In Group 1, regardless of ethnicity, 29 (90.6%) patients developed LN within 5 years or less from the onset of SLE symptoms, while the remaining 3 (9.4%) developed LN after 5 years. In contrast, in Group 2, 24 (55.8%) patients developed LN within 5 years or less while 19 (44.2%) developed LN after 5 years. (P-value = 0.002).

Further stratification was based on ethnicity and antibody (AB) status to investigate the time to develop LN from SLE symptom onset: African ancestry with positive AB, African with negative AB, Asian with positive AB, Caucasian with positive AB and Asian & Caucasian with negative AB. Analysis showed that of 29 (38.7%) African ancestry patients with the autoantibody combination, 19 (65.5%), developed LN within 5 years. In comparison, 46, (61.3%) patients, independent of ethnicity and AB status, developed LN after 5 years (P-value = 0.01).

Conclusion: Patients with the unusual autoantibody combination of Sm, Ro & RNP developed LN significantly earlier than patients who did not have this combination. This autoantibody combination was significantly over represented in the African ancestry patients with this autoantibody combination are at increased risk of developing LN soon after SLE symptom onset and merit close monitoring for the development of renal disease.

References:

Table 1.1 Ethnicity with Autoantibody status showing the rate of progression into Lupus Nephritis.

<table>
<thead>
<tr>
<th>Duration of LN onset</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 5 years after SLE onset</td>
<td>More than 5 years after SLE onset</td>
</tr>
<tr>
<td><strong>Ethnicity with AB status</strong></td>
<td></td>
</tr>
<tr>
<td>African with positive</td>
<td>19</td>
</tr>
<tr>
<td>African with negative</td>
<td>9</td>
</tr>
<tr>
<td>Asian with positive</td>
<td>5</td>
</tr>
<tr>
<td>Caucasian with positive</td>
<td>5</td>
</tr>
<tr>
<td>Other negatives</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
</tr>
</tbody>
</table>

Graph 1. Ethnicity with Autoantibody status showing the rate of progression into Lupus Nephritis (P-value= 0.01)

Disclosure of Interests: Majed Albirdisi: None declared, David d’cruz Grant/research support from: GlaxoSmithKline, Shrish Sangle: None declared, Natasha John: None declared DOI: 10.1136/annrheumdis-2020-202200.4414

THU0256 CARDIAC INVOLVEMENT IN NEWLY DIAGNOSED SPANISH PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: DATA FROM THE RELIS COHORT

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Background: Cardiac involvement is one of the most important causes of disability and mortality in patients with systemic lupus erythematosus (SLE). Transthoracic echocardiography (TTE) is a sensitive and specific technique in detecting cardiac abnormalities, particularly mild pericarditis, valvular lesions and myocardial dysfunction in SLE.

Objectives: Using data of patients from the inception cohort Registro Español de Lupus Eritematoso Sistémico (RELES), we aimed to analyze the echocardiographic features of cardiac involvement of systemic lupus erythematosus (SLE).

Methods: Prospective observational study on a multicenter Spanish inception cohort. Patients with SLE, diagnosed by the American College of Rheumatology (ACR) criteria, since January 2009, who had at least one TTE performed were selected. Demographic data, diagnostic criteria, follow-ups, treatments and SLE-DAI were analyzed.

Results: We included 289 patients diagnosed with SLE with TTE performed. The mean age was 40.5 ± 19 years, of which 86.9% (251) were women and 82.4% (238) Caucasian. The ACR score at diagnosis was 4.98 ± 0.1. Most frequent SLE manifestations were arthritis (59.2%), photosensitivity (49.5%), malar rash (39.1%) and serositis (31.1%). The main immunological findings were: ANA (97.6%), anti-DNA (66.4%), hypocomplementemia (58.6%), antiphospholipid antibodies (31.4%) and protein C and S deficiency (12.5%). One third (31.5%) of the TTE performed were pathological, of these, 18.3% had pericardial effusion, 13.3% valvulopathy, 6.5% myocardial dysfunction, 5.2% pulmonary hypertension and 3.2% myocardial regurgitation. Regarding valvulopathies, 9.5% presented valvular dysfunction, 3.2% valvular thickening and 0.6% vegetation. The most frequently injured valve was the mitral (91.1%), followed by the aortic (2.8%). The majority of patients (88.26%) were asymptomatic at the time of TTE. However, patients with pathological TTE had more dyspnea than those in the normal TTE group (24.7% vs. 5.8%, p<0.001). Presenting a pathological TTE was associated with higher SLICC score (p<0.001), greater number of admissions (p<0.001) and mortality (p=0.002). A higher SLEDAI was also associated with higher mortality (p<0.001).

