Conclusions NSPA expression in the cell surface of kidney and liver cells and not the P0 provides a potential target for anti-P pathogenic effects, which might contribute to lupus hepatitis and nephritis.

Funding Source(s): Programa de Financiamiento Basal (AFB 17/0005) and FONDECYT Nº 1160513.

Background Systemic lupus erythematosus (SLE) is an autoimmune disorder often characterized by the development of glomerulonephritis. The use of mycophenolate mofetil (MMP) is highlighted as induction and maintenance therapy in lupus nephritis. We evaluated the treatment outcome of MMP in lupus nephritis patients from a real clinical practice.

Methods Patients with biopsy proven lupus nephritis (class III, IV, and V) between November 2005 and August 2017 in Sevance Hospital were extracted, and those patients who were treated with MMP at least 3 months were included in this study. The remission rate of lupus nephritis and risk factors for failure of remission were evaluated using Kaplan-Meier analysis and Cox proportional hazards model.

Results Of 116 patients included in this study, 89 (76.7%) patients achieved remission of lupus nephritis after treatment with MMP. The median time to remission was 4.2 months (interquartile range 0.9 9.1). Normal complement level, negative result of anti-dsDNA antibody, and nephrotic range proteinuria were risk factors for remission failure in univariate analysis (p=0.017, 0.001, and 0.007, respectively). Nephrotic range proteinuria and negative result of anti-dsDNA antibody are independently associated with remission failure in multivariate analysis (OR 3.19, p=0.004 and OR 1.62, p=0.028, respectively).

Conclusions Patients with lupus nephritis showed a favourable clinical outcome after MMP treatment. However, additional therapy would be required in patients with nephrotic-range proteinuria and without anti-dsDNA antibody.

Funding Source(s): None

Abstract 126 Table 1 Lupus nephritis histology class and anti-dsDNA and anti-P presence

<table>
<thead>
<tr>
<th>Lupus Nephritis Class</th>
<th>Anti-dsDNA present and no anti-P</th>
<th>Anti-dsDNA and anti-P present</th>
<th>Anti-dsDNA and anti-P absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISN/RPS</td>
<td>n=26</td>
<td>n=20</td>
<td>n=4</td>
</tr>
<tr>
<td>Proliferative Class III or IV</td>
<td>17</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>Mixed Proliferative Class</td>
<td>7</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>III or IV and Membranous Class</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Abs 127 Hepatic involvement as a presentation in pediatric lupus: A retrospective study of 3 cases

Background Studies looking for clinical association of anti-ribosomal P (anti-P) autoantibodies and lupus nephritis (LN) describe contradictory results. It is clear that anti-dsDNA antibodies contribute to LN pathogenesis and their titers fluctuate together with those of anti-P, suggesting a linked generation or an anti-dsDNA cross-reaction with the P antigen. We reexamined the anti-P involvement in LN in relation with the possibility of anti-dsDNA and anti-P cross-reactivity.

Methods Anti-P and anti-dsDNA were analyzed by ELISA. SLE sera (n=24) from patients with and with no LN were divided into 4 groups: A (anti-dsDNA positive, anti-P negative), B (both positive), C (anti-dsDNA negative, anti-P positive) and D (both negative). Anti-dsDNA cross-reaction was assessed against recombinant wild type and P-epitope-lacking P0 proteins using purified IgGs from SLE patients. Anti-P cross-reaction with dsDNA was analyzed testing affinity purified anti-P antibodies with an anti-dsDNA ELISA. LN biopsies (n=26) were classified according to ISN/RPS and relations to the anti-dsDNA and anti-P simultaneous presence were examined.

Results Neither anti-dsDNA nor anti-P antibodies showed evidence of cross-reaction in any of the applied tests. LN biopsy proven patients: age (media±SD) 34±11 years; 88% female. No LN histologic class with anti-P relationship was observed: 9 patients had Class V (pure n=2) or mixed with Class III/IV (n=7); 1 patient was anti-P positive (p value>0.05). Two patients with LN had neither anti-dsDNA nor anti-P antibodies. The NIH Activity Index (8.9±4.9) and NIH chronicity index scores (1±1.1) and tubule-interstitial lesions were similar between anti-P positive or negative LN patients.

Conclusions Cross-reactivity between anti-dsDNA and anti-P antibodies cannot explain the contradictory results of anti-P association with LN. Based on the present and our previous studies failing to find anti-P association with LN, it seems unlikely that anti-P contribute to the renal damage. Other factors beyond anti-dsDNA and anti-P might participate in LN.

Funding Source(s): FONDECYT grant # 1160513 and CONICYT Basal grant # AFB170005
Abstracts

128 THE LUPUS SEVERITY INDEX IS A PREDICTOR OF DAMAGE AND DEATH IN LUPUS PATIENTS


CHU de Québec – Université Laval; Université Laval; Division of Rheumatology, Cumming School of Medicine, University of Calgary; University of Western Ontario; McMaster University; University of Ottawa; Faculty of Medicine, Department of Internal Medicine, University of Manitoba

Background Predictors of poor outcome in systemic lupus erythematosus (SLE) may lead to the identification of high-risk patients at the onset of disease (incident cases) and/or when we first assess them in our clinics (prevalent cases). We tested whether the Lupus Severity Index (LSI) can help characterize high versus low risk lupus patients.

Methods Population: Patients from six lupus centers were recruited according to a standard data collection protocol. We characterized incident cases and prevalent cases as those with a diagnosis made within or after the previous 15 months. Data collected: Demographic, socioeconomic, disease specific and medication data were collected at baseline and annually. We collected: the American College of Rheumatology (ACR) and the Systemic Lupus International Collaborating Clinics (SLICC) classification criteria, the SLE Disease Activity Index (SLEDAI), the Systemic Lupus Activity Questionnaire (SLAQ), and the SLICC Damage Index (SDI). The LSI was derived from the ACR classification criteria and used as a predictor variable. Statistical analyses: Kruskal-Wallis test and Spearman correlations were used to see the association of LSI with categorical and continuous variables respectively. The baseline LSI was used to predict outcomes at follow-ups using logistic regressions and Spearman correlations for dichotomous and continuous variables respectively.

Results We enrolled 639 lupus patients and 440, 324 and 168 were re-evaluated at 1, 2 and 3 years. Baseline characteristics (table 1) [median (IQR)] were: age=49.0 (36.8–58.5) years, female=92%, Caucasian=74%, disease duration=10.1 (2.7–58.5) years. There were 129 (20%) incident cases and 471 (74%) prevalent cases with missing information in 39 (6%). Twelve patients died during follow-up. To better understand, table 1 summarizes baseline associations between LSI and several characteristics for the incident and prevalent cases. We found that age, sex, ethnicity (Asian worse LSI), SLICC classification criteria, SLEDAI, prednisone use and daily dose were associated with LSI in both incident and prevalent groups while the SDI and the use of immunosuppressors drugs was associated with LSI only in the prevalent cases. In follow-up, baseline LSI predicted SDI in prevalent cases (p=0.02) with a trend in incident cases (p=0.07). LSI predicted death in the prevalent group.

Conclusions The LSI is easy to derive from the ACR classification criteria and a useful measure of severity in lupus. The LSI is associated with baseline characteristics, some of them...