Duplex Study of the Carotid and Femoral Arteries of Patients with Systemic Lupus Erythematosus: A Controlled Study

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ABSTRACT. ***Objective.*** To identify atherosclerosis in the common carotid (CCA) and common femoral arteries (CFA) of patients with systemic lupus erythematosus (SLE) and matched controls.

Methods. Fifty-one consecutive patients with SLE were enrolled in the study. Controls were matched by age, sex, ethnicity, and atherosclerosis risk factors. All patients and controls underwent ultrasonic biopsy (U-B) of the CFA and CCA, a noninvasive screening technique that detects early atherosclerotic plaques and changes. The U-B features were classified and scored as follows: class A: normal (score 0); class B: interface disruption (score 2); class C: intima-media granulation (score 4); class D: plaque without hemodynamic disturbance (score 6); class E: stenotic plaque (score 8); and class F: plaque with symptoms (score 10). Total score was calculated. Classes A and B indicate an intact media; classes D to F point to a significant medial involvement; class C signifies a borderline lesion with a potential for regression to normal or progression to a plaque.

Results. Mean ages were 40.5 years for SLE patients and 41 years for controls (p = 0.6). Ninety-six percent of the patients and controls were women. The mean disease duration of SLE was 8.65 years. Frequencies of risk factors among the SLE patients compared to controls were hypertension (30% vs 24%), smoking (23% vs 24%), and dyslipidemia (17.7% vs 17%). No patient had diabetes mellitus or family history of cardiovascular disease. A 3.17-fold increased rate of atherosclerotic plaques was detected in the SLE patients compared with controls (95% CI 1.08–10.9). Twenty-eight percent of SLE patients had at least a single class D–F lesion in one of the 4 vessels tested, compared with 10% in the control group (p = 0.02). In addition, the mean total U-B score of the SLE patients was significantly higher than that of the controls (5.65 vs 3.14; p = 0.02). Univariate analyses showed that the development of plaques in SLE was associated with a history of ischemic heart disease, hypertension, cardiovascular accident, and anemia. Multivariate analysis found plaques to be strongly associated with age, particularly in those older than 50 (OR 2.66, p = 0.000).

Conclusion. Patients with SLE have a high rate of atherosclerotic changes compared to controls. The development of atherosclerosis is strongly associated with age. (J Rheumatol 2004;31:909–14)

Key Indexing Terms:
SYSTEMIC LUPUS ERYTHEMATOSUS DUPLEX ULTRASONIC BIOPSY
ATHEROSCLEROSIS CAROTID ARTERY FEMORAL ARTERY

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Systemic lupus erythematosus (SLE) is a chronic autoimmune inflammatory disease of unknown cause that can affect the skin, joints, kidneys, lungs, nervous system, serous membranes, and other organs. During the last 3 decades, studies have identified atherosclerosis as a major contributor to the morbidity and mortality of patients with SLE[1]. Urowitz, *et al*[2] described the bimodal pattern of mortality in SLE, where late death in SLE is related to cardiovascular diseases. In a large mortality study[3], 25% of all deaths were due to myocardial infarction (10.5%), cerebrovascular event (4%), rupture of abdominal aortic aneurysm (1%), congestive heart failure (2%), and sudden death (8.1%). Moderate to severe atherosclerosis was detected in 54% of 40 autopsies of SLE patients[3]. The prevalence of ischemic heart disease in SLE patients ranges

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between 5% and 10%, and the mean age at diagnosis of ischemic heart disease was 48 years in one study[4].

These data suggest that SLE is associated with premature and accelerated atherosclerosis. Early detection and management of atherosclerosis may reduce morbidity and improve survival of patients with SLE.

Ultrasonic biopsy (U-B) is a noninvasive technique able to recognize early atherosclerotic changes in the blood vessel wall[5,6]. The technique identifies alterations in the morphology of the posterior wall and atherosclerotic plaques. The sonographic findings are classified into 6 classes, A to F, each of which is scored on a scale from 0 to 10 (Table 1). In Class B there are only intimal changes with an intact media. Class C is a borderline lesion, with potential for both full regression to normal and progression to a plaque. The U-B classes have a predictive value for development of symptomatic cardiovascular disease[5,6]. Classes D and E are associated with an increased risk of subsequent cardiovascular events even when risk factors are corrected and antiplatelet prophylaxis is given[6].

