A case report of ventricular dysfunction post pericardiocentesis: stress cardiomyopathy or pericardial decompression syndrome?

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Abstract
We report a case of transient biventricular dysfunction post therapeutic pericardiocentesis, with classic features of stress cardiomyopathy (SCM). In our patient, the clinical and echocardiographic features were more in keeping with Takotsubo-type SCM than pericardial decompression syndrome (PDS). Our case is instructive in challenging our understanding of the aetiology of LV dysfunction complicating pericardiocentesis, and in highlighting the importance of careful clinical evaluation (altered heart rate and dyspnoea) in suspecting acute LV dysfunction after initial clinical improvement with pericardial aspiration.

Keywords: Pericardiocentesis, Stress cardiomyopathy, Pericardial decompression syndrome, Ventricular dysfunction, Echocardiography

Background
We report a case of reversible biventricular dysfunction following successful pericardiocentesis with classic features of stress or “Takotsubo” cardiomyopathy (SCM). Reports of SCM after pericardiocentesis are rare [1], as distinct from so-called pericardial decompression syndrome (PDS) which encompasses a spectrum of features of cardiac decompensation after large volume pericardiocentesis, including pulmonary oedema, adult respiratory distress syndrome, severe bi-ventricular failure and cardiogenic shock [2]. Our case is instructive in challenging our understanding of the aetiology of LV dysfunction complicating pericardiocentesis, and in highlighting the importance of careful clinical observations (heart rate and dyspnoea) in suspecting acute LV dysfunction after initial clinical improvement with pericardiocentesis.

Case report
A 62-year-old male presented with progressive dyspnoea for 10 days. He had a background of stage IV metastatic non-small lung carcinoma treated for 6 months with non-cardiotoxic chemotherapy (carboplatin and gemcitabine), and recently commenced on target therapy (Erlotinib). Clinical examination revealed signs consistent with cardiac tamponade, including significant pulsus paradoxus, tachycardia (heart rate 101), tachypnea (respiratory rate 25), elevated jugular venous pressure and muffled heart sounds. He was normotensive at 130/90mmHg. The patient was extremely anxious and spontaneously expressed concern about his imminent death.

His electrocardiogram (ECG) (Fig. 1) demonstrated electrical alternans and bedside transthoracic echocardiography (TTE) revealed a large pericardial effusion with features of cardiac tamponade, including diastolic compression of both right atrium and ventricle (Fig. 2, Additional file 1: Video 1 and Additional file 2: Video 2) and large mitral inflow variation (Fig. 3). Urgent pericardiocentesis was performed with a restricted aspiration of only 600 ml drained initially over the first hour, and a total drainage of 1.8 l of heavily blood-stained pericardial fluid over 36 h. During initial aspiration of pericardial fluid there was immediate symptomatic relief and haemodynamic improvement (heart rate [HR] decreased to 80/min, respiratory rate [RR] decreased to 15 breaths/min and BP increased to 150/70 mmHg).
Overnight (9 h post procedure) the patient developed chest discomfort, dyspnea, tachycardia (HR 110) and tachypnoea (RR 24). TTE the next morning showed no re-accumulation of pericardial fluid, but detected new severe impairment in function of both ventricles, with akinesis of the apex and peri-apical region (Figs. 4 and 5, Additional file 3: Video 3 and Additional file 4: Video 4). Biomarkers demonstrated a rise in highly sensitive troponin from 8 to 224ng/L, but creatinine kinase did not rise significantly (107 to 116U/L). ECG after chest pain demonstrated resolution of the electrical alternans, with new loss of R waves in the anterior leads (Fig. 6).

Based on a presumptive diagnosis of SCM, angiotensin converting enzyme inhibitor and long acting beta-blocker were commenced, chemotherapy withheld and the patient discharged for early clinical and echocardiographic review. Serial follow up TTEs showed normalization of bi-ventricular function after two weeks (Figs. 7 and 8, Additional file 5: Video 5 and Additional file 6: Video 6), and restoration of R waves on subsequent
ECGs (Fig. 9). Subsequent computed tomography examination showed normal coronary arteries with a calcium score of zero and no evidence of LAD laceration or dissection.

