higher in the male group. Also, there is a tendency towards more effective inhibition of radiographic progression in men.

Disclosure of Interests: None declared

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AB0246

COMORBIDITIES APPEARING UNDER BIOLOGIC THERAPY: PREVALENCE AND ASSOCIATED FACTORS

T. El Joumani¹, T. Latifa¹, I. Hmamouchi^{2,3}, S. Ahid⁴, R. Abouqal⁵, L. Achemlal⁶, I. El Bouchti⁷, A. El Maghraoui⁸, I. Ghoulani⁹, H. Hassikou¹⁰, T. Harzy¹¹, I. Linda¹², O. Mkini¹³, R. Niamane¹⁴, R. Bahiri¹⁵, H. Rkain¹, F. Allali¹. ¹El Ayachi Hospital, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco, Department of Rheumatology B, Rabat, Morocco; ²Faculty of Medicine and Pharmacy, Mohammed V University, Rabat, Morocco, Laboratory of Biostatistical, Clinical and Epidemiological Research, Rabat, Morocco; ³Provincial Hospital of Temara, Morocco, Department of Rheumatology, Rabat, Morocco; ⁴Mohammed V University, Rabat, Morocco, Research Team of Pharmacoeconomics & Pharmacoepidemiology, Rabat, Morocco; ⁵Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco, Laboratory of Biostatistical, Clinical and Epidemiological Research, Rabat, Morocco; ⁶Military Hospital Mohammed V, Ibn Sina University Hospital, Rabat, Morocco, Department of Rheumatology, Rabat, Morocco; ⁷Arrazi University Hospital, Marrakech, Morocco, Department of Rheumatology, Rabat, Morocco; ⁸Private Medical Office, Rabat, Morocco, Rheumatology, Rabat, Morocco; ⁹University Hospital of Agadir, Morocco, Department of Rheumatology, Agadir, Morocco; ¹⁰Military Hospital Moulay Ismail, Hassan II University Hospital, Meknes, Morocco, Department of Rheumatology, Meknes, Morocco; ¹¹Hassan II University Hospital, Fes, Morocco, Department of Rheumatology, Fes, Morocco; ¹²Mohammed VI University Hospital, Department of Rheumatology, Oujda, Morocco; ¹³Ibn Rochd University Hospital, Department of Rheumatology, casablanca, Morocco; ¹⁴Military Hospital Avicenne, Mohammed VI University Hospital, Department of Rheumatology, marrakech, Morocco; ¹⁵El Ayachi Hospital, Ibn Sina University Hospital, Department of Rheumatology A, Salé, Morocco

Objectives: The aims of our study are to determine the new comorbidities appearing under biologic therapy, their prevalence, and the factors implicated in their appearance.

Methods: It's a multicentric historical-prospective cohort including 10 rheumatology departments of Moroccan University Hospitals. The data were collected from the national register of patients under biologic therapy supervised by the Moroccan Society of Rheumatology. An electronic follow-up questionnaire is completed every 6 months by the investigator.

Results: The study included 418 patients: 224 with Rheumatoid Arthritis (RA) who represented 53.6% and 194 with spondyloarthropathy (SP) who represented 46.4%.

The prevalence rate of comorbidities appearing after one year of treatment with biologic therapy was 15.7% in RA and 8.4% in SP. The rate of cardiovascular diseases was 12.9% (arterial hypertension, myocardial infarction and/or ischemic stroke), 6.4% was the same value of hypercholesterolemia and depression, diabetes 3.3%, ulcer 3.2%, and osteoporosis 9.7%. The sedentary rate was 54.8% and smoking was about 3.3%.

No patient had developed hypertriglyceridemia or chronic obstructive pulmonary disease.

In the group of patients with RA, the average age of the patients who had developed a new comorbidity was 51.8 ± 11.3 years, women represented 87.5%, the average of Body Mass Index (BMI) was 27.6 ± 5.9 and the average duration of the disease was 14.1 ± 9.2 years. The disease activity score (DAS28) had an average of 3.15 ± 1.47 .

59.6% of patients used Rituximab, 23.8% Tocilizumab, 8.1% Etanercept, 5.8% Adalimumab, 0.18% Infliximab, and 0.9% Golimumab.

In the group of patients with SP, the average age of the patients who had developed a new comorbidity was 40.2 ± 13.7 years, men represented 63.4%, and the average of BMI was 24.3 ± 4.94 .

The activity of the disease, had an average Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) of 2.62 ± 1.79 and ankylosing Spondylitis Disease Activity Score (ASDAS) of 1.93 ± 1.09 .

Regarding the type of biologic therapy, 33.2% of patients used Etanercept, 30.1% Adalimumab, 24.9% Infliximab, 9.8% Golimumab, 1.6% Secukinumab, and 0.5% Tocilizumab.

Conclusion: Our study showed a high prevalence of cardiovascular disease in patients under biologic therapy. This can be explained by the sedentary lifestyle secondary to rheumatic disease.

