	Treatment Period	
	0 to ≤52 Weeks	APR-Exposure Period 0 to ≥156 Weeks Cumulative Events*
	APR30 n=1,905 Pt-Yrs=1,524.5	APR30 n=1,905 Pt-Yrs=3,527.5
	EAIR/100 Pt-Yrs	EAIR/100 Pt-Yrs
Major adverse cardiac events§		
Acute myocardial infarction	0.1	0.1
Myocardial infarction	0.1	0.1
Subarachnoid hemorrhage	0.1	0.1
Cardiac arrest	0.0	0.1
Cerebral infarction	0.0	0.1
Malignancies		
Hematologic	0.0	0.1
Non-melanoma skin cancer	0.9	0.5
Solid tumors‡	0.3	0.4
Serious infections	0.7ǂ	1.0#
Pneumonia	0.1	0.1
Urinary tract infection	0.1	0.1
Appendicitis	0.1	0.1
Diverticulitis	0.1	0.1
Sepsis	0.0	0.1
Bronchitis	0.0	0.1

*Each patient's total exposure is defined as the time interval between the date of the first and last dose of APR30, regardless of when treatment was initiated, through February 2015.
§No adjudication of major adverse cardiac events for the APR-exposure period.
‡Including malignant melanoma.
ǂSerious infections occurring in patients included pneumonia (n=2), urinary tract infection (n=2), appendicitis (n=1), and diverticulitis (n=1).
#Serious infections occurring in ≥2 patients included pneumonia (n=5), appendicitis (n =3), bronchitis (n=3), diverticulitis (n=2), sepsis (n=2), and urinary tract infection (n=2).
APR30=apremilast 30 mg BID; EAIR=exposure-adjusted incidence rate; PBO=placebo; Pt-Yrs=patient-years.

Conclusions: Incidence of MACE, malignancies, and SIs was low in pts with psoriasis and PsA receiving APR30 for ≥156 wks. No new safety signals or SOIs were observed over time with APR30.

Disclosure of Interest: A. Kavanaugh Grant/research support from: Abbott, Amgen, AstraZeneca, BMS, Celgene Corporation, Centocor-Janssen, Pfizer, Roche, and UCB, M. Augustin Consultant for: AbbVie, Almirall, Amgen, Biogen, Boehringer Ingelheim, Celgene Corporation, Centocor, Eli Lilly, GSK, Janssen-Cilag, LEO Pharma, Medac, Merck, MSD, Novartis, Pfizer, UCB, and XenoPort, Speakers bureau: AbbVie, Almirall, Amgen, Biogen, Boehringer Ingelheim, Celgene Corporation, Centocor, Eli Lilly, GSK, Janssen-Cilag, LEO Pharma, Medac, Merck, MSD, Novartis, Pfizer, UCB, and XenoPort, E. Lespessailles Grant/research support from: Amgen, Celgene Corporation, Eli Lilly, and Novartis, Speakers bureau: Amgen, Celgene Corporation, Eli Lilly, and Novartis, K. Papp Grant/research support from: AbbVie, Allergan, Amgen, Anacor, Astellas, Baxalta, Biogen-Idec, Boehringer Ingelheim, Bristol-Myers Squibb, Celgene Corporation, Dermira, Dow Pharma, Eli Lilly, Galderma, Genentech, Glenmark, GSK, Janssen, Kyowa Hakko Kirin, LEO Pharma, MedImmune, Merck, Merck Serono, Mylan, Novartis, Pfizer, Regeneron, Roche, Sanofi-Aventis, Stiefel, Takeda, UCB, and Valeant, Consultant for: AbbVie, Akros, Allergan, Amgen, Anacor, Astellas, AstraZeneca, Baxalta, Baxter, Biogen-Idec, Boehringer Ingelheim, Bristol-Myers Squibb, Can-Fite BioPharma, Celgene Corporation, Celtic, Cipher, Dermira, Dow Pharma, Eli Lilly, Formycon, Forward Pharma, Funxional Therapeutics, Galderma, Genentech, Genexion, Glenmark, GSK, Janssen, Kyowa Hakko Kirin, LEO Pharma, Lypanosys, MedImmune, Merck, Merck Serono, Mitsubishi Pharma, Mylan, Novartis, Novommune, Pan Genetics, Pfizer, Regeneron, Roche, Sanofi-Aventis, Stiefel, Sun Pharma, Takeda, UCB, Valeant, Vertex, and Xoma, M. Paris Employee of: Celgene Corporation, R. Chen Employee of: Celgene Corporation, D. Gladman Grant/research support from: AbbVie, Amgen, BMS, Celgene Corporation, Janssen, Novartis, Pfizer, and UCB, Consultant for: AbbVie, Amgen, BMS, Celgene Corporation, Janssen, Novartis, Pfizer, and UCB, D. Pariser Grant/research support from: Abbott Laboratories, Amgen, Astellas Pharma US, Asubio Pharmaceuticals, Basilea Pharmaceutical, Celgene Corporation, Dow Pharmaceutical Sciences, Eli Lilly, Galderma Laboratories, Graceway Pharmaceuticals, Intendis, Johnson & Johnson, Novartis Pharmaceuticals, Novo Nordisk A/S, Ortho Dermatologics, Photocure ASA, Stiefel Laboratories, and Valeant Pharmaceuticals, K. Peris Consultant for: Eli Lilly, LEO Pharma, MEDA, and Roche

