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FRI0135 HAVE PREVALENCE OF JOINT SURGERY DECREASED WITH THE USE OF BIOTHERAPY IN RHEUMATOID ARTHRITIS?

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Background: Biological response modifiers have greatly expanded therapeutic arsenal of rheumatoid arthritis (RA) leading to a better control of inflammation, a reduced long-term complications and a prevention of joint damage.
Objectives: Our objective was to assess the impact of use of biologics on joint surgery during RA.
Methods: This is a retrospective study including patients with RA according to American College of Rheumatology (1987) followed- over 15 years period [2000–2014]. We excluded patients who underwent joint surgery without direct relevance to RA. The significance level was set at 0.05.
Results: A total of 500 RA patients (422 women and 78 men) were enrolled in this period. The mean age was 53.3 years (21–83) and the mean disease duration was 12 years (2–40). Rheumatoid factor was positive in 71.4% cases. A high disease activity was noted at diagnosis with a mean disease activity score of 5.90 ±1.38. The mean Health Assessment Questionnaire index was 1.62 [0.2 à 3]. All patients received at least 2 conventional disease-modifying antirheumatic drugs, one of which was methotrexate. Twenty seven per cent of RA patients (135 patients) received biologics: 35 patients received Rituximab (7%) and 100 patients (20%) received anti TNF α (infliximab, etanercept and adalimumab in 10%, 6.8% and 3.2% respectively). The trend curve of biologics use showed a linear increase with spikes of use in 2008, 2011 and 2014. A surgical act was considered necessary in 59 cases (11.8%) mainly total knee arthroplasty (56%). The mean duration between the onset of RA and surgery was 7.02 (1–33). Patients who received biologics had less joint surgery without significant association (p=0.350). The joint surgery showed a decrease in the number of procedures from 2004, concomitantly with promoting biologics.
Conclusions: Our study concluded that joint surgery was less frequent in RA patients who received biologics without a significant association.
Disclosure of Interest: None declared
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FRI0136 PERSISTENCE OF MONOTHERAPY OR COMBINATION THERAPY WITH DISEASE-MODIFYING AGENTS IN PATIENTS WITH PSORIATIC ARTHRITIS IN A REAL-WORLD SETTING

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Background: Until recently, treatment for moderate to severe psoriatic arthritis (PsA) mainly focused on conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) and tumour necrosis factor inhibitors (TNFis). However, the persistence of TNFis alone or in combination with csDMARDs is not well understood.
Objectives: To assess real-word treatment patterns among patients with PsA receiving TNFi monotherapy, csDMARD monotherapy, or TNFi and csDMARD combination therapy.
Methods: This retrospective study utilised data from patients with PsA aged ≥18 years, enrolled in the Corrona PsA registry between March 21, 2013, and July 31, 2017, treated with a TNFi and/or csDMARD (index therapy), and with ≥6 months of follow-up time. Patients were stratified by prevalent (initiation before enrollment) or incident (initiation after enrollment) therapy use; cohorts were based on index therapy: TNFi monotherapy, csDMARD monotherapy, or combination therapy. Outcomes of interest were the percentage of patients who were persistent on their index therapy or had a therapy change (discontinued, switched, or restarted) 12 months after the index visit.
Results: There were 1266 patients in this study: 1144 prevalent and 122 incident (table 1). Patient characteristics at the index date were similar among patients; however, csDMARD monotherapy patients had higher disease activity than either TNFi group. Among prevalent patients, TNFi monotherapy patients were likely to be female (59%) and younger (51.9 years), nearly all patients had psoriasis, and BSA was similar and ≤5. At month 12, among patients with a follow-up visit within the 9–15–month window, the vast majority of prevalent patients and half of incident patients were persistent on their index therapy, and one quarter to one third of incident patients discontinued or switched therapy (table 1).

