CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 28 (2016) 145–148

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Biventricular transient systolic dysfunction after mitral valve replacement: Pericardial decompression syndrome

Sebnem Albeyoglu, Mustafa Aldag*, Ufuk Ciloglu, Hakan Kutlu, Sabri Dagsali

Department of Cardiovascular Surgery, Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey

ARTICLE INFO

Article history:
Received 29 July 2016
Received in revised form
27 September 2016
Accepted 27 September 2016
Available online 30 September 2016

Keywords:
Mitral valve
Pericardial effusion
Decompression
Heart failure
Systolic
Case report

ABSTRACT

BACKGROUND: Pericardial decompression syndrome is defined as paradoxical hemodynamic instability, left ventricular or bi ventricular systolic dysfunction and pulmonary edema after pericardial fluid drainage. Pericardial Decompression Syndrome is an unexpected clinical scenario with an incidence less than 5% in all surgically or percutaneously managed pericardial tamponade patients. The aim of this manuscript was to describe a case with cardiac tamponade in whom acute biventricular heart failure and pulmonary edema developed after surgical creation of a pericardial window, and to discuss this case in light of the literature.

CASE REPORT: A 43-year-old woman who underwent mitral valve replacement three weeks ago admitted to our hospital with dyspnea, tachycardia, and atrial fibrillation. Large quantity of pericardial fluid (35 mm in the posterior wall, 25 mm in the anterior wall) with partial compression of the right ventricle and 50% left ventricle ejection fraction (LVEF) was determined via transthoracic echocardiography (TTE). After creation of pericardio-pleural window, more than 1000 ml of serosanguineous fluid were quickly removed from the pericardial space. During the following hours of the decompression, the patient's condition deteriorated and overt pulmonary edema developed. On the second day, biventricular systolic dysfunction, global diffuse hypokinesia and 15–20% LVEF was observed via TTE. High-dose inotropic support and diuretics was continued. During follow up she was progressively weaned off inotropes, LVEF were raised to 35%. Two weeks later, repeated TTE showed normal biventricular systolic function and LVEF was 50%.

CONCLUSION: We recommend gradual removal of pericardial effusion under hemodynamic monitoring, especially in patient with postcardiotomy tamponade.

1. Introduction

Pericardial Decompression Syndrome (PDS) is an unexpected clinical scenario with an incidence less than 5% in all surgically or percutaneously managed pericardial tamponade patients [1,2]. Pericardial decompression syndrome is defined as paradoxical hemodynamic instability, left ventricular (LV) or bi ventricular systolic dysfunction and pulmonary edema after pericardial fluid drainage [1–3].

The aim of this manuscript was to describe a case with cardiac tamponade in whom acute biventricular heart failure and pulmonary edema developed after surgical creation of a pericardial window, and to discuss this case in light of the literature. Raising awareness of PDS may help its diagnosis and management of this potentially devastating but reversible complication after cardiac surgery.

2. Presentation of case

A 43-year-old woman, had an operation for mitral valve disease 3 weeks ago, after discharged from the hospital, was admitted to our cardiovascular surgery department with a 3 days' history of worsening dyspnea, tachycardia, and palpitation. Physical examination revealed heart rate was 110 beats per minute (chronic atrial fibrillation), blood pressure was 110/76 mmHg, and respiratory rate was 25 breaths per minute, and jugular venous distension was noted. Her chest X-ray revealed left pleural effusion, and enlarged heart silhouette. Electrocardiogram (ECG) showed atrial fibrillation with rapid ventricular response. She was further investigated with a transthoracic echocardiogram, which confirmed the presence of large quantity of pericardial fluid (35 mm in the posterior wall, 25 mm in the anterior wall) with partial compression of the

Abbreviations: CPAP, continuous positive airway pressure; ECG, electrocardiogram; ICU, intensive care unit; PDS, pericardial decompression syndrome; PCWP, pulmonary capillary wedge pressure.

