

A Case of Biventricular Failure after Pericardial Window for Large Pericardial Effusion

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INTRODUCTION

Pericardial tamponade resulting in hemodynamic compromise requiring either pericardiocentesis [Vandyke 1983] or subxiphoid pericardial window has been reported in literature [Armstrong 1984]. There are no large case series, only scattered case reports. Cardiac tamponade is known to affect the diastolic function of the heart but rare reports have documented systolic impairment of the left and right ventricle in the setting of tamponade [Vandyke 1983; Armstrong 1984]. We report a case of a transient biventricular systolic dysfunction in a patient with early cardiac tamponade after surgical drainage of pericardial effusion.

CASE REPORT

Our patient is a 37-year-old Hispanic woman who underwent subxiphoid pericardial window for a large pericardial effusion complicated postoperatively by biventricular systolic failure. Her medical history was significant for atrial fibrillation and rheumatic mitral stenosis and tricuspid regurgitation. She had undergone mitral valve replacement and tricuspid valve annuloplasty 3 months prior to current presentation. She presented three months after surgery with nausea, abdominal pain, and worsening dyspnea and palpitations. Clinical examination was significant for atrial fibrillation with rapid ventricular response, no pulsus paradoxus or elevated jugular venous pressure, clear lungs, and no peripheral edema. Her 2D echocardiogram at admission showed a large circumferential pericardial effusion with paradoxical septal motion, <50% tricuspid valve inflow variation, reduced left ventricular ejection fraction (EF) <50%, and hepatic vein collapse with inspiration. Also noted was minimal early diastolic

right ventricular (RV) collapse without right atrial collapse and moderate fibrinous strands around the heart. The patient was taken to the operating room for subxiphoid pericardial window; 1500 mL of serous fluid was drained without any immediate complications. Eleven hours postoperatively she developed shortness of breath with hypotension (blood pressure 72/48), tachycardia (heart rate in 120-130), and hypoxia (pulse oximetry 70% on 31/min nasal cannula). Her arterial blood gas analysis was consistent with severe metabolic acidosis. Her EKG revealed atrial fibrillation with RVR and a new RBBB. The patient was intubated and mechanical ventilation was initiated. A Swan-Ganz catheter was placed which showed CVP 29, PCWP 42, PAP 48/34, CO 2.41/min, CI 1.5 L/min/m², and SVR 1900. Emergent bedside echocardiogram showed trace pericardial effusion, severe left ventricular systolic dysfunction with EF <20%, and septal-apical akinesis and anterior and posterolateral wall hypokinesis. The RV function was also depressed. The patient required aggressive hemodynamic support with intra-aortic balloon pump (IABP), dobutamine, dopamine, and norepinephrine. She showed a gradual recovery, and was extubated on postoperative day 4. Her IABP was removed on postoperative day 5, her vasopressor and inotropic support was withdrawn by postoperative day 7, and she made a gradual recovery.

DISCUSSION

Acute biventricular failure following drainage of pericardial effusion is not commonly described. There have been few case reports in the past about occurrence of cardiogenic pulmonary edema and isolated right ventricular failure following large volume pericardiocentesis and surgical subxiphoid drainage for malignant and non-malignant pericardial effusion. Different hypotheses have been brought forward to explain this rare entity.

The most commonly accepted hypothesis is the sudden increase in preload due to relief of extrinsic pressure and ventricular dilatation in the setting of elevated systemic vascular resistance resulting in acute ventricular decompensation [Manyari 1983]. The presence of underlying myocardial ischemia or infarction may worsen this situation; Chamoun et al applied Laplace's law to explain this transient phenomenon

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[Chamoun 2003]. A sudden increase in ventricular diameter after removal of pericardial fluid increases the myocardial wall stress acutely and may thus precipitate ventricular failure.

Since the right ventricle is affected more due to extrinsic restriction caused by pericardial fluid, a larger increase in right ventricle output is expected after fluid drainage in comparison to the left ventricle. It may then cause elevated left atrial and pulmonary capillary wedge pressure, ultimately leading to left ventricle failure and pulmonary edema [Vandyke 1983].

Transient decrease in coronary perfusion has been described in canine models of cardiac tamponade in proportion to increasing intra-pericardial pressures. It may thus induce myocardial ischemia and stunning, which may contribute to myocardial systolic dysfunction in these patients.

Rowan et al described the role of sympathetic activity in paradoxical hypotension seen in some cases of pericardial tamponade after relief of obstruction [Rowan 2006]. In their case report, post-pericardiocentesis serum catecholamine levels dropped in concordance with the patient's blood pressure. Since mean arterial pressure (MAP) = cardiac output (CO) × systemic vascular resistance (SVR), a drop in SVR after drainage of pericardial tamponade may contribute to a drop in MAP. Low cardiac output in the setting of cardiac tamponade results in excessive catecholamine release to maintain MAP by increasing SVR. These catecholamines may also be responsible for masking an underlying left or right ventricular dysfunction. Once the tamponade is relieved and catecholamine levels fall toward normal, underlying ventricular dysfunction may declare itself. It may also explain the slight delay in our patient's clinical deterioration, but we do

not have catecholamine levels measured in this case.

A transient and reversible but severe systolic dysfunction of myocardium, although rarely described, may be seen in patients with large pericardial effusions, especially when they are removed rapidly. Several possible explanations include sudden increase in preload in the setting of high SVR, transient myocardial ischemia and stunning due to elevated intrapericardial pressures, and excessive catecholamine levels produced to maintain MAP. All of these may be clinically relevant.

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