Relationship Between Cardiac Valvular and Arterial Calcification in Patients with Rheumatoid Arthritis and Systemic Lupus Erythematosus

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ABSTRACT. ***Objective.*** Cardiac valvular calcification has been linked with systemic atherosclerosis in the general population. The prevalence and relationship with arterial calcification in patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) is unknown. We investigated the prevalence of valvular calcification in patients with RA and SLE and its relationship with arterial atherosclerotic calcification.

Methods. We compared aortic valve calcification (AVC), mitral valve calcification (MVC), and systemic vascular bed calcification using multidetector computed tomography in 110 patients (mean age 46.5 ± 9.4 yrs, 97 women) with RA (n = 58) or SLE (n = 52) and 60 age and sex-matched healthy controls.

Results. Patients with RA and SLE, combined, had significantly higher prevalence of AVC (21.8% vs 3.3% in controls; $p < 0.01$), MVC (19.1% vs 0% in controls; $p < 0.01$), and arterial calcification in different vascular beds (all $p < 0.05$). AVC was not associated with any specific clinical characteristics, but MVC was associated with older age, hypertension, C-reactive protein level, and duration of disease. The presence of MVC was independently associated with coronary calcification and calcification in any vascular bed upon adjustment with clinical measures.

Conclusion. Our study demonstrated that cardiac valvular calcification is more prevalent in patients with RA and SLE compared with healthy controls. The presence of MVC, but not AVC, independently predicted the occurrence of premature atherosclerosis with arterial calcification in patients with RA and SLE. (First Release Feb 1 2011; J Rheumatol 2011;38:621–7; doi:10.3899/jrheum.100844)

Key Indexing Terms:
RHEUMATOID ARTHRITIS
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SYSTEMIC CALCIFIED ATHEROSCLEROSIS

Cardiac valvular disease is prevalent in patients with systemic inflammatory diseases including rheumatoid arthritis (RA)[1] and systemic lupus erythematosus (SLE)[2,3,4]. In these patients, pathological changes to valves include valve thickening that results in regurgitation or stenotic lesion, noninfective vegetation, valvular nodules and calcification. In particular, the left side heart valves, the aortic valve and mitral valve, are most frequently affected[2,4].

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Aortic valve calcification (AVC)[5] and mitral valve calcification (MVC)[6] are commonly seen in the general population and their prevalence increases with aging. The presence of AVC and MVC as detected by multidetector computed tomography (MDCT) is associated with systemic calcified atherosclerosis[5]. Although the mechanisms remain unclear, it has been suggested that both valvular and arterial calcification may be a result of systemic inflammation[7].

Patients with RA and SLE have an increased incidence of cardiovascular events[8,9,10,11] that has been attributed to chronic inflammation and consequent development of premature atherosclerosis[8,12]. Nonetheless few studies have evaluated the prevalence of AVC and MVC in patients with RA and SLE. The correlation of valvular calcification with systemic calcified atherosclerosis has not been studied in these patients.

Our aim was to evaluate the prevalence of AVC and MVC in patients with RA and SLE compared with age- and sex-matched healthy controls using MDCT. The potential association between valvular calcification and systemic calcified atherosclerosis was also assessed.

MATERIALS AND METHODS

Study population. From January 2006 to January 2008, 110 consecutive Chinese patients age > 18 years who met the classification criteria for RA[13] (n = 58) and SLE[14] (n = 52) were enrolled. A further 60 age and sex-matched controls who did not meet the classification criteria for any inflammatory disease were recruited from a local health-check program. No study subject had a history of cardiovascular disease including stroke, myocardial infarction, peripheral vascular disease, or angina.

The study was approved by the institutional ethical review board and all subjects gave written informed consent.

Study protocols. Data on baseline demographics, clinical characteristics, and blood sampling were obtained from subjects prospectively on the same day. Cardiovascular risk factors including history of smoking, diabetes, hypercholesterolemia, and hypertension were assessed. Body height and weight, blood pressure, and body mass index were measured as described[15]. Hypertension was defined in the presence of a resting systolic or diastolic blood pressure ≥ 140/90 mm Hg on 2 occasions or prescription of antihypertensive medication. Diabetes was defined as a serum fasting glucose ≥ 7.1 mmol/l or prescription of an oral hypoglycemic agent. Hypercholesterolemia was defined as a fasting total serum cholesterol level ≥ 4.9 mmol/l or prescription of appropriate medication. Smoking status was recorded as either smoker (past and current) or nonsmoker. For patients with RA, duration of disease, presence of rheumatoid factor, and use of current medication were recorded. Similarly, demographic data were retrieved for patients with SLE, including duration of disease, anti-double stranded DNA antibodies, and use of current medication.

