Abstract: P742

Right heart failure as a first manifestation of myocarditis

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Introduction: Myocarditis is an inflammation of the heart muscle, which in its most severe, fulminant form can manifest with cardiogenic shock and cardiac arrest. It usually involves both ventricles equally, but rarely can present with right heart failure as a first manifestation.

Case report: A 29-year-old male patient, with a history of hyperthyroidism, was admitted to the Intensive cardiology care unit for the evaluation of the etiology of pulmonary hypertension with signs of predominant right heart failure, with pleural effusions and mild pretibial edema. The echocardiography showed dilated right ventricle (RV) with reduced ejection fraction (RV diameter=4.2cm, tricuspid annular plane systolic excursion=0.8cm, fractional area change RV=22%), "D" shape of interventricular septum; dilated inferior vena cava (IVC=2,7cm); ejection fraction of the left ventricle was globally reduced (end-diastolic volume=132ml, end-systolic volume=89ml, EF=32%) with normal LV filling pressure (E/e'=7). CT pulmonary angiography excluded pulmonary embolism, however, revealed anterior mediastinal mass dimension 4.6x5.8x8 cm, without compression on any vascular and heart structures. The differential diagnosis of thymoma/lipoma was proposed. CT coronary angiography was normal. Meanwhile, standard therapy for heart failure was administered, altogether with thoracocentesis of the right pleural space showing transudate. MRI of the thorax was proposed for the evaluation of both, heart and anterior mediastinal mass. However, hospital course was complicated with cardiac arrest due to ventricular fibrillation. After successful cardiopulmonary resuscitation, due to progression of cardiogenic shock despite vasopressor support, veno-arterial extracorporeal membrane oxygenation (VA ECMO) was placed. Next day, an endomyocardial biopsy was performed, revealed active myocarditis and myocardial fibrosis, most likely as a result of earlier myocarditis. Polymerase chain reaction analysis revealed herpes simplex virus in the heart biopsy samples, thus acyclovir was administered. Despite the VA ECMO, there was progression of cardiogenic shock mainly due to further failure of right ventricle with subsequently low cardiac output. Hospital course was complicated by Acinetobacter pneumonia and the development of disseminated intravascular coagulation. A fatal outcome occurred on the 31st day of hospitalization. An autopsy confirmed acute myocarditis. Edematous fat tissue with thymus lobules and enlarged lymph nodes were found in the anterior mediastinum.

Conclusion: Clinical course of myocarditis may be different depending on the predominant involvement of the left or right ventricle or both. The most difficult complications of myocarditis are cardiogenic shock and cardiac arrest. However, like in previous publications, despite the introduction of ECMO in the treatment of cardiogenic shock, loss of right ventricle function is the most powerful predictor of death in these patients.
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