An Uncommon But Dangerous Complication After Successful Pericardiocentesis: Pericardial Decompression Syndrome

Gamze Babur Güler1, Ekrem Güler1, Gültekin Günhan Demir1, Hacı Murat Güneş1

1 İstanbul Medipol Üniversitesi Tıp Fakültesi, Kardiyololoji Anabilim Dalı, İstanbul, Türkiye

ABSTRACT

Pericardial decompression syndrome (PDS) is an infrequent but mostly fatal complication after successful pericardiocentesis. We present a case of a 27 year-old female patient with suspected malignancy and cardiac tamponade treated with pericardiocentesis followed by acute pulmonary edema and cardiac arrest 2 hours later. We also reviewed possible mechanisms involved in the PDS pathophysiology in the context of literature data.

Keywords: Pericardiocentesis, decompression, tamponade

Başarılı Perikardiyosentez Sonrası Nadir ve Tehlikeli Bir Komplikasyon: Perikardiyal Dekompresyon Sendromu

Gamze Babur Güler1, Ekrem Güler1, Gültekin Günhan Demir1, Hacı Murat Güneş1

1 İstanbul Medipol Üniversitesi Tıp Fakültesi, Kardiyololoji Anabilim Dalı, İstanbul, Türkiye

ÖZET

Perikardiyal dekompresyon sendromu (PDS), başarılı perikardiyosentez sonrası görülebilecek nadir ancak ölüm bir tablodur. 27 yaşında malignite sebebiyle takip edilen, kardiyak tamponad sebebiyle perikardiyosentez yapılan ve sonraki 2. saatte dispne, akut pulmoner ödem ve kardiyak arrest gelişen vakayı sunduk. PDS patofizyolojisinde etkili mekanizmalar ve olası tedavi literatür bağlamında gözden geçirildi.

Anahtar Kelimeler: Perikardiyosentez, dekompresyon, tamponad

Geliş Tarihi: 16.09.2015 - **Kabul Tarihi:** 08.10.2015

Introduction

Pericardiocentesis is a life-saving procedure commonly performed in cardiology practice. Injuries of cardiac wall, coronary arteries and lungs are well-known complications; however pericardial decompression syndrome (PDS), a serious but less known complication of pericardiocentesis (1). We aimed to present a case of PDS following pericardiocentesis in a patient with suspected malignancy and cardiac tamponade and also to review the current literature.

Case presentation

A 27 year-old female patient with ovarian mass was evaluated for malignancy and presented with progressive shortness of breathe and palpitation symptoms. The patient has orthopnea, jugular venous pressure was increased and the heart sounds were muffled on auscultation. On physical examination blood pressure was 100/60 mmHg, heart rate 106 bpm, respiratory rate was 25/min and saturated oxygen was 94 %. Chest-X ray and computed tomography showed enlarged cardiac silhouette and bilateral pleural effusion (Figure 1, Figure 2). Echocardiographic examination revealed pericardial effusion surrounding the entire heart. Signs of cardiac tamponade were detected and pericardiocentesis was scheduled. After obtaining informed consent and local anesthesia, initially 500 ml and then 480 ml of serous fluid was drained via subxyphoid route. Symptoms of the patient improved right after the procedure but two-hours later a new-onset dyspnea, tachycardia and hypotension developed. The patient was intubated for progressive respiratory failure and control echocardiography showed no pericardial effusion. However left ventricular ejection fraction which was normal before the index procedure, was measured 20 % with global hypokinesia. The patient was followed-up with inotropic support but shortly afterwards bradycardia and cardiac arrest occurred and cardiopulmonary resuscitation was undertaken. The patient died despite all supportive treatment.

Discussion

We described a case of PDS presenting with pulmonary edema and cardiogenic shock following pericardiocentesis. Various definitions have been used in the literature for PDS (2).

Estimated prevalence for this mortal complication was reported as 4,8 percent (3). In the analysis of a case-series of 35 patients with PDS, it was reported that the onset of PDS after the procedure ranged from seconds to 48 hours. Mortality was seen in 10 patients (29%) and it was only associated with surgical drainage. Various clinical presentations of PDS were recorded as follows; 10 (29%) with cardiogenic pulmonary edema without shock, 14 (40%) with left ventricular failure, three (9%) with right ventricular failure, seven (20%) with biventricular failure, and one (3%) with non-cardiogenic pulmonary edema (4). In our case pulmonary edema and subsequent cardiogenic shock occurred 2 hours after the procedure.

The underlying mechanism of PDS remains unclear however autonomic dysfunction, hemodynamic overload and ischemia hypotheses were postulated. According to the sympathetic overdrive hypothesis, transient tachycardia and inotropic effect caused by excessive catecholamine release diminishes following pericardial drainage. Altered sympathetic tone uncovers masked left ventricular dysfunction (5).

Increased coronary vascular resistance caused by increased pericardial pressure result in a decline in coronary blood flow (6). It is assumed that mechanical pressure of increased pericardial pressure on coronary arteries leads to myocardial stunning and hibernation which end up as transient systolic dysfunction. Moreover malignant myocardial infiltration and chemotherapy suppress systolic functions

particularly in PDS cases associated with malignancy (2). Basmaji et al. performed nuclear perfusion imaging in their PDS case and detected fixed perfusion defects without any inducible ischemia. Coronary angiography revealed no significant stenosis and EF was 57 % in magnetic resonance imaging (7). In our case it was not possible to investigate ischemia and myocardial disease due to failed resuscitation.

Hemodynamic hypothesis suggests pulmonary edema as a consequence of preload-afterload mismatch. Rapid drainage of pericardial effusion increases sudden increase in venous return and causes overloaded left ventricle (8). Besides, increased right cardiac output in comparison with left cardiac output might have produced ventricular dysfunction (9).

