Abstract: P610

Myocarditis as a cause of reversible dilated cardiomyopathy

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Topic(s):
Myocarditis

Citation:
We report the case of a 37 year-old-male with a clinical background of arterial hypertension and a sporty lifestyle. In January 2018, patient was admitted to emergency department complaining of shortness of breath related to exercise, weakness and progressive dyspnea for almost a week. He referred cold symptoms four weeks prior to admission. He denied drug or alcohol abuse. No family history of cardiomyopathy was reported. On initial examination, patient was hypertensive (147/111 mmHg), tachycardic with 110 rpm heart rate and slightly tachypneic.

ECG showed sinus rhythm and negative T waves in precordial and lateral leads, without alterations on ST segment. Chest X-ray (Figure A) demonstrated cardiomegaly with mild bilateral pleural effusion and blood tests showed the following: mildly increased CRP (12 mg/L), acute renal failure with a mild increase of creatinine (1.4 mg/dl) and increased levels of hepatic parameters (bilirubin 2 mg/dl and GPT 504 U/L).

Echocardiography (Figure B) demonstrated a severely dilated left ventricle with global left ventricular hypokinesia, ejection fraction (EF) 15% and two masses, one located in the left atrium and another attached to the LV apex. During his hospital stay, anticoagulation was initiated with heparin and he was treated with diuretics and heart failure treatment, including IECAs, B –blockers and spironolactone, improving his clinical course.

Coronary angiography was made with normal coronary arteries. Cardiac MRI was also performed, finding dilated cardiomyopathy (DCM) with severely reduced EF and intracardiac masses suggestive of thrombus without pathological enhancements. Considering the MRI findings, diagnosis of myocarditis versus idiopathic DCM was suspected.

Finally, an endomyocardial biopsy detected parvovirus B19 IgG antibodies. Parvovirus B19 IgM and other antibodies were negative. Additionally, a subcutaneous ICD was implanted because of the severe dilatation and dysfunction. During the following weeks, patient clinically improved and was discharged under periodical follow-up by Cardiology. Four months later, an echocardiogram (Figure C) was repeated showing complete recovery of the diameter and EF of heart. Nowadays, patient remains asymptomatic with good functional capacity.

Our differential diagnosis was between myocarditis and idiopathic DCM. However, the evolution and complete recovery of heart diameters and EF only four months later made the diagnosis of myocarditis more probable. Myocarditis is a complex disease which can present in various ways rendering diagnosis and treatment difficult. During the last years there is growing evidence that myocarditis and DCM are closely related due to the existence of an important inflammatory component in the pathogenesis of DCM. Our patient represents a case of reversible DCM in the context of myocarditis.
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