Takotsubo Cardiomyopathy Complicated with Acute Pericarditis and Cardiogenic Shock

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Takotsubo cardiomyopathy (TC) is a relatively uncommon stress-induced cardiomyopathy that accounts for 2.2% of all acute myocardial infarctions. It occurs most commonly in postmenopausal women between the ages of 55–70. The most common complications that have been described are cardiogenic shock and left ventricular outflow tract obstruction, stroke and apical thrombus formation. There have been multiple prior case reports of TC; however, our case is the first to report acute pericarditis as one of its complications.

Key words: heart ■ vascular

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CASE REPORT

An 84-year-old African-American female with a past medical history of oral hypoglycemic-treated diabetes mellitus, medication-controlled hypercholesterolemia and hypertension was brought to the emergency department complaining of new-onset chest pain. She described the pain as "sharp," nonradiating, persistent and substernal. The pain was alleviated with rest and worsened with exertion. She denied any previous hospitalization for cardiac disease, dyspnea, diaphoresis, dizziness or fever. Notwithstanding, the patient reported that the onset of pain occurred two hours prior to her admission to the emergency room, directly after coming to the startling realization that her house had caught fire. Both her social and family histories were noncontributory.

A physical exam demonstrated normal S1 and S2 with no abnormal heart sounds. Furthermore, her lungs were clear to auscultation bilaterally. A chest x-ray and head CT performed on presentation to the ER were unremarkable. Initial laboratory tests were significant for troponin I that was elevated at 0.61 ng/ml. Additionally, an electrocardiogram (ECG) revealed 1.5-mm ST segment elevations in V2-V4, I and AVL leads (Figure 1).

This patient was admitted with a working diagnosis of myocardial infarction (MI) and was started on nitroglycerine sublingual, aspirin 162 mg, morphine 20 mg, IV heparin drip and IV pantoprazole sodium 20 mg. A cardiac catheterization revealed normal coronary arteries (Figure 2); however, left ventriculography showed sever left ventricular hypokinesis, mild mitral regurgitation, an ejection fraction of 20% with associated basal hyperkinesis and left ventricular apical ballooning. Takotsubo cardiomyopathy (TC) was diagnosed on the basis of these characteristic findings.

The patient's hospital course was significant for the development of cardiogenic shock and concomitant hypotension (blood pressure in the 90s), which manifested on hospital day 2. The patient was admitted to the CICU and required inotropic support for four days. On day 9, the patient developed pleuritic chest pain that she described as nonradiating, and 6/10 in severity. She stated that the pain decreased in severity with "sitting up and leaning forward." A subsequent physical exam revealed a cardiac rub with normal S1 and S2. A repeat ECG revealed diffuse ST segment elevations, and subsequent 2-D echo revealed mild pericardial effusion with an improved left ventricular ejection fraction of 40–50%. The patient was treated with NSAIDs for post-MI pericarditis. The patient's condition dramatically improved over the next few days and upon discharge the patients EF was noted to return to 70%, and her chest pain was resolved.

DISCUSSION

TC, also known as transient left ventricular apical ballooning syndrome, is a relatively uncommon stress-

induced cardiomyopathy. It was originally described as a heart syndrome characterized by transient regional left ventricular dysfunction involving the apex and mid-ventricle in the absence of obstructive epicardial coronary disease and other known cardiomyopathies.[1] While the original study noted that only 27% of patients diagnosed with the condition had a recent emotional or physical stress that was believed to play a key role in this cardiomyopathy, further studies have shown that up to 97% of patients were afflicted with these aforementioned stressors.[2] The following acute coronary syndrome presentations were noted in the original study; ECG changes: ST elevation (90%), Q-wave formation (27%) and T-wave inversion (97%). It was also found that 56% of patients had elevated cardiac enzymes.[1]

A case study from the Mayo Clinic used the criteria in the original study to diagnose TC and found that subsequent work-up further supported the diagnosis. The prognosis is favorable with total recovery of the ejection fraction in a period of a few weeks.[3] The severity of the illness appears to be out of proportion to the treatment that is required. The current recommendations treating this acute illness are largely supportive with specific recommendations for patients with TC and cardiogenic shock. While the use of beta blockers and phenylephrine are encouraged, there appears to be a limited role for inotropic agents because of their ability to worsen left ventricular outflow tract (LVOT) obstruction.[7]

