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POS0445 **PHYSICIAN AND PATIENT REPORTED EFFECTIVENESS OUTCOMES ARE SIMILAR IN TOFACITINIB AND TNF INHIBITORS IN RHEUMATOID ARTHRITIS PATIENTS: DATA FROM A RHEUMATOID ARTHRITIS REGISTRY IN CANADA**

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Background: Tofacitinib (TOFA) is an oral, small molecule drug used for rheumatoid arthritis (RA) treatment as an alternative option to biologic disease modifying antirheumatic drugs (bDMARDs) including tumor necrosis factor inhibitors (TNFi).

Objectives: We aimed to evaluate physician and patient reported effectiveness outcomes in TNFi compared to TOFA, using real-world data from the Ontario Best Practices Research Initiative (OBRI).

Methods: RA patients enrolled in the OBRI initiating their TOFA or TNFi (Adalimumab, Certolizumab, Etanercept, Golimumab, Infliximab, and Biosimilars) between 1st June 2014 (TOFA approval date in Canada) and 31st Dec 2019 were included. Patients were required to have physician and patient reported effectiveness outcomes data available at treatment initiation and 6-month ($\pm$ 2 months) follow-up. These included clinical disease activity index (CDAI), rheumatoid arthritis disease activity index (RADAI), HAQ-DI, sleep problem, and anxiety/depression scores. Multiple imputation (Imputation Chained Equation, N=20) was used to deal with missing data for covariates at treatment initiation. To deal with confounding by indication, we estimated propensity scores for covariates with an absolute standard difference greater than 0.1 between the two treatment groups.

Results: A total of 419 patients were included. Of those, 226 were initiating a TNFi and 193 TOFA, and had a mean (SD) disease duration of 8.0 (8.7) and 12.6 (9.6) years, respectively. In the TNFi group, 81.9% were female and mean age (SD) at treatment initiation was 56.6 (13.4) years. In the TOFA group, 85% were female and mean (SD) age at treatment initiation was 60.3 (11.2) years. The TNFi group was less likely to have prior biologic use (21.7%) compared to the TOFA group (67.9%). At treatment initiation, physical function measured by HAQ-DI was significantly lower in TNFi compared to the TOFA group (1.2 vs. 1.4).

The rate of CDAI LDA/remission at 6 months was 36.7% and 33.2% in TNFi and TOFA group, respectively. The generalized linear mixed models (GLMM) adjusting for propensity score quantile, showed that there was no significant difference in CDAI LDA/remission (ORs: 0.85, 95% CI: 0.51, 1.43), RADAI (coefficient: 0.48, 95% CI: -0.18, 1.14), HAQ-DI (coefficient: -0.01, 95% CI: -0.18, 0.16), sleep problems (coefficient: -0.25, 95% CI: -0.95, 0.45), and anxiety/depression scores (coefficient: 0.12, 95% CI: -0.35, 0.58) between the two treatment groups (TOFA used as reference).

Conclusion: In this real-world data study, we found that, physician and patient reported effectiveness outcomes are similar in the TNFi and TOFA groups 6 months after treatment initiation in patients with RA.

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POS0446 **FILGOTINIB-TREATED RHEUMATOID ARTHRITIS PATIENTS WITH HIGH BASELINE NEUTROPHIL-TO-LYMPHOCYTE RATIO SHOW BETTER CLINICAL RESPONSE RATES AND PATIENT-REPORTED OUTCOMES**

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Background: Rheumatoid arthritis (RA) is a systemic inflammatory disease which includes increased innate and myeloid immune cell activation. Filgotinib (FIL), an oral JAK1 inhibitor, has shown safety and efficacy in three phase 3 studies (FINCH1-3) in adults with moderately-to-severely active RA. The baseline (BL) neutrophil-to-lymphocyte ratio (NLR) in RA has been associated with a positive response to anti-tumor necrosis factor (TNF) therapy¹ and a negative response to DMARD triple therapy². We previously reported a BL signature of clinical response in the FINCH2 (bDMARD-IR) population which included a neutrophil component³.

Objectives: We conducted a post-hoc analysis to explore whether the BL NLR was associated with response to treatment in the FINCH studies.

