Background: The symptom burden of typical rheumatoid arthritis (RA) symptoms are associated with pain, fatigue, and health-related quality of life. The symptom burden of typical RA patients has been thoroughly investigated. However, the role of RA in systemic lupus erythematosus (SLE) has been scarcely studied.

Objectives: We explored whether the response of specific biomarkers after treatment-initiation could increase the understanding of SDFR-development in ACPA-negative RA.

Methods: Twelve biomarkers (SAA/CRP/MMP-1/MMP-3/resistin/leptin/IL-6/TNF-R1/YKL-40/EFG/VEGF/VCAM-1) were measured at diagnosis and 1-2 years after DMARD-initiation and compared between patients achieving and not achieving SDFR. Differences in biomarker levels might reflect distinct underlying biological pathways leading towards disease resolution in this subgroup of ACPA-negative RA-patients. Subsequently, the course of specific biomarkers after treatment-initiation could attain more insight in the understanding of SDFR-development in ACPA-negative RA.

Objectives: We explored whether the response of specific biomarkers after treatment-initiation could increase the understanding of SDFR-development in ACPA-negative RA.

Results: RA-patients that achieved SDFR (n=63) were characterized by a stronger decline in MMP-1, MMP-3, SAA and CRP in the first 12-months after DMARD-start, compared to RA-patients not achieving SDFR. MMP-1 and MMP-3 declined 1.30-times (95%CI; 1.17-1.46) and 2.24-times (95%CI; 1.16-4.35) stronger in SDFR-patients. In SAA and CRP, this decline was 2.12-times (95%CI; 1.08-4.14) and 2.24-times (95%CI; 1.16-4.35) stronger. Baseline levels of MMP-3, SAA and CRP and strongly correlated with decline in 12-months; \( \rho = -0.6991 \) and \( -0.9205 \) respectively \( -0.9008 \). RA-patients with high baseline levels characterized the subgroup with the strongest decline in biomarker levels in the first year after DMARD-initiation.

Conclusion: The subgroup ACPA-negative RA-patients achieving SDFR were characterized by higher baseline levels and a stronger serological response of MMP-3, SAA and CRP after DMARD-initiation. Because MMP-3 and SAA are expressed by fibroblast-like synoviocytes, these cells may differ in RA-patients achieving disease resolution; molecular studies on synovial tissue could be a next step.

Disclosure of Interests: None declared

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POS1443

RELATIONSHIP BETWEEN FIBRINOGEN TO ALBUMIN RATIO AND DISEASE ACTIVITY IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Systemic lupus erythematosus (SLE) is a systemic inflammatory autoimmune disease characterized by excessive production of immune complexes and proinflammatory cytokine. Low complement, neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR) have been used as inflammatory biomarkers to assess the disease activity of SLE. Recently, the fibrinogen to albumin ratio (FAR) has emerged as an effective indicator to reflect the systemic inflammation in many diseases. However, the role of FAR in SLE has been scarcely studied.

Objectives: This study was to investigate the association between FAR and SLE Disease Activity Index 2000 (SLEDAI-2K) in SLE.

Methods: We enrolled 140 SLE patients who were divided into four groups (disease activity group: high: DMARD, moderate: DMARD, low: DMARD, inactive: DMARD; and disease activity group: high: DMARD, moderate: DMARD, low: DMARD, inactive: DMARD) according to their disease activity. The associations between the FAR and disease activity were examined using multiple linear regression analysis. The cutoff values of FAR were determined by the receiver operating characteristic (ROC) curve.

Results: The FAR was significantly higher in the disease activity group compared to the inactive disease activity group (95%CI: 1.55-2.83; \( p < 0.001 \)); cutoff values: FAR = 0.79 for high disease activity and 0.61 for low disease activity. The FAR was also significantly associated with disease activity (95%CI: 1.08-4.14; \( p = 0.002 \)).

Conclusion: The fibrinogen to albumin ratio (FAR) is a useful biomarker for assessing disease activity in SLE.

Disclosure of Interests: None declared

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POS1442

THE SUBGROUP OF ACPA-NEGATIVE RHEUMATOID ARTHRITIS PATIENTS ACHIEVING SUSTAINED DMARD-FREE REMISSION IS CHARACTERIZED BY A DIFFERENT RESPONSE IN SEROLOGICAL MARKERS WITHIN THE FIRST YEAR OF DMARD-INITIATION

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Background: Whilst ~40% of ACPA-negative RA-patients achieve sustained DMARD-free remission (SDFR; the sustained absence of synovitis after DMARD-cessation), the biological pathways involved in SDFR-development are incompletely understood. Previously, high baseline levels of MMP-3, SAA and CRP were found in ACPA-negative RA-patients achieving SDFR. Differences in biomarker levels might reflect distinct underlying biological pathways leading towards disease resolution in this subgroup of ACPA-negative RA-patients. Subsequently, the course of specific biomarkers after treatment-initiation could attain more insight in the understanding of SDFR-development in ACPA-negative RA.

Objectives: We explored whether the response of specific biomarkers after treatment-initiation could increase the understanding of SDFR-development in ACPA-negative RA.

Methods: One-hundred-thirty-one consecutive ACPA-negative RA-patients were studied on SDFR-development during (median) 7.2-years follow-up.

Twelve biomarkers (SAA/CRP/MMP-1/MMP-3/resistin/leptin/IL-6/TNF-R1/YKL-40/EFG/VEGF/VCAM-1) were measured at diagnosis and 1-2 years after DMARD-initiation and compared between patients achieving and not achieving SDFR. Differences in biomarker levels might reflect distinct underlying biological pathways leading towards disease resolution in this subgroup of ACPA-negative RA-patients. Subsequently, the course of specific biomarkers after treatment-initiation could attain more insight in the understanding of SDFR-development in ACPA-negative RA.

Results: RA-patients that achieved SDFR (n=63) were characterized by a stronger decline in MMP-1, MMP-3, SAA and CRP in the first 12-months after DMARD-start, compared to RA-patients not achieving SDFR. MMP-1 and MMP-3 declined 1.30-times (95%CI; 1.17-1.46) and 2.24-times (95%CI; 1.16-4.35) stronger in SDFR-patients. In SAA and CRP, this decline was 2.12-times (95%CI; 1.08-4.14) and 2.24-times (95%CI; 1.16-4.35) stronger. Baseline levels of MMP-3, SAA and CRP and strongly correlated with decline in 12-months; \( \rho = -0.6991 \) and \( -0.9205 \) respectively \( -0.9008 \). RA-patients with high baseline levels characterized the subgroup with the strongest decline in biomarker levels in the first year after DMARD-initiation.

Conclusion: The subgroup ACPA-negative RA-patients achieving SDFR were characterized by higher baseline levels and a stronger serological response of MMP-3, SAA and CRP after DMARD-initiation. Because MMP-3 and SAA are expressed by fibroblast-like synoviocytes, these cells may differ in RA-patients achieving disease resolution; molecular studies on synovial tissue could be a next step.

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