Impairment of arterial distensibility in premenopausal women with systemic lupus erythematosus

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Abstract

Background and aim: Systemic lupus erythematosus (SLE) is a chronic inflammatory autoimmune disease affecting young women and is associated with increased frequency of atherosclerotic vascular diseases. Pulse wave velocity (PWV) is an index of arterial stiffness and a marker of cardiovascular events. This study aimed to investigate arterial distensibility using carotid-femoral (aortic) PWV measurements in premenopausal women with SLE.

Methods: We recruited 24 premenopausal women with SLE (SLE duration: 5.3±4.6 years) and 24 age- and sex-matched controls. Aortic PWV was determined by using an automatic device, the Complior Colson (France), which allowed on-line pulse wave recording and automatic calculation of PWV.

Results: The carotid-femoral PWV (8.98±2.05 vs. 8.05±0.94 m/s), systolic blood pressure (117.08±17.12 vs. 106.87±11.96 mmHg), pulse pressure (45.62±11.91 vs. 38.33±9.04 mmHg), heart rate (81.41±9.20 vs. 71.12±10.32 beat/min) and serum glucose levels (89.68±8.12 vs. 73.80±10.72 mg/dl) were significantly higher in premenopausal women with SLE, as compared with control subjects (p=0.04, p=0.02, p=0.02, p=0.001, p <0.001, respectively). We found a significant correlation between PWV and age, body mass index, waist-to-hip ratio, heart rate and blood pressure.

Conclusion: Arterial distensibility is decreased in premenopausal women with SLE.

Key words: pulse wave velocity, arterial stiffness, systemic lupus erythematosus, inflammation

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Introduction

Systemic lupus erythematosus (SLE) is associated with several cardiovascular manifestations, in part due to accelerated atherosclerosis. Women with SLE have an increased risk of coronary artery disease [1, 2]. The pathogenesis of accelerated atherosclerosis in SLE is not completely understood and likely multifactorial. The risk factors for atherosclerosis (e.g. diabetes mellitus, hypertension, hyperlipidaemia, obesity, cigarette smoking) are common among patients with SLE, in part due to the adverse effects of steroids [3-7]. Atherosclerosis is an inflammatory disease with immune cell activation, inflammation, plaque formation and rupture [8]. In humans and animal models of SLE, the degree of systemic inflammation correlates with development of atherosclerosis [3, 9, 10]. Autoimmune vascular damage in SLE may predispose to atherosclerotic plaque formation by a number of possible factors: e.g. deposition of immune complexes, anti-beta 2-glycoprotein I, increased asymmetric dimethylarginine, plasminogen activator inhibitor and transforming growth factor beta-1 [11-15].

Systemic inflammation is an important factor in the initiation or the progression of atherosclerosis [16]. Damage to the arterial wall due to inflammation and atherosclerosis causes decreased arterial distensibility and compliance

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[17-19]. Decreased compliance of arterial vessels may produce increased shear stress [20]. Non-invasive ultrasound techniques are used to evaluate the vascular system and cardiovascular condition. One such technique, pulse wave velocity (PWV), which is defined as the arterial pulse's velocity of moving along the vessel wall, is an indicator of arterial distensibility. PWV is inversely correlated with arterial distensibility and relative arterial compliance [21]. Inflammation may impair vascular function and lead to an increase in arterial PWV. This study aims to investigate arterial distensibility using carotid--femoral (aortic) PWV measurements in premenopausal women with SLE.

Methods

Study protocol

Patient population

We recruited 24 women (SLE duration: 5.3±4.6 years) with SLE diagnosed according to standard criteria [22] and 24 age- and sex-matched controls. The median SLE Disease Activity Index was 2.0 at the time of study. All patients were receiving corticosteroid treatment and hydroxychloroquine. All subjects were non-smokers. All subjects gave their consent for inclusion in the study. The investigation conforms with the principles outlined in the Declaration of Helsinki. Exclusion criteria were: previous myocardial infarction, constrictive, restrictive or dilated cardiomyopathy, heart failure, valvular heart disease, peripheral arterial disease, cerebrovascular disease, renal failure, anaemia (Hct <30%), conduction or rhythm disorders recorded on standard ECG, systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg [23], body mass index ≥35 kg/m^2 and waist/hip ratio ≥1 cm. None of our patients was treated at the time of examination with antihypertensive agents or statins.

Body mass index and waist-hip ratio measurements

Body mass index (kg/m^2) was calculated by dividing body weight in kilograms by the square of body height in metres. The circumference of the waist divided by the circumference of the hip gave the waist-to-hip ratio.

