Reduction in SLEDAI is associated with improved arterial stiffness in systemic lupus erythematosus

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Abstract

Lipid abnormalities are an important cause of premature atherosclerosis in patients with systemic lupus erythematosus (SLE). This longitudinal study investigates the changes in lipid profile and arterial stiffness with SLE disease activity index (SLEDAI) reduction.

Fifty one female SLE patients with baseline SLEDAI ≥ 6 and SLEDAI reduction >3 at 1-year follow-up were included. Neutrophil-to-lymphocyte ratio (NLR), erythrocyte sedimentation rate (ESR), high-sensitivity C-reactive protein (hsCRP), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglyceride (TG), and mean brachial-ankle pulse wave velocity (baPWV) were measured and compared between baseline and 1-year follow-up. Correlations between inflammation biomarkers, SLEDAI, mean baPWV and lipid profile were assessed.

We observed significant decreases in ESR, mean baPWV, TG and TC to HDL-C ratio compared with baseline at 1-year follow up, while HDL-C, hsCRP, and NLR were not significantly changed. Significant correlations were found between the reductions in ESR and TG, and SLEDAI and mean baPWV, with adjustment to age, disease duration, blood pressure, and medications (prednisone, immunosuppressants and ARB/ACEI).

SLE patients experiencing SLEDAI reductions showed improvements in arterial stiffness. This finding may provide insight into the beneficial effects of reducing SLEDAI on atherosclerosis risk in SLE.

Abbreviations: baPWV = brachial-ankle pulse wave velocity, CIMT = carotid intima-media thickness, ESR = erythrocyte sedimentation rate, HDL-C = high-density lipoprotein cholesterol, hsCRP = high-sensitivity C-reactive protein, LDL-C = low-density lipoprotein cholesterol, MAP = mean arterial pressure, NLR = neutrophil-to-lymphocyte ratio, PP = pulse pressure, SLE = systemic lupus erythematosus, SLEDAI = SLE disease activity index, TC = total cholesterol, TG = triglyceride.

Keywords: brachial-ankle pulse wave velocity, inflammation, lipid profile, SLEDAI, systemic lupus erythematosus

1. Introduction

Systemic lupus erythematosus (SLE) is an inflammatory autoimmune disease influencing multiple organs including the heart, brain, skin, joints, kidneys, and blood vessels.[1] Compared with healthy controls, patients with SLE exhibit an elevated rate of atherosclerosis (1.8–2.5 fold), a hallmark of cardiovascular disease.[2,3] Traditional Framingham risk factors have failed to fully explain the increased cardiovascular risk accompanying SLE[4,5]; thus, the exact mechanism of atherosclerosis in SLE is

not yet fully elucidated. Furthermore, it is now commonly recognized that atherosclerosis occurs in SLE in an interplay between traditional cardiovascular risk factors and SLE-specific risk factors (e.g., disease duration, medication, and renal involvement).[6]

The SLE disease activity index (SLEDAI) stratifies SLE disease activity, and high SLEDAI was reported to be predictive of increased carotid intima-media thickness (CIMT), carotid plaques, and cardiovascular events.[7–9] This can be attributed to increased dyslipidemia with elevated SLEDAI.[10–12] However, patients with high SLEDAI are more likely to receive high doses of glucocorticoids, hydroxychloroquine, or other immunosuppressive drugs. These medications decrease SLEDAI and systemic inflammation, which potentially decreases the risk of atherosclerosis; however, high doses of glucocorticoids will adversely increase traditional atherosclerotic risk factors, such as dyslipidemia.[6,13,14] Hydroxychloroquine, immunosuppressive drugs like cyclophosphamide, and other medications frequently used in SLE such as ARB/ACEI also influence lipid metabolism.[15–17] Nevertheless, many of the previous studies, which investigated the correlations between lipid profile, atherosclerosis, and SLEDAI[7,18,19]; did not consider the potential effects of medications on the lipid profile. Furthermore, these studies were cross-sectional and based on a western population, which had different prevalence of cardiovascular disease and genetic background from the Asian population.[20,21] Studies are needed to assess the correlations of SLEDAI to lipid profile and atherosclerosis with adjustment of medications, especially in Asian population.

