<0.05, q = 0.02), supporting the idea that they should be involved in the pathophysiology of the disease.

Conclusion: These preliminary data confirm that the innate immune cells could play an important role in AxSpA. MAIT cells are at the forefront of the expression of IL-17A before γδ T, CD4+T and CD8+T. Neutrophils do not appear to participate in the production of IL-17A, but the high expression of AS linked genes in these cells suggests their involvement in AxSpA.

REFERENCES:

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HIGH-DIMENSIONAL MULTIPARAMETRIC CHARACTERIZATION OF THE REGULATORY T CELLS LANDSCAPE IN SPONDYLOARTHRITIS

Davide Simone, India Brough, Lye Chen, Frank Penkava, Anna Ridley, Hui Shi, Hussein Al Mossawi Grant/research support from: UCB, Paul Bowness Grant/ research support from: Merck, QSK, Celgene

FR0363
AUTOANTIBODIES TO THREE NOVEL PEPTIDES IN EARLY AXIAL SPONDYLOARTHRITIS IN TWO INDEPENDENT COHORTS

Dana Quaden1, Patrick Vandormaal1, Piet Geusens1,2,3, Johan Vanhoof2, Kurt de Vlam1,5, Veerle Somers1. 1Hasselt University, Biomedical Research Institute, Diepenbeek, Belgium; 2ReumaClinic, Genk, Belgium; 3Maasstricht University Medical Center, Internal Medicine, Rheumatology, Maastricht, Netherlands; 4University Hospitals Leuven, Division of Rheumatology, Leuven, Belgium; 5University Hospitals Leuven, Skeletal Biology and Engineering Research Center, Department of Development and Regeneration, Leuven, Belgium

Background: Diagnosis of axial spondyloarthritis (axSpA) is challenging since clinical manifestations, such as inflammatory back pain, peripheral arthritis, enthesitis and inflammatory bowel disease, often overlap with other disorders. Current laboratory markers for axSpA, Human Leukocyte Antigen (HLA)-B27 and C-reactive protein (CRP) are not sufficiently specific for diagnosis. Despite being considered a “seronegative” disease, emerging evidence supports the involvement of antibodies in axSpA. In order to identify novel autoantibodies in axSpA patients, we recently screened an axSpA cDNA phage display library for reactivity with immunoglobulin G (IgG) antibodies in plasma of early axSpA patients. This resulted in autoantibodies to 9 novel University of Hasselt (UH) axSpA peptide targets, corresponding to fragments of known proteins and novel linear peptides.

Objectives: The aim of this study was to determine the diagnostic potential of autoantibodies to the 9 novel UH axSpA peptides in axSpA patients and controls from 2 independent cohorts.

Methods: Using enzyme-linked immunosorbent assays (ELISA), presence of autoantibodies to the 9 novel UH axSpA peptides on phage particles was determined in 76 early axSpA patients, 75 chronic low back pain patients (LBP), 60 early rheumatoid arthritis patients (RA) and 94 healthy controls (HC). Antibody reactivity was further validated in 174 patients from the Leuven Spondyloarthritis (Biologics) Cohort ((Bio)SPAR), including 79 early axSpA patients.

Results: Antibody reactivity against at least one of the 9 novel UH axSpA peptides was found in 54% (41/76) of early axSpA patients, 26% (24/94) of HC (p=0.0002), 39% (29/75) of LBP (p=0.0731) and 38% (23/60) of RA patients (p=0.0845) from the UH cohort, as compared to 43% (74/174) of the axSpA patients from the (Bio)SPAR cohort. By combining the three UH axSpA peptides with highest positive likelihood ratios (LR+) into a panel, antibodies against these peptides were found in 14% (22/155) of early axSpA patients from the combined UH and (Bio)SPAR cohorts and in only 5% (4/75) of persons with LBP (p=0.0484), resulting in a specificity of 95%. The LR+ for confirming axSpA using antibodies to these 3 UH axSpA peptides was 2.7, which is the same as for the currently used laboratory marker CRP. Assuming a 5% pretest probability of axSpA in persons with LBP, a combination of the presence of inflammatory back pain (LR+ 3.1) and a positive test result for the laboratory markers HLA-B27 (LR+ 9.0) and CRP (LR+ 2.5) provides a disease (posttest) probability of 79%. When we added a positive test result for the presence of antibodies to the 3 UH axSpA peptides (LR+ 2.7), posttest probability could be increased to 91%.

