CASE REPORT

Management of a patient with pericardial decompression syndrome and HOCM

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Received 25 June 2015

Revised 9 May 2016

Accepted 15 May 2016

SUMMARY

A 44-year-old man, with a history of arterial hypertension, was referred with increasing shortness of breath due to a large pericardial effusion and imminent tamponade. Emergency ultrasound-guided pericardiocentesis resulted in the rapid withdrawal of 2760 cc of serous fluid. 3 hours later, the patient developed acute pulmonary oedema, which was successfully treated. Hypertrophic obstructive cardiomyopathy was later diagnosed and malignancy was excluded as a cause of the effusion. Clinicians performing pericardiocentesis need to be aware of pericardial decompression syndrome (PDS), a rare but serious complication. Although the underlying mechanisms causing PDS are not fully understood, patients with high left ventricular (LV) filling pressures are at particular risk. In other words: diastolic dysfunction of the LV is a risk factor for the occurrence of PDS.

BACKGROUND

Pericardial decompression syndrome (PDS) or acute pulmonary oedema following pericardial drainage, is a rare but life-threatening complication of pericardiocentesis. The underlying mechanisms causing PDS are not yet fully understood but many authors agree to a multifactorial genesis of this disorder.^{1–3} In the scientific literature, PDS has mostly been associated with neoplasia.^{1 2}

CASE PRESENTATION

We report the case of an otherwise healthy 44-year-old man, with a history of systemic hypertension controlled by ramipril, referred to our emergency department (ED) from a cardiology practice, with shortness of breath in the setting of imminent pericardial tamponade. The patient had a 1-week history of dyspnoea, with noticeable worsening over the last days before presentation. Although his vitals were still normal on admission, we nevertheless decided—based on the clinical distress of the patient—to rapidly perform an echocardiography-guided pericardiocentesis through a subxiphoidal window, leading to immediate clinical relief after the withdrawal of 2760 cc serosanguineous fluid. After 3 hours, the patient reported anew of increasing dyspnoea at rest. Chest X-ray showed newly spreading massive pulmonary oedema, which led us to immediately start diuretics and oxygen inhalation. These measures brought quick relief of the dyspnoea (figures 1–3). The next day, the patient received 80 mg of gentamicin prophylactically⁴ through an intrapericardial catheter. Ibuprofen 400 mg three

Figure 1 Chest posterioranterior immediately after pericardiocentesis.

times a day was also started. Mildly elevated C reactive protein (CRP) and leucocytes in the blood analysis additionally prompted us to start a course of ceftriaxone 2 g daily (for 5 days). Cytological evaluation of the pericardial effusion demonstrated erythrocytes and leucocytes, but no malignant cells; in addition, microbiological culture yielded no positive results. Serial echocardiography was noticeable for minimal effusion without haemodynamic relevance, and blood CRP and leucocytes normalised on day 3. Interestingly, echocardiography also revealed hypertrophic obstructive cardiomyopathy (figures 4 and 5), with a maximal left ventricular (LV) outflow tract (LVOT) pressure gradient at rest of 83 mm Hg (mean pressure gradient: 52 mm Hg), positive systolic anterior motion, left-ventricular ejection fraction of 60% and a septum thickness of between 30 and 40 mm. The patient had no history of cardiac disease. We also performed a cardiac

Figure 2 Chest anteroposterior 3 hours later, note spreading pulmonary oedema.

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To cite: Fozing T, Zouri N, Adam O, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-211550

Figure 3 Chest posterioranterior after treatment with diuretics, 12 hours after [figure 2](#) had been taken.

Figure 4 Apical four-chamber view echocardiogram: note massive left ventricular hypertrophy and residual pericardial effusion after drainage (arrow).

Figure 5 Massive left ventricular outflow tract obstruction with maximum resting gradient of 83.79 mm Hg.

catheter examination in order to rule out coronary heart disease. However, massive diastolic dysfunction with left ventricular (LV) end-diastolic pressure around 36 mm Hg was confirmed, as expected. Neoplasia could also be excluded as the cause of pericardial effusion through further investigations (thoracic and abdominal CT scans) and the patient was finally referred to our thoracic surgery division for septal myectomy. The latter was completed 4 weeks later. Follow-up echocardiography preoperatively and 6 weeks after myectomy revealed, respectively, minimal and no pericardial effusion. Ibuprofen was discontinued after 4 weeks of treatment in total. Endomyocardial biopsy samples gathered during myectomy showed only hypertrophic myocytes and fibrous tissue.