To fulfill these gaps, this study investigated the association between the changes in SLEDAI, lipid profile and arterial stiffness, a surrogate marker of early sub-clinical atherosclerosis, in a longitudinal Chinese cohort, with adjustment of medications. This study may better address the association of SLEDAI itself to lipid profile and arterial stiffness, and also provide more direct evidence for the reduction of SLEDAI on the improvement of lipid profile and arterial stiffness.

2. Methods

2.1. Study population

This study included adult (>18 years old) female patients enrolled in the Chinese SLE Treatment and Research group (CSTAR) registry who presented at the outpatient clinic of the Peking Union Medical College Hospital between September 2013 and September 2015. All subjects met the 2012 Systemic Lupus erythematosus International Collaborating Clinics (SLICC) classification criteria.[22] Subjects in the CSTAR had annual clinical assessments, including assessments of SLE disease activity and cardiovascular disease risk factors. The traditional cardiovascular risk factors evaluated in this study include age, sex, menopause, smoking status, body mass index (BMI), and diabetes history. SLE-related parameters including disease duration, the Systemic Lupus International Collaborating Clinics Classification (SLICC)/ACR damage index, courses of prednisone (total cumulative duration of prednisone usage for a year was calculated based on the medical records and patient self-reporting) and immunosuppressant treatments were also assessed. We included subjects who had a SLEDAI ≥6 (active disease) at baseline and experienced a SLEDAI reduction >3 at 2 consecutive time points that were 1 year apart. The exclusion criteria included current smoker, and the presence of any other autoimmune diseases, infectious diseases, chronic kidney disease (eGFR≤60 ml/minute/1.73 m^2), and any known cardiovascular diseases. To remove the potential effects of statins on low-density lipoprotein cholesterol (LDL-C), subjects receiving statins during the 6 months prior to the baseline and the end of the study were excluded. As diabetes mellitus significantly influences lipid metabolism, patients with diabetes mellitus were also excluded.[23] In addition, we excluded subjects with low ankle-brachial indexes (ABI<0.9), which could lead to inaccurate brachial-ankle pulse wave velocity (baPWV) values.[24] To detect a moderate correlation (correlation coefficient ≥ 0.4) with 95% level of confidence and 80% statistical power, a sample size ≥47 was calculated with online sample size calculators (https://sample-size.net/correlation-sample-size/).

2.2. Laboratory analysis

Fasting venous blood was drawn at baseline and at the 1-year follow-up. A complete blood cell count was determined using laser scattering and the chemical dyeing of cells (ADVIA 2120 hematology analyzer, Siemens Healthcare Diagnostics, Erlangen, Germany). Enzymatic analyses or the transmission turbidity method (Beckman Coulter AU5800; Beckman Coulter Inc., Brea, CA, USA) were used to determine total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), LDL-C, triglyceride (TG), creatinine, high-sensitivity C-reactive protein (hsCRP), and complement 3 levels. Glycosylated hemoglobin (HbA1c) levels were evaluated by ionic exchange high-performance liquid chromatography (Variant II automatic analyzer, Bio-Rad Laboratories, Hercules, CA, USA). Line immunoassay was performed to measure anti-dsDNA antibody levels (Euroline ANA profile assay, Euroimmun AG, Luebeck, Germany).

BaPWV was measured with an automated waveform analyzer (VP-2000, Omron-Colin, Japan) as previous described.[25] Blood pressure was measured as described in Ding et al.[25] Mean arterial pressure (MAP) was calculated by MAP=[systolic blood pressure+(2 × diastolic blood pressure)]/3. Pulse pressure (PP) was calculated by PP=systolic blood pressure-diastolic blood pressure. Hypertension was defined as systolic blood pressure ≥140 mm Hg, diastolic blood pressure ≥90 mm Hg, or the current use of anti-hypertensive medications. Dyslipidemia was defined as either one of the following:[26]

1. hypercholesterolemia: TC ≥6.22 mmol/L (240 mg/dl);
2. high LDL-C: LDL-C ≥4.14 mmol/L (160 mg/dl);
3. hypertriglyceridemia: TG ≥2.26 mmol/L (200 mg/dl); and
4. low HDL-C: HDL-C < 1.04 mmol/L. BMI=weight (kg)/height (m^2).