* Corresponding author at: Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Department of Cardiovascular Surgery, Tibbiye Cad. No: 25, Siyami Ersek Hastanesi, Selimiye-Uskudar, Istanbul, 34668, Turkey.
E-mail address: mustafa.aldag@saglik.gov.tr (M. Aldag).

http://dx.doi.org/10.1016/j.ijscr.2016.09.045

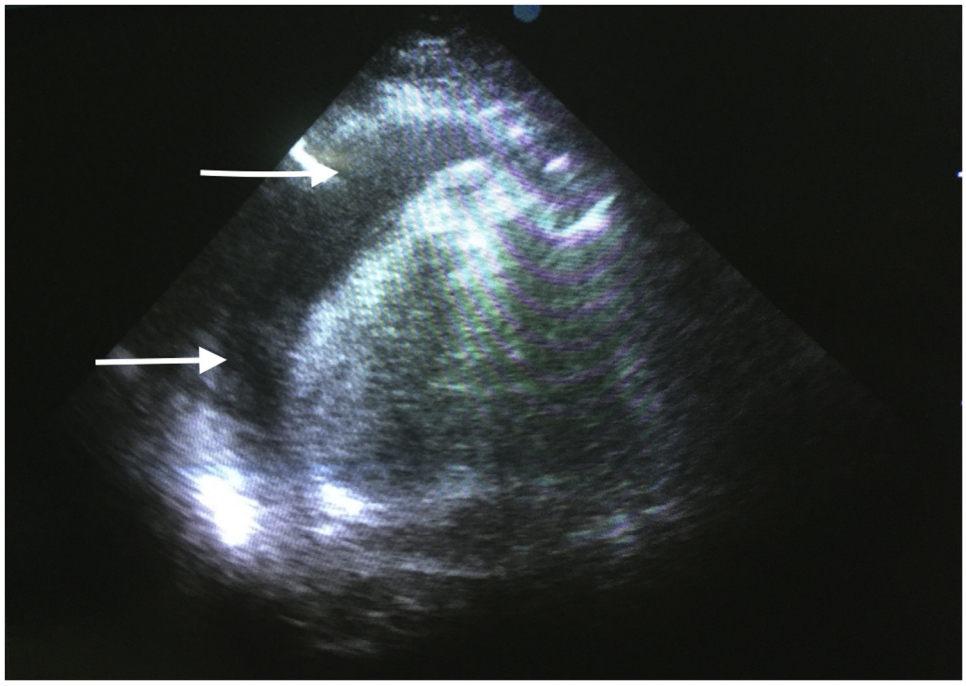

Fig. 1. Pericardial effusion causing cardiac tamponade.

