ABSTRACT

Background:

Cardiovascular disease is a major cause of morbidity and mortality in systemic lupus erythematosus (SLE) patients. Interestingly, SLE was suggested to be a coronary artery disease equivalent. Accurate risk stratification would require a simple, non-invasive index integrating all traditional and emerging risk factors. Vascular stiffness – proven to have better predictive value for cardiovascular events than traditional risk factors in hypertensives and patients with coronary artery disease – fulfills these requirements.

Aim of the study:

We sought to determine whether arterial stiffness is increased in SLE patients compared to healthy controls and to correlate the arterial stiffness in SLE patients with cardiovascular risk factors.

Subjects and Methods:

This study included 100 subjects divided into 50 SLE patients and 50 age- and gender-matched healthy individuals. All individuals underwent standard clinical evaluation. Assessment of aortic stiffness was performed by calculation of aortic elastic indices using M-mode transthoracic echocardiography (TTE). Endothelial function was assessed using brachial flow mediated dilation (FMD). Carotid duplex ultrasound was performed to measure quality arterial stiffness (QAS) parameters using Esaote MyLab 60 (phased array sector probe PA230, frequency range 1-4 MHz). We calculated carotid-femoral pulse wave velocity (cf-PWV) as the carotid-femoral travel distance divided by the transit time ($\Delta L/\Delta t$).

Results:

Our study included 50 SLE individuals (47 females, median age 29, range 17 – 45 years) and 50 age- and gender-matched healthy individuals. SLE patients had higher median aortic stiffness index (SI) and lower strain and distensibility, compared to controls (p value for all <0.001). SLE patients had significantly impaired FMD compared to controls: the median (range) in SLE patients was 8.82 (2.5 - 21.87), compared to 19 (12 - 37.5) in controls (z = -7.695, p < 0.001). Regarding QAS parameters, SLE patients had significantly lower median carotid distension, distensibility coefficient, and compliance coefficient, with higher median carotid SI, carotid pulse wave velocity (PWV), and augmentation index (AI), compared to controls (p value for all ≤ 0.001). SLE patients had a higher median cf-PWV 6.5 m/sec (4.8 – 11.8), compared to a median of 4.6 m/sec (3.8 – 6.9) in controls (z = -8.193, z =

Linear regression analysis to adjust for hypertension and diabetes mellitus yielded a statistically significant difference between both groups for all of the above parameters (p = 0.014 for maximum carotid IMT and <0.001 for remaining parameters), with the exception of the maximum carotid augmentation index (p = 0.184).

Carotid AI and FMD were significantly associated with hypertension, with hypertensive patients having higher median AI compared to non-hypertensives (z = -2.749 & -2.298, p = 0.006 & 0.022, respectively). There was a positive association between SLE duration and each of carotid PWV, carotid SI, and cf-PWV (r = 0.363, 0.361, and 0.302, respectively; p = 0.01, 0.011, and 0.033, respectively).

Conclusion:

SLE patients have significantly impaired FMD and increased arterial stiffness compared to healthy controls. This is true even after adjusting for hypertension and diabetes mellitus, highlighting the fact that SLE is an independent cardiovascular risk factor. SLE duration is an important predictor of arterial stiffness. These findings emphasize the need for early diagnosis of SLE and aggressive risk factor modification.

Key words: Systemic lupus erythematosus, arterial stiffness, flow-mediated dilatation

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