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Meeting abstract

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2033 Frequent detection of myocardial inflammation in autoimmune diseases

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Introduction

Autoimmune diseases that are associated with active myocarditis include systemic lupus erythematosus, rheumatoid arthritis, Takayasu's arteritis, systemic sclerosis and autoimmune thyroid disease. Patients may show myocarditis and/or pericarditis causing both short- and long-term morbidity and mortality.

Purpose

To detect the presence of possible myocardial inflammation in patients with autoimmune diseases using CMR, immunohistological and PCR techniques.

Methods

Seventeen patients, aged 20–55 yrs with various autoimmune diseases (2 with Takayasu's arteritis, 9 with systemic lupus erythematosus, 3 with rheumatoid arthritis, 2 with autoimmune thyroid disease and 1 with systemic sclerosis) presented with chest pain, shortness of breath or palpitations were included in the study. All patients were in immunosuppressive treatment. Two of them had slight increase of myocardial troponin I (2.5–3.5 ng/ml). After exclusion of coronary artery disease by coronary angiography, the presence of myocardial inflammation and the left ventricular systolic function were evaluated by Cardiovascular Magnetic Resonance (CMR). Myocardial inflammation was documented using T2-weighted (T2-w), T1-weighted (T1-w) before and after contrast media injection and late enhanced images. In 8/17 patients diagnosed by CMR as having myocardial inflammation, myocardial biopsy was also performed. Biopsy specimens were evaluated by both immunohistological and polymerase reaction techniques (PCR).

Results

Myocardial inflammation was identified in 12/17 patients using CMR. In the T2-w images the signal ratio of myocardium to skeletal muscle (latissimus dorsi) was 1.66 ± 0.58 (normal values 1.28 ± 0.05), indicative of myocardial oedema. From the T1-w images the relative myocardial enhancement was 10.8 ± 12.4 (normal values 2.3 ± 0.69), indicative of myocardial inflammation. Epicardial late gadolinium enhanced areas were also identified in 12/17 (in 5 patients in the intraventricular septum (IVS), in 3 in the inferolateral wall (INFL) and in 4 in both IVS and INFL). The ejection fraction of left ventricle was decreased in 3/12 patients with CMR evidence of myocarditis (one with Takayasu's arteritis EDV = 210 ml, ESV = 160 ml, EF 24%, one with autoimmune thyroid disease EDV = 190 ml, ESV = 120 ml, EF 37% and one with rheumatoid arthritis EDV = 170 ml, ESV = 100 ml, EF 41%). The rest of them had normal ejection fraction. At biopsy, immunohistology revealed inflammation in 4/8 (50%). PCR evaluation of myocardial specimens documented the presence of viral or microbial genomes in 7/8 (87.5%). Histologically proved myocardial inflammation and pos-

itive myocardial PCR results were in agreement with 50% and 87.5% of positive CMR examinations respectively. Herpes virus was identified in 3/8, Adeno in 1/8, Coxsackie B6 in 1/8, echo in 1/8, Parvo-B19 in 2/8, CMV in 1/8 and Chlamydia trachomatis in 6/8). Coexistence of more than one viral genomes, was identified in 5/8 (62.5%). In one patient with rheumatoid arthritis only CMR was positive for myocardial inflammation.

Conclusion

Myocardial inflammation with possible deterioration of LV function is a frequent finding in autoimmune diseases. CMR can be used as a reliable, noninvasive tool to early diagnose myocardial inflammation in these patients and guide further treatment.