Objectives: To evaluate the changes of peripheral lymphocyte subsets, conventional drugs and remission rate in patients with systemic lupus erythematosus (SLE) after immunomodulatory therapy.
Methods: A total of 89 patients with SLE from the Second Affiliated Hospital of Shanxi Medical University from January 2016 to April 2018 were enrolled, who were divided into well-controlled group and untargeted control group taking a full consideration of the patient's symptoms, signs and related laboratory findings. We measured the absolute counts of B, NK, CD8+T and helper T 1 (Th1), helper T 2 (Th2), helper T 17 (Th17) and Treg cells in peripheral blood of patients before immunomodulatory therapy and during the 3 months and 6 months of follow-up and 93 sex- and age- matched control individuals using flow cytometry. Moreover, the ratios of various cells to Treg cells were calculated.
Results: Compared with healthy controls, Treg cells in SLE patients were significantly lower before the treatment with immunomodulator, while the ratios of various pro-inflammatory lymphocytes to Treg cells (such as Th2/Treg, Th17/Treg, CD8+T/Treg, etc.) were higher. After 3 months and 6 months with immunomodulatory therapy, the absolute number of Treg cells in peripheral blood of SLE patients increased obviously reaching to normal level. Accordingly, the ratios of various pro-inflammatory lymphocytes to Treg cells recovered. At the same time, the dose of glucocorticoid and disease-modifying antirheumatic drugs (DMARDs) decreased distinctly. Additionally, the well-controlled group was able to maintain a high remission rate, and the untargeted control group could achieve a higher response rate after immunomodulatory treatment.
Conclusion: The imbalance between pro-inflammatory lymphocytes and Treg cells caused by the significant decrease of Treg cells may be the main cause of SLE. And immunomodulatory therapy we came up with may reverse the imbalance of proinflammatory lymphocytes and Treg cells, which is an potential and effective treatment for SLE.

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AB0471 **VITAMIN D SUPPLEMENTATION IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS WITH VITAMIN D DEFICIENCY AND INSUFFICIENCY: THE EFFECT ON DISEASE ACTIVITY, FATIGUE AND INTERFERON SIGNATURE GENE EXPRESSION**

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Background: Vitamin D deficiency is highly prevalent among patients with systemic lupus erythematosus (SLE) [1]. Evidence from multiple studies has shown that vitamin D deficiency in SLE is associated with a higher disease activity [2]. There is conflicting evidence with regards to the relationship between fatigue and vitamin D level [3,4].
Objectives: The principal aim of this study was to establish any potential effect on the level of fatigue, disease activity (measured by SLE disease activity index-2K (SLEDAI-2K)) and interferon signature gene expression, from vitamin D supplementation to SLE patients with vitamin D deficiency or insufficiency.
Methods: 33 SLE patients, 13 with vitamin D deficiency and 20 with vitamin D insufficiency, gave informed consent to participate in this 12 month prospective study. Their participation consisted of an interview, filling of the Fatigue Severity Scale (FSS), and blood tests. The patients were advised to take vitamin D3 8000IU daily for 8 weeks if they were vitamin D deficient, or 8000IU daily for 4 weeks if they were insufficient. This was followed by 2000IU daily maintenance. The patients were re-assessed after 6 and 12 months of vitamin D supplementation. RNA was extracted from whole blood taken from the patients at baseline and after 6 months of vitamin D supplementation. The expression of 12 interferon signature genes was measured in the extracted RNA by using QuantiGene Plex technology. Approval to carry out this study was obtained from the University Research Ethics Committee.
Results: 87.9% of SLE patients studied were female. The mean age was 47.6 years and the mean duration of SLE was 13.8 years. Table 1 shows the results obtained for several variables at baseline, after 6 months and after 12 months. The expression of all 12 interferon signature genes measured, was noted to decrease following 6 months of vitamin D supplementation. This reached statistical significance for two of the genes measured (OAS1, p=0.014; SOCS1, p=0.003).

Variable	Time	Mean	Standard Deviation	p-value
SLEDAI-2K	Baseline	3.97	3.359	0.210
	6 months	3.36	2.924	0.009
	12 months	2.61	2.193	
Anti-dsDNA level (IU/mL)	Baseline	210.31	234.416	0.032
	6 months	194.08	214.129	0.030
	12 months	190.50	208.131	
Prednisolone daily dose (mg)	Baseline	1.93	3.458	0.043
	6 months	2.30	4.089	0.042
	12 months	1.33	2.931	
FSS	Baseline	4.21	1.536	0.206
	6 months	3.93	1.577	0.053
	12 months	3.81	1.859	
25-hydroxyvitamin D (ng/mL)	Baseline	21.91	6.336	<0.001
	6 months	32.88	6.594	0.001
	12 months	28.48	9.291	

Abstract AB0471 Table 1. Table showing results at baseline, after 6 months and after 12 months of vitamin D3 supplementation.

Conclusion: The results indicate that vitamin D supplementation in SLE patients who are deficient or insufficient, results in an improvement in disease activity, and possibly also in the level of fatigue. This could be explained by the decrease in the expression of interferon signature genes.

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