

Patient with purulent pericarditis: a case doomed to fail?

Chory z ropnym zapaleniem osierdza:
czy przypadek był skazany od początku na niepowodzenie?

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Abstract

Exudative pericarditis, and in particular a purulent type, is a rare condition which requires emergency medical intervention. In our paper, we present a case report concerning a patient with purulent pericarditis.

Key words: purulent pericarditis, echocardiography

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INTRODUCTION

Exudative pericarditis, and in particular a purulent type, is a rare condition which requires emergency medical intervention. It has a very high mortality rate, of up to 40% if treated and 100% if untreated [1, 2]. Exudative pericarditis risk factors include immunosuppression, chronic diseases, previous chest surgeries and injuries as well as alcohol addiction. In its acute form, it progresses rapidly, it is accompanied by fever and the patient's general condition is grave [3, 4]. According to current guidelines, purulent pericarditis should be treated by draining the fluid from the pericardial sac, combined with antibiotic therapy, initially empirically selected, and later consistent with the pericardial fluid culture [5].

CASE REPORT

A 52 year-old male smoker, not treated previously for cardiovascular diseases and not taking any medication, was admitted to the Department of Internal Medicine at the County Hospital due to progressive general fatigue of two weeks' duration, dyspnoea, initially effort dyspnoea and for the last week resting dyspnoea, cough with sputum and swollen shanks. He had vomited chyme for a few days, and had had no temperature. On admission, the patient's condition was considered medium-severe. His blood pressure was

100/80 mm Hg; he had regular cardiac rhythm, accelerated to 100/min, medium-pitched quiet heart sound, with no audible murmurs or friction rub. He presented massive, symmetrical oedemas of the shanks, with no symptoms of venous thrombosis. Other signs included: resting dyspnoea, tachypnoea — 30/min, and the following findings on auscultation: vesicular sounds over the lungs, low-pitched wheezes over the left lung, symmetrical crepitations near the base of the lungs. Laboratory tests revealed features of inflammation: leukocytosis 33,000/ μ g with predominance of segmented neutrophils 87%, CRP 149 mg/dL, ESR 7 mm. Blood tests revealed: haemoglobin concentration 12 g/dL, haematocrit 36%, thrombocytes 230,000/mL, features of liver damage with elevated aminotransferase levels (ALAT 4115 U/L, ASPAT 6135 U/L), GGTP 189 U/L, significantly prolonged INR 3.73, D-Dimer concentration 1.3 μ g/mL, troponin I — negative, total bilirubin concentration slightly elevated to 1.9 mg/dL, total serum protein 5.8 g%, normal serum glucose concentration 99 mg/dL, hyponatremia with serum sodium concentration of 123 mmol/L, normal potassium level, serum creatinine 1.2 mg/dL, eGFR (MDRD) 62 mL/min, normal arterial blood gas values with 93% oxygen saturation. Results of examinations for suspected viral hepatitis B and C were negative. The electrocardiogram showed normogram, regular sinus rhythm

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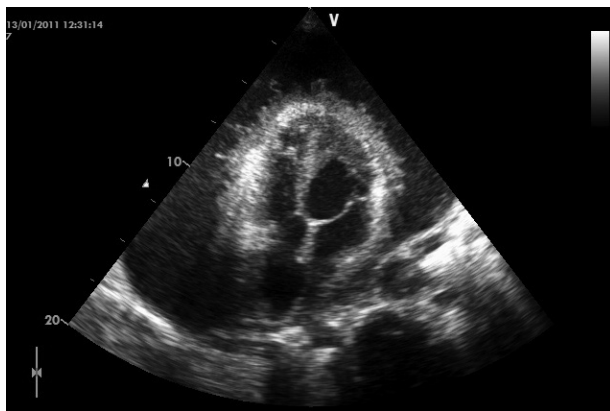


Figure 1. Transthoracic echocardiography, apical four-chamber view (a large amount of fluid in the pericardial cavity with a deposition of fibrin in the form of mobile outgrowths on the epicardial surface, right ventricular hypertrophy)

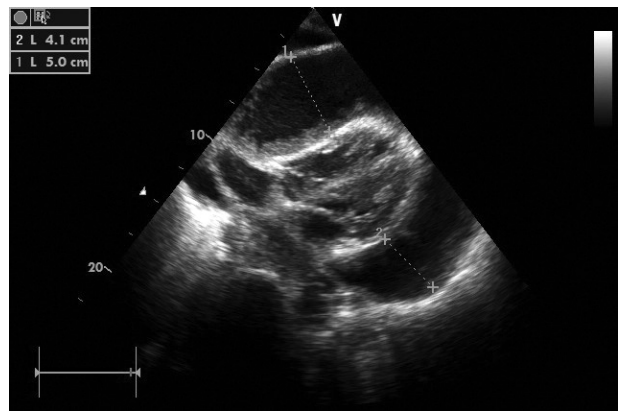


Figure 2. Transthoracic echocardiography, substernal view (fluid in the pericardial cavity of up to 5.0 cm in front of the right ventricle and of 4.1 cm behind the lateral wall of the left ventricle)

of 100/min, low voltage of the QRS complexes in the limb: in leads I and aVL up to 2 mm, in the remaining limb leads up to 4 mm, in precordial leads up to 8 mm, concave ST-segment elevation by 1 mm in leads V_2 – V_4 . The chest X-ray showed parenchyma concentrations above the left diaphragmatic leaf with a small amount of fluid in the left pleural cavity and a significantly enlarged cardiac silhouette. Echocardiography showed a very large amount of fluid in the pericardial sac up to 4.0–4.5 cm, a large deposition of fibrin on the epicardial surface, resembling mobile outgrowths of various lengths and widths, significant right ventricular (RV) wall hypertrophy up to 10 mm, with no features of cardiac tamponade in echocardiography (diastolic collapse of right atrium and RV, exaggerated respiratory variation in mitral and tricuspid blood flow velocities). Other findings included: rigid inferior vena cava dilated up to 25 mm, retained left ventricular contractile function, trace mitral regurgitation and mild tricuspid insufficiency (Figs. 1, 2).