Our aim was to detect intimal and medial changes in the common carotid (CCA) and common femoral arteries (CFA) of patients with SLE and matched controls using U-B. In addition, we wanted to identify clinical and/or laboratory variables associated with the presence of atherosclerotic plaques in patients with SLE.

MATERIALS AND METHODS

Patients. Fifty-one consecutive SLE patients were enrolled in the study. There were no exclusion criteria. All patients fulfilled the American College of Rheumatology (ACR) for classification of SLE[7] and were followed at the Soroka Medical Center Lupus Clinic. A complete history, examination, and laboratory evaluation were performed and recorded in a standard protocol.

Controls. Controls were matched by age, sex, ethnicity, and atherosclerosis risk factors (arterial hypertension, smoking, diabetes mellitus, family history of arteriosclerosis, hyperlipidemia, and total cholesterol and LDL cholesterol levels). Arterial hypertension was defined as systolic blood pressure > 140 and diastolic blood pressure > 95 mm Hg. Hyperlipidemia was defined as LDL level > 130 mg/dl or total cholesterol level > 240 mg/dl.

U-B study. All patients and controls underwent ultrasound study of the CCA and CFA, using an ATL Ultra-Mark 9, HDI duplex scanner, with a high resolution 10 MHz probe. Both CCA and CFA were examined in the longitudinal plane, over 3 cm, immediately proximal to the bifurcation. By this technique the 3 planes of the posterior vessel wall could be identified and graded[5]. Measurements of the intima-media (I-M) complex thickness were made online on a frozen image using the built-in calipers. The complex thickness was defined as the average of 2 measurements selected at a distance of ± 30 mm proximal to the bifurcation where the adventitial line was the most uniform, measured on the far wall of the artery between the leading edge of the I line and the most distant edge of the adventitial line.

Ultrasound variables (dynamic range, depth range, power, reject, edge, grey scale, and smoothness) were constant during all examinations.

The ultrasonographer was blinded to the type of the examined subjects. Reproducibility and intraobserver variation were previously checked by repeat scanning of controls performed on different days. The coefficient of variation for the interobserver variability was 8.3% (r = 0.84, 95% confidence limits ± 0.13 of the observed values) and the intraobserver variability was 5.7% (r = 0.87; 95% confidence limits ± 0.06 mm of the observed values).

Outcome measures. The outcome measures included: (1) I-M thickness of the posterior wall from intima to adventitia inclusive; (2) mean total U-B score of the 4 vessels, ranging between 0 and 40, where 0 indicates that all 4 vessels are in class A and 40 signifies symptomatic plaques in all vessels; (3) frequency of various U-B classes for each blood vessel; (4) frequency of the highest score (of the 4 vessels) for each patient, with each patient being given the highest of the scores of the 4 blood vessels examined by U-B.

Predictors for atherosclerotic plaques in SLE patients. SLE patients were stratified into 3 groups: Group I (no atherosclerosis) included patients with U-B classes A and B only in all vessels and no medial involvement; Group II (atherosclerosis plaque group) included SLE patients with classes D to F in at least one of the 4 blood vessels and significant medial involvement with atherosclerotic plaques; and Group III (intermediate group) included patients with class C, a borderline lesion with potential for progression or regression.

Statistical analysis. Statistical analysis was carried out using the Stata program[8]. Descriptive statistics, chi-square tests, Fisher's exact tests, and t tests were used to compare cases and controls. Univariate analysis and multivariate linear regression models were performed to identify clinical and laboratory variables associated with atherosclerosis.

RESULTS

Table 2 shows the demographic features, atherosclerosis risk factors, levels of total and LDL cholesterol, and disease activity scores (SLE Disease Activity Index, SLEDAI, and Systemic Lupus International Collaborating Clinics, SLICC) of the SLE patients and controls. The mean age at U-B study, ethnic origin, and sex of SLE patients and controls were not statistically different. No patient or control

Table 1. Ultrasonic biopsy (U-B) classification.

