Transient left ventricular dysfunction after therapeutic pericardiocentesis - Takotsubo cardiomyopathy or pericardial decompression syndrome

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Abstract

We present a case of reversible left ventricular (LV) dysfunction with characteristic stress or «Takotsubo» cardiomyopathy (SCM) after therapeutic pericardiocentesis in a patient with tubercular pericardial effusion. SCM following pericardiocentesis is uncommon, as opposed to the well-defined entity, pericardial decompression syndrome (PDS). PDS is defined as a paradoxical deterioration of hemodynamics and development of severe biventricular dysfunction, cardiogenic shock, and pulmonary edema after uneventful, often large volume pericardiocentesis in patients of pericardial effusion.

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Introduction

Patients with cardiac tamponade routinely undergo pericardiocentesis which involves minimally invasive draining of the pericardial fluid. Although this procedure is relatively safe, rarely serious complications like perforation of the right ventricle, coronary laceration, pneumothorax, or even death may occur. In some patients, transient and reversible LV dysfunction after the procedure has been described [1,2]. This myocardial dysfunction may be global and can involve both the right and left ventricle [1]. Our case adds to our knowledge of the etiology of LV dysfunction complicating pericardiocentesis, as well as emphasizes the necessity of thorough evaluation in suspecting acute LV dysfunction after pericardiocentesis.

Case Report

A 52-year-old male, with poorly controlled diabetes mellitus presented to us with insidious onset, and gradually progressive shortness of breath for the last 20 days. He also reported having low-grade fever, associated with weight loss, and night sweats for the last two months. There was no history of cough, with or without expectoration. His examination revealed a heart rate (HR) of 110 beats per minute, blood pressure (BP) 80/60 mm Hg, and temperature of 37.4°C. He was tachypneic, had distended neck veins. On auscultation, air entry was reduced on bilateral lung bases, and heart sounds were muffled. Electrocardiogram (ECG) showed low voltage complexes and sinus tachycardia. Chest X-ray showed mild pleural effusion in bilateral lung fields and cardiomegaly. An emergency echocardiogram was done which demonstrated massive pericardial effusion, with diastolic right atrium (RA) and right ventricle (RV) collapse. There were no regional wall motion abnormalities (RWMA) and left ventricular function was normal (Figure 1, Video 1**)**. After obtaining informed consent, and taking universal aseptic precautions, urgent pericardiocentesis was performed. Around 600 mL of straw-colored pericardial fluid was removed and sent for etiological evaluation. The patient was relieved of symptoms immediately after the procedure, and his vitals returned to normal.

Around 4 h after pericardiocentesis, the patient again developed resting angina, dyspnea, and tachycardia. A repeat ECG showed qS complexes, with an ST elevation of 1 mm in the precordial leads. Cardiac biomarkers, troponin T, and creatine kinase-

MB were newly elevated to 0.44 ng/L and 312 U/L, respectively. A repeat echocardiogram showed minimal pericardial effusion, severe LV dysfunction (LV ejection fraction 30% by Simpson method) with RWMA in the apex, mid and apical septum, and lateral wall (Figure 2, Video 2). At this point, differential diagnoses of ischemic heart disease, SCM, coronary laceration, cardiac perforation, vasovagal response, and pericardial decompression syndrome were considered. Since at the time of fluid aspiration the fluid was non-hemorrhagic, we believed that coronary laceration or cardiac perforation leading to LV dysfunction was less likely. We performed a coronary angiogram, which turned out to be normal except for insignificant plaque in the mid-left anterior descending artery (Figure 3). To further elicit the diagnosis, we performed an LV angiogram which showed apical ballooning in systole, thus indicating SCM (Figure 4).

The patient was managed medically in the intensive care unit with tablet bisoprolol 2.5 once daily, tablet ramipril 2.5mg once daily, tablet aspirin 75mg once daily, and tablet atorvastatin 40mg once daily. The fluid analysis suggested a tubercular pathology for which guideline-directed anti-tubercular treatment was started. A repeat echocardiogram after 2 weeks showed normal LV function (LV ejection fraction 55%) with no RWMA.

Discussion

For patients with cardiac tamponade, pericardiocentesis is a life-saving minimally invasive therapeutic intervention. Although it is a relatively safe procedure, associated with minimal complications, clinicians must be aware of the potential for post-procedural problems. Acute worsening of symptoms and paradoxical hypotension developing post-procedure should raise concern for two under-diagnosed and under-reported diagnoses: PDS and SCM [3].