A preliminary diagnosis of exudative pericarditis of undetermined aetiology was established, with RV insufficiency, secondary liver function damage and pneumonia. Initially, a pericardial puncture was not performed due to the absence of features suggesting cardiac tamponade and because of a significantly prolonged coagulation time (INR 3.73). Therapeutic treatment involved loop diuretics (furosemide in increased doses of a maximum of 400 mg/day in continuous infusion), spironolactone 200 mg/day, hydrochlorothiazide 25 mg/day, a small dose of carvedilol of 12.5 mg/day, the combination of amoxicillin with clavulanic acid 3×1.2 g (IV) consistent with the sputum culture (*Haemophilus influenzae* and *Streptococcus pneumoniae* were cultured), nonsteroidal anti-inflammatory drugs (ibuprofen 3×200 mg orally). The patient was consulted by the Cardiology Referral Department. The consultation recommended further conservative treatment. The patient's condition did not improve, laboratory

tests continued to reveal coagulation disturbances with a prolonged INR level above 3.0. In order to prepare the patient for pericardiocentesis and control INR to reach the level of 1.5, vitamin K in a dosage of 20 mg/day (IV) was introduced. On the second day after the administration of vitamin K, the patient's condition suddenly deteriorated, resting dyspnoea was exacerbated, and massive haemoptysis occurred, with subsequent cardiac arrest associated with the atrioventricular dissociation which led to the patient's death. Post mortem results revealed: acute, protracted purulent pericarditis with 700 mL of purulent fluid in the pericardial sac, pulmonary embolism — right pulmonary artery embolus with haemorrhagic infarct in the lower lobe of the right lung.

DISCUSSION

On the basis of reports provided in the medical literature, we know that in a case of purulent pericarditis, neglecting to drain the fluid from the pericardial sac and not introducing antibiotic therapy consistent with the fluid culture in practice dooms the patient to fail [5–8]. This is confirmed by the presented patient with exudative pericarditis, in whose case the purulent aetiology was also considered, along with other causes such as neoplasm, tuberculosis or virus. Due to his cachectic condition and evident social neglect, the patient was in the risk group including such factors as immune system disorders, malnutrition, and alcohol abuse. In our opinion, such a large amount of fluid in the pericardium with the presence of fibrin as well as RV adaptation to the elevated pressure in the pericardial sac visible in echocardiography suggested a neoplastic aetiology more strongly than a purulent one which is most often characterised by an acute and sudden course. According to our assessment, the patient was in a medium-severe but stable condition, presenting no clinical and echocardiographic features of cardiac tamponade. We were convinced that an exploratory puncture of the pericar-

dium should be performed with the minimum possible risk for the patient, consistent with current procedures. Moreover, we were afraid of intraoperative haemorrhagic complications because of advanced coagulation disorders presented by the patient. In order to control coagulation parameters, the patient was treated with vitamin K (IV). Perhaps the administration of fresh frozen plasma would have obtained a better effect. At this point, a question arises as to whether the applied treatment to achieve the INR level of 1.5 required in order to safely perform the puncture did not in fact iatrogenically induce a coagulation mechanism, and consequently directly contributed to the pulmonary embolism.

Again, medicine teaches us humility. This case showed us how complicated medical mechanisms are; how difficult it is for a physician to make crucial decisions in complicated cases, and how important co-operation between various centres is in everyday medical practice.

Conflict of interest: none declared

References

1. Goodman IJ. Purulent pericarditis. *Curr Treat Options Cardiovasc Med*, 2000; 2: 343–350.
2. Keersmaekers T, Elshot SR, Sergeant PT. Primary bacterial pericarditis. *Acta Cardiol*, 2002; 57: 387–389.
3. Sweet DD, Isac G, Morrison B et al. Purulent pericarditis in patients with rheumatoid arthritis treated with etanercept and methotrexate. *CJEM*, 2007; 9: 40–42.
4. Schuett AB, Davis M, Ray T et al. Pericardial tamponade masquerading as septic shock. *J Gen Intern Med*, 2007; 22: 269–271.
5. Maisch B, Seferovic PM, Ristic AD et al. Guidelines on the diagnostic and management of pericardial diseases executive summary. The Task force on the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*, 2004; 25: 587–610.
6. Pankuweit S, Ristic AD, Seferovic PM et al. Bacterial pericarditis: diagnosis and management. *Am J Cardiovasc Drugs*, 2005; 5: 103–112.
7. Leoncini G, Iurilli L, Queirolo A et al. Primary and secondary purulent pericarditis in otherwise healthy adults. *Interact Cardiovasc Thorac Surg*, 2006; 5: 652–654.
8. Farhat F, Dubreuil O, Durand PG et al. Constrictive pericarditis following a pyopericardium due to *Staphylococcus aureus*. *Interact Cardiovasc Thorac Surg*, 2003; 4: 626–628.

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