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Case Report

Snowballing, Flaccid and Flabby Myocardium in the Framework of Bacteremia: Exploration of This Perplexing Conglomeration

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Abstract

Introduction:

Takutsubo (TTS) cardiomyopathy is a syndrome characterized by apical ballooning of left ventricle along with transient hypokinesis. It is also referred to broken heart syndrome or stress cardiomyopathy. Although physical/emotional stressful event usually the most common predisposing factor, its eventuation in the context of ESBL bacteremia has not been reported in the literature. This outlandish relationship is presented in this clinical case and further debunked to assess its clinical significance.

Clinical Case:

77-yr-old female with PMH of CAD s/p stent, PAF, DM, CHF, GERD, HTN and HPL presented to the ER with chest pain and shortness of breath. Work up showed total CK:209, troponin :1672.9, & BNP: 12,700. Urinalysis revealed leukocyte esterase positive UTI. Echocardiogram uncovered severe global hypokinesis with sparing of the basal myocardium and EF = 25%. Physical examination showed + JVD, bilateral inspiratory crackles and irregularly irregular rhythm. CT chest showed pulmonary edema and pneumonia. She also developed acute hypoxic respiratory failure secondary to UTI-sepsis, and was transferred to ICU for further evaluation and management.

Treatment:

In ICU, she was managed with IV phenylephrine, IV antibiotics, IV diuretics (Vancomycin & Cefepime), and beta-blockers. As culture results came back positive as ESBL E. coli, Klebsiella pneumonia, Cefepime is replaced with Ertapenem (14 days). After 5-days, her symptoms resolved. Echocardiogram 5 weeks later revealed a moderately dilated left atrium with normal LV Function, EF 55%.

Takehome Message:

This clinical case emphasizes the unusual relationship between sepsis and TTS cardiomyopathy in elderly patients. Emotional stress, inflammation, estrogen deficiency and endothelial dysfunction are the prevailing risk factors. Spasmodically, sepsis comes into play where cytokine induced myocardial depression preponderates, thus paving the way for TTS. Elderly female presenting with chest pain and shortness of breath should undergo EKG, coronary angiography and echocardiography. Empiric IV antibiotics followed by culture specific antibiotics to trample the source of infection can potentially tweak the disease course of TTS. Apart from this, conservative management with GDMT, calcium channel blockers and anticoagulants should result in symptomatic relief within few days as disease process runs its due course and fizzles out. Efforts should be made to limit the use of cardiotoxic drugs that can stonewall the conventional disease process and digress from faster recovery of cardiac function. Follow up with serial imaging studies is recommended to monitor regional wall abnormalities, ejection fraction, valvular dysfunction and thrombus formation.

Keywords: bacteremia; sepsis; ballooning of left heart; hypokinesis; congestive heart failure; takutsubo cardiomyopathy and atrial fibrillation

Introduction

Stress cardiomyopathy or broken heart syndrome is represented by ballooning of the apical myocardium along with regional wall motion abnormalities [1]. According to American College of Cardiology, broken heart syndrome is classified as primary acquired cardiomyopathy[2]. Broken heart syndrome is conceived from the fact that severe emotional stressful event is galvanizing event for engendering myocardial damage in older age women[1]. It is crucial to comprehend the fact that, stress induced catecholamine surge in the blood is primarily accountable for provoking the myocardial wreckage in TTS[3]. Uncommonly, intracranial disorders such as stroke, anxiety and depression can stimulate brain-heart axis, instigate autonomic dysfunction and spawn myocardial stunning, there by laying the foundation for TTS[4]. Occasionally, sepsis can be encountered as an etiological factor, when cytokine burst can directly exert toxic effects on the heart muscle and incite TTS. TTS is a diagnosis of exclusion and there should be absence of coronary artery disease, atherosclerosis, myocarditis, dissection or coronary spasm. TTS is a reversible cardiomyopathy, where culmination of its typical clinical course results in recuperation of cardiac pump function to its physiological state within days to weeks[5]. Consequently, management of heart failure along with treatment of associated comorbidities would result in symptomatic resolution. It goes without saying that some clinical cases deviate from predictable trajectory, and thus endure high-speed disease progression and eventually suffer cardiac or extra-cardiac complications. It would be prudent to monitor these patients with serial echocardiography scans to detect recurrence after discharge.

