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Fulminant autoimmune myocarditis in Hashimoto thyroiditis

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Topic(s):
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A 79 year old patient presented in 2017 with severe fatigue and intermittent fever combined with cough since 5 months. In her history there was a mammary carcinoma on the right in 1998 followed by a second carcinoma on the left (2006). Regular controls revealed a tumour free status. Hypertrophic non obstructive cardiomyopathy with normal left ventricular function was known since 1975. In 2013 septal hypokinesia led to coronary angiography revealing with unobstructed coronaries. As hs-troponin (221 ng/l) was elevated at admission, acute coronary syndrome was suspected. Firstly echocardiography was performed demonstrating impaired systolic function of both ventricles. Therefore cardiac catheterization was planned but had to be postponed as the patient was in a hyperthyroid condition. Autoantibodies against TSH receptor, thyreoglobulin and thyroid peroxidase were elevated and Hashimoto’s thyroiditis was diagnosed.

Cardiac MRI with stress perfusion was performed revealing systolic biventricular failure (LV-EF 32%, RV-EF 35%), septal, inferior and anterior hypokinesia (figure A) with normal ventricular volumes and mass and left atrial dilatation. The TIRM sequences gave the impression of a myocardial edema (midventricular anterior and inferior). Stress perfusion revealed a perfusion defect in the areas with transmural LGE (septal, anterior wall). In addition LGE was seen in the subepicardial layers of the mid-inferoseptal, inferior and basolateral wall with pericardial involvement combined with a minor pericardial Effusion (figure B). The transmural scar was suspected to be ischemic while the inferior and lateral lesions (white arrow) were supposed to result from an acute myocarditis. Due to patient’s discomfort T1 mapping was not performed.

Coronary artery disease was ruled out by coronary angiography, a myocardial biopsy showed hypertrophic cardiomyopathy with an acute lymphocytic myocarditis but no virus causative. Two days later the patient was successfully resuscitated during a wide complex tachycardia, the basic rhythm was atrial fibrillation with heart rates from 23 to 170 beats/min. A CRT-ICD was implanted but 8 days later the patient suffered from acute severe biventricular decompensation and died after successful termination of a wide QRS tachycardia in electromechanical decoupling.

1. It is known that Hashimoto’s disease results in a higher risk of coronary artery disease, however the association to an acute fulminant myocarditis is very rare.
2. In our patient we suppose that the impaired heart with septal scars due to a long history of hypertrophic cardiomyopathy succumbed the acute autoimmune myocarditis.
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