Background: The palatine tonsils are secondary lymphoid-organs that serve as the first line of defense against pathogens. Whether history of tonsillectomy (TE) is associated with the phenotype of Sjögren’s syndrome (SjS) has not been investigated to date.

Objectives: To test whether TE is linked to SjS phenotype and disease activity scores.

Methods: A total of 183 patients from the Optimising Assessment in Sjögren’s Syndrome (OASIS) cohort with SjS or non-SjS sicca syndrome were analysed. Patients with SjS fulfilled 2016 ACR/EULAR classification for primary SjS; sicca patients had objective and/or subjective dryness, but were anti-Ro/SSA negative and had no physician diagnosis of SjS. One SjS patient who had TE around the time of symptom onset was excluded.

Results: Of the total cohort, 116 were diagnosed with SjS (86.2% SSA/Ro positive) and 47 with non-SjS sicca syndrome. Overall, 29% (53/183) had TE: 24.1% (46/189) of SjS patients (28/116) and 37.3% of the sicca patients (25/67). The prevalence of TE was higher in sicca than in SjS (p=0.043). The median age at TE was 8 (range 3-50) years and did not differ between SjS and sicca patients (p=0.629).

Neither age at first symptoms (p=0.093) nor disease duration (p=0.623) were associated with TE in patients with SjS. SjS patients with TE showed a higher average histological focus score (2.1 (1.2-2.8) vs. 1.3 (0.6-4.3); p=0.049), and were more likely to have activity in the glandular (53.6 vs. 20.5%; p=0.001) and constitutional (93.9 vs. 14.9%; p=0.014) domains of the ESSDAI, and lower levels of IgG (12.2 (7.3-35.6) vs. 15.6 (7.5-56.4) g/l; p=0.012) and IgA (2.3 (0.9-6.6) vs. 2.9 (0.7-9.4) g/l; p=0.032). There was no difference in EQSD utility values (p=0.718), VAS global health was significantly lower in the patients with SjS who had TE (58 (10-78) vs. 70 (10-97); p=0.021). There was no association between the status of TE and autoantibodies (SSA, SSB, RF), lachrymal and salivary glands function (Schirmer’s test, unstimulated saliva flow), complement (C3, C4), serum levels of free light chains, IgG, IgA, and IgM of the sicca patients (ESSDAI, all p-values >0.1). Of 181 patients, 12.7% (23/283) had appendectomy (AE); 10.5% (12/114) of the SjS patients and 16.4% (11/67) of the sicca patients (p=0.258). The exception of lower unstimulated saliva flow (0.086 (0.01-0.43) vs. 0.11 (0.01-0.3) ml/min; p=0.026) in SjS patients with AE, there were no differences in disease phenotype between SjS patients with and without AE (all p-values >0.1).

Conclusion: History of TE in SjS is associated with higher average focus scores and with glandular swelling. It could be speculated that the absence of palatine tonsils is compensated by enhanced lymphocytic infiltrates in the salivary glands. Further research is required to determine if TE is a risk factor for both SjS and non-SjS sicca and to determine the role of the tonsils in the generation of hypergammaglobulinemia in SjS.

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SAT0214 ULTRASONOGRAPHIC CHANGES OF SALIVARY GLANDS IN PRIMARY SJOGREN’S SYNDROME: A LONGITUDINAL PROSPECTIVE STUDY

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Background: In the diagnosis of primary Sjögren’s syndrome (SjS), salivary gland ultrasound is useful tool. Until now, there is no data for ultrasonographic changes of major salivary glands over time. The aim of this study was to evaluate the changes in abnormalities of salivary gland ultrasound (SGUS) over time in patients with pSS.

Methods: Patients with pSS (n=70) and idiopathic sicca syndrome (n=18) underwent SGUS twice at baseline and 2 years later. The semi-quantitative SGUS score (0-48) was used, which comprises five parameters: parenchymal echogenicity, homogeneity, hypoechoic areas, hyperechogenic reflections, and clearness of posterior borders. The intraglandular power Doppler signal (PDS) was also assessed. The changes of these SGUS variables were compared in patients with pSS and idiopathic sicca syndrome.

