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OP0021

### PREDICTING SEVERE INFECTION IN REPEAT CYCLES OF RITUXIMAB AND EFFECTS OF HYPOGAMMAGLOBULINAEMIA FOR THE TREATMENT OF RHEUMATIC AND MUSCULOSKELETAL DISEASES

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**Background:** Rituximab (RTX) is effective in treating various rheumatic and musculoskeletal diseases (RMDs). Repeat cycles are often required for disease control but may lead to hypogammaglobulinaemia. Low IgG at baseline has been associated with increased risk of severe infection event (SIE) post-RTX. However, there are limited data on predictors of SIEs in repeat cycles including immunoglobulin levels and B-cell numbers as well as outcomes of hypogammaglobulinaemia.

**Objectives:** To assess predictors of SIEs in repeat RTX cycles and effects of hypogammaglobulinaemia in terms of SIEs rates, humoral response and its persistence post-cessation of RTX.

**Methods:** A retrospective study was conducted in the first 700 consecutive ARD patients treated with at least a cycle of RTX in Leeds. IgM, IgA and IgG levels were measured at baseline and 4-6 months after each cycle. For cycles 2-4 (C2-4), predictors for SIEs were analysed using mixed-effects logistic regression analysis.

**Results:** 550 patients were female, mean(SD) age 56(16) years and median (IQR) disease duration 7.9(3.4-15.0) years. 507(72%) had RA, 94(13%) SLE, 49 (7%) AAV, 14(2%) inflammatory myopathies, 9(1%) pSS, 5(1%) APS, 6(1%) SSC and 16(3%) other CTDs. 364(52%) were biologic-naïve and 514(73%) were on concomitant DMARDs. Total follow-up: 2880 patient-years (PY). 281 SIEs were recorded in 176 patients (9.8/100 PY). In C1, we had validated that low IgG was predictive of SIE within 12 months of C1. For cycles 2-4, in multivariable analysis, non-RTX-specific comorbidities [chronic lung OR (95% CI) 2.4 (1.3-4.4), diabetes 2.9 (1.2-6.9), heart failure 6.3 (1.4-28.1), previous cancer 3.0 (1.3-6.7) and severe infection 6.3 (3.0-13.4)] and RTX-specific variables [higher corticosteroid dose 1.08 (1.02-1.14), higher IgM 1.3 (1-1.7) and longer retreatment time 1.01 (1-1.02)] were associated with increased odds of SIEs, but not B-cell numbers or depletion status. Higher IgG reduced the risk 0.88 (0.8-0.96). Of 103 patients with low IgG for at least 4 months duration, SIEs rates were higher in those with low baseline IgG (16.4 PY) or acquired it during/post-RTX (21.3 PY) versus those with normal IgG (9.7 PY), 5/8(64%) had impaired humoral response to pneumococcal and haemophilus following vaccination challenge and only 4/11(36%) had IgG normalised after switching therapies. Overall, 7(1%) of the patients required Ig replacement based on recurrent sino-pulmonary SIEs and/or low IgG.

**Conclusion:** Immunoglobulin should be monitored at baseline and before each RTX cycle to identify patients at risk of SIEs. Vigilance is needed for those with lower IgG as this is a consistent predictor of SIE and may affect infection outcomes when patients are switched to a different bDMARD. For those at risk of SIEs, reduction of corticosteroid dose could reduce risk. Low B-cell numbers were not predictive of SIEs.

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OP0022

### DO MRI-DETECTED EROSIONS IN PATIENTS WITH CLINICALLY SUSPECT ARTHRALGIA PREDICT PROGRESSION TO RHEUMATOID ARTHRITIS? A LONGITUDINAL STUDY

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**Background:** Radiographic joint erosions are a hallmark of Rheumatoid Arthritis (RA). MRI is more sensitive than radiographs in detecting erosions. It is unknown if MRI-detected erosions are predictive for RA-development in patients with Clinically Suspect Arthralgia (CSA).

