Abstract 178 Table 1 Association of laboratory and clinical values (SLAM) with SLE flares

	Correlation coefficient	p	RR	95% CI
ESR	0.34	<0.05	1.56	0.36–0.87
ANA	0.22	>0.05	1.12	0.43–1.50
AntiDNA Ab	0.13	>0.05	0.98	0.67–2,13
Low Hb level	0.48	<0.05	1.99	0.45–0.80
Low leucocytes	0.23	>0.05	1.01	0.56–1.33
Low lymphocytes	0.56	<0.05	2.05	0.33–0.67
Antiphospholipid syndrome	-	-	2.30	0.61–0.88
Pulmonary Involvement	-	-	1.88	0.23–0.82

During a 12 moths fallow–up, 55 flares were enregistered, including 11 cases of severe flares, with a SLEDAI increase from 3 to 17 points. So, the total incidence of flares was 0,53 patient/year and the incidence for severe flares was 0,10 patient/year. In order to assess the risk of flares, we have studied several potential risk factors, as shown in the table.

Conclusions the incidence of flare in a 12 months period was 53,9%, including 10,8% of severe flares. Low Hb and lymphocytopenia are at risk for flares and antiphospholipid syndrome and pulmonary involvement were the main clinical risk factors in our cohort.

179 BIOMARKERS OF ATHEROSCLEROSIS IN SLE IMPROVE AFTER TREATMENT WITH MYCOPHENOLATE MOFETIL

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Background and aims Women with SLE have an increased risk of atherosclerosis that is not adequately explained by traditional risk factors. We previously discovered that a "high risk" score on a panel of biomarkers, *PREDICTS*, confers 28-fold increased odds for carotid plaque in SLE women, and is also associated with IMT progression. The biomarkers included are pro-inflammatory HDL, sTWEAK ≥373 pg/mL, homocysteine ≥12 mmol/L, leptin ≥34 ng/dL, age ≥48 years, and DMII. It is unknown, however, whether these biomarkers are modifiable by SLE disease modifying agents.

Methods This prospective observational study included UCLA cohort patients started on new immunosuppressive agents. Plasma samples were taken at baseline and 12 weeks. HDL antioxidant function was measured by changes in fluorescence intensity of a substrate incubated with LDL and patient HDL. Plasma leptin and sTWEAK were measured using ELISA. Homocysteine was measured in the UCLA clinical labs.

Results 16 subjects started mycophenolate mofetil (MMF), 18 azathioprine (AZA), and 25 hydroxychloroquine (HCQ). In MMF treated subjects, HDL function (p=0.009, paired t-test) and sTWEAK (p=0.05) significantly improved after 12 weeks, while leptin and homocysteine did not significantly change. In HCQ treated subjects, HDL function improved (p=0.05). In the AZA group there were no significant changes in any of the biomarkers. Overall, the mean number of PREDICTS biomarkers at week 12 significantly decreased in the MMF group (p=0.03).

Conclusions The mean number of "high-risk" cardiac biomarkers significantly improved after initiation of MMF. Further longitudinal studies will determine whether changes in biomarkers reflect decreased cardiovascular events.

180 PLASMA MYELOPEROXIDASE IS INVERSELY ASSOCIATED WITH FUTURE ATHEROSCLEROSIS PROGRESSION AND INFLAMMATORY HDL FUNCTION IN SLE

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Background and aims Women with SLE have increased atherosclerosis (ATH) that is not adequately explained by traditional risk factors. We previously discovered that a "high risk" score on a panel of biomarkers, *PREDICTS*, confers 28-fold increased odds for carotid plaque in SLE women. The biomarkers included in PREDICTS are sTWEAK, pro-inflammatory HDL (piHDL), homocysteine, leptin, age ≥48, and DMII. It is unknown, however, whether other biomarkers of oxidative stress also predict progression of ATH in SLE. The enzyme myeloperoxidase (MPO) catalyses formation of reactive oxygen species and generates piHDL. The aim of this study was to determine whether MPO levels might predict future progression of ATH in SLE.

Methods B-mode and Doppler scanning of carotid arteries was performed at baseline and 24–36 months. Baseline plasma MPO levels were measured using ELISA.

Results Repeat carotid ultrasounds and MPO measurements were completed on 202 SLE women. Plaque progression (defined as new or increased plaque) was seen in 42 subjects (21%). Baseline MPO levels were significantly lower in SLE patients with plaque progression vs. those without (p<0.001). Baseline MPO levels were also inversely correlated with piHDL function at follow-up (r=−0.33, p<0.001). Using logistic regression, the variables associated with plaque progression in SLE included high PREDICTS (OR 27.0 p<0.001), MPO levels in the lowest half (OR 4.2, p=0.005), and non-Caucasian ethnicity (OR 4.5, p=0.003).

Conclusions Plasma MPO levels are inversely associated with plaque progression in SLE. Lower baseline MPO levels are also associated with future formation of inflammatory piHDL, suggesting that this could be one mechanism to explain the association.

181 EPIDEMIOLOGIC PROFILE OF ERECTILE DYSFUNCTION IN SLE: A MULTI-CENTRECENTER STUDY IN LATIN AMERICAN PATIENTS

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