Conclusion: Antibodies to 3 UH axSpA peptides were significantly more present in early axSpA patients compared to LBP and could provide a novel tool for objective diagnosis of a subset of axSpA patients.

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T REGULATORY CELLS AS BIOMARKER OF DISEASE ACTIVITY AND RESPONSE IN PSORIATIC ARTHRITIS PATIENTS: RESULTS FROM APREMILAST-TREATED COHORT

Fulvia Cecarelli1,2, Ilaria Pacella1, Arianna Forniti1, Fulvia Ceccarelli1,2, Ilenia Pacella1, Arianna Forniti1, Francesca Spinelli3, Enrica Cipriano1, Carlo Perricone1, Simona Truglia1, Francesca Miranda1, Rossana Scrivo1, Cristiano Alessandri1, Vincenzo Barnaba2, Rossana Scrivo1, Cristiano Alessandri1, Vincenzo Barnaba2, Guido Valensin1, Silvia Piconese2, Fabrizio Conti1, 1Sapienza Università di Roma, Sapienza Arthritis Center, Reumatologia, Dipartimento di Medicina Interna e Specialità Mediche, Roma, Italy; 2Sapienza Università di Roma, Sapienza Arthritis Center, Reumatologia, Dipartimento di Medicina Interna e Specialità Mediche, Roma, Italy; 3Sapienza Università di Roma, Cellular and Molecular Immunology Unit, Dipartimento di Medicina Interna e Specialità Mediche, Roma, Italy.

Background: The PDE4-inhibitor apremilast has been recently introduced in the treatment of Psoriatic Arthritis (PsA). It acts by down-regulating intracellular inflammatory mediators synthesis by elevating cAMP levels. Tregs, a subset of FOXP3+ CD T cells, play a key role in preventing immune responses and could exert their suppressive function via cAMP (1). Reduced frequencies of circulating Tregs have been observed in inflammatory disorders, nonetheless very few data are available on PsA patients.

Objectives: We evaluated peripheral Tregs in a cohort of PsA patients treated by apremilast.

Methods: Seventeen PsA patients (M/F 3/14; median age 56.0 years, IQR 20.0; median disease duration 15.0 years, IQR 11.0) with polyarticular subset treated by apremilast, were evaluated at baseline (T0) and after 6 (T1) and 12 weeks (T2). Clinimetric evaluation included DAS28, CDAI, DAPSA, CDAS, SDAS and DAPSA. Moreover, US assessment was performed at wrists, metacarpophalangeal (MCP), and proximal interphalangeal (PIP) joints: synovial effusion/hypertherpy and power Doppler were scored by a semi-quantitative scale (0-3), obtaining a total score (0-198). Treg frequency was assessed in peripheral blood mononuclear cells by flow cytometry, as the percentage of CD127low FOXP3+ in live CD4+ T cells.

Results: At baseline we identified a median%Tregs of 4.6 (IQR 1.1), inversely correlating with DAS28 (r=-0.5, p=0.02), DAPSA (r=-0.7, p=0.003), CDAI (r=-0.6, p=0.01), SDAS (r=0.6, p=0.003) and US inflammatory score (r=0.6, p=0.01) (Figure 1). Treatment with apremilast was able to induce a significant improvement in all activity indices at T1 (DAS28, P=0.009; DAPSA, P=0.01; CDAI, P=0.005; SDAS, P=0.006; US score, P<0.0001) and T2 (DAS28, P=0.001; DAPSA, P=0.007; CDAI, P=0.006; SDAS, P=0.006; US score, P=0.0005). Furthermore, responding patients according with EULAR criteria at T2 showed significantly higher%Treg at baseline in comparison with non-respondper patients (median 4.8, IQR 1.2 versus median 2.9, IQR 1.1; P=0.02).

Conclusion: The results of this study demonstrate that%Treg is a marker of disease activity in PsA patients. Moreover, the baseline value of Tregs could predict the response to apremilast after 12 weeks of treatment.

REFERENCE:

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