DISCUSSION

PDS is a known, although rare, complication of pericardial fluid drainage.^{1–3} Most of the cases encountered in the current scientific literature describe patients having malignant effusion.^{1–3} Cases of PDS associated with infectious,³ endocrine⁵ and traumatic⁶ effusions are less frequent. In our case, neoplasia could be ruled out (clinically, CT scan, and laboratory analysis) and the effusion fully resolved after non-steroidal anti-inflammatory drugs (NSAID) and adjuvant antibiotic therapy (intravenous antibiotics had been initially administered to the patient because of transient CRP and leucocyte count elevation); therefore, we hypothesised that the effusion had been inflammatory or parainfectious in origin. Certainly more sensitive tests such as PCR and next generation sequencing next-generation sequencing could have helped identify the causative organism behind this effusion, but we instead decided—in keeping with our routine practice—to use an *ex juvantibus* approach to solving this case: the combination of gentamicin, which is also used as a sclerosing agent, ceftriaxone as a systemic antibiotic and ibuprofen as an NSAID, led to a full recovery and prevented disease relapse after the effusion had been drained. Finally, it may be stated that the patient's outcome justified this approach.

In reviewing the current literature, the main hypotheses for the pathogenesis of PDS can be summarised by three hypotheses:

1. The haemodynamic hypothesis suggests that rapid release of the tamponade leads to a sudden increase in pulmonary venous return, that is, greater preload, while adaptive

systemic vascular resistance (after-load) remains high, partly due to high catecholamine levels in the setting of pericardial tamponade. Pulmonary oedema, thus, results from a preload/after-load mismatch.¹⁻³

2. In the ischaemic hypothesis, myocardium damage occurs due to impaired coronary artery blood flow from compression by the pericardial fluid. Altered intramyocardial blood distribution then causes stunning of the myocardium, myocardial ischaemia and subendothelial haemorrhage. The resulting myocardial dysfunction persists long after relief of the tamponade and subsequently induces pulmonary oedema.¹⁻³
3. In the sympathetic overdrive hypothesis, pericardial fluid is a potent stimulus for the sympathetic nervous system. Removal of that stimulus may uncover a latent myocardial dysfunction previously compensated by high catecholamine levels.¹⁻³

In our case, no impairment of systolic LV-function was identified; rather, massive diastolic dysfunction, that is, elevated filling pressures in conjunction with LVOT obstruction, was noted. Increased venous return after effusion drainage combined with this LVOT obstruction (elevated after load) and non-compliant left ventricle seem to have had a synergistic effect in causing PDS, such as defined in the haemodynamic hypothesis.

This case also features two seemingly distinct diseases occurring in the same patient. Although cases of concurrent hypertrophic cardiomyopathy and pericarditis have been presented in the literature,⁷ we are not aware of any direct link between these two entities.

We found no clear guidelines and no recommendations in the literature for the quantity of fluid that may be safely drained concurrently. Wolfe and Edelman⁸ described patients with PDS after drainage of 650 mL of effusion; however, in our daily practice and in the literature, drainage of more than 2000 mL at one time has been described, without development of PDS.⁶ Vandyke *et al*⁹ proposed that the fluid should be drained gradually in steps of <1 L at a time, over 24 hours, to allow adaptive changes in myocardium to take place. However, Al Banna and Husain³ also reported the case of a patient developing biventricular systolic dysfunction and pulmonary oedema after slowly draining 150 mL of infectious pericardial effusion over 4 hours.

Acknowledgements The authors would like to thank Axel Tost, Mahmud Atassi and Tarek Jarkas, for their kind support.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

Learning points

- In conclusion, although pericardial decompression syndrome (PDS) remains a complication mostly described after the drainage of malignant pericardial effusion, it is also encountered in effusion of other aetiologies.
- To date, it is still unclear how much fluid at one time and at what flow speed should be drained to avoid PDS.
- In reviewing the current literature and in considering our case, decompression celerity, quantity of drained fluid and left-ventricular dysfunction (either systolic and diastolic) seem to play, among other factors, a major role in the incidence of PDS.
- In situations where a relevant diastolic dysfunction is already present, increased venous return after drainage, resulting in a preload/after-load mismatch, appears to be the main pathophysiological mechanism causing PDS, as stated in the haemodynamic hypothesis. In other words: diastolic dysfunction of the LV is a risk factor for the occurrence of PDS.
- As a result of this variety of factors, clinicians might not always be able to avoid PDS, especially in an emergency. They need to be on the lookout for this complication and to closely monitor their patients until the tamponade physiology fully resolves. It is also possible that varying degrees of this condition go unnoticed in daily clinical practice.

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