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 was 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:

Disclosure of Interests: None declared
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Table 1. Changes of cytokines, dickkopf-1, sclerostin and noggin with anti-TNF treatment.

<table>
<thead>
<tr>
<th>Cytokine/Protein</th>
<th>Pre-Anti-TNF</th>
<th>Post-Anti-TNF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TNF-alpha</td>
<td>34.4(31.4-37.03)</td>
<td>30.7(12.8-35.6)</td>
<td>0.004</td>
</tr>
<tr>
<td>IL-17</td>
<td>159.2(151.9-178.4)</td>
<td>183.5(156.3-304.6)</td>
<td>0.033</td>
</tr>
<tr>
<td>IL-23</td>
<td>36.5(25.6-52.9)</td>
<td>41.3(38.6-55.1)</td>
<td>0.068</td>
</tr>
<tr>
<td>IL-33</td>
<td>1278(106.8-161)</td>
<td>1470(128.5-213.4)</td>
<td>0.016</td>
</tr>
<tr>
<td>COX2</td>
<td>0.176 (0-0.374)</td>
<td>0.202(0.051-1.151)</td>
<td>0.469</td>
</tr>
<tr>
<td>TNF-alpha</td>
<td>34.4(31.4-37.03)</td>
<td>30.7(12.8-35.6)</td>
<td>0.004</td>
</tr>
<tr>
<td>Dickkopf-1</td>
<td>446.7(356.9-529.3)</td>
<td>881.6(663.1-972.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Sclerostin</td>
<td>312.4 (140.8-142.7)</td>
<td>405.7(126.3-452.5)</td>
<td>0.018</td>
</tr>
<tr>
<td>Noggin</td>
<td>48.3(1704.153.9)</td>
<td>312(113-103.7)</td>
<td>0.264</td>
</tr>
</tbody>
</table>

POS0041

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

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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 spondylarthritides (SpA) receiving bDMARDs in real-world clinical practice.

Methods: Prospective study of all patients who start a bDMARD in a tertiary centre Hospital and have 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-29th, 25th, 22nd, 18th-24th) 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 29% 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 = 2.72 (1.14-5.3), p=0.020] or with RDCI ≥1 vs. RDCI =
Spondyloarthritis - clinical aspects (other than treatment)

**POS0942**

THE NEGATIVE IMPACT OF UNDIAGNOSED DEPRESSION IN AXIAL Spondyloarthropathy: RESULTS OF A SCREENING STUDY

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**Background:** Previous research in axial spondyloarthropathy (axSpA) has shown this population to have a high prevalence of depression. This co-morbidity has been previously shown to impact disease activity in patients with rheumatic disease.

**Objectives:** The purpose of this study was to screen for early signs of depression using two validated tools, the Patient Health Questionnaire-9 (PHQ-9) and the Hospital Anxiety and Depression Scale for depression (HADs-D) in patients with known axSpA.

**Methods:** AxSpA patients attending the Rheumatology department in St James’ Hospital between February and October 2020 were invited to take a self-administered survey which included the PHQ-9 and the HADs-D. Scores from the HADs-D yielded a numerical result which was then categorised as normal, borderline or abnormal. PHQ-9 numerical results were categorised as normal, mild, moderate, moderate/severe or severe. Patients with a known diagnosis of depression were excluded. In addition to baseline demographics, patient reported outcomes from the clinic visit were also recorded.

**Results:** In total 71 axSpA patients took part in the survey. The population was 70.4% (50) males and 29.5% (21) females. The mean age was 57.6 years and 54.3% (39) were smokers. Prior to depression screening, PHQ-9 and HADs-D scores were calculated. A one-way analysis of variance (ANOVA) was used to determine significance of variation in outcomes between patient outcomes as determined by the HADs-D and PHQ-9. A p-value of <0.05 was deemed significant. Consent was obtained prior to participation. Approval was received from the St James’/Tallaght Hospital Joint Ethics Committee.

**Results:** In total 71 axSpA patients took part in the survey. The population was 70.4% (50) males and 29.5% (21) female, with an average age 47.9 years and mean disease duration 19.7 years (mean outcomes: BASDAI 4.08, BASFI 3.62, BASMI 3.54, ASQoL 6.79). Overall, 7 (9.9%) participants recorded abnormal HADs-D scores, while 17 (23.9%) recorded moderate to severe PHQ-9 scores indicative of underlying depression. AxSpA females had higher mean HADs-D scores, with significantly worse BASMI (7.9 vs 4.8, p=0.01) than males, with abnormal scores in 19% (4) of females and 6% (3) of males. No significant differences were found in PHQ-9 scores between genders.

Analysis revealed significantly worse BASDAI (6.27 vs 3.42, p<0.01) and ASQoL scores (12.57 vs 5.26, p<0.01) in axSpA patients with abnormal compared to normal HADs-D scores. No significant differences were noted in BASFI, BASMI or baseline demographics. A similar pattern was noted on analysis of PHQ-9 scores, with significantly worse BASDAI (7.9 vs 2.55, p<0.01), BASFI (8.05 vs 2.33, p<0.01) and ASQoL (19.5 vs 2.62, p<0.01) noted in those scoring as severe compared to normal. No significant differences were detected in BASMI scores or baseline demographics.

**Conclusion:** A high percentage of axSpA patients recorded higher HADs-D and PHQ-9 scores concerning for undiagnosed depression. These patients were noted to have significantly worse disease activity and quality of life as compared to patients with normal scores. Clinicians treating axSpA should consider screening for depression in this population.

**Disclosure of Interests:** Sinead Maguire Speakers bureau: Speaker fee from Jansen, Grant/research support from: Recipient of the Gilead Inflammation Fellowship Grant, Finbar Barry O’Shea: None declared

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**Reference:**