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Acute Heart Failure after Evacuation of Large Volume of Pericardial Effusion by Pericardiostomy

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Abstract: In this report, we present two cases of acute left ventricular failure after large volume pericardiocentesis. Both cases presented with the symptoms of massive pericardial effusion which documented with Trans-Esophageal Echocardiography (TEE). The case No. 1 had the history of concomitant aortic and Mitral Valve Replacement (MVR and AVR), 2 months before the present admission. The case No. 2 had severe valvular PS and underwent valvotomy, PS repair and PFO occlusion, 2 weeks ago. An emergent pericardiostomy was performed for both cases and approximately 1000 mL of serous fluid was removed. However, after 2 h, the patients complained of symptoms of LV failure. High-dose inotropic support and other supportive measures were considered. The first case dead and the second survived.

Key words: Ventricular failure, pericardial effusion, acute heart failure, evacuation

INTRODUCTION

Pericardial tamponade, a life-threatening condition caused by the accumulation of fluid in the pericardial sac, is treated by drainage (Kabukcu *et al*., 2004). Surgical placement of a subxiphoid tube is the preferred technique for draining a small amount of effusion in patients with quickly developing pericardial tamponade, such as those with acute traumatic hemopericardium. For patients with massive effusion and slowly developing pericardial tamponade, there are 2 principal methods: Percutaneous catheter drainage and surgical tube drainage (Kabukcu *et al*., 2004).

The infrequency of effusive and compressive pericardial disease limits the feasibility of large, randomized studies to compare the effectiveness of treatment strategies. The choice of drainage method depends on the cause of the effusion, the patient's general health, the physician's experience and preference, and the facilities available. Because the management of cardiac tamponade is governed to such a large extent by institutional practice, it remains controversial (Kabukcu *et al*., 2004; Harada *et al*., 2002).

Acute left ventricular failure is one of rare complications of evacuation of large volume of pericardial effusion by pericardiocentesis or pericardiostomy (Ceron *et al*., 2003; Chamoun *et al*., 2003; Bastian *et al*., 2000). It seems that the chronic external support of the heart by the pericardial fluid, when rapidly released, may result to overdilatation of the heart, leading to systolic dysfunction and failure (Dosios *et al*., 2003).

We present here 2 cases of large volume evacuation of pericardial effusion followed by transient severe acute Left Ventricular (LV) systolic failure in the absence of any prior history of LV dysfunction. To our knowledge, few cases of acute heart failure after large volume pericardiocentesis have been reported.

CASE REPORT

Case No. 1: A 32-year-old woman with severe aortic insufficiency, moderate MR, moderate MS, and mild LV enlargement underwent concomitant Aortic and Mitral Valve Replacement (MVR and AVR), 2 months before the present admission. She had mild RV dysfunction after mentioned operation and had been discharged with good general condition, sinus rhythm and had pre- and post-operative LVEF of 35%. She had been taking digoxin, warfarin, and furosemide. During the week preceding the index admission she had developed progressive dyspnea with superimposed lower limbs edema. On admission, she had normal routine tests and renal and hepatic functional tests; Transthoracic Echocardiography (TTE) showed a large pericardial effusion with partial compression of the right and left heart. The pressure gradient across the prosthetic mitral and aortic valves was within expected levels. Her Electrocardiography (ECG) showed atrial fibrillation with rapid ventricular response and no electrical alternance (Fig. 1).

The patients underwent emergent pericardiostomy with the diagnosis of massive pericardial effusion with evacuation of more than 1000 mL of serous fluid. After

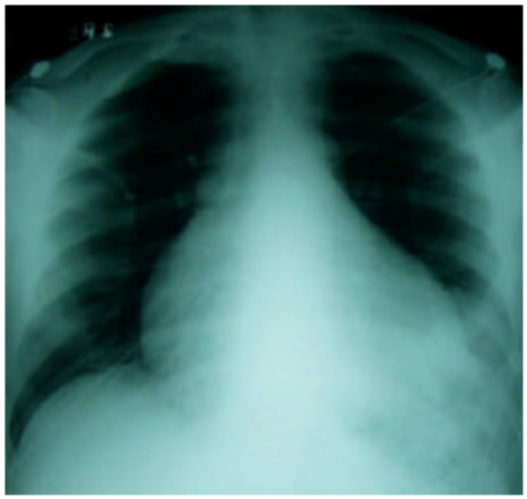

Fig. 1: Chest x-ray of the patient with prominent pericardiac effusion

pericardiostomy, she had tachypnea, tachycardia (HR = 140), BP = 80/60 and Hb = 9.4.

Two hours after this approach, tachycardia worsened and then, QRS widened. TTE showed that the function of mitral and aortic prosthetic valves is normal but there were moderate PE, global hypokinesia, LVEF<10% and RVEF<15%. Serial ABG analysis showed the progressive acidosis. An Intra-Aortic Balloon Pump (IABP) was inserted, the patient received high-dose inotropic support with dobutamine and diuretics.

Two days after pericardiostomy, despite the presence of IABP and high-dose inotropic support, QRS widened and severe bradycardia and hypotension appeared. Temporary pacemaker was replaced. However, VT and cardiogenic shock appeared, the CPR measures were failed and she dead.