Disclosure of Interests: None declared

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Rheumatoid arthritis - non biologic treatment and small molecules

AB0247

ANALYSIS OF THE IMPACT OF TOFACITINIB TREATMENT ON WEIGHT IN PATIENTS WITH RHEUMATOID ARTHRITIS

J. Wollenhaupt¹, J. Morel², C. Daien², A. Ruysen-Witrans³, C. Lukas², C. Richez⁴, A. Shapiro⁵, D. Chapman⁶, M. Cros⁷, J. L. Rivas⁸, G. Citera⁹. ¹Struenseehaus Centre for Rheumatology and Clinical Immunology, Hamburg, Germany; ²CHU Montpellier and University of Montpellier, Department of Rheumatology, Montpellier, France; ³Hopital Purpan, CHU Toulouse, Department of Rheumatology, Toulouse, France; ⁴Groupe Hospitalier Pellegrin-CHU de Bordeaux, Department of Rheumatology, Bordeaux, France; ⁵Pfizer Inc, Inflammation and Immunology, Peapack, NJ, United States of America; ⁶Pfizer Inc, Inflammation and Immunology, New York, NY, United States of America; ⁷Pfizer Inc, Inflammation and Immunology, Paris, France; ⁸Pfizer SLU, Inflammation and Immunology, Madrid, Spain; ⁹Instituto de Rehabilitación Psicosfísica, Section of Rheumatology, Buenos Aires, Argentina

Background: A prior post hoc analysis of tofacitinib clinical trial data reported improvements in rheumatoid arthritis (RA) outcomes with tofacitinib vs placebo (PBO) through Month (M)6, regardless of baseline (BL) body mass index (BMI).¹

Objectives: To assess change from BL (Δ) in BMI, and disease activity by BL BMI status, in patients (pts) with moderate/severe RA receiving tofacitinib through M12.

Methods: This post hoc analysis included data pooled from Phase 3 and 3b/4 studies of pts who were methotrexate-naïve (NCT01039688) or inadequate responders to conventional synthetic (cs) or biologic DMARDs (NCT00960440; NCT00847613; NCT00814307; NCT00856544; NCT00853385; NCT02187055; NCT02831855). Pts received ≥ 1 dose of tofacitinib 5 or 10 mg twice daily (BID) or 11 mg once daily (QD), $\pm$ csDMARDs, or PBO. Least squares (LS) mean Δ BMI (linear mixed model repeated measures; observed cases) was summarised for all treatment groups at M3/6/12 (M3/6 only for tofacitinib 11 mg QD and PBO). Other assessments at M3/6/12 included Δ BMI $\pm$ BL concomitant glucocorticoids (GCs) or antidepressants (descriptive statistics), LS mean Δ DAS28-4(ESR) stratified by BL BMI status (<25 , ≥ 25 – <30 , ≥ 30), and correlations between LS mean Δ BMI and Δ DAS28-4(ESR).

Results: In total, 2349, 1611, 694 and 681 pts received tofacitinib 5 mg BID, 10 mg BID, 11 mg QD or PBO, respectively. Demographics/baseline characteristics were generally similar across treatments, except for some numerical differences in the tofacitinib 11 mg QD group, eg fewer female pts, more White pts and fewer pts receiving concomitant GCs, compared with other treatment groups. At M3/6, LS mean BMI significantly increased from BL with all tofacitinib doses vs PBO (all $p < 0.05$); LS mean Δ BMI was greatest with 10 mg BID and lowest with 11 mg QD (Figure 1a). LS mean Δ BMI was greater in pts receiving tofacitinib as monotherapy vs combination therapy at M3/6/12 (Figure 1b). Δ BMI was generally similar in pts receiving treatment $\pm$ concomitant GCs or antidepressants (data not shown). Improvements in DAS28-4(ESR) were observed in each BL BMI status group at M3/6/12 and were greatest with all tofacitinib doses vs PBO. LS mean Δ DAS28-4(ESR) was generally numerically highest for pts with BMI <25 and numerically lowest for pts with BMI >30 , for all tofacitinib doses. LS mean Δ DAS28-4(ESR) was generally greatest with tofacitinib 10 mg BID and 11 mg QD vs 5 mg BID across BL BMI status groups (Figure 1c). Across treatments, model-adjusted associations between LS mean Δ DAS28-4(ESR) and Δ BMI were weak (correlation coefficients all <0.3 ; Table 1).

Table 1. Correlations between LS mean Δ DAS28-4(ESR) and Δ BMI through M12

	Tofacitinib 5 mg BID			Tofacitinib 10 mg BID			Tofacitinib 11 mg QD			PBO		
	N	Correlation coefficient		N	Correlation coefficient		N	Correlation coefficient		N	Correlation coefficient	
M3	2021	0.1169		1348	0.1240		641	0.0907		554	0.0783	
M6	1918	0.1305		1270	0.1397		611	0.0438		150	0.1556	
M12	1455	0.1213		874	0.1826		-	-		-	-	

Slopes for associations between LS mean Δ BMI and Δ DAS28-4(ESR) were significantly different from 0 at M3/6/12 with tofacitinib 5 and 10 mg BID (all $p < 0.05$). Correlations were analysed by a general linear model method, which included BL age, gender, race and RA duration. For pts receiving tofacitinib 11 mg QD in ORAL Shift (NCT02831855), only data to M6 were included. Pts who advanced from PBO to tofacitinib were not analysed post-advancement-DAS28-4(ESR), Disease Activity Score in 28 joints, erythrocyte sedimentation rate; N, number of pts analysed