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Takotsubo Cardiomyopathy

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What Topic?

Takotsubo Cardiomyopathy (TTC)

- First discovered in Japan in the 1990's, TTC represents a transient type of left ventricular wall dysfunction.
- TTC gets its name from the shape that the left ventricle (LV) takes on during apical ballooning.
- Tatkotsubo comes from the Japanese words meaning octopus (takot) and jar (subo). (See figure 3)
- TTC has a frequency of 5.2 per 100,000 females and 0.6 per 100,000 males. (Agarwal et al., 2017)
- 2-3% of ST-segment Elevated Myocardial Infarction (STEMI) in women are actually TTC. (Sattar et al., 2020)
- TTC is associated with a physical or emotional trigger which stimulates a massive catecholamine release mediated by the sympathetic nervous system. (Chen & Dilsizian, 2017)

Why TTC?

Clinicians must differentiate TTS from other acute coronary syndrome disorders. Although TTC presents similar to an acute myocardial infarction, patients do not have evidence of ischemia on cardiac catheterization. (Sattar et al., 2020)

A recent systematic review and meta-analysis suggests a possible correlation with surgery and TTC. (Agarwal et al, 2017) Nurse anesthesia providers must include TTC in the differential diagnosis upon encountering acute coronary syndrome like events in the operating room.

Regardless of how TTC develops, Advanced Practice Registered Nurses must understand how diagnosis nuances may affect care. Although current treatment recommendations are similar to many Heart Failure treatment recommendations, certain TTC associated complications provide treatment nuances. (Sattar et al., 2017)

People who develop TTC may have long-term physical and structural impairment after LVEF recovery. (Scally et al., 2018) These changes may represent a new phenotype of heart failure. (Scally et al., 2018) Lastly, TTC disproportionately affects women which represent 90% of all cases. (Pellicca et al., 2017)

Understanding Pathophysiology

TTC often has an identifiable stress-producing event, physical or emotional, associated with onset. This event stimulates the sympathetic nervous system via the limbic system and induces excessive amount of catecholamine release. (See figure 1) Pelliccia, Kaski, Crea, and Camici (2017) note that emotional stress activates portions of the brain which increase the bioavailability of cortisol, epinephrine, and norepinephrine. Additionally, endothelial dysfunction is believed to contribute to microvascular coronary artery spasms in TTC through an imbalance of vasodilating and vasoconstricting factors. (Pellicca et al., 2017) Estrogen is believed to be an endothelial and cardiovascular protective hormone. (Akashi et al., 2008) Therefore, a lack of estrogen, present in post-menopause may explain why TTC is more common in post-menopausal women. (Pellica et al., 2017)(See Figure 2) Endothelial tissue secrets vasodilating and vasoconstricting substances which therefore influences myocardial perfusion. (Pellica et al., 2017)

Chen and Dilszian (2017) expand upon known pathophysiology and postulate what happens at a cellular level to further explain TTC pathophysiology.

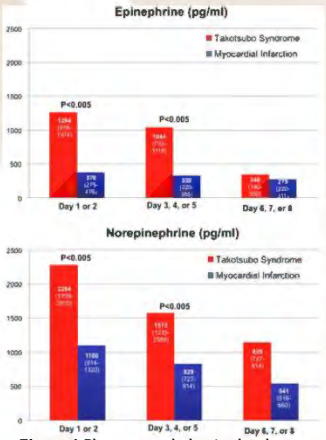

Figure 1 Plasma catecholamine levels in patients with TTC. (Massachusetts Medical Society, 2005)

Chen and Dilsizian (2017) describe two leading in-depth hypotheses of TTC pathogenesis. The first hypothesis is that there is a regional myocardial stunning due to a transient vascular dysfunction such as coronary vasospasm, microvascular dysfunction, or aborted myocardial infarction. (Chen & Dilsizian, 2017) Myocardial stunning is thought to be a metabolic adaptation that inhibits glucose utilization despite blood flow returning to normal. (Chen & Dilsizian, 2017) These metabolic alterations appear to be most pronounced during the acute phase of TTC and resolve during the recovery phase. (Chen & Dilsizian, 2017)

The second hypothesis for pathogenesis is that a cardiac sympathetic innervation or disruption occurs. (Chen & Dilsizian, 2017) Researchers utilize I-metaiodobenzylguanidine (mIBG) as a radiotracer analyzing cardiac sympathetic activity and notice a decrease in cardiac sympathetic innervation in patients with TTC. (Chen & Dilsizian, 2017) Researchers postulate that the abnormal epinephrine and norepinephrine serum concentrations could cause a switch in the Beta-2 receptors to initiate Gi coupling instead of normal Gs coupling. (Chen & Dilsizian, 2017) This temporary switch in theory allows myocytes to recover inotropic function. (Chen & Dilsizian, 2017)

Signs and Symptoms

- Chest pain (Agarwal et al., 2017)
- Dyspnea (Agarwal et al., 2017)
- Elevated troponin level (Agarwal et al., 2017)
- Electrocardiogram abnormalities (Chen and Dilsizan, 2017)
- ST-segment elevation (Agarwal et al., 2017)
- T-wave abnormalities (Agarwal et al., 2017)
- Less likely to see ST-segment elevation and T-wave abnormalities in perioperative TTC presentation (Agarwal et al., 2017)
- Apical rounding then basal and mid-ventricular contraction on left ventricle angiogram (Sattar et al., 2020)

Treatment Recommendations

Treatment of TTC is focused on acute management of symptoms. Sattar et al. (2020) highlight treatment recommendations for general management, TTC without complications, TTC with hypotension and cardiogenic shock, TTC with Heart Failure, and TTC with thromboembolism.