Blood pressure and pulse wave velocity measurements

In each subject the carotid-femoral PWV and arterial blood pressure were measured by the same observer in each subject in the supine position after at least 20 min of rest. Blood pressure was measured using a mercury sphygmomanometer with a cuff appropriate to the arm circumference (Korotkoff phase I for systolic blood pressure and V for diastolic blood pressure). In each subject two blood pressure measurements were performed, and their mean was used for computation of [1] pulse pressure = systolic blood pressure – diastolic blood pressure, and mean blood pressure = (systolic blood pressure + 2 × diastolic blood pressure)/3.

Arterial distensibility was assessed by automatic carotid-femoral (aortic) PWV measurement using the Complior Colson device (Createch Industrie, France); the technical characteristics of this device have been described, and indicate inter- and intra-observer repeatability coefficient values >0.9 [24]. The Complior system is designed to determine the arterial distensibility from the pulse wave time interval and velocity measurements. This system is made of a Complior PC board installed in an IBM APTIVA. The Complior software makes use of the computer resources for the calculation, the display and the recording of the data. Two acoustic sensors deliver the signals of the pulse wave to the acquisition board. A pedal triggers the data acquisition. The Complior kit is made of: (I) a Complior data acquisition board installed in an APTIVA IBM PC computer; the PC runs with a 486DX2 50 MHz and has a 270 Mo hard drive, (II) the Complior software version 3.0 installed in the IBM PC, (III) two acoustic sensors, (IV) a trigger pedal, and (V) a Canon BJ-200ex printer. The computer is configured for 220V AC mains. PWV along the aorta can be measured by using two ultrasound or strain--gauge transducers [non-invasively using a TY-306 Fukuda pressure sensitive transducer (Fukuda, Tokyo, Japan)] fixed transcutaneously over the course of a pair of arteries separated by a known distance: the femoral and right common carotid arteries. During pre-processing analysis the gain of each waveform was adjusted to obtain an equal signal for the two waveforms. During PWV measurements, after pulse waveforms of sufficient quality were recorded, the digitisation process was initiated by the operator and automatic calculation of the time delay between two upstrokes was started. Measurement was repeated over 10 different cardiac cycles, and the mean value was used for the final analysis.

Laboratory measurements

Overnight-fasting blood samples were taken in the morning from the antecubital vein. Biochemical parameters (i.e. serum fasting blood glucose, triglyceride, total cholesterol, HDL and LDL cholesterol) were measured using an Abbott C8000 (Abbott, Japan) automatic analyser. Glucose was analysed using the hexokinase method. Total cholesterol, HDL, LDL cholesterol and triglycerides were measured by enzymatic methods in all samples. Blood cells were counted using the HMX (Beckman Coulter, USA) analyser.

Statistical analysis

Statistics were obtained using the ready-to-use program SPSS version 8.0. All the values were expressed as mean ± standard deviation. The obtained results were assessed by independent samples test. Correlations were calculated with the Pearson test. A p value < 0.05 was considered significant.

Results

The carotid-femoral PWV, systolic blood pressure, pulse pressure, heart rate and serum glucose levels were significantly higher in premenopausal women with SLE than in control subjects (p=0.04, p=0.02, p=0.02, p=0.001, p <0.001, respectively) (Table I). We found a significant correlation between PWV and age, body mass index, waist-to-hip ratio, heart rate and blood pressure (Table II) (Figure 1).

Discussion

In the present study, we found that the carotid-femoral PWV, systolic blood pressure, pulse pressure, heart rate and serum glucose levels were increased in premenopausal women with SLE, as compared with control subjects. We found a significant correlation between PWV and age, body mass index, waist-to-hip ratio, heart rate and blood pressures.

Table I. Basic data, haemodynamic values and laboratory data in control subjects and patients with systemic lupus erythematosus

