Results: Increased frequencies of ILC2 and ILC3 were observed in patients compared to controls, while decreased frequency of ILC1 was found in patients compared with controls (p=0.008, p=0.004, and p=0.006, respectively). We also found the expression of T cell surface markers, CD4 or CD8, on ILCs and their subsets. The results showed that decreased frequencies of $CD4^+CD8^+$ ILCs, $CD4^+CD8^-$ILC1, $CD4^+CD8^+$ ILC2, $CD4^+CD8^-$ ILC2, and $CD4^+CD8^-CD336^-$ ILC3 were found in patients compared with healthy controls (p=0.001, p=0.017, p=0.001, p=0.004, and p=0.002, respectively). Furthermore, frequencies of $CD4^+CD8^+$ ILCs and $CD4^+CD8^+$ ILC2 were positively correlated with the SLEDAI-2000 score (r=−0.548, p=0.005 and r=−0.613, p=0.001, respectively). Frequencies of $CD4^-CD8^+$ ILCs and $CD4^-CD8^+$ ILC1 were positively related with serum C3 level (r=0.519, p=0.008 and r=0.528, p=0.007, respectively), and were positively related with serum C4 level (r=0.623, p=0.001 and r=0.643, p=0.001, respectively).
Conclusions: In the present study, we demonstrated that frequencies of circulating ILCs and its subsets were altered in SLE patients and some subpopulations were negatively correlated with SLE disease activity.

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Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2018-eular.5058

FRI0299 MUCOSAL-ASSOCIATED INVARIANT T CELL DEFICIENCY IN SYSTEMIC LUPUS ERYTHEMATOSUS IS REALTED TO AN INTRINSIC DEFECT IN THE CA2+/ CALCINEURIN/NFAT1 SIGNALLINGPATHWAY

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Background: Mucosal-associated invariant T (MAIT) cells contribute to protection against certain microorganism infections and play an important role in mucosal immunity. However, the role of MAIT cells remains enigmatic in autoimmune diseases.
Objectives: Here, we examined the level and function of MAIT cells in patients with rheumatic diseases.
Methods: Patients with systemic lupus erythematosus (SLE; n=54), rheumatoid arthritis (RA; n=66), Behçet's disease (n=9), ankylosing spondylitis (n=21), and healthy controls (n=136) were enrolled in the study. MAIT cell, cytokine and programmed death-1 (PD-1) levels were measured by flow cytometry.
Results: Circulating MAIT cell levels were significantly reduced in systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) patients. In particular, this MAIT cell deficiency was more prominent in CD8 +and double-negative T cell subsets, and significantly correlated with disease activity, such as SLE disease activity index (SLEDAI) and 28-joint disease activity score (DAS28). Interestingly, MAIT cell frequency was significantly correlated with natural killer T (NKT) cell frequency in SLE patients. IFN-gamma in MAIT cells was impaired in SLE patients, which was due to an intrinsic defect in the Ca2+/calcineurin/NFAT1 signalling pathway. In SLE patients, MAIT cells were poorly activated by alphagalactosylceramide-stimulated NKT cells, thereby showing the dysfunction between MAIT cells and NKT cells. Notably, an elevated expression of PD-1 in MAIT cells and NKT cells was associated with SLE. In RA patients, MAIT cell levels were significantly higher in synovial fluid than in peripheral blood.
Conclusions: Our study primarily demonstrates that MAIT cells are numerically and functionally deficient in SLE. In addition, we report a novel finding that this MAIT cell deficiency is associated with NKT cell deficiency and elevated PD-1 expression. These abnormalities possibly contribute to dysregulated mucosal immunity in SLE.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2018-eular.6201

FRI0300 POLYMORPHISM OF 5,10-METHYLENETETRAHYDROFOLATE REDUCTASE (C677T) IN PATIENTS WITH ANTIPHOSPHOLIPID SYNDROME, ITS ASSOCIATION WITH CARDIOVASCULAR LESIONS

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Background: Many studies have been conducted to determine the role of genetic polymorphism in the occurrence of cardiovascular diseases. The pathogenetic significance of MTHFR polymorphism is the subject of intensive research, especially its connexion with lesions of the cardiovascular system. The frequency of C677T polymorphism in the 5,10-MTHF gene is poorly known in patients with antiphospholipid syndrome **(**APS), and its relationship with vascular lesions has not been studied yet.
Objectives: The present study aimed to analyse the C677T mutation of the MTHFR gene and its association with endothelial dysfunction and clinical manifestations of cardiovascular lesions in APS.
Methods: We studied 82 patients with APS, including 34 (41.6%) with primary antiphospholipid syndrome (PAPS) and 48 (58.4%) with secondary antiphospholipid syndrome (SAPS). The analysis of the MTHFR C677T mutation was performed by PCR followed by digestion according to Frosst et al. All patients were assessed for the endothelium-dependent vasodilatation of brachial artery (EDVD), the thickness of the intima-media of the common carotid artery (IMT), the presence of atherosclerotic plaque (AP) and clinical manifestations of cardiovascular lesions
Results: There were 10.8% of homozygotes (677-TT), 37.8% of heterozygotes (677-CT) and 51.4% of homozygotes (677-CC) in the control group, and the frequency of T-alleles amounted to 29.7%. The incidence of T-alleles was higher among the patients with APS than in the control group and was 35.4%. The prevalence of of homozygotes (677-TT), heterozygotes (677-CT) and homozygotes (677-CC) was not significantly different between the PAPS and SAPS groups (44,1%, 38,2%, 17,7% and 45,8%, 39,6%, 14,6% respectively p<0,05). The frequency of T-alleles was higher in PAPS group than in SAPS group (36,8% against 34,4%, respectively p<0,05). The analysis of structural and functional vascular lesions in homozygotes (677-CC), homozygotes (677-TT) and heterozygotes (677-CT) did not reveal significant differences in both mean values and the proportion of individuals with IMT thickness (0,86±0,03 mm, 0,88±0,05 mm, 0,90 ±0,03 and 35,3%, 38,5%, 51,7% respectively p<0,05) with decrease of EDVD (7,09±0,49, 6,32±1,0, 6,92±0,58 and 47,0%, 53,8%, 48,3% respectively p<0,05) and the presence of AP (26,5%, 23,1%, 48,3% respectively p<0,05). Although there was a tendency of IMT thickness increase and EDVD decrease for T-carriers. The proportion of persons with IMT thickness (>0.90 mm) and the decrease of EDVD BA (≤8.0%) among the homozygote 677-TT was 3%–6.5% higher than that of the 677-CC homozygote. The frequency of clinical manifestations of cardiovascular lesions (myocardial infarction, stroke, TIA) was in 1,2–1,8 times more often among the homozygotes 677-TT than 677-CC homozygote.
Conclusions: The mutation of the C677T of the MTHFR gene is not a risk factor for the development of atherosclerotic vascular damage in patients with APS, due to the lack of associative interrelationships between the decrease of EDVD, increase of IMT, clinical manifestations on the one hand, and the MTHFR polymorphism on the other
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2018-eular.5232

FRI0301 SERUM EXOSOMES INVOLVED IN THE PROGRESSION OF LUPUS NEPHRITIS

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Background: Systemic lupus erythematosus (SLE is a chronic autoimmune disease that systemically affects several important organs[.[1] Lupus nephritis (LN is one of the most severe complications of SLE[.[2] Exosomes are important mediators of biological information and play a part in the occurrence and development of various diseases including LN[.[3]
Objectives: The aim of study was to find whether exosomes participate in the pathogenesis of lupus nephritis.