REFERENCES:

- [1] Clauw DJ. Fibromyalgia: a clinical review. *JAMA*. 2014;311:1547-55.
- [2] Nascimento, et al. Gender role in pain perception and expression: an integrative review. *BrJP*. 2020; 3: 58-62
- [3] Wolfe F, et al. Fibromyalgia criteria and severity scales for clinical and epidemiological studies: A modification of the ACR preliminary diagnostic criteria for fibromyalgia. *J Rheumatol* 2011;38:1113–22.
- [4] Burckhardt CS, et al. The fibromyalgia impact questionnaire: development and validation. *J Rheumatol* 1991;18:728–33.
- [5] Salaffi F, et al. Diagnosis of fibromyalgia: comparison of 2011/2016 ACR and AAPT criteria and validation of the modified Fibromyalgia Assessment Status. *Rheumatol* 2020; 0:1-8.
- [6] Wolfe F, et al. Fibromyalgia prevalence, somatic symptom reporting, and the dimensionality of polysymptomatic distress: results from a survey of the general population. *Arthritis Care Res*. 2013; 65:777–85

Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.3482

OP0311

THE ROLE OF COMORBID PATHOLOGY IN THE PROGRESSIVE COURSE OF ANCA-ASSOCIATED SYSTEMIC VASCULITIS

A. Chudinov¹, I. Belyaeva², V. Mazurov², O. Inamova¹. ¹Clinical rheumatological hospital #25, rheumatology, Saint-Peterburg, Russian Federation; ²North-West State Medical University named after I.I. Mechnikov, rheumatology, Saint-Peterburg, Russian Federation

Background: ANCA-associated systemic vasculitis (AAV) is characterized by a high incidence of complications and high damage index. Comorbid pathology at the onset can significantly worsen the prognosis AAV. The most significant comorbid conditions in patients with AAV are coronary artery disease, hypertension and dyslipidemia.

Objectives: The aim of this study was to determine the role of comorbid pathology in the progressive course in patients with AAV.

Methods: Patients with granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA) and eosinophilic granulomatosis with polyangiitis (EGPA) were observed during the first 3 years of the disease and included in this study between 2010 and 2018. At the onset of AAV 75% of patients had significant comorbidities (coronary artery disease, hypertension, dyslipidemia, chronic obstructive pulmonary disease, peptic ulcer, diabetes mellitus, autoimmune thyroiditis and others).

Results: In total 209 [165 [79%] female and mean age 51.8 ± 13.2 years) AAV patients (94 GPA; 46 MPA; and 69 EGPA) were included in the analysis. Formation of chronic kidney disease was significantly more frequent in the group of AAV patients with hypertension at the onset, than in patients without hypertension (respectively 37% and 23.6%, p=0.041). Development of thromboembolic complications was significantly more frequent in the group of AAV patients with coronary artery disease at the onset of AAV, than in patients without coronary artery disease (respectively 34% and 14.8%, p=0.034). Dyslipidemia also was risk factor for cardiovascular complications (OR – 3.81, 95% CI (2.43; 8.2) p=0.009). Presence of diabetes mellitus in the AAV onset was risk factor for infectious complications (OR – 1.77, 95% CI (1.14; 3.45) p=0.038).

Conclusion: Our study has shown that comorbid pathology increase risk of serious complications and can significantly worsen the prognosis AAV. Prevention of development of comorbid conditions and control of lipid levels, hypertension levels are necessary to prevent the formation of irreversible organ damage.

Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.4094

Stress, inflammation & autoimmunity

OP0312

METABOLIC REPROGRAMMING IN MEMORY CD4+ T CELLS IS ASSOCIATED WITH REACTIVE OXYGEN INDUCED IMMUNE CELL DYSFUNCTION DURING AGING

Y. Chen^{1,2}, Y. Ye³, H. Wu⁴, P. L. Krauß^{1,2}, P. Löwe^{1,2}, M. Pfeifferberger^{1,2}, L. Ehlers^{1,2}, A. Damerau^{1,2}, P. Hoff¹, F. Buttgerit^{1,2}, T. Gáber^{1,2}. ¹Charité – Universitätsmedizin Berlin, corporate member of Freie Universität Berlin, Humboldt-Universität zu Berlin, and Berlin Institute of Health, Department of Rheumatology and Clinical Immunology, Berlin, Germany; ²German Rheumatism Research Centre (DRFZ) Berlin, a Leibniz Institute, Berlin, Germany; ³Quanzhou First Hospital Affiliated to Fujian Medical University, Department of Gastroenterology, Quanzhou, China; ⁴Julius-Maximilians Universität Würzburg, Institut für Systemimmunologie, Würzburg, Germany

Background: Inflamm-aging is a chronic, sterile, low-grade inflammatory status, characterized by an increase of proinflammatory cytokines which participate in the development of most age-related diseases such as cancer, Alzheimer's disease, type 2 diabetes mellitus, stroke, cardiovascular diseases, and rheumatoid arthritis (RA). As cellular metabolism modulates T cell function, it can be assumed that metabolic changes accompany the differentiation of memory CD4+ T cells into senescent CD4+ T cell and contribute to memory CD4+ T cells dysfunction during aging.