C-reactive protein (CRP) was measured using a Hitachi 747 analyzer (Boehringer Mannheim, Mannheim, Germany) and a particle-enhanced immunoturbidimetric assay (Roche Diagnostics, Mannheim, Germany)[15].

Multidetector computed tomography imaging. All subjects underwent imaging of valvular and systemic vascular calcification using a 64-slice MDCT machine (Lightspeed, VCT, GE Healthcare, USA)[16,17,18]. In brief, all scans were performed with the subjects in the supine position and included regions from the aortic arch to the fundus of the heart. Prospective electrocardiogram-gated cardiac scan was obtained with the following scan parameters: rotation time 0.35 s, slice thickness 2.5 mm, 120kV, 250 mA, trigger delay 70% R-R interval. Patients were instructed to hold breath for 30 s during scanning.

The acquired MDCT images were reviewed at a designated postprocessing workstation (Advantage Windows 4.02, GE Healthcare). Complete data were available from all the scans, without misregistration of slices due to artifacts of motion, respiration, or asynchronous electrocardiographic triggering. To ensure continuity and consistency of interpretation of calcium scores, 2 expert investigators (SW, GCO) blinded to subjects' clinical status analyzed all the scans. Interobserver and intraobserver variability correlation coefficients of calcium score measurements were 0.92 and 0.91, respectively.

Analysis of valvular and systemic vascular bed calcification by MDCT. Valvular calcium was defined as an area of 4 adjacent pixels with ≥ 130 Hounsfield units. Quantification of the extent of calcium in the aortic and mitral valves was assessed using an Agatston scoring protocol that detects areas of calcification based on the preset threshold automatically. Presence of AVC was defined as calcium deposition located in the 3 aortic cusps (left coronary, right coronary, noncoronary) including the valvular commissures and free edge of the leaflets. MVC was defined as calcium deposits within the anterior and posterior mitral valve leaflets as well as the mitral valve annulus.

Detailed measurements of systemic vascular calcification have been reported[16,17]. In brief, the coronary calcium score was calculated as the sum of the calcium score in the left main coronary artery, left anterior descending artery, left circumflex coronary artery, right coronary artery, and posterior descending artery. Carotid artery calcium score was calculated as the sum of calcium scores in the bilateral carotid arteries, including the common carotid arteries, internal carotid arteries, and bulb. Ascending calcium score was defined as the sum of calcium scores in the ascending and the aortic arch. Descending calcium score was calculated as the sum of scores in the rest of the thoracic descending aorta in the remaining scanning region. Total calcium score was the sum of coronary, carotid, and aortic scores.

Statistical analysis. Continuous variables are expressed as mean ± standard deviation or median (interquartile range) and compared using either Student's t or Wilcoxon's rank-sum test as appropriate. Categorical variables are reported as frequencies and compared using the chi-square or the Fisher exact test if at least one cell had an expected cell count below 5. The potential association of cardiovascular risk factors, CRP, and duration of disease with valvular calcification was explored by multivariate analysis. The association between individual vascular bed calcification and the presence of valvular calcification was tested with multivariable logistic regression and adjustment of the aforementioned factors individually together with age and sex. All statistical analyses were performed using SPSS for Windows (V. 15.0; SPSS, Chicago, IL, USA). A p value < 0.05 was considered statistically significant.

RESULTS

Clinical characteristics. The mean age of the study population was 47.2 ± 8.3 years and 144 (84.7%) were female. Baseline characteristics of controls and patients with RA and SLE are shown in Table 1. Control subjects were more likely to have a higher body mass index, a history of hypercholesterolemia, and a lower CRP level.

There were no significant differences in age, sex, or cardiovascular risk factors between patients with RA and SLE (Table 1). Patients with RA nonetheless had a higher CRP level and shorter duration of disease.

Among patients with RA, 42 (72%) had a positive rheumatoid factor and 45 (78%) were prescribed treatment: 8 (14%) with corticosteroid and 37 (64%) methotrexate.