Methods: Clinical data of 3273 RA patients (pts) enrolled in the FINCH clinical trials (FINCH3, methotrexate (MTX)-naïve: NCT02886728; FINCH1, MTX-Inadequate Responder (IR): NCT02889796; FINCH2, bDMARD-IR: NCT02873936) were retrospectively analyzed for a relationship between the BL NLR and composite clinical endpoints (ACR-N, DAS28CRP, or CDAI) or PROs (Pain VAS, FACIT Fatigue, HAQ-DI) through week 24. Pts were classified as High or Low BL NLR using a cutpoint (2.7) identified as an independent predictor of treatment failure in a published RA study². Adjusted clinical outcomes were estimated based on mixed effects linear regression models including geographic region and demographics covariates.

Results: 57% of pts enrolled in the FINCH trials were classified as BL NLR-High (Table 1) and FINCH3 NLR-High pts showed higher BL DAS28(CRP). FINCH1 and FINCH3 FIL+MTX-arm NLR-High pts demonstrated significantly better DAS28(CRP) response compared to NLR-Low pts (Figure 1). DAS28(CRP) differences between NLR-High and NLR-Low were detectable as early as Week 2 for FIL200mg + MTX and were sustained through Week 24. FINCH1 and FINCH3 FIL200mg + MTX NLR-High pts also demonstrated sustained clinical and PRO improvements over NLR-Low, including CDAI, ACR-N, Pain VAS, FACIT Fatigue, and HAQ-DI. The strength of these associations was dose-dependent; pts that received FIL100mg + MTX demonstrated weaker but directionally consistent trends in both populations. No significant association between NLR subgroup and clinical efficacy was observed in FINCH2 FIL+MTX-arm pts, FIL-monotherapy (FINCH3) pts, or adalimumab+MTX (FINCH1) pts.

Conclusion: In FINCH1 (MTX-IR) and FINCH3 (MTX-naïve), FIL200mg + MTX-arm NLR-High pts demonstrate better sustained clinical response and PRO scores compared to NLR-Low pts. These data are the first to report an association between the BL NLR and therapeutic response in large randomized RA clinical trials. Future studies on pathobiologies reflected by the NLR biomarker may clarify its potential to guide RA disease management.

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Table 1. Demographics of patients enrolled in FINCH clinical trials by baseline NLR category.

	NLR-Low (NLR < 2.7) (N=1407, 43%)	NLR-High (NLR > 2.7) (N=1866, 57%)	Total (N=3273, 100%)	P-value
Age at enrollment				
Mean (SD)	53.00 (12.74)	53.19 (13.24)	53.11 (13.02)	0.668 ²
Female	1,157 (82.2%)	1,448 (77.6%)	2,605 (79.6%)	0.001 ¹
Seropositivity	1,073 (76.3%)	1,575 (84.4%)	2,648 (80.9%)	< 0.001 ¹
Prior bDMARD #				
Mean (SD)	0.23 (0.73)	0.28 (0.82)	0.26 (0.78)	0.037 ²
Duration of RA				
Mean (SD)	5.93 (7.39)	6.61 (8.07)	6.32 (7.79)	0.013 ²
Baseline oral corticosteroid	537 (38.2%)	895 (48.0%)	1,432 (43.8%)	< 0.001 ¹
Baseline DAS28(CRP)	5.55 (0.90)	5.89 (0.97)	5.74 (0.95)	0.001 ²
Baseline HAQ-DI	1.53 (0.60)	1.62 (0.65)	1.58 (0.63)	0.001 ²

¹Pearson's Chi-squared test. ²Linear Model ANOVA.

Figure 1. Association of BL NLR with DAS28(CRP). Dots represent DAS28(CRP) estimate from a fully adjusted mixed-effects model, shaded area shows the 95% confidence interval. Outlined circles and proximal text show significantly different values between NLR-High and NLR-Low.

