P56 CORONARY ARTERY CALCIFICATION IN DANISH SLE PATIENTS WITHOUT KNOWN CARDIOVASCULAR DISEASE: COMPARISON WITH THE GENERAL POPULATION, MYOSITIS AND DIABETES PATIENTS

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Objective Patients with systemic lupus erythematosus (SLE) have an increased risk of cardiovascular disease (CVD), which is thought to be due to accelerated atherosclerosis. The aim of this study was to determine the extent of coronary artery calcification (CAC) in SLE patients compared to a subset of the general population and to patients with idiopathic inflammatory myopathies (IIM) and diabetes mellitus (DM).

Methods 104 SLE patients, 2,237 controls from the general population, 69 IIM patients and 175 DM patients were screened for CAC. In all cohorts patients with symptoms of or known CVD were excluded.

CAC was measured in Agatston score and classified as any (score>0), high (>399 U) or extremely high (>1000 U). To compare the groups multivariate logistic regression was used.

Results The SLE patients were younger and more often females, but compared to the general population, IIM and DM patients, they had more severe calcifications (high CAC score: 33% versus 19%, 25% and 30%, respectively and extreme high CAC scores: 20% versus 6%, 8% and 13%. Adjusted for age and sex, SLE patients had substantially higher odds ratios (OR) for high and extreme high CAC compared to the general population, IIM patients and DM patients (high CAC: OR 6.2, 3.2 and 3.0, respectively and extreme high CAC: 10.7, 8.7 and 4.7, respectively).

Conclusion In the setting of individuals without previous known CVD, SLE patients independently of age and sex had more severe coronary atherosclerosis, not only compared to the general population, but also compared to patients with IIM and even to patients with DM.

P57 STRESS PERFUSION CARDIAC MAGNETIC RESONANCE IMAGING (CMR) CHANGED MEDICAL MANAGEMENT IN SLE PATIENTS WITH CHEST PAIN

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Objective Chest pain and/or discomfort are more commonly reported by SLE patients than by their doctors. The causes for these complaints are multifactorial, but it is especially important to promptly recognize and treat heart disease, a well-documented major cause of impaired health and shortened life expectancy in SLE.

Methods SLE patients who presented with chest pain and/or discomfort were investigated using stress cardiac magnetic resonance imaging (CMR). Patients with contraindications to stress CMR were either excluded or investigated using CMR without adenosine provocation. Patients with findings at stress CMR indicating coronary artery disease (CAD) or coronary microvascular dysfunction (CMD) were referred to the cardiology department for further work-up. Patients with pericarditis

Abstract P57 Table 1 Baseline characteristics

Variables	Proportion (%) or median	IQR	Number of observations
Female,%	85		20/20
Age, years	42	32–58	20/20
Chest pain characteristics[1],%			20/20
Pleuritic chest pain	50		
Angina	15		
Non-angina-non-pleuritic chest pain	25		
Chest pain equivalent symptoms	10		
Lupus nephritis,%	35		20/20
Plasma creatinine, micromol/L	73	61–86	20/20
Albumin-creatinine ratio in urine, mg/mmol	2.7	1.0–19	15/20
APS,%	5.0		20/20
VTE,%	20		20/20
Sjögren's syndrome[2]	5.0		20/20
C-reactive protein, mg/L	2.5	1.0–4.0	18/20
Sedimentation rate, mm	26	14–47	18/20
Plasma albumin, g/L	35	30–37	18/20
Leucocyte count, $x10^9$/L	6.2	4.0–7.0	18/20
Lymphocyte count, $x10^9$/L	1.2	0.8–1.6	18/20
Platelet count, $x10^9$/L	320	220–360	18/20
ANA pos,%	100		12/20
Anti-dsDNA pos[3],%	67		18/20
Anti-Sm pos,%	24		17/20
Anti-RNP86 pos,%	18		17/20
aPL pos,%	12		17/20
Lupus anticoagulant pos,%	14		14/20
Anti-Ro52_SSA pos,%	53		17/20
Anti-Ro60_SSA pos,%	71		17/20
Anti-SSB pos,%	35		17/20
C3, g/L	0.80	0.60–0.95	16/20
C4, g/L	0.12	0.10–0.17	13/20
Prednisolone equivalent dose, mg	7.5	5.0–15	20/20
HCQ treatment,%	75		20/20

[1]= Chest pain was divided into four categories; pleuritic (worse with breathing or positional), angina (precipitated by exercise and/or relieved by nitrates/rest), non-angina-non-pleuritic chest pain (neither angina nor pleuritic chest pain) and chest pain equivalents (dyspnea precipitated by exercise suggestive of coronary involvement). [2]= Defined according to Vitali et al. (1). [3]= Either according to multiplex or immunofluorescence. ANA = Antinuclear antibodies. aPL = Antiphospholipid antibodies including anti-cardiolipin and/or beta-2 glycoprotein 1 antibodies of either IgM or IgG type. APS = Antiphospholipid syndrome defined according to Miyakis et al. (2). DsDNA = Double stranded DNA. HCQ = Hydroxychloroquine. IQR = Interquartile range. VTE = Venous thromboembolism (defined as pulmonary embolism or deep vein thrombosis).

were managed by the rheumatology department. Medical files were reviewed in detail.[1–3]

Results Twenty consecutive SLE patients (85% female) with a median age of 42 (IQR 32–58) years were included, out of which 15 (75%) underwent adenosine stress CMR. SLE characteristics and traditional cardiovascular risk factors are reported in tables 1 and 2. Chest pain was characterized as pleuritic in 50%, non-angina-non-pleuritic in 25%, angina like in 15% and chest pain equivalent (dyspnea precipitated by exercise suggestive of coronary involvement) in 10%. Treatable heart disease was found in 40% of patients through CMR, including CMD (27%), CAD (20%) and/or pericarditis (15%). Note that more than one cardiac disease was found in some patients. Referral for stress CMR changed medical management in 35% of patients.