	SLE	Control	p
Age [years]	33.6±9.6	30.2±9.2	0.22
Body mass index [kg/m^2]	25.50±5.31	23.04±3.89	0.07
Waist-to-hip ratio	0.82±0.00	0.80±0.00	0.23
Systolic blood pressure [mmHg]	117.08±17.12	106.87±11.96	**0.02**
Diastolic blood pressure [mmHg]	71.66±8.80	68.54±8.90	0.22
Mean blood pressure [mmHg]	87.06±11.42	81.30±9.07	0.05
Pulse pressure [mmHg]	45.62±11.91	38.33±9.04	**0.02**
Heart rate [beat/min]	81.41±9.20	71.12±10.32	**0.001**
Pulse wave velocity [m/s]	8.98±2.05	8.05±0.94	**0.04**
Pulse wave propagation time [s]	68.00±12.36	72.64±7.68	0.21
Total cholesterol [mg/dl]	153.88±38.36	149.10±23.43	0.72
LDL cholesterol [mg/dl]	86.93±25.28	83.70±19.55	0.73
HDL cholesterol [mg/dl]	53.88±15.80	46.70±10.97	0.21
Triglyceride [mg/dl]	79.41±34.86	72.50±24.49	0.58
Glucose [mg/dl]	89.68±8.12	73.80±10.72	**<0.001**

Abbreviations: SLE – systemic lupus erythematosus, LDL – low density lipoprotein, HDL – high density lipoprotein

Table II. Correlation between PWV and basic data and haemodynamic values

	p	r
Age [years]	<0.001	0.68
Body mass index [kg/m^2]	0.03	0.30
Waist-to-hip ratio	0.02	0.31
Systolic blood pressure [mmHg]	<0.001	0.50
Diastolic blood pressure [mmHg]	0.007	0.38
Mean blood pressure [mmHg]	0.001	0.48
Pulse pressure [mmHg]	<0.001	0.48
Heart rate [beat/min]	0.001	0.44
Total cholesterol [mg/dl]	0.19	0.25
LDL cholesterol [mg/dl]	0.07	0.36
HDL cholesterol [mg/dl]	0.29	0.21
Triglyceride [mg/dl]	0.17	0.27
Glucose [mg/dl]	0.05	0.38

Abbreviations: PWV – pulse wave velocity, LDL – low density lipoprotein, HDL – high density lipoprotein

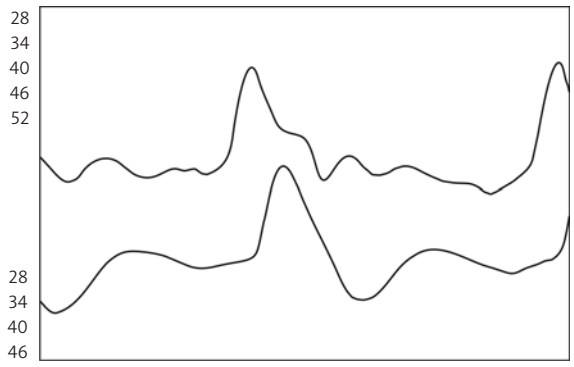

Figure 1. The carotid-femoral pulse wave velocity (PWV) measurement PWV is calculated from measurements of pulse transit time and the distance (the distance between two recording sites is measured on the surface of the body in metres) travelled by the pulse between two recording sites

Pulse wave velocity is a technique in which large artery elasticity is assessed from analysis of the peripheral arterial waveform [24] and is calculated from measurements of pulse transit time and the distance travelled by the pulse between two recording sites. Pulse wave velocity is more a marker of compliance than distensibility and refers to aortic compliance more than arterial compliance. It is an index of arterial elasticity and a surrogate marker for coronary atherosclerosis. SLE is a chronic inflammatory autoimmune disease affecting young women associated with increased atherosclerotic vascular diseases [20]. In SLE, immune complexes may be a cause of arterial damage which initiates atherogenesis [25]. These complexes bind to receptors on the endothelium, triggering upregulation of adhesion molecules such as E-selectin, intercellular adhesion molecules 1 (ICAM-1) and vascular cell adhesion molecules 1 (VCAM-1). Finally, inflammatory cells and oxidised low density lipoprotein (LDL) can be activated [25]. This inflammation may impair vascular function and lead to an increase in aortic PWV.

Complement (C) activation may increase endothelium permeability, and C-reactive protein (CRP) may be associated with vascular damage [26]. Lower leukocyte count and presence of antibodies to native DNA are associated with higher PWV among the SLE women [20]. An inverse correlation has been shown between use of hydroxychloroquine, a drug with cardioprotective properties, and PWV [20]. Higher creatinine levels were also associated with PWV in the SLE women [20]. Kidney disease is associated with hypertension and dyslipidaemia, which are PWV risk factors [20]. In the SLE group, we did not find any correlation between PWV and hydroxychloroquine use, urea, creatinine and inflammation markers such as C3, C4, rheumatoid factor, CRP, sedimentation and leukocytes ($p > 0.05$).