Objectives: Therefore, we hypothesized that metabolic reprogramming in memory CD4+ T cells might represent an essential factor promoting immune cell dysfunction during aging, thereby fuelling to the pathogenesis of age-related diseases including RA

Methods: To this end, we analysed memory CD4+ T cells isolated from PBMCs of young donors (20-32 years) and old donors (52-67 years) by using MACS™ technology. *Ex vivo* memory CD4+ T cells were analysed by Seahorse™ Technology to determine proton efflux rate (PER) as a measure of glycolysis (glycoPER) and oxygen consumption rate (OCR) as a measure of mitochondrial respiration (mitoOCR). Cytokine expression and secretion was measured by flow cytometry and multiplex assay with and without Mitotempo an inhibitor of reactive oxygen species (ROS). Finally, TCR-stimulated memory CD4+ T cell proliferation was determined using CFSE and Ki-67 after 3 days and 4 days by flow cytometry. ROS and mitochondrial activity were analysed after 24 h using DCF-DA and CellROX Deep Red and Mitotracker by flow cytometry.

Results: In a quiescent state, memory CD4+ T cells from elderly individuals demonstrated a decrease in basal glycolysis and compensatory glycolysis, and an increase in the ratio of basal mitochondrial oxygen consumption rate (mitoOCR) to glycolytic proton efflux rate (glycoPER) while their mitochondrial profile was equivalent to that of young donors while the amount of mitochondria was higher with no increase in steady-state ATP level. In this line and in comparison to the younger reference group, memory CD4+ T cells from aged donors presented a greater spare respiratory capacity after TCR-activation and a marked increase in intracellular ROS production. Interestingly, we did not observe an impact of aging on memory CD4+ T cell proliferation as determined by CFSE and Ki-67. Although the capacity of intracellular cytokine expression did not differ between the compared groups, the levels of secreted IFN-γ, IP-10, IL-6, IL-9, and MCAF were significantly higher in the supernatants of memory CD4+ T cells taken from aged donors but were sensitive to ROS inhibition.

Conclusion: These findings suggest that metabolic reprogramming in human memory CD4+ T cells during aging results in an increased expression of proinflammatory cytokines as a result of ROS production and mitochondrial dysfunction. This process may culminate in T cell dysfunction and thus contribute to the pathogenesis of inflamm-aging and the development of age-related diseases such as rheumatoid arthritis (RA).

Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.3937

Hyperinflammation and Covid19

OP0313

PRELIMINARY CRITERIA FOR MACROPHAGE ACTIVATION SYNDROME ASSOCIATED WITH CORONAVIRUS DISEASE-19

S. Amikishiyev¹, M. G. Gunver², M. Bektas¹, S. Aghamuradov¹, B. Ince¹, N. Koca³, E. S. Torun¹, N. Aliyeva¹, S. San¹, C. Cetin¹, B. Ç. Yalçın Dulundu¹, R. Deniz⁴, F. Kemik⁴, B. F. Agargun⁴, U. A. Gulseren⁴, B. Besisik⁴, O. Alkan⁴, C. Bağrıaçık⁴, Y. B. Tor⁴, Y. Catma⁴, G. Durak⁵, S. Mese⁶, A. Agacfidan⁶, M. Kose⁴, M. Erelel⁷, A. A. Çağatay⁸, S. Ş. Yavuz⁸, S. K. Besisik⁹, F. Esen¹⁰, A. Gül¹. ¹Istanbul Faculty of Medicine, Istanbul University, Division of Rheumatology, Istanbul, Turkey; ²Istanbul Faculty of Medicine, Istanbul University, Department of Biostatistics, Istanbul, Turkey; ³Istanbul Faculty of Medicine, Istanbul University, Division of Rheumatology, Istanbul, Turkey; ⁴Istanbul Faculty of Medicine, Istanbul University, Department of Internal Medicine, Istanbul, Turkey; ⁵Istanbul Faculty of Medicine, Istanbul University, Department of Radiology, Istanbul, Turkey; ⁶Istanbul Faculty of Medicine, Istanbul University, Department of Medical Microbiology, Division of Virology and Fundamental Immunology, Istanbul, Turkey; ⁷Istanbul Faculty of Medicine, Istanbul University, Department of Chest Diseases, Istanbul, Turkey; ⁸Istanbul Faculty of Medicine, Istanbul University, Department of Infectious Diseases and Clinical Microbiology, Istanbul, Turkey; ⁹Istanbul Faculty of Medicine, Istanbul University, Division of Hematology and Therapeutic Apheresis Unit, Istanbul, Turkey; ¹⁰Istanbul Faculty of Medicine, Istanbul University, Department of Anesthesiology, Istanbul, Turkey

Background: COVID-19 runs a severe disease associated with acute respiratory distress syndrome in a subset of patients, and a hyperinflammatory response developing in the second week contributes to the worse outcome. Inflammatory