addition, monocyte subset counts were determined, based on CD14 and CD16 expression. To obtain absolute counts, proportions determined by flow cytometry were corrected by the total CD4+ T-cell and monocyte counts. Th1, Th17 and monocyte subsets were determined in 21 GCA patients, 19 PMR patients and 19 healthy controls (HC). Th2 cells were determined in 10 GCA patients, 10 PMR patients and 10 HC. All GCA and PMR patients were newly-diagnosed and treatment-naïve. HC were age- and sex-matched and without any immunomodulatory medication.

Results: Both absolute counts and percentages of peripheral Th1 cells, Th17 cells and Th2 cells did not differ between GCA/PMR patients and HC. The monocyte subset in GCA and PMR was mainly attributed to an expansion of the classical monocyte subset. Counts of monocyte subsets were not strongly correlated with counts of either Th1 or Th17 cell counts. In GCA patients, the ESR correlated positively with counts of intermediate monocytes ($R = 0.63$), but this was not observed in PMR patients.

Conclusion: Compared to most previous work, we report similar circulating Th1 and Th17 cell counts in HC, but lower counts in treatment-naïve GCA patients than previously reported (Table 1). Furthermore, numbers of Th1 and Th17 cells in peripheral blood showed no relationship with monocyte subsets. As our protocol for defining Th1 and Th17 cells appears to be similar to the other studies, we propose differences in patient selection. Alternatively, Th1 and Th17 skewing should be studied at the site of inflammation, since Th1 and Th17 skewing cytokines are all highly expressed by macrophages at the inflammatory site. This study shows the importance of replicating previous research, as key concepts of disease pathology are derived from data on disturbed Th cell distribution.

Table 1. Observed median percentages of circulating Th1 and Th17 cells in GCA patients and age-matched HC. Shown are outcomes from several previous studies as well as the present study. Deng et al, Terrier et al and Saadoun et al found elevated Th1 cell percentages in GCA, whereas Samson et al found them lower. All four previous studies showed elevated Th17 percentages in GCA.

Reference	Th1%		Th17%	
	GCA	HC	GCA	HC
Deng et al., <i>Circulation</i> , 2010	20	12	2.2	0.4
Terrier et al., <i>Arthritis Rheumatol</i> , 2012	22	12	2.5	0.5
Saadoun et al., <i>Arthritis Rheumatol</i> , 2015	22	17	2.5	0.6
Samson et al., <i>Arthritis Rheumatol</i> , 2012	10	14	0.7	0.3
This study	11	12	0.6	0.5

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AB0047 T-CELL IMMUNOGLOBULIN AND MUCIN DOMAIN-CONTAINING PROTEIN 3 EXPRESSION IN REGULATORY T CELLS OF ANKYLOSING SPONDYLITIS PATIENTS AND THE CORRELATION WITH DISEASE ACTIVITY

Y. Wang¹, X. Huang¹, J. Chen¹, M. Liu¹, W. Deng¹, Z. Huang¹, T. Li¹.

¹Guangdong Second Provincial Central Hospital, Department of Rheumatology and Immunology, Guangzhou, China

Background: Ankylosing spondylitis (AS) is a chronic inflammatory autoimmune disease. Regulatory T cells have been found in peripheral blood of AS patients. However, there is a controversy regarding the relative number and function of regulatory T cells in AS. T-cell immunoglobulin and mucin domain-containing protein 3 (Tim-3) is a negative immune regulator that participates in immune responses and widely expresses on a variety of immune cells. Many studies have shown that Tim-3 participates in tumors, infections, hematological system diseases and autoimmune diseases (such as systemic lupus erythematosus, rheumatoid arthritis, autoimmune hepatitis, etc). While there has not yet clinical trials with large samples to verify whether or not Tim-3 is involved in the incidence of AS and the relationship between the disease activity of AS and the expression of Tim-3 in regulatory T cells.

Objectives: The present study aimed to identify the Tim-3 expression in regulatory T cells in AS patients, and the association between Tim-3 and the disease activity of AS.

Methods: There were 47 patients diagnosed as AS and 51 age- and sex-matched healthy controls (HCs) from Guangdong Second Provincial Central

Hospital enrolled in the study. The clinical information of the AS patients was recorded in detail and the disease activity was calculated. The positive expression rates and medium fluorescence intensity (MFI) of Tim-3 in regulatory T cells were examined by flow cytometry. Statistical approaches were used to analyze the experimental data of patients and controls.

Results: The Treg cells level was significantly decreased in AS patients compared to the healthy controls (5.14 ± 0.27 vs 4.38 ± 0.23 , $P = 0.032$, Fig 1 A). Tim-3 expression in regulatory T cells was lower in AS patients than the HCs (63.29 ± 2.39 vs 52.56 ± 3.49 , $P = 0.013$, Fig1 B). And Tim-3 MFI in regulatory T cells was significantly decreased too (1355.04 ± 171.44 vs 859.19 ± 105.11 , $P = 0.016$, Fig1 C).

The Treg cells ratio negatively correlated with BASDAI ($r = -0.395$, $p = 0.014$) and BASFI ($r = -0.391$, $p = 0.015$) in AS patients. However, the Tim-3 expression in the Treg cells has no correlation with diseases activity in patients.

Figure 1 The Treg cells frequency, Tim-3 expression and MFI in AS patients compared to the healthy controls.

Conclusion: According to the test results, we could confirm that regulatory T cells participate in the progression of AS, and it has negative relationship with disease activity. Tim-3 can express in Treg, but whether Tim-3 can be used as a potential target for AS treatment in future need further verified.

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AB0048

THE EXPRESSION OF T LYMPHOCYTE SUBSETS IN PERIPHERAL BLOOD OF PATIENTS WITH AUTOIMMUNE UVEITIS AND THE SHORT-TERM EFFICACY AND SAFETY EVALUATIONS OF LOW-DOSE IL-2 COMBINED THERAPY

T. Yan¹, J. Li¹. ¹The Second Hospital of Shanxi Medical University, Taiyuan, China

Background: Autoimmune uveitis is a group of inflammatory diseases that affect the uveal tract such as iris, cilia and choroid. In addition to diabetic retinopathy and age-related macular degeneration, uveitis is one of the main causes of blindness in developed countries^[1]. The disease is autoimmune-mediated, and abnormal immune responses are induced by pathogenic antigens such as retinal soluble antigens and retinal interphotoreceptor retinoid-binding protein (IRBP), and autoimmune inflammation is caused by specific cytotoxic effects, immune complex responses and delayed hypersensitivity reactions. It has been found that the disorder of lymphocyte subsets, mainly due to the number and function defects of regulatory T cells (Tregs), may be involved in the development of uveitis. IL-2 is a key cytokine in T cell differentiation. As a new type of immunomodulator, IL-2 has achieved preliminary efficacy in the treatment of systemic lupus erythematosus, ankylosing spondylitis, Sjogren's syndrome and other diseases^[2-4], but there is no clinical evidence of IL-2 in the treatment of autoimmune uveitis. The purpose of this study was to investigate the expression of T lymphocytes in patients with autoimmune uveitis and the effect of low dose IL-2 on their immune status.

Objectives: To investigate the expression of peripheral blood lymphocyte in patients with autoimmune uveitis and evaluate the short-term efficacy and safety of low-dose IL-2 combined with methylprednisolone.

Methods: A total of 108 patients with autoimmune uveitis and 93 healthy subjects who visited our hospital from January 2016 to April 2019 were collected. Twenty-three patients were treated with a low dose of IL-2 (50WU/day for 5 consecutive days) on the basis of conventional treatment