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POS0447

PHYSICAL FUNCTION IN PATIENTS WITH RA, STRATIFIED BY SEROSTATUS AND TREATMENT LINE, FOLLOWING SC ABATACEPT: POST HOC ANALYSIS OF AN OBSERVATIONAL, 2-YEAR STUDY CONDUCTED IN ROUTINE CLINICAL PRACTICE (ASCORE)

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Background: RA is characterised by the production of autoantibodies, including RF and anti-citrullinated protein antibodies (ACPAs).¹ Seropositive disease is associated with poorer prognosis in patients with RA,² and response to different treatments has been shown to vary based on ACPA status.³ ASCORE (Abatacept SubCutaneOus in Routine Clinical Practice; NCT02090556) was a 2-year, observational, prospective, multicentre study of SC abatacept for the treatment of RA.⁴

Objectives: This *post hoc* analysis of the ASCORE study evaluated patient-reported outcomes, assessed using HAQ-DI, by RF/ACPA serostatus and treatment line over 24 months of treatment with abatacept.

Methods: Eligible patients, aged ≥18 years, with active moderate-to-severe RA (ACR/EULAR 2010 criteria) who were IV abatacept-naïve and initiated SC abatacept 125 mg once weekly, were enrolled into two cohorts: biologic (b)DMARD-naïve patients and those with ≥1 prior bDMARD treatment failure. This *post hoc* analysis assessed mean change from baseline in HAQ-DI score at 6, 12, 18 and 24 months in response to treatment with abatacept stratified by baseline serostatus (RF/ACPA double positive [+/-]; RF/ACPA single positive [+/-]; RF+/ACPA- or RF-/ACPA+) or RF/ACPA double negative [-/-]) and by line of therapy (all patients, patients receiving abatacept as a first-line bDMARD or as a ≥ second-line bDMARD [data not shown], and those receiving abatacept following 1 [data not shown] or ≥2 prior bDMARDs). Estimates of mean difference with 95% CIs between patients with different serostatus were calculated using a *t*-test for all patients and within different lines of therapy.

Results: Among 2892 eligible patients in ASCORE, 1748 patients with RF/ACPA status available at baseline were included in this analysis (1079 +/-, 326 +/- and 343 -/-). Of these, 791 patients received abatacept as a first-line bDMARD therapy and 957 as a ≥ second-line bDMARD therapy (505 patients had received ≥2 prior bDMARDs). Among all patients, mean change from baseline in HAQ-DI score at 6 months was greater for patients with +/- RA (mean difference [95% CI]: -0.2 [-0.3, -0.0]; *p*=0.0068) or +/- RA (mean difference [95% CI]: -0.2 [-0.3, -0.0]; *p*=0.0315) versus those with -/- RA at baseline (Figure 1). Similarly, mean change (95% CI) in HAQ-DI score at 6 months was greater for patients with +/- RA versus -/- RA among those receiving abatacept as first-line therapy (-0.2 [-0.4, -0.0]; *p*=0.0407) or following treatment with ≥2 bDMARDs (-0.3 [-0.5, -0.0]; *p*=0.0265) (Figure 1). Among patients treated with abatacept following ≥2 prior bDMARDs, mean change in HAQ-DI score was higher among patients with +/- RA versus -/- RA at 18 months (data not shown) and 24 months (Figure 1). No other significant differences were observed by serostatus or line of therapy at any other time points.

Conclusion: Patients with RA who were RF+/ACPA+ at baseline showed an enhanced initial response to abatacept compared with those who were RF-/ACPA-. Over 24 months of treatment in this real-world setting, abatacept was equally effective as a first- or ≥ second-line therapy.

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Figure 1. Mean change from baseline in HAQ-DI score over 6 and 24 months by baseline RF and ACPA serostatus in all patients with RA receiving SC abatacept and by line of therapy

n is the number of subjects with both baseline and post-baseline measurements. Estimates of mean difference with 95% CI between patients with different serostatus (+/-, +/-, or -/-) were calculated using a *t*-test for all patients and within different lines of therapy.

+/- RF/ACPA double positive; +/- RF/ACPA single positive (RF+/ACPA- or RF-/ACPA+); -/- RF/ACPA double negative; ACPA=anti-citrullinated protein antibody; bDMARD=biologic disease-modifying antirheumatic drug.