Conclusions: Draft classification criteria for GPA, MPA and EGPA have been created which reflect current practice and have good sensitivity and specificity.

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From NSAIDs to bDMARDs in SpA: what is new?___

OP0022

IS A PRIMARY GOOD RESPONSE TO NSAIDS PREDICTIVE OF THE SUBSEQUENT RESPONSE TO THE FIRST TNF INHIBITOR IN PATIENTS WITH RECENT AXIAL SPONDYLOARTHRITIS?

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Background: Good response to NSAIDs is a SpA feature included in classification criteria for axial spondyloarthritis (axSpA). Among patients eligible for a TNF inhibitor (TNFi), some patients may have never responded to NSAIDs (non-responders to NSAIDs) while others initially responded (responders to NSAIDs) but have secondarily escaped and need to be treated with biologics.

Objectives: Our aim was to determine if the initial response to NSAIDs is an independent predictive factor of a subsequent good response to the first TNFi in axSpA.

Methods: **Patients:** Patients from the prospective observational DESIR cohort of early axSpA cohort who started a TNFi over the 5 years of follow-up.

NSAIDs response and TNFi response definitions

NSAIDs response was defined by the item 'good response to NSAIDs according to Amor's criteria' at the inclusion visit. TNFi response was defined by the BASDAI50 response between the 'baseline' visit (last cohort visit before TNFi initiation) and the 'follow-up' visit (visit taking place after at least 8 weeks of TNFi treatment).

Analysis: We compared the characteristics of the NSAIDs responder to the non-responders and their response to the first TNFi.

We performed a multivariate logistic regression modelling the impact of an NSAID response to the TNFi response. We included known predictive factors of TNFi response in this model (age, gender, HLAB-B27, activity of the disease [ASDAS-CRP], CRP, X-ray and MRI sacroiliitis). To account for selection bias and for confirmation purpose, we applied a propensity score with Inverse Probability Weighting (IPW) method to predict TNFi response (SAS, version 9.2).

Results: Among the 708 patients of the cohort, 236 were included in the analysis. At the inclusion, the main characteristics were the following: 106 (44.7%) males, mean age 33.8±3.9 years, mean BASDAI 54.5±17.3 and 202 (85.6%) were NSAIDs responders.

The NSAIDs responder and non-responder groups were comparable at M0 except for HLA-B27 positive status: 59.9% vs 40.1%, $p=0.041$, CRP level: 13.4±20.3 mg/L vs 6.3±6.6 mg/L, $p=0.027$, history of psoriasis: 17.8% vs 35.3%, $p=0.001$ and BASDAI: 53.0±18.1 vs 61.8±13.2, $p=0.001$, in responder and non-responder patients, respectively.

The percentage of TNFi responders was 32.2% (65/202) and 23.5% (8/34) in the NSAIDs responder and non-responder groups, respectively (univariate analysis [OR 1.54 [95% CI: 0.7 to 3.6], $p=0.313$).

The multivariate logistic regression found the following independent factors of the TNFi response: gender [adjusted OR (aOR)=2.9 [CI95%: 1.4–6.0], $p=0.004$], age [aOR=0.9 [95% CI: 0.91 to 0.99], $p=0.026$], HLA-B27 status [aOR=2.5 [CI95%: 1.2–5.3], $p=0.02$], ASDAS-CRP score [aOR=1.6 [95% CI: 1.1 to 2.4], $p=0.016$], and MRI sacroiliitis [aOR=2.0 [CI95%: 1.0–4.2], $p=0.054$]. Response to NSAIDs was not significantly associated to the response to the TNFi [aOR=1.93 [95% CI: 0.6 to 6.3], $p=0.275$]. The IPW aOR confirmed the non-association between NSAIDs good response and TNFi good response: 1.60 [CI95%: 0.7–3.3], $p=0.20$.

Conclusions: The good response to NSAIDs according to the Amor's criteria does not seem to be an independent predictive factor of a good response to the first TNFi in early axSpA patients.

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OP0023

EFFECT OF ANTERIOR UVEITIS, PSORIASIS AND INFLAMMATORY BOWEL DISEASE ON DRUG-SURVIVAL FOR TNF-INHIBITORS IN ANKYLOSING SPONDYLITIS

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Background: Tumour necrosis factor inhibition (TNFi) is the mainstay treatment for ankylosing spondylitis (AS) with a high disease activity. Whereas disease activity and sex have been shown to affect drug-survival for TNFi in AS, the impact of typical AS-comorbidities, such as anterior uveitis, psoriasis and inflammatory bowel disease (IBD), on the drug-survival of TNFi in AS is less well understood.

Objectives: To determine the impact of comorbidity with anterior uveitis, psoriasis and IBD on drug-survival in patients with AS starting treatment with a first TNFi.

Methods: Swedish biologics-naïve patients with AS starting a 1st TNFi July 1 2006 – December 31 2015, were identified in the Rheumatology Quality Register, and followed from treatment start until treatment discontinuation. Censoring occurred at the first of: December 31 2015, death, emigration, or loss of follow-up. Comorbidities, and potential confounders, were identified through linkage to six other national registers.