Classically, PDS has been described as paradoxical hypotension with pulmonary edema and ventricular dysfunction, developing shortly after transient improvement of symptoms post pericardiocentesis. The onset might range from minutes after drainage to up to 48 h later, with a fatality rate of up to 29% [4]. Cardiac enzymes do not reliably predict PDS, in one case series, they were elevated in 8 out of 10 patients, whereas in other studies, they were not elevated in the majority of PDS patients [5,6]. As can be seen, this syndrome presents in a variety of ways and may be linked to a variety of factors. After pericardiocentesis, the incidence of PDS, or acute left or right systolic dysfunction, has been observed to vary from 5% to 36% of cases [7].

Different hypotheses have been proposed to understand the pathophysiology of PDS [1,3]:

1. *Hemodynamic hypothesis:* Sudden hemodynamic transition after rapid removal of pericardial fluid causes increased venous return, thus increased right ventricular filling. This leads to compression of LV and pulmonary edema.
2. *Ischemic hypothesis:* The pericardial effusion compresses the epicardial coronary vasculature causing high perfusion pressures, and 'myocardial stunning'. Rapid draining of pericardial fluid results in transient myocardial dysfunction leading to further inability to handle sudden fluid shifts.
3. *Autonomic imbalance hypothesis:* The baseline sympathetic tone is elevated in patients with tamponade physiology, which increases LV contractility and HR to maintain cardiac output. Once the pericardial fluid is drained rapidly, this sympathetic overactivity unmasks the underlying LV dysfunction.

The classical echocardiographic and LV angiographic feature of SCM is transient LV apical ballooning. A sudden sympathetic surge and catecholamine excess is a well-known trigger for the development of the so-called Takotsubo SCM [8]. This surge causes increased peripheral arterial resistance, increased afterload, and thus increased LV end-diastolic pressures. Also, this catecholamine

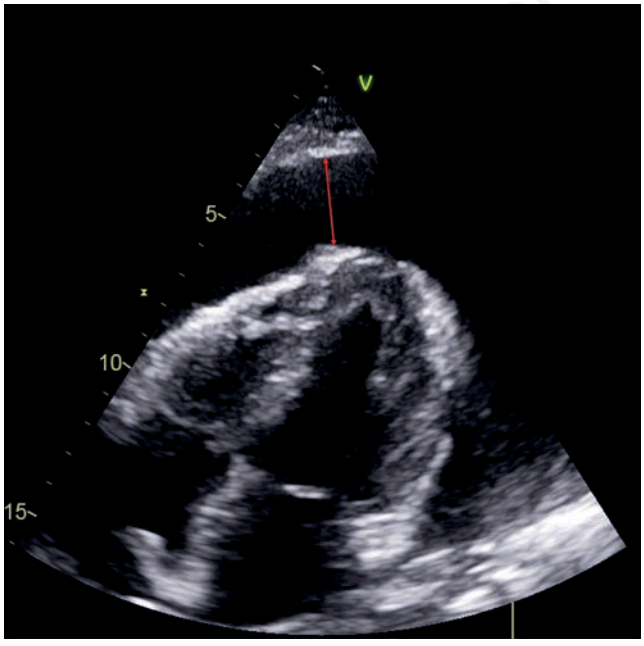

Figure 1 (Video 1). Echocardiogram showing massive pericardial effusion (red arrow) with normal left ventricle systolic function, and no regional wall motion abnormalities.

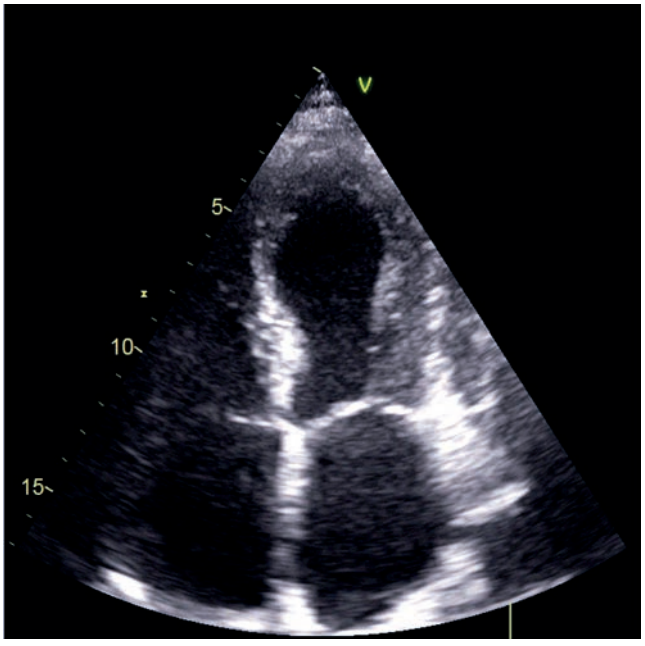

Figure 2 (Video 2). Apical four chamber view showing apical ballooning of left ventricle, with regional wall motion abnormalities in the apex, mid and apical septum, and lateral wall.