Patients with SLE had a mean anti-dsDNA level of 23.1 ± 34.7 IU/ml. Medication included hydroxychloroquine (n = 30; 58%), corticosteroid (n = 15; 26%), azathioprine (n = 27; 54%), cyclophosphamide (n = 4; 8%) and mycophenolate mofetil (n = 14; 24%).

Prevalence of aortic valve, mitral valve, and systemic vascular calcification. Patients with RA and SLE (combined) had significantly higher prevalence of AVC (21.8% vs 3.3% in controls; p < 0.01) and MVC (19.1% vs 0%; p < 0.01) as well as calcification in individual vascular beds including carotid (11.8% vs 1.7%; p = 0.02), coronary (32.7% vs 1.7%; p < 0.01), ascending aorta (11.8% vs 0%; p < 0.01), descending aorta (43.6% vs 6.7%; p < 0.01), and any vascular bed (66.4% vs 8.3%; p < 0.01) compared with controls (Figure 1). Similarly, the extent of AVC, MVC, carotid artery, coronary artery, ascending aorta, descending aorta, and any vascular bed calcification was significantly higher in patients with RA and SLE (Table 1).

As shown in Figure 1, patients with RA and SLE had a similar incidence of AVC (29.3% vs 13.5%, respectively; p = 0.06), MVC (20.7% vs 17.3%; p = 0.81), carotid (15.5% vs 7.7%; p = 0.25), coronary (27.6% vs 38.5%; p = 0.31), descending aorta (46.6% vs 40.4%; p = 0.57), and any vascular bed calcification (67.2% vs 65.4%; p = 0.84). The

Table 1. Clinical characteristics and extent of valvular and vascular calcification in controls and in patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). Categorical data presented as number of patients (%).

	Controls, n = 60	RA/SLE, n = 110	p	RA, n = 58	SLE, n = 52	p
Age, yrs	48.5 ± 5.7	46.5 ± 9.4	0.08	47.7 ± 9.6	45.1 ± 9.0	0.16
Female gender, %	47 (78.3)	97 (88.2)	0.12	49 (84.5)	48 (92.3)	0.25
SBP, mmHg	119.4 ± 12.0	121.9 ± 17.0	0.26	123.2 ± 17.9	120.4 ± 16.1	0.40
DBP, mmHg	74.2 ± 9.8	76.2 ± 9.7	0.23	76.3 ± 11.1	76.0 ± 8.0	0.89
BMI, kg/m^2	23.6 ± 2.5	22.1 ± 3.7	< 0.01	22.0 ± 3.7	22.3 ± 3.7	0.71
Smoker, %	9 (15.0)	9 (8.2)	0.20	5 (8.6)	4 (7.7)	1.00
Hypertension, %	7 (11.7)	20 (18.2)	0.38	10 (17.2)	10 (19.2)	0.81
Diabetes, %	0 (0)	4 (3.6)	0.30	2 (3.4)	2 (3.8)	1.00
Hypercholesterolemia, %	22 (36.7)	6 (5.5)	< 0.01	2 (3.4)	4 (7.7)	0.42
CRP, mg/l	1.7 ± 1.2	11.1 ± 1.6	0.02	16.6 ± 20.0	6.2 ± 6.4	< 0.01
Duration of disease, yrs	—	11.8 ± 8.2	—	9.8 ± 8.0	14.0 ± 8.0	< 0.01
Mean calcium score*						
Aortic valve	0 (0)	0 (0)	0.05	0 (5.8)	0 (0)	0.83
Mitral valve	0 (0)	0 (0)	0.03	0 (0)	0 (0)	0.82
Carotid	0 (0)	0 (0)	0.03	0 (0)	0 (0)	0.24
Coronary	0 (0)	0 (3.5)	0.01	0 (2.0)	0 (6.8)	0.61
Ascending	0 (0)	0 (0)	0.05	0 (2.0)	0 (0)	0.12
Descending	0 (0)	0 (12.5)	0.04	0 (17.3)	0 (7.5)	0.21
Any vascular	0 (0)	3.5 (23.8)	0.01	4.0 (32.3)	3.0 (27.8)	0.42

* Median (interquartile range). BMI: body mass index; CRP: C-reactive protein; DBP: diastolic blood pressure; SBP: systolic blood pressure.

extent of AVC, MVC, and all vascular bed calcifications was also similar (Table 2). Patients with RA nonetheless had a higher incidence of calcification in the ascending aorta (25.9% vs 9.6%; p = 0.05) (Figure 1).