The patient presented three months later with reaccumulation of pericardial effusion and tamponade. Therapeutic pericardiocentesis was performed with 500 ml of blood stained pericardial fluid drained immediately, with 1.9 L in total over 36 h. On this presentation he was relaxed and well adjusted in regards to his diagnosis. No LV dysfunction was detected on serial follow-up echocardiograms after the second pericardiocentesis (Fig. 10).

Discussion

Our patient developed biventricular apical dysfunction following successful and judicious pericardiocentesis, with features typical of stress or “Takotsubo cardiomyopathy”. The case is instructive for its comparison with PDS and the clinical pattern of initial improvement followed by deterioration respectively due to pericardial aspiration and myocardial pathology.

In light of the timing of onset of biventricular impairment immediately post procedure PDS is an important differential diagnosis. Other differentials such as laceration to the ventricle or left anterior descending (LAD) coronary artery were clinically unlikely. The former was
excluded by the absence of new pericardial bleed post procedure. Laceration of the LAD was also clinically unlikely given relatively small rise in cardiac enzymes and absence of large infarct, the presence of concurrent RV dysfunction, spontaneous recovery of ventricular function in a short period of time; additionally CT scan showed no evidence of haematoma or injury to the LAD.

Accordingly, we reviewed the literature describing SCM and PDS. Whereas SCM has been rarely reported after pericardiocentesis, much has been published on PDS. The incidence of PDS or new left or right systolic dysfunction has been reported to range from 5% to 36% of patients post pericardiocentesis [3, 4], especially after malignant pericardial effusions. Although the first case report of PDS in 1983 noted APO with preserved LV function [5], most subsequent reports describe severe impairment of left, right or bi-ventricular function, which may be segmental or global (Tables 1 and 2).

A number of mechanisms have been proposed to explain the pathogenesis of LV systolic dysfunction in PDS.
Acute withdrawal of exaggerated sympathetic drive during relief of tamponade may trigger paradoxical haemodynamic instability [5]. Mechanical, inter-ventricular volume mismatch may also contribute, with sudden relief of pericardial constraint leading to abrupt, disproportionate increase in RV volume and a paradoxical rise in pulmonary artery pressure, resulting in raised LV end diastolic pressure and transient left heart failure [5–9]. Others have proposed myocardial stunning from coronary perfusion mismatch with acute distension of cardiac chambers after decompression [6, 10, 11]. Taken together, it is likely that a combination of hormonal and mechanical pathophysiologic mechanisms contribute to LV dysfunction and the final clinical sequelae in PDS.

The classic echocardiographic feature in SCM is transient LV apical ballooning, although other segmental patterns have been described [12, 13]. A stressor leading to sympathetic overdrive and excessive catecholamine release is the currently accepted trigger in the development of SCM [12]. The catecholamine surge precipitates 1) peripheral arterial vasospasm leading to increased afterload and transient increase in LV end-systolic pressure; 2) acute multiple coronary artery vasospasm leading to myocardial ischaemia, and 3) direct catecholamine-
Fig. 9 ECG 2 months post event demonstrating resolution of ischemic changes in ECG in Figure 6