Class	Description	Score
A	Normal, 3 US layers clearly separated. No disruption of lumen-intimal interface for at least 0.5 cm.	0
B	Interface disruption: lumen-intimal interface disruption at intervals < 0.5 cm.	2
C	Intima-media granulation: granular echogenicity of deep, normally unechoic intimal-medial layer.	4
D	Plaque without hemodynamic disturbance: wall thickening and increased density involving all US layers. No hemodynamic disturbance on duplex.	6
E	Stenotic plaque: as in D but with hemodynamic disturbance on duplex.	8
F	Stenotic plaque and presence of symptoms.	10

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Table 2. Demographics and atherosclerotic (AS) risk factors of patients with SLE and controls.

	SLE, n = 51	Controls, n = 47	p
Age, mean yrs (SD)	40.5 (14)	41.1 (14)	0.59
F:M	49:2	45:2	0.92
Ethnicity (%)			0.47
Ashkenazi Jews	32	39	
Sephardic Jews	53	57	
Arab	13	4	
Other	2		
SLE duration, mean yrs (SD)	8.65 (1.1)		
AS risk factors			
Hypertension	30%	24%	0.63
DM	0	0	1
Smoking	23%	24%	1
Family history	0	0	1
Hyperlipidemia	17.7%	17%	1
Mean LDL cholesterol, mg % (SD)	111	116	0.76
Mean total cholesterol, mg % (SD)	184	188	0.64
Mean SLEDAI (SD)(range)	8.7 (8.2) (0–36)		
Mean SLICC, (SD) (range)	2.1 (2.6) (0–12)		

DM: diabetes mellitus.

had diabetes mellitus. Mean SLEDAI score of the patients was 8.7 (range 0–36), and mean SLICC score was 2.1 (range 0–12).

The main clinical features of the SLE patients, occurring at any time during the SLE disease duration, included butterfly rash (43%), photosensitivity (66.6%), arthritis (58.8%), ischemic heart disease (11.8%), valvular heart disease (11.8%), congestive heart failure (4.0%), vein thrombosis (5.9%), livedo reticularis (17.6%), nephrotic syndrome (25.5%), renal failure (27.0%), cerebrovascular disease (cerebrovascular accident, CVA; 11.8%), seizures (5.8%), and cognitive dysfunction (8%).

Laboratory manifestations included antinuclear antibodies (98%), anti-DNA antibodies (74.5%), IgG anticardiolipin antibodies (aCL) (51%), and IgM aCL (53%).

The drugs given to the SLE patients during the course of the disease were nonsteroidal antiinflammatory drugs (39%), glucocorticosteroids (88%), antimalarials (90%), cyclophosphamide (23.5%), methotrexate (17.6%), and azathioprine (49%).

I-M thickness. Table 3 summarizes the I-M thickness of the CCA and CFA of patients and controls. The thickness of each vessel wall was measured at 2 different sites. Table 3 shows that I-M thicknesses of the vessels of cases and controls were not statistically different.

Total U-B score. The total U-B score for patients with SLE was 1.8 times higher than that of the controls. The mean total U-B score was 5.65 (± 6.4) for the SLE patients compared to 3.14 (± 5.82) in the controls (p = 0.02).

Table 4 shows the number of SLE patients and controls with the highest U-B score of the 4 vessels. Patients with SLE had significantly higher rates of AS plaques with an odds ratio (OR) of 3.17 [95% confidence interval (CI) 1.08–10.9, p = 0.034).

Table 3. Intima-media thickness (mm) of the posterior wall from intima to adventitia of common femoral (CFA) and common cartoid arteries (CCA) of SLE patients and controls.

	SLE Patients	Controls	p
Right CCA	1.42	1.44	0.92
Left CCA	1.31	1.31	0.98
Right CFA	1.38	1.23	0.27
Left CFA	1.27	1.23	0.58

Table 4. U-B classes of SLE patients and controls.

