ILC1(Lin–IL7R−+KLRG1+ILCs), ILC2(Lin−IL7R−+CRTH2+ILCs) and ILC3(Lin−IL7R−+CRTH2−CD11c+ILCs) [3]. Little research on ILC in the pathogenesis of Primary Sjögren Syndrome (primary SS).

**Objectives:**
Our purpose is to explore the function and role of ILC in the pathogenesis of Primary Sjögren Syndrome and its correlation with clinical markers.

**Methods:**
20 patients with pSS and 15 age-matched healthy non-immune-related diseases controls were enrolled. The frequency of ILCs, B cells, CD4+ T and CD8+ T cells from PBMCs were detected by flow cytometry. Analysis of the subsets of ILCs in each group which compared with B cells and T cell subsets respectively and correlation with clinical serologic markers. Analyze the levels of IL-4, IL-7, IL-33, IL-22 and IFN-γ in each group by ELISA.

**Results:**
Compared with the control group, the percentage of ILC was decreased significantly in primary SS (P<0.0001). Meanwhile ILC1 was significantly increased in primary SS (P<0.0001), ILC2 was decreased significantly in primary SS (P=0.0009) and ILC3 has no significant difference in the primary SS (P=0.05).

The frequency of ILCs in all patients positively correlated with antinuclear antibody titer (ANA) (r=0.295, P=0.0133), moreover, the frequency of ILC2 in primary SS was positively correlated with B cells (r=0.3896; P<0.05), and the serum IgG was negatively correlated with ILC2 of all patients (r=0.2091; P=0.0427). Compared with Healthy control group, the level of IL-22 was significantly higher in primary SS (P<0.0003), however, the levels of IL-4, IL-9, IL-33 and IFN-γ were not significant different with healthy control group (P>0.05).

**Conclusion:** The frequency of ILCs is related to ANA(D) of primary SS patients and ILCs play a critical role in the pathogenesis of primary SS. Its function and mechanism are worth further exploration.
Disclosure of Interests: None declared


AB0051

SEMAPHORIN 3A: A POSSIBLE MARKER FOR DISEASE ACTIVITY IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Sema 3A is concerned in the pathogenesis of many autoimmune diseases because it is involved in regulation of immune responses and maintenance of self-tolerance. Regulatory T cells play an important role in maintaining immunological self-tolerance by suppressing autoreactive T cells. Sema3A promotes regulatory T cells by enhancing IL-10 production

Objectives: The current study aimed at testing the possible role of Semaphorin 3A (Sema 3A) in activity and in remission in rheumatoid arthritis patients and to assess whether this level correlates with interleukin 10 (IL-10) level.

Methods: Sixty Egyptian patients with rheumatoid arthritis (RA) were divided into three groups according to modified Disease Activity Score (DAS28), RA in high activity (group II, n=20), RA in moderate activity (group III, n=20) and RA in remission (group IV, n=20) and compared with 20 normal individuals (group I).

Results: Serum levels of Sema 3A and IL-10 were measured and correlated with ESR, CRP, Rheumatoid factor, DAS28 and Health Assessment Questionnaire (HAQ).

Conclusion: reduced serum level of Sema 3A was found to be correlated with disease activity and indicating its usefulness marker for RA disease activity.

Disclosure of Interests: None declared


AB0052

GENE POLYMORPHISM TNFAIP3 RS6920220 IS ASSOCIATED WITH A SPECIFIC CYTOKINE PATTERN IN PATIENTS WITH EARLY RHEUMATOID ARTHRITIS

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Background: Genetic factors are known to substantially influence the production of cytokines. Objectives: This prospective study aimed to explore whether the polymorphisms of immune response genes were associated with cytokines/chemokine production in patients with early rheumatoid arthritis (RA) and whether a biological therapy affected this association.

Methods: 44 eRA patients (36 females; median age 55.0 [46; 60.0]; median disease duration 7.0 [4.0; 11.0] months; DAS28 5.9 [4.8; 6.4]) were included. 89% were positive for IgM rheumatoid, 92% - anti-CCP positive. "Gene candidate" approach was used to search for the following gene polymorphisms (SNPs): PTPN22 (+1858 C/T, rs2476601), CTLA4 (-308A/G, rs1800629), TNFAIP3 (rs675520, rs6920220, rs10499194), IL6 (+174G/C, rs1800795), IL6R (+358A/C, rs8192284), TNFA (-308A/G, rs1800629), MCP1/CCL2 (+2581A/G, rs1204611), IL10 (-592A/C, rs1800872, -1082 A/G, rs1800896). Serum levels of 27 cytokine/chemokines were measured using the xMAP multiplexing technology at baseline, and at 12 weeks after initiation of therapy. All patients were treated with methotrexate and/or biological therapy in accordance with the treat-to-target strategy (REMAFCA study).

Results: Among all studied SNPs only polymorphism rs6920220 of gene TNFAIP3 was associated with a certain cytokine/chemokine production across all time-points: baseline, 12 and 24 weeks. At baseline, the carriers of GG genotype (27 pts) showed significantly higher serum levels of the following mediators, compared to the patients with GA/AA genotypes (17 pts): IL8 (42.6±23.0 pg/ml vs 25.5±8.8 pg/ml, p=0.001), IL-17 (20.4±15.8 pg/ml vs 11.5±12.1 pg/ml, p=0.017), MIP-1α (19.1±10.5 pg/ml vs 9.8±2.7 pg/ml, p=0.036), MIP-1β (155.4±45.0 pg/ml vs 117.6±47.2 pg/ml, p=0.013), PDGF-BB (6967.5±3275.7 pg/ml vs 4600.6±1826.0 pg/ml, p=0.018). At week 12, IL-6 serum levels were the only ones to remain associated with polymorphism rs6920220 (p=0.015). At week 24, all of GG genotype had higher serum levels of several cytokines/chemokines, compared to the patients with GA/AA genotypes: IL-8 (47.9±26.2 pg/ml vs 33.7±19.3 pg/ml, p=0.05), IP-10 (2451.7±1825.2 pg/ml vs 1404.9±941.1 pg/ml, p=0.037), MIP-1β (150.7±68.2 pg/ml vs 89.7±33.8 pg/ml, p=0.004), PDGF-BB (6150.9±2867.6 pg/ml vs 3915.4±1343.5 pg/ml, p=0.046). There was no statistically significant difference in production of IL-8, IP-10, MIP-1β, and PDGF-BB prior to and after 24 weeks of therapy.

Conclusion: The results suggest that polymorphism rs6920220 of gene TNFAIP3 is linked to a particular cytokine/chemokine pattern. Biological therapy used according to the treat-to-target strategy for 24 weeks does not lead to the reduced production of IL-8, IP-10, MIP-1β, and PDGF-BB in the carriers of GG genotype.

Disclosure of Interests: None declared