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Meeting abstract

2038 CMR detects transient and permanent myocardial injury in Churg-Strauss syndrome

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Objective

Cardiac magnetic resonance imaging (CMR) has the potential to visualize various forms of transient and permanent myocardial damage in inflammatory diseases. We report on CMR in patients with clinical evidence of myocardial involvement in Churg-Strauss syndrome. In this vasculitis, inflammatory damage of small cardiac vessels can result in myocarditis and cardiomyopathy.

Methods

We studied 10 patients (7 women, median 44 years) with biopsy-proven Churg-Strauss syndrome and clinical evidence of cardiac involvement in a 1.5 T clinical scanner. Left ventricular (LV) size and function were measured using ECG-triggered SSFP sequences during breathhold. We quantified myocardial water content with triple-inversion T2-weighted fast spin echo images (TR = 2 RR, TE = 64 ms, slice thickness 20 mm). Early contrast enhancement was measured on T1-weighted fast spin echo images before and early after administration of 0.1 mmol/kg Gadolinium-DTPA (nonbreathhold acquisition over 4 minutes TR = 1 RR, TE = 14 ms, slice thickness 8 mm). After a second contrast dose delayed enhancement images for fibrosis detection were acquired. Myocardial signal intensity was quantified on T2 and T1-weighted images and compared to skeletal muscle as a reference. Values were compared to a large data set from normal volunteers. Elevated myocardial signal in T2 compared to skeletal muscle was considered edema, whereas elevated early myocardial enhancement compared to skeletal muscle was considered positive for inflammation. Patients were followed clinically and in CMR for a median time of 3 years. In total 41 scans were performed.

Results

All patients showed acute and chronic myocardial tissue abnormalities on CMR even when systolic LV function (mean EF 46 ± 15%) was still preserved. In the initial scan three out of ten patients had myocardial edema and six patients had increased early enhancement. Eight patients had predominantly subendocardial myocardial fibrosis. This distribution pattern differed from that commonly seen in viral myocarditis. Even among those patients with normal LV function (mean EF 61 ± 4%) subendocardial fibrosis was present in three out of four patients. Ejection fraction did not differ in those with or without fibrotic lesions (EF 46 ± 15% vs. 50 ± 16% p = n.s.). On follow-up left ventricular function decreased in four out of ten patients (mean EF 41 ± 15%, $p < 0.05$), all of them had delayed enhancement lesions. Edema and early enhancement were transient findings depending on inflammatory activity whereas fibrotic lesions persisted.

Conclusion

Multi-sequential cardiac magnetic resonance imaging detects myocardial involvement in systemic vasculitis even beyond the assessment of wall motion.