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Acute heart failure following decompression of tuberculosis induced pericardial tamponade

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Summary
Acute heart failure following pericardial decompression is a potentially fatal complication. We present a case with tuberculosis induced cardiac tamponade. After the emergent drainage of the pericardial cavity the patient remained hemodynamic unstable. Echocardiography revealed global biventricular systolic dysfunction and low cardiac output. Inotropic support was initiated and the patient gradually improved. Possible mechanisms implicated in this complication are the interventricular volume mismatch after the release of pericardial constraint in the presence of vasoconstriction due to high catecholamine levels or an acute increase in "wall stress" due to the acute distension of the cardiac chambers. Also systolic dysfunction may be already present but masked by the reduced chamber sizes and the tachycardia during tamponade. Physicians must be aware of this complication.

Acute heart failure following pericardial decompression is a rare but potentially fatal complication. We present a case with tuberculus (TB) pericarditis and tamponade who developed cardiogenic shock after myocardial decompression.

A 25-year-old-man, presented with a 20-day history of fever, dyspnea and chest pain. His medical history was unremarkable except from cigarette smoking.

On physical examination tachypnea, tachycardia (110/min), hypotension (85/60 mmHg) and cyanosis were found. Left bronchial breathing with dullness on percussion bellow that level was noted. Electrocardiography showed sinus tachycardia with nonspecific ST-T wave abnormalities and electric alternans. Chest-X-ray demonstrated a large left pleural effusion. Blood count and biochemistry were within normal limits.

Transthoracic echocardiography showed a large pericardial effusion with signs of tamponade. An emergent rapid drainage of the pleural and pericardial cavity followed (1200

Abbreviations: ICU, Intensive Care Unit; LVEF, Left ventricular ejection fraction; LV, Left ventricular; RT, Right ventricular; TB, Tuberculosis.

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and 1800 cc, respectively) and a sample of pericardial tissue was sent for histological examination. Fluid cytology and biochemistry showed lymphocytic exudates without malignant cells. Gram stain was negative.

After the decompression of the heart the patient remained hemodynamic unstable and was transferred to the intensive care unit (ICU). Echocardiography revealed global biventricular systolic dysfunction (left ventricular ejection fraction, LVEF = 25%) and minor pericardial effusion (0.3 cm around the heart). The cardiac output, estimated from the velocity-time integral of the left ventricular outflow tract, was 3 l/min. Inotropic support with dobutamine (20 μg/kg min) was initiated. Three hours after ICU admission the patient developed atrial fibrillation with rapid ventricular response (180/min), hypotension and desaturation. Synchronised monophasic electrical cardioversion led to cardiac arrest with ventricular fibrillation. Sinus rhythm was restored immediately after defibrillation (360J). The patient improved gradually and could be weaned off the ventilator and the inotropic support over the next five days.

On the second day of his ICU stay the histological examination of the pericardial tissue revealed acute granulomatous necrotic pericarditis. A four regimen antituberculus therapy (isoniazid, rifampicin, pyrazinamide and streptomycin) plus systemic corticosteroids were initiated. Echocardiography performed before ICU discharge showed significant improvement of the biventricular function and the LVEF rose to 45%. The culture of pericardial fluid was positive for B. Koch.

Discussion

Accumulation of excessive pericardial effusion with pericardial tamponade can impair ventricular blood inflow. It is a medical emergency demanding immediate drainage. Rarely some patients develop severe systolic myocardial dysfunction after relief of pericardial tamponade despite the expected improvement of diastolic ventricular function.

Usually this complication is observed after drainage of malignant pericardial effusions.[1–4] However it has been observed after relief of benign pericardial tamponade. Table 1 summarizes all reported cases that developed severe hemodynamic derangement after pericardiocentesis of benign pericardial tamponade.[5,1,6–8] To our knowledge, this is the first case of tuberculus pericarditis presenting with low cardiac output syndrome after relief of tamponade. Tuberculosis is responsible for up to 7% of cases of pericardial tamponade.

Severe myocardial dysfunction in our patient could not be due to tuberculus myocarditis since this is usually related to sudden cardiac death (tuberculuc myocarditis accounts for 2.4% of cases of sudden cardiac death related to tuberculosis). In the literature there are only a handful of cases reported and most of them were diagnosed postmortem.[9] Furthermore the rapid improvement of myocardial function with inotropic support before antituberculosis therapy could be effective is also against such a possibility.

Possible mechanisms implicated in the post pericardial evacuation low cardiac output syndrome are:

(1) During rapid large volume pericardiocentesis, the release of pericardial constraint could lead to a disproportionate increase in right ventricular (RV) end-diastolic volume compared with left ventricular (LV) end-diastolic volume. This interventricular volume mismatch in the presence of vasoconstriction due to high catecholamine levels could lead to an increase in LV end-diastolic pressure and transient LV systolic failure.[2,3,5,6]
(2) An acute increase in "wall stress" due to the acute distension 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 mechanism.[1] During tamponade there is a diminished coronary flow which can lead to some degree of myocardial stunning causing systolic dysfunction.[6] "Stunning" of the myocardium is a condition of transient impaired regional systolic function, following an episode of ischemia.[4]
(3) Systolic dysfunction may be already present, masked by the reduced chamber sizes and the tachycardia during tamponade. Alternatively, high levels of sympathetic

Table 1 Patients characteristics with hemodynamic derangement after pericardiocentesis of benign pericardial tamponade.

Gender	Age	Aetiology of pericardial effusion	Preexisting cardiac condition	Complication after pericardiocentesis
Male[5]	50	Trauma	Good LV function	Acute pulmonary edema
Female[1]	36	Post pericardiotomy syndrome	Atrial fibrillation, good LV function, RV dilatation, severe TR, pulmonary hypertension	Cardiogenic shock
Female[6]	27	Unknown	Good LV function	Cardiogenic shock
Female[7]	16	Mediastinal irradiation	Unknown	Cardiogenic shock
Male[8]	69	Nonspecific pericarditis	Unknown	PLCOS
Female[8]	31	HIV	UnKnown	PLCOS
Male[a]	25	Tuberculosis	Good LV function	Cardiogenic shock

Abbreviations: LV = left ventricle, RV = right ventricle, TR = tricuspid regurgitation, PLCOS = post operative low cardiac output syndrome.
[a]Current case.

tone and endogenous catecholamines during tamponade may mask preexisting myocardial dysfunction, which is then accentuated after pericardial decompression.[10]

Irrespective of the etiology of pericardial tamponade physicians must be aware of the possibility that some patients might develop acute heart failure after decompression. The therapy of low cardiac output syndrome is to restore blood flow to the hypoperfused tissue. If, however, myocardial stunning is severe, involves large parts of the left ventricle and thus impairs global left ventricle function, it can be reversed with inotropic agents.

Conflict of interest statement

There are no conflicts of interest.

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