We calculated survival curves and hazard ratios (HRs) for each comorbidity and the risk of TNFi discontinuation. HRs were adjusted for sex, age, CRP, peripheral arthritis, type of TNFi and BASDAI at baseline. Additional models were also adjusted for other chronic morbidities (cardiovascular disease, affective disease, diabetes, malignancies, chronic lung disease and chronic kidney failure), and for socioeconomic status (length of education, household income, sick-leave, country of birth and civil status), respectively.

Results: 2577 patients (71% men) were identified, 27% had a previous history of anterior uveitis, 7% IBD and 6% psoriasis. A history of anterior uveitis was associated with a lower risk of TNFi discontinuation (HR 0.72; 0.62–0.83), whereas presence of psoriasis was associated with an increased risk (HR 1.48; 1.18–1.86). No association was found between presence of IBD and risk of TNFi discontinuation. Models adjusting for disease activity, morbidities, and socioeconomic status resulted in an attenuated association for psoriasis (table 1). The impact of each comorbidity on drug-survival is visualised in figure 1.

Abstract OP0023 – Table 1. HR for association between inflammatory comorbidities and risk of discontinuation of TNFi treatment in AS

Comorbidity	A	B	C	D
Anterior uveitis	0.72 (0.62–0.83)	0.71 (0.60–0.84)	0.72 (0.61–0.85)	0.75 (0.62–0.90)
Psoriasis	1.48 (1.18–1.86)	1.35 (1.04–1.76)	1.28 (0.98–1.67)	1.33 (0.99–1.78)
IBD	0.91 (0.71–1.16)	0.84 (0.65–1.09)	0.82 (0.63–1.07)	0.83 (0.62–1.10)

A)univariable; B-D) adjusted for baseline measures; C) also adjusted for other morbidity and D) also adjusted for socioeconomic status.

Abstract OP0023 – Figure 1 Survival probability (Kaplan-Meier plots) for persisting on a first-line TNFi, for patients with ankylosing spondylitis, dependant on having a history of (A) anterior uveitis, (B) psoriasis or (C) inflammatory bowel disease.

Conclusions: Comorbidities typically associated with AS affect TNFi drug survival, but in different directions. Possible explanations include a differential effect of TNFi on these morbidities themselves, or other inherent differences in the AS inflammatory phenotype. The association with psoriasis decreased after adjustment, suggesting the influence of other factors. The role of inflammatory comorbidities in determining response and persistence of TNFi should be further examined.

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OP0024

WHAT HAPPENS TO PATIENTS WITH ANKYLOSING SPONDYLITIS WHO START A FIRST BIOLOGIC? A SWEDISH STUDY OF TREATMENT TRAJECTORIES IN CLINICAL PRACTICE

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Background: Clinical trials have shown that 40%–50% of patients with ankylosing spondylitis (AS) achieve a good response ($\geq 40\%$ improvement) to treatment with tumour necrosis factor inhibitors (TNFi). By contrast, observational studies indicate that less than two out of three patients continue their first biological disease modifying anti-rheumatic drug (bDMARD) up to two years. During the past six years, five TNFi and one IL-17-inhibitor have been marketed for AS in Sweden. Yet, an assessment of long-term treatment trajectories in AS is lacking. In particular, there is a need for a better understanding of the proportion of patients who fail their first bDMARD, when this happens, and which drugs become their ensuing treatments.

Objectives: To describe contemporary five-year treatment trajectories for patients initiating a first ever bDMARD in AS, and to explore whether the dose of bDMARD is gradually tapered in those patients who remain stable on their bDMARD treatment.

Methods: Swedish patients with AS starting a first ever bDMARD in 2010–2015 were identified in the Swedish Rheumatology Quality Register (SRQ). At the end of each full year (1–5 years) after treatment start, the treatment status of each patient was determined. Censoring occurred at the first of: death, emigration, 31 December 2015 or loss to follow-up. In addition, data on collected prescriptions for the subcutaneous bDMARDs were retrieved from the National Prescribed Drug Register. For patients remaining on their first subcutaneous bDMARD, according to SRQ, the proportion of patients collecting $\geq 75\%$ of the yearly 'defined daily doses' (DDD) at a pharmacy during each full year was determined.

Results: 1698 patients with AS starting a first bDMARD in the study-period were identified, all of which were TNFi. After the end of the first year, 74% remained on their first bDMARD and after five years 38% (figure 1). Of the 72% of patients who remained on any bDMARD after five years, 85% were on their first or second bDMARD. After five years 24% were not on any DMARD and around five percent of the patients discontinuing bDMARD treatment did so due to low disease activity or remission. Among those remaining on their first subcutaneous bDMARD the proportion collecting $\geq 75\%$ of the yearly DDD gradually decreased from 88% to 56% over the 5 years of follow-up (black line in figure 1).

