SAT0372 CHANGE OF SUBCLINICAL ATHEROSCLEROSIS AFTER FIVE YEARS ANTI-TNF TREATMENTS IN PSORIATIC ARTHRITIS

Abdulsamet Erden[1], Uğur Canpolat[2], Oğuz Abdullah Uyaroğlu[3], Cem Çöteli[2], Levent Kiliç[1], Ali Akdoğan[1], Umut Kalyoncu[1], Omer Karadag[1], Ali İhsan Ertenli[1], Sedat Kiraz[1], Kudret Aytemir[2], Şule Apraş Bilgen[1]. *[1]Hacettepe University Faculty of Medicine, Department of Internal Medicine, Division of Rheumatology, Ankara, Turkey; [2]Hacettepe University Faculty of Medicine, Department of Cardiology, Ankara, Turkey; [3]Hacettepe University Faculty of Medicine, Department of Internal Medicine, Ankara, Turkey*

Background: Although cardiovascular (CV) diseases are very common in inflammatory arthritis like psoriatic arthritis (PsA), long-term impact of medication on CV outcomes is lacking.
Objectives: The aim of our study was to evaluate the long-term effects of anti-TNF-a drugs on subclinical atherosclerosis assessed by the flow-mediated dilatation (FMD) and carotid intima media thickness (IMT).
Methods: A total of 30 patients with PsA according to classification of psoriatic arthritis (CASPAR) criteria1 and 28 healthy controls were enrolled in this cross-sectional study between June 2011-July 2012. 22 out of 30 PsA patients completed the study. Demographic data (sex, age), PsA and psoriasis duration, joint pattern (monoarthritis, oligoarthritis or polyarthritis) and other PsA involvements (nail, enthesis, dactilytis) were noted. Tender joint count, swollen joint count and disease activity score (DAS)-28 were used for joint activity assessment. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were used for acute phase reactants. Sex and age matched healthy controls were selected as the control group for this study. Flow mediated dilatation (FMD) from brachial artery and carotid intima media thickness (IMT) were measured by an experienced cardiologist both at initial and 5-year follow-up visits.
Results: Psoriasis duration of PsA patients was 180±114 months. PsA disease duration was 108±33 months. The mean duration between two evaluations was 62±9 months. At first evaluation, 14 (63.6%) patients had peripheral joint, 1 (4.5%) patient had axial, and 7 (31.8%) patients had both peripheral and axial involvement among patients. Dactilytis in 6 (27.3%), enthesis in 7 (31.8%) and nail in 12 (54.5%) patients were other clinical involvements. FMD% was lower in PsA patients than healthy controls [9.3±3.9 vs 12.9±1.8, p<0.001] and carotid IMT was more obvious in PsA patients than healthy controls [0.64±0.17 vs 0.54 ±0.09, p=0.017] (Figure). All PsA patients used anti-TNF alpha treatment during the follow-up period. 68.1% of the PsA patients were in remission during the control. At 5-year follow-up visits, there was no CV event in study groups. However, FMD% was lower in PsA patients than healthy controls [7.6±4.8 vs 12.9±1.8, p<0.001] and carotid IMT was also similar between PsA patients and healthy controls [0.61±0.33 vs 0.54±0.09, p=0.306]. After 5-year follow-up visits, there was no statistically significant difference in FMD% compared to baseline [p = 0.254]. ΔFMD% was found to be moderately correlated with the ΔBASDAI (r= -0.45).
Conclusion: Our results showed that there was a significant impact of anti-TNF-a drugs on progression of subclinical atherosclerosis at the vascular wall level, but no impact on the endothelial dysfunction. Further large-scale randomized studies are needed to confirm our findings.

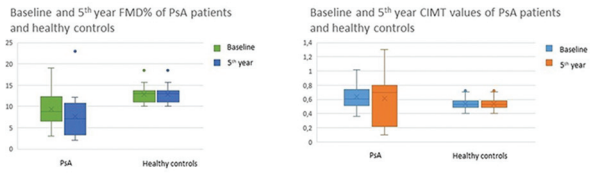

Figure.

Disclosure of Interests: Abdulsamet Erden: None declared, Uğur Canpolat: None declared, Oğuz Abdullah Uyaroğlu: None declared, Cem Çöteli: None declared, Levent Kiliç: None declared, Ali Akdoğan: None declared, Umut Kalyoncu Grant/research support from: MSD, Roche, UCB, Novartis and Pfizer, Consultant for: MSD, Abbvie, Roche, UCB, Novartis, Pfizer and Abdi Ibrahim, Speakers bureau: MSD, Abbvie, Roche, UCB, Novartis, Pfizer and Abdi Ibrahim, Omer Karadag: None declared, Ali İhsan Ertenli: None declared, Sedat Kiraz: None declared, Kudret Aytemir: None declared, Şule Apraş Bilgen: None declared
DOI: 10.1136/annrheumdis-2019-eular.6044

SAT0373 EFFECT OF FILGOTINIB ON PATIENT-REPORTED OUTCOMES IN ACTIVE PSORIATIC ARTHRITIS: RESULTS FROM EQUATOR, A RANDOMIZED, PHASE 2 STUDY

