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Short Communication

Acute Left Ventricular Failure after Large Volume Pericardiocentesis

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Summary: This paper reports on two cases of large volume pericardiocentesis followed by transient severe acute left ventricular (LV) systolic failure in the absence of any prior history of LV dysfunction. Acute LV volume overload due to interventricular volume mismatch is believed by most authors to be the cause for this phenomenon. Another plausible physiopathologic explanation is the acute increase in "wall stress" (Laplace's law) due to acute distention of the cardiac chambers secondary to a sudden increase in venous return at high filling pressures, combined with a "vacuum" effect of the evacuated pericardial space.

Key words: pericardial effusion, pericardiocentesis, cardiogenic shock, echocardiography

Case No. 1

A 36-year-old woman with severe rheumatic mitral stenosis and tricuspid insufficiency, pulmonary hypertension, and chronic atrial fibrillation underwent mitral valve replacement and surgical repair of the tricuspid valve at an outside hospital 2 months before the present admission. She had been taking digoxin, warfarin, and furosemide. During the week preceding the index admission she had developed progressive dyspnea. On admission, transthoracic echocardiography (TTE) showed a large pericardial effusion with partial compression of the right heart, right ventricular (RV) dilatation, moderate to severe tricuspid regurgitation (with severely increased estimated RV systolic pressure) and normal left ventricular (LV) systolic function. The pressure gradient across the prosthetic mitral valve was within expected limits. Her electrocardiogram (ECG) showed atrial fibrillation with rapid ventricular response and no electrical alternans. Because of prolonged prothrombin time due to coumadin therapy and lack of clinical signs of tamponade, conservative approach with steroids was instituted first. However, dyspnea worsened, and 3 days later the patient underwent pericardiocentesis under echocardiographic guidance, with evacuation of 1070 ml of serous fluid. During the following hours, the patient's condition deteriorated. She became tachypneic, her heart rate increased, and blood pressure decreased. On the next morning she developed overt cardiogenic shock. Repeat TTE showed severe LV dysfunction with regional wall motion abnormalities without pericardial effusion. Her ECG was unchanged from admission. Ultimately, the pericardial fluid was consistent with an exsudate with increased proteins and inflammatory cells consistent with postpericardiectomy pericarditis. Because of the presence of regional wall abnormalities, the patient underwent urgent coronary angiography that demonstrated angiographically normal coronary arteries. An intra-aortic balloon pump was inserted, the patient received high-dose inotropic support with dobutamine and diuretics, and her clinical condition improved. After inotropic support was discontinued, repeat TTE 4 days later showed significant improvement in LV function. Two weeks later she was discharged from the hospital on beta blockers, digoxin, warfarin, and furosemide. At discharge, TTE showed normal LV function without regional wall motion abnormalities and with normally functioning prosthetic mitral valve.

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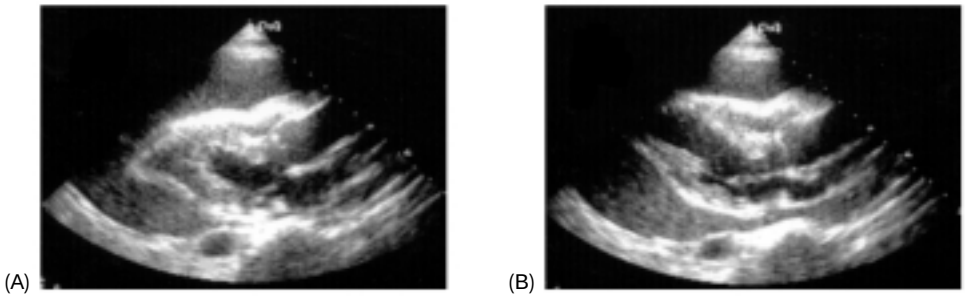

FIG. 1 Parasternal long-axis views in end-diastole (A) and end-systole (B). Prepericardiocentesis frames demonstrating normal left ventricular systolic function with large pericardial effusion and right ventricular diastolic compression. The walls of the left ventricle are thickened.

Case No. 2

A 46-year-old female patient with a history of metastatic breast cancer presented to the emergency department with progressively worsening dyspnea over the previous 3 weeks. Her physical examination was noticeable for jugular venous distension, hypotension (blood pressure of 80/50 mmHg), tachycardia (heart rate of 110 beats/min), and muffled heart sounds. Pulsus paradoxus was noted with a decrease of > 20 mmHg in her systolic blood pressure with deep inspiration. Her ECG showed sinus tachycardia with electrical alternans. An emergent TTE confirmed the presence of a large pericardial effusion with right heart compression. The walls of the left ventricle were thickened, and LV systolic function was preserved without regional wall motion abnormalities (Fig. 1A, B). An emergent pericardiocentesis was performed and 1,000 ml of serosanguinous fluid was removed. Immediate significant clinical improvement was noted. The pericardial draining catheter was left in place. A postpericardiocentesis TTE showed no residual pericardial effusion, with normal LV systolic function. Her postpericardiocentesis ECG showed normal sinus rhythm without electrical alternans. The pericardial fluid was exsudative, with significant presence of inflammatory and carcinomatous cells consistent with a malignant effusion. Over the following 24 h, the patient complained again of shortness of breath. Repeat TTE showed severely depressed LV systolic function with akinesis of the mid anterior, anteroseptal, and septal segments. There was only minimal pericardial effusion. The left ventricle appeared dilated relative to the initial echocardiogram, and the LV walls appeared thin (Fig. 2A, B). Low-dose captopril, digoxin, and furosemide were initiated. Over the following week her shortness of breath resolved. The pericardial catheter was removed after drainage fell to < 40 ml of sanguinous fluid per day. A TTE showed a decrease in LV end-diastolic and end-systolic diameter with normalization of LV systolic function. There was a trivial pericardial effusion.