Case No. 2: A 16-year-old girl with severe valvular PS (gradient = 170 mmHg) underwent PS valvotomy and PFO occlusion, 2 weeks ago. She discharged a week after operation with good general condition and gradient of 60-70 mm Hg (residual PS). She had taking propranolol 10 mg TDS.

On current admission (2 weeks after operation) she had tachypnea with initiation from a week ago with superimposed lower limbs edema.

TTE documented a massive pericardial effusion with partial compression of the right heart and Right Ventricular (RV) dilatation. The pressure gradient across the pulmonary valve was 30-40 mm Hg. Her ECG showed sinus tachycardia with electrical alternance.

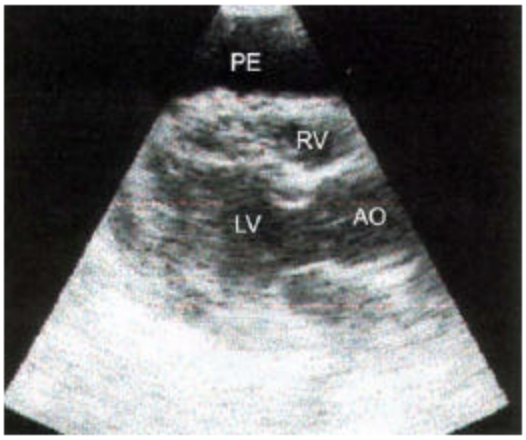

Fig. 2: There is prominent Pericardiac Effusion (PE) on the echocardiogram. AO: Aorta, LV: Left Ventricule, RV: Right Ventricule

An emergent pericardiostomy was performed and 1000 mL of serous fluid was removed. Immediate significant clinical improvement was noted.

A post-pericardiostomy TTE showed no residual pericardial effusion with normal LV systolic function (LVEF = 45-50%). The pressure gradient across the pulmonary valve was 25-40 mm Hg. Her post-ericardiostomy ECG showed normal sinus rhythm (HR = 80) without electrical alternance.

However, after 2 h, the patient complained of rapidly onset tachycardia, hypotension, and tachypnea. Repeated TTE showed severely depressed LV systolic function with akinesis of the anteroseptal and septal segments. There was not pericardial effusion. The left ventricle was dilated and its walls appeared thinner relative to the initial echocardiogram. The patient underwent ventricular and respiratory support with diagnosis of pulmonary edema and respiratory failure due to LV dysfunction. Low-dose captopril, digoxin and furosemide were initiated. Over the following week, the symptoms resolved. The patient was weaned off ventilator and discharged 5 days later (Fig. 2).

DISCUSSION

We describe a severe and fatal cardiac complication following evacuation of large volume of pericardial effusion by pericardiostomy.

The subxiphoid pericardiostomy is an expeditious, easy and inexpensive procedure, which can be applied to a wide spectrum of pericardial effusions. The technique provides accurate diagnosis and effective, durable treatment with an operation-related mortality ranging between 0 and 5% and a recurrence rate 0-9.1% (Dosios *et al*., 2003; Warren, 2003).

Several complications have been described after pericardiocentesis or pericardiotomy to relieve cardiac tamponade. Most of these complications are directly related to the surgical technique. Other complications, such as congestive heart failure or ventricular failure, are related to the failure of adaptive mechanisms to cope with the hemodynamic consequences of pericardial stress relief (Chamoun *et al*., 2003; Newland *et al*., 2002). Hemodynamic derangement as a complication of peri cardiocentesis has been rarely reported and includes cases of cardiogenic shock, cardiogenic pulmonary edema and Adult Respiratory Distress Syndrome (ARDS) (Chamoun *et al*., 2003; Newland *et al*., 2002; Geffroy *et al*., 2004).

It has been speculated that the mechanism of transient LV dysfunction after pericardiocentesis is related to the adaptive cardiac mechanisms to the increased intrapericardial pressure (Chamoun *et al*., 2003; Geffroy *et al*., 2004; Sunday *et al*., 1999). It seems that the chronic external support of the heart by the pericardial fluid, when rapidly released, may result to overdilatation of the heart, leading to systolic dysfunction and failure (Dosios *et al*., 2003).

External compression of the heart by large pericardial ef fusion leads to a decrease in stroke volume and cardiac output. During rapid large-volume pericardiocentesis, the release of pericardial constraint could lead to a disproportional increase in RV end-diastolic volume compared with LV end-diastolic volume (Spodick, 1997). This interventricular volume mismatch in the presence of vasoconstriction due to high catecholamine levels (Braverman and Sundaresan, 1994) could lead to an increase in LV end-diastolic pressure and transient LV systolic failure. Another proposed mechanism is myocardial stunning due to mismatch of oxygen supply across the myocardial wall. We believe an acute increase in wall stress (Laplace's law) due to the acute distention 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 physio-pathologic factor (Chamoun *et al*., 2003). The echocardiographic findings in the second case support this hypothesis.

Our first patient had long-standing LV dilatation due to severe aortic insufficiency that may have exacerbated after evacuation of the pericardial effusion.

CONCLUSION

Despite the preexisting heart conditions in the previously reported cases, it is likely that the mechanism underlying such hemo dynamic derangement is rapid evacuation of a large volume of pericardial effusion. We support previous recommendations for setting limitations for rate and volume of pericardial fluid evacuation to prevent this rare but potentially disastrous complication.

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