AB0276

COMPARISON OF HEALTH ASSESSMENT QUESTIONNAIRE DISABILITY PROGRESSION IN RHEUMATOID ARTHRITIS PATIENTS WITH PROPOSED SDAI, CDAI, AND RAPID3-BASED MINIMAL DISEASE ACTIVITY AND PATIENTS WITH LOW DISEASE ACTIVITY: RESULTS FROM A JAPANESE NATIONAL DATABASE

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Background: By OMERACT, a core-set definition of minimal disease activity (MDA) required a tender joint count (TJC) of 0, swollen joint count (SJC) of 0, and erythrocyte sedimentation rate (ESR) ≤ 10 mm/hour or the fulfilment of 5 of 7 core criteria, namely, pain ≤ 2 , SJC ≤ 1 , TJC ≤ 1 , health assessment questionnaire (HAQ) ≤ 0.5 , physician's global ≤ 1.5 , patient's global ≤ 2 , and ESR ≤ 20 .¹

In addition to the original Disease Activity Score 28 (DAS28)-based MDA definition (DAS28 ≤ 2.85),¹ we proposed a Simplified Disease Activity Index (SDAI) ≤ 5.3 , Clinical Disease Activity Index (CDAI) ≤ 4.8 , and Routine Assessment of Patient Index Data 3 (RAPID3) ≤ 5 , each value being two points higher than the respective remission criterion, as cut-offs for the MDA index in routine care for rheumatoid arthritis (RA).²

Objectives: To compare HAQ disability progression in patients with proposed SDAI, CDAI, and RAPID3-based MDA and patients with low disease activity (LDA) for each index.

Methods: We evaluated RA patients with functional remission (HAQ ≤ 0.5) registered with the Japanese National Database. We excluded patients with any missing values for patient SJC, TJC, physician's global, patient's global, pain, CRP, ESR, HAQ, or MDHAQ, which require the MDA to be assessed and the DAS28, SDAI, CDAI, and RAPID3 scores to be determined. HAQ disability progression from 2015 to 2016 was analysed in patients with MDA vs non-MDA and in those with LDA (or remission) vs non-LDA (or remission).

The interclass correlation of the disease activity categories of LDA and MDA in DAS28, CDAI (SDAI), and RAPID3 were also compared.

Results: In total 3798 patients were analysed, 76.5% of whom met the core-set definition of MDA and 40.3% of whom were assessed as being in Boolean remission. Patients with a core-set definition of MDA had less HAQ progression over one year (356 $\pm$ 71 days) than those without the core-set definition, at 0.036 (95% CI: 0.026–0.045) and 0.066 (0.050–0.082) (p=0.002), respectively. The progression of HAQ in each disease activity state is summarised in table 1.

Patients in the DAS28, SDAI, CDAI, and RAPID3-based MDA group showed less HAQ progression. The same results were found for LDA. For the MDA categories, the interclass correlation for CDAI (SDAI) vs DAS28, CDAI (SDAI) vs RAPID3, and DAS28 vs RAPID3 was 0.585 (0.617), 0.568 (0.557), and 0.361, respectively, and 0.449 (0.442), 0.411 (0.410), and 0.371 for LDA, respectively.

Abstract AB0276 – Table 1

Index-based MDA	HAQ progression, mean (95% CI)	Index based LDA	HAQ progression, mean (95% CI)
DAS28 ≤ 2.85	0.032 (0.022–0.041)	DAS28 ≤ 3.2	0.034 (0.023–0.045)
DAS28 > 2.85	0.064 (0.051–0.077)	DAS28 > 3.2	0.052 (0.041–0.063)
p	<0.001	p	0.011
SDAI ≤ 5.3	0.036 (0.026–0.045)	SDAI ≤ 11	0.040 (0.031–0.048)
SDAI > 5.3	0.058 (0.044–0.072)	SDAI > 11	0.074 (0.049–0.099)
p	0.008	p	0.010
CDAI ≤ 4.8	0.036 (0.027–0.046)	CDAI ≤ 10	0.039 (0.031–0.048)
CDAI > 4.8	0.056 (0.042–0.070)	SDAI > 10	0.073 (0.049–0.097)
p	0.020	p	0.011
RAPID3 ≤ 5	0.038 (0.029–0.047)	RAPID3 ≤ 6	0.037 (0.029–0.046)
RAPID3 > 5	0.058 (0.042–0.074)	RAPID3 > 6	0.068 (0.050–0.086)
p	0.031	p	0.003

Conclusions: Among patients with functional remission, both the MDA and LDA categories showed less HAQ progression over one year.