Results: The median (interquartile range) total SGUS scores at baseline was 27 (14) in patients with and 4 (3) in those with idiopathic sicca syndrome (p<0.001). In the pSS group, the total SGUS scores and the SGUS scores for bilateral parotid glands were significantly increased during median 23.4 month follow-up (p=0.013 and p=0.011, respectively). Homogeneity and hypoechoic areas were the domain to show statistically significant progression of SGUS scores. None of the SGUS scores changed significantly in the patients with idiopathic sicca syndrome. In patients with pSS, baseline and follow-up PDS sum scores of four salivary glands were significant higher in worsening SGUS group (n=13) than no change/improvement SGUS group (n=55).

Conclusion: The structural abnormalities in major salivary glands assessed using SGUS scores progressed significantly in patients with pSS. In pSS group, 18.6% patients had worsening SGUS scores during 2 years. Intra-glandular hypervascularity was associated with worsening of salivary gland abnormalities.

References:

SAT0216 HISTORY OF TONSILLECTOMY IS ASSOCIATED WITH GLANDULAR INFLAMMATION IN SJÖGREN’S SYNDROME


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SAT0215 DISEASE SEVERITY, COMORBIDO CONDITIONS, TREATMENT PATTERNS, AND FLARES IN ADULTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS IN THE UNITED KINGDOM: A REAL-WORLD OBSERVATIONAL RETROSPECTIVE COHORT ANALYSIS

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Background: There is limited real-world evidence describing the presentation and treatment patterns of systemic lupus erythematosus (SLE) in the United Kingdom (UK).

Objectives: To characterize disease severity, comorbid conditions, treatment patterns, and flares in a longitudinal cohort of adults with SLE in the UK.
Methods: Patients aged ≥18 years with SLE were identified in the Clinical Practice Research Datalink – Hospital Episode Statistics database from January 1, 2005, to December 31, 2017. Patients were required to have ≥12 months of data before and after index date (date of earliest SLE diagnosis available). SLE disease severity and flares were classified as mild, moderate, or severe using adapted claims-based algorithms that use SLE-related conditions (eg, end-stage renal disease), medications (eg, antimalarials, immunosuppressants, and corticosteroids), and health service use (eg, hospitalizations and emergency department visits).

Results: Of 802 patients with SLE, 369 (46.0%) had mild, 345 (43.0%) had moderate, and 88 (11.0%) had severe SLE at baseline. In total, 692 (86.3%) patients were treated with SLE medications in the first year after SLE diagnosis. Among the total population (802), 557 (69.5%) patients received antimalarials, 203 (25.3%) received immunosuppressants, and 416 (51.9%) received corticosteroids and prednisolone; patients may have received ≥1 type of drug. Information on biologic use in hospitals is unavailable in these data. The mean (standard deviation [SD]) time to initiating any medication from index date was 177 (385.3) days (Figure 1A). The median time to first flare from index date was 63 days (95% confidence interval 57–71) (Figure 1B). A majority of patients (750/802, 93.5%) experienced ≥1 flare during follow-up; the first flare was mild for 73.2% of patients (549/750), moderate for 15.5% (116/750), and severe for 11.3% (85/750). The mean (SD) annual overall flare rate for 15.5% (116/750), and severe for 11.3% (85/750). The mean (SD) annual overall flare rate in the first year after index date was 3.5 (2.5) (mild flares: 2.6 [2.5]; moderate flares: 0.7 [1.5]; severe flares: 0.2 [0.6]) (Figure 2). A shorter median time to first flare was significantly associated with moderate or severe disease (P<0.001) and the presence of comorbid conditions (P<0.001).

Conclusion: Our findings suggest some delay in SLE treatment initiation in the UK. Most patients with SLE experience flares within 2 months from diagnosis. Early treatment may delay or reduce the severity of the first flare after diagnosis and may translate to slower disease progression, lower organ damage accrual, and better outcomes.

References: