patients with surgery, 1 patient with radiotherapy and 5 patients were not treated: 3 patients had to short follow-up or were lost, 1 died, 2 developed a DLBCL, 4 were stable and 7 were in complete remission. The 5 HL were treated with chemotherapy and all were in complete remission.

Conclusions: IBCL and DLBCL were the most common type of lymphomas in SLE patients. Data suggest a role for EBV but not for exposition to immunosuppressant in the pathogenesis of SLE-associated lymphoma. The outcome of lymphoma in the setting of SLE seems not different from the outcome of lymphoma in the general population. A case-control study is ongoing to study the risk factors associated with the occurrence of lymphoma in SLE

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SAT0460

LONG-TERM IMMUNE PROTECTION FOLLOWING PNEUMOCOCCAL 13-VALENT/23-VALENT POLYSACCHARIDE VACCINE IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

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Background: Systemic lupus erythematosus (SLE) patients are at increased risk for *Streptococcus pneumoniae* infection. Although pneumococcal vaccination is an attractive method to prevent invasive pneumococcal infection, vaccination coverage remains dramatically low in SLE. Moreover, the efficacy of vaccination may be reduced in SLE patients and sequential pneumococcal vaccination using new conjugated pneumococcal vaccines in combination with 23-valent pneumococcal polysaccharide vaccine (PPV23) is now advocated.

Objectives: We aimed to determine the efficacy of the prime-and-boost vaccination strategy using the 13-valent pneumococcal conjugate (PCV13) and 23-valent polysaccharide (PPV23) vaccines in SLE.

Methods: Consecutive SLE patients admitted from April to December 2015 in our daycare hospital unit (Paris, France) were enrolled to receive PCV13 vaccine followed by PPV23 vaccine 8 weeks later. Immune protection, defined by an antigen-specific IgG concentration ≥ 1.3 $\mu\text{g/mL}$ for at least 70% of 7 pneumococcal serotypes (4, 6 B, 9 V, 14, 18 C, 19 F, 23 F), was assessed at baseline, 2 and 12 months. The primary endpoint was immune protection 12 months (long-term) after PCV13 shot.

Results: 37 consecutive adult SLE patients admitted in our daycare hospital unit (Department of Internal Medicine, Bichat Hospital, Paris, France) were screened for pneumococcal vaccination. Among them, 8 patients refused to be vaccinated, 7 patients accepted the vaccination but refused to complete the 12 months immune response follow up and 1 patient had already been vaccinated against pneumococcal infection. Eventually, 21 (40²⁵⁻⁷⁵ years; 85.7% female) SLE patients were included in the study and received the sequential PCV13/PPV23 pneumococcal vaccines. Only 12 patients (57.1%) reached the primary endpoint. Nine patients had no long-term protection with a seroconversion that never ($n=4$, not protected, NP) or only transiently ($n=5$, short-term protected, STP) occurred. B-cells defects and immunosuppressive treatment were associated with the NP status. A lower IgG2 serum level and a higher ratio of pre-germinal/post-germinal centre B cells were associated with the STP status. The serotype 19 F IgG titer measured 2 months after PCV13 had the best ability (sensitivity of 100% [95% CI: 47.8–100]; specificity of 91.7% [95% CI: 61.5–99.8]) to predict long-term protection.

Conclusions: The benefit of sequential PCV13/PPV23 vaccination in SLE is limited. Several factors are associated with long-term immune protection and may help to design selective schedule strategy and/or new vaccines.

Disclosure of Interest: None declared

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SAT0461

ULTRASONOGRAPHY OF SALIVARY GLANDS IN PRIMARY SJOGREN'S SYNDROME AND ASSOCIATION WITH DISEASE ACTIVITY, SEROLOGICAL MARKERS AND BIOPSY OF MINOR SALIVARY GLANDS

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Background: Parenchymal inhomogeneity of the salivary glands (SG) is the most relevant ultrasonographic (US) feature for diagnosis primary Sjogren's syndrome (pSS).

Objectives: To analysis parenchymal echostructure of SG in patients with established pSS and association USSG score with disease activity, serological markers and biopsy of minor salivary glands (MSG).

