

Conclusion: The presence of active RA, both in terms of clinical and ultrasonographic indices, correlates with an increased CSA of the palmar digital nerves. This alteration is probably due to inflammatory mechanisms of the perineural tissues at the level of the MCPj. Active synovitis during RA can somehow be framed as a condition capable of causing neuropathic damage to the palmar digital nerves.

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POS0566

CATASTROPHIZING IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Catastrophizing is conceptualized as a negative cognitive-affective response to an anxiety-provoking stimulus, especially anticipated or actual pain. Catastrophizing can be assessed quickly using a validated questionnaire: the Pain Catastrophizing Scale (PCS)¹. Catastrophizing plays a role in maintaining chronic pain and is associated with several pain-related outcomes in osteoarthritis and low back pain.

Objectives: To assess the prevalence of catastrophizing and associated factors in rheumatoid arthritis (RA).

Methods: We performed an observational, prospective, bi-centric study. All patients aged 18 or over with RA and fulfilling the ACR-EULAR 2010 criteria were consecutively included. Sociodemographic data, information on the disease and its treatments were collected as well as questionnaires for disease activity (DAS28), function (HAQ), quality of life (SF12, EQ5D), anxiety and depression (HADS, GAD7), fibromyalgia (FIRST), insomnia (ISI) and catastrophizing scores (PCS). Statistical analysis included the samples t-test, one-way variance analysis, the Spearman's correlation test, the Chi² test, Fisher's exact test, the Wilcoxon test, multivariate linear regression (considering catastrophizing as a continuous variable) and multivariate logistics regression (considering catastrophizing as a categorical variable: PCS ≥ 20 = high level catastrophizing).

Results: From September 2019 to March 2020, 201 patients with RA were included: 78.1% were women and the median age was 63.0 years. In all, 64.1% of patients were RF+, 65.7% ACPA+, and 46% had erosive disease. Median DAS28 CRP was 2.9 [2.1-4.0], with 45% of patients in remission, 14.8% with low, 31.2% moderate and 9% high activity. The majority of patients (92%) had a disease lasting for more than 2 years.

The prevalence of a PCS score ≥20 was 48.0% [41.0;54.9]. The median PCS score was 18 [7-28]. In multivariate logistics regression, high-level catastrophizing was significantly associated with DAS28-CRP (OR= 1.61 [1.18-2.20]), HADS anxiety score (OR=1.25 [1.11-1.40]) and the HADS depression score (OR=1.19 [1.07-1.33]). In multivariate linear regression, catastrophizing was significantly associated with the HADS anxiety score (p< 0.0001), HADS depression score (p=0.0055), HAQ (p=0.0015) and the ISI insomnia score (p=0.005).

Conclusion: Almost half the patients with RA were high catastrophizers. Catastrophizing is linked to anxiety, depression, disease activity, function impairment and insomnia. It may be interesting to detect catastrophizing in order to improve the management of our patients.

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POS0567

HEPCIDIN IS POTENTIAL BIOMARKER TO DISTINGUISH BETWEEN IRON DEFICIENCY ANEMIA AND ANEMIA OF INFLAMMATION IN RHEUMATOID ARTHRITIS

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Background: Anemia of inflammation (AI) and iron deficiency anemia (IDA) are the two most prevalent forms of anemia in patients with rheumatoid arthritis (RA). Diagnosis becomes challenging if AI is associated with true ID (AI/ID), as there is still a lack of a gold standard for differentiation between AI and AI/ID. However, as therapies to overcome anemia differ, proper diagnosis and understanding of underlying pathophysiological regulations are necessary.

Objectives: The aim of the study was to evaluate the clinical efficiency of hepcidin, a key regulator of iron metabolism, in the diagnosis of IDA, as well as the differential diagnosis of AI/ID and AI in patients with RA.

Methods: The study was undertaken 96 patients with RA, 67 of them were diagnosed anemia according to WHO criteria (104.3±21.4g/l). Anemic patients and anemia-free patients with RA (n=29) were comparable (p>0.05) in age (44.4±14.8 and 49.8±9.3 years), disease duration (73.5±65.4 and 59.8±48.3 months) and DAS28 (6.3±1.6 and 5.9±1.9). All cases were subjected to following tests: complete blood count with peripheral smear, serum C-reactive protein, serum interleukin-6, iron studies, serum soluble transferrin receptor (sTfR), and serum hepcidin. Patients with RA and anemia were divided two groups: 25 patients with IDA and 42 - with AI. The AI cases were subdivided into pure AI and AI with coexistent ID (n=15).

