(95%CI: 6.36 -17.04). Most prevalent comorbidities and complications were cardiovascular disorders (62.6%), metabolic disorders (39.6%), polyarthritis (39.0%) and high blood pressure (38.5%). Renal impairment was identified in 34.8% of patients (all renal ICD-10 codes + text strings). Lastly, 31.0% and 15.0% of patients presented with antiphospholipid antibody syndrome or Gougerot-Sjögren syndrome, respectively. Patients received cyclophosphamide (4.8%), immunosuppressants (35.8%), glucocorticoids (51.3%), hydroxychloroquine (47.1%), belimumab (15.0%), and/or rituximab (8.6%). Over the study period, patients were hospitalized on average 10.8 times (median 4.0).

Conclusions A digital platform connected to multidata hospital sources is more time efficient in characterizing complex pathologies like lupus than manual chart reviews. This type of solution also provides a breadth and granularity of information not available in traditional RWE claims data sources. Learning from this single-center study will be deployed to multihospital system in France to test the robustness of the approach.

P30 POLYAUTOIMMUNITY PHENOMENON IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE): SECONDARY SJOGREN SYNDROME (SSS)

Cristiana Sieiro Santos, Clara Moriano Morales, Carolina Álvarez Castro, Elvira Diez Alvarez. *Rheumatology Dept., Complejo Asistencial Universitario de León, León, Spain*

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Background Sjogren's syndrome (SS) is a chronic autoimmune disease characterized by lymphocytic infiltration of the exocrine glands. It can be associated with other connective tissue diseases (CTDs), including systemic lupus erythematosus (SLE).

Objectives To determine the incidence of secondary Sjogren (sSS) in patients diagnosed with systemic lupus erythematosus (SLE-SS) and compare clinical and serological features of SLE-SS to SLE-only patients.

Methods We performed a prospective observational study including patients seen at Rheumatology department diagnosed with systemic lupus erythematosus (SLICC criteria) between 1990–2020. A total of 453 patients diagnosed with SLE were assessed for fulfilment of the criteria for SS using: (European questionnaire and Schirmer test), fluorescein staining test/non-stimulated whole-salivary flow, and anti-Ro/La antibodies and lip biopsy. Anti-Ro/SSA and anti-La/SSB antibodies and RF were measured at entry into the cohort and at SS assessment. SS/SLE was defined according to the American-European Consensus Criteria (AECC). We defined as SLE-SS the case that only fulfilled SLE classification criteria at first and then, during follow-up, the disease progressed and met classification criteria for sSS.

Results SLE-sSS, occurred in 11% of the patients with SLE. In comparison to SLE-non sSS, the SLE-sSS group was older at inclusion, onset and with a longer disease course. Sicca syndrome, oral ulcers, pulmonary involvement, and peripheral neuropathy were more frequent. Anti-SSA, anti-SSB, rheumatoid factor and total IgG were higher in the SLE-sSS group (for all comparisons).

Conclusion SLE-SS appears to be a subgroup of patients with distinct clinical and serologic. The frequency of SLE-sSS increased with age. The subset of patients with SLE-SS has higher frequency of oral ulcers, anti-Ro and anti-La antibodies and a lower frequency of renal disease, anti-dsDNA antibodies, anti-SM and lower hypocomplementemia of C3 and C4.

P31 A NOVEL DISEASE ENDOTYPE IN SLE CHARACTERIZED BY B CELL HYPERACTIVITY, SM/RNP REACTIVITY AND HIGHER DISEASE ACTIVITY

[1]Hugo J van Dooren, [2]Yemil Atisha-Fregoso, [1]Annemarie L Dorjée, Tom WJ Huizinga[1]Tom WJ Huizinga, [2]Meggan Mackay, [2]Cynthia Aranow, [1]Rene EM Toes, [2]Betty Diamond, [1]Jolien Suurmond. *[1]Dept.t of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; Center for autoimmune, musculoskeletal and hematopoietic diseases; [2]The Feinstein Institutes for Medical Research, Manhasset, NY, USA*

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Objective Uncontrolled plasma cell (PC) expansion and hypergammaglobulinemia are hallmarks of B cell hyperactivity in the autoimmune disease Systemic Lupus erythematosus (SLE). While the presence of autoantibodies, in particular anti-nuclear antibodies (ANAs), is another hallmark of the disease, their link to B cell hyperactivity remains unknown. Our goal was to investigate the relationship between PC expansion, hypergammaglobulinemia, and specific autoantibody production.

Methods SLE patients from two cohorts were included as primary analysis cohort (US) and replication cohort (Europe), respectively. B cell subsets were determined by flow cytometry in fresh blood, whereas Ig and autoantibody levels were determined in serum by ELISA. Disease endotypes were calculated based on the frequencies of PC relative to total B cells, CD27 + B cells in healthy individuals.

Results We identified a subgroup of SLE patients with higher frequencies of circulating PC. In particular, this was observed in the frequency relative to memory B cells (high PC/CD27). Importantly, this phenotype was consistent over time in most patients. This disease endotype was characterized by higher levels of total IgG and IgA and a broader anti-nuclear antibody (ANA) response, suggesting that the high PC/CD27 endotype reflects B cell hyperactivity. This subgroup of SLE patients frequently displayed presence of Sm/RNP autoantibodies (29 vs 80%, OR: 9.167 (2.966–26.04)). Furthermore, patients with this disease endotype exhibited a more severe disease course. Interestingly, these characteristics were less prominent or absent in groupings based on total plasma cell percentages or absolute plasma cell counts, suggesting the PC/CD27 frequency in particular represents a distinct disease endotype. Results were validated in the independent replication cohort.

Conclusions Our study presents a novel disease endotype in SLE, characterized by a distinct distribution of the B cell compartment, hypergammaglobulinemia, and distinct serological (auto)antibody characteristics. This patient phenotype therefore signifies B cell hyperactivity and the higher disease activity in this patient group suggests a potential role in the pathogenesis of SLE. These findings may assist in the development of B cell targeted therapies for SLE patients.