Progression of subclinical atherosclerosis in systemic lupus erythematosus of low disease activity: three-year follow-up and comparison to rheumatoid Arthritis

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Background: Both systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) are characterised by accelerated atherosclerosis compared to the general population. Prospective studies have shown that atherosclerosis progression is halted in patients with RA of low disease activity, but it is unclear if maintaining lupus low disease activity state mitigates accelerated atherosclerosis due to SLE.

Objectives: To prospectively assess the risk and determinants of atherosclerosis progression in SLE versus RA patients of low disease activity.

Methods: We performed carotid and femoral artery ultrasound to detect atherosclerotic plaques at baseline on 345 participants with SLE, RA, and healthy controls, individually matched for age and gender, after excluding patients with atherosclerotic cardiovascular disease, malignancy and diabetes. After 3 years of follow-up, patients with SLE (n=89) and RA (n=64) maintaining low disease activity for >75% of the follow-up time, and their matched controls (n=72) underwent repeat ultrasound to identify those with atherosclerosis progression, as defined by the development of new plaques compared to baseline. We applied multiple logistic regression models to assess the odds of atherosclerosis progression between SLE, RA, and control participants, and used the stepwise backward elimination algorithm (p<0.1) to examine potential associations with SLE damage index, antiphospholipid antibodies, corticosteroids, hydroxychloroquine, immunosuppressives, and disease duration in patients with SLE, adjusting for use of antiplatelet agents,
statins, and traditional cardiovascular risk factors with the European Society of Cardiology’s SCORE risk estimation of 10-year fatal cardiovascular disease.

**Results:** Atherosclerotic plaque progression was detected in 21% of SLE patients, 17% of RA patients, and 8% of controls (p=0.078). After controlling for SCORE, antiplatelet agent use and statins, the rate of atherosclerosis progression compared to healthy controls was significantly higher in SLE (OR=3.05, 95% Confidence Interval (CI) 1.06–8.79, p=0.039), but not in RA (OR=2.11, 95% CI: 0.72 to 6.23, p=0.176). In patients with SLE, longer disease duration at baseline (OR=1.11, 95% CI: 1.02 to 1.21, p=0.015), antiphospholipid antibody positivity (OR=7.04, 95% CI: 1.57 to 31.58, p=0.011), cumulative corticosteroid dose during follow-up (OR=1.16, 95% CI: 0.99 to 1.35, p=0.069), treatment with antiplatelet agents (OR=0.21, 95% CI: 0.05 to 0.99, p=0.049), and the SCORE prediction (OR=1.67, 95% CI: 0.91 to 3.08, p=0.099) were included in the multivariable model as determinants of atherosclerosis progression. No disease-related determinants were significantly associated with atherosclerosis progression in patients with RA.

**Conclusions:** Unlike RA, atherosclerosis progression is accelerated in SLE even in the setting of low disease activity. In addition to SCORE, longer disease duration at baseline, antiphospholipid antibody positivity, and higher cumulative corticosteroid dose during follow-up increase the odds of atherosclerosis progression in patients with SLE of low disease activity.

**References:**


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