Fig. 10 Time line of clinical events
| Report           | Age/Gender | Clinical Scenario | Chronicity of effusion | Type of pericardiocentesis | Nature of pericardial fluid | Fluid drained | Time to onset of symptoms | Symptom | Signs          |
|------------------|------------|-------------------|------------------------|----------------------------|------------------------------|---------------|--------------------------|---------|----------------|---|
| VanDyke (1983)   | 42 M       | Unwell for 10 days| Days                   | P                          | Exudate (malignant)         | 680 mls       | Minutes                  | Dyspnoea| LVF            |   |
| Shenoy (1984)    | 57 M       | Recent myocardial infarction| Days             | P                          | Transudate                  | 1000 mls      | Minutes                  | Dyspnoea| LVF            |   |
| Glasser (1988)   | 33 M       | Respiratory tract infection 3 months prior, history of Down's and Ventricular Septal Defect| Weeks         | S                          | Transudate                  | 2000 mls      | Minutes                  | Dyspnoea| LVF            |   |
| Downey (1991)    | 50 M       | Traumatic (3 weeks post motor vehicle accident) | Weeks         | P                          | Not specified               | 450 mls then 1500 mls | Minutes                  | Dyspnoea| LVF            |   |
| Wolfe (1993)     | 46 F       | 2 weeks, history of breast cancer prior | Weeks         | P                          | Exudate                     | 650 mls       | Weeks                    | Dyspnoea| LVF            |   |
| Wolfe (1993)     | 50 F       | 2 weeks, history of breast cancer prior | Weeks         | P                          | Exudate                     | 650 mls       | Weeks                    | Dyspnoea| LVF            |   |
| Hamaya (1993)    | 16 F       | Unwell, lymphoma with pericardial effusion for 3 years | Months        | P                          | Not specified               | 700 mls       | Weeks                    | Dyspnoea| CS, and no APO|   |
| Braverman (1994) | 27 F       | Unwell for 3 weeks (Atrial Septal Defect closure 13 years prior) | Weeks         | P then S                   | Transudate                  | 500 mls then 100 mls | Days                    | Dyspnoea, pleuritic chest pain | LVF, RVF, CS |   |
| Anguera (1996)   | 68 F       | History of bowel cancer, anorexia and dyspnoea for 1 month | Weeks         | P                          | Malignant                   | 800 mls       | Minutes                  | -       | CS             |   |
| Sunday (1999)    | 60 F       | 3 days of dyspnoea, lung cancer with pericardial involvement | Days          | S                          | Exudate                     | 700 mls       | Minutes                  | Dyspnoea| CS, LVF         |   |
| Chamoun (2003)   | 36 F       | 2 months post Mitral valve replacement and Tricuspid repair | Days          | P                          | Exudate                     | 1070 mls      | Hours                    | Dyspnoea| CS, LVF         |   |
| Chamoun (2003)   | 46 F       | Metastatic cancer | Weeks         | P                          | Exudate                     | 1000 mls      | Hours                    | Dyspnoea| CS, LVF         |   |
| Geffroy (2004)   | 53 M       | 1 month post chemotherapy for cancer | Weeks         | S                          | Exudate                     | 1500 mls      | Not specified            | Dyspnoea, hypoxia | CS, LVF, RVF |   |
| Ligero (2006)    | 41 F       | Lung cancer with hepatic metastases | Days          | P                          | Exudate                     | 1000 mls      | Hours                    | Dyspnoea| LVF, RHF        |   |
| Bernal (2007)    | 45 F       | Acute myeloid leukemia | Days          | P                          | Exudate                     | 500 mls       | Hours                    | Dyspnoea| CS, LVF         |   |
| Dosios (2007)    | 66 F       | Hematoma, 10 day history of dyspnoea | Days          | S                          | Exudate                     | 500 mls initially | Hours                  | -       | CS             |   |
| Sevimli (2008)   | 42 F       | Infective - tuberculous pericarditis | Days          | S                          | Exudate                     | 500 mls       | Hours                    | Dyspnoea| CS and LVF      |   |
| Khalili (2008)   | 32 F       | 2 months post aortic and mitral valve replacement surgery | Weeks         | P                          | Transudate                  | 1000 mls      | Hours                    | Dyspnoea| CS             |   |
| Name                | Age | Gender | Diagnosis                                                                 | Procedure | Drainage | Time | Symptoms          | Outcome |
|---------------------|-----|--------|---------------------------------------------------------------------------|-----------|----------|------|-------------------|---------|
| Flores (2009) [30]  | 80  | M      | Unwell for weeks, multiple myeloma, stent 2 weeks prior                  | Weeks P   | Transudate 1200 mls | Days | Dyspnoea          | CS and LVF |
| Karamichalis (2009) | 19  | F      | 2 months post motor vehicle accident                                     | Weeks P   | Exudate 1600 mls   | Hours | Dyspnoea          | LVF     |
| Lee (2010) [18]     | 14  | M      | Infective – tuberculous pericarditis                                     | Days P    | Exudate Not specified | Hours | Dyspnoea          | CS, LVF |
| Lim (2011) [32]     | 44  | F      | Hypothyroidism related heart failure. Dyspnoea and fatigue for 4 months | Weeks S   | Exudate 1.3L       | 9 h  | -                 | CS      |
| Abdelsalam (2012) [10] | 65  | F      | Stage IV Non small cell lung cancer for 6 months, 1 week of dyspnoea     | Weeks S   | Malignant Complete drainage of pericardial effusion intraoperatively | Seconds | Asystole during surgery | CS |
| Weijers (2013) [11] | 69  | F      | Weight loss and dyspnoea                                                | - P       | -                  | 800 mls | 6 h               | LVF     |
| Liang (2014) [1]    | 56  | F      | Polymyositis. Progressive dyspnoea on exertion                           | - P       | -                  | 275 mls | Several hours     | Nil     |
| Versaci (2015) [16] | 78  | F      | 3 months post mitral valve repair                                        | Days P    | Possibly transudate 500 mls | Hours | Dyspnoea          | LVF     |