2.3. Statistical analysis

We compared SLEDAI, white blood cell (WBC), neutrophil-to-lymphocyte ratio (NLR), erythrocyte sedimentation rate (ESR), hsCRP, TC, LDL-C, HDL-C, TG, TC/HDL-C, HbA1c, MAP, mean baPWV, prednisone dosage per day, of which the majority had non-normal distribution (Shapiro-Wilk test, $P > .05$), between baseline and the 1-year follow-up using Wilcoxon signed-rank tests, which is a non-parametric statistical test for paired samples. McNemar test was used to test the significance of differences for paired nominal data (hydroxychloroquine, cyclophosphamide, mycophenolate mofetil, and ARB/ACEI usage) at baseline and 1-year follow-up.

Correlations between reductions in SLEDAI, ESR, hsCRP, WBC, and NRL and the changes in mean baPWV and lipid profile were tested using Spearman rank-order correlation test. To remove the potential influence of confounding variables on these correlation tests, covariate adjusted correlation analysis was performed with R package "psych",[27] and the following confounding factors were adjusted[15–17,28,29]: age, disease duration, changes in blood pressures (systolic blood pressure, diastolic blood pressure, mean arterial pressure, and blood pressure), and changes in medications (prednisone dosage, hydroxychloroquine usage, cyclophosphamide usage, mycophenolate mofetil usage, and ARB/ACEI usage). All the analyses were performed with R (version 3.5.3). P value less than .05 was considered as significant.

3. Results

A total of 51 SLE patients, with a median age of 31 years (range 24.0–41.5 years) (Table 1), fulfilled the inclusion criteria and were included in this study. All subjects were female. The median (Q1–Q3) disease duration of the patients was 238 weeks (range 54–432 weeks). We assessed the traditional cardiovascular risk factors at baseline (Table 1). Three (5.9%) patients were >55 years old, 15 (29.4%) patients had hypertension, 28 (54.9%) had dyslipidemia, 6 (11.8%) had a family history of premature coronary heart disease, and 2 (3.9%) had a BMI $\geq 28\,kg/m^2$.

Table 2 summarized the changes of biomarkers of inflammation, lipid profile and treatments between baseline and 1-year follow-up. The median SLEDAI at 1-year follow-up was 4 (2–6),

significantly different from a score of 10 (8–13) at baseline ($P < .01$). The median ESR was also significantly decreased between baseline and 1-year follow-up, with an absolute reduction of 7 mm/hour (1.5–35.0 mm/hour) ($P < .01$). Further-

Table 1

Baseline clinical characteristics of SLE subjects.

Characteristics	Baseline (N = 51)
Age [years, M (Q1,Q3)]	31 (24.0~41.5)
Menopause, n/N (%)	12/51 (23.5%)
Hypertension, n/N (%)	15/51 (29.4%)
Dyslipidemia, n/N (%)	28/51 (54.9%)
Body mass index [kg/m², M (Q1,Q3)]	20.7 (19.1~25.2)
Family history of premature coronary heart disease	6/51 (11.8%)
Serum Creatinine [mmol/L, M (Q1,Q3)]	62 (53.5~74.0)
eGFR [ml/min/1.73 m², M (Q1, Q3)]	109 (92.0-123.5)
Disease duration [months, M (Q1,Q3)]	238 (54~432)
SLEDAI [M (Q1,Q3)]	10 (8-13)
SLICC/ACR DI scores ≥1, n/N (%)	7/51 (13.7%)
Positive anti-dsDNA antibody, n/N (%)	44/51 (86.3%)
Positive lupus anticoagulant, n/N (%)	5/46 (10.9%)
Positive anti-cardiolipin antibody, n/N (%)	4/48 (8.3%)
Positive anti-β2GP1antibody, n/N (%)	8/48 (16.7%)
Complement 3 [g/L, M (Q1,Q3)]	0.60 (0.45~0.89)
Complement 4 [g/L, M (Q1,Q3)]	0.089 (0.059~0.164)
Prednisone usage, n/N (%)	50/51 (98.0%)

SLEDAI = systemic lupus erythematosus disease activity index, SLICC/ACR DI scores = SLICC/ACR Damage Index scores, eGFR = estimated glomerular filtration rate, calculated using the 2009 Chronic Kidney Disease Epidemiology Collaboration (CKD- EPI) equation. Premature coronary heart disease (female < 65 years old, male < 55 years old in first-degree relatives). Data were presented as median (interquartile range) or percentage (n/N). N = 59 if not otherwise specified.

Table 2

Changes in Biomarkers of Inflammation, Treatments, Lipoproteins and baPWV Values between baseline and 1-year follow-up (N = 51).

