THE CORRELATION BETWEEN PREGNANCY, DISEASE ACTIVITY AND ADVERSE PREGNANCY OUTCOMES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS


Background: Patients with systemic lupus erythematosus (SLE) can present with acute disease flares/exacerbations during pregnancy and postpartum period. These flares can cause adverse pregnancy outcomes (APO).

Methods: 168 pregnancy data involving 136 patients with SLE meeting the ACR criteria were examined. Cumulative clinical, laboratory and serological parameters were described and disease activity and flares were calculated using SLEDAI-2K disease activity index during preconceptional six month period, during all trimesters of pregnancy and during six month period after delivery. Patients with low lupus disease activity scores (LLDAS) during each of these periods were identified. Fetal/neonatal death, premature birth due to preeclampsia, eclampsia or HELLP syndrome, neonates small for gestational age were determined as adverse pregnancy outcomes. Relationship of APO with disease activity was studied and patients with APO were compared to patients without APO.

Results: Mean SLEDAI-2K scores was 13.2± 2.0 (16) during preconceptional six month period, 13.2± 2.6 (16) during conception period, 1.7±3.9 (22) during first trimester, 1.4±2.7 (0-16) during second trimester, 1.5±3.3 (2-20) during third trimester and 3.5±5.4 (0-26) during postpartum six month period. Mean postpartum six month period SLEDAI-2K score was higher compared to the mean pregnancy SLEDAI-2K score (p<0.05). LLDAS was sustained in 79% of all pregnancies. 19% of pregnancies resulted in flares. 42% of these flares were severe and 58% were mild or moderate. 49% of severe flares occurred during the postpartum six month period and this percentage was significantly higher compared to each trimester (p<0.05). Most of the flares during pregnancy and postpartum period had mucocutaneous (37%), renal (35%) and hematological (25%) involvement.

APO was observed in 34% of pregnancies (n=57). APO (+) group was characterized by significantly longer disease duration and higher disease activity in all periods compared to APO (-) group (142±70 vs 170±88 months, p<0.05). In APO (+) group, the proportion of patients with severe disease activity during all pregnancy periods and postpartum period was significantly low (%18 vs SS 35, p<0.05), while the proportion of patients with sustained LLDAS was much higher (%88 vs 70).

Conclusion: Postpartum six-month period appears to have the highest risk for disease flares during SLE pregnancy. Disease activity during pregnancy morbidity, regular follow up of patients during pregnancy and postpartum period by Rheumatology and Gynecology and Obstetrics departments is necessary.

References:

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PHENOTYPIC DIFFERENCES BETWEEN SJO¨GR¨EN’S SYNDROME PATIENTS WITH LOW AND HIGH-GRADE INFLAMMATION BASED ON SALIVARY GLAND FOCUS SCORE

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Background: Patients with primary Sjögren’s Syndrome (SS) is characterized by the presence of lymphocytic infiltration around the ductal epithelium of the salivary and lacrimal glands. The peripherial inflammatory lesions and the enclosed B cell component are responsible for the glandular and extraglandular manifestations of the disease. Previous studies have shown that the severity of inflammation observed within the salivary glands is correlated with the occurrence of extraglandular manifestations. However, in these studies either the number of patients is small or the SS criteria are not well defined. To explore the association between the degree of inflammation within the salivary glands and the phenotype of the disease, large and well characterized cohorts of SS patients is required.

Objectives: To compare the phenotypic features of SS patients with low and high degree of inflammation within the minor salivary glands as reflected by the focus score (FS).

Methods: From a total cohort of 1723 consecutive SS patients who fulfill the 2016 EULAR/ACR criteria and are followed up in 4 clinical centers ([Universities of Pisa, Athens, Harokopio and Ioannina, PAHI]), those who had performed a lip biopsy and the focused score was available were chosen into low grade (FS<3) or high grade (FS≥3). Glandular (dry mouth, dry eyes, parotid gland enlarge-ment) and extra-glandular manifestations (Raynaud’s phenomenon, arthralgia/myalgia, arthritis, palpable purpura, liver involvement, kidney involvement, lung involvement, neurologic involvement, long standing lymphadenopathy and lymphoma) as well as serologic features (ANA, RF, anti-SSA/SSB, anti-La/SBB) were considered. Between the 2 groups, statistical analysis for categorical variables was performed by Fisher exact or chi-square tests and for continuous variables with t test or Mann-Whitney accordingly.