**Abbreviations:** P percutaneous, S surgical, CS cardiogenic shock (hypotension, tachycardia), LVF Left heart failure, RVF right heart failure
| Report            | LV function pre tap | LV function post tap | RV function post tap | Regional wall motion abnormality | Bio marker ECG | Coronary artery imaging | Inotrope, IABP or Intubation | Death | LV recovery |
|-------------------|---------------------|----------------------|---------------------|----------------------------------|----------------|------------------------|-----------------------------|-------|-------------|
| VanDyke (1983)    | Normal              | Normal (EF 67%)      | -                   | Nil                              | Normal         | Normal                 | Intubation                  | No    | Normal LV   |
| Shenoy (1984)     | Mild LV impairment  | Normal               | Septal hypokinesis  | Normal                            | T wave abnormality and ST elevation V5-6 | - | - | No | Normalised few days later |
| Glasser (1987)    | Pulmonary capillary wedge pressure normal | - | - | - | Intubation                          | No | Clinical improvement |
| Downey (1991)     | Inferred to be normal | Normal               | -                   | - | Normalised few days later          | No    | Normal LV   |
| Wolfe (1993)      | Normal, EF > 50%    | EF 30%               | Severe global hypokinesis of LV | - | - | No | Normalised after 7 days |
| Wolfe (1993)      | Normal, EF > 50%    | EF 25%               | Antero-apical akinesis and apical dyskinesis | - | - | No | Normalised after 2 weeks |
| Hamaya (1993)     | Normal              | -                   | Not provided         | Normal                            | ST elevation   | Inotropes and intubation | No | - |
| Braverman (1994)  | EF 20%              | EF 20%               | EF <15%             | Not provided                       | - | - | - | EF 45% in 9 days then normalised after a few weeks |
| Anguera (1996)    | Mildly impaired. Normal capillary wedge pressure | Severely dilated and severely impaired contractility, EF <15% | Paradoxical septal motion | - | - | Normal coronary arteries | Inotropes | No | Complete recovery of biventricular fn after 10 days |
| Sunday (1999)     | EF 65%              | EF 30%               | Severely impaired contractility | Global hypokinesis | - | - | Intubation | Yes | No |
| Chamoun (2003)    | Normal, EF > 50%    | EF 20%               | Regional wall motion abnormality | - | SR | Normal coronary arteries | Inotropes and IABP | No | Normalised 2 weeks later |
| Chamoun (2003)    | Normal, EF > 50%    | EF 20%               | Akinosis of mid anterior wall and septum /dilatation of LV | - | SR | Normal coronary arteries | Inotropes and intubation | No | Normalised 2 weeks later |
| Geffroy (2004)    | Normal, EF > 50%    | EF <15%              | Akinetic and dilated RV | Elevated Old RBBB | Normal coronary arteries | Inotropes and intubation | Yes | - |
| Ligero (2005)     | Normal, EF 75%      | EF 25%               | Severe impairment    | Akinesis of anterior, septum and apex | Normal CK | Normal coronary arteries | Inotropes | No | Normalised 10 days later |
| Bernal (2007)     | Normal, EF 60-65%   | EF 30%               | Akinosis of mid anterior wall, anteroseptal akinesis with apical sparing | Elevated Sinus tachycardia | CMR: no myocardial infarction | Inotropes and intubation | No | Normalised 1 weeks later |
| Dosios (2007)     | Normal LV fn        | EF 25%               | Moderately dilated, impaired | Global hypokinesis | Elevated | - | Inotropes and intubation | Yes | - |
|                  | EF 20%              | -                   | -                   | -                               | - | - | No | No |