General Management (TTC without Complications) (Sattar et al., 2020)

- Emotional or physical stress management
- Dual antiplatelet therapy, discontinue upon discharge
- Angiotensin-converting enzyme inhibitor (ACE)/angiotensin receptor blocker (ARB)

TTC with Cardiogenic Shock (without left ventricular outflow tract obstruction (LVOTO)) (Sattar et al., 2020)

- Cautious fluid resuscitation
- Inotropic agent trial (dopamine or dobutamine)
- Intra-aortic balloon pump (IABP) for persistent hypotension or signs of end organ damage
- Vasopressors contraindicated in moderate to severe LVOTO

TTC with Cardiogenic shock and LVOTO (Sattar et al., 2020)

- Inotropic agents contraindicated
- Moderate to severe LVOTO-fluid resuscitation
- Beta blockers to improve LVOTO gradient
- May consider alpha agonist
- IABP considered with caution in patients not responsive to fluid resuscitation

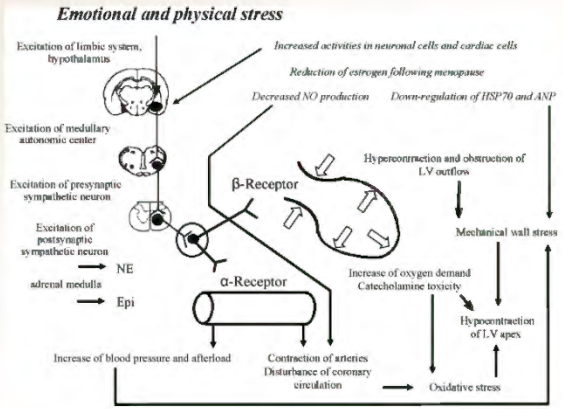

Figure 2 Proposed model of TTC pathophysiology (Yoshihiro et al., 2008)

Treatment Continued

TTC with Heart Failure (Sattar et al., 2020)

- Oxygen and respiratory support as needed
- Preload and afterload reduction only in cases without LVOTO
- ACE inhibitor/ARB
- Continue treatment until systolic function improves, about four weeks

TTC with Thromboembolism (Sattar et al., 2020)

- Ventricular thrombus present in 1.3% of patients with TTC
- Anticoagulation for three months in presence of LV thrombus
- No clear guidelines to prevent LV thrombus, ten days anticoagulation recommended

Long-Term Prognosis

Recent evidence suggests that TTC may have long-term complications despite LVEF recovery. Scally et al. (2017) report statistically significant variations in exercise capacity and metabolic performance, LV functional and structural variations, and cardiac inflammatory biomarkers among individuals recovered from TTC and those who have not had TTC. Scally et al. (2017) propose that their findings represent a new phenotype of heart failure secondary to TTC.

However, Scally et al. (2017) note further research must establish a causative relationship with their observed changes.

Nursing Implications

Advanced practice nurses (APRNs) must understand the pathophysiology of TTC to provide excellent care. Nurse Anesthetists in particular ought not exclude TTC in the differential diagnosis in a rapidly deteriorating patient.

Understanding the pathophysiology of TTC guides APRN's understanding of TTC treatment modalities.

TTC, unlike other cardiovascular pathologies, often has an emotional trigger. Soube et al. (2017) reported that 31.7% of patients in their study had an identifiable emotional trigger. Nurses are uniquely prepared clinicians who can blend medical diagnoses with caring practices. APRNs must holistically approach patients with a TTC diagnosis to ensure that care meets a patient's physical and emotional needs.

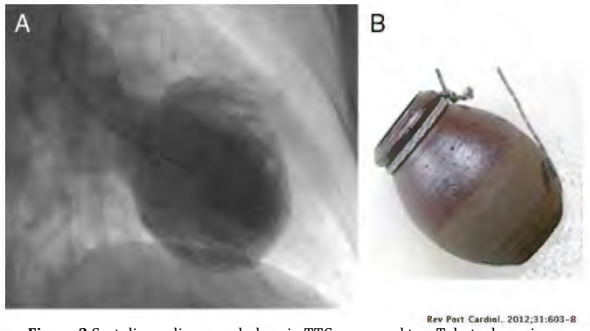

Figure 3 Systolic cardiac morphology in TTC compared to a Takotsubo or jar for capturing octopus (Cesario et al., 2012)

Conclusion

Takotsubo Cardiomyopathy is a recent discovery in cardiology. Because it presents like acute coronary syndrome yet acts like heart failure, clinicians must understand TTC's pathophysiology in order to provide appropriate treatment. (Sattar et al., 2017)

Unlike many cardiovascular pathologies, TTC often has a direct link to an emotional trigger. (Sobue et al., 2017) Care must include emotional support especially in instances where an emotional trigger is identified. (Sattar et al., 2017) Nurses are well prepared to offer excellent, holistic care to this population.

TTC needs further research to develop current understanding. This is most notable with long-term prognosis. TTC was once considered resolved after LVEF returned to normal. (Scally et al., 2018) However, recent literature suggests that there are long-term cellular metabolic variations associated with TTC. (Scally et al., 2018)

References QR Link

Additional Sources

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