Results: Eight hundred and eight minor salivary gland biopsies were available and evaluated based on focus score at the initial evaluation of SS patients, of whom 753 had low grade (FS<3) and 153 high grade (≥3) inflammation. The median disease duration after SS diagnosis was not statistically significant different for the 2 groups (median:4 years, range:0-36 years). SS patients with high grade inflammation displayed higher prevalence of salivary gland enlargement (SLEDAI-2K scores) (40% vs 25%, p=0.002), long standing lymphadenopathy (22% vs 14%, p=0.02), ANA (97% vs 88%, p<0.0001), anti-La/SBB (52% vs 32%, p<0.0001), RF (61,5% vs 48%, p=0,003), peripheral neuropathy (PN) (5,3% vs 1,5, p=0,01) and of lymphoma (26% vs 8%, p<0,0001, OR=4,142, 95%CI=2,65 to 6,47) compared to those with low grade inflammation.

Conclusion: SS patients with FS ≥3 at the initial evaluation, display higher prevalence of lymphoma as well as higher B cell hyperactivity and certain clinical manifestations (SLE, PNS, lymphadenopathy) that constitute risk factors for lymphoma development.

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COMPARISON OF MUSCULOSKELETAL INFLAMMATION BASED ON SALIVARY GLAND FOCUS SCORE IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS GROUPS: HAND ARTHRITIS AND HAND ARTHRALGIA

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Background: Articular involvement can reach up to 95% within the multisystemic manifestations of SLE. Originally, a non-erosive pattern of articular inflammation was described, but the emergence of more sensitive imaging techniques, such as Magnetic Resonance Imaging (MRI), show synovitis (S), erosions (E), bursitis, tenosynovitis (T), bicipital tendinitis (B), and bone marrow edema (BME) and tenosynovitis (TS) in patients with systemic lupus erythematosus (SLE). Nowadays, a specific validated pattern of articular involvement associated with this disease does not yet exist, although it has begun to be studied (1,2).
Objectives: To assess the extent of structural joint involvement in proximal interphalangeal joints (PIP), hand and wrist; E, BME, S, TS and peritendinitis (PT) by MRI, in patients diagnosed with SLE with hand arthritis or arthralgia.

Methods: All patients with SLE who manifested hand pain and/or swelling in the prior 6 months were consecutively included in the study. They were divided into two groups: arthritis or arthralgia, according to the physical examination by an expert rheumatologist. All patients underwent an MRI with contrast injection on their non-dominant hand. The images obtained were evaluated following RAMRIS criteria extended to PIP and Tenosynovitits score for RA by two expert musculoskeletal radiologists, blind to the groups.

Results: 32 patients were included: arthritis: n = 13, arthralgia: n = 19, with a mean age of 50.91 ± 13.37 years and a disease evolution time of 10.21 ± 8.26 years. The average SLEDAI score 6.30 ± 3.40 for the arthritis groups and 3.79 ± 2.14 for the arthralgia group. The average SLICC score was 0.23 ± 0.42 in the arthritis group and 0.1 ± 0.31 in the arthralgia group. E was found in 7 patients with arthritis (53.84%) (15.38% in PIP, 0% in hand and 53.84% in wrist) and in 13 patients with arthralgia (68.42%) (0% in PIP, 10.52% in hand and 68.42% in wrist). BME was observed in 4 patients with arthritis (30.76%) (769% in PIP, 0% in hand and 30.76% in wrist) and in 5 patients with arthralgia (26.31%) (5.26% in PIP, 10.52% in hand and 15.78% in wrist). S was observed in 12 patients with arthritis (92.30%) (61.58 in PIP, 76.92% in hand and 84.61% in wrist) and in 8 patients with arthralgia (42.10%) (31.57% in PIP, 36.84% in hand and 36.84% in wrist). TS was observed in 6 patients with arthritis (46.15%) (38.46% in flexor tendons and 23.07% in extensor tendons) and in 8 patients with arthralgia (42.10%) (31.57% in flexor tendons and 21.05% in extensor tendons). PT was found in 6 patients with arthritis (23.07%) and in no patient with arthralgia (0%).

Conclusion: MRI allows us to diagnose musculoskeletal involvement in SLE, morphologically similar to rheumatoid arthritis (erosion, bone marrow edema, synovitis and tenosynovitis), which usually are underestimated on plain radiography. This study also shows the important erosive burden of arthritis in SLE, which has not been well characterised yet. In addition, it demonstrates the underestimated physical examination to diagnose active inflammatory damage, such as subclinical synovitis, in patients with inflammatory arthralgias.