This case is unique in the sense that, an unfathomable combination of sepsis and TTS has been rarely reported in the literature. By presenting this case, we would like to lay emphasis on this unlikely collaboration, so that clinicians will have heightened awareness for prompt evaluation and management, thus opening the doors for improved prognosis as well as optimal clinical outcomes.

Clinical Case

A 77-year-old white female with a past medical history of coronary artery disease with a history of LAD stent in 2010, paroxysmal atrial flutter, diabetes, diastolic CHF, paroxysmal atrial fibrillation, GERD, GI bleeding, iron deficiency anemia, hypertension, and hyperlipidemia presented to the ER with complaints of chest pain and shortness of breath. She states that over the last week, she has felt short of breath and weak with exertion, and last night she became acutely short of breath and felt her heart racing too fast. The patient arrived on supplemental oxygen at an 8L nasal cannula. Due to increased work of breathing, the patient required initiation of BiPAP. The patient has a history of atrial fibrillation, which is treated with sotalol. She is not chronically anticoagulated due to a recent GI bleed and was planned for a Watchman device. She received a dose of furosemide in the ER and was transferred to the ICU. She is currently in atrial flutter with a controlled rate on amiodarone.

Vitals : Temp: 38.0 °C HR: 149 RR: 40 BP: 100/56 SpO2: 100% on BiPaP 16/6 Fio2 60% Neck: Supple. + JVD; Cardiovascular: +Irregular rate and rhythm; Respiratory: Lungs with basilar crackles bilaterally. Labs: WBC 15.89, hemoglobin 8.6, hematocrit 31.4, creatinine 0.7, potassium 4.6, lactic acid 5.21, total CK 209, troponin 1672.9, BNP 12,700 Urinalysis: leukocyte esterase large, nitrite positive, blood large, bacteria. CT Chest: Mild smooth interlobular septal line thickening in both lungs, consistent with mild interstitial pulmonary edema. Patchy Tree-in-bud opacities, ground-glass, and consolidation throughout both lungs, consistent with pneumonia. **Echocardiogram 2 months ago:** Mild MR, mildly dilated left atrium, mildly increased LV wall thickness, estimated EF of 60%. Echocardiogram today: Moderately dilated left atrium. There is severe global hypokinesis with sparing of the basal myocardium suggestive of Takutsubo cardiomyopathy, Estimated EF = 25%.

Patient was managed in the ICU with acute hypoxic respiratory failure secondary to Urosepsis and CHF Exacerbation, initiated on pressors with IV phenylephrine, antibiotic coverage with IV cefepime and vancomycin. Urine cultures: ESBL E. coli, Klebsiella pneumonia. Cefepime discontinued. Started on ertapenem based on sensitivities. Patient was managed with watchful diuresis for decompensated heart failure and remains in rate-controlled atrial fibrillation with beta-blockers. After 5 days of symptomatic management, the patient denied shortness of breath upon discharge. Oxygenating well on room air. Follow-up Chest x-ray: no focal consolidation, pulmonary vascular congestion, or changes. Patient transitioned to oral diuretics. Midline was placed before discharge. Plan to continue ertapenem for a complete course of 14 days. Echocardiogram 5 weeks later revealed a moderately dilated left atrium with normal LV Function, EF 55%.