**Objectives:** We investigated the prognostic value of MRI-detected erosions (any MRI-erosion, or MRI-erosion characteristics that were recently identified as specific for RA) in CSA.

**Methods:** Patients presenting with CSA (n=491) underwent contrast-enhanced 1.5T MRI of the wrist, metacarpophalangeal (MCP) and metatarsophalangeal (MTP) joints at baseline. MRIs were scored according to RAMRIS. Presence of any MRI-erosion (erosion score  $\geq 1$ ) and RA-specific erosion characteristics as identified previously (grade  $\geq 2$  erosions, erosions in MTP5, erosions in MTP1 if aged <40) were studied with clinically apparent inflammatory arthritis development as outcome (median follow-up 17 months). Analyses were corrected for age, CRP, ACPA and MRI-detected inflammation.

**Results:** Erosions were present in 20.6% of patients. Presence of erosions was not associated with arthritis development (HR multivariable analysis 0.85 (95% CI 0.52-1.40)). Also the different erosion characteristics were not predictive in CSA-patients (grade  $\geq 2$  HR 1.29 (95% CI 0.40-4.14), erosions in MTP5 HR 0.89 (95% CI 0.38-2.09) and MTP1 if aged <40 HR 0.98 (95% CI 0.23-4.21)). MRI-erosions were more prevalent in ACPA-positive than in ACPA-negative patients (32.3% versus 18.8%, p=0.02). However, no association with arthritis development was observed in both subgroups.

**Conclusion:** MRI-detected erosions in hands and feet of patients with CSA were not predictive for arthritis development. These data warn against overinterpretation of MRI-detected erosions in CSA.

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LB0001

### EFFICACY AND SAFETY OF FILGOTINIB FOR PATIENTS WITH RHEUMATOID ARTHRITIS WITH INADEQUATE RESPONSE TO METHOTREXATE: FINCH PRIMARY OUTCOME RESULTS

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**Background:** Filgotinib (FIL) is an orally administered, potent and selective inhibitor of Janus kinase 1 (JAK1) that has shown good efficacy and was well tolerated for treatment of rheumatoid arthritis (RA).

**Objectives:** To evaluate efficacy and safety of FIL treatment in patients with RA who have had an inadequate response to methotrexate (MTX).

**Methods:** This phase 3, double-blind, active- and placebo (PBO)-controlled study randomized patients with active RA (3:3:2:3) to FIL 200 mg, FIL 100 mg, active comparator (adalimumab [ADA] 40mg every 2 weeks), or PBO daily for up to 52 weeks; results through week 24 are presented. Patients were also receiving MTX for  $\geq 12$  weeks with a stable dose of MTX for  $\geq 4$  weeks before initiation of study drug. Primary efficacy endpoint was proportion of patients achieving ACR20 response at week 12; additional clinical assessments were ACR50 and ACR70 responses, DAS28-CRP score  $\leq 3.2$  and  $< 2.6$ , van der Heijde modified total Sharp score (mTSS), and patient-reported outcomes were HAQ-DI, SF-36 PCS, and FACIT-Fatigue. Safety endpoints included types and rates of adverse events. Logistic regression adjusting for stratification factors with nonresponder imputation was used for superiority test of FIL vs PBO for ACR response and other binary endpoints. Mixed-effect model adjusting for baseline value, stratification factors, treatment, visit, and treatment by visit interaction as fixed effects with observed cases was used for continuous endpoints. Non-inferiority test of FIL to ADA (preserving  $> 50\%$  of ADA response) was performed for DAS28-CRP  $\leq 3.2$  and  $< 2.6$ .

