Medical Expertise

"Development of the European Network in Orphan Cardiovascular Diseases"
„Rozszerzenie Europejskiej Sieci Współpracy ds Sierocych Chorób Kardiologicznych"

EXPERT: Dr Anna Prokop-Staszecka, MD, PhD, Pulmonologist

Affiliation: *Department of Pulmonary Diseases, John Paul II Hospital, Krakow, Poland*

CASE SUMMARY

63 year old female patient with a history of coronary artery disease, after myocardial infarction (2009), with diabetes type 2, obesity, hypertension, persistent atrial fibrillation and COPD and chronic kidney disease, after the removal of the uterus, admitted to the Department Cardiology because of shortness of breath at rest, from a few days prior to admission.
The bedside echocardiography showed significant amount of fluid in the pericardial cavity - up to 36mm behind the posterior wall of the heart. Pericardiocentesis was performed evacuating approximately 100ml of bloody content. Microbiological negative, exudative character. Also the fluid from the right pleural cavity was collected- exudate. Cytology of fluid from the aforementioned body cavities showed no tumor cells. Due to the high levels of inflammatory parameters implemented antibiotic and anti-inflammatory drugs. Because elevated levels of CA125 abdominal CT (without contrast due to allergy) was performed, there was no irregularity in the genital tract, with the presence of lymph nodes 10mm in diameter above the spinal branch of the right dome of the diaphragm.
After transient improvement the amount of fluid in the pericardium increased. The puncture was performed again. Cytology without tumor cells. Parameters of inflammation significantly decreased. The patient clinically stable without recurrence of dyspnea. It was decided to attempt electrical cardioversion of atrial fibrillation. TEE was performed 9.01.2014r. – there was a thrombus in the left atrial appendage and persistent foramen ovale with a continuous left-to-right shunt.
14.01.2013r. A patient once again returned to the hospital with shortness of breath, tachycardia and fever. TBC tests were performed, collagenosis, amyloidosis - negative. Anti-inflammatory treatment (Diclak and Colchicine) was maintained. In the next days the patient's condition has stabilized. Fever and shortness of breath did not return, the amount of fluid in the pericardium is maintained as in the study of 13.01.2014r.
In the steady state, no fever, no shortness of breath at rest, with atrial fibrillation discharged home. Currently, the patient feels well. In the control echocardiography there is a small amount of liquid up to 5 mm in front of the anterior wall.

DISCUSSION

Pericardial effusion is a common finding in everyday clinical practice. The first challenge to the clinician is to try to establish an etiologic diagnosis. Sometimes, the pericardial effusion can be easily related to a known underlying disease, such as acute myocardial infarction, cardiac surgery, end-stage renal disease or widespread metastatic neoplasm. When no obvious cause is apparent, some clinical findings can be useful to establish a diagnosis of probability. The presence of acute inflammatory signs (chest pain, fever, pericardial friction rub) is predictive for acute idiopathic pericarditis irrespective of the size of the effusion or the presence or absence of tamponade. Severe effusion with absence of inflammatory signs and absence of tamponade is predictive for chronic idiopathic pericardial effusion, and tamponade without inflammatory signs for neoplastic pericardial effusion. Epidemiologic considerations are very important, as in developed countries acute idiopathic pericarditis and idiopathic pericardial effusion are the most common etiologies, but in some underdeveloped geographic areas tuberculous pericarditis is the leading cause of pericardial effusion. The second point is the evaluation of the hemodynamic compromise caused by pericardial fluid. Cardiac tamponade is not an "all or none" phenomenon, but a syndrome with a continuum of severity ranging from an asymptomatic elevation of intrapericardial pressure detectable only through hemodynamic methods to a clinical tamponade recognized by the presence of dyspnea, tachycardia, jugular venous distension, pulsus paradoxus and in the more severe cases arterial hypotension and shock. In the middle, echocardiographic tamponade is recognized by the presence of cardiac chamber collapses and characteristic alterations in respiratory variations of mitral and tricuspid flow. Medical treatment of pericardial effusion is mainly dictated by the presence of inflammatory signs and by the underlying disease if present. Pericardial drainage is mandatory when clinical tamponade is present. In the absence of clinical tamponade, examination of the pericardial fluid is indicated when there is a clinical suspicion of purulent pericarditis and in patients with underlying neoplasia. Patients with chronic massive idiopathic pericardial effusion should also be submitted to pericardial drainage because of the risk of developing unexpected tamponade. The selection of the pericardial drainage procedure depends on the etiology of the effusion. Simple pericardiocentesis is usually sufficient in patients with acute idiopathic or viral pericarditis. Purulent pericarditis should be drained surgically, usually through subxiphoid pericardiotomy. Neoplastic pericardial effusion constitutes a more difficult challenge because reaccumulation of pericardial fluid is a concern. The therapeutic possibilities include extended indwelling pericardial catheter, percutaneous pericardiostomy and intrapericardial instillation of antineoplastic and sclerosing agents. Massive chronic idiopathic pericardial effusions do not respond to medical treatment and tend to recur after pericardiocentesis, so wide anterior pericardiectomy is finally necessary in many cases. (1,2)

EXPERT'S OPINION

Medical treatment of pericardial effusion is mainly dictated by the presence of inflammatory signs and by the underlying disease if present. Pericardial drainage is mandatory when clinical tamponade is present.

Patients with acute inflammatory signs (fever, chest pain, pericardial friction rub)

John Paul II Hospital in Kraków
Jagiellonian University, Institute of Cardiology
80 Prądnicka Str., 31-202 Kraków;
tel. +48 (12) 614 33 99; 614 34 88; fax. +48 (12) 614 34 88
e-mail: rarediseases@szpitaljp2.krakow.pl
www.crcd.eu

should receive aspirin or non-steroid anti-inflammatory drugs. In the setting of acute inflammatory pericarditis steroids should be avoided as they increase the possibility of relapses. Colchicine is an established indication in patients with relapsing pericarditis and has also been suggested to be useful in the first episode of acute pericarditis in order to avoid the appearance of recurrences The patients with acute viral or idiopathic pericarditis can be managed on an out-of-hospital basis unless they have clinical predictors of poor prognosis (cardiac tamponade, severe pericardial effusion, immunosuppression, oral anticoagulant therapy or fever > 38°C. When specific etiology is found (bacterial, tuberculous) the treatment should be directed against the causative agent with pericardial drainage if hemodynamic compromise is present. Strict control in the first weeks or months is necessary because of the possibility of evolution to constrictive pericarditis. When acute idiopathic or viral pericarditis is accompanied by moderate to severe effusion new echocardiographic controls should be performed (initially every week) until resolution of the disease. (1,3).

REFERENCES

1. Sagrista-Sauleda J. Sarrias Merce A. Soler-Soler J. Diagnosis and management of pericardial effusion. World J Cardiol. May 26, 2011;3(5):135–143.
2. Maisch B, Seferović PM, Ristić AD, Erbel R, Rienmüller R, Adler Y, Tomkowski WZ, Thiene G, Yacoub MH. Guidelines on the diagnosis 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.
3. Piehler JM, Pluth JR, Schaff HV, Danielson GK, Orszulak TA, Puga FJ. Surgical management of effusive pericardial disease. Influence of extent of pericardial resection on clinical course. J Thorac Cardiovasc Surg. 1985;90:506–516.