
Background: Fatigue in rheumatoid arthritis (RA) is hypothesized to be caused by inflammation. Still ~50% of fatigue in RA cannot be explained by the disease activity score (DAS), nor by genetic or psychological factors. Objectives: Since MRI can detect joint inflammation more sensitively than DAS, we hypothesized that residual inflammation detected by MRI could aid in explaining fatigue in RA at diagnosis and during follow-up.

Methods: 526 consecutive RA-patients were followed longitudinally. Fatigue was formed at inclusion, after 12 and 24-months in 199 patients and were scored by inflammation measure.

Results: Fatigue was scored by MRI inflammation measure.

Conclusion: Sensitive measurements of joint inflammation did not aid in explaining fatigue in RA at diagnosis and during follow-up. The 2-year course of MRI-inflammation associated with the course of fatigue (linear mixed models) and whether decrease in MRI-inflammation in year-1 associated with subsequent improvement in fatigue in year-2 (cross-lagged models). Similar analyses were done with DAS as inflammation measure.

Results: At diagnosis, higher DAS-scores were associated with more severe fatigue (p<0.001). However, patients with more MRI-inflammation were not more fatigued (p=0.94). During 2-year follow-up, DAS decrease associated with improvement in fatigue (p<0.001), but MRI-inflammation decrease did not (p=0.056). DAS decrease in year-1 associated with fatigue improvement in year-2 (p=0.012), as did MRI-inflammation decrease (p=0.030), with similar effect strength.

Conclusion: Sensitive measurements of joint inflammation did not aid in explaining fatigue in RA at diagnosis and follow-up. This supports the concept that fatigue in RA is partly uncoupled from inflammation.

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POS0439

THE INFLUENCE OF ESCALATED PGA SCORE ON DISEASE ACTIVITY, DAILY ACTIVITY, AND QUALITY OF LIFE, AND SOLUTION FOR OPTIMAL PGA LEVEL THAT DESERVES CLINICAL REMISSION IN PATIENT WITH RHEUMATOID ARTHRITIS

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Background: Patient’s global assessment (PGA) is one most difficult component as a part of disease activity index for treatment of rheumatoid arthritis (RA), that often causes an obstacle to achieving clinical remission. Moreover, PGA level affects activities in daily lives.

Objectives: The influence of escalated PGA score on disease activity, daily activity, and quality of life for patient with RA was investigated, and the optimal PGA level for both disease activity and daily activities was investigated from real world data.

Methods: A total of 24,075 times of monitoring for RA was performed in the institute. Monitored items included TJC, SJC, PGA, EGA, CRP, and calculated values of DAS28, CDAI, SDI, composite index of Boolean evaluation (Boolean), pain score with visual analog scale (PS-VAS), Health Assessment Questionnaire Disability Index (HAQ-DI), and quality of life score (QOLS) calculated from Euro-QOL questionnaire with 5th dimensions. Each measured item was calculated as mean value according to the PGA score, which was measured at the same time. The PGA score was classified by one increment significantly by one from zero to ten. The mean values of DAS28, CDAI, SDI, remission rate of these indices and Boolean remission rate, and mean values of PS-VAS, HAQ-DI, and QOLS were statistically evaluated.

Conclusion: 0.5 was determined as remission (HAQ remission). Sensitivity and specificity regarding attainment HAQ remission according to each level of PGA score were calculated, and cutoff index (COI) was determined with receiver operating characteristic (ROC) curve. For PS-VAS, specificity and specificity of Boolean evaluation regarding each level of PS-VAS after classification divided by one increment was calculated, and comparable level (PS-VAS remission) was determined with reference of the curve. ROC was performed according to PGA level, and COI was determined with a similar manner.