Aortic and mitral valve calcification in patients with RA and SLE. Clinical characteristics of RA and SLE patients with and without AVC and MVC are shown in Table 2. In patients with RA and SLE, no clinical parameters were associated with the presence of AVC although MVC was significantly associated with older age, history of hypertension, higher systolic blood pressure, CRP level, and longer duration of disease.

Multivariate analysis of independent predictors of AVC and MVC, adjusted for age and sex, was performed in patients with RA and SLE. The occurrence of AVC was not associated with any clinical characteristics (p > 0.05), but MVC was independently associated with older age (Table 3). Multivariate adjustment with age and sex revealed that duration of disease independently predicted the presence of MVC but not the remaining cardiovascular risk factors or CRP (Table 3).

Aortic and mitral valve calcification association with systemic vascular calcification. The percentage of RA and SLE patients with vascular calcification stratified according to the presence of AVC and MVC is shown in Figure 2. The presence of AVC was not associated with calcification in any of the vascular beds. Nonetheless the presence of MVC was significantly associated with carotid calcium score (28.6% vs 15.7%; p = 0.02), coronary calcium score (76.2% vs 22.5%; p < 0.01), descending aorta calcium score (66.7% vs 38.2%; p = 0.03), and calcium score in any vascular bed (95.2% vs 59.6%; p = 0.03).

The association of AVC with calcification in each vascular bed was not related to any clinical parameters after multivariate adjustment for age and sex (p > 0.05). In contrast, MVC was significantly associated with calcification in coronary artery and any systemic vascular bed after adjustment for the majority of clinical parameters individually in addition to age and sex (Table 4).

DISCUSSION

We have demonstrated that patients with RA and SLE have a higher prevalence of AVC and MVC than age and sex-matched controls. Importantly, this study is the first to show that the presence of MVC, but not AVC, detected by MDCT is associated with calcification in the majority of the vascular beds in patients with RA and SLE. The presence of MVC in patients with RA and SLE independently predicted the presence of coronary calcification and calcification in any systemic vascular bed after multivariate adjustment.

Valvular calcification in patients with RA and SLE. The prevalence and extent of AVC and MVC detected by MDCT were greater in patients with RA and SLE than in controls. No difference was observed between patients with RA and SLE. The low incidence of AVC (3.3%) and absence of MVC in the young controls is comparable with previous

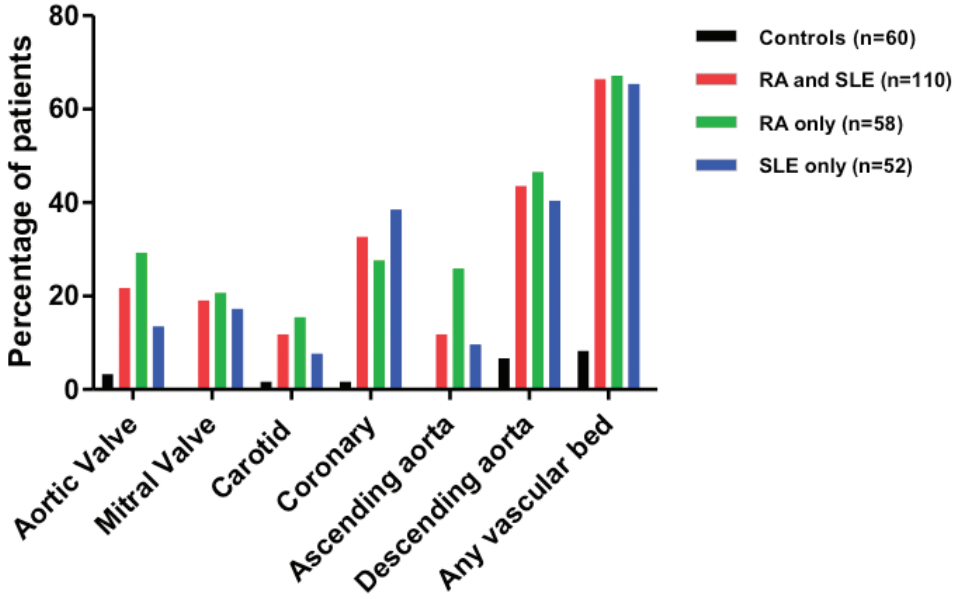

Figure 1. Percentage of controls and patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) with aortic, mitral valve, and individual systemic vascular calcification.