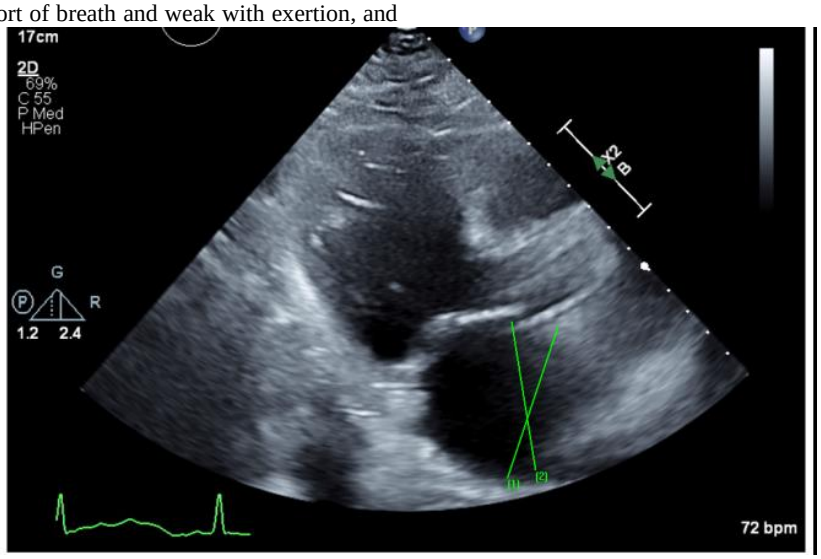

Figure 1: Echocardiography on admission: Moderately dilated left atrium. There is severe global hypokinesis with sparing of the basal myocardium. Estimated EF = 25%. Previous echocardiography 2 months ago showed Mild MR, mildly dilated left atrium, mildly increased LV wall thickness, estimated EF of 60%.

Discussion

We present a case where 70-year-old with PMH of CAD, PAF, DM, & CHF presented with chest pain and shortness of breath. Workup including CXR [pneumonia], urine analysis [ESBL E.Coli & Kliebsella UTI] and echocardiography [global hypokinesis and EF 25%] were promptly performed. Patient is managed with IV antibiotics, diuretics and beta-blockers for UTI, CHF and AFIB respective. After 5 days of symptomatic management, the symptoms resolved and patient is discharged. Echocardiography after 5 weeks showed normal LV function with EF 55%. This case represents a classic case of Takutsubo cardiomyopathy (TTS) in the setting of UTI-sepsis, which underwent spontaneous resolution within 5 weeks.

The TTS syndrome was first recognized in Japan in 1990, and name is derived from the octopus trap like pot used to catch octopuses by Japanese fisherman [6]. After its identification in Japan, first case of TTS presenting with chest pain, ECG and cardiac enzymes has been in detected in United States in 2003[7]. Emotional and physical stressful events as harbinger for TTS has been documented in 26.8% and 37.8% cases[8]. The most common age of presentation reported is 67.7 years[9]. It has more predilection to occur in females as compared to males [9:1][10]. There are some racial differences in susceptibility for TTS, with highest in Asians (57.2%) followed by Caucasians (40%) and other races (2.8%)[9].

In evaluation of 199,890 patients with TTS, 83% of them were older age postmenopausal women, with most of them ending up with complications including atrial fibrillation [20.7%], cardiac arrest [3.4%], congestive cardiac failure [35.9%], cardiogenic shock [6.6%] and stroke [5.3%][11]. The incidence of TTS was documented to be around 1-2% of acute coronary syndrome[12]. Out of all the patients admitted to hospital with acute coronary syndrome, TTS has been found in 2% of the cases[13]. With no specific therapies available to abort the disease progression, patients who come through unscathed during first episode of TTS, have higher recurrence at 6 months [1.2%] and 6 years [5%][13].

In 75-80% percentage of cases, there is apical ballooning and hypokinesis of the left ventricle, however atypical cases including biventricular, basal and focal are occasionally encountered[14, 15]. The clinical criteria for diagnosis of TTS includes occurrence of chest pain/shortness of breath following a severe stressful event with spilling of troponin into the blood circulation, ST-elevation/T-wave inversion, left ventricular dysfunction, regional wall motion abnormalities, hypokinesis of the left ventricle, and absence of coronary artery disease, myocarditis or pheochromocytoma[14]. Occasionally, right ventricular wall motion abnormalities are encountered, with apico-lateral, anterio-lateral and inferior areas effected along with pleural effusion[16].

Studies indicate that TTS has more proclivity to occur in females as compares to males, with the mean age of presentation being around 66 years [10]. TTS has been noticed in 2% of the ACS patients under coronary angiography in which ejection fraction has been suddenly relegated to less than 30%[17]. TTS has been in witnessed in 0.02% of hospitalizations in postmenopausal women with history of smoking, alcohol and hyperlipidemia[18].

