Case Report

A case of cardiac tamponade with a “small” pericardial effusion and superior vena cava syndrome

Tapas Mondal, Taheera Ramji and Ashkan Javidan*

Department of Pediatrics, McMaster University, Hamilton, ON, Canada

Received 19 October 2012

Revised 7 December 2012

Accepted 18 December 2012

Abstract. In this article, we report a case of a previously healthy 11-year-old male who presented to our university hospital with a mediastinal mass, a left and right pleural effusion, and a small pericardial effusion of 5 mm, detected on computed tomography scan and echocardiography. On assessment, he had all the clinical features of superior vena cava syndrome and tamponade, including muffled heart sounds, tachycardia, and pulsus paradoxus. The patient developed increasing respiratory distress requiring tracheal intubation and mechanical ventilation, and despite drainage of his larger pleural effusion, continued to experience symptoms of respiratory distress. Finally, the small pericardial effusion was confirmed on echocardiography and drained after a clinical diagnosis of cardiac tamponade, leading to dramatic improvement in the patient’s cardiovascular status. Curiously, on pericardiocentesis, much less fluid was obtained than expected for his presenting symptoms and clinical course.

Keywords: Cardiac tamponade, pericardial effusion, mediastinal cancer, lymphoblastic lymphoma, pericardiocentesis, pediatrics

1. Introduction

Although cardiac tamponade can be uncommon, it is a life threatening medical emergency requiring prompt diagnosis and treatment. It is generally a result of slow or rapid compression of the heart secondary to accumulating pericardial fluid [1]. Both the quantity of fluid and the rate of accumulation are causal factors in the clinical presentation of cardiac tamponade. A small, acute effusion can induce the same presentation in a patient as a large chronic effusion [2]. In both situations, fluid accumulates under pressure and compresses the

cardiac chambers. This increased intra-pericardial pressure approximates intracardiac pressure, reducing myocardial diastolic compliance and limiting cardiac inflow and filling, ultimately manifesting as hemodynamic compromise [3]. The result is a decline in cardiac output and ventricular stroke volume leading to hypotensive shock, circulatory collapse, and potentially, death if not diagnosed early [1,3]. The objective of this article is to report on a rare, acute case of pediatric cardiac tamponade, secondary to a small pericardial effusion and mediastinal mass and to provide a brief discussion of confounding factors that contribute to such a rare pathogenesis. Numerous reports have described cardiac tamponade secondary to a mediastinal mass; however, to our knowledge, this is the first case of tamponade caused by a small pericardial effusion [4–8].

*Corresponding author: Ashkan Javidan, Department of Pediatrics, McMaster University Health Sciences Centre-3A35, 1200 Main Street West, Hamilton, ON, Canada, L8S 2A5. Tel.: +1 905 962 7763; Fax: +1 905 521 2333; E-mail: javidaa@mcmaster.ca.

2. Case report

An 11-year-old male presented to the emergency department with a 1-wk history of fever and malaise. For 3 d prior to presentation, he also experienced dyspnea, particularly when lying in the supine position, facial swelling, decreased energy, anorexia, nausea and one episode of emesis. Over the past 48 h, his mother had noticed increasing periorbital swelling. There was no previous history of hospitalizations or surgery. He was not on medications. His family history was unremarkable and his growth and development to date were normal.

On initial examination, he was alert and oriented but visibly short of breath. There was a noticeable plethoric appearance with periorbital edema. He was tachycardic with a heart rate of 115 bpm, tachypneic with respiratory rate of 35 breaths/min, blood pressure of 126/88 mmHg and oxygen saturation 96% in room air. There was upper body venous distension and engorgement. He had a friction rub. Pulses were palpable and found to be regular. On cardiac and respiratory examination, he had muffled heart sounds with subcostal indrawing and no audible breath sounds in the right lung field. Neurological and abdominal examinations were normal.

Initial evaluation by the pediatric team included a chest X-ray showing a large, right-sided pleural effusion as well as a mediastinal mass. A computed tomography (CT) scan of his chest was performed the same day, which confirmed the large, right-sided pleural effusion, a small, left-sided pleural effusion, and the anterior mediastinal mass measuring $10 \times 8.6 \times 4.4$ cm, causing severe narrowing of the superior vena cava and compression of the right main stem bronchus. No cardiac invasion or chamber compression was present due to the mediastinal mass, either on echocardiogram or CT scan. There was also a small pericardial effusion present, which measured as 5 mm in diameter (Fig. 1). Secondary to these findings, he was admitted to the pediatric intensive care unit for further management and observation. The presentation of periorbital edema in the absence of generalized edema led to a clinical suspicion of superior mediastinal syndrome and the patient was not administered diuretics. Analgesics were administered to alleviate discomfort.