Note: EF = Ejection Fraction, CMR = Cardiac Magnetic Resonance Imaging, LV = Left Ventricular, RV = Right Ventricular, IABP = Intra-Aortic Balloon Pump, CK = Creatine Kinase, RBBB = Right Bundle Branch Block, SR = ST segment, Normal = Normalised.
| Author (Year) | EF (2008) | EF 35% | <10% | EF <15% | Global hypokinesis | Normal, EF > 50% | Severe mid and apical hypokinesis of both Ventricles (diagnosis: Takotsubo’s cardiomyopathy) |
|--------------|-----------|--------|------|---------|--------------------|------------------|---------------------------------------------------------------------|
| Khalili (2008) | 60%       | 13%    | -    | Normal | Normal             | Normal            | Normal, EF >50% | LV balloononing, typical feature of Takotsubo’s cardiomyopathy |
| Flores (2009) | 2008      | EF 60% | 13%  | -       | Normal             | Normal            | Normal, EF >50% | LV balloononing, typical feature of Takotsubo’s cardiomyopathy |
| Karamichalis (2009) | EF normal, 73% | EF 46% | -    | Normal, EF 69% | Severe mid and apical hypokinesis of both Ventricles (diagnosis: Takotsubo’s cardiomyopathy) | Normal            | Normal, EF >50% | LV balloononing, typical feature of Takotsubo’s cardiomyopathy |
| Lee (2010)    | Vigorous  | EF 10-15% | Dilated and impaired fn | Takotsubo pattern of akinesia | ST elevation | Normal            | No | Yes | No |
| Weijers (2013) | Normal | Poor LV fn | - | General hypokinesia and anterior and septal akinesia | Normal, EF 69% (on MRI) | Impaired | Normal, EF >50% | Normal, EF >50% |
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β-adrenoceptor-mediated myocardial stunning in the apex [14]. These three pathophysiologic pathways are thought to contribute to the ischaemia, morphologic features and potential haemodynamic sequelae that can be seen in SCM.

More recent case reports have made reference to LV apical ballooning related to PDS as similar to SCM [10, 11, 15–17], and have postulated the physiological stressor being cardiac tamponade along with emotional stress [16]. It is therefore possible that the transient ventricular systolic dysfunction in PDS is actually a variant form of stress cardiomyopathy. We carefully reviewed 25 cases of heart failure post pericardiocentesis in the literature (Tables 1 and 2), and we believe that seven cases (two considered to be SCM [1, 18] by the authors and five classified as PDS [10, 16, 17, 19, 20]) could be considered to have echocardiographic features of SCM.

SCM has relatively characteristic clinical presentation, with rise of cardiac enzymes [21], and often associated with ischaemic ECG changes (up to 44 % of those with SCM have T-wave inversion and 41 % ST elevation [13, 21]). The clinical manifestations in PDS are more variable, ranging from asymptomatic in some to severe low cardiac output states in others. The primary clinical symptom in PDS has been reported as dyspnoea (Table 1). This is in contrast to chest pain being predominant in SCM (69–83 % of presentations) [13, 21]. In the majority of cases of PDS in the literature (Table 2) there was no cardiac enzyme rise, and ischaemic type changes on ECG were seen in a minority (seven of twenty five cases). In all the cases where ischaemic ECG changes where present except for one, there was concomitant apical and peri-apical regional wall motion abnormality, which could be classified as SCM also.