TTS has the tendency to develop in patients with pre-existing cardiovascular disorders who are threatened with stressful scenarios. To shed light on these stressful situations, clinical researchers delineated that excessive emotional/physical stress [domestic abuse, death of a family member, financial loss & new medical diagnosis], estrogen deficiency, microvascular dysfunction, inflammatory disorders, spasm of coronary vessels, impaired myocardial fatty acid metabolism and aborted myocardial infarction [1, 19, 20]. Furthermore, other risk factors such as type D personality disorder, anxiety and depression are also documented in the literature. These subset of patients have heightened sympathetic drive and lower threshold for firing of sympathetic neurons in response to stressful events, thus making them best suited to establish TTS [21]. In these patients, neuro-cardiogenic stunning can spring up, where autonomic dysfunction provoked by neuropsychiatric disorders can catalyze left ventricular dysfunction, ECG changes and spilling of cardiac enzymes – reminiscent of TTS [3, 22]. Very rarely, consumption of sushi has been documented to instigate broken heart syndrome[23].

A TTS like syndrome is had been documented in clinical scenarios such as sepsis neurological conditions [seizures, stroke, transient ischemic attack, tumors, traumatic brain injury], pheochromocytoma, cancer, asthma, COPD, endoscopy, cardioversion, drugs [Cocaine, amphetamine, dopamine, dobutamine, epinephrine, and norepinephrine] and anesthesia [1, 24, 25]. Higher incidence of TTS in postmenopausal women can be attributed to estrogen deficiency. Estrogen loss lays the foundation for increased proclivity for developing TTS in these women due to loss of cardiac protective function offered by estrogen. To further enlighten this cardio-protective function, estrogen greases the wheels for sustenance of vasodilation while at the same time offering immunity against atherosclerosis and endothelial dysfunction[19]. Keeping that in mind, it is not surprising that postmenopausal women are more susceptible to bear the brunt of increased vasoconstriction, endothelial dysfunction in the setting of emotional stressors[26].

As the emotional stressors occur, they are sensed by the neocortex and limbic system (hippocampus and amygdala) which sends afferent fibers to the locus coeruleus located in the rostral pons. As locus coeruelus is activated, it then stimulates the hypothalamic-pituitary-adrenal axis and posterior hypothalamus, which becomes the primary motivation for adrenal medullary secretion of epinephrine and norepinephrine into the blood stream. The sympathetic nerve terminals in the heart muscles and coronary circulation release epinephrine and norepinephrine, thus activating the α and β-adrenergic receptors in the heart. Studies have shown that there is increased circulating levels of catecholamines along with increased concentration of the catecholamines at the myocardial interface [27, 28]. This latter finding can be explained by a synergic effect of enhanced exocytosis from the presynaptic cleft along with decreased reuptake from uptake transporter [29]. Studies report that, levels of epinephrine, norepinephrine and dopamine levels can reach as high as 3 times above upper limit of normal [27]. As this cathecholamine surge transpires, they provoke beta-2 adrenargic receptor transmutation, where Gs [stimulatory type of G-protein] is revamped into Gi [inhibitory type of G-protein], due to which negative inotrophy can be the final consequence [30]. On top of these, enhanced sarcolemmal permeability, free radical overproduction, decreased anti-oxidant synthesis, calcium overloand and enhanced lipid mobility might also contribute to bolster the myocardial injury[31]. Studies have shown that excess stimulation of beta-1/2 adrenergic receptor induced catecholamine surge has resulted in widespread cardiomyocyte death with sparing of the cardiac stem cells as well as myocardial insulin resistance[9, 32, 33] These cellular changes can be the radical stimulus for eventuation of left ventricular dysfunction in TTS.