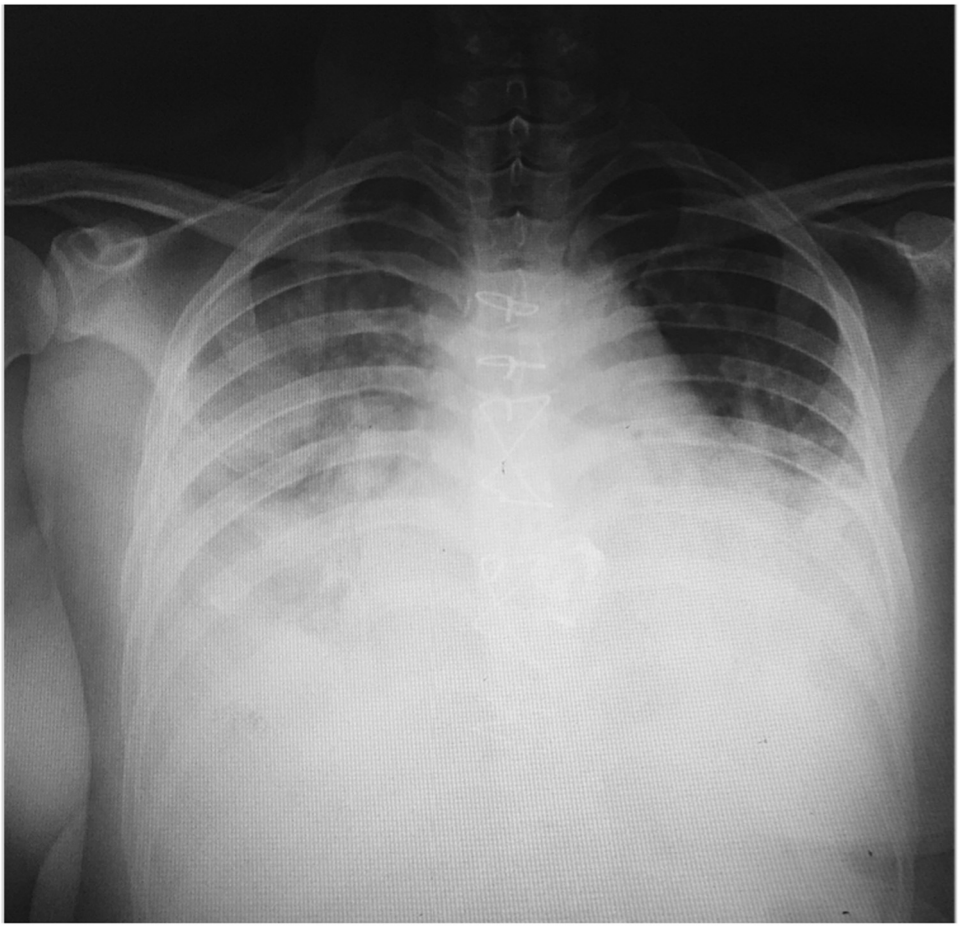

Fig. 2. Chest X-ray shows bilateral pulmonary edema.

right ventricle (Fig. 1) and good systolic function of the left ventricle (ejection fraction was 60%). The pressure gradient across the prosthetic mitral valve was within expected limits. Blood count and biochemistry were within normal limits. Hemodynamic mon-

itoring with a Swan–Ganz catheter confirmed the diagnosis of tamponade. The prothrombin time was prolonged due to warfarin usage and fresh frozen plasma and vitamin K intravenously was applied slowly. After acceptable levels of INR achieved, an emergent rapid drainage of the pleural and pericardial cavity with left anterior mini thoracotomy was carried out. After creation of pericardiopleural window, by excising a large piece of pericardium anterior to the phrenic nerve, more than 1000 ml of serosanguineous fluid were quickly removed from the pericardial space during the procedure, and both right atrium and pulmonary capillary wedge pressures (PCWP) transiently dropped to near-normal values. The chest was closed as per routine, two chest tubes was placed, one in the pericardial cavity and the other in the left pleural cavity.

After the decompression of the heart patient was transferred to intensive care unit and monitored, during the following hours of the decompression, the patient's condition deteriorated. She became increasingly breathless, tachypneic, tachycardic, and blood pressure decreased progressively. Overt pulmonary edema developed within hours, bilateral rales were audible on physical examination, and PCWP increased to 28 mm Hg. A chest X-ray performed at that time showed bilateral pulmonary edema (Fig. 2). Intravenous dobutamine, nitroglycerin, norepinephrine and furosemide therapy was initiated for hemodynamic support and pulmonary edema. On the second day of intensive care unit (ICU), a transthoracic echocardiogram repeated, very little pericardial effusion without hemodynamic compromise, but systolic dysfunction of both ventricles, global diffuse hypokinesia, 15–20% LVEF was found. High-dose inotropic support with dobutamine and diuretics was continued, continuous positive airway pressure (CPAP) therapy was used for her respiratory distress at times.

During follow up she was progressively weaned off inotropes, ejection fraction raised to 35% on control TTE. Two weeks later, she was discharged from the hospital on the therapy of beta blockers, digoxin, warfarin, and furosemide. At discharge, repeated TTE showed normal LV and RV function, LV ejection fraction was 50%, and normally functioning prosthetic mitral valve.

3. Discussion

Acute cardiac failure is a very unusual complication after removal of pericardial effusion for cardiac tamponade. We describe a case with a normal LV systolic function at baseline who developed transient biventricular heart failure immediately after the pericardial window drainage. This syndrome was first described by Vandyke et al. in 1983 and Angouras et al. proposed the use of the term "pericardial decompression syndrome" [1,3] to gain wider recognition of this potentially fatal complication. The incidence of this rare complication was reported to be 4.8% (5 of 104 patients with pericardial effusion who underwent surgical subxiphoid pericardiostomy) by Angouras et al. [2,3].

Causes underlying the PDS can be identified in very few patients. Pathologies which affects myocardial performance such as malignant myocardial infiltration or chemotherapy induced cardiomyopathy may be considered causative factors [1,4]. Yet, in the majority of cases there is no obvious cause for the development of paradoxical ventricular dysfunction after decompression, as observed in our case.

Various hypotheses have been raised to explain the pathophysiology of this phenomenon, but a single pathway has not yet been elucidated. Possible mechanisms implicated in the post-pericardial decompression syndrome are: A rapid drainage of a large pericardial effusion, could lead to a disproportionate increase in right ventricular (RV) end-diastolic volume compared with left ventricular (LV) end-diastolic volume. This interventricular volume mismatch in the presence of vasoconstriction due to high catecholamine levels could lead to an increase in LV end-diastolic pressure and transient LV dysfunction for the previously diseased heart [2,5].

