- 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 OR (95% CI)	Model B OR (95% CI)
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