Myocardial perfusion is associated with impaired strain in systemic lupus erythematosus: a cardiovascular magnetic resonance study

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Introduction: Systemic lupus erythematosus (SLE) is a systemic autoimmune disorder that commonly affects the heart, resulting in a 7 to 9 times greater incidence of cardiovascular disease (CVD) in SLE patients compared to healthy controls. Female patients with SLE between 35 and 44 years old have an incidence of myocardial infarction over 50 times greater than that observed in the Framingham cohort. The clinical utility of cardiovascular magnetic resonance (CMR) first-pass perfusion for assessment of myocardial ischaemia is well-established. We hypothesised that CMR including stress first-pass perfusion would be able to detect coronary microvascular disease and subtle functional abnormalities in SLE.

Objective: We aimed to detect myocardial ischaemia and regional dysfunction in SLE using adenosine stress perfusion CMR.

Methods: 29 SLE patients (28 female, mean age 42 ± 9 years) and 29 matched controls (28 female, mean age 42 ± 9 years) without previously known cardiovascular disease underwent CMR at 1.5T including cine, tagging, perfusion, and late gadolinium enhancement (LGE) imaging. Comorbid status, disease activity index and duration of disease were recorded for each subject.

Results: Myocardial perfusion reserve index (MPRI) was lower in SLE compared to controls (1.4 ± 0.2 vs. 1.9 ± 0.4, $p<0.001$). A third of lupus patients had visual evidence of non-segmental subendocardial perfusion defects, in keeping with microvascular dysfunction. No segmental perfusion defects were observed to suggest presence of coronary artery disease. There was no significant difference in LV size, mass and ejection fraction between SLE patients and controls. Peak systolic circumferential strain (-17.2 ± 1.7 vs. -19.4 ± 1.2, $p<0.001$) and peak diastolic strain rate (78 ± 24 vs. 118 ± 15 s^{-1}, $p<0.001$) were impaired in SLE patients. Left atrial size was larger in SLE (32 ± 5 vs. 26 ± 4 mm, $p<0.001$), in keeping with diastolic dysfunction. Focal fibrosis on LGE was found in 10 (43%) SLE patients compared to none of controls, and represented overall a small fraction of fibrosis (2.7 ± 0.3 %). In SLE, MPRI had a significant correlation with peak systolic strain ($R= -0.76$, $p<0.001$) and peak diastolic strain rate ($R= 0.65$, $p<0.001$).

Conclusions: Myocardial perfusion is impaired in patients with SLE with no know heart disease. In these patients, impaired MPRI was associated with abnormal myocardial deformation characteristics. It is likely that chronic disease activity and myocardial inflammation results in abnormalities in microvascular function which predate the development of myocardial functional derangements. CMR is an important tool for assessment of subclinical myocardial disease in SLE.

Myocardial structure, function and perfusion in SLE patients and controls

	Controls N=29	SLE N=29	P value
LVEDV indexed to BSA, ml/m^2	80 ± 15	78 ± 12	0.71
LVESV indexed to BSA, ml/m^2	22 ± 6	20 ± 6	0.23
LVEF, %	74 ± 5	72 ± 6	0.54
LV Mass indexed to BSA, g/m^2	51 ± 10	48 ± 10	0.18
LA size, mm	26 ± 4	32 ± 5	<0.001
Mid SA circumferential strain	-19.4 ± 1.2	-17.2 ± 1.7	<0.001
Peak diastolic circumferential strain rate (s^{-1})	118 ± 15	78 ± 24	<0.001
Presence of LGE (%)	0	10 (43)	–
MPRI	1.9 ± 0.4	1.4 ± 0.2	<0.001
Proportion of non-segmental perfusion defects (%)	0	9 (31)	

Continuous data are mean ± SD unless otherwise indicated.
LA, left atrium; LGE, late gadolinium enhancement; LV, left ventricle/ventricular; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; MPRI, myocardial perfusion reserve index; SA, short axis; SLE, systemic lupus erythematosus

Functional correlates of MPRI in SLE