Catecholamine surge in the myocardial milieu can provoke endothelial dysfunction, which is more pronounced in estrogen deficient postmenopausal women exposed to severe emotional/physical stress[34]. This can be harbinger for inciting imbalance in vasocontriction and vasodilation in the coronary vasculature, an underlying factor for triggering myocardial ischemia and transient left ventricular dysfunction in TTS patients [29]. To corroborate these findings, estrogen supplementation in animals blunted the stress induced hypothalamic-pituitary-adrenal axis induced sympathetic outflow, spiking in blood pressure& heart rate, attenuated apical LV ballooning and upregulated the cardio-protective

peptides including atrial natriuretic peptide and heat shock protein-70 (HSP-70)[20, 35].

Excess catecholamines in the blood can increase heart rate and contractibility, due to which myocardial ischemia can be the culminating by-product secondary to supply-demand discordance [36]. Over and above that, catocholamine surge in blood circulation might set in motion a cluster of metabolic changes in the heart muscle including lipid heaping in cardiomyocytes, uncoupling of oxidative phosphorylation, electrolyte depletion, mineral deficiencies [Zinc & Selenium] and free radical toxicity[37, 38]

Direct release of catecholamines from the sympathetic nerve terminals onto the surface of myocardial fibres an trigger contract band necrosis due to cAMP mediated calcium overload which is characterized by interstitial inflammatory infiltrate, dense eosinophilic transverse bands and hypercontracted sarcomeres[39]. As compared to pheochromocytoma, TTS has less severe catocholamine elevation, which gives rise to less cardiac damage, mild spiking of cardiac markers and lack of late enhancement in cardiac magnetic resonance imaging.[40, 41]. A significant proportion of patients were also found to have coronary spasm and microvascular dysfunction, a synergistic and lethal combination that catalyzes the progression of cardiac dysfunction in TTS [42-44].

In our patient, sepsis induced TTS is the likely clinical scenario. It is been speculated that, sepsis induces TTS though multifarious mechanisms including, increased coronary circulation release of pro-inflammatory cytokines (TNF-α, IL-1 & IL-6) triggered myocardial slowing through nitric oxide, reduced calcium sensitivity of myocardium and mitochondrial dysfunction[45].

In a typical clinical profile, patients are exposed to very stressful event such as major life event, earthquake, or COVID-19 pandemic before the symptoms comes to light [1, 14, 46]. However, not all patients fit in this category, as some patients might have low-level stressful event that has been going on for quite a bit of time. Nevertheless, with a catastrophic stressful event, superfluous cathecholamine surge in the blood stream triggering cardiac injury will precede the symptom onset. Resultantly, patients without typical cardiac risk factors present with symptoms mimicking acute coronary syndrome such as chest pain, dyspnea[1, 46].

It is not unforeseen that the disease progression marches forwards swiftly in few vulnerable patients, thus TTS exposes itself with severe manifestations such as heart failure, cardiogenic shock, tachyarrhythmias, bradyarrhythmias, mitral regurgitation, cardiac arrest[1]. Apical ballooning of the left ventricular apex gives rise to excess pooling of blood, a water shed movement that sets in motion ominous events starting with stagnation, activation of pro-coagulant pathways and deactivation of anti-coagulant pathways, where synergistic intersection of these pathways ultimately culminates in thrombus formation. This LV thrombus being motile, can be thrusted forward in the direction of blood flow, thus showing heightened propensity to embolize into the brain circulation. These antecedent events make the patient more prone to present with transient ischemic event or stroke[1].

The diagnosis of TTS can be performed by various tests including cardiac biomarkers, EKG, echocardiography, coronary angiography and magnetic resonance imaging. It goes without saying that, TTS is officially diagnosed when reversible systolic dysfunction that occurs following stressful event gradually pulls through and rehabilitates to near normal function, without evidence of coronary spasm, dissection, atherosclerosis or thromboembolism[47, 48]. Although it is been argued that rejuvenation occurs over 6-8 weeks, it usually does not follow any specific time frame with case-by-case variation more evident.

