Transient Left Ventricular Dysfunction Following Pericardiocentesis. An Unusual Complication to Bear in Mind

To the Editor:

The case of an 80 year old male is presented. Type 2 diabetic, recently diagnosed with latent multiple myeloma and a history of severe right coronary lesion, revascularised through angioplasty and stent implant 2 years before. The echocardiography performed 2 years previously presented normal global and segmental contractility of the left ventricle (LV) and mild chronic pericardial effusion (maximum diameter, 8 mm). The patient attended the emergency unit with intense asthenia and maintained hypotension over the previous week. In the physical exam, the patient presented a blood pressure of 90/40 mm Hg, heart rate 120 beats/min, raised jugular venous pressure, lower limb oedema, and paradoxical pulse. The electrocardiogram displayed diffuse low voltage QRS and the chest x-ray, general cardiomegaly. With these findings, an emergency echocardiogram was performed (Figure), which confirmed the clinical suspicion of cardiac tamponade showing severe and diffuse pericardial effusion (maximum diameter, 53 mm), paradoxical movement in the interventricular septum, right atrial collapse and transmitral flow variation >25% with respiration. An emergency pericardiocentesis was performed, with immediate drainage of 1200 mL of serous-sanguineous fluid and rapid improvement of the clinical and haemodynamic parameters. In the immediate echocardiographic monitoring, the normality of the diameters was checked and the LV systolic function (end diastolic diameter, 47 mm; LV ejection fraction of the LVEF >60%), with mild residual persistent pericardial effusion (maximum diameter, 11 mm). The cytologic, biochemical, microbiological, and immunological studies did not identify specific effusion aetiology.

Two days after the pericardiocentesis, the patient presented rapidly progressing dyspnea, with clinical symptomatology of pulmonary oedema and signs of low cardiac output. The emergency echocardiogram displayed severe global contractile dysfunction of the LV (LVEF, 13%). The ECG and myocardial enzymes obtained sequentially did not display data of acute ischemia. The patient required treatment with intravenous furosemide and inotropic support with intravenous dobutamine and dopamine, which were then phased out. Contractility of the LV normalised on the sixth day (LVEF, 64%). A coronary catheterisation was performed which displayed a lesion in the posterolateral branch of the
Letters to the Editor

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