Highest U-B Score	SLE Patients, %	Controls, %
0 (Class A)	37	64
2 (Class B)	24	9
4 (Class C)	12	17
6 (Class D)	24	8
8 (Class E)	4	2

p = 0.027.

Twenty-eight percent of the SLE patients had an atherosclerotic plaque in at least one of the 4 vessels compared with only 10% in the control group (Table 4). Similarly, 67% of the controls were class A in all vessels compared with only 37% of the SLE patients. Although 6 SLE patients had a history of CVA, no association between the location of the plaque and the anatomic site of the CVA was found. Therefore, those plaques were given the score of 8 and not 10.

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Table 5 shows the U-B scores of CFA and CCA in the cases and controls. There is a trend toward a higher frequency of atherosclerotic plaques in the SLE group compared to controls.

Association between clinical and laboratory variables and atherosclerotic plaques. Sixty-five demographic (age, disease duration, ethnicity), clinical (all main clinical features of SLE), laboratory (complete blood count, chemistry, lipid profile, anti-DNA, complements, IgG and IgM aCL), and therapeutic variables (steroids, cytotoxic drugs) were included in the univariate analysis. The analysis was conducted to compare variables of the SLE patients without atherosclerosis (Group I) with those with overt plaques (Group II) and those with intermediate lesions (Group III). Table 6 shows various variables of SLE patients found to be associated in the univariate analysis with the presence of plaques. Carotid and femoral plaques were strongly associated with age, hypertension, coronary artery disease, and CVA.

Sixteen variables (Table 6) were included in 2 multivariate regression models. In the first mode, patients were classified into 2 groups: those age 50 or above and those below age 50. In this model the development of atherosclerosis was associated only with age > 50 (OR 2.66, 95% CI 1.63–4.35, p = 0.000).

In a second model, the age of patients was included without stratification into age groups. In this model, atherosclerosis was significantly associated with age (regression coefficient = 0.031, p = 0.000) and marginally associated with hypertension (p = 0.086). No other variable was associated with atherosclerosis in either model.

DISCUSSION

Later morbidity and mortality in SLE is largely caused by atherosclerosis[1-3]. The atherosclerotic process is usually widespread and may affect the aorta, coronary arteries, the common carotid, cerebral, and main arteries of the extremities[2]. Carotid duplex scanning is a noninvasive method for screening for both symptomatic and asymptomatic atherosclerosis. The detection of atherosclerotic plaques in the carotid arteries is associated with the presence of coronary artery disease, and atherosclerotic plaque is an independent predictor for myocardial infarcts[5,6,9]. Early detection of asymptomatic atherosclerotic disease allows early intervention and possibly prevention of symptomatic cardiovascular disease. Few studies have reported the frequency of athero-

Table 5. U-B classification of the CFA and CCA of SLE patients and controls. Results are expressed as percentages.

	Right CCA		Left CCA		Right CFA		Left CFA	
Class	SLE	Controls	SLE	Controls	SLE	Controls	SLE	Controls
A and B	85	87	81	76	75	89	79	90
C	8	9	16	17	8	8	8	4
D and E	8	4	4	6	8	2	16	4

Table 6. Variables associated with atherosclerotic plaques (univariate analysis). Results are expressed as percentages unless otherwise defined.

Variable	Group I (No AS)	Group II (AS)	Group III (Intermediate)	p
Mean age, yrs	32.5	55.6	46.1	0.00
Disease duration > 5 yrs	45	72	33	0.17
MI/IHD	3	35	0	0.006
Arthritis	50	85	50	0.09
Brain hemorrhage	0	15	0	0.06
Organic brain syndrome	3	23	0	0.07
CVA	3.1	31	0	0.02
Claudication	0	15	0	0.06
Ischemic ulcers	3	15	33	0.07
Hypertension	23	54	17	0.009
PVD	0	15	0	0.07
Smoking	17	31	33	0.47
Renal failure	17	43	33	0.18
Anemia	89	31	60	0.001
Mean total cholesterol, mg/dl	170	209	193	0.06
Mean LDL level, mg/dl	102	126	114	0.25

MI/IHD: myocardial infarction/ischemic heart disease, CVA: cardiovascular accident, PVD: pulmonary vascular disease.