Blood pressure and heart rate are known determinants of measured aortic PWV [27, 28]. At the time of the carotid--femoral PWV measurement, the premenopausal women with SLE had a higher systolic blood pressure, pulse pressure and heart rate than the control subjects. Inflammation, corticosteroid use (corticosteroids can elevate hyperglycaemia and blood pressure and many have associated mineralocorticoid effects) and increased plasma glucose levels are associated with aortic PWV [29, 30]. Increased body-mass index, increased waist-to-hip ratio, and kidney disease with resulting increased blood pressure may play a role in the development of reduced arterial distensibility in premenopausal women with SLE. Arterial distensibility depends on variation in blood pressure level, especially pulse pressure. With increasing age, there is a gradual shift from diastolic blood pressure to systolic blood pressure and pulse pressure. Stiffness becomes higher at high blood pressure and lower at low blood pressure, through mechanical change in arterial wall stretching and resulting change in the contribution of elastin and collagen fibres to the elastic modulus [31].

Increased resting heart rate also increases cardiovascular mortality [32]. The higher resting heart rate in premenopausal women with SLE may reflect inactivity due to joint inflammation. Albaldejo et al. [33] reported, using cardiac pacing, a non-significant positive correlation between PWV and heart rate. Increased heart rate shortens the time available for recoil, which results in arterial stiffening [34].

Increased body-mass index and waist-to-hip ratio might adversely affect the cardiovascular system through association with hyperlipidaemia, hypertension and inflammation. Obesity is a common side effect of long--term corticosteroid use. It could be a marker of inactivity and insulin resistance [20, 35]. In this study, we found a significant correlation between carotid-femoral (aortic) PWV, body-mass index and waist-to-hip ratio. These findings showed that arteries become less elastic with increased body-mass index and waist-to-hip ratio.

In conclusion, the present study showed that arterial distensibility is decreased in premenopausal women with SLE.

Limitations

Despite the method which we used for measuring the stiffness of the aorta, indirectly, it is the best described method. The pressure waveforms are easily recorded in both areas, the distance between two areas is long enough, and elasticity of the arterial wall could be reflected on a large scale as in the aorta [24]. We took great care to exclude subjects with known cardiovascular disease or risk factors which may influence aortic stiffness and PWV values. Finally, the number of patients with SLE included in our study is relatively small; therefore, our results need confirmation in larger studies.

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Upośledzenie podatności naczyń tętniczych u miesiączkujących kobiet z toczniem rumieniowatym układowym

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Streszczenie

Wstęp i cel: Toczeń rumieniowaty układowy (ang. *systemic lupus erythematosus*, SLE) jest przewlekłą chorobą zapalną o podłożu autoimmunologicznym, która często występuje u młodych kobiet i jest związana ze zwiększoną częstością występowania miażdżycy naczyń. Prędkość fali tętna (ang. *pulse wave velocity*, PWV) jest miarą sztywności naczyń tętniczych i ma znaczenie prognostyczne w przewidywaniu wystąpienia powikłań sercowo-naczyniowych. Niniejsze badanie miało na celu ocenę podatności naczyń tętniczych u miesiączkujących kobiet z SLE.

Metody: Badaniem objęto 24 miesiączkujące kobiety z SLE (czas trwania choroby 5,3±4,6 roku) i 24 kobiety zdrowe, dopasowane pod względem wieku do grupy SLE. Podatność aorty zmierzono za pomocą automatycznego urządzenia Complior Colson (Francja), które pozwala na jednoczesną rejestrację fali tętna i automatyczne obliczanie PWV.

Wyniki: Wartości szyjno-udowego PWV (8,98±2,05 *vs* 8,05±0,94 m/s), skurczowego ciśnienia tętniczego (117,08±17,12 *vs* 106,87±11,96 mmHg), ciśnienia tętna (45,62±11,91 *vs* 38,33±9,04 mmHg), częstotliwości rytmu serca (81,41±9,20 *vs* 71,12±10,32 uderzeń/min) i stężenia glukozy (89,68±8,12 *vs* 73,80±10,72 mg/dl) były istotnie wyższe u kobiet z SLE niż w grupie kontrolnej (odpowiednio: p=0,04, p=0,02, p=0,02, p=0,001, p <0,001). Wykazano również istotną korelację pomiędzy PWV a wiekiem, indeksem masy ciała, wskaźnikiem talia/biodro, częstotliwością rytmu serca i wartościami ciśnienia krwi.

Wniosek: Podatność naczyń tętniczych jest obniżona u miesiączkujących kobiet z SLE.

Słowa kluczowe: podatność naczyń, prędkość fali tętna, toczeń rumieniowaty

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