Abstract THU0688 – Figure 1 Hazard. Ratios for Incidence of T2D in Pts With RA on Different DMARDs.

(HR>1 favours abatacept, HR<1 favours other therapies)

Conclusions: Pts with RA treated with abatacept had greater risk factors for T2D at baseline. However, the adjusted risk of new-onset T2D was lower among abatacept pts versus other bDMARDs.

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Disclosure of Interest: E. Alemao Shareholder of: Bristol-Myers Squibb, Employee of: Bristol-Myers Squibb, Z. Guo Employee of: Bristol-Myers Squibb, L. Ferri Employee of: Bristol-Myers Squibb, L. Burns Employee of: Bristol-Myers Squibb

DOI: 10.1136/annrheumdis-2018-eular.2033

THU0689

VARIABILITY OF PAIN LEVELS IS EXPLAINED BY SELF-REPORTED DISEASE ACTIVITY IN RHEUMATOID ARTHRITIS AND AXIAL SPONDYLOARTHRITIS: A 3-MONTHS PROSPECTIVE STUDY OF 165 PATIENTS

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Background: Pain is a central characteristic of rheumatoid arthritis (RA) and axial spondyloarthritis (axSpA), but it is multifactorial. Some patients describe high fluctuation of pain intensity, but fluctuation of pain and its association with disease activity has been little investigated.

Objectives: To compare the variability of pain and of self-reported disease activity in patients with RA or axSpA through repeated assessments, and to describe clinical characteristics of patients with high variability of pain.

Methods: Data were extracted from a prospective cohort study (ActConnect) including patients with clinician-confirmed RA or axSpA owning a smartphone.¹ Baseline characteristics were collected. Over 3 months, weekly self-assessments of pain and patient global assessment of disease activity on 0–10 points numerical rating scales (NRS) were obtained. For each patient, the mean of the inter-assessment differences of NRS (average of absolute change – AAC) of pain and disease activity were calculated. RA and AxSpA patients were compared by t-test. High variability was defined as the upper tercile of AAC. Pearson's correlation was used to evaluate the correlation between variability of pain and of self-reported disease activity. Univariate and multivariate logistic regression compared patients with high vs low variability of pain, without imputation of missing data, on R, including weekly self-reported flare over follow-up.

Results: Eighty-six patients with RA and 79 with axSpA were included, mean age was respectively 48.7 (SD 12.7) and 41.7 (SD 10.2) years, mean disease duration was 8 (SD 8.8 and 8.6 respectively) years, 81% and 44% respectively were female. Mean DAS28 and BASDAI were respectively 2.27 (SD 1.18) and 3.21 (SD 2.06) at baseline; 44% and 62% respectively of patients were receiving a biologic.

Mean levels of pain and patient global assessment of disease activity were non-significantly higher in AxSpA than RA patients (table 1) and variability of pain and disease activity were also higher in AxSpA (table 1). Correlation between AAC of pain and activity was 72% in RA, and 84% in AxSpA. In multivariate analyses, self-reported flares were the only determinant of pain AAC (Odds ratio 2.25 [1.27–4.38] p=0.01 and 3.26 [1.67–6.35] p<0.001 respectively for RA and axSpA).

Abstract THU0689 – Table 1

	RA	AxSpA	p value
Baseline pain	2.90 (SD 2.36)	3.40 (SD 2.30)	0.16
AAC of pain	1.02 (SD 0.74)	1.20 (SD 0.70)	0.10
Baseline PGA of disease activity	3.09 (SD 2.27)	3.73 (SD 2.48)	0.08
AAC of PGA of disease activity	0.98 (SD 0.67)	1.22 (SD 0.70)	0.02

Conclusions: In this population of overall well-controlled patients, close repeated assessments of pain showed relatively low fluctuations of pain in RA and axSpA (around 1 point on a 0–10 scale). Patients with axSpA reported slightly more pain, self-reported disease activity and fluctuations of these outcomes, than patients with RA. Fluctuations in pain were highly correlated to fluctuations in patient global assessment of disease activity, indicating significant overall between these 2 patient-reported outcomes. Self-reported flares were the main element explaining fluctuations in pain, confirming the validity of self-reported flares.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.4938

THU0690

RETROSPECTIVE ANALYSIS OF TEMPORAL ARTERY BIOPSY AND ITS IMPACT IN THE MANAGEMENT OF GIANT CELL ARTERITIS

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Background: Prevalence of Giant cell arteritis (GCA) as per primary care data is 17/10000 for patients>55 years of age¹. British society of rheumatology (BSR) guidelines recommend that suspected cases of GCA should be started on treatment with high dose corticosteroids immediately and then referred for temporal artery biopsy². Temporal artery biopsy has diagnostic yield around 13%³. We have done a retrospective analysis of patients who had temporal artery biopsy in order to assess the compliance with BSR guidelines and assess the diagnostic yield.