Laura C. Coates[1], Philip J. Mease[2], Dafna D. Gladman[3], Filip van den Bosch[4], Chantal Tasset[5], Luc Meuleners[5], Robin Besuyen[6], Jingjing Gao[7], Mona Trivedi[7], Thijs Hendrikx[6], Philip Helliwell[8]. *[1]University of Oxford, Oxford, United Kingdom; [2]Swedish Medical Center/Providence St Joseph Health and University of Washington, Seattle, United States of America; [3]University of Toronto and Krembil Research Institute, Toronto Western Hospital, Toronto, Canada; [4]Ghent University Hospital, Ghent, Belgium; [5]Galapagos NV, Mechelen, Belgium; [6]Galapagos BV, Leiden, Netherlands; [7]Gilead Sciences, Inc., Foster City, United States of America; [8]Leeds Institute of Rheumatic and Musculoskeletal Medicine, Leeds, United Kingdom*

Background: Filgotinib (FIL) is an oral, selective Janus kinase 1 inhibitor under clinical investigation in a number of inflammatory diseases. FIL significantly improved multiple disease domains vs placebo (PBO) in patients with active psoriatic arthritis (PsA) in the multicenter, double-blind, phase 2 EQUATOR trial (NCT03101670) [1].
Objectives: To evaluate the effect of FIL vs PBO on patient reported outcomes (PROs) in EQUATOR and the extent to which effects on composite disease endpoints translate to clinically relevant improvements for patients.
Methods: Patients were randomized 1:1 to FIL 200 mg or PBO once daily for 16 weeks [1]. Patient's Global Assessment of Disease Activity (PtGADA), pain intensity (visual analog scale), Pruritus Numerical Rating Scale (NRS), Health Assessment Questionnaire Disability Index (HAQ-DI), 36-Item Short Form Survey (SF-36) Physical Component Summary (PCS) and Mental Component Summary (MCS), and Functional Assessment of Chronic Illness Therapy-Fatigue scale (FACIT-F) were assessed at week 16. Analysis of covariance was used to compare changes from baseline in outcomes between groups. Proportions of patients achieving normative PRO scores or minimal clinically important differences (MCIDs) were compared using Cochran-Mantel-Haenszel tests [2, 3].
Results: FIL significantly improved multiple PROs vs PBO at week 16 (Table). Proportions of patients reaching normative PRO values for FACIT-F and SF-36 PCS (≥40 or 50, respectively), and achieving MCIDs in HAQ-DI and SF 36 PCS, were significantly greater for FIL vs PBO (Table). Significant improvement in 6/8 SF 36 domains was observed at week 16 with FIL vs PBO (Fig a). Improvement in most individual FACIT F items was also observed (Fig b).
Conclusion: In EQUATOR, FIL-treated patients with active PsA reported greater and clinically meaningful improvements in most PROs at week 16 vs PBO, mirroring improvements previously reported with FIL in disease activity measures [1].

Table

Table

Week 16		FIL (n=65)	PBO (n=66)	Treatment difference (95% CI)	p
Mean change from baseline					
PtGADA (mm)		−27.2 (22.1)	−13.5 (25.8)	−15.2[a] (−22.3, −8.1)	<0.0001
Pain (mm)		−31.6 (21.3)	−11.1 (29.7)	−18.9[a] (−26.7, −11.1)	<0.0001
Pruritus NRS		−2.5 (2.1)	−0.6 (2.2)	−2.2[a] (−3.1, −1.4)	<0.0001
HAQ-DI		−0.6 (0.5)	−0.3 (0.5)	−0.3[a] (−0.4, −0.1)	0.0009
SF-36 PCS		7.4 (6.6)	2.4 (6.6)	4.7[a] (2.6, 6.8)	<0.0001
SF-36 MCS		4.3 (8.3)	3.2 (9.2)	1.2[a] (−1.7, 4.0)	0.4128
FACIT-F		8.2 (7.3)	5.5 (8.1)	3.2[a] (0.8, 5.5)	0.0086
Response rate, n/N (%)					
HAQ-DI	MCID ≥0.35	41/63 (65)	26/62 (42)	23.2[b] (5.7, 38.8)	0.0085
SF-36 PCS	Score ≥50	11/64 (17)	4/63 (6)	10.9[b] (−0.7, 22.5)	0.0471
	MCID ≥2.5	49/65 (75)	26/66 (39)	36.0[b] (19.2, 50.0)	<0.0001
SF-36 MCS	Score ≥50	13/46 (28)	14/47 (30)	−1.5[b] (−19.4, 16.6)	0.9879
	MCID ≥2.5	32/65 (49)	40/66 (61)	−11.4[b] (−27.4, 5.5)	0.2607
FACIT-F	Score ≥40	18/58 (31)	7/57 (12)	18.7[b] (3.6, 33.0)	0.0105
	MCID ≥4	43/65 (66)	37/66 (56)	10.1[b] (−6.5, 25.9)	0.1921

[a]Least-squares mean. [b]Arithmetic mean.
CI, confidence interval

REFERENCES

[1] Mease P, et al. Lancet 2018;392:2367–77.
[2] Mease P, et al. J Rheumatol 2011;38:2461–5.