Measurement	Baseline	Follow-up	Difference-value	P value
SLEDAI	10 (8.0–13.0)	4 (2.0–6.0)	6 (4.0–10.0)	<.01*
WBC [×10⁹/L, M (Q1,Q3)]	5.49 (4.23–7.55)	5.97 (4.32–7.11)	0.11 (−1.84–1.28)	.97
NLR [M (Q1,Q3)]	2.67 (1.82–4.13)	2.30 (1.76–3.13)	0.35 (−0.72–1.86)	.20
hsCRP [mg/dl, M (Q1,Q3)]	1.32 (0.54–3.13)	0.99 (0.48–2.38)	0.12 (−0.29–0.98)	.17
ESR [mm/h, M (Q1,Q3)]	17 (10.0–49.5)	8 (5.0–14.5)	7 (1.5–35.0)	<.01*
TC [mmol/L, M (Q1,Q3))]	4.33 (3.87–5.64)	4.52 (3.69–5.10)	0.15 (−0.40–0.99)	.10
LDL-C [mmol/L, M (Q1,Q3)]	2.55 (2.29–3.67)	2.56 (2.06–3.06)	0.21 (−0.41–0.81)	.08
HDL-C [mmol/L, M (Q1,Q3)]	1.24 (0.95–1.44)	1.30 (1.08–1.50)	−0.19 (−0.3–0.17)	.10
TG [mmol/L, M (Q1,Q3)]	1.57 (1.09–2.94)	1.29 (0.84–1.77)	0.41 (−0.10–1.35)	<.01*
TC/HDL-C [M (Q1,Q3)]	3.78 (3.28–4.92)	3.41 (2.70–4.16)	0.72 (0.07–1.65)	<.01*
HbA1c [%, M (Q1,Q3)]	5.4 (5.1–5.9)	5.2 (5.0–5.7)	0 (−0.2–0.4)	.37
SBP [mmHg, M (Q1,Q3)]	123 (110-136)	116 (106-124)	7 (−5 to 18)	<.01*
DBP [mmHg, M (Q1,Q3)]	77 (65-84)	72 (65-80)	2 (-7 to 13)	.39
PP [mmHg, M (Q1,Q3)]	48 (42-55)	43 (38-48)	2 (−1 to 8)	.01
MAP [mmHg, M (Q1,Q3)]	93 (83–101)	84 (80–94)	3 (−5 to 13)	.09*
BaPWV [cm/s, M (Q1,Q3)]	1257 (1101–1464)	1176 (1070–1361)	71 (−54 to 137)	.01*
Prednisone dosage per day [mg, M (Q1,Q3)]	20.0 (7.5–40.0)	5.0 (5.0–7.5)	12.5 (2.5–33.8)	<.01*
12-month cumulative prednisone [g, M (Q1,Q3)]	−	4.56 (2.46–9.80)	−	−
Hydroxychloroquine usage, n (%)	40 (78.4)	48 (94.1)	−8 (15.26)	.04*
Cyclophosphamide usage, n (%)	29 (54.24)	12 (23.73)	17 (−0.2 to 0.4)	<.01*
Mycophenolate mofetil usage, n (%)	9 (18.64)	18 (35.59)	−9 (−4 to 18)	.08
ARB/ACEI, n (%)	24 (47.46)	23 (44.07)	1 (3.39)	1.00

McNemar test was used to test the significance of difference of hydroxychloroquine, cyclophosphamide, mycophenolate mofetil, and ARB/ACEI usage at baseline and 1-year follow-up. The difference of other factors between baseline and 1-year follow-up were tested by Wilcoxon signed-rank test.

* P value < .05.

SLEDAI = systemic lupus erythematosus disease activity index, WBC = white blood cell, NLR = neutrophils/lymphocyte ratio, hsCRP = high sensitivity C-Reactive Protein, ESR = erythrocyte sedimentation rate, TC = total cholesterol, LDL-C = low-density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, TG = triglyceride, TC/HDL-C = total cholesterol to high-density lipoprotein cholesterol ratio, HbA1c = hemoglobin A1c, SBP = systolic blood pressure; DBP: diastolic blood pressure, PP = pulse pressure, MAP = mean arterial pressure, baPWV = mean brachial-ankle pulse wave velocity, ARB/ACEI = angiotensin~converting enzyme inhibitor/angiotensin receptor blocker.

more, other inflammation biomarkers, such as NLR and hsCRP were not significantly changed. We also observed a significant decrease in the mean baPWV compared with baseline (P=.01), indicating decreased systemic arterial stiffness at 1-year follow-up. As for the lipid profile, a significant increase in TG (P<.01) and TC/HDL-C (P<.01), a predictor of coronary heart disease risk,[30] were observed at 1-year follow-up. Trends toward decreasing TC (P=.06), HDL-C (P=.10) and LDL-C (P=.08) were also observed. These results collectively suggested a more favorable lipid profile at 1-year follow-up.