The following morning, he developed increasing respiratory distress, which initially required bilevel positive airway pressure support before escalating eventually to tracheal intubation and invasive mechanical ventilation. A chest tube was emergently inserted

Fig. 1. Initial computed tomography chest demonstrating large right-sided pleural effusion, small left side pleural effusion, anterior mediastinal mass and 5 mm pericardial effusion measured during diastole.

on the right side of the chest to drain his pleural effusion with a large amount of resultant drainage (Fig. 2). Upon drainage of the large right-sided pleural effusion, the patient continued to remain irritable when lying down and was left in a semi-upright position. Two boluses of intra-venous fluid (5 mL/kg) were administered through a lower limb peripheral intravenous catheter. A pericardial effusion was verified on echocardiogram that morning (Fig. 3). Interestingly, there were significant differences observed in the size of the pericardial effusion during inspiration and expiration. During inspiration, with increased ventricular filling and coinciding with diastole, the effusion was minimal. Despite this perplexing presentation, there was a clinical diagnosis of tamponade based on findings of pulsus paradoxus, right ventricular diastolic collapse on echocardiogram (Fig. 4) and, pulsed-wave Doppler echocardiography for mitral inflow velocities, displaying significant respiratory variation (Fig. 5).

Pericardiocentesis was promptly performed with evidence of improvement of the right ventricular motion during the procedure. There was also a dramatic clinical improvement in the patient's irritability and respiratory function. He was able to lie supine with no difficulty. Surprisingly, only 30 mL of serosanguinous fluid was removed before improvement in cardiac function and resolution of pulsus paradoxus. The catheter was left in place for an additional 24 h with minimal drainage and then discontinued. Fig. 6 shows resolution of the tamponade a day later as shown on the echocardiogram. Fig. 7 outlines the repeat CT scan of his chest, demonstrating resolution of the pericardial effusion; the pericardiocentesis catheter tip is also seen. Biopsy of the mediastinal mass and samples of the pleural, pericardial and cerebrospinal fluid were examined with bone marrow aspiration samples, and it was concluded that the

Fig. 2. Chest X-ray demonstrating chest tube insertion for drainage of right pleural effusion. Large anterior mediastinal mass also verified.

Fig. 3. Echocardiogram (parasternal long axis view) demonstrating a small pericardial effusion immediately prior to pericardiocentesis.

Fig. 4. Echocardiogram demonstrating right ventricular diastolic collapse. Taken at the same time as figure 3.

Fig. 5. Pulsed-wave Doppler echocardiography of mitral inflow velocities demonstrating significant respiratory variation suggestive of cardiac tamponade; clinical equivalent is pulsus paradoxus.

Fig. 6. Echocardiogram one day post pericardiocentesis demonstrating resolution of pericardial effusion with pericardiocentesis drainage tube in place.

Fig. 7. Repeat computed tomography chest demonstrating resolution of pericardial effusion with pericardiocentesis catheter in place.

patient had precursor T-cell lymphoblastic lymphoma. He was started on chemotherapy as per protocol and tolerated treatment fairly well. There has not been any re-accumulation of his pericardial effusion since his initial diagnosis.

2. Discussion

Pericardial effusions and cardiac tamponade are rare entities in childhood, though not uncommon with mediastinal masses, particularly of the malignant type [4]. Pericardial fluid accumulations can vary in composition depending on the cause of the effusion. The fluid may be fibrinous, serous, purulent or hemorrhagic. Tamponade occurs when this fluid has accumulated to a level that compromises cardiac function [1]. In most healthy children, there is normally 10–15 mL of fluid found in the pericardial space, which serves as lubrication for the visceral and parietal layers of the pericardium [6]. The pericardium and pericardial fluid provide an important support system for hemodynamics and optimal cardiac function. The parietal pericardium contributes to increasing pressure in the right atrium and ventricle, and the subsequent resting diastolic pressure. The pericardium as a whole ensures uniform myocardium contraction by distributing force across the heart [1]. With increasing fluid in the pericardial space, intrapericardial pressure begins to slowly rise until an internal reserve volume threshold is reached, after which, both pressure and the rate of expansion rise rapidly. Once the intrapericardial pressure surpasses ventricular diastolic pressures, there is an increase in ventricular interdependence and subsequent reduction of left ventricular compliance and preload [1,3]. The result is compromised diastolic ventricular filling, elevated systemic and

pulmonary venous pressures and, ultimately, impaired cardiac output. While rapidly accumulating pericardial fluid readily induces these symptoms of cardiac tamponade, slowly progressing effusions can develop asymptotically with large accumulations of fluid due to pericardial compliance [9]. In essence, tamponade is an effect of decreased atrial and ventricular filling due to increasing pericardial pressure. Early diagnosis and intervention are imperative for successful management of tamponade [2,6,9].