References:


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Table 1. Additive effect of other aPLs adjusting for LAC

<table>
<thead>
<tr>
<th>Model</th>
<th>LAC +</th>
<th>aCL-G</th>
<th>aCL-M</th>
<th>aB2GPI-A</th>
<th>aB2GPI-M</th>
<th>aB2GPI-G</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ANY thrombosis</td>
<td>VENOUS thrombosis</td>
<td>ARTERIAL thrombosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>age adjusted RR 95%(CI)</td>
<td>P-value</td>
<td>age adjusted RR 95%(CI)</td>
<td>p-value</td>
<td>age adjusted RR 95%(CI)</td>
<td>p-value</td>
<td></td>
</tr>
<tr>
<td>Model 1: LAC + aCL-G</td>
<td>3.78 (2.08, 6.85)</td>
<td>&lt;0.0001</td>
<td>5.03 (2.25, 11.24)</td>
<td>&lt;0.0001</td>
<td>3.32 (1.43, 7.73)</td>
<td>0.0053</td>
</tr>
<tr>
<td>LAC (-) vs (+)</td>
<td>0.64 (0.22, 1.83)</td>
<td>0.4045</td>
<td>0.48 (0.11, 2.14)</td>
<td>0.3351</td>
<td>1.09 (0.31, 3.89)</td>
<td>0.8962</td>
</tr>
<tr>
<td>Model 2: LAC + aCL-M</td>
<td>3.82 (2.13, 6.87)</td>
<td>&lt;0.0001</td>
<td>4.57 (2.07, 10.98)</td>
<td>0.0002</td>
<td>3.69 (1.61, 8.46)</td>
<td>0.0021</td>
</tr>
<tr>
<td>LAC (-) vs (+)</td>
<td>0.51 (0.16, 1.65)</td>
<td>0.2576</td>
<td>0.68 (0.16, 2.93)</td>
<td>0.6019</td>
<td>0.61 (0.14, 2.64)</td>
<td>0.5075</td>
</tr>
<tr>
<td>Model 3: LAC + aB2GPI-A</td>
<td>3.34 (1.88, 5.96)</td>
<td>&lt;0.0001</td>
<td>4.14 (1.9, 9.00)</td>
<td>0.0003</td>
<td>3.29 (1.47, 7.38)</td>
<td>0.0038</td>
</tr>
<tr>
<td>LAC (-) vs (+)</td>
<td>2.39 (0.56, 9.70)</td>
<td>0.2459</td>
<td>2.44 (0.32, 18.4)</td>
<td>0.3858</td>
<td>2.2 (0.29, 14.41)</td>
<td>0.4415</td>
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<tr>
<td>Model 4: LAC + aB2GPI-M</td>
<td>3.52 (1.94, 6.39)</td>
<td>&lt;0.0001</td>
<td>4.21 (1.86, 9.57)</td>
<td>0.0006</td>
<td>3.48 (1.48, 7.79)</td>
<td>0.0038</td>
</tr>
<tr>
<td>LAC (-) vs (+)</td>
<td>0.91 (0.32, 2.61)</td>
<td>0.8655</td>
<td>1.1 (0.31, 3.88)</td>
<td>0.8849</td>
<td>0.96 (0.22, 4.18)</td>
<td>0.957</td>
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<tr>
<td>Model 5: LAC + aB2GPI-G</td>
<td>3.53 (1.97, 6.31)</td>
<td>&lt;0.0001</td>
<td>4.17 (1.9, 9.14)</td>
<td>0.0004</td>
<td>3.58 (1.58, 8.1)</td>
<td>0.0023</td>
</tr>
<tr>
<td>LAC (-) vs (+)</td>
<td>0.91 (0.49, 1.73)</td>
<td>0.7782</td>
<td>1.17 (0.51, 2.68)</td>
<td>0.7010</td>
<td>0.74 (0.29, 1.9)</td>
<td>0.5285</td>
</tr>
<tr>
<td>Model 6: LAC + aB2GPI-A</td>
<td>3.16 (1.77, 5.65)</td>
<td>&lt;0.0001</td>
<td>3.68 (1.68, 8.08)</td>
<td>0.0012</td>
<td>3.19 (1.41, 7.2)</td>
<td>0.0052</td>
</tr>
<tr>
<td>LAC (-) vs (+)</td>
<td>1.68 (1.01, 2.79)</td>
<td>0.0441</td>
<td>2.01 (1.02, 3.97)</td>
<td>0.0434</td>
<td>1.4 (0.67, 2.91)</td>
<td>0.3738</td>
</tr>
</tbody>
</table>

Conclusion: Our study shows that LAC is still the best predictor of risk of any, arterial and venous thrombosis in SLE. Moreover, aB2GPI IgA positivity appeared to add also a significant risk to any and venous thrombosis. Therefore, the clinical significance of IgA anti-β2GPI deserves further investigation in SLE patients.

References:

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