On the account of ventricular dilation, stretching and mild-moderate necrosis, there can be elevation of cardiac markers including creatinine kinase-MB [CKMB], troponin, brain natriuretic peptide [BNP] and N-terminal inactive molecule [NT-proBNP] [20]. The amount of release of cardiac enzymes into the blood is usually proportional to the extent of cardiac muscle injury and pump dysfunction. The levels of troponin elevation is usually at a lesser magnitude as compared to myocardial infarction due to presence of mild or absent myocardial necrosis in TTS [49, 50]. In contraposition, the intensity of BNP elevation is generally greater than myocardial infarction secondary to omnipresence of higher amplitude of myocardial stretch and chamber dilation in TTS [51, 52].

As TTS presents with similar clinical presentation as acute coronary syndrome, EKG changes that comes to light can range from ST elevation, T wave in version and left bundle branch block [53, 54]. Some clinical researchers categorized into three stages namely stage I [ST elevation] in initial few hours, stage II in 1-6 days [T wave inversion, QTc prolongation, Torsade de pointes and ventricular tachycardia] and stage III [Resolution of T wave and QTc changes] in weeks to months[46, 53, 54]. Apart from these, J wave, fragmented QRS and low-voltage QRS complexes are occasionally encountered, although not common [46, 55].

Echocardiography is usually performed can be useful in delineating location [apical, biventricular, basal or focal], extent, severity, presence of thrombus, left ventricular outflow tract obstruction, pericardial effusion, right ventricular involvement, and mitral regurgitation[1, 19, 46]. By the way of explanation, myocardial edema and increased LV wall thickness are primarily responsible for bringing to pass NSTEMI related wall motion abnormalities[56].

Examination of 145 patients between 1998-2012 revealed that apical ballooning along with hypokinesis is present 90% patients, with most of them having apical nipple sign[57]. In conjunction with this, there is well-conserved small territory of contractile activity located in the most apical portion of left ventricle, labelled as apical nipple sign[57]. As TTS closely mimics ST elevation myocardial infarction, presence of apical nipple sign can be a striking distinguishing feature between these two clinical entities[57].

Cardiac magnetic resonance imaging (cMRI) with gadolinium contrast might offer clues to the diagnosis of TTS by unearthing a fibrotic band at the junction of hyperkinetic and hypokinetic ventricular wall, a culmination that unfolds due to conflicting contractile forces impinging on the ventricular wall[46]. Moreover, myocardial edema, right ventricular involvement, efflux of collagen into the extracellular matrix, and pericardial effusion are also not uncommon findings[46, 56]. cMRI can be utilized for drawing a distinction for broken heart syndrome from apical hypertrophic cardiomyopathy by prolonged T1 mapping times, absence of late gadolinium enhancement and extracellular volume bulging to collagen accumulation[56].

Histopathological examination of myocardium in TTS might reveal contraction band necrosis, myocardial lysis, focal fibrosis and inflammatory infiltrate based in the stage and severity of the disease process [1, 19].

Since TTS presents with similar symptoms with acute coronary syndrome, an initial coronary angiography should be performed to rule our thrombosis, embolism, atherosclerosis, spasm or dissection[46]. In the absence of these abnormalities, the presence of non-infarct pattern on cardiac MRI points towards TTS[46]. In this clinical scenario, (GDMT) guideline directed medical therapy including aspirin, beta-blocker, ACE inhibitor, calcium channel blockers, and lipid lowering agent should be prescribed for 3-6 months in hemodynamically stable patients [1, 9, 19, 58]. In a small subset of patients who have a history of thromboembolism or thrombus, supplementing anticoagulation therapy should improve therapeutic

outcomes[1]. As QTc prolongation is a more to develop during hospitalization, a cautious prudence warrants avoiding QTc prolonging drugs and regular ECG monitoring[59, 60].

Resurrection of cardiac enzymes, EKG changes and LV function usually takes placed within few days to few weeks[9]. On top of this, therapy should be tailored towards treatment of congestive heart failure, and cardiogenic shock, associated with TTS, which might entail inotropes, intra-aortic balloon pump, and left ventricular assist devices [1, 14]. As the patients gets stabilized, upon discharge, these patients should be advised to undergo serial cardiac imaging studies to evaluate ejection fraction, regional wall motion abnormalities, and thrombus formation[1].

