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P055 INTERLEUKIN-7 IN AORTIC ADVENTITIA OF PATIENTS WITH RHEUMATOID ARTHRITIS AND CORONARY ARTERY DISEASE

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Introduction Rheumatoid Arthritis (RA) patients have increased cardiovascular risk (CV) due to accelerated atherosclerosis (ATS), which significantly contributes to excess mortality in RA.¹ The increased CV risk cannot be fully explained by traditional risk factors and systemic chronic inflammation appears to play a crucial role. Interestingly, IL-7, a proinflammatory cytokine involved in RA pathogenesis, appears to play a role also in ATS² but its effect on cardiovascular disease (CVD) in RA has not been studied yet.

Objectives To examine serum IL-7 levels and expression of IL-7, IL-7R, CD3 and CD20 in aortic adventitia of RA and non-RA patients with coronary artery disease (CAD) and to search for relationships between systemic IL-7 levels and expression of vascular markers, cardiovascular risk factors including metabolic and inflammatory parameters.

Methods We examined 19 RA and 20 non-RA patients undergoing coronary artery bypass graft surgery included in the Feiring Heart Biopsy Study. Serum IL-7 levels were measured by chemiluminescence (MSD). Biopsies from the adventitia of thoracic aorta from a subset of patients (12 RA and 14 non-RA) were stained for IL-7, IL-7R, CD3 and CD20 by immunohistochemistry and scored per mm² of tissue.

Results Non-RA patients had lower IL-7 serum levels than RA (3.4±3.3 vs. 6.7±3.5, p<0.05). Independently of RA diagnosis, IL-7 significantly correlated with CRP (rho=0.450, p=0.008), triglycerides (TG, rho=0.566, p=0.005), glucose (rho=0.642, p=0.001) and hypertension (p=0.036). Levels of IL-7 were associated with New York Heart Association class (rho=0.429, p=0.014) and this was stronger in non-RA patients (rho=0.577, p=0.010). No associations were found with smoking or markers of CVD severity (i.e., numbers of arteries with stenosis or previous myocardial infarcts (MI)). The number of IL-7 +and IL-7R+cells/mm² in adventitia were significantly higher in RA (134.2±45.5 and 144 ±49.9 respectively) than non-RA patients (46.9±22.8 and 54.4±20.2, p<0.005) and were associated with serum IL-7 levels (rho=0.551 and rho=0.588, p<0.01). Both IL-7 +and IL-7R+cells were associated with a positive history of MI (p=0.047 and p=0.005) and IL-7R+cells with the number of previous MIs (rho=0.408, p=0.038). Only in RA patients, IL-7R+cells showed a trend for correlation with TG (rho=0.771, p=0.072). IL-7 +and IL-7R+cells correlated with CD3 (rho=0.688, p=0.013 and rho=0.630, p=0.028), but no

correlation was found with CD20. Cholesterol and HDL levels were associated with IL-7 +cells only in non-RA patients (rho=0.729, p=0.04 and rho=0.733, p=0.038).

Conclusions Among patients with CAD, those with RA had higher serum IL-7 and a greater expression of both IL-7 and IL-7R in aortic adventitia. Systemic levels of IL-7 were related to its vascular expression. Thus, the IL-7/IL-7R axis may play a role in the accelerated ATS observed in RA; further studies are needed to elucidate the precise role of IL-7 in CV risk in RA.

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P056 IMPORTANT ROLE OF CD11C+ DENDRITIC CELLS IN INFLAMMATORY ARTHRITIS

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Introduction The main function of dendritic cells (DCs) is to present antigen to T-cells and therefore they play an important role in bridging the innate and the adaptive immune response. However, DCs can be divided in different subsets, which have intrinsic differences that lead to functional specialisation in the generation and maintenance of autoimmunity. Therefore the aim of our study was to investigate the role of CD11c⁺ conventional DCs and monocytes derived DCs in inflammatory arthritis.

Methods We analysed histological sections of K/BxN serum transfer arthritis as well as hTNFtg arthritis for the presence of CD11c⁺ cells by immunohistochemistry. We also performed synovial biopsies and analysed the cellular composition of the inflammatory infiltrate with respect to DCs. We used CD11c-diphtheria toxin receptor (DTR) transgenic mice, which express the human diphtheria-toxin receptor under the CD11c promoter, allowing for specific depletion of CD11c⁺ cells by administration of diphtheria toxin (DT). K/BxN serum transfer arthritis was induced, and mice were given either DT or PBS or in wt and BARF3 deficient mice. In addition CD11c DTR mice were crossed into hTNFtg animals and also received either DT or PBS. The severity of arthritis was determined clinically and histologically.

Results We show that Cd11c⁺ cells are present in significant numbers in the synovia of K/BxN and TNF driven arthritis. Both CD8⁺CD11c⁺ and CD11b⁺CD11c⁺, can be found in synovial tissue. In K/BxN serum transfer arthritis, clinical scores showed that CD11c-DTR transgenic mice that received DT had significantly reduced paw swelling and loss of grip strength compared to PBS treated animals. Histological analysis found reduced inflammation after the depletion of CD11c⁺ cells in K/BxN arthritis. In addition local bone destruction and the number of osteoclasts was significantly reduced. Analysis of K/BxN arthritis in wt mice and BATF3^{-/-} mice, which lack CD8⁺CD11⁺ DCs revealed no difference in arthritis severity between the two groups. In addition to K/BxN arthritis, we found that also in TNF-driven arthritis