**Results:** Of 1,759 patients randomized, 1,755 received study drug and were analyzed, with 475 FIL 200mg; 480 FIL 100mg; 325 ADA; and 475 PBO, of which 89.5%, 90.4%, 88.9%, and 81.3%, respectively, completed week 24 study drug. Most patients (81.8%) were female, mean (standard deviation [SD]) duration of RA was 7.8 (7.6) years, and mean (SD) DAS28-CRP was 5.7 (0.9). At week 12, significantly more patients in the FIL 200mg and 100mg arms achieved an ACR20 response compared to PBO (Table 1). Similarly, compared to PBO, more patients receiving FIL achieved ACR50 and ACR70 responses, DAS28-CRP scores  $\leq 3.2$  and  $< 2.6$ , had lower radiographic progression, and reported improvements in HAQ-DI, SF-36 PCS, and FACIT-Fatigue scores (Table 1). Non-inferiority of FIL 200mg to ADA was met based on DAS28-CRP  $\leq 3.2$ . The FIL safety profile was consistent with prior studies through week 24 (Table 2).

Table 1. Efficacy at Week 12 (Primary Analysis) and Week 24\*

	FIL 200 mg (N = 475)		FIL 100 mg (N = 480)		ADA 40 mg Q2W (N = 325)		PBO (N = 475)	
	Week 12	Week 24	Week 12	Week 24	Week 12	Week 24	Week 12	Week 24
ACR20, %	76.6***	78.1	69.8***	77.7	70.8	74.5	49.9	59.2
ACR50, %	47.2***	57.9	36.3***	52.7	35.1	52.6	19.8	33.3
ACR70, %	26.3***	36.2	18.5***	29.4	14.2	29.5	6.7	14.9
DAS28-CRP $\leq 3.2$ , %	49.7***	60.6	38.8***	53.1	43.4	50.5	23.4	33.7
DAS28-CRP $< 2.6$ , %	33.9***	48.4	23.8***	35.2	23.7	35.7	9.3	16.2
mTSS, mean change from BL	0.08	0.13***	0.11	0.17***	0.13	0.16	0.25	0.38
HAQ-DI, mean change from BL	-0.69***	-0.82	-0.50***	-0.75	-0.61	-0.76	-0.42	-0.62
SF-36 PCS, mean change from BL	9.2***	10.4	8.5***	10.3	8.4	10.4	5.9	7.7
FACIT-Fatigue, mean change from BL	9.2***	10.5	9.1***	10.8	8.8	10.3	6.8	8.4

\*All patients who were randomized and received at least 1 dose of study drug were included in efficacy analyses. P-values are shown only for primary time points (all at week 12 except mTSS, which was at week 24).  
\*\*\*P < 0.01 vs PBO; \*\*P < 0.05 vs ADA non-inferiority test; \*P < 0.05 vs ADA non-inferiority test; †P < 0.01 vs ADA superiority test; ‡Comparison not adjusted for multiplicity.  
ACR20/50/70, 20%/50%/70% improvement in American College of Rheumatology criteria; ADA, adalimumab; BL, baseline; DAS28-CRP, Disease Activity Score based on 28 joints with Creative Biotech; FIL, filgotinib; FACIT, Functional Assessment of Chronic Illness Therapy; HAQ-DI, Health Assessment Questionnaire Disability Index; mTSS, modified total Sharp score; SD, standard deviation; SF-36 PCS, Short-Form 36 Physical Component Summary; Q2W, every 2 weeks.

Table 2. Safety Events of Interest through Week 24

Patient with event, n (%)	FIL 200 mg (N = 475)	FIL 100 mg (N = 480)	Q2W (N = 325)	PBO (N = 475)
	Serious AEs	21 (4.4)	24 (5.0)	14 (4.3)
Serious infections	8 (1.7)	8 (1.7)	8 (2.5)	4 (0.8)
Herpes zoster	2 (0.4)	2 (0.4)	2 (0.6)	2 (0.4)
Adjudicated MACEs	0	1 (0.2)	1 (0.3)	2 (0.4)
Venous thrombotic events	1 (0.2)	0	0	2 (0.4)
Malignancies	0	1 (0.2)	1 (0.3)	3 (0.6)
Deaths	2 (0.4)	1 (0.2)	0	2 (0.4)

AE, adverse event; MACE, major adverse cardiovascular event

**Conclusions:** The selective JAK1 inhibitor FIL, at doses of 200mg and 100mg led to significant improvement in signs and symptoms of RA, prevented radiographic progression, and improved physical function compared to PBO, and was well tolerated among patients with RA with prior inadequate response to MTX. Efficacy of FIL 200mg was non-inferior to ADA based on DAS28-CRP  $\leq 3.2$ .