The pathogenesis of this syndrome and its complications are still unknown, but some studies have suggested that myocardial damage appears to occur by catecholamine overload that leads to a stunned myocardium.[2,5] One study looked at a molecular explanation for the role of emotional and psychological stress in TC. It found a variety of molecules to be elevated and postulated that activation of alpha and beta adrenoceptors were the primary trigger of emotional stress-induced molecular changes in the heart.[4]

TC is a relatively uncommon stress-induced cardiomyopathy that accounts for 2.2% of all acute myocardial infarctions. It occurs most commonly in postmenopausal women between the ages of 55–70.[5]

There have been multiple prior case reports of TC; however, our case is the first to report acute pericarditis as one of its complications. The most common complications that have been described are cardiogenic shock and LVOT obstruction, stroke and apical thrombus formation.[6] Although the etiology of myocardial damage in TC appears to occur by catecholamine overload, its true source remains unclear. At the present time, there seems to be no proposed theory of myocardial damage that could explain post-TC pericarditis.

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Figure 1. Anterolateral ST segment elevations

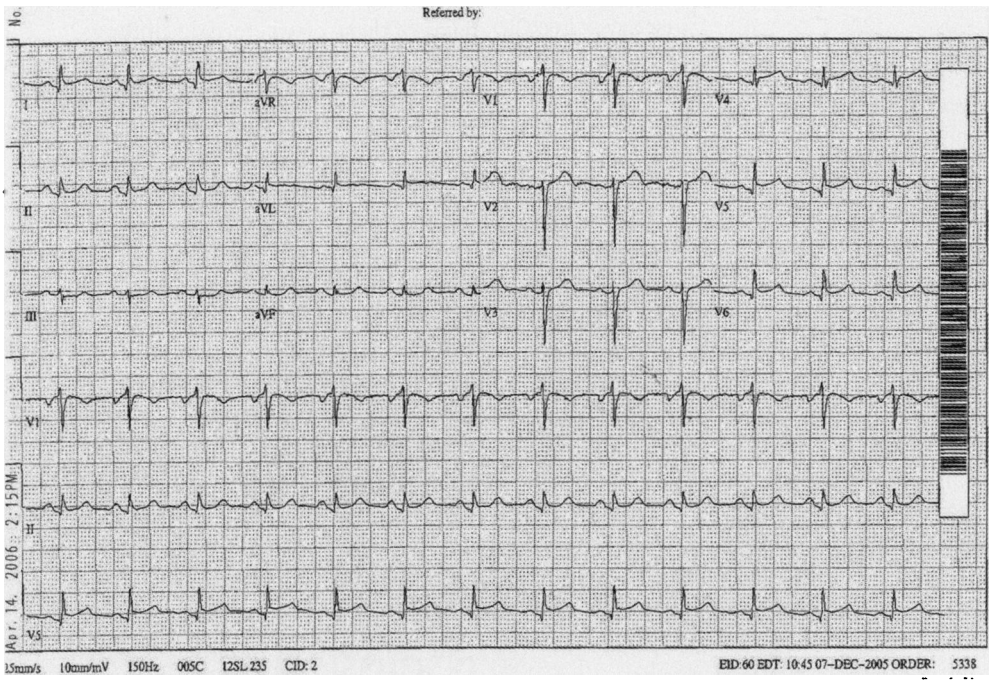
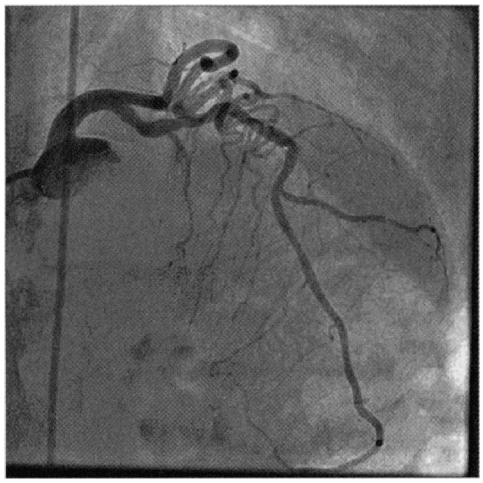


Figure 2. Cardiac catheterization revealing normal coronary arteries



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ART IN MEDICINE

Emphysema

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Slow asphyxiation, constant drain
Mighty once, now slave to a cane
Oceans of tears, unable to contain
Kind, elusive oxygen, for thee, my lungs strain
Incapacitated, bedridden, like a dragon slain
No respite, inescapable fate, cyanotic pain
Grim reaper, grinning above, with scalpel and chain