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sclerotic plaques in patients with SLE. Petri, *et al*[10] found the frequency of AS plaques in SLE to be 8%. Manzi, *et al*[11] identified focal plaques in 40% of the carotid arteries of women with SLE. However, 15% of the women had known coronary artery disease or CVA. In the older age group, plaques were detected in 71% of women with SLE aged 55–64 years. In a recent study[12], 37% of SLE patients had atherosclerotic plaques in the carotid arteries.

Our study is the first to scan the CFA in addition to the CCA. We also used U-B, which allows detection of early changes that occur in the vessel wall before the development of an overt plaque. This includes lumen-intimal interface disruption (U-B class B) and granular echogenicity of deep I-M layer (U-B class C).

In a series of 2000 asymptomatic individuals[5], all subjects in the 10–29 age group were in class A, 7% in the 30–49 age group were in class B, and only 5% of individuals in the age group 40–49 had atherosclerotic plaques. Two hundred four asymptomatic subjects underwent a cardiac stress test. No patients in classes A and B had ischemic changes. However, a pathological stress test was observed in 6.3%, 25%, and 42% of subjects in classes C, D, and E, respectively. Fifty-five percent of symptomatic patients (class E) had ischemic changes in a stress test.

The significance of class B is not clear. B class differs from A class by the ultrasonic wall structure analysis and therefore, despite identical present-time clinical significance, gets a different score. In a 6 year followup, 27% of subjects progressed from class A to class B, while 13% progressed from class B to class C[5].

In our study, patients with SLE were found to have a higher frequency of abnormal CCA and CFA. This was documented by the total U-B scores and the frequency of atherosclerotic plaques. The total U-B score for patients with SLE was 1.8 times higher than that of the controls. In addition, only 37% of the SLE patients had normal CCA and CFA by U-B scan (class A). A 3.17-fold increased rate of atherosclerotic plaques was detected in the SLE patients compared with controls (28% in SLE compared with 10% in controls). Similarly, Roman, *et al*[12] found the frequency of atherosclerotic plaques in their SLE patients to be 2.2 times higher than controls (37% vs 16%).

A few studies[13,14] have shown that patients with SLE had a higher I-M thickness compared with controls. However, those reports were based on few cases of SLE patients. We measured the I-M thickness at 2 different sites on each vessel wall. Our I-M thickness is higher than other published data. This is explained by our measurement technique that included the adventitial layer within the measured wall thickness. This way of measuring is inaccurate among an aged population with irregularity in the outer layer. However, our young study patients had very little adventitial irregularity. We found this wall thickness measurement to be more reproducible when online, onscreen analysis is done. The I-M thickness measurements of the CFA and CCA of our SLE patients and controls were not significantly different. Roman, *et al*[12] found the I-M thickness was significantly greater in controls. The data indicate that I-M thickness is not a sensitive instrument for identifying early and asymptomatic atherosclerosis.

By univariate analysis, we found atherosclerotic plaqueswere associated with age, coronary artery disease, CVA, and hypertension. A trend was found to suggest higher levels of LDL and total cholesterol among patients who had atherosclerotic plaques.

Previous reports identified a link between plaques and various clinical variables. In a univariate analysis, Maksimowicz, *et al*[15] found an association between plaques and traditional risk factors including male gender, family history, smoking, hypertension, and high mean cholesterol. Another study[12] reported a link between plaques and pulmonary hypertension, Raynaud's phenomenon, postmenopausal status, hypertension, and duration of SLE. However, age eliminated all other factors in multivariate analysis.

In our study, the multvariate linear regression model found plaques to be associated only with age of the SLE patients. This observation indicates that plaques are more likely to develop late in the course of SLE. This finding indirectly supports the bimodal pattern of mortality in SLE, described by Urowitz, *et al*, 26 years ago[2].

In summary, patients with SLE have a high frequency of abnormal CCA and CFA, manifested by early (pre-plaques) changes as well as atherosclerotic plaques. The development of atherosclerotic plaques is mainly associated with age, particularly in those over 50.

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