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Japanese Octopus Traps & Broken Hearts: Takotsubo Cardiomyopathy

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Introduction

-Takotsubo cardiomyopathy (TTC) is a non-ischemic cardiomyopathy in which there is a "transient left ventricular systolic dysfunction, sparing the basal segments of the left ventricle" which results in an apical ballooning appearance of the left ventricle (LV) (Lesser, Maron & Sharkey, 2011).

-The name originates from the apical ballooning shape, found via left ventriculography or echocardiogram, that is similar to a Japanese octopus trap called "takotsubo" (Reeder & Prasad, 2015). Other common names include apical ballooning syndrome, stress-induced cardiomyopathy or broken heart syndrome (Reeder & Prasad, 2015).

Proposed Underlying Pathophysiology

Increased Catecholamine Levels

-The first includes increased levels of circulating catecholamines that cause decreased contractility and myocardial stunning (Pelliccia, Greco, Vitale, Rosano, Gaudio & Kaski, 2014). It is believed that increased catecholamines damage myocytes through cyclic adenosine monophosphate (CAMP) calcium overload and increased levels of free radiacls (Pelliccia, Greco, Vitale, Rosano, Gaudio & Kaski, 2014).

Coronary Artery Vasospasm & Microvascular Dysfunction.

-Another common hypothesis includes coronary artery vasospasm and coronary artery microvascular dysfunction producing the transient left ventricular dysfunction found in TTC (Liang, Cha, Oh, Prasad, 2013). Arterial vasospasm of large epicardial coronary arteries and microvascular coronary arteries are known to have the potential to cause significant cardiac disease as Prinzmetal's angina

Significance of Pathophysiology

-Ultimately, regardless of the cause treatment must be provided to avoid worsening of LV dysfunction, which can lead to ventricular arrhythmias, permanent ventricular dysfunction and/or death. Since TTC initially presents in a similar fashion as ACS, the treatment of the patient should follow ACS guidelines initially until ACS can be ruled out and TTC made. Frequently this involves coronary angiography, either emergently or urgently, to rule out obstructive coronary artery disease (Scantlebury & Prasad, 2014).

Treatment

-Fortunately, conservative therapy that mimics general heart failure treatment is successful at resolving most cases of TTC (Reeder & Prasad, 2015). Due to the lack of a unified, accepted theory for the underlying pathophysiology of TTC a large amount of treatment is supportive in nature, attempts to avoid the provocation of proposed causes and mirrors treatment for heart failure. No evidence based treatment currently exists for TTC (Reeder & Prasad, 2015).

-If profound shock is present treatment may include mechanical therapy, such as insertion of an intra-aortic balloon pump (IABP), or inotropic therapy if necessary.

-Ideally inotropes should be avoided given a surge in catecholamines is theorized to be one of the causative factors of the disease (Reeder & Prasad, 2015).

-Initially beta blockers, angiotension-convering enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) and diuretics should be used to treat LV dysfunction (Kono & Sabbah, 2014).

-Anticoagulation should be used in severe cases of LV failure to prevent thromboembolism (Reeder & Prasad, 2015) which could cause stroke or other end-organ ischemia.

-Beta blockers have been used in some patients after recovery of LV function due to their catecholamine-inhibiting nature however there is no conclusive standard of care on TTC treatment (Reeder & Prasad, 2015).

-ACEIs/ARBs, diuretics and anticoagulation should be discontinued after LV recovery unless there are other concomitant diseases requiring their use (Andrade & Stainback, 2014).

Summary of Treatment

-Conservative therapy that mimics general heart failure treatment
-Profound shock treatment → IABP & inotropic therapy if necessary
-Avoidance of inotropes if possible due to their possible role in the disease
-Beta blockers, ACEIs or ARBs and diuretics for LV dysfunction
-Individualized discontinuation of treatment with recovery

-Table 1 content taken from Scantlebury & Prasad, 2015

Mayo Clinic Criteria for Takotsubo Cardiomyopathy
1. Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid-segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular 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 and myocarditis

Pathophysiology

Signs & Symptoms

-TTC frequently mimics a ST-segment elevated myocardial infarction (STEMI) and/or acute coronary syndrome (ACS) with acute chest pain, dyspnea, syncope and/or signs of heart failure (Reeder & Prasad, 2015) with severe cases presenting in cardiogenic shock (Liang, Cha, Oh & Prasad, 2013). Treatment for TTC is generally supportive, with the disease being predominantly benign in nature, though some individuals presenting with severe symptoms experience high mortality rates (Liang, Cha, Oh & Prasad, 2013).

-The onset of the disease is often caused by an intense emotional stressor such as the loss of a loved one, an interpersonal conflict or a catastrophic medical diagnosis (Sharkey, Pink, Lesser, Garberich, Maron & Maron, 2015) but can have physical triggers such as infection, surgery or respiratory failure, while some have no obvious inciting events or diseases. Women, particularly post-menopausal women, are at a much greater risk than males for developing TTC with approximately 90% of cases occurring in females (Minhas, Hughey & Kolias, 2015).

-The similarity in symptoms is both a blessing and a curse as patients who experience the aforementioned symptoms typically seek medical attention relatively quickly as they often associate the symptoms with a heart attack. These same symptoms can easily be interpreted as ACS or a STEMI by healthcare providers, as there is no unique sign or symptom to distinguish between the two differential diagnoses, so individuals presenting with similar symptoms are often treated alike until proven otherwise (Scantlebury & Prasad, 2014).