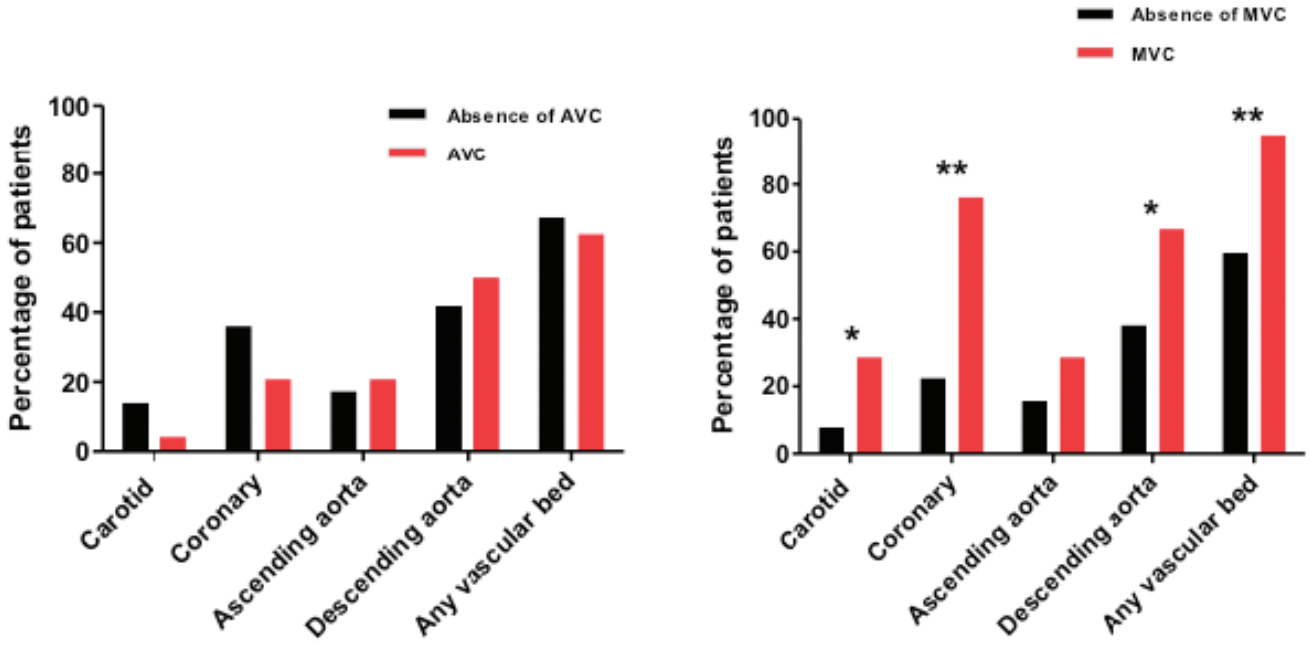

Figure 2. Percentage of patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) with individual vascular calcification stratified according to the presence of aortic valve calcification (AVC; left panel) and mitral valve calcification (MVC; right panel). *p < 0.05; **p < 0.01.

data where valvular calcification increased with age[19,20]. The high incidence of AVC and MVC observed in young patients with RA and SLE (age 46.5 yrs) was comparable only with patients aged > 60 years in the general population and in those with diabetes. This supports the hypothesis that accelerated valvular calcification occurs in patients with systemic inflammatory disease[19,21,22].

In contrast to the general population[5], our study showed that the presence of AVC in patients with RA and SLE is not associated with age, sex, or cardiovascular risk factors. The presence of AVC was not associated with CRP level or duration of disease, although the presence of MVC in patients with RA and SLE was associated with older age, history of hypertension, higher CRP, and longer disease duration.

Table 2. Clinical characteristics of controls stratified by presence of aortic valve calcification (AVC) and patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) stratified by the presence of AVC and mitral valve calcification (MVC).