Conclusion CMR demonstrated treatable heart diseases in 40% of investigated SLE patients; most commonly CMD and CAD. These results are important, since many of these patients would likely have been misdiagnosed as pleuro-pericarditis based only on the clinical picture. Thus, CMR merits further use in SLE, to correctly diagnose and treat chest symptoms. Larger studies are needed to confirm our results.

Abstract P57 Table 2 Traditional cardiovascular risk at baseline

Variables	Proportion or median	IQR	Number of observations
Risk of CVD according to (3),%:			17/20
Low to moderate risk	59		
High risk	6		
Very high risk[1]	35		
CVD,%	25		20/20
Creatine-based eGFR, mL/min/1.7	78	70–93	20/20
Diabetes,%	0		20/20
Smoking status,%			19/20
Current	0		
Previous	53		
Never	47		
Systolic BP, mmHg	120	110–130	20/20
Systolic hypertension (>130 mmHg),%	25		20/20
Diastolic BP, mmHg	79	70–82	20/20
Diastolic hypertension (>80 mmHg),%	40		20/20
Antihypertensive treatment,%	45		20/20
BMI, kg/m^2	25	21–28	20/20
Classes of BMI,%			20/20
<20 kg/m^2	25		
20–24.9 kg/m^2	20		
25–29.9 kg/m^2	35		
30–34.9 kg/m^2	15		
>35 kg/m^2	5		
Total cholesterol, mmol/L	4.7	4.2–5.0	14/20
LDL, mmol/L	2.6	2.2–2.7	13/20
HDL, mmol/L	1.5	1.2–1.7	14/20
Triglycerides, mmol/L	1.3	1.0–1.9	14/20
Cholesterol lowering treatment,%	20		20/20

[1]= Due to previous CVD, but also because of eGFR 30–44 with albumin-creatinine ratio >30 mg/mmol in one case. BMI = Body mass index. BP = Blood pressure. CVD = Cardiovascular disease defined as myocardial infarction, unstabila angina, chronic coronary syndrome, stroke, TIA, aortic aneurysm or peripheral arterial diseases. eGFR = Estimated glomerular filtration rate. HDL = High-density lipoprotein. IQR = Interquartile range. LDL = Low-density lipoprotein

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P58 MENOPAUSAL AGE AND THE EFFECT OF MENOPAUSE ON DISEASE ACTIVITY IN SLE

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Objective To investigate menopausal age in women with SLE and controls, and to assess if SLE-related measurements, blood lipid levels, and hs-CRP differed with menopausal status among SLE patients.

Methods In a cross-sectional study, we compared self-reported menopausal age between 200 well-characterized women with SLE and 118 population controls.

Within the SLE cohort (n=200), we identified 30 pre- and 30 post-menopausal women, individually matched for age ±3 years. Disease activity indices (SLAM, SLEDAI), damage (SLICC/ACR Damage Index (DI)), lipid profiles, and CRP were compared between the two groups.

Results

1) ***Age at menopause in SLE and controls***. Mean age among SLE patients and controls was 59.2 (SD 9) and 61.1 (SD 7.7) years, respectively, median disease duration of SLE patients was 17.7 years. Menopause occurred at a younger age in SLE than in controls (48.1 vs 50.6 years, p<0.0001). Even younger menopausal age was reported in the sub-groups with lupus nephritis (46.7 years) and previous cyclophosphamide treatment (45.9 years, table 1).

2) Comparisons between pre- versus post-menopausal status among women with SLE. Mean ages in the matched pre- and post-menopausal groups were 44.4 and 44.9, respectively (p=0.7). Three post-menopausal and two pre-menopausal patients were on statin treatment. We observed no difference regarding disease activity, damage, or lipids between the groups, but hs-CRP was higher among pre-menopausal women. (6.1 vs. 2.7 mg/L, p=0.04, table 2).

Conclusions Women with SLE enter menopause earlier than women from the general population, regardless of lupus nephritis or cyclophosphamide exposure. However, both a diagnosis of lupus nephritis and previous cyclophosphamide treatment, are associated with even younger age at menopause.

Premenopausal women with SLE have similar disease activity, damage scores, and blood lipid levels but higher hs-CRP compared to postmenopausal SLE women of similar age. Our data indicate that menopausal status does not affect disease burden or the lipid profile in SLE patients.

Larger and longitudinal studies are needed to understand if the higher hs-CRP in postmenopausal patients is associated with a systemic inflammatory state and if the underlying