Medications such as prednisone may influence lipid profile; therefore, we compared the treatments between baseline and 1-year follow-up. Significant differences in hydroxychloroquine usage (P=.04), cyclophosphamide usage (P<.01), and prednisone dosage per day (P=.04) were observed, while the mycophenolate mofetil usage and ARB/ACEI usage were not significantly different.

We next tested the relationships between inflammation biomarkers, lipid profile and baPWV (Supplementary Table 1, http://links.lww.com/MD/F189). Significant correlations were observed between the changes in ESR and TG (r=0.32, P=.02). Contrary to our expectation, the reduction in baPWV was not significantly correlated with the reduction in SLEDAI (r=0.22, P=.13, supplementary Table 1, http://links.lww.com/MD/F189 and supplementary figure 1, http://links.lww.com/MD/F188) or the changes in inflammation biomarkers. Previous studies reported that lipid profile was closely influenced by age, disease duration and medications (prednisone, hydroxychloroquine, cyclophosphamide, and mycophenolate mofetil).[15,28] BaPWV was significantly associated with blood pressure (systolic blood pressures, diastolic blood pressure, MAP, and PP).[29] In our dataset, we found changes in lipid profile were significantly associated with changes in cyclophosphamide usage, mycophenolate mofetil usage and ARB/ACEI usage (Supplementary Table 2, http://links.lww.com/MD/F190 and 3, http://links.lww.com/MD/F191). Significant correlations were also found between changes in baPWV and changes in systolic blood pressure, diastolic blood pressure, and MAP (Supplementary Table 2, http://links.lww.com/MD/F190). To remove the potential effect of those factors on the correlation analysis between the changes in SLEDAI and inflammation biomarkers with lipid profile and baPWV, we further analyzed the correlation adjusted by age, disease duration, blood pressure, and medications (prednisone dosage, cyclophosphamide usage, mycophenolate mofetil usage, and ARB/ACEI usage) (Table 3). With adjustment, the correlations between reduction in ESR (r=0.33, P=.02) with a decrease in TG remained significant. A reduction in SLEDAI was significantly associated with baPWV (r=0.36, P=.01). Of note, no significant correlation between changes in SLEDAI and lipid profile was observed with or without adjustment (Table 3, Supplementary Table 1, http://links.lww.com/MD/F189).

4. Discussion

Among SLE patients with a reduction in disease activity, we observed significant decreases in TG, TC/HDL-C, and arterial stiffness levels. Significant correlation between the reductions in ESR and reduction in TG was also observed. These findings provide a potential explanation for the beneficial effect of reducing inflammation on the cardiovascular disease risk in SLE. To our knowledge, this is the first study to analyze the correlations between disease activity, inflammation status, lipid profile, and arterial stiffness with longitudinal data. This approach reduced the impact of individual differences on the results compared with previous cross-sectional analyses.[10,18,19]

BaPWV is indicative of stiffness of the major arteries and the elasticity of arterial walls and is used as an early surrogate marker of arteriosclerosis.[31] In SLE patients, increased SLEDAI has been identified as predictive of increased carotid plaques and cardiovascular events.[7–9] Two groups specifically studied the relationships between baPWV and SLEDAI, and found SLEDAI was an independent risk factor for increased arterial stiffness.[25,32] In this study, reduced SLEDAI was significantly associated with a decrease in arterial stiffness levels, after adjustment for age, disease duration, blood pressure and several medications, which are confounding factors influencing baPWV or lipid profile.[15–17,28,29] This result directly suggested that the reduction of disease activity would improve arterial stiffness in SLE patients.

