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Improvement in Left Ventricular Systolic Function After Pericardiocentesis: Case Report

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Compression of the heart by pericardial fluid may produce impaired filling of the ventricle in diastole. If not countered by compensatory mechanisms, depressed cardiac output occurs. Cardiac tamponade may ensue causing reduced arterial blood pressure, narrow pulse pressure, tachycardia, elevated neck veins, pulsus paradoxus, and reduced perfusion of vital organs. Myocardial contractility, at least early on, is preserved, and ejection fraction increases in an effort to maintain cardiac output (1). Animal research has suggested that severe degrees of tamponade may result in transient left ventricular dysfunction (2,3). However, the effect of cardiac tamponade on left ventricular systolic function remains controversial (4,5). We report the case of a patient with a cardiomyopathic process and cardiac tamponade who experienced immediate improvement in left ventricular contractile function after pericardiocentesis.

Case Report

A 24-year-old black homosexual male was admitted for complaints of exertional dyspnea, three pillow orthopnea, and paroxysmal nocturnal dyspnea over the preceding month. He denied chest discomfort, leg edema, fever, chills, or cough. His medical history was significant for acquired immunodeficiency syndrome, Pneumocystis carinii pneumonia, and hepatitis B infection. He had no history of intravenous dmg abuse or alcoholism.

The patient appeared ill. His temperature was 36.7°C, pulse 125 beats/min and regular, blood pressure 110/90 mm Hg, and respiratory rate 34 breaths/min. A pulsus paradoxus of 16 mm Hg was present. Inspiratory and expiratory crackles were audible to the midlung fields bilaterally. Cardiac examination revealed diminished S1 and S2 capacity and the possible presence of an S3 gallop. Jugular venous pressure was elevated to the angle of the mandible. No murmurs were present. The point of maximal impulse was not palpable. The abdomen was soft. The liver percussed out to a span of 14 cm. Distal pulses were 1+/4 and symmetric.

Admitting electrocardiogram showed a sinus tachycardia with diffuse nonspecific ST and T wave changes. Chest x-ray revealed an enlarged cardiac silhouette and pulmonary vascular congestion. There was a question of a right middle lobe infiltrate. Chest x-ray one month earlier had been normal. Room air blood gases revealed a PaO2 of 88 torr and a mild respiratory alkalosis.

Cardiac tamponade was suspected based on the physical examination and radiographic evidence of an enlarged globular heart. An echocardiogram (Fig 1) showed a moderate-sized pericardial effusion with right atrial collapse consistent with early cardiac tamponade. There was also evidence of severe global left ventricular hypokinesis with an estimated ejection fraction of 12%. Pericardiocentesis was performed under ultrasound guidance. Approximately 435 mL of clear fluid was drained with

Fig 1—Two-dimensional echocardiogram illustrating depressed systolic function with pericardial effusion prior to pericardiocentesis. (A = aorta, LA = left atrium, LV = left ventricle, PE = pericardial fluid, RV = right ventricle, S = septum.)

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immediate subjective improvement in dyspnea. Respiratory rate decreased to 24 breaths/min. A portable echocardiogram three hours later revealed an estimated ejection fraction of 30% (Fig 2). Repeat echocardiogram at 72 hours and at one week follow-up revealed ejection fractions of 27% and 32%, respectively, with improved wall motion by echocardiography. Pericardial fluid was sent for routine analysis plus bacterial, fungal, viral, and acid-fast bacilli cultures. Viral serology was also analyzed. The etiology of the pericardiac tamponade could not be determined.

Discussion

Cardiac tamponade is a complex circulatory state which impairs left ventricular filling during diastole (2). The decreased ventricular filling pressures may contribute to depressed stroke volumes and low cardiac output. Several compensatory mechanisms may become operational to maintain peripheral perfusion. Adrenergic stimulation causes tachycardia and increases systolic contractility to improve cardiac output. Peripheral resistance increases in an attempt to maintain blood pressure. However, this vasoconstriction may increase afterload and further decrease cardiac output. There is a lack of agreement regarding the effect of cardiac tamponade on left ventricular systolic function and ejection fraction (1, 6).

Experimental studies report conflicting data concerning the effect of tamponade on left ventricular systolic function. Investigators have considered left ventricular contractile function to be either impaired or decreased appropriately to the underfilling of the ventricle. However, in the only studies done in humans, Grose et al (4) found that ejection fractions were well maintained during tamponade and that left ventricular systolic function was preserved. Our patient had unequivocal echocardiographic (Figs 1 and 2) improvement in left ventricular systolic function almost immediately after pericardiocentesis, which suggests that his left ventricular systolic function was actually impaired by cardiac tamponade.

The hypotension that occurs in tamponade, as well as the increased left ventricular end diastolic pressure, may cause decreased myocardial perfusion and subsequent ischemia. Compression of epicardial coronary vessels by a tense effusion would further contribute to myocardial ischemia (1). Our patient was already suffering from a severe cardiomyopathy, and possible ischemia due to the aforementioned mechanisms could have further depressed his ventricular systolic function. Pericardiocentesis could have decreased myocardial ischemia with subsequent improvement in ventricular systolic function.

Our institution previously reported a patient with probable effusive-constrictive pericarditis and a pericardial effusion who experienced normalization of left ventricular systolic function after limited pericardiotomy (7) (ejection fractions were 29% before and 55% four days after pericardiotomy). Our case, as well as the previous one (7), illustrate that left ventricular function can be influenced by the state of the pericardium and its related pathology. Further work is necessary to assess the interaction of both the pericardium and pericardial fluid on ventricular function in various cardiac disease states.

References