DOI: 10.1136/annrheumdis-2017-eular.5065

AB0744 DOES TIMING OF INITIATION OF ANTI-TNF AGENTS AFFECT THE QUALITY OF LIFE OUTCOMES IN PATIENTS WITH PSORIASIS AND PSORIATIC ARTHRITIS?

P.W. Barnes[1], N. Rich-Garg[2], D. Choi[3], A. Deodhar[2]. *[1]School of Medicine; [2]Division of Arthritis & Rheumatic Diseases; [3]Public Health & Preventative Medicine, Oregon Health and Science University, Portland, United States*

Background: Psoriatic Arthritis (PsA) affects up to 30% of people with psoriasis[1]. While Tumor Necrosis Factor inhibitors (TNFi) are effective agents for PsA, the relationship between early treatment and patient reported outcomes in a real world setting has not been reported previously.

Objectives: To assess whether timely treatment with TNFi leads to better improvement in quality of life outcomes than delayed treatment.

Methods: This was a retrospective analysis of patients with PsA and/or Psoriasis (PsO) using TNFi with or without methotrexate, and who had a minimum of 2 visits at the Center of Excellence for Psoriasis and Psoriatic Arthritis at our university. Detailed demographic and clinical characteristics of this cohort have been published previously[2]. Demographics, quality of life measures (e.g. Routine Assessment of Patient Index Data – RAPID3, Psoriasis Quality of Life – PQoL12, Short Form 12 – SF-12), and clinical data (percent of body surface area involved with PsO – BSA%) were collected from patient-reported questionnaires and electronic medical records. Only those patients who had a chronological overlap of treatment exposure and QoL measures such as RAPID3, BSA, SF12 and PQOL were included. To ascertain treatment effects, a mixed-effects model was fitted to estimate the trend of each QoL outcome of a patient separately. Then, for all estimated trends of an outcome, a linear regression model was employed to explore the association between the magnitude of estimated trends and timeliness of TNFi treatments.

Results: The quality of life measures were not affected by how early after the disease onset TNFi treatment was started (in other words, no statistically significant associations between the effectiveness of TNFi treatment and disease duration) for RAPID3 (p=0.285), SF-12 (p=0.674), or BSA (p=0.078). For PQoL, there was a significant association between the trend of treatment effects and timeliness of treatment. A day of delay into treatment was resulted in a reduction of 4.4×10^{-4}/day in the trend of PQoL scores (p=0.007).

Conclusions: In this sample of PsA & PsO patients, timing of starting TNFi in patients with PsA had significant impact on improvements in the PQoL but not other quality of life measures such as RAPID3, SF-12 and BSA. A relatively short treatment history might have led to the negative correlations.

References:

[1] McLaughlin M, et.al. Early treatment of psoriatic arthritis improves prognosis. Practitioner 2014 258 (1777) 21–24.
[2] Truong B et.al. Demographics, clinical disease characteristics and quality of life in a large cohost of psoriasis patients with and without psoriatic arthritis. Clin, Cosm and Inv Dermatology 2015: 8: 563–569.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.1535

AB0745 SUBCLINICAL ATHEROSCLEROSIS EVOLUTION IN PSORIATIC ARTHRITIS PATIENTS TREATED WITH ANTI-TNF ALPHA: 5 YEARS FOLLOW UP

A. Ortolan[1], M. Puato[2], G. Boschetti[3], M. Lorenzin[1], A. Hoxha[1], L. Punzi[1], R. Ramonda[1]. *[1]Rheumatology Unit, Department of Medicine DIMED, University of Padova; [2]Clinica Medica III, Department of Medicine DIMED, Universityof Padova; [3]Clinica Medica III, Department of Medicine DIMED, University of Padova, Padova, Italy*

Background: Psoriatic arthritis (PsA) is associated with increased morbility and mortality and an accelerated atherosclerosis. Influence of anti-TNFalpha treatment (a widely used therapy in PsA) in subclinical atherosclerosis is still unclear.