PDS treatment includes supportive therapy for pulmonary edema and cardiogenic shock. Gradual drainage was suggested for prevention of PDS. However there is no data regarding the efficacy and safety of the amount of drainage fluid. Current hypotheses suggest impaired cardiac functions even in the absence of PDS symptoms. Further investigation of optimal drainage rate of pericardial fluid and its impact on cardiac functions with new echocardiographic or invasive imaging methods will help better understanding of PDS.

Conclusion

Utilization of new echocardiographic imaging modalities might help prevention of PDS by determining optimal rate and amount of drainage fluid.

References

1-Karamichalis JM, Gursky A, Valaulikar G, Pate JW, Weiman DS. Acute pulmonary edema after pericardial drainage for cardiac tamponade. Ann Thorac Surg 2009;88:675-677.

2-Angouras DC, Dosios T. Pericardial decompression syndrome: a term for a well-defined but rather underreported complication of pericardial drainage. Ann Thorac Surg 2010;89:1702-1703.

3-Dosios T, Theakos N, Angouras D, Asimacopoulos P. Risk factors affecting the survival of patients with pericardial effusion submitted to subxiphoid pericardiostomy. Chest 2003;124:242–246.

4-Pradhan R, Okabe T, Yoshida K, Angouras DC, DeCaro MV, Marhefka GD. Patient characteristics and predictors of mortality associated with pericardial decompression syndrome: a comprehensive analysis of published cases. Eur Heart J Acute Cardiovasc Care 2014 Sep 1.

5-Wolfe MW, Edelman ER. Transient systolic dysfunction after relief of cardiac tamponade. Ann Intern Med 1993;119:42– 44.

6-Skalidis EI, Kochiadakis GE, Chrysostomakis SI, Igoumenidis NE, Manios EG, Vardas PE. Effect of pericardial pressure on human coronary circulation. Chest 2000;117:910-912.

7-Basmaji SG, Peretz-Larochelle M, Bernier ML. Pericardial decompression syndrome: A rare and potentially dramatic complication of pericardiocentesis. Int J Cardiol 2015;178:297-298.

8-Vandyke Jr WH, Cure J, Chakko CS, Gheorghiade M. Pulmonary edema after pericardiocentesis for cardiac tamponade. N Engl J Med 1983;309:595– 596.

9-Konstam MA, Levine HJ. Pulmonary edema after pericardiocentesis. N Engl J Med 1984;310:391.

Figure Legends

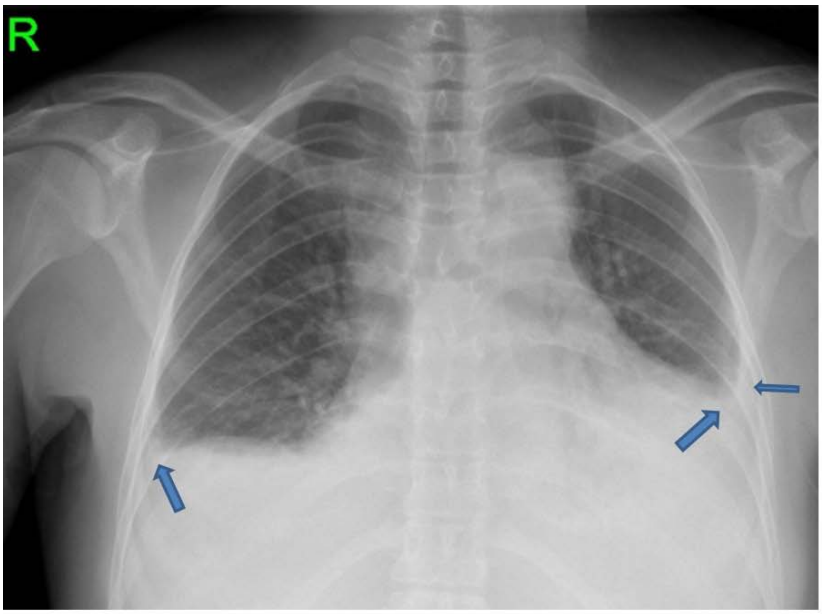

Figure 1: Chest X-ray taken on antero-posterior view show Cardio Thoracic Index (CTI)> 0,5 and bilateral pleural effusion (blue arrow).

DOI: 10.5578/khj.10324

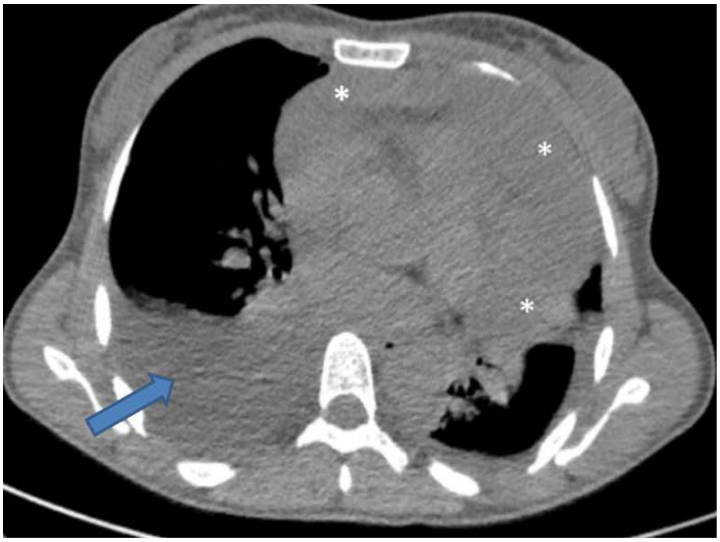

Figure 2 : CT image showing pericardial effusion (*) and pleural effusion (blue arrow) .