Though cardiac tamponade is a clinical diagnosis, echocardiography is the most useful, non-invasive technique available to evaluate whether it is present as a result of pericardial effusion. It allows for rapid confirmation in unstable patients and can assess the relative role of tamponade contributing to hemodynamic compromise [3,8,10]. Finally, echocardiography reveals abnormalities in the right ventricle and atrium (diastolic collapse) that suggest a hemodynamically significant cardiac tamponade [3,10]. Cardiac tamponade is an indication for urgent pericardiocentesis, which is useful both as a diagnostic and therapeutic technique. Echocardiographic-guided pericardiocentesis with catheter drainage is the treatment strategy employed for effusions resulting in hemodynamically significant effusions [6,9].

We have presented a case of tamponade caused by a relatively small amount of pericardial fluid accumulation. Usually, attention is focused on pericardial fluid and pericardial pressures causing tamponade. It is uncommon for tamponade to be present as an outcome of a small pericardial effusion; however, of note is the fact that neoplastic disease is the underlying cause of 39% of pediatric cases of pericardial effusion [6]. Thus, there is a remarkable chance that filling pressure could already be compromised as in superior vena cava syndrome, allowing tamponade to occur with the accumulation of very little pericardial fluid [5]. This is what occurred in our case. The patient's mediastinal mass caused baseline impedance of ventricular filling. The small amount of added pericardial fluid would only exacerbate the situation, leading to even further compromise of ventricular filling and therefore, tamponade. Management of such a case is equally important. Even though diuretics have a role in chronic pericardial effusions, in this case, careful fluid resuscitation is necessary to maintain cardiac filling [6]. In addition, it is prudent in this setting that intravenous fluid be given through the inferior vena cava distribution, namely, infused in the lower limbs and not the upper limbs [5].

This case highlights the need to suspect cardiac tamponade, not based on the amount of pericardial effusion present, but rather, the presence of clinical findings in keeping with tamponade. It is important to remember that, if ventricular filling is already compromised, for example, secondary to compression of the superior vena cava or inferior vena cava, it is possible to present with symptoms of tamponade with a small pericardial effusion that has collected acutely. Management should be tailored appropriately in this situation so as to maintain cardiac filling.

References

- [1] Spodick DH. Pathophysiology of cardiac tamponade. *Chest* 1998;113(5):1372–8.
- [2] Saito Y, Donohue A, Attai S, Vahdat A, Brar R, Handapangoda I, et al. The syndrome of cardiac tamponade with “small” pericardial effusion. *Echocardiography* 2008;25(3):321–7.
- [3] Cohen ML. Experimental cardiac tamponade: correlation of pressure, flow velocity, and echocardiographic changes. *J Appl Physiol* 1990;69(3):924–31.
- [4] Bień E, Stefanowicz J, Aleszewicz-Baranowska J, Polczyńska K, Szolkiewicz A, Stachowicz-Stencel T, et al. Cardio-vascular disorders at the time of diagnosis of malignant solid tumours in children-own experiences. *Med Wieku Rozwoj* 2005;9(3 Pt 2): 551–9 (in Polish).
- [5] Gowda RM, Khan IA, Mehta NJ, Gowda MR, Hyde P, Vasavada BC, et al. Cardiac tamponade and superior vena cava syndrome in lung cancer-a case report. *Angiology* 2004;55(6):691–5.
- [6] Kühn B, Peters J, Marx GR, Breitbart RE. Etiology, management, and outcome of pediatric pericardial effusions. *Pediatr Cardiol* 2008;29(1):90–4.
- [7] Refaat MM, Katz WE. Neoplastic pericardial effusion. *Clin Cardiol* 2011;34(10):593–8.
- [8] Serlo WS, Heikkinen E. Cardiac tamponade caused by a mediastinal teratoma. *Scand J Thorac Cardiovasc Surg* 1983;17(3): 323–5.
- [9] Spodick DH. Acute cardiac tamponade. *N Engl J Med* 2003; 349(7):684–90.
- [10] Hoit BD. Pericardial disease and pericardial tamponade. *Crit Care Med* 2007;35(8 Suppl):S355–64.