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to RTX (6,6). The demographic and response data DAS 28 are shown in Table 1. There are no differences in the years of evolution,% of women, FR or ACPA positive, erosions and disease activity, as measured by DAS 28, before the switch, between The two groups (anti-TNF vs non-anti-TNF). Patients in the anti-TNF group were slightly younger than non-anti-TNF.When the DAS 28 response is evaluated at 3 and 6 months, modifying the treatment is effective (DAS 28 beginning 4,40 vs DAS 28 6 months 2.8 p<0.001). When assessing the response to change, there is no difference in the DAS 28 response at 3 months or 6 months, if you switched to anti-TNF or non-anti-TNF (3.18 vs 2.52 p=0.122)

When comparing the patients with anti TNF alpha vs TCZ, 62.5% of the patients with TCZ are in remission compared to 38, 5% (p=0.047).

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Table 1							
	Total n=61	Swich anti-TNF alpha n=26	Swich No anti-TNF alpha n=35	р			
Age (years)	60.08±11,5	56.19±8,4	62.97±12,8	0.023			
Sex (% M)	80.3	84.6	77.1	0.532			
Years evolution (years)	17.44±9,4	18.92±11,6	16.34±7,4	0.298			
Erosion (%)	90.2	92.3	88.6	1.000			
FR+ (%)	77.0	76.9	77.1	1.000			
ACPA+ (%)	91.8	88.5	94.3	0.642			

Table 2

	Total n=61	Swich anti-TNF alpha n=26	Swich No anti-TNF alpha n=35	р
DAS 28 Swich	4.40±1,38	4.61±1,5	4.21±1,3	0.216
DAS 28 3 months	3.18±1,52	3.52±1,09	2.96±1,7	0.167
DAS 28 6 months	2.8±1,54	3.18±1,74	2.52±1,3	0.122
DAS 28 < 2.6 6 m (%)	52.5	38.5	62.5	0.047

Conclusions: In this retrospective study in daily clinical practice, it is evident that the change in treatment after failure of the first biological one, without differences if the change is to an anti TNF or another treatment. The percentage of patients who are remission at 6 months is higher if the change is at TCZ. Given the small number of patients, larger studies would be needed to confirm the results. References:

[1] Johnston SS et col. Comparison of Biologic Disease-Modifying Antirheumatic Drug Therapy Persistence Between Biologics Among Rheumatoid Arthritis Patients Switching from Another Biologic. Rheumatol Ther.

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AB0382 OUTCOMES OF ETANERCEPT THERAPY IN ELDERLY RHEUMATOID ARTHRITIS PATIENTS: AN INVESTIGATION OF THE AKITA ORTHOPEDIC GROUP ON RHEUMATOID ARTHRITIS REGISTRY

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Background: The Akita Orthopedic Group on Rheumatoid Arthritis (AORA) encompasses 32 physicians and 28 clinics providing medical care to rheumatoid arthritis (RA) patients in Akita Prefecture, Japan. The patient registry for this group (the AORA registry) reflects actual clinical data on RA therapy in Akita, where the proportion of elderly (age ≥65 years) residents in the population is the highest in Japan. Etanercept (ETN) is a tumor necrosis factor inhibitor reportedly associated with fewer adverse event-related treatment discontinuations than other drugs of this class. However, few evaluations of the efficacy and safety of ETN have been reported for elderly populations.

Objectives: Based on data from the AORA registry, we aimed to investigate the continuation rate for ETN therapy, reasons for discontinuation, and therapeutic effects among elderly RA patients living in Akita.

Methods: Among 204 AORA-registered patients starting ETN therapy between January 2009 and August 2014, data for the 73 patients (35.8%) who were ≥65 years old at the initiation of therapy were evaluated. Mean age was 72.4±4.7 years (range 65-83 years), and 79.5% were women. Mean disease duration was 15.1±12.6 years (range 9 months-55 years), 13.7% of patients were switching from another biologic agent, 49.3% could perform activities of daily living (ADL) independently, and 65.8% had at least one of the following comorbidities: hypertension, diabetes, respiratory disorder, cardiovascular disease, and cerebrovascular disease. We evaluated the 1-year cumulative continuation rate for ETN therapy using the Kaplan-Meier method, and investigated the characteristics of patients who discontinued treatment because of adverse events (AE cohort) or lack of efficacy (LOE cohort). We evaluated efficacy in 55 patients for whom Disease Activity Score - C-reactive protein assessments were possible, based on European League Against Rheumatism (EULAR) criteria.