	RA/SLE					
	AVC			MVC		
	Absent, n = 86	Present, n = 24	p	Absent, n = 89	Present, n = 21	p
Age, yrs	46.9 ± 9.9	45.0 ± 7.2	0.30	44.3 ± 8.0	55.6 ± 9.6	< 0.01
Female gender, %	74 (86.0)	23 (95.8)	0.29	81 (91.0)	16 (76.2)	0.12
SBP, mmHg	122.4 ± 17.8	120.0 ± 13.9	0.55	120.0 ± 15.4	130.4 ± 21.1	0.04
DBP, mmHg	76.6 ± 10.0	74.7 ± 8.7	0.41	75.8 ± 9.5	77.6 ± 10.8	0.49
BMI, kg/m^2	22.1 ± 3.6	22.2 ± 3.9	0.91	22.2 ± 3.8	21.6 ± 3.2	0.47
Smoker, %	8 (9.3)	1 (4.2)	0.68	6 (6.7)	3 (14.3)	0.37
Hypertension, %	18 (20.9)	2 (8.3)	0.23	12 (13.5)	8 (38.1)	0.02
Diabetes, %	3 (3.5)	1 (4.2)	1.00	3 (3.4)	1 (4.8)	0.58
Hypercholesterolemia, %	5 (5.8)	1 (4.2)	1.00	5 (5.6)	1 (4.8)	1.00
CRP, mg/l	12.3 ± 17.5	9.2 ± 8.5	0.32	9.3 ± 9.6	20.4 ± 25.8	0.04
Duration of disease, yrs	11.2 ± 8.4	9.0 ± 5.3	0.25	10.9 ± 7.8	15.7 ± 8.9	0.03

For abbreviations see Table 1.

Table 3. Risk factors multivariate analysis for mitral valve calcification (MVC) adjusted with age and gender.

	MVC		
	HR	95% CI	p
Age	1.14	1.08–1.22	< 0.01
Female gender	0.32	0.09–1.09	0.07
Body-mass index*	0.91	0.78–1.07	0.24
Smoker*	1.67	0.21–13.04	0.63
Hypertension*	1.67	0.48–5.75	0.42
Diabetes*	0.46	0.03–6.70	0.57
Hypercholesterolemia*	0.21	0.02–2.50	0.21
C-reactive protein*	1.15	0.78–1.70	0.48
Duration of disease*	1.07	1.00–1.14	0.04

* Adjusted hazard ratios together with age and gender. CI: confidence interval; HR: hazard ratio.

Multivariate adjustment revealed that age and duration of disease were the only independent predictors for MVC in patients with RA and SLE. It is unknown whether there is an association between conventional cardiovascular risk factors and valvular calcification in patients with RA and SLE. No association between cardiovascular risk factors and systemic vascular atherosclerosis was observed in patients with systemic inflammatory disease[8,12]. This may suggest the independent contribution of RA and SLE disease status per se to the development of premature valvular calcification and atherosclerosis. We also showed that duration of disease, but not systemic inflammation assessed by CRP, independently predicted the presence of AVC and MVC. This could be partly explained by the limited ability of a single-time CRP measurement to reflect the chronic inflammatory burden in patients with RA and SLE.

Table 4. Unadjusted and adjusted odds of mitral valve calcification (MVC) in each vascular bed in patients with rheumatoid arthritis and systemic lupus erythematosus.