Inflammation plays a fundamental role in the process of atherosclerosis.[33] Traditional markers of inflammation, such as ESR and hsCRP, have been reported to be important risk factors for atherosclerosis in the general population.[34,35] In a cross-sectional study with 161 SLE patients, Ding et al identified that ESR was significantly associated with baPWV.[25] Two other studies with a smaller number of SLE patients also showed that

Table 3

Correlation between changes in inflammation and changes in lipid profile and baPWV values with adjustment for age, disease duration, blood pressure and medications.

Measurements	SLEDAI		ESR		hsCRP		WBC		NLR	
	Corr	P	Corr	P	Corr	P	Corr	P	Corr	P
TC	0.10	.49	0.21	.15	−0.02	.90	0.02	.88	0.09	.52
LDL-C	0.22	.12	−0.14	.34	0	.96	0.12	.40	0.17	.22
HDL-C	0.11	.43	−0.04	.77	0.27	.06	0.05	.71	−0.01	.93
TG	0	.95	0.33	.02*	0.05	.76	0.01	.92	0.22	.13
TC/HDL-C	−0.11	.46	0.24	.10	−0.20	.16	−0.07	.62	0.12	.40
baPWV	0.36	.01*	0.19	.19	0.22	.12	0.10	.47	−0.10	.49

Correlation analyses were performed with Spearman rank-order correlation test with adjustment for age, disease duration, changes in blood pressures (systolic blood pressure, diastolic blood pressure, mean arterial pressure and blood pressure), and changes in medications (prednisone dosage, hydroxychloroquine usage, cyclophosphamide usage, mycophenolate mofetil usage and ARB/ACEI usage).
* p value < 0.05.
TC = total cholesterol, LDL-C = low-density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, TG = triglyceride, TC/HDL-C = total cholesterol to high-density lipoprotein cholesterol ratio, baPWV = mean brachial-ankle pulse wave velocity, SLEDAI = systemic lupus erythematosus disease activity index, ESR = erythrocyte sedimentation rate, hsCRP = high sensitivity C-Reactive Protein, WBC = white blood cell, NLR = neutrophils/lymphocyte ratio.

ESR and hsCRP were significantly correlated with carotid to femoral PWV (cfPWV).[36,37] However, our analyses showed no significant correlation between the changes in markers of inflammation and baPWV (Table 3). The inconsistency between our result and that of previous studies might be due to the longitudinal nature of our study. Our results here suggest that lowering traditional inflammation markers may not be able to decrease the risk of atherosclerosis, despite the crucial role of inflammation on the development of atherosclerosis. Furthermore, it is still unclear whether the traditional markers of inflammation are drivers or side-effects of the atherosclerosis process.

A few studies further investigated the association between markers of inflammation and the lipid profile in SLE patients. Mok et al studied 289 SLE patients and found that patients with high hsCRP levels (>3.0 mg/L) had significantly increased TC/HDL-C ratios, TG/HDL-C ratios, and a trend towards an increased LDL-C/HDL-C ratio ($P = .06$) compared with low hsCRP group (≤3.0 mg/L).[38] Chung et al. reported that higher ESR was significantly associated with a low concentration of HDL-C in a group of 110 SLE patients.[39] While another study by Ali Abdalla et al with 48 SLE patients showed no significant correlation between ESR and lipid profile (TC, HDL-C, LDL-C, TG, and VLDL).[40] In contrast, our results showed that hsCRP was not associated with lipid profile, while reductions in ESR were significantly associated with a reduction in TG, suggesting that ESR may serve as surrogate markers for cardiovascular risk in SLE patients. Nevertheless, given that our findings were based on a limited number of patients, and inflammation markers like hsCRP and ESR fluctuate with active infection, the results should be treated with caution.[41]

Our study has several limitations. First, our study was an exploratory study based on a relatively small number of patients. Second, we did not account for patients who underwent lifestyle modifications that could result in lipid and arterial stiffness changes between baseline and the 1-year follow-up. Third, we analyzed the correlation between reduction in SLEDAI and changes in lipid profile and baPWV with adjustment to several medications.[17,28,29] However, as our study had a limited number of patients, we were unable to study the effect of one SLE medication on lipid and baPWV with precise control of other medications. A future longitudinal study is needed to fully investigate the relationship between changes in medications and lipid profile and arterial stiffness in SLE.

In summary, SLE patients experiencing a reduction in SLEDAI may have improved arterial stiffness. Reduction in ESR was correlated with decreases in TG. These findings may provide further insight into the beneficial effects of inflammation-lowering treatments on atherosclerosis risk in SLE.

Acknowledgments

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