An acute increase in "wall stress" due to the acute distension of the cardiac chambers secondary to increased venous return at high filling pressures, combined with a negative pressure in the pericardial cavity immediately after large volume pericardiocentesis may be another mechanism [5,6].

In our patient, both ventricles were hypokinetic and CVP returned to a high following the transient decrease during evacuation of effusion; this supports that these mechanisms might be responsible for the cardiac failure however we cannot rule out other proposed mechanisms that might have lead to cardiac failure.

Myocardial stunning due to mismatch of oxygen supply for the acutely increased wall stress in myocardium might have resulted. Tamponade resulted in a disproportionately greater decrease of the coronary blood flow, but any occult dysfunction may be masked by the reduction in chamber sizes and associated tachycardia [2,5,6].

The autonomic nerve system is also likely to have influenced the cardiac function before and after pericardiocentesis. Wolfe and Edelman [4] argued in a case report that the removal of the stimulus for sympathetic outflow (the drainage of pericardial effusion) might well lead to the unmasking of left ventricular dysfunction, which was obscured due to the high catecholamine levels producing transient tachycardia and inotropic effect.

Many literature articles support, post pericardial drainage low cardiac output syndrome can complicate any method of surgical pericardial drainage even in patients with apparently normal myocardium and preoperative hemodynamic status [1,7]. Therefore, these patients require close postoperative monitoring for the first 24 h, preferably in the ICU, and aggressive heart failure treatments should be managed if low cardiac output develops.

4. Conclusion

In conclusion, despite the multiple etiologies of pericardial effusions and preexisting heart conditions in the previously reported cases, it is likely that the mechanism underlying such hemodynamic derangement is rapid evacuation of a large volume of pericardial effusion. We recommend gradual removal of pericardial effusion under hemodynamic monitoring, especially in patient with postcardiotomy tamponade.

This study has been reported in line with the SCARE criteria [8].

Informed consent

Written informed consent was obtained from patient who participated in this case.

Ethics committee approval

Ethics committee approval was received for this study from the The Institutional Ethics Committee of Siyami Ersek Hospital.

Conflict of interest

The authors declare that there are no conflicts of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution

S. Albeyoglu: Study concept, data collection, writing paper. M. Aldag: Design, data analysis, writing paper. U. Ciloglu: Data Collection, data analysis. H. Kutlu: Data Collection, data analysis. S. Dagsali: Data analysis and interpretation, Concept.

Guarantor

Sebnem Albeyoglu M.D., Mustafa Aldag M.D.

Acknowledgement

None to declare.

References

[1] Feridoun Sabzi, Reza Faraji, Predictors of post pericardiotomy low cardiac output syndrome in patients with pericardial effusion, J. Cardiovasc. Thorac. Res. 7 (1) (2015) 18–23.

[2] T. Dosios, N. Theakos, D. Angouras, P. Asimacopoulos, Risk factors affecting the survival of patients with pericardial effusion submitted to subxiphoid pericardiostomy, Chest 124 (July (1)) (2003) 242–246.

[3] A. Chamoun, R. Cenz, A. Mager, A. Rahman, C. Champion, M. Ahmad, Y. Birnbaum, Acute left ventricular failure after large volume pericardiocentesis, Clin. Cardiol. 26 (2003) 588–590.

[4] C. Ligero, R. Leta, A. Bayes-Genis, Transient biventricular dysfunction following pericardiocentesis, Eur. J. Heart Fail. 8 (2006) 102–104.

[5] E.I. Skalidis, G.E. Kochiadakis, S.I. Chrysostomakis, et al., Effect of pericardial pressure on human coronary circulation, Chest 117 (2000) 910–912.

[6] M.M. Shenoy, S. Dhar, R. Gittin, A.K. Sinha, M. Sabado, Pulmonary edema following pericardiotomy for cardiac tamponade, Chest 86 (1984) 647–648.

[7] B. Neelakandan, N. Jayanthi, R. Kanthimathi, Subxiphoid drainage for pericardial tamponade, J. Thorac. Cardiovasc. Surg. 111 (1996) 489.

[8] R.A. Agha, A.J. Fowler, A. Saetta, I. Barai, S. Rajmohan, D.P. Orgill, SCARE Group, The SCARE statement: consensus-based surgical case report guidelines, Int. J. Surg. (2016) (article in press).