

metabolic biomarkers, increasing age, Korean ethnicity, higher disease activity and increased serum leptin performed similarly (AUC ROC=0.75).

**Conclusions:** SLE patients who develop incident MetS exhibit a more inflammatory disease phenotype, with higher corticosteroid exposure in the preceding visit. Increased serum leptin concentration is independently associated with future onset of MetS. These factors can help predict those at increased risk of developing future MetS and may help target patients for more focused cardiovascular disease prevention.

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**OP0119** **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<sup>1</sup> 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, anti-phospholipid 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.

**REFERENCE:**

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**OP0120** **INFLUENCE OF EPIDEMIOLOGY AND ETHNICITY ON SYSTEMIC EXPRESSION OF PRIMARY SJÖGREN SYNDROME IN 9974 PATIENTS**

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**Objectives:** To analyse the influence of epidemiology and ethnicity on the clinical systemic presentation at diagnosis of primary Sjögren syndrome (SjS).

**Methods:** The Big Data Sjögren Database included 10 475 worldwide patients from 22 countries fulfilling the 2002 criteria. Age at diagnosis, gender and ethnicity (77% White, 14% Asian, 6% Hispanic, 1% Black/African American, 2% others) were correlated with systemic involvement at diagnosis (retrospectively scored in 9974 patients using ESSDAI/clinESSDAI)

**Results:** Men had higher mean ESSDAI (8.0 vs 5.9, p<0.001) and clinESSDAI (8.4 vs 6.1, p<0.001) in comparison with women; the domains more active in men included lymphadenopathy (p<0.001), glandular (p<0.001), pulmonary (p=0.001), PNS (p<0.001) and CNS (p<0.001). Highest scores were also reported in patients with young-onset disease (<35 years) with respect to older onset (>65 years) for ESSDAI (6.5 vs 5.6, p=0.002) and clinESSDAI (6.4 vs 5.8, p=0.034). Highest ESSDAI scores were reported in Black/African American, followed by White, Asian and Hispanic patients (6.7 vs 6.3 vs 5.4 vs 4.9, respectively, p<0.001). BAA patients showed the highest frequency of activity in lymphadenopathy, articular, PNS, CNS and biological domains, Whites in glandular, cutaneous and muscular, Asians in pulmonary, renal and haematological and Hispanics in the constitutional domain.

**Conclusions:** This study provides the first evidence for a strong influence of epidemiology and ethnicity on the systemic phenotype at diagnosis of primary SjS

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**OP0121** **LUPUS ANTICOAGULANT IS ASSOCIATED WITH THROMBOTIC EVENTS IN HEALTHY CARRIERS: RESULTS FROM A PROSPECTIVE LONGITUDINAL STUDY**

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**Background:** Antiphospholipid syndrome (APS) is an autoimmune disease characterised by thrombotic events and/or pregnancy morbidity in combination with persistent positivity for antiphospholipid antibodies (aPL) [lupus anticoagulant (LA), anticardiolipin (aCL), anti b2 glycoprotein I (anti-β2GPI) in medium/high titers]. These antibodies could be identified not only in other pathological