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SAT0027

CIRCULATING CD3+CD31+CXCR4+ T CELLS IN RHEUMATOID ARTHRITIS PATIENTS: CORRELATION WITH RETINAL MICROVASCULAR DAMAGE AND POTENTIAL EFFECT OF ABATACEPT THERAPY

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Background: T-cells play a role in pathogenesis of rheumatoid arthritis (RA) and in its cardiovascular (CV) comorbidities [1]. CD3+CD31+CXCR4+ T-cells may be involved in damaged endothelium repair [2]. The percentage of these cells in the peripheral blood was reported to be lower in RA than in healthy controls, as an effect of disease activity rather than of traditional CV risk factors [3]. Abatacept (ABA), a T-cell co-stimulation blocker, is approved for the treatment of RA. In addition to its effect on disease activity, it may have a CV protective action [4].

Objectives: To evaluate CD3+CD31+CXCR4+T-cells in a cohort of RA patients in correlation with disease activity, CV parameters, and the potential effect of ABA therapy.

Methods: Thirty-one RA patients (median[10th-90thpercentile] age=60[40-70] years, baseline C-reactive protein (CRP)-DAS28=4[3-5.4], body mass index (BMI)=21[17.3-28.6] kg/m², rheumatoid factor (RF) positive:55%, anti-citrullinated peptide autoantibodies (ACPA) positive:77%) were enrolled. Thirteen patients were evaluated before and after 6 months of ABA therapy. Among them, in 5 patients without known CV risk factors (history of arterial hypertension, diabetes, hypercholesterolemia, previous CV events and smoking), a morphological evaluation of retinal arterioles was performed by adaptive optics, a validated technique quantifying microvascular damage [5]. The response to treatment was evaluated with the EULAR response criteria. Phenotypic analysis of peripheral blood T lymphocytes was made by flow-cytometry.

Results: At baseline, no correlation was found between relative CD3+CD31+CXCR4+T-cell number and age, BMI, CRP DAS28, RF and ACPA titer. However, a negative correlation was observed with retinal wall thickness (Figure1). After ABA therapy (n=13), no significant differences were found between good-moderate responders (n=10) and non-responders (n=3), but the two groups seemed to show an opposite trend (T0 vs T6; from 23.8[4-45.4] to 11.9[5-38.7] and from 20.4[15.4-32.3] to 27.4[17.8-41] % of CD3+ cells, respectively). All 5 patients without known CV risk, evaluated also with adaptive optics, had a good EULAR response. A trend for reduction of CD3+CD31+CXCR4+T-cells after ABA therapy was also observed (19[7.9-45.6] vs 12.4[4.6-26.1] % of CD3+), as well as of the retinal wall thickness (14.4[12.9-16.2] vs 14.2[11.7-14.9] μm). A significant reduction of retinal wall cross-sectional area was observed (5123.3[4027.8-6145.5] vs 4852.3[3554.9-5788.2] μm²; p=0.04).

Conclusion: In a subgroup of patients without known CV risk factors, CD3+CD31+CXCR4+T-cell number was inversely related to the possible presence of subclinical CV involvement, as evaluated by retinal microvascular damage. This improved after ABA therapy. These findings may suggest a possible value of CD3+CD31+CXCR4+T-cell number in the

evaluation of microvascular damage, and a possible beneficial effect of ABA on the microcirculation in RA patients.

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Figure 1.

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SAT0028

PERIPHERAL BLOOD REGULATORY T-LYMPHOCYTES OF PATIENTS WITH RHEUMATOID ARTHRITIS BEFORE AND AFTER ALFACALCIDOL THERAPY

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Background: The identification of regulatory T cells (Treg) changed the Th1/Th2 dichotomy response in the pathogenesis of autoimmune diseases. Treg suppresses various autoreactive responses and maintain auto-tolerance in the immune system. The vitamin D receptor is widely expressed in immune cells, including monocytes, macrophages, dendritic cells, NK cells, and T and B lymphocytes, and plays an important role in the regulation of the immune system, and in the modulation of immunologically mediated diseases. Alfacalcidol, a synthetic analogue of hormone D, used in the prevention and treatment of osteoporosis, but the last results indicate its potential therapeutic effects in autoimmune diseases.

Objectives: Assess the presence of regulatory T cells in patients with active rheumatoid arthritis (RA) patients before and after 12 weeks of 2 mcg of alfacalcidol administration daily, and in healthy controls.

Methods: The study included 16 patients with RA, both sexes, with active disease (DAS 28 (SE) > 3.2, despite therapy with LMTBs for at least a month and 20 healthy volunteers (the same distribution of sex and age) - a control group of healthy subjects. In addition to their regular LMTB therapy, patients with RA received daily alfacalcidol (2mcg/day) for 12 weeks. Phenotypic characterization of peripheral blood lymphocytes was performed by direct and indirect immunofluorescence technique using the BD FACS Aria III flow cytometer. Descriptive and analytical statistics were used in data analysis.

Results: A small percentage of activated Treg cells (HLA-DR + compared to total T-reg lymphocytes) in the peripheral blood of patients with active RA was detected in the Treg lymphocytes in relation to the control group of healthy subjects, which is close to the statistical significance level (4.76% versus 6.5% = 0.07). In contrast, the percentage of total Treg cells (Treg versus total CD4 +) was slightly higher in patients compared