Results: The mean serum hepcidin concentration was significantly increased in pure AI patients (123.85±25.8ng/mL) as compared to those in IDA patients (63.9±22.8ng/mL, P < 0.05) and anemia-free patients with RA (88.1±39.09ng/mL). Also, compared to pure AI patients [normal sTfR levels (<3 µg/mL)], the serum hepcidin concentration was reduced significantly in AI patients with ID [high sTfR levels (≥3 µg/mL)] with a mean of 79.0±23.97ng/mL.

Conclusion: Hepcidin measurement can provide a useful tool for differentiating AI from IDA and also help to identify an iron deficiency in AI patients. This might aid in the appropriate selection of therapy for these patients.

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POS0568

OBESITY RATES AND BMI ARE SIMILAR IN AGE AND SEX MATCHED RHEUMATOID ARTHRITIS, PSORIATIC ARTHRITIS AND SPONDYLOARTHRITIS PATIENTS

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Background: Obesity has been suggested to be a chronic inflammatory condition and therefore, obesity may be considered as a risk factor for developing immune-mediated diseases, including inflammatory arthritis. In larger biologic registries, obesity was found frequently in psoriatic arthritis than other inflammatory diseases such as rheumatoid arthritis and spondyloarthritis. [1-4]. However, obesity may be a reason of aging, moreover, there are strong sex differences between those diseases.

Objectives: The aim of this study was to evaluate the obesity rates in sex and aged matched patients with inflammatory arthritis before the initiation of biological therapy.

Methods: HUR-BIO (Hacettepe University Biologic Registry) is a prospective, single center database of biological treatments since 2005 and to date 5635 patients have been recorded. Demographic, clinical and laboratory data before bDMARD of SpA, RA and PsA patients were noted. The patients were divided into two groups: non-obese patients (<30kg/m²) and obese (≥30kg/m²) patients. When investigating the changes in BMI by diagnosis, the effects of gender and age were adjusted using two-way ANOVA and ANCOVA tests. The selection was made for the gender and age indifferences of the relevant groups by using propensity score.

Results: 5059 patients' (1834 RA, 2741 SpA and 484 PsA) BMI data before the bDMARD treatments were available and analysed. Baseline characteristics of RA, SpA and PsA patients were given in Table 1. 72.3% of the RA patients were seropositive. HLAB27 was positive in 64.7% and 22.9% of the SpA and PsA patients. Anti-TNF therapy was started as first bDMARD in 57.2% of the RA patients, others were started with non-Anti-TNF bDMARDs. In SpA (99.2%) and PsA (100%) patients anti-TNFs were the first biologics. Overall, the proportion of obese patients was significantly higher in RA and PsA than in SpA patients (Table 1) and age and sex affected BMI significantly (p<0.001) (Figure 1). After adjusting age and sex indifferences between groups, the difference between the BMI of the patients disappeared (Table 1).

Table 1. Baseline characteristics and BMI of the patients

		RA	SpA	PsA	p
All bDMARD patients	N	1834	2741	484	
	Female, n (%)	1470 (80.2)	1257 (45.9)	334 (69.0)	0.000*
	Age, years*	52.9±13.4	43.1±11.4	47.4±12.2	0.000*
	Disease duration, years*	11 (7-17)	8 (5-13)	7 (3-12)	0.000*
	Body mass index*	29.6 ± 6.5	27.7 ± 5.4	29.2 ± 5.8	0.000*
Age and sex matched group	Obesity, n (%)	811 (44.2)	815 (29.7)	199 (41.1)	0.000*
	N	481	483	484	
	Female, n (%)	315 (65.7)	324 (67.1)	334 (69.0)	0.545
	Age, years*	47 (36-59)	48 (39-57)	47 (38-56)	0.691
	Disease duration, years*	10 (6-15)	5 (5-13)	7 (3-12)	0.000*
	Body mass index*	28.5 ± 6.1	28.5 ± 5.8	29.2 ± 5.8	0.150
	Obesity, n (%)	183 (38.0)	176 (36.4)	199 (41.1)	0.316

* Mean ±S.D *Median (IQR)