Objectives: The aim of this study was to evaluate subclinical atherosclerosis progression before, during and after 5 years of anti-TNFalpha treatment.

Methods: Twenty-seven consecutive PsA patients were evaluated before TNF blockers therapy (T0), after 2 years (T1) and after 5 years (T2) of treatment. Subclinical atherosclerosis was evaluated through carotid duplex scanning, analyzing intima-media thickness (IMT) and flow-mediated dilation (FMD). IMT values were expressed as IMT mean (cumulative mean of all the IMT mean in every analyzed carotid segment) and M-MAX (cumulative mean of all the higher IMT in every analyzed carotid segment). Response to therapy was studied by the evaluation of tender and swollen joints (Tj and Sj), DAS 28 (disease activity score), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Metrologic and metabolic data were collected. For the statistical evaluation of parameters over time (T0 vs T1, T1 vs T2) Student's T test for paired data was used.

Results: From T0 to T1 a deterioration in IMT-mean and M-MAX ($p<0.01$) was

Table

	T0	T1	T2
IMT-mean	0.72±0.15	0.91±0.37*	0.92± 0,34
M-MAX	0.89±0.18	1.06±0.39*	1.10±0.35**
FMD	5.40±1.93	5.37±1.66	5.40±1.89
Tender joints (n)	8.10±5.56	2.09±2.32*	1.72± 2.05
Swollen joints (n)	3.85±3.84	0.25±0.72*	0.50±0.92
CRP	11.25±9.16	2.91±1.72*	2.73 ±2.51
DAS28	4,16±0.67	2.30±0.82*	2.40±0,9

Legend: IMT= intima-media thickness; M-MAX= cumulative mean of all the higher IMT in every analyzed carotid segment; FMD= flow-mediated dilation; CRP= C Reactive Protein; DAS28=Disease Activity Score 28joints; *= p<0.01 with respect to the previous determination; **=p<0.05 with respect to the previous determination

noted. At T2 IMT-mean remained stable, M-MAX worsened further ($p<0.05$). No significant variation in FMD was observed (Table). Noteworthy, from T0 to T2 systolic blood pressure and Body Mass Index remained stable (p=ns), while diastolic blood pressure decreased (p=0,001). A good response to PsA treatment was confirmed by a significant decrease (T0 vs T1) in Tj, Sj, DAS 28 and CRP ($p<0.01$); treatment efficacy was preserved from T1 to T2 (p=ns) (Table).

Conclusions: Our data revealed that in patients with PsA, despite treatment with TNF blockers, there is still a gradual, albeit slight progression of subclinical atherosclerosis assessed by ultrasonography. Other inflammatory mechanisms not related to TNF may be responsible of the progression in atherosclerotic disease.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.5545

AB0746 PRELIMINARY RESULTS OF A TWO-YEAR FOLLOW UP OF SUBCLINICAL ATHEROSCLEROSIS IN PATIENTS WITH PSORIATIC ARTHRITIS

C. Vergara-Dangond[1], M. Robustillo-Villarino[1], V. Núñez-Monje[1], M. Aguilar-Zamora[1], L. Montolio-Chiva[1], E. Valls-Pascual[2], A. Ybáñez-García[1], Á. Martínez-Ferrer[2], J.J. Alegre-Sancho[1]. [1]*Rheumatology;* [2]*Hospital Universitario Dr. Peset, Valencia, Spain*

Background: Psoriatic arthritis (PsA) is a disease associated with an increased cardiovascular (CV) risk, due to early atherosclerosis, which is comparable to a rheumatoid arthritis population. However, there is a lack of studies that evaluate the progression of subclinical atherosclerosis over a year in these patients.

Objectives: To explore the progression of the vascular damage by different techniques in patients with PsA and the factors related with these changes.