Results: Number of measures counted 10428, 3096, 3110, 2346, 998, 1773, 751, 703, 655, 139, and 73 for each PGA level. PGA level from 3 to 5, and 5 to 10 were put together for number adjustment. Mean DAS28, CDAI, and SDI demonstrated significant increase as PGA level increased, and remission rate of the all indices including Boolean demonstrated significant decrease as PGA level increases (p<0.01%). Boolean remission rate demonstrated zero percent from two, and CDAI and SDI remission rate demonstrated from zero five, whereas DAS remission rate showed gradual decrease then zero percent was not shown in any level. Mean value of PS-VAS and HAQ-DI score demonstrated also significant decrease as PGA level increases, and QOLS demonstrated significant decrease as PGA level increases (p<0.01%). Increase of HAQ-DI score and decline of QOLS demonstrated more steep from PGA level 3 whereas no significant difference demonstrated from zero to one. HAQ remission counted 15,703, whereas no HAQ remission counted 8,335. Using ROC, COI of the PGA level was 2.0, whereas sensitivity and specificity were 63.6% and 66.3%, respectively. The estimated PS-VAS remission level was 10mm. Optimal PGA level for PS-VAS remission was set as 1.0, and sensitivity and specificity regarding PS-VAS remission were 87.1% and 71.3%, respectively.

Conclusion: Increase of PGA affects daily activities and quality of life. The optimal level that increases deterioration risk significantly was supposed to be from 3. Optimal level of PGA score for attaining the PS-VAS remission was 1.0, whereas the optimal PGA level for HAQ-DI remission is 0.5, despite sensitivity and specificity for the HAQ remission were lower than these for the PS-VAS remission.

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POS0440

REDUCTION IN MONOCYTE COUNT PREDICTS SUSTAINED REMISSION IN RHEUMATOID ARTHRITIS PATIENTS TREATED WITH ANTI-TNF THERAPY

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Background: Sustained remission is the ultimate goal in the management of rheumatoid arthritis (RA) but is infrequently achieved. After conventional therapy, TNFi (tumour necrosis factor inhibitor) has a strong track record for achieving remission. Real-world data suggests variable effect of RA and TNFi on various cellular components of the full blood count (FBC), but their relationship with loss of remission (LOR) is unclear.

Objectives: To investigate whether cellular changes in the FBC can predict LOR (remission defined as DAS28-ESR≤2.6) in patients with RA receiving TNFi, adjusted using key clinical factors.

Methods: Real-world clinical and routine laboratory data were analysed from two independent cohorts of adult RA patients, who were started on their first TNFi (from September 2009 to December 2019), and then entered into remission. Data was extracted in October 2020. A linear mixed model was used to investigate longitudinal changes of different components of FBC and CRP, stratified by LOR, and grouped by

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POS0438

IS IMAGING DETECTED JOINT INFLAMMATION HELPFUL IN EXPLAINING FATIGUE IN RHEUMATOID ARTHRITIS AT DIAGNOSIS AND DURING THE DISEASE COURSE? – A LARGE MRI STUDY

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Background: Fatigue in rheumatoid arthritis (RA) is hypothesized to be caused by inflammation. Still ~50% of fatigue in RA cannot be explained by the disease activity score (DAS), nor by genetic or psychological factors.

Objectives: Since MRI can detect joint inflammation more sensitively than DAS, we hypothesized that residual inflammation detected by MRI could aid in explaining fatigue in RA at diagnosis and during follow-up.

Methods: 526 consecutive RA-patients were followed longitudinally. Fatigue was assessed yearly on a numerical rating scale. Hand and foot MRIs were performed at inclusion, after 12 and 24-months in 199 patients and were scored for inflammation (synovitis, tenosynovitis and osteitis combined). We studied whether RA-patients with more MRI-inflammation were more fatigued at diagnosis (linear regression), whether the 2-year course of MRI-inflammation associated with the course of fatigue (linear mixed models) and whether decrease in MRI-inflammation in year-1 associated with subsequent improvement in fatigue in year-2 (cross-lagged models). Similar analyses were done with DAS as inflammation measure.

Results: At diagnosis, higher DAS-scores were associated with more severe fatigue (p<0.001). However, patients with more MRI-inflammation were not more fatigued (p=0.94). During 2-year follow-up, DAS decrease associated with improvement in fatigue (p<0.001), but MRI-inflammation decrease did not (p=0.096). DAS decrease in year-1 associated with fatigue improvement in year-2 (p=0.012), as did MRI-inflammation decrease (p=0.039), with similar effect strength.

Conclusion: Sensitive measurements of joint inflammation did not aid in explaining fatigue in RA at diagnosis and follow-up. This supports the concept that fatigue in RA is partly uncoupled from inflammation.

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