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Takotsubo Cardiomyopathy: The Pathophysiology of Broken Heart Syndrome

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Introduction
Takotsubo cardiomyopathy (TTC) is a syndrome that was identified in the early 1990s in Japan based on an acute coronary syndrome (ACS) presentation without the typical coronary occlusion that accompanies ACS. Although now, Takotsubo cardiomyopathy of broken heart syndrome, has become a topic of interest in the world of cardiology. It has upended the medical world with its identical presentation to a ST-elevated myocardial infarction (STEMI) with a symptomatic angiogram, 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):
• Severe chest pain or discomfort
• Cold sweating
• Shortness of breath

Common treatments:
• Nitrates
• Beta-blockers
• Diuretics
• Intravenous adenosine

Left Ventricular Apical Ballooning
The term takotsubo, a Japanese term, describes a narrow neck-fishing pot useful to catch octopus (see Figure 2). This narrow neck and bulb out at the pot are characteristic of the left ventricular ballooning. The apex is saturated with catecholamines, causing myocardial ischemia and ST-segment elevation flattened identified during STEMI (ST-elevated myocardial injury) and TTC.

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 TTC.

Underlying Pathophysiology
Massive Catecholamine Release
During stress, the sympathetic nervous system is over-drive, producing an excess of endogenous catecholamines like epinephrine and norepinephrine. This excess endogenous catecholamines inside the beta-adrenergic cells located in the heart that increase heart rate and contractility and induce hypotrophy on the cells. This is seen in the ballooning of the left ventricular apex most common in TTC.

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; the criterion is met when a patient is diagnosed, and treated with TTC. The underlying pathophysiology has not been clearly defined, so further research on the condition is highly warranted. TTC is significant in its causative factor being endogenous - although risk factors such as hyperlipidemia, smoking, and a keloid level has been identified, the intracellular catecholamine surge is not a modifiable factor.

Parasympathetic Dysfunction

Nordrhein-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 the 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 of TTC.

Implications for Care
Treatment of TTC involves the treatment of a non-STEMI:
- Nitrates: infusion to vasodilate and decrease preload/afterload
- Heparin infusion
- Beta-blockers: decrease catecholamine stimulation and cardiac demand
- Insoprost and intra-aortic balloon pump (IABP): May be temporarily necessary to ensure hemodynamic stability
- Diuretics: prevent pulmonary edema
- Analgesics: such as acetaminophen should be considered, as stress is the root of TTC pathophysiology
- Stents, thrombolysis, and chronic heart disease medications are no longer recommended and the treatment of TTC.

Nursing care should focus on education of TTC’s reversibility and also to identify long-term complications such as end-organ shock or thrombosis formation and subsequent Cox which can occur from the abnormal ventricular wall movement. Supporting the patient not only medically but emotionally can help with desire progression and lead to restoration of myocardial function.

Conclusion
Takotsubo cardiomyopathy is a complex condition. Although necessary, increased cost, patient stress, and superficial hospital use occur as the condition mimics acute coronary syndrome and must be treated as such until the diagnosis of TTC can be achieved. An excess in catecholamines produced by stress 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 cardiac shock as well as thrombosis formation. Emotional support along with antispasms therapy is recommended because the catecholamine levels decrease, the myocardium has proven to restore itself back to the pre-stress state of function. Healing a broken heart may be complicated, but with evidence-based treatment and direct nursing care, TTC is entirely reversible.

References

Figure 2: "Fireman’s hat" ST-segment elevation seen in ST-S and also in Takotsubo cardiomyopathy.