	Carotid		Coronary		Ascending Aorta		Descending Aorta		Any Vascular Bed	
	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p
Unadjusted	4.69 (1.38–15.90)	0.01	11.0 (3.60–33.86)	< 0.01	2.14 (0.71–6.47)	0.18	3.24 (1.19–8.82)	0.02	13.9 (1.75–105.80)	0.01
Age	4.54 (1.27–16.17)	0.17	4.54 (1.27–16.17)	0.02	2.58 (0.70–9.47)	0.15	1.54 (0.49–4.85)	0.48	8.12 (1.02–67.25)	0.04
Gender	3.94 (1.12–13.99)	0.03	10.31 (3.33–31.91)	< 0.01	2.09 (0.68–6.45)	0.20	3.31 (1.19–9.21)	0.02	16.00 (1.96–130.42)	0.01
BMI*	2.10 (0.63–11.37)	0.17	4.50 (1.23–16.45)	0.02	2.74 (0.74–10.23)	0.13	1.62 (0.51–5.20)	0.42	10.0 (1.14–87.55)	0.04
Smoker*	2.33 (0.54–10.02)	0.25	4.37 (1.21–15.89)	0.03	2.58 (0.68–9.75)	0.16	1.65 (0.51–5.33)	0.41	11.50 (1.22–108.56)	0.03
Hypertension*	2.21 (0.51–9.58)	0.29	4.25 (1.17–15.42)	0.03	2.65 (0.70–10.03)	0.15	1.59 (0.50–5.09)	0.43	10.55 (1.20–93.02)	0.03
Diabetes*	2.41 (0.56–10.47)	0.24	4.34 (1.19–15.81)	0.03	2.57 (0.69–9.57)	0.16	1.59 (0.50–5.07)	0.43	8.98 (1.04–97.15)	0.05
Hyperlipidemia*	2.36 (0.53–10.58)	0.26	4.26 (1.17–15.42)	0.03	2.83 (0.75–10.64)	0.12	1.65 (0.51–5.30)	0.40	11.9 (1.29–110.10)	0.03
CRP*	1.95 (0.39–9.76)	0.42	4.29 (0.94–19.53)	0.06	2.45 (0.55–10.96)	0.24	1.97 (0.44–8.45)	0.39	8.5 (1.07–82.45)	0.04
Duration of disease*	1.87 (0.42–8.40)	0.41	3.88 (1.04–14.85)	0.04	4.15 (0.98–17.60)	0.06	1.65 (0.50–5.40)	0.41	9.00 (1.03–73.9)	0.04

* Adjusted hazard ratios together with age and gender. For abbreviations see similar to Table 3.

Association between valvular calcification and systemic calcified atherosclerosis. Our studies[16,17] and others[23,24] have demonstrated a significantly higher prevalence and extent of systemic calcified atherosclerosis in RA and SLE patients compared with controls. The current study also showed that patients with RA and SLE have comparable calcification in most vascular beds, although patients with RA had higher prevalence of calcification in the ascending aorta ($p = 0.05$). The potential differences in the preferential systemic arterial calcification between patients with RA and SLE require future detailed evaluation.

Previous histopathologic studies have demonstrated that valvular and arterial calcification share a similar pathogenesis[25]. Both may develop in the presence of systemic inflammation with subsequent lipid core formation[26] and endothelium injury[27]. The correlation of both AVC and MVC with systemic calcified atherosclerosis has been shown in the general population using MDCT[5]. Our results also demonstrated a strong association between MVC and vascular calcification in the carotid, coronary, and the descending aorta in patients with RA and SLE. In the presence of MVC, there was an adjusted relative 4-fold risk for coronary calcification and 8- to 12-fold risk of calcification in any vascular bed compared with absence of MVC, after adjustment with individual cardiovascular risk factors. In contrast, presence of AVC was not associated with calcification in any vascular bed in the current cohort of patients with systemic inflammatory disease. This confirms previous findings of no correlation between AVC and coronary calcification in patients with SLE[3]. The reason for the lack of association between AVC and systemic calcification in patients with systemic inflammatory disease is unknown. A single CRP measurement cannot accurately represent the chronic inflammatory burden in patients with RA and SLE. Thus these study results do not preclude the role of inflammation leading to premature AVC; the exact mechanism for this requires investigation.

Clinical implications. The presence of AVC and MVC are associated with cardiovascular events in the general population[6,28]. The prognostic role of cardiovascular events in the presence of AVC and MVC detected by MDCT in patients with RA and SLE has not been studied previously. In a study of 107 patients with SLE, the presence of AVC and mitral annulus calcification detected by transthoracic echocardiogram was associated with death, although most examples were of noncardiac origin[29]. In addition, multivariate adjustment was not available and therefore the value of valvular calcification as an independent predictor of mortality in patients with SLE could not be established. The potential prognostic value of the presence of AVC and MVC in patients with RA and SLE requires verification in studies of a large population.

This study comprised a small population of patients with RA and SLE. Independent risk factors for development of AVC and MVC could not be confidently determined. Although well validated as a surrogate marker for atherosclerosis, systemic vascular calcification measured by MDCT cannot identify noncalcified plaque. Thus the total atherosclerotic burden in patients with RA and SLE was not fully determined.

Our study demonstrated that cardiac valvular calcification is more prevalent in patients with RA and SLE compared with age- and sex-matched controls. The presence of MVC, but not AVC, independently predicted the occurrence of premature atherosclerosis with arterial calcification in patients with RA and SLE.

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