Methods: Pre-post study with analytical components. 44 patients with PsA (CASPAR criteria) and peripheral joint involvement of more than one year since diagnosis were consecutively included. We gathered demographic (age, gender, BMI), clinical (traditional CV risk factors, previous CV event), and analytical variables (atherogenic index [AI], GFR [MDRD], fibrinogen, glycated hemoglobin, CRP, ESR, ultrasensitive CRP, apoB/apoA1 ratio) and basal CV risk was estimated with SCORE tool. Other variables were collected retrospectively from patients electronic medical record. The extracranial branches of carotid artery were explored by ultrasonography (US) using an Esaote MyLab70XVG with a 7–12 MHz linear transducer and an automated program measuring intima-media thickness (IMT) through radiofrequency (^{RF}QIMT), and the presence of atheroma plaques, as per the Mannheim consensus, was registered. Pulse wave velocity (PWV) was determined, as an arterial stiffness marker, by a validated MobilOGraph® device. Patients were followed during a 2-year period between may 2014 until december 2016. All of the tests were repeated after 2 years. Statistical analysis was performed using SPSS 17.0 software.

Results: We analyzed 38 patients, excluding those with high CV risk (previous CV event, GFR<60mg/dl, and/or type II or type I diabetes with organ affection) and followed during 2 years. At baseline, the mean and median of age was 59,2 and 60,5 years (39–88), respectively, mostly women (65,8%). The median BMI was 28 (17–35). 28,9% were smokers and 36,8% had hypertension. 26,3% received glucocorticoids, 57,9% NSAIDs, 84,2% DMARDs and 31,6% biologic therapies. The median CRP, ESR and DAS 28 were 5mg/L (1–19,1), 7mm/h (2–28) and 2,17 (1,24–3,7), respectively. The median SCORE was 1 (0–7), the PWV was 8 m/s (5,6–13,5) and basal IMT was 728 μ(462–1087); the presence of atheroma plaques was detected in 35,1% of the patients.

After 2 years, plaque appearance was seen in 15% more of patients, as well as worsening of PWV and IMT in 56,8% and 38,9% of patients, respectively.These changes were not significant. No patient developed a CV event.

In the bivariate analysis, PWV progression at 2 years related with advanced age ($p<0,002$), and with elevated SCORE ($p<0,044$), and showed a tendency with higher arterial systolic pressure (ASP). We also observed a tendency to an association between plaque appearance, age and ASP; as well as lower apolipoprotein A1 levels with IMT.

Conclusions: The progression over time of the vascular damage in patients with PsA relates with traditional CV risk factors. These preliminary results must be confirmed in a posterior analysis with a greater number of patients.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.3002

AB0747 THE EFFECT OF CERTOLIZUMAB PEGOL ON RADIOGRAPHIC PROGRESSION OVER 4 YEARS OF TREATMENT IN PATIENTS WITH PSORIATIC ARTHRITIS

D. van der Heijde[1], R. Fleischmann[2], J. Wollenhaupt[3], A. Deodhar[4], L. Bauer[5], B. Hoepken[5], L. Peterson[6], P.J. Mease[7]. [1]*Leiden University Medical Center, Leiden, Netherlands;* [2]*UT Southwestern Medical Center and Dallas Metroplex Clinical Research Center, Dallas, United States;* [3]*Schön Klinik, Hamburg, Germany;* [4]*Oregon Health and Science University, Portland, United States;* [5]*UCB Pharma, Monheim, Germany;* [6]*UCB Pharma, Raleigh;* [7]*Swedish Medical Center and University of Washington, Seattle, United States*

Background: The RAPID-PsA trial (NCT01087788) investigated the efficacy and safety of certolizumab pegol (CZP) in patients (pts) with psoriatic arthritis (PsA). It demonstrated that CZP treatment inhibits radiographic progression over 96 weeks (wks).[1]

Objectives: We report the long-term effect of CZP treatment on radiographic progression in pts with PsA over 4 years.

Methods: The RAPID-PsA phase 3 trial was double-blind and placebo-controlled to Wk24, dose-blind to Wk48, and open-label (OL) to Wk216. Pts had active PsA and had failed ≥1 DMARD. Pts randomized to CZP (200mg Q2W or 400mg Q4W, following 400mg loading dose at Wks0, 2, 4) continued their assigned dose in the OL period. Radiographs taken at baseline (BL), and at Wks96, 168, 216, were read in a single reading campaign using the modified Total Sharp Score (mTSS) for PsA by 2 readers, blinded to patient information and time point sequence. The mean of the scores of the 2 readers was used. Outcomes reported are the least squares (LS) mean mTSS score, change from BL (CFB) in mTSS score, and the percentage of pts assessed for radiographic damage who achieved mTSS non-progression (defined either as CFB in mTSS score ≤0.5 or ≤0), for all Wk0 CZP-treated pts, irrespective of dose regimen. mTSS score and CFB were estimated using Mixed Model Repeated Measures (MMRM) estimates; proportions of pts with radiographic non-progression are presented as observed case.