Conclusion: Cardiac involvement in SLE is not only related to damage accrual but can also be an early manifestation (beyond pericarditis), especially in active SLE. TTE assessment should be considered as a part of routine examination for SLE due to the high prevalence of heart disease even in asymptomatic patients.

References:


THU0257 ESTIMATED 10-YEARS CARDIOVASCULAR RISK IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS: PRELIMINARY RESULTS FROM THE “CARDIOVASCULAR OBESITY AND RHEUMATIC DISEASE (CORDIS)” STUDY GROUP OF THE ITALIAN SOCIETY OF RHEUMATOLOGY.

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### THU0258

**SOJGREN’S SYNDROME WITH AND WITHOUT AND AUTOIMMUNE THYROIDIS: IS THERE ANY DIFFERENCE?**

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**Background:** Sjögren’s syndrome (SS) is a systemic autoimmune disease mainly affecting exocrine glands and characterized by a progressive lymphocytic infiltration of salivary and lacrimal glands with consequent loss of function and development of sicca symptoms. Autoimmune thyroiditis (AT) is the most frequent autoimmune disease associated with SS and detectable in about 10 - 30% of cases. Interestingly, patients with concomitant SS and AT seem to display a more attenuated phenotype compared to patients with solely SS. It is also noteworthy that up to 30% of patients with AT experience sicca symptoms without a clear diagnosis SS. At the light of these evidences, it is unclear whether SS and AT represent two distinct nosological entities or different expressions of the same pathology.

**Objectives:** Aim of this study is to evaluate the prevalence of AT in a large monocentric cohort of patients with SS and to define its clinical and laboratory characteristics compared to isolated SS.

**Methods:** Consecutive patients with SS (AECG criteria) referring to our Sjögren Clinic (Sapienza University of Rome) were enrolled and divided in two groups: SS with AT (group 1) and SS without AT (group 2). Group 1 was further divided in two subgroups depending on the presence (1a) or absence (1b) of anti Ro/SSA antibodies. The following clinical and laboratory data were retrospectively collected for all patients: concomitant celiac disease, arthralgia, lung involvement, purpura, lymphoma, presence of ANA, anti-Ro/SSA, anti-La/SSB, rheumatoid factor, cryoglobulins, leukopenia and hypergammaglobulinemia. These characteristics were compared between the following groups: group 1, group 2, group 1a and 1b. For statistical Chi Square and Fisher’s test analysis were performed.

**Results:** Six-hundred and three SS patients were enrolled (group 1 n=135; group 2 n=381; group 1a n=96; group 1b n=39). The prevalence of AT was 135/603 (22.3%). When comparing SS patients with or without AT (group 1 vs group 2) the frequency of both celiac disease and rheumatoid factor was significantly higher in group 2 compared to group 1 (p=0.05). No case of lymphoma was recorded in group 1 while 14 cases of lymphoma were ascertained in group 2 (p=0.08). Conversely, celiac disease was higher in group 1 compared to group 2 (p=0.01). No other differences between these groups were identified. Stratifying SS patients with AT according to the presence (group 1a) or not (group 1b) of anti Ro/SSA antibodies, ANA, rheumatoid factor and hypergammaglobulinemia were significantly more positive in group 1a compared to group 1b (OR 0.0002, p=0.002, p=0.02, respectively); no clinical differences were identified.

**Conclusion:** In this study, we confirm the presence of a less aggressive disease in patients with SS and AT compared to solely SS. The higher prevalence of rheumatoid factor and lymphoma occurrence in SS without AT, strictly suggest a more severe phenotype in this subset. Although is known that in SS patients with or without Ro/SSA antibodies and RF there is a more aggressive disease, in SS with AT the presence or absence of such autoantibodies does not seem to associate with any difference in clinical severity. Follow up studies are presently being carried out in order to provide confirmation of a less severe phenotype and a better disease outcome in patients associated SS and AT.

**References:**
