Background: Ankylosing Spondylitis (AS) is an inflammatory rheumatic disease that affects the spine and sacroiliac joints (SIJ). Non-steroidal anti-inflammatory drugs (NSAIDs), modifying antirheumatic drugs (DMARDs), and recently anti-TNF blockers, which are more costly than NSAIDs but used in resistant cases, are generally used in its treatment (1).

Objectives: While SIJ-MRI is one of the imaging methods in the diagnosis of the disease, it is not yet used in its follow-up (2). We wanted to define when the activated SIJ-MRI findings, which showed early sacroiliitis, regressed according to the treatment option.

Methods: Among the 8100 SIJ-MRIs taken in our hospital in the last 5 years, those that were reported normally were excluded from the study. Among the remaining 1150 patients with active or chronic SIJ findings, a total of 87 patients who were diagnosed with AS and had active SIJ findings in the first imaging and had a second SIJ-MRI examination for any reason were included in our retrospective, cross-sectional and observational study. According to the treatment option, how long the active SIJ-MRI findings disappeared was calculated in months using the Kaplan-Meier method.

Results: Of the 87 patients examined in the study, 41 were women and 46 were men. The average age is 32.7. Active SIJ-MRI findings disappeared in a mean of 30.6 months for 11 of 24 patients who did not use any medication, in a mean of 33.9 months for 6 of 16 patients using NSAIDs, in a mean of 40.7 months for 14 of 34 patients using sulfasalazine, in a mean of 28.2 months for 3 of 6 patients using adalimumab, in a mean of 24.4 months for 3 of 5 patients using methotrexate, 11.9 month for 2 of 1 patient using certolizumab. There was no statistically significant difference between the groups.

Conclusion: The signs of active sacroiliitis disappeared in less time in patients who did not receive any treatment than those using NSAIDs and sulfasalazine. Patients using anti-TNF blockers had earlier results than those using no medication. The reason for this may be the progressive course of patients who are switched to the next level of treatment and their late response to treatment, as well as the mechanism of the disease (2-3). This situation brings to the agenda the tendency of physicians to over-treat the disease. More randomized controlled studies are needed regarding new treatment options.

REFERENCES:

- Braun J. Axial spondyloarthritis including ankylosing spondylitis' Rheumatology (Oxford). 2018 1;57(suppl_6):vi1-vi3.
- Braun J, Baraliakos X, Golder W, Brandt J, Rudwaleit M, Listing J, Bollow M, Sieper J, van der Heijde D. Magnetic resonance imaging examinations of the spine in patients with ankylosing spondylitis, before and after successful therapy with infliximab. *Arthritis & Rheumatism* Vol. 48, No. 4, April 2003, pp 1126–1136.
- Jee WH, McCauley TR, Lee SH, Kima SH, Ima SA, Had KY. Sacroiliitis in patients with ankylosing spondylitis: association of MR findings with disease activity. *Magnetic resonance imaging* 2004;22;245–250.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.4031

POS0940

THE IMPACT OF TUMOUR NECROSIS FACTOR-ALPHA INHIBITOR TREATMENT ON WNT SIGNALING INHIBITORS, NOGGIN AND CYTOKINE LEVELS IN AXIAL SPONDYLOARTHRITIS

N. Atas¹, B. Çakır², F. Bakır³, M. Uçar⁴, H. Satış¹, G. T. Güz⁵, H. Babaoglu¹, R. Bilici Salman¹, A. Avanoğlu Güler¹, H. Karadeniz¹, S. Haznedaroğlu¹, B. Goker¹, M. A. Oztürk¹, A. Tufan¹. ¹Gazi University, Division of Rheumatology, Department of Internal Medicine, Ankara, Turkey; ²Alab Laboratory, Department of Clinical Biochemistry, Ankara, Turkey; ³Lokman Hekim University, Department of Clinical Biochemistry, Ankara, Turkey; ⁴Gazi University, Department of Radiology, Ankara, Turkey; ⁵Gazi University, Department of Internal Medicine, Ankara, Turkey

Background: Axial spondyloarthritis (axSpA) is a common chronic inflammatory disease of the axial skeleton. Some cytokines have important roles in initiation and progression of disease and are elevated in active disease. Additionally, Wnt signaling pathway inhibitors and noggin also appear to be involved in pathogenesis of ankylosing spondylitis. Anti-tumor necrosis factor-α (TNF) agents have dramatically improved the clinical outcome of axSpA; however, acceptable clinical improvement is not achieved in all patients and capacity of anti-TNF to slow or prevent structural damage still remains controversial.

Objectives: To evaluate the effect of anti-TNF on inflammatory and noninflammatory milieu in patients with axSpA.

Methods: In this prospective study we included 30 biologic treatment naive adult patients with axSpA and 30 healthy controls. All patients with high disease activity were treated with anti-TNF therapy for 6 months. Laboratory and clinical evaluation of all patients were performed at baseline and after 6 months of anti-TNF treatment. Following cytokines and wnt/BMP antagonists were measured; TNF-Alpha, COX-2, IL-6, IL-17, IL-22, IL-23, IL-33, dickkopf-1, sclerostin, noggin.