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LB0002

### RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED, MULTIPLE-DOSE, PHASE 2B STUDY TO DEMONSTRATE THE SAFETY AND EFFICACY OF TILDRAKIZUMAB, A HIGH-AFFINITY ANTI-INTERLEUKIN-23P19 MONOCLONAL ANTIBODY, IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS

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**Background:** Tildrakizumab (TIL), a high-affinity anti-interleukin-23p19 monoclonal antibody, is approved for moderate-to-severe plaque psoriasis treatment and is under investigation for psoriatic arthritis (PsA).

**Objectives:** To evaluate the 24-week efficacy and safety results from the randomised, double-blind, placebo-controlled, multiple-dose, phase 2b TIL study in patients with active PsA (NCT02980692).

**Methods:** Patients with active PsA were randomised 1:1:1:1:1 to receive TIL (200 mg once every 4 weeks [Q4W] [n = 78], 200 mg every 12 weeks [Q12W] [n = 79], 100 mg Q12W [n = 77], 20 mg Q12W to week 24 [n = 78]), or placebo (PBO) Q4W to week 24 (n = 79). Stable concomitant methotrexate or leflunomide use was permitted but not mandated. The primary efficacy endpoint was the proportion of patients who achieved a 20% reduction from baseline in American College of Rheumatology response criteria (ACR20) at week 24. Other outcome measurements included proportion of patients achieving ACR50/70 response and Psoriasis Area and Severity Index (PASI) 75, PASI 90, and changes in swollen and

Table 1. Demographics and baseline disease characteristics

	TIL 200 mg Q4W (N = 78)	TIL 200 mg Q12W (N = 79)	TIL 100 mg Q12W (N = 77)	TIL 20 mg Q12W (N = 78)	PBO (N = 79)
<b>Patient demographics</b>					
Age, years, median	50.0	49.0	50.0	47.5	47.0
Female, n (%)	46 (59.0)	37 (46.8)	47 (61.0)	41 (52.6)	44 (55.7)
White, n (%)	76 (97.4)	78 (98.7)	75 (97.4)	75 (96.2)	74 (93.7)
BMI, kg/m <sup>2</sup> , median	30.0	29.3	27.8	28.9	27.8
<b>Baseline disease characteristics</b>					
Swollen joint count, median (range)	8.0 (3.0–35)	7.0 (3.0–45)	8.0 (0–38)	8.0 (3.0–38)	8.0 (3.0–42)
Tender joint count, median (range)	13.5 (3.0–64)	15.0 (4.0–63)	19.0 (3.0–59)	14.0 (4.0–54)	15.0 (3.0–64)
BSA $\geq 3\%$ , n (%)	53 (67.9)	44 (55.7)	54 (70.1)	41 (52.6)	42 (53.2)
Physician GADA, mean $\pm$ SD	54.0 $\pm$ 16.1	55.4 $\pm$ 16.2	57.3 $\pm$ 17.3	59.4 $\pm$ 14.4	59.5 $\pm$ 15.6
Patient GADA, mean $\pm$ SD	57.8 $\pm$ 18.3	61.1 $\pm$ 20.7	60.3 $\pm$ 20.2	61.9 $\pm$ 17.4	65.2 $\pm$ 18.1
Patient's pain assessment, mean $\pm$ SD	55.4 $\pm$ 19.1	59.6 $\pm$ 23.5	59.2 $\pm$ 22.1	60.9 $\pm$ 19.7	64.2 $\pm$ 20.4

BMI, body mass index; BSA, body surface area; GADA, global assessment of disease activity; Q4W, every 4 weeks; Q12W, every 12 weeks; PBO, placebo; SD, standard deviation; TIL, tildrakizumab.