Methods: This study included 205 pSS patients (mean age 53.9 $\pm$ 11.5, disease duration 5.6 years) and 87 healthy controls (mean age 52.3 $\pm$ 14.7). All pSS patients fulfilled the AECG diagnostic criteria. The disease activity was evaluated by EULAR SS disease activity index (ESSDAI), Sjögren's Syndrome Disease Damage Index (SSDDI) and EULAR Sjogren's syndrome patient reported index (ESSPRI). The presence of ANAs assessed by indirect immunofluorescence assay on Hep-2 cells, anti SSA and anti SSB (commercial ELISA kit) and RF (nephelometry). The parotid and submandibular glands on both sides were examined by US using a GE LogiqE9 with a linear high-frequency transducer (6–15 MHz). Inhomogeneity of the SG were graded according to the De Vita scoring system. Grading 0 was for a homogeneous gland, 1 for mild inhomogeneity, 2 for evident inhomogeneity and 3 for a grossly inhomogeneous gland. The global SGUS score (0–6) was the sum of the scores of each pair of SG. Statistical analysis was performed by SPSS v19. Data were compared using *t*-test, χ^2 test and Mann-Whitney U test. The diagnostic accuracy of inhomogeneity was evaluated by area under the receiver operating characteristics curves (AUC-ROC). A multivariate linear regression analysis was performed to determine the factors associated with SGUS score.

Results: Xerophthalmia and xerostomia were presented in 185/205 (90.2%) and 186/205 (91.2%), respectively. According to ESSDAI, the majority of pSS patients 88/205 (43%) had moderate disease activity. Seventy-eight per cent of pSS patients were anti-SSA positive, 44% anti-SSB positive. Biopsy of MSG was positive in 140/172 (81.4%) pSS patients. US abnormalities were established in 197/205 (96%) pSS patients and in 16/85 (18%) controls ($p<0.0001$). The median SGUS was significantly higher in pSS patients in comparison with control group [median (range) 4 (0–6) vs. 0 (0–2), $p<0.0001$]. The diagnostic accuracy of parenchymal inhomogeneity was high, AUC-ROC 0.89 (0.02) with cut-off ≥ 2 (Sp 89.5%, Sn 89.3%). Out of 197 pSS patients with abnormal findings, the most patients had US score 4 (47%), while 81% in control group had score 0. After adjustment for potential confounders variables, dry mouth ($B=1.192$, $\beta=1.542$; $p=0.04$), ESSDAI ($B=0.184$, $\beta=1.203$; $p=0.008$) and biopsy of MSG ($B=1.006$, $\beta=2.735$; $p=0.05$) were significantly associated with advanced US changes of SG. Dry eyes, SSDAI, ESSPRI and serological markers were not associated with USSG score ($p>0.05$).

Conclusions: Our findings confirmed that parenchymal inhomogeneity of the salivary glands is the reliable feature for primary Sjogren's syndrome. Presence of xerostomia, objective disease activity as by ESSDAI and pathological findings of biopsy of minor salivary glands had predictive value for advanced US change of salivary glands.

Disclosure of Interest: None declared

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SAT0462

PD-1+CXCR5-CD4+T CELLS MAY PLAY AN IMPORTANT ROLE IN THE SEVERITY OF SYSTEMIC ERYTHEMATOSUS LUPUS

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Background: CD4⁺ T cells are central mediators in specific autoimmune diseases; however, it remains challenging to define their key effector functions in systemic erythematosus lupus (SLE), a chronic immune-mediated damage to the whole system. Programmed death 1 (PD-1), a negative T cell regulator to maintain peripheral tolerance, induces negative signals to T cells during interaction with its ligands. PD-1⁺CD4⁺T cells could be divided into PD-1⁺CXCR5⁺CD4⁺T peripheral helper cells and PD-1⁺CXCR5⁺CD4⁺ T (Tfh) cells according to CXCR5 expression. PD-1⁺CXCR5⁺CD4⁺T peripheral helper (Tph) cells were proven to promote B cell responses and antibody production in rheumatoid arthritis Rao DA, Nature, 2017.¹ Tfh cells were required for the generation of most memory B cells and long-lived plasma cells in SLE. However, what the role of Tph cells in the pathogenesis of SLE was unknown.

Objectives: We assessed that whether Tph cells were associated with clinical profiles of patients with SLE.

Methods: This cohort study included 36 patients with SLE from the Division of Rheumatology, the first Affiliated Hospital, college of medicine, Zhejiang University. All SLE patients fulfilled the American College of Rheumatology revised classification criteria. 26 Age- and sex-matched healthy individuals had no connective tissue disorders, neoplasms or current infections. Here we used flow cytometry to analyse PD-1⁺CXCR5⁺CD4⁺T cells in peripheral blood from patients with SLE. Correlation between Tph cells and other parameters was investigated by Spearman's correlation coefficient test, and comparisons between groups were performed using nonparametric Mann-Whitney test.

Results: Firstly, we revealed a markedly expanded population of Tph cells (8.95 $\pm$ 6.35 vs. 2.67 $\pm$ 1.22, $p<0.0001$) in the circulation of patients with SLE ($n=36$),