Results: The 1-year cumulative continuation rate for ETN therapy was 87.2%, and 24 patients discontinued treatment. The AE and LOE cohorts contained 12 and 7 patients, respectively. The AE cohort had a mean age of 75.5 years at the start of treatment, with a mean disease duration of 20.7 years, 8.3% of patients switching from another biologic agent, 16.7% performing ADL independently, and a comorbidity rate of 100%. Corresponding values for the LOE cohort were: mean age, 71.4 years; disease duration, 12.7 years; switching from another biologic agent, 42.9%; performing ADL independently, 57.1%; and comorbidity rate, 28.6%. Efficacy was noted for 81.8% of all patients with 52 weeks of ETN therapy, achieving good efficacy in 21 cases and moderate efficacy in 24 cases. Conclusions: Retention rate and efficacy were considered satisfactory in elderly RA patients receiving ETN therapy. The risk of adverse events was suggested to increase with increasing age, declining ADL, and presence of comorbidities. These factors require attention when prescribing ETN therapy.

References:

[1] Cho SK, Sung YK, Kim D, et al. Drug retention and safety of TNF inhibitors in elderly patients with rheumatoid arthritis. BMC Musculoskelet Disord. 2016;

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AB0383 SUBCLINICAL BRAIN DAMAGE IN PATIENTS WITH RHEUMATOID ARTHRITIS AND ITS RELATIONSHIP TO TNF **BLOCKER THERAPY**

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Background: RA is a chronic disease with a yet unclarified etiology, which causes the activation of pro-inflammatory pathways that bring about joint and systemic inflammations (1). In recent years, the pathophysiology of brain damage that can occur in RA has drawn attention. Emphasis is being put on the possibility that brain damage occurs due to blood-brain barrier (BBB) damage that is linked to chronic inflammation

Objectives: In this study, we aimed to investigate the peripheral blood levels of brain-specific proteins such as S100 beta and GFAP (glial fibrillary acidic protein), the differences in these proteins in patients who did and did not undergo TNF blocker therapy and their relationship with cranial MR lesions, disease activity and cognitive functions with the purpose of determining CNS (central nervous system) damage in patients with rheumatoid arthritis (RA).

Methods: 58 RA patients (47 (81.0%) females, 11 (19.0%) males) and 34 healthy controls (24 (70.6%) females, 10 (29.4%) males) were included in the study. All RA patients were on synthetic DMARD therapy at the beginning. While 30 patients continued sDMARD therapy, 28 patients with high disease activity were started on TNF blocker therapy. All demographic characteristics of the patients were recorded. Disease activity was evaluated using DAS28. The Mini-Mental State Examination (MMSE) was used to evaluate cognitive functions, and the Fazekas Scale was used to assess the cranial MRI lesions. The peripheral blood S100 beta, GFAP, claudin, IL-17, IL-1 beta levels of the patients were measured at the beginning and on the 6th month.

Results: Demographic characteristics were similar between the two groups and no statistical difference was detected between the patient group and the control group in terms of sex, age, and BMI. (p>0.05) S100 beta and GFAP levels were higher to a significant degree compared to the control group. (p<0.05) In the group that was started on TNF blocker therapy, S100 beta and GFAP levels were detected to have decreased significantly 6 months after treatment compared to the start of treatment. (p<0.05) No difference was found between the RA and control groups in terms of hyperintense lesions seen in the cranial MRI. (p>0.05) As the lesions in the deep white matter seen in the cranial MRI of RA patients increased, their S100 beta levels were also seen to increase. (p<0.05)

Conclusions: In conclusion, next to decreasing disease activity and joint erosions by suppressing inflammation, anti-TNF therapy in RA can also suppress potential brain damage linked to subclinical BBB (blood-brain-barrier) dysfunction. Further studies with broader participation and longer patient follow-up are needed to reinforce this hypothesis.

References:

[1] McInnes, I.B. and Schett, G. (2011). Thepathogenesis of rheumatoidarthritis. N Engl J Med 365, 2205 2219.doi:10.1056/ NEJMra1004965.

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CLINICAL AND RADIOLOGICAL EVOLUTION IN RHEUMATOID ARTHRITIS (RA) PATIENTS AFTER DEINTESIFICATE BIOLOGICS

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Background: RA is the most common chronic inflammatory arthritis. About 30% of patients are treated with biological therapy (BT). Deintensification of BT for patients in clinical remission, is a strategy used in clinical practice to reduce side effects and burden.

Objectives: The primary endpoint was evaluate clinical and radiological behavior of the RA in patients receiving BT at reduced doses.