Objectives: To assess the yield of temporal artery biopsy and its impact in the management of Giant cell arteritis

Methods: The medical records of patients who had temporal artery biopsy from September 2015 to August 2017 were retrospectively reviewed. We documented demographics, management pre and post biopsy, and timeframes between referral, treatment and biopsy. Data was searched through the clinic letters, referral letters and pathology results.

Results: Total number of patients identified was 45. Among them 23 (51%) patients had typical clinical features of GCA and 21 (49%) had atypical features as per the referral letter. Age distribution found was as following: 50 years and below-3; 51 to 60 years- 7; 61 to 70 years- 15; 71 to 80 years-12; 81 to 90 years- 7 and above 90 years-1. Timeframe between biopsy referral and biopsy was as following: within 1 week-27; between 1–2 weeks- 10; between 2–3 weeks- 6 and more than 3 weeks –1. Ophthalmologists performed biopsy for 37 patient and vascular surgeons for 8 patients. Referral was made by primary care for 7 patients, acute medicine for 12 patients, neurology for 3 patients and by rheumatologist for 23 patients. Treatment was initiated in 37 (82%) patients prior to biopsy. Biopsy was positive in 11 (25%) patients, negative in 30 (66%) patients and 4 (8%) showed healed inflammatory changes (HIC). Age distribution of biopsy positive patients was as following - age 61 to 70 years - 4; age 71 to 80 years - 4; age 81 to 90 years - 3. Out of the 11 biopsy positive patients 7 were started on treatment prior to the biopsy and remaining 4 were initiated on treatment after the results. The distribution of duration of treatment before biopsy was as following - within 1 week - 2; between 1 to 2 weeks - 3; between 2 to 3 weeks - 2.

Conclusions: - Majority of the patients (80%) had temporal artery biopsy done within 2 weeks of referral

- Diagnostic yield of temporal artery biopsy was found nearly twice the existing data

- Biopsy was positive in the age group between 61 to 90 years
- Positive biopsy was seen up to 3 weeks of treatment with high dose corticosteroids
- Duration of treatment pre biopsy and age group appear to impact the diagnostic yield of temporal artery biopsy.

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Acknowledgements: Department of rheumatology, Solihull hospital, Heart of England NHS trust

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.4937

THU0691

ASSOCIATION BETWEEN PERIODONTITIS AND CLINICAL RESPONSE IN RHEUMATOID ARTHRITIS PATIENTS UNDER BIOLOGICAL TREATMENT

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Background: Previous studies showed that periodontitis (PD) was a propagation factor for the severity of rheumatoid arthritis (RA) and our previous epidemiological study revealed that PD was associated with discontinuation risk of etanercept.

Objectives: To investigate the association between PD and the risk of 3 month clinical non-response using the Disease Activity Score (DAS)-based European League Against Rheumatism (EULAR) response criteria in RA patients under biological therapy.

Methods: We enrolled 111 RA patients treated with biologics, including etanercept (n=16), adalimumab (n=44), golimumab (n=7), tocilizumab (n=23), abatacept (n=14), and rituximab (n=7). A qualified periodontist performed the periodontal assessment, and the 3 month clinical response was determined DAS-based EULAR response criteria. We quantified the association between PD and the risk of non-response by calculating odds ratios (ORs) with 95% confidence intervals (CIs) using the logistic regression analysis, after adjusting for confounders including age, sex, tobacco use, RA disease duration, biologic treatment duration, rheumatoid factor and anti-citrullinated peptide antibody, erythrocyte sedimentation rate and C-reactive protein, concurrent medication, and diabetes.

Results: Of 111 RA patients, 83 (74.8%) had PD. 37 (44.6%) of PD patients received periodontal treatment within three months. After adjusting for potential confounders, PD patients had a higher risk of non-response to treatment than non-PD patients (OR, 4.20; 95% CI, 1.06–16.68; p=0.041). Compared with non-PD patients, the risk of non-response was significantly greater in PD patients who did not receive periodontal therapy (OR, 5.12; 95% CI, 1.16–22.56; p=0.031), but not in PD patients who received periodontal therapy (OR, 3.28; 95% CI, 0.72–15.06; p=0.126). Among those who were under tumour necrosis factor inhibitor therapy (n=67), the risk of clinical non-response was markedly higher in those with PD (OR, 9.65; 95% CI, 1.33–70.04; p=0.025), particularly in those who did not receive periodontal therapy (OR, 14.39; 95% CI, 1.59–130.38; p=0.018).