Although TTS is self-limiting and resolves within few weeks, some patients have protracted clinical course, thus making them liable to complications. With that in mind, hypotension, right ventricular involvement with pleural effusion, atrial/ventricular tachyarrhythmias, mitral regurgitation, left ventricular outflow tract obstruction, left ventricular mural thrombus, rupture of ventricular thrombus polymorphic ventricular tachycardia, torsade de pointes, cardiac arrest, and systemic thromboembolism are reported in the literature[14, 46]. Resultantly, a part of management entails delivering symptomatic therapy directed towards these complications.

Patients with TTS are deemed to have favorable prognosis as near normal recovery of cardiac function is a universal phenomenon rather than an exception. Efforts should be made to limit the use of cardiotoxic drugs that can stonewall the usual disease process and digress from faster recovery of cardiac function. With being said, it is conceivable that 95% of the patients recuperate to regain their LV pump function within 6-8 weeks[61]. However, few patients experience recurrence of 1-2%, thus increasing their mortality and morbidity[14, 62]. In those where disease process marches forward swiftly, there is increased propensity of mortality 2-3% [20]. According to Kashou, A.H. et al broken heart syndrome should not be presumed be a benign disorder, rather a profound pathological disorder where short-term myocardial dysfunction during the first hit might kick off concealed shattering of ventricular electrical conduction system, thus setting the stage for ventricular arrhythmias during second hit in the near future[63].

In patients with severe TTS, complications that can expected can range from cardiogenic shock, congestive heart failure, cardiac arrest, respiratory arrest, acute renal failure, stroke, TIA and sepsis [10, 64, 65]. Acute neurological disorders, male sex, right ventricular involvement, moderate-to-severe mitral regurgitation, EF < 45%, very high BNP levels, and troponin (> 10-fold) are considered as harbingers for worse clinical outcomes and prognosis[4, 66, 67]. Specifically, the all-cause mortality and adverse cardiovascular events occur in 5.6% and 9% cases [19]. Clinical case studies suggest that even in cases who completely recover from first episode of TTS can experience lingering chest pain, lethargy and shortness for a substantial period (>2 years), a prognostic factor that is associated with long-term morbidity and mortality[46].

Take-home messages

- Initially identified in Japan, TTS procured its pseudonym from the octopus trapping pot used by Japanese fisherman.
- It is more liable to develop in postmenopausal women at older age due to their lack of estrogen.
- It is portrayed by ballooning of the apical myocardium consorted with transient hypokinesis or dyskinesis. There is no associated coronary artery disease or atherosclerotic vascular narrowing.
- Apical form is most common type seen, while biventricular, basal, focal forms less commonly seen.
- Triggering factors including severe physical/emotional stressful event, inflammation, myocardial stunning, sepsis, microvascular dysfunction, and estrogen deficiency were implicated.
- In sepsis patients, TTS can be an unexpected finding, whose revelation can be unfolded by myocardial depression secondary to cytokines, altered coronary circulation, mitochondrial dysfunction, and attenuated calcium sensitivity.
- Excess catecholamine surge from sympathetic nerve terminals infringing directly on the heart muscules will ravage the cardiomyocytes, sarcolemma, and intercalated discs, a turning point that sparks off contraction band necrosis.
- Its clinical presentation closely mirrors coronary artery disease, thence patients present with chest pain and dyspnea.
- Nevertheless, in some vulnerable patient's disease process takes a quantum leap, thus bringing to light complications such as cardiogenic shock, thrombus and left ventricular outflow tract obstruction.
- As the diagnosis is suspected, cardiac markers, EKG, echocardiography, coronary angiography and cardiac magnetic resonance imaging can be quite resourceful.
- In clinically stable patients, GDMT for management of heart failure would be therapeutically beneficial, with anticoagulants reserved for those with LV thrombus formation.
- Empiric IV antibiotics followed by customization to culture specific antibiotics for sepsis would be life-saving and would potentially alter the disease course of TTS.
- Serial imaging studies at regular intervals to monitor regional wall motion abnormalities, pump function and thrombus formation needs to be undertaken given the recurrence rate.
- In patients with severe complications, increased mortality and morbidity can be expected.

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Consent for publication: Consent taken

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