 patients for 3 domains (TWP1, absenteeism, presenteeism), and in PsA patients for 2 domains (absenteeism, activity impairment).

Conclusions: Golimumab s. c. 1 x monthly is an effective treatment in patients with RA, AS and PsA. All scores of the WPAI showed a significant (p<0.05) reduction in mean score values in each indication. Golimumab leads to an improvement of work productivity and daily activities in all patients already within the first 3 months of treatment and provided sustained improvement in WPAI in patients with RA, PsA and AS.

Disclosure of Interest: I. Klaudius Employee of: MSD Sharp & Dohme GmbH, K. Krueger: None declared, S. Remstedt: None declared, A. Thiele: None declared


SAT0188 TOCILIZUMAB IN EARLY RHEUMATOID ARTHRITIS DELIVERS CLINICAL AND ULTRASOUND-CONFIRMED RAPID AND DEEP REMISSION WITH ABOLITION OF PD – TREMERA STUDY

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Background: Tocilizumab (TCZ) has shown impressive outcomes in early RA (ERA) with clinical remission rates of up to 80%. The speed and depth of remission of TCZ in treatment-naive ERA have not been specifically evaluated.

Objectives: To evaluate the rate and depth of clinical and imaging response and remission, and timing of optimal response in ERA.

Methods: A prospective, open-label, RCT of active (DAS28 $>$3.2), new-onset (symptom duration ≤12 months) treatment-naive RA (2010 ACR/EULAR RA classification criteria), randomised 1:1, and treated with either TCZ 8 mg/kg (4-wkly) monotherapy or TCZ 8 mg/kg (4-wkly) and methotrexate (MTX) combination for 48 weeks.

Clinical response/remission rates, and patient reported outcomes (PRO) were evaluated at early (wks 4 and 12) timepoints, and wks 24 and 48. High resolution ultrasound (US) was performed at baseline, wks 12, 24 and 48. Odds ratios were calculated using Firth logistic regression.

Results:

- 20 pts [16 female; 13 RF+, 15 ACPA+; mean(SD) age 55.25(12) years] were recruited; baseline mean(SD) DAS28-ESR 5.98(1.21), HAQ 1.64(0.67).
- High-hurdle endpoints: at wk4: 30%, 95% and 35% and 95% achieved DAS28-ESR rem, EULAR and ACR50 response respectively; and continued to increase, peaking at wk48 with 67%, 93% and 63% respectively [OR wk4 1.0, 0.3, 0.7 and wk48 1.4, 3.0, 4.0 respectively]. Sustained DAS28-ESR remission (8 successive weeks) was observed in 40%; chisq=0.5, p=0.462; mean (90% CI) time to remission 38.3 (33.2, 43.3) wks.

PRO: Baseline median(IQR) VASGH 56.5(29,71.5) improved by wk4 to 24 (14.54), maintained wk48 24.5(3.5,46.5); VASDA from 63.5(42,77) to wk4 19.5(4,53), maintained wk48 17.5(2.5,49.5). Similarly, median(IQR) HAQ at baseline 1.63(1.25,2.13) decreased by wk4 1.13(0.19,2.0) and wk48 0.75 (0.19,1.38).

Ultrasound: Baseline mean grey scale (GS) and power Doppler (PD) (as well as DAS28-ESR) are shown in the figure below, illustrating rapid reduction by wk4 that continued throughout the study period to all achieving PD=0 by wk48. Baseline median (IQR) erosions 0 (0, 0) remained unchanged throughout study. Baseline GS and PD appeared to associate with achievement of DAS44ESR remission (p=0.038 and p=0.043 respectively).

No meaningful numerical differences between the two treatment arms were observed. These data indicate convincing patient-relevant, imaging-determined depth of remission in a new-onset, treatment-naive RA cohort.

REFERENCE:

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SAT0189 DYNAMICS OF CIRCULATING TNF DURING ADALIMUMAB TREATMENT OF RHEUMATOID ARTHRITIS USING A NOVEL DRUG-TOLERANT TNF ASSAY

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Background: Tumor necrosis factor- (TNF) inhibitors are effective in the treatment of rheumatoid arthritis (RA); these include adalimumab, which binds TNF to form inactive complexes. Once in remission, a proportion of patients can successfully discontinue adalimumab treatment, indicating that blocking TNF is no longer necessary for disease control. We developed a novel assay that can quantify TNF in the presence of large amounts of TNF-inhibitor, i.e. a ‘drug-tolerant’ assay.

Objectives: To investigate, for the first time, the relationship between TNF levels and disease course during adalimumab treatment.

Methods: The new drug-tolerant competition enzyme-linked immunosorbent (ELISA) assay was used to quantify TNF levels on initiation and during 2 years of adalimumab treatment in 206 consecutive RA patients. The relationship between TNF levels and clinical response was evaluated.

Results: Circulating TNF levels were close to the detection limit at baseline, but TNF levels increased on average >50-fold upon adalimumab treatment (figure 1A; black lines show median (IQR)), and reached a stable level in time in the majority of patients (figure 1B; representatives of n=206), regardless of disease activity. During treatment, TNF was in complex with adalimumab, and recovered as inactive 31 adalimumab:TNF complexes. Low TNF levels at week four were associated with a higher frequency of anti-drug antibodies (ADAs) at subsequent time points (figure 1C) and significantly less methotrexate (MTX) use at baseline. Furthermore, week four TNF levels were significantly correlated with SDAI score, with significantly lower TNF levels in patients who did not reach remission (Spearman r = -0.18; p=0.015; figure 1D).

Conclusions: TNF levels, mostly in complexed form, do not appear to decline in patients that reach remission, and may therefore not be predictive for treatment...