Results: 409 PsA pts were randomized, of whom 273 received CZP from Wk0. The LS mean BL mTSS score was 16.0 and there was little increase from BL in mTSS score to Wk216 (Table). Amongst those who completed the study to Wk216, the majority of CZP-treated pts achieved radiographic non-progression to Wk216, both with non-progression defined as CFB in mTSS score ≤0.5 or ≤0 (Table). The change in LS mean mTSS score over time for Wk0 CZP-treated pts was consistently low throughout the trial: 0.14 (95% CI: 0.02–0.26) per 48 wks from BL to Wk96, and 0.18 (95% CI: 0.08–0.28) per 48 wks from Wk96 to Wk216.

Table: mTSS score and radiographic non-progression to Week 216 for patients treated with CZP from Week 0

	Week 0 CZP dose combined (N=273)			
	Week 0	**Week 96**	**Week 168**	**Week 216**
mTSS score, LS mean (SE) [95% CI] [a]	16.0 (2.2) [11.6–20.4]	16.2 (2.2) [11.8–20.6]	16.6 (2.3) [12.1–21.0]	16.7 (2.3) [12.2–21.1]
CFB in mTSS score, LS mean (SE) [95% CI] [a]	–	0.3 (0.1) [0.0–0.5]	0.6 (0.2) [0.3–1.0]	0.7 (0.2) [0.3–1.1]
Non-progression rate, n (%) [b]				
Patients with CFB in mTSS ≤0.5, n/N (%)	–	180/214 (84.1)	158/196 (80.6)	145/186 (78.0)
Patients with CFB in mTSS ≤0, n/N (%)	–	157/214 (73.4)	131/196 (66.8)	121/186 (65.1)

[a] Mixed Model Repeated Measures (MMRM) estimates; [b] Observed case; percentage of patients with non-progression, as a proportion of the number assessed for progression. CFB: change from baseline; LS: least squares; SE: standard error.

Conclusions: There was little radiographic progression in CZP-treated PsA pts, as measured by mTSS, throughout the 4-year RAPID-PsA trial.

References:

[1] Mease P. RMD Open 2015;1:e000119;doi:10.1136/rmdopen-2015–000119.

Acknowledgements: This study was funded by UCB Pharma. We thank the patients and their caregivers in addition to the investigators and their teams who contributed to this study. Editorial services were provided by Costello Medical Consulting.

Disclosure of Interest: D. van der Heijde Consultant for: AbbVie, Amgen, Astellas, AstraZeneca, BMS, Boehringer Ingelheim, Celgene, Daiichi, Eli Lilly, Galapagos, Gilead, Janssen, Merck, Novartis, Pfizer, Regeneron, Roche, Sanofi, UCB Pharma, Employee of: Director of Imaging Rheumatology B.V., R. Fleischmann Grant/research support from: Genentech Inc, Roche, Abbott, Amgen, UCB Pharma, Pfizer, Bristol-Myers Squibb, Eli Lilly, Sanofi-Aventis, MSD Pharmaceuticals, Novartis, AstraZeneca, Janssen, Consultant for: Roche, Abbott, Amgen, UCB Pharma, Pfizer, Bristol-Myers Squibb, Eli Lilly, Sanofi-Aventis, Novartis, AstraZeneca, Janssen, J. Wollenhaupt Grant/research support from: UCB Pharma, Consultant for: UCB Pharma, A. Deodhar Grant/research support from: AbbVie, Amgen, Eli Lilly, GlaxoSmithKline, Janssen, Novartis, Pfizer, UCB Pharma, Consultant for: Eli Lilly, Janssen, Novartis, Pfizer, UCB Pharma, L. Bauer Employee of: UCB Pharma, B. Hoepken Employee of: UCB Pharma, L. Peterson Employee of: UCB Pharma, P. Mease Grant/research support from: (Abbott) AbbVie, Amgen, Bristol-Myers Squibb, Celgene, Janssen, Eli Lilly, Novartis, Pfizer, Sun, UCB Pharma, Consultant for: (Abbott) AbbVie, Amgen, Bristol-Myers Squibb, Celgene, Crescendo, Corrona, Dermira, Janssen, Eli Lilly, Merck, Novartis, Pfizer, Sun, UCB Pharma, Zynerba, Speakers bureau: (Abbott) AbbVie, Amgen, Bristol-Myers Squibb, Celgene, Crescendo, Genentech Inc, Janssen, Novartis, Pfizer, UCB Pharma

DOI: 10.1136/annrheumdis-2017-eular.1961