The interclass correlation for MDA was more acceptable than that for LDA. Index-based MDA, which provides more stringent criteria than LDA, may serve as an alternative target for LDA in patients who have difficulty achieving remission.

REFERENCES:

- Wells GA, et al. J Rheumatol 2005;32:2016–24.
- Yokogawa N, et al. Ann Rheum Dis 2017;76(supplement 2):525.

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AB0277

LEPTIN AND ADIPONECTIN LEVELS IN RHEUMATOID ARTHRITIS PATIENTS

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Background: Rheumatoid Arthritis (RA) is a chronic inflammatory autoimmune disorder characterised by systemic inflammation of joints, in particular the synovial membrane, cartilage and bone. Leptin (LEP) and adiponectin (ADIP) belong to a distinct group of biological molecules, adipokines which are secreted by white adipose tissue and have a central role in energy storage and metabolism.

Objectives: To compare fasting serum LEP and ADIP in RA to healthy controls and to investigate whether fasting LEP and ADIP serum levels change following a change in RA treatment.

Methods: Male and female subjects aged 18 years old or above with RA, fulfilling the 2010 ACR/EULAR classification criteria and having DAS28 score >4.2, were recruited and followed up for 90 days. LEP and ADIP levels were measured using ELISA. Fasting glucose, lipids and insulin levels were also measured as indicating factors for co-morbidities. The associations between RA patients at baseline and controls were assessed by uncorrelated/unrelated t-test analyses while associations between RA patients at baseline, day 30 and day 90 were assessed by one-way correlated/related analysis of variance.

Results: We studied 30 RA patients with mean (SD) age of 56.33 (13.84) years, of whom 24 (80%) were female and 15 healthy individuals with mean (SD) age of 53.80 (13.97) years, of whom 12 (80%) were female. LEP and ADIP levels for control subjects were 28.36 (15.08) ng/mL (p=NS vs RA at baseline) and 14.76 (4.42) μ g/mL (p<0.05 vs RA at baseline) (table 1). Over 90 days DAS28 and HAQ score improved significantly in RA patients. LEP levels for RA patients were 29.65 (18.17) ng/mL at baseline, 30.81 (17.65) ng/mL at day 30 and 25.90 (15.01) ng/mL at day 90 (p=NS) whilst ADIP levels for RA patients were 16.78 (8.73) μ g/mL at baseline, 16.65 (8.70) μ g/mL at day 30 and 15.44 (7.24) μ g/mL at day 90 (p=NS) (table 2).

Abstract AB0277 – Table 1. Demographics and baseline adipokines for RA patients and control subjects. All values are mean (SD) unless otherwise indicated where we use n (%). (**Levene's test for equality of variances value p<0.05)

Variable	RA Patients	Control subjects	Levene's test for equality of variances (p)
Gender (M/F)	6 (20%)/24 (80%)	3 (20%)/12 (80%)	1.000
Age (years)	56.33 (13.84)	53.80 (13.97)	0.850
LEP (ng/mL)	29.65 (18.17)	28.36 (15.08)	0.421
ADIP (μ g/mL)	16.78 (8.73)	14.76 (4.42)	0.023**

Abstract AB0277 – Table 2. Change over time in adipokine levels and RA parameters for RA patients at baseline, day 30 and day 90. All values are mean (SD). (**One Way ANOVA significance value p<0.05)

Variable	LEP (ng/mL)	ADIP (μ g/mL)	RA parameters			
			DAS28	HAQ score	PAIN score	HEALTH score
RA patients (day 0)	29.65 (18.17)	16.78 (8.73)	5.59 (0.56)	1.20 (0.70)	1.80 (0.73)	1.59 (0.55)
RA patients (day 30)	30.81 (17.65)	16.65 (8.70)	4.39 (0.93)	0.84 (0.66)	1.07 (0.82)	1.11 (0.84)
RA patients (day 90)	25.90 (15.01)	15.44 (7.24)	3.92 (1.08)	0.76 (0.74)	1.07 (0.82)	1.32 (0.81)
One Way ANOVA (p)	0.515	0.791	0.000**	0.045**	0.000**	0.046**

Conclusions: ADIP was significantly higher in RA patients at baseline compared to control subjects and there was a trend towards normalisation of levels as inflammation improved. The role of ADIP in active RA remains unclear and further examination of the site of origin of ADIP as well as its role in pro- and anti-inflammatory pathways warrants further study.

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