Discussion

Hemodynamic derangement as a complication of pericardiocentesis has been rarely reported and includes cases of cardiogenic shock,[1–3] cardiogenic pulmonary edema,[4–6] and adult respiratory distress syndrome (ARDS).[7] In all cases except one,[7] patients underwent rapid evacuation of a large volume of longstanding pericardial effusion. Table I shows the age, gender, etiologies of pericardial effusion, preexisting cardiac conditions, and volumes of drained pericardial fluid in patients who developed severe hemodynamic derangement after pericardiocentesis.

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. External compression of the right heart by large pericardial effusion leads to a decrease in stroke volume and cardiac output.

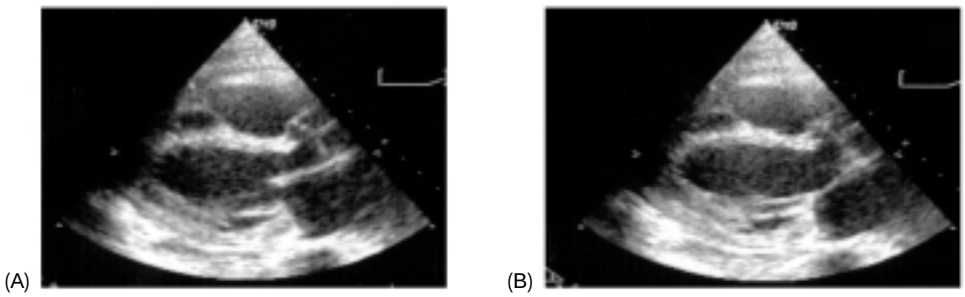

FIG. 2 Parasternal long-axis views in end-diastole (A) and end-systole (B). Twenty-four h postpericardiocentesis frames demonstrating severely depressed left ventricular systolic function. The left ventricle is dilated and wall thickness is decreased. No pericardial effusion is seen.

TABLE I Summary of reported cases of pericardiocenthesis that led to severe hemodynamic derangement

Reference No.	Gender	Age	Etiology of pericardial effusion	Preexisting cardiac condition	Volume of pericardiocentesis
1.	Female	27	Unknown	Good LV function	1000 ml
2.	Female	16	Mediastinal irradiation	Unknown	700 ml
3.	Female	50	Malignant	Good LV function	650 ml
3.	Female	46	Malignant	Good LV function, RV dilatation due to prior pulmonary embolism	650 ml
4.	Male	50	Trauma	Good LV function	650 ml + 1500 ml
5.	Male	57	Malignant pericardial effusion	Previous MI without signs of heart failure	1000 ml
6.	Male	42	Malignant pericardial effusion	Good LV function	680 ml
Present case No. 1	Female	36	Post pericardiotomy syndrome	Atrial fibrillation, good LV function, RV dilatation, severe TR, pulmonary hypertension	1070 ml
Present case No. 2	Female	46	Breast malignancy	Good LV function	1000 ml

Abbreviations: LV = left ventricular, MI = myocardial infarction, TR = tricuspid regurgitation, RV = right ventricular.

Compensatory mechanisms include tachycardia and an increase in RV preload through expansion of the intravascular volume.[4] 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.[4, 7] Pulmonary artery pressure usually falls post pericardiocentesis but may occasionally rise with a temporary mismatch of ventricular outputs.[8] This interventricular volume mismatch in the presence of vasoconstriction due to high catecholamine levels[1, 6] 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.[2] 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 physiopathologic factor. The echocardiographic findings in the second case (Figs. 1 and 2) support this hypothesis. This phenomenon may be similar to the well-described unilateral pulmonary edema after evacuation of a pneumothorax or a large pleural effusion.

Our first patient had long-standing RV dilatation and severe pulmonary hypertension due to rheumatic valve disease that may have exacerbated the encroachment on the left ventricle after evacuation of the pericardial effusion. It is possible that the absence of any previous identifiable conditions causing RV pressure or volume overload in our second patient had led to a more benign postpericardiocentesis course.

Conclusion

Despite the multiple etiologies of pericardial effusions and preexisting heart conditions in the previously reported cases, it is likely that the mechanism underlying such hemodynamic 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.[2, 4–6]

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