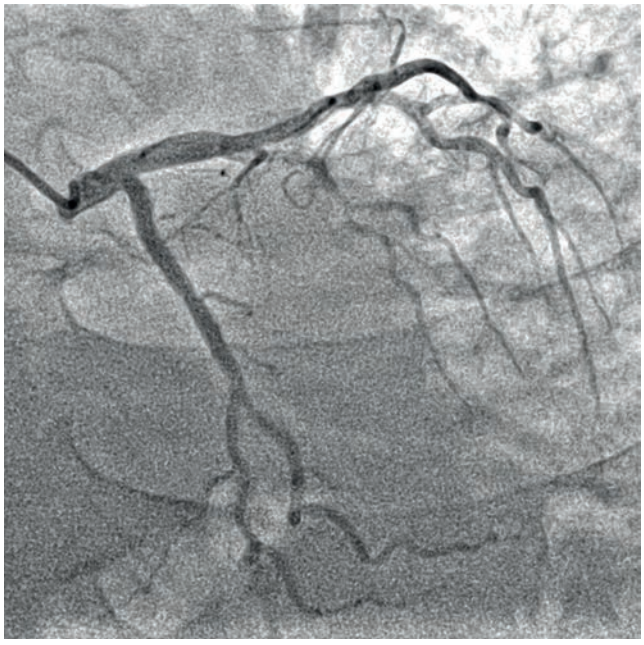

Figure 3. Right anterior oblique caudal projection demonstrating insignificant plaque in the mid-left anterior descending artery.

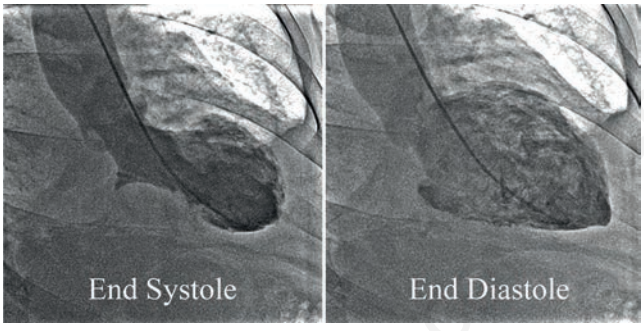

Figure 4. Left ventricle angiogram showing apical ballooning.

excess leads to sudden coronary vasospasm causing myocardial ischemia, and direct beta-receptor mediated stunning of the apex. These mechanisms with underlying non-obstructed coronaries, contribute to the transient ischemia, apical ballooning, and development of hemodynamic sequelae of SCM [9].

Recent literature has described LV apical ballooning of PDS as identical to seen in SCM. The postulated trigger is increased sympathetic tone in patients with tamponade physiology, which is further worsened by the anxiety and pain during the procedure[7]. In our case, the classical presentation of the patient after pericardiocentesis- chest pain, raised cardiac enzymes, newly developed ST elevations, LV dysfunction with apical ballooning, normal coronary angiogram, and subsequent improvement of LV function after 2 weeks suggest SCM as the likely diagnosis.

The risk factors for developing PDS or SCM post pericardiocentesis remain less researched, however, it is established that PDS developing after surgical pericardiostomy is associated with higher mortality as compared to PDS developing after needle pericardiocentesis [4]. Experts recommend that pericardial fluid removal should be gradual, and only done till the tamponade physiology is corrected. The remaining fluid must be drained slowly, to let the myocardium and coronary flow adapt to hemodynamic shifts [1,7]. At present, there are no evidence-based guidelines to prevent or treat PDS. Also, it remains unknown if PDS and SCM are different entities or a consequence of common underlying pathophysiology.

Patients with pericardial effusion with tamponade physiology are routinely treated in cardiology centers worldwide. However, it is worthwhile to be mindful of these rare complications (PDS or SCM) of simple pericardiocentesis, so that these can be anticipated and avoided.

Conclusions

The underlying pathophysiology of PDS remains yet to be precisely described. It is likely that the underlying catecholaminergic excess of tamponade physiology (a reflex to maintain adequate cardiac output) causes SCM. A reduction of this sympathetic stimulation after removal of pericardial fluid, leads to cardiogenic shock and pulmonary edema, as described in classical PDS. Hence, further studies are needed to elicit the pathophysiology of PDS and SCM as overlapping entities and then establish evidence-based guidelines to prevent and treat them.

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