Chronic Stress resulting in ‘Broken Heart’ Syndrome
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Citation

Abstract
Broken heart syndrome/ tako-tsubo cardiomyopathy is a recently described stress-induced cardiomyopathy that is often associated with symptoms suggestive of acute coronary ischemia including chest pain, ST changes and elevated cardiac enzymes. In most cases the syndrome is triggered by profound physical or psychological stress and has an increased incidence in post menopausal women. Here we describe a patient who was admitted with nonspecific abdominal pain and symptoms of ileus that went onto develop Tako-tsubo cardiomyopathy as confirmed by left ventriculography. Unlike previously reported cases, our patient appeared to develop the syndrome as a result of chronic rather than acute stress.

CASE REPORT
A 64 yrs old Asian female presented to the emergency room complaining of a cramping abdominal pain of 6 hour duration. Other than diffuse abdominal tenderness without rebound tenderness, physical examination was unremarkable. Further workup revealed a WBC count of 14800 cells/cm$^3$ and abdominal x-ray showing a mild ileus. EKG and cardiac enzymes were within normal limits. The patient was admitted for observation but within 3 hours the patient developed retrosternal chest pain that radiated into her neck and left arm. Electrocardiogram showed ST-elevation in V1 and V2 and cardiac enzymes were elevated including a troponin of 1.14ng/mL leading to emergency cardiac catheterization.

Although catheterization showed completely normal coronary arteries with no culprit lesions, left ventriculography revealed a severely reduced ejection fraction with diffuse aneurysmal dilation of the mid and apical ventricular segments consistent with tako-tsubo cardiomyopathy (Figure 1). An in depth interview of the patient revealed that she had been under increased stress over the last several months due to the death of a parent.

The patient received supportive care as well as aspirin, beta blocker, ACE inhibitor and statin therapy. She was discharged home on the third day of her admission in good condition and was advised to continue on pharmacological treatment and follow up for repeat echocardiogram in 2 months.

DISCUSSION
Tako-tsubo cardiomyopathy, also known as stress-induced cardiomyopathy and “broken heart syndrome”, was first described in 1990 by Sato et al and is becoming increasingly recognized as an acute cause of cardiac dysfunction. The classic clinical presentation is similar to that of an acute myocardial infarction with substernal chest pain, ST elevations in the precordial leads and modest elevations of
cardiac enzymes. However, when cardiac catheterization is performed, no culprit lesions can be identified.

Although the most common presenting symptom is acute substernal chest pain, some patients may present with dyspnea or even shock. Most patients are between 60 to 75 years old, postmenopausal women, and present with an acute medical illness or report being under intense emotional stress. Death of a close relative is the stressor reported most frequently earning it the name “broken heart syndrome.” Although the incidence of Tako-tsubo cardiomyopathy is currently unknown, it has been reported to account for approximately 1 percent of suspected acute coronary syndromes and the incidence has been shown to increase following disasters such as the Mid-Niigata Prefecture earthquake. In-hospital mortality ranges from 0 to 8 percent and most patients who survive the acute episode recover normal LV function within two to four weeks.

The name tako-tsubo cardiomyopathy comes from the peculiar shape of the left ventricle seen during intra-cardiac ventriculography which resembles a Japanese pot used for catching octopuses called a “tako-tsubo”, literally meaning “octopus pot.” (Figure 2) The shape is caused by a hyperkinetic basal segment of the ventricle combined with a hypo or akinetic mid and apical segment. Because of the dilation and abnormal wall motion, the left ventricular ejection fraction is usually moderately to severely reduced. Although the exact etiology of this dysfunction is unknown, the leading hypothesis suggests that increased circulating catecholamines lead to a diffuse cardiac micro vascular vasospasm leading to cardiac wall motion abnormalities and impaired cardiac output. This theory is supported by a small study that showed that 13 patients diagnosed with tako-tsubo cardiomyopathy had circulating catecholamine levels 2 to 3 times higher than controls presenting with typical ST elevation myocardial infarctions of similar severity.

Criteria for diagnosis suggested by Mayo clinic includes transient akinesia or dyskinesia of the left ventricle, new electrocardiographic abnormalities including either ST elevations or T wave inversion, the absence of obstructive coronary artery disease on catheterization, and the absence of cardiomyopathy, head trauma, intracranial bleed or a pheochromocytoma. Initial treatment strategies are largely supportive but addressing and reducing the patients’ physical or emotional stress is also important. Although pharmacologic treatment remains somewhat controversial due to the lack of any trial data, there is general consensus among most experts advocating the use of beta blocker as well as an angiotensin-converting enzyme inhibitor at least until the patients left ventricular function has recovered. For hemodynamically unstable patients, reported treatments have included inotropic therapy, vasopressor support, and intra-aortic balloon counterpulsation.

Unlike the previously reported cases our patient presented with atypical abdominal pain and ileus and developed acute MI like changes under observation. Furthermore, although the death of a parent is the most commonly reported stressor, since this occurred almost one month earlier suggests that chronic stress may also predispose a patient to tako-tsubo cardiomyopathy.

References
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