Characteristic at index date	Prevalent TNFi mono N=421	Prevalent csDMARD mono N=347	Prevalent combo N=376	Incident TNFi mono N=43	Incident csDMARD mono N=56	Incident combo N=23
Sex (female), n (%)	244 (59)	158 (46)	185 (50)	19 (44)	24 (43)	11 (48)
Age, mean (SD)	51.9 (12.3)	57.7 (13.3)	53.9 (11.3)	50.7 (14.0)	55.8 (14.1)	52.3 (13.5)
BMI (obese), n (%)	202 (51)	182 (54)	204 (56)	18 (44)	37 (70)	8 (47)
Current psoriasis, n (%)	366 (93)	300 (90)	312 (90)	38 (93)	49 (94)	15 (68)
HAQ, mean (SD)	0.2 (0.4)	0.3 (0.4)	0.3 (0.4)	0.3 (0.4)	0.3 (0.3)	0.4 (0.6)
CDAI, mean (SD)	9.7 (6.6)	11.6 (9.2)	11.7 (8.4)	12.6 (7.3)	16.2 (8.5)	15.7 (13.9)
DAS28 CRP, mean (SD)	2.4 (0.9)	2.7 (1.0)	2.8 (1.0)	3.0 (0.7)	3.4 (0.8)	2.8 (1.4)
Enthesitis, n (%)	74 (18)	52 (15)	75 (20)	14 (32)	8 (14)	3 (13)
Dactylitis, n (%)	27 (6)	25 (7)	12 (3)	7 (16)	14 (25)	4 (17)
BSA (%), mean (SD)	4.9 (8.7)	4.8 (9.3)	5.0 (11.9)	6.2 (8.8)	5.4 (8.9)	1.3 (2.1)
Nail psoriasis VAS, mean (SD)	5.6 (12.4)	8.1 (22.6)	7.2 (14.3)	11.9 (18.8)	5.9 (15.2)	10.3 (23.3)
History of biologic, n (%)	421 (100)	0	375 (100)	2 (5)	0	0
History of csDMARD, n (%)	192 (46)	347 (100)	369 (98)	13 (30)	0	13 (57)
Treatment pattern, n (%)						
Persistent, n/patients with a follow-up visit within 9-15 month window	233/251 (92.8)	196/225 (87.1)	186/214 (86.9)	13/26 (50.0)	15/35 (42.9)	8/15 (53.3)
Time on drug (mo), mean (SD)	68.6 (46.4)	82.5 (78.1)	49.8 (37.7)	15.9 (5.4)	16.9 (8.7)	16.0 (5.0)
Discontinuation	3 (1.2)	8 (3.6)	17 (7.9)	5 (19.2)	9 (25.7)	4 (26.7)
Switch (to another biologic)	2 (0.8)	2 (0.9)	4 (1.9)	1 (3.8)	4 (11.4)	2 (13.3)
Restart	0	3 (1.3)	1 (0.5)	0	0	0

Conclusions: Most patients who were prevalent on therapy at the time of enrollment in Corrona remained persistent on their therapy for 12 months in this study, while roughly half of patients initiating therapy after enrollment remained persistent over the same period. Young, female patients were more likely to receive TNFi monotherapy; the TNFi monotherapy cohort was associated with the least disease activity. The incident group was not different from the prevalent group. Although the prevalent group is more likely to have patients who responded to treatment, the data suggest that most therapy changes occur within the first year of PsA treatment.
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FRI0137 EFFICACY, SAFETY AND IMMUNOGENICITY FROM WEEK 30 TO WEEK 54 IN A RANDOMISED, DOUBLE-BLIND PHASE III STUDY COMPARING A PROPOSED INFLIXIMAB BIOSIMILAR (PF-06438179/GP1111) WITH REFERENCE INFLIXIMAB

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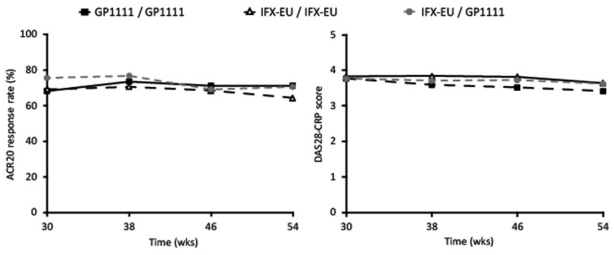
Background: PF-06438179/GP1111 (GP1111) is an infliximab (IFX) biosimilar in development for the treatment of immune-mediated inflammatory diseases, including rheumatoid arthritis (RA). The efficacy, safety and immunogenicity of GP1111 and European reference IFX (IFX-EU) have been reported to be similar over 30 weeks (Wks).
Objectives: To evaluate the efficacy, safety and immunogenicity of GP1111 and IFX-EU with longer-term treatment, and after treatment transition from IFX-EU to GP1111.
Methods: A randomised, double-blind, parallel-group study compared GP1111 with IFX-EU in biologic-naïve, adult patients with moderate-to-severe active RA on a stable dose of methotrexate (MTX). Patients were randomised (1:1) to GP1111 or IFX-EU (3 mg/kg IV at Wks 0, 2, 6, and then every 8 wks, with one dose escalation to 5 mg/kg allowed at or after Wk 14 for inadequate responders)

