

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

	Normal controls (N=35)	Controls with CVRFs (N=18)	RA (N=22)	RA with CVRF (N=44)	p Value
Age (years)	51.2±13.3	53.4±11.8	49.2±9.8	51.2±10.7	0.19
Females (%)	21 (60.0)	10 (55.0)	16 (72.7)	31 (70.5)	0.06
BMI (kg/m ²)	23.3±2.7	27.3±4.2	24.5±2.8	28.4±7.4	<0.001
LVEDV (ml) indexed to BSA	72.3±12.2	73.0±15.4	74.3±19.4	70.1±14.2	0.72
LVESV (ml) indexed to BSA	20.7±13.2	18.6±5.3	21.9±9.3	19.5±8.3	0.65
LVEF	73.8±4.5	74.5±5.2	71.2±5.7	72.9±7.1	0.17
LA size	2.7±0.5	3.0±0.6	3.2±0.5	3.2±0.6	<0.001

Biventricular volumes and function, LGE, myocardial strain and vascular function were assessed by CMR. Aortic distensibility and pulse wave velocity 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 4 groups (table 1). RA patients with CVRFs showed the greatest reduction in mid short axis circumferential systolic strain, peak diastolic strain rate, and vascular indices. RA patients without CVRFs showed a similar degree of vascular dysfunction and deformational abnormality as controls with CVRFs (table 2). Aortic distensibility ($R_s=-0.25$, $p=0.048$) and total pulse wave velocity ($R_s=0.41$, $p<0.001$) correlated with RA disease duration.

Conclusions CMR demonstrates impaired myocardial deformational characteristics and impaired vascular function in RA and in

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MYOCARDIAL AND VASCULAR DYSFUNCTION IN PATIENTS WITH RHEUMATOID ARTHRITIS ASSESSED WITH CARDIOVASCULAR MAGNETIC RESONANCE: EVIDENCE OF INCREASED VASCULAR RISK

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Introduction Rheumatoid arthritis (RA) is a multi-system, auto-immune disorder and is one of the strongest known risk factors for cardiovascular disease (CVD) morbidity and mortality. Endothelial dysfunction, accelerated atherosclerosis, vascular inflammation and myocarditis are thought to contribute to this excess CVD. 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 to determine their relation to the presence of cardiovascular risk factors (CVRFs) and RA disease duration.

Methods 22 RA patients with no CVRFs (16 female, mean age 51±13), 44 RA patients with CVRFs (31 female, mean age 53±12), 35 normal controls (31 female, mean age 49±10), and 18 controls with CVRFs (10 female, mean age 51±11), underwent CMR at 1.5 Tesla. All patients with previously known CVD were excluded. CVRFs and duration of disease were recorded for each subject.

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

	Normal controls (N=35)	Controls with CVRFs (N=18)	RA (N=22)	RA with CVRF (N=44)	p Value
Mid short axis systolic circumferential strain	-19.2±1.1	-17.6±1.2	17.26±1.4	-16.9±1.2	<0.001
Peak diastolic strain rate	143.9±19.7	113.6±27.9	101.9±23.5	103.1±20.3	<0.001
Ascending aortic distensibility (10-3 mm Hg ⁻¹)	3.2±1.8	2.1±1.5	2.2±1.5	2.0±1.3	0.002
Proximal descending aortic distensibility (10-3 mm Hg ⁻¹)	3.7±1.3	3.1±1.4	2.7±1.2	2.1±1.5	<0.001
Distal descending aortic distensibility (10-3 mm Hg ⁻¹)	5.7±2.0	4.5±1.5	4.0±1.5	3.6±1.7	<0.001
Aortic arch pulse wave velocity (m/s)	4.2±2.6	6.2±1.9	6.8±3.1	7.2±2.2	<0.001
Descending aortic pulse wave velocity (m/s)	3.8±1.5	6.2±1.8	6.9±2.9	7.7±2.3	<0.001
Total pulse wave velocity (m/s)	4.4±1.6	6.7±1.5	7.7±2.9	8.3±2.8	<0.001

patients with CVRFs. The cardiac abnormalities due to RA appear to be independent and incremental to those due to traditional CVRFs.