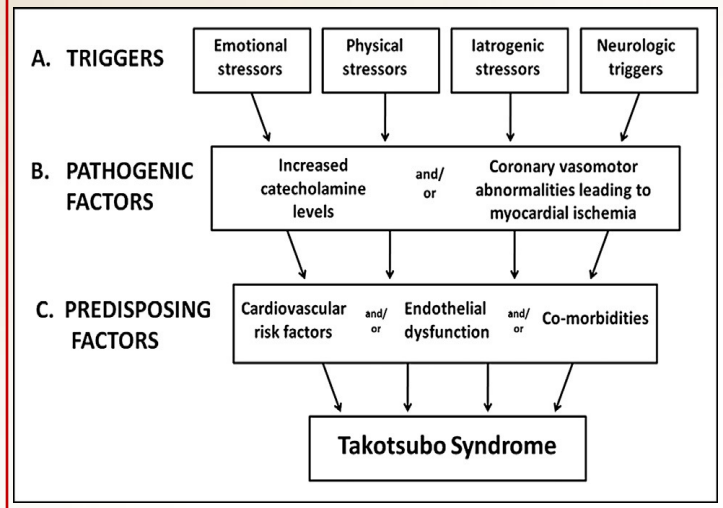


Figure 1 Pelliccia, Greco, Vitale, Rosano, Gaudio, & Kaski, 2014

Diagnostic Tests & Features

-Typical cardiac diagnostic tests for ACS, STEMI and TTC frequently share similar results, including ST-segment changes on EKG, elevated cardiac biomarkers and wall motion abnormalities on echocardiogram.

-Emergent or urgent coronary angiography is frequently required to exclude the diagnosis of ACS or STEMI as delays in treatment for these diagnoses can lead to great increases in morbidity and mortality (Hafiz, Naidu, DeLeon, Ismal, Alkhatib, Lorenz, E'Elia, Rosenthal & Marzo, 2013).

-A diagnostic feature of TTC found on coronary angiography and echocardiography is wall motion abnormalities of the LV mid-segments with or without apical involvement that extend beyond a single coronary artery distribution (Scantlebury & Prasad, 2014).

Implications for Nursing Care

Monitoring

-In the acute care setting, care for patients with TTC should include close hemodynamic and electrocardiographic monitoring as shock and malignant arrhythmias are known to occur, and are the biggest contributors of death from TTC (Liang, Cha, Oh, Prasad, 2013).

Treatment

-Administration of appropriate medications ACEIs/ARBs, diuretics, beta blockers and possibly anticoagulation should be provided to promote recovery of LV function (Andrade & Stainback, 2014).

Trigger Identification & Avoidance

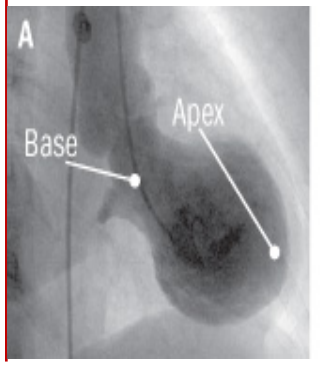
-A thorough history and assessment may provide clues to a possible emotional or physical trigger, if one exists (Inggs & Christensen, 2015).

-If a trigger is identified education specific to avoiding that trigger may prove beneficial, as 1 in 20 patients can suffer a recurrence of the disease (Lesser, Maron & Sharkey, 2011).

Education

-Medication and disease education should be provided to improve compliance with treatment to help increase the chances of a full recovery (Lesser, Maron & Sharkey, 2011).

-Education regarding anginal symptoms (Pore & Burley, 2012) should be provided as TTC patients could experience occlusive coronary artery disease in the future and they should not omit seeking treatment because they have had or are recovering from TTC.



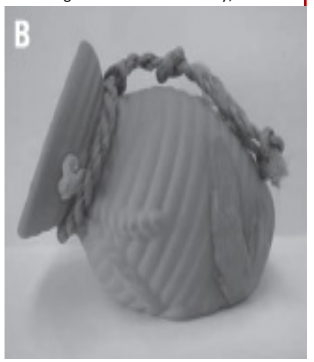


Figure 2 Harvard University, 2016

Conclusions

-While TTC remains a relatively benign condition with a good prognosis for complete recovery, the three-fold increase in recognized cases between 2007-2012 demands awareness of the condition (Minhas, Hughey & Kolias, 2015).

-While most cases carry low mortality rates, cardiogenic shock and cardiac arrest can accompany TTC in severe cases contributing to increased mortality rates in certain cases (Liang, Cha, Oh & Prasad, 2013).

-This varied presentation of the disease can make the diagnosis of TTC difficult, with a diagnosis typically made after invasive cardiovascular testing is complete (Reeder & Prasad, 2015).

-While both increased levels of circulating catecholamines and coronary artery vasospasm/dysfunction are strong theories for the cause of TTC a definitive cause is yet to be identified (Lesser, Maron & Sharkey, 2011).

-Interestingly TTC is usually triggered by a physical or emotional stressor in the patient's life and occurs predominantly in females, though mortality rates in males can be up to four times higher than females (Khera, Light-McGroary, Kahr, Horwitz & Girotra, 2016).

-Treatment primarily mirrors that of congestive heart failure and can usually be discontinued after return of LV function (typically within weeks of condition onset) with recurrence rates of the condition around 5% (Lesser, Maron & Sharkey, 2011).

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