Abstract OP0024 – Figure 1 Proportion of patients with ankylosing spondylitis on each treatment after 1–5 years of follow-up from initiating their first bDMARD

Conclusions: While a minority of patients with AS remain on their first-line bDMARD after 5 years of treatment, most are still on any bDMARD. Further, despite a low rate of discontinuation due to remission, one fourth of patients no longer use any bDMARDs treatment after 5 years. Patients remaining stable on treatment gradually but substantially decrease their use over time.

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OP0025

IDENTIFICATION OF PREDICTORS OF STRUCTURAL DAMAGE PROGRESSION IN THE SACROILIAC JOINTS IN PATIENTS WITH EARLY AXIAL SPONDYLOARTHRITIS ON A LONG-TERM ANTI-TNF TREATMENT

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Background: Several observational studies showed a low, but still detectable progression of structural damage in the sacroiliac joints (SIJ) in patients with axial spondyloarthritis (axSpA) over 2 to 5 years. Few predictors of progressions, such as elevated C-reactive protein (CRP) and active inflammation on magnetic resonance imaging (MRI), have been identified, mostly in patients not treated with TNF inhibitors. To date, it is not clear whether these predictors also work in patients treated with anti-TNF agents and whether anti-TNF therapy is able to retard such a progression.

Objectives: To evaluate the radiographic progression in the sacroiliac joints (SIJ) and to identify predictors of such a progression during long-term (up to six years) treatment with tumour necrosis factor (TNF) blocker etanercept in patients with early axSpA.

Methods: In the ESTHER trial¹ a total of 76 patients with early (up to 5 years symptom duration) and active axSpA were randomised to be treated with either etanercept or sulfasalazine for one year. Between year 1 and year 6, all patients who continued in the study were treated with etanercept. X-rays of SIJ were collected at baseline and every 2 years thereafter. Two trained readers, who were blinded for all clinical data, scored independently the SIJ x-rays in a concealed and randomly selected order, according to the grading system of the modified New York (mNY) criteria (grade 0 to 4). Patients with bilateral sacroiliitis of grade ≥ 2 or unilateral of grade ≥ 3 were classified as radiographic axSpA (r-axSpA), and as non-radiographic axSpA (nr-axSpA) otherwise. The sacroiliitis sum score (0–8) was calculated as a sum of means of both readers for the left and right SIJ. Active and chronic inflammatory changes on MRI of SIJ were assessed at baseline, year 2 and year 4 according to the Berlin MRI scoring system. A longitudinal mixed model analysis was performed to identify predictors of the radiographic sacroiliitis progression.

Abstract OP0025 – Table 1. Longitudinal mixed model analysis of the association between structural damage in the sacroiliac joints (sacroiliitis sum score) and disease-related parameters in patients with early axSpA treated with etanercept for up to 6 years.

Disease-related parameters	Univariable analysis β (95% CI)	Model 1 β (95% CI)	Model 2 β (95% CI)
Elevated CRP (>5mg/l)	0.44 (0.13 – 0.75)	-	0.45 (0.11 – 0.78)
SIJ osteitis score on MRI (0-24)	0.05 (0.01 – 0.08)	0.04 (0.01 – 0.08)	-
Age, years	0.00 (-0.02 – 0.01)	-0.00 (-0.02 – 0.01)	-0.00 (-0.02 – 0.01)
Male sex	-0.03 (-0.35 – 0.27)	-0.18 (-0.53 – 0.16)	0.00 (-0.33 – 0.33)
HLA-B27 positivity	-0.07 (-0.47 – 0.32)	-0.08 (-0.55 – 0.38)	-0.08 (-0.54 – 0.36)
Treatment duration with ETN, years	-0.04 (-0.13 – 0.03)	-0.03 (-0.15 – 0.09)	-0.01 (-0.12 – 0.09)
Symptoms duration, years	-0.01 (-0.07 – 0.05)	0.01 (-0.07 – 0.11)	0.01 (-0.07 – 0.10)

axSpA, axial spondyloarthritis; CRP, C-reactive protein; CI, confidence interval; ETN, etanercept; MRI, magnetic resonance imaging, SIJ, sacroiliac joints.

Results: Totally, 55 patients with axSpA contributing with 159 SIJ radiographs were included in the analysis. At baseline, 19 patients were classified as r-axSpA and 36 as nr-axSpA based on the independent SIJ reading. Radiographic progression from nr- to r-axSpA was observed in 5 (18%) patients between baseline and year 2. Progression decelerated to 4.1% between year 2 and 4, and no further progression was observed up to year 6. The mean $\pm$ SD change of sacroiliitis sum score was 0.13 ± 0.73 , -0.26 ± 0.76 and -0.09 ± 0.67 , in the time intervals baseline-year 2, year 2 year 4, and year 4 year 6, respectively. In the longitudinal mixed model analysis, elevated CRP (model 2) and osteitis on MRI (model 1) were independently and significantly associated with a higher sacroiliitis sum score (table 1).