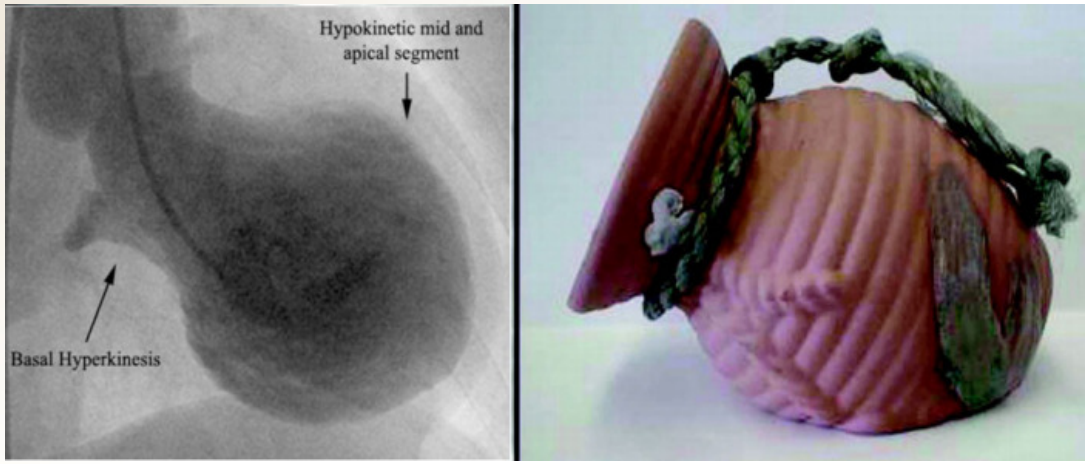

Figure 2: On the left: the dilated ballooning of the left ventricular apex classically seen in Takotubo cardiomyopathy. *On the right*: the Japanese pot in which Takotsubu cardiomyopathy was named after. "Tako" meaning octopus and "tsubo" meaning pot. Notice the narrowed neck with enlarged base of the pot.

Implications for Care

Treatment of TC mimics the treatment of a non-STEMI

- aspirin
- nitroglycerin infusion→ vasodilation and decreased preload/afterload
- heparin infusion
- Beta-blockers →decrease catecholamine stimulation and cardiac demand
- Inotropes and intra-aortic balloon pump (IABP) → may be temporarily necessary to ensure hemodynamic stability
- diuretics → prevent pulmonary edema
- anxiolytics → such as lorazepam should be considered, as stress is the at the root of TC's pathophysiology
- Stents, thrombolytics, and chronic heart disease medications are no longer recommended for the treatment of TC.

Nursing care should focus on education on TC's reversibility and also to identify long-term complications such as cardiogenic shock or thrombus formation and subsequent CVA which occur from the abnormal ventricular wall movement. Supporting the patient not only medically but emotionally can help with disease progression and lead to restoration of myocardial function.

Conclusion

Takotsubo cardiomyopathy is a complex condition. Although necessary, increased cost, patient stress, and superfluous hospital resource use occur as the condition mimics acute coronary syndrome and must be treated as so until the diagnosis of TC can be achieved. An excess in catecholamines produced by acute events of physical or emotional stress seem to be evident in all current research, but a dysfunction in the parasympathetic nervous system and coronary vasospasms have also been attributing factors. Quick recognition and treatment with aspirin, heparin, nitrates, beta-blockers, possibly IABP, as well as vasopressors are indicated to increase cardiac function, to enhance end-organ perfusion and prevent cardiogenic shock as well as thrombus formation. Emotional support along with anxiolytic therapy is recommended because as the catecholamine levels decrease, the myocardium has proven to restore itself back to the pre-stressor state of function. Healing a broken heart may be complicated, but with evidence-based treatment and excellent nursing care, TC is entirely reversible.

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