

Table 1 Demographic, clinical and CMR features in SLE, SLE with CVRFs, controls and controls with CVRFs

	Normal controls (N=39)	Controls with CVRFs (N=11)	SLE (N=11)	SLE with CVRFs (N=19)	p Value
Age (years)	44.6±11.8	51.7±8.9	37.0±7.0	46.7±10.7	0.02
Females (%)	38 (97.4)	11 (100)	11 (100)	18 (94.7)	0.77
BMI (kg/m ²)	22.5±2.3	27.5±5.2	24.4±2.5	30.5±6.4	<0.001
LVEDV (ml) indexed to BSA	76.6±12.1	79.3±20.2	80.7±16.1	70.3±14.6	0.22
LVESV (ml) indexed to BSA	21.1±5.4	19.8±6.1	23.0±10.5	20.3±6.4	0.66
LVEF	72.5±4.1	75.1±5.3	72.6±7.3	71.7±3.7	0.27
LA size	2.7±0.5	2.9±0.5	3.0±0.4	3.3±0.6	<0.001

Introduction Systemic lupus erythematosus (SLE) is a systemic autoimmune disorder that commonly affects the heart. The impact of SLE on the heart is a 7–9 times greater incidence of cardiovascular disease (CVD) in SLE patients compared to healthy controls. Moreover, 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 exact cause of this excess CVD burden in SLE is poorly understood, but is thought to be multi-factorial. Cardiovascular magnetic resonance (CMR) has the capacity of simultaneously assessing non-invasively cardiac function, altered vascular distensibility, myocardial strain and fibrosis.

Objective The purpose of this study was to assess cardiac and vascular function and myocardial strain in patients with SLE and to determine their relation to the presence of cardiovascular risk factors (CVRFs) and SLE disease duration.

Table 2 Systolic circumferential strain, aortic distensibility and pulse wave velocity in SLE, SLE with CVRFs, controls and controls with CVRFs.

	Normal controls (N=39)	Controls with CVRFs (N=11)	SLE (N=11)	SLE with CVRFs (N=19)	p Value
Mid short axis systolic circumferential strain	-19.4±1.1	-18.4±1.4	-16.7±1.2	-16.3±1.0	<0.001
Peak diastolic strain rate	144.5±14.5	126.9±20.7	99.6±23.2	89.4±17.3	<0.001
Ascending aortic distensibility (10–3 mm Hg ⁻¹)	3.6±2.0	3.1±1.9	3.2±1.1	2.4±1.2	0.11
Proximal descending aortic distensibility (10–3 mm Hg ⁻¹)	4.1±1.5	3.4±1.7	3.8±1.0	2.8±1.0	0.006
Distal descending aortic distensibility (10–3 mm Hg ⁻¹)	6.2±2.5	4.7±1.8	5.0±1.2	3.6±1.2	<0.001
Aortic arch pulse wave velocity (m/s)	4.2±2.0	5.5±1.9	6.0±1.2	7.5±2.1	<0.001
Descending aortic pulse wave velocity (m/s)	3.7±1.5	5.9±1.6	6.6±1.6	8.1±1.7	<0.001
Total pulse wave velocity (m/s)	4.3±1.4	5.4±2.4	6.3±1.3	8.5±1.9	<0.001

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SYSTEMIC LUPUS ERYTHEMATOSUS IS ASSOCIATED WITH IMPAIRED MYOCARDIAL STRAIN AND VASCULAR FUNCTION, INCREMENTAL TO THAT CAUSED BY TRADITIONAL RISK FACTORS: A CARDIOVASCULAR MAGNETIC RESONANCE STUDY

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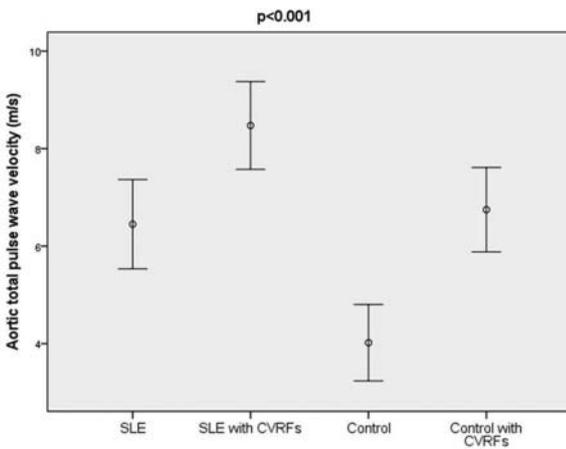


Figure 1 Total pulse wave velocity in SLE, SLE with CVRFs, controls and controls with CVRFs. Error bars represent 95% CI of mean.

Methods 11 SLE patients with no CVRFs (11 female, mean age 37 ± 7), 19 SLE patients with CVRFs (18 female, mean age 47 ± 11), 39 normal controls (39 female, mean age 45 ± 12), and 11 controls with CVRFs (11 female, mean age 52 ± 9), underwent CMR at 1.5 T. All patients with previously known CVD were excluded. CVRFs, disease activity index and duration of disease were recorded for each subject. Biventricular volumes and function, LGE, myocardial strain and vascular function were assessed by CMR. Aortic distensibility and pulse wave velocity (PWV) were measured in the ascending aorta, proximal descending aorta and distal descending aorta.

Results There were no differences in left ventricular (LV) volumes and LV ejection fraction between the four groups (table 1). SLE patients with CVRFs showed the greatest reduction in mid short axis circumferential systolic strain, peak diastolic strain rate, and vascular indices. SLE patients without CVRFs showed a similar degree of vascular dysfunction and deformational abnormality as controls with CVRFs. Aortic distensibility ($R_s = -0.59$, $p < 0.001$) and total pulse wave velocity ($R_s = 0.29$, $p = 0.01$) correlated with SLE disease duration (table 2).

Conclusions Evidence of impaired circumferential systolic strain and vascular function in SLE is demonstrated on CMR assessment, which is independent and incremental to that due to traditional CVRFs.