Significant predictor of 3 month non-response	Univariable analysis OR (95% CI)	Multivariable	
		Model A	Model B
Periodontitis			
No	Reference:	Reference:	Reference:
Yes	1.39 (0.56–3.45)	4.20 (1.06–16.68)	
Not treated	1.77 (0.66–4.74)		5.12 (1.16–22.56)
Treated	1.01 (0.36–2.90)		3.28 (0.72–15.06)
RA duration, incremental year	1.03 (1.00–1.06)	1.13 (1.04–1.24)	1.13 (1.03–1.23)
Baseline DAS28	1.00 (0.86–1.15)	1.64 (0.99–2.73)	1.69 (1.01–2.82)
Baseline ESR	1.06 (1.02–1.09)	1.06 (1.004–1.11)	1.06 (1.003–1.11)

Conclusions: In RA patients under biological therapy, an increased risk of clinical non-response to treatment was observed in patients with PD, especially among those who did not receive periodontal treatment.

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Acknowledgements: The authors would like to thank the Biostatistics Task Force of Taichung Veterans General Hospital, Taichung, Taiwan, ROC for statistical support.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.1104

THU0692

RISK OF END-STAGE RENAL DISEASE REQUIRING DIALYSIS IN ANKYLOSING SPONDYLITIS PATIENTS STARTING MEDICAL THERAPY: A NATIONWIDE, POPULATION-BASED, COHORT STUDY

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Background: From the year 2000 Taiwan has had the highest incidence and prevalence of end-stage renal disease (ESRD) among the regions investigated by the US Renal Data System. Also, previous studies had suggested a possible association between IgA nephropathy and ankylosing spondylitis (AS) because an increased prevalence of microscopic hematuria and a higher proportion of elevated serum IgA levels were found in AS patients. However, whether the risk of ESRD was increased in treated AS patients or not is still unknown.

Objectives: To examine the risk of ESRD requiring dialysis in patients with AS who started medical therapy.

Methods: Using 2003–2012 claims data from the Taiwanese National Health Insurance Research Database, we identified 38,259 AS patients who received at least 3 courses of AS-related medical therapy (i.e., non-steroidal anti-inflammatory drugs, methotrexate, salazopyrine or corticosteroid) and started therapy from 2005 to 2012. The first date of medical therapy was defined as the index date. After excluding those who had a history of chronic renal disease (ICD9-CM 585, 586) or receiving dialysis before the index date, we identified 37 070 newly-treated AS cases. We randomly selected 3 70 700 non-AS individuals matching (1:10) AS cases for age, sex and the year of the index date without a history of chronic renal failure or dialysis before the index date. After adjusting for age, sex, moderate to severe renal disease, diabetes mellitus, hypertension, and annual cumulative defined daily dose (cDDDs) of traditional non-steroidal anti-inflammatory drugs (NSAIDs), selective cyclooxygenase-2 inhibitors (COX2i), and preferential COX2i, we calculated the adjusted hazard ratios (aHRs) with 95% confidence intervals (CIs) using the Cox proportional hazard model to quantify the risk of ESRD in AS patients compared with non-AS controls. We re-selected 6,621 AS patients and 6621 non-AS subjects by further matching (1:1) for cDDDs of three groups of NSAIDs to re-estimate the aHRs for ESRD.

Results: 51 (0.14%) of 37,070 AS patients and 1,17 (0.38%) of non-AS individuals developed ESRD after a follow-up of 1 58 846 and 1,707,757 person-years respectively. The aHR for ESRD was 0.4 (0.30–0.54) in AS patients compared with non-AS individuals. However, after further matching for cDDDs of NSAIDs, the aHR of ESRD was 0.80 (0.34–1.86). Significant risk factors included diabetes mellitus, hypertension, renal disease, and use of COX2i.

Conclusions: The risk of ESRD was not significantly different between treated AS patients and age, sex, index date, and NSAIDs use matched non-AS individuals.

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Acknowledgements: The authors would like to thank the Biostatistics Task Force of Taichung Veterans General Hospital, Taichung, Taiwan, ROC for statistical support.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.4095