Results: The mean age of patients with axSpA and healthy controls were 38.1±13.3 and 37.7±7.7 years, respectively (p>0.005). At baseline, the median (IQR) TNF-α was higher in axSpA patients when compared to healthy controls, 34.4 pg/ml (31.4-37.03) vs 18.1 pg/ml (12.1-28.4), (p<0.001), while the median (IQR) dickkopf-1 and sclerostin were lower in axSpA patients, 446.7 pg/ml (356.9-529.3) vs 1088.7 pg/ml (951.7-1244.4), (p<0.001) and 312.4 pg/ml (140.8-412.7) vs 412.3 pg/ml (295.4-512.8), (p<0.001), respectively. IL-17, IL-22, IL-33, dickkopf-1 and sclerostin increased with anti-TNF treatment (table 1).

Conclusion: Elevation of some cytokines which are important in pathogenesis of axSpA and nonincrease in noggin with anti-TNF drugs may affect effectiveness of anti-TNF treatment.

Table 1. Changes of cytokines, dickkopf-1, sclerostin and noggin with anti-TNF treatment.

	Pre-Anti-TNF	Post-Anti-TNF	P value
IL-6	45(39.1-68.8)	476(273-61.1)	0.750
IL-17	93.3 (85.1-104.8)	102.1(86.6-114.6)	0.026
IL-22	159.2 (151.9-178.4)	183.5(156.3-304.6)	0.033
IL-23	36.5 (26.1-52.9)	41.3(28.4-55.5)	0.658
IL-33	127.8 (106.6-186.1)	147.06(128.5-213.4)	0.016
COX2	0.176 (0-0.374)	0.202(0.051-1.151)	0.469
TNFα	34.4(31.4-37.03)	30.7(12.8-35.6)	0.004
Dickkopf-1	446.7(356.9-529.3)	881.3(663.1-972.2)	<0.001
Sclerostin	312.4 (140.8-412.7)	405.1(276.3-452.5)	0.018
Noggin	48.3(17.04-153.9)	31.2(11.3-103.7)	0.264

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.4130

POS0941

IN SPONDYLOARTHRITIS PATIENTS THE PRESENCE OF COMORBIDITIES IS AN INDEPENDENT PREDICTOR OF INSUFFICIENT RESPONSE TO THERAPY WITH BIOLOGIC AGENTS AND TREATMENT DISCONTINUATION

I. Flouri¹, N. Koukgas¹, N. Avgustidis¹, A. Repa¹, A. Eskitzis¹, A. Molla Ismail Sali¹, S. Pitsigavdaki¹, K. Pateromichelaki¹, E. Kalogiannaki¹, G. Bertisias¹, P. Sidiropoulos¹. ¹University Hospital of Heraklion, Crete, Rheumatology Department, Heraklion, Crete, Greece

Background: Long-term observational studies of patients under biologic disease-modifying anti-rheumatic drug (bDMARD) therapies in routine clinical practice can provide us with important data regarding patients with comorbidities, who are usually excluded from randomized controlled studies.

Objectives: To study the impact of comorbidities in the outcome (response and persistence to therapy) of patients with spondyloarthritis (SpA) receiving bDMARDs in real-world clinical practice.

Methods: Prospective study of all patients who start a bDMARD in a tertiary centre University Hospital after their consent. All patient comorbidities [among a list of approximately 100 pre-specified major comorbidities] are registered by treating physicians at baseline and during follow-up.

Comorbidities were studied as total Comorbidities Count (CC) and rheumatic disease comorbidity index (RDCI). Statistical analyses were performed using logistic and Cox regression models, adjusting for the potential confounding of age, sex, disease duration, diagnosis (axial vs. peripheral SpA), number of previous conventional synthetic and biologic DMARDs, year of therapy start, and co-administered methotrexate and corticosteroids (yes/no). Analyses of response to therapy also included baseline BASDAI or ASDAS indices as confounding variables.

Results: A total of 603 biologic treatments (1st: 298, 2nd: 157, ≥3rd: 148) were analyzed. Half (51%) of the patients were female, 413 patients had axial SpA (AxSpA) and 190 peripheral SpA (perSpA). At baseline, median (IQR) age: 48 (38-57) years, disease duration: 11 (4-19) years, CC: 2 (1-4) and RDCI: 1 (0-2). Both comorbidity indices were significantly higher in perSpA compared to AxSpA (p<0.001).

At 6 months of therapy, 31% of patients with AxSpA achieved BASDAI50 and 39% had ASDAS-ESR < 2.1. Higher CC was an independent predictor of insufficient response according to BASDAI50 [OR (95%) = 0.70 (0.52-0.94), p=0.019] and higher RDCI was predicting failure to achieve ASDAS-ESR < 2.1 [OR (95%) = 0.59 (0.37-0.94), p=0.027]. Other independent predictors of non-response were age, longer disease duration and (for ASDAS-ESR<2.1) higher baseline disease activity.

During 1405 patient-years of follow-up, 349 (58%) treatments were discontinued. The adjusted hazard ratio for bDMARD discontinuation within the first 2 years of treatment due to insufficient response was doubled in patients with CC ≥2 versus those with CC ≤1 [HR = 2.27 (1.14-4.53), p=0.020] or with RDCI ≥1 (vs. RDCI =