

Therapeutic effect of NSAID on active inflammatory SI joint lesions in patients with early axial SpA

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Background/Aims: The purpose of the present study was to examine the therapeutic effect of NSAIDs on active inflammatory lesions (bone marrow oedema [BMO]) in the sacroiliac(SI) joint in early axial spondyloarthritis (SpA). The aim was to propose optimal duration of treatment in initially diagnosed axial SpA patients with a good response to NSAIDs. **Methods:** We enrolled 19 patients with axial SpA who were initially diagnosed in our hospital, and prescribed full-dose NSAIDs for 12 weeks. Twelve patients completed the 12-week protocol. We collected demographic, clinical, laboratory, and radiologic data at the time of enrolment and after 6 and 12 weeks of NSAID treatment. Baseline, 6-week, and 12-week data were compared using the Friedman test. **Results:** The total SPondyloArthritis Research Consortium of Canada (SPARCC) score decreased significantly at 6 and 12 weeks (P value for 6 weeks and 12 weeks, 0.001, 0.025, respectively). The SPARCC score was significantly correlated with the ESR and CRP (P value for ESR and CRP, 0.041, 0.001, respectively). Univariate and multivariate regression analyses showed that the body mass index was significantly associated with changes in the SPARCC score. **Conclusions:** Active inflammation in the SIJ was significantly attenuated by 6 weeks of full-dose NSAIDs. The SPARCC score was well-correlated with CRP and ESR. Therefore, at least 6 weeks of full-dose NSAIDs can decrease active SIJ lesions, and initial full-dose NSAIDs may prevent further structural damage as shown by reverting early radiologic change (BMO).

Table 2. Comparison of SPARCC score of SIJ at 6 and 12 weeks

	Baseline	Follow up MRI at week 6	Follow up MRI at week 12	p*	Baseline -6 weeks	Baseline -12 weeks	6 weeks -12 weeks
SPARCC score of BMO (0-48)	10.2 [9.0;15.5]	5.0 [3.8; 6.5]	5.5 [1.8; 9.5]	0.009	0.001	0.017	0.833
SPARCC score of intense edema (0-12)	0.8 [0.0; 2.0]	0.0 [0.0; 0.2]	0.0 [0.0; 0.5]	0.226	0.105	0.144	0.705
SPARCC score of deep edema (0-12)	3.2 [0.5; 5.8]	0.8 [0.0; 1.2]	0.0 [0.0; 1.5]	0.049	0.008	0.116	1.000
Total SPARCC of SIJ (0-72)	16.8 [11.8;22.0]	6.0 [3.8; 8.2]	6.5 [1.8;14.0]	0.012	0.001	0.025	1.000

* P value for Friedman test

BMO: bone marrow oedema, MRI: magnetic resonance imaging, SIJ: sacroiliac joint

Table 4. Correlation between SPARCC score of SIJ and disease activity parameters

		ESR	CRP	BASDAI	ASDAS-CRP	ASDAS-ESR
SPARCC score of the SIJ	Rho	0.368	0.554	-0.211	0.224	0.176
	P	0.041	0.001	0.281	0.252	0.369

Table 5. Univariate and multivariate regression analysis of factors related with changes in SIJ SPARCC scores

Variables	Univariate			Multivariate		
	β	SE	P	β	SE	P
Age	-0.122	0.274	0.665			
HLA-B27 positivity	0.929	5.439	0.868			
BMI	2.490	0.801	0.011	2.168	0.706	0.013
Smoking	-7.271	4.937	0.172			
ESR	0.164	0.151	0.303			
CRP	-0.110	0.278	0.701			
BASDAI	-2.016	1.595	0.235			
ASDAS-CRP	-2.652	3.722	0.493			
ASDAS-ESR	-0.357	3.764	0.926			
BASFI	-1.178	1.059	0.292			
EQVAS	0.150	0.108	0.197			
EQ-5D-5L	32.753	19.368	0.122			
Baseline high SPARCC score (>20)	10.833	5.169	0.063	8.256	3.900	0.063

PD-1 expression on Tfh and Tfr cells is a Disease Activity Marker in Sjögren's Syndrome

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Background/Aims: Sjögren's syndrome (SS) is a chronic, autoimmune disease characterized by lymphocytic infiltration of exocrine glands, resulting in glandular dysfunction and sicca symptoms. We examined follicular helper T (Tfh) and follicular regulatory T (Tfr) cell populations from peripheral blood of patients with SS, and investigated whether specific subsets of Tfh or Tfr cells were associated with disease activity or salivary gland inflammation in SS. **Methods:** Peripheral blood samples were obtained from patients with SS and healthy donors (HDs). Tfh and Tfr cell populations were determined from peripheral blood mononuclear cells by flow cytometry. Activated Tfh and Tfr cells were evaluated by Programmed cell death protein 1 (PD-1) and Inducible T-cell costimulator (ICOS) expression. Serum interleukin (IL)-21 levels were determined by Enzyme-Linked Immunosorbent Assay. Salivary gland inflammation was assessed by presence of focal lymphocytic sialadenitis and the number of foci. **Results:** Twenty-three patients with SS and 11 HDs were enrolled in this study. Tfh and Tfr cells were increased in patients with SS, but did not show statistical significance. The ratio of Tfr to Tfh cells was similar between SS patients and HD. Expression of PD-1 on blood Tfh and Tfr cells were significantly associated with disease activity assessed by EULAR SS Disease Activity Index (ESSDAI) ($r=0.492$, $p=0.019$ and $r=0.451$, $p=0.034$, respectively). However, ICOS expression on blood Tfh and Tfr cells were not associated with ESSDAI ($r=-0.053$, $p=0.818$ and $r=0.147$, $p=0.522$, respectively). PD-1 expression on Tfh and Tfr cells were significantly associated with serum IL-21 levels. CCR7loPD-1hi Tfh and CCR7loPD-1hi Tfr cell subsets seemed to correlate with focus score. **Conclusions:** Blood activated Tfh and Tfr cells, represented by intensity of PD-1 expression, are associated with disease activity of SS. PD-1 may be a therapeutic target in SS.

