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POS1132
HYDROXYCHLOROQUINE, WHICH REGULATES IFN, IS HIGHLY EFFECTIVE IN SLE WITH ELEVATED SERUM S100 PROTEIN

Keywords: Systemic lupus erythematosus, Cytokines and chemokines

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Background: Serum S100 protein, a damage-associated molecular pattern factor, has been associated with interferon (IFN) [1]. In a previous study, we found that hydroxychloroquine (HCQ), which regulates IFN activity, modulates serum S100 protein in SLE patients [2].

Objectives: This study determined if the therapeutic effect of HCQ is altered in SLE patients with different serum S100 protein levels.

Methods: This single-center, prospective, exploratory study evaluated SLE patients with different serum S100 protein levels. Patients were divided into three groups, namely, high, intermediate, and low, according to baseline S100A8 levels. P-values were determined using Wilcoxon distributions are represented as medians (interquartile range).

Results: Sixty-seven patients (63 females, mean age 41.8 years) were divided into three groups, namely, high, intermediate, and low, according to baseline S100A8 and S100A9 protein levels. The high and low groups were analyzed (Table 1). The S100A8 high group was more likely to have a history of lupus nephritis and active skin lesions. The S100A8 low group had a low serum complement titer (C3) and white blood cell count. The S100A8 high group was more likely to achieve LLDAS after HCQ treatment (high group, 76.9 % vs. low group, 56.3 %). SLEDAI and SLE-DAS improved in the S100A8 high group after 3 months of HCQ treatment. There was no significant improvement in SLE-DAS score. Changes in SLEDAI and SLE-DAS scores before and 3 months after HCQ treatment were analyzed separately by baseline S100A8 levels. P-values were determined using Wilcoxon signed-rank sum test. 0M, 0 months; 3M, 3 months.

Acknowledgements: NIL.
Disclosure of Interests: None Declared.
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POS1133
TREATMENT OF SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS WITH UPADACITINIB RESULTS IN THE COORDINATED INHIBITION OF TYPE I IFN-RELATED BIOMARKERS: BIOMARKER ANALYSIS OF THE M19-130 (SLEEK) PHASE 2 STUDY

Keywords: Cytokines and chemokines, Biomarkers, Systemic lupus erythematosus

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Background: Activation of Type I interferons (IFNs) and a wide array of innate and adaptive immune mediators are hallmarks of the pathogenesis of systemic lupus erythematosus (SLE) and activation of IFN pathways correlates with disease activity [1]. In a phase 2 study of SLE patients, upadacitinib (UPA, Janus kinase inhibitor) given alone or in combination with elosubrutinib (ABBV-599, Bruton’s tyrosine kinase inhibitor) resulted in significant improvement in disease activity as measured by British Isles Lupus Assessment Group-Based Combined Lupus Assessment (BICLA) and SLE Responder Index-4 (SRI-4) at weeks 24 and 48.

The statistical analyses were conducted using the Mann-Whitney U test. Nonparametric distributions are represented as medians (interquartile range).

Changes in SLEDAI and SLE-DAS scores before and 3 months after HCQ administration were analyzed separately by baseline S100A8 levels. P-values were determined using Wilcoxon signed-rank sum test. 0M, 0 months; 3M, 3 months.

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Table 1. Characteristics of SLE patients.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All (N = 67)</th>
<th>S100A8 High (N = 22)</th>
<th>S100A8 Low (N = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female, n (%)</td>
<td>63 (94.0)</td>
<td>22 (100)</td>
<td>21 (95.5)</td>
</tr>
<tr>
<td>Age, years, mean (SD)</td>
<td>41.8 (13.0)</td>
<td>43.5 (10.3)</td>
<td>44.8 (10.5)</td>
</tr>
<tr>
<td>Disease duration, years, mean (SD)</td>
<td>14.8 (11.1)</td>
<td>18.5 (12.5)</td>
<td>16.9 (11.1)</td>
</tr>
<tr>
<td>History of lupus nephritis, n (%)</td>
<td>30 (47.8)</td>
<td>5 (22.7)</td>
<td>25 (59.1)</td>
</tr>
<tr>
<td>Prednisone, mg/d*</td>
<td>57 (78.1)</td>
<td>21 (95.5)</td>
<td>36 (81.8)</td>
</tr>
<tr>
<td>Disease activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cutaneous lesion, n (%)</td>
<td>5 (3.0–73)</td>
<td>5 (4.5–10)</td>
<td>0 (0–4.4)</td>
</tr>
<tr>
<td>Joint lesion, n (%)</td>
<td>39 (58.2)</td>
<td>16 (72.7)</td>
<td>23 (52.3)</td>
</tr>
<tr>
<td>SLEDAI score*</td>
<td>2 (1–4)</td>
<td>2 (1–3)</td>
<td>2 (1–4)</td>
</tr>
<tr>
<td>SLE-DAS score*</td>
<td>4 (2–6)</td>
<td>4 (2–4)</td>
<td>4 (2–6)</td>
</tr>
<tr>
<td>LLDAS, n (%)</td>
<td>18 (26.9)</td>
<td>7 (11.8)</td>
<td>11 (25.6)</td>
</tr>
<tr>
<td>Anti-dsDNA antibodies, IU/ml*</td>
<td>7.4 (5.0–16.3)</td>
<td>5 (5–13.3)</td>
<td>7.8 (5.9–19.5)</td>
</tr>
</tbody>
</table>

The statistical analyses were conducted using the Mann-Whitney U test. Nonparametric distributions are represented as medians (interquartile range).

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