Generally SCM has a benign course, with recovery of LV function and good prognosis [12], whilst PDS has poorer outcomes and increased mortality [4]. Reports of PDS suggested normalization of LV dysfunction in 12 of 25 cases classified as PDS. Of the 12 cases that did recover LV function, four had LV impairment with classic SCM pattern of LV impairment on echocardiogram [16, 17, 19, 20]. The normalisation of LV function in our patient 2 weeks subsequently is more in keeping with SCM.

Current literature has not specifically addressed risk factors for the development of ventricular dysfunction after pericardiocentesis. In our patient, the malignant nature of the effusion, the presence of tamponade and larger size of pericardial effusion [4], may have increased his predisposition to develop ventricular dysfunction. Amount and rate of fluid removed on initial decompression are also associated with development PDS [4, 5], however there are no guidelines regarding the maximum amount of pericardial fluid that can be drained immediately. There is consensus to stop initial drainage with improvement of symptoms or hemodynamic parameters, followed by gradual decompression through indwelling catheter [5].

Our patient’s apical systolic dysfunction post pericardiocentesis was associated with chest discomfort, transient loss of R waves and rise in cardiac enzymes are typical of classic SCM. The clinical sequence of HR and RR improving immediately post decompression and then increasing again hours after the procedure, was a useful clinical marker of myocardial dysfunction, prompting investigation which identified new ventricular impairment. It is likely that the frequency of transient LV dysfunction is underestimated in these patients.

Conclusion

We report a case of transient biventricular dysfunction post pericardiocentesis, with classic features of SCM. LV dysfunction post pericardiocentesis and in PDS is more prevalent than previously thought, and some previous reports of PDS may also be potentially considered as SCM complicating pericardiocentesis. In addition to judicious and gradual decompression to avoid ventricular dysfunction or PDS, patients undergoing therapeutic pericardiocentesis should have careful haemodynamic monitoring, as changes in parameters such as heart rate and respiratory rate can raise suspicion of acute LV impairment.

Consent

Written informed consent was unable to be obtained from the patient for publication of this Case report and any accompanying images, as he has passed away. His next of kin are not contactable after their subsequent return to their home country of China. Professor L. Kritharides, Head of Department, approves the publication of this report, with all patient identifiers kept confidential and material presented solely for educational purposes arising from the clinical encounter.

Additional files

**Additional file 1:** Video 1. Apical four chamber view demonstrating large pericardial effusion on presentation, with tamponade and diastolic compression of right heart chambers. Note the LV function is normal.

**Additional file 2:** Video 2. Parasternal long view demonstrating large pericardial effusion on presentation, with tamponade and diastolic compression of right heart chambers. Note the LV function is normal.

**Additional file 3:** Video 3. Apical four chamber view performed on the following morning after the patient experienced chest discomfort. The main finding is severe biventricular impairment, with akinesis of the apex and periapical areas. There is small residual pericardial effusion.
Apical four chamber view performed nearly 2 weeks after the pericardiocentesis and subsequent stress cardiomyopathy. They show normalization of both left and right ventricular function.

Parasternal long view performed nearly 2 weeks after the pericardiocentesis and subsequent stress cardiomyopathy. They show normalization of both left and right ventricular function.

Additional file 5: Video 5. Apical four chamber view performed nearly 2 weeks after the pericardiocentesis and subsequent stress cardiomyopathy. They show normalization of both left and right ventricular function.

Additional file 6: Video 6. Parasternal long view performed nearly 2 weeks after the pericardiocentesis and subsequent stress cardiomyopathy. They show normalization of both left and right ventricular function.

Abbreviations

SCM: Stress Cardiomyopathy; LV: Left ventricle; RV: Right ventricle; PDS: Pericardial decompression syndrome; APO: Acute pulmonary oedema; ECG: Electrocardiogram; TTE: Transthoracic echocardiography; bpm: Beats per minute; HR: Heart rate; RR: Respiratory rate.

Competing interests

The authors declare that they have no competing interests.

Authors’ contributions

All authors have approved the final article, contributed to conception, literature review, analysis and interpretation of the material, to the drafting of the manuscript and its critical revision; All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Received: 9 April 2015 Accepted: 30 June 2015

Published online: 16 July 2015

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