During a 12 months follow-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 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.

**179 BIOMARKERS OF ATHEROSCLEROSIS IN SLE IMPROVE AFTER TREATMENT WITH MYCOPHENOlate MOFETIL**

M McMahon*, B Skaggs, J Grossman, L Sahakian, B Hahn. UCLA David Geffen School of Medicine, Rheumatology, Los Angeles, USA

10.1136/lupus-2017-000215.179

**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 associated with IMT progression. The biomarkers included are pro-inflammatory HDL (piHDL), homocysteine, leptin, age ≥48, and DMII. It is unknown, however, whether these 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.

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

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10.1136/lupus-2017-000215.180

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

<table>
<thead>
<tr>
<th>Correlation coefficient</th>
<th>p</th>
<th>RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR</td>
<td>0.34</td>
<td>&lt;0.05</td>
<td>1.56</td>
</tr>
<tr>
<td>ANA</td>
<td>0.22</td>
<td>&gt;0.05</td>
<td>1.12</td>
</tr>
<tr>
<td>AntiDNA Ab</td>
<td>0.13</td>
<td>&gt;0.05</td>
<td>0.98</td>
</tr>
<tr>
<td>Low Hb level</td>
<td>0.48</td>
<td>&gt;0.05</td>
<td>1.99</td>
</tr>
<tr>
<td>Low leucocytes</td>
<td>0.23</td>
<td>&gt;0.05</td>
<td>1.01</td>
</tr>
<tr>
<td>Low lymphocytes</td>
<td>0.56</td>
<td>&gt;0.05</td>
<td>2.05</td>
</tr>
<tr>
<td>Antiphospholipid syndrome</td>
<td>-</td>
<td>-</td>
<td>2.30</td>
</tr>
<tr>
<td>Pulmonary involvement</td>
<td>-</td>
<td>-</td>
<td>2.30</td>
</tr>
</tbody>
</table>

**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.
Background and aims Although SLE has a higher prevalence in women, the disease usually has a more aggressive course in men. Therefore, the aim of this study was to describe the prevalence of erectile dysfunction (ED), as well as associated demographic and clinical features in men with SLE, by means of a systematic, standardised evaluation.

Methods We performed a transversal study in six tertiary care centres in Latin America. We included male patients ≥16 years who fulfilled ≥4 ACR criteria for SLE, and had regular sexual activity in ≤6 months. Patients with other rheumatic diseases (except for APS), chronic viral infections and late-onset SLE were excluded. All patients answered the IIEF-5 Questionnaire, which has been validated in Spanish. Other relevant demographic, clinical and serological characteristics were documented.

Results We included 157 subjects. The prevalence of ED in our study population was 68%, the majority were classified as mild to moderate (17.3±0.36 points; normal score: 22–25).
Background and aims Myositis, especially acute myositis, is a rare manifestation of systemic lupus erythematosus (SLE). Here we report a case of acute myositis concomitant with lupus pleuritis.

Methods a case report and review of literature.

Results A 29-year-old woman with an 8 year history of SLE was admitted to our hospital because of pleuritic chest pain. Her initial diagnosis as SLE was made by malar rash, photosensitivity, oral ulcer, oligoarthritis, leukopenia and the positivity for antinuclear antibodies as well as anti-Sm. She has shown recurrent pleuritis afterwards. The Chest CT revealed bilateral pleural and pericardial effusion. Bacterial cultures and viral antibody tests were negative, and the daily dose of prednisolone was increased from 5 mg to 20 mg. Despite the improvement in the pleuritic chest pain, she developed acute myalgia with the elevated value of serum muscle enzymes, positive signals in the muscle/fascia by the ultrasound and the myopathic changes in the electromyogram examination. After the administration of intravenous steroid pulse therapy for 3 days followed by prednisolone 40 mg/day, all the myositis signs and symptoms subsided, which was also confirmed by the ultrasound.

Conclusions The present case suggests that acute myositis may develop as a manifestation of SLE exacerbation and the ultrasound evaluation may be useful in the diagnosis and the follow-up of myositis.

Background and aims To study the effect of the metabolic syndrome(MetS) on organ damage and mortality in patients with SLE.

Methods Consecutive patients who fulfilled ≥4 ACR criteria for SLE were assessed for the presence of the MetS in 2010. The MetS was defined by the updated joint consensus criteria, using the Asian criteria for central obesity. Longitudinal data on organ damage, vascular events and mortality were retrieved from our database. The association of the MetS with new organ damage and mortality was studied by logistic regression.

Results 577 SLE patients were studied (93% women; age 41.2 ±13.4 years; SLE duration 9.3±7.2 years). The mean follow-up time of the patients was 66.3±1.8 months. 85 (14.7%) patients qualified the MetS. New organ damage and vascular events developed in 128 (22%) and 23 (4.0%) patients, respectively. Thirty-nine (6.8%) patients died. Patients with MetS, compared to those without, had significantly higher SDI accrual at their last visits (0.70±1.0 vs 0.26±0.6; p<0.001). New vascular events (11% vs 2.8%; p=0.001), all-cause mortality (14% vs 5.5%; p=0.003), death due to vascular complications (7.1% vs 0.2%; p<0.001) were significantly more common in patients with MetS than those without. Logistic regression revealed that the MetS was significantly associated with new damage in the ocular, renal, cardiovascular and endocrine system, adjusted for age, sex, SLE duration and the antiphospholipid antibodies. The presence of the MetS showed a significant increase in vascular mortality after adjustment for the same covariates (OR 30.3 [3.42–268]; p=0.002).

Conclusions The MetS is significantly associated with new organ damage, vascular events and mortality in patients with SLE.