for 30 weeks (treatment period 1). The primary endpoint was a≥20% improvement in ACR response (ACR20) at Wk 14. At Wk 30 (treatment period 2 [TP2]), patients receiving IFX-EU were blindly re-randomised (1:1) to remain on IFX-EU or transition to GP1111 for 24 wks. Here we report longer-term efficacy, safety and immunogenicity data from Wks 30–54.

Results: 650 patients were randomised initially (GP1111, n=324; IFX-EU, n=326). At Wk 30, 566 patients entered TP2 (continued GP1111, n=280; continued IFX-EU, n=143; switched from IFX-EU to GP1111, n=143). ACR20 rates and DAS28-CRP scores were comparable between groups at all TP2 visits after re-randomisation in the TP2 population (figure 1). Incidences of TP2 treatment-emergent adverse events (AEs) (36.8%, 33.6%, and 37.8%), serious AEs (4.6%, 7.7% and 2.8%) and infusion-related reactions (3.2%, 8.4% and 4.2%) were comparable between the GP1111/GP1111, IFX-EU/IFX-EU, and IFX-EU/GP1111 groups, respectively. Pre-dose ADA rates at Wk 30 (TP2) were 47.1%, 53.8% and 45.5% for the GP1111/GP1111, IFX-EU/IFX-EU, and IFX-EU/GP1111 groups, respectively. Overall, post-dose ADA rates in TP2 were comparable between groups (52.1%, 60.1%, and 58.0% respectively).



Abstract FRI0137 – Figure 1. ACR20 response rate and change in DAS28-CRP score at Wk 30 and 54 for the overall population during TP2

Conclusions: Results from TP2 (Wks 30–54) continued to show the absence of clinically meaningful differences in efficacy, safety and immunogenicity between patients with RA remaining on GP1111 or IFX-EU, or when blindly switched from IFX-EU to GP1111.

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FRI0138 TREAT TO TARGET STRATEGY PLUS CERTOLIZUMAB IN COMPARISON TO CONTINUED, FIXED CSDMARD PLUS CORTICOSTEROIDS IN PATIENTS WITH RHEUMATOID ARTHRITIS AND INADEQUATE RESPONSE TO CSDMARDS (REMISSION BY INTRA-ARTICULAR INJECTION PLUS CERTOLIZUMAB, THE RICE STUDY): A MULTI-CENTRE RANDOMISED CONTROLLED TRIAL

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Background: Treatment of rheumatoid arthritis (RA) includes the use of conventional (cs) or targeted synthetic (ts) and biologic disease-modifying anti-rheumatic drugs (DMARDs) and oral or subcutaneous (SC) glucocorticoids (GC).

Objectives: We aimed to test the hypothesis that an improved outcome can be achieved by employing a treat to target (T2T) strategy optimising csDMARD, oral, and SC-GC treatment in parallel to a new onset certolizumab pegol (CZP) in RA patients with an incomplete response to csDMARD as compared to a conventional step up strategy with CZP.

Methods: We designed a randomised controlled trial in four specialised rheumatological units. 43 patients with active RA (≥6 tender,≥6 swollen joints, and ESR ≥20 mm/h or CRP ≥7 mg/L) despite csDMARD treatment for ≥3 months and naïve to biologic DMARDs were randomly allocated either to CZP plus a treat to target strategy (T2T group, n=21) or add on of CZP to a fixed dose of the already established csDMARD with or without established GCs (fixed dose group, n=22). Patients of both groups received 400 mg CZP at week 0, 2, and 4 (loading dose), followed by 200 mg every 2 weeks. The T2T strategy consisted in a step up in, or to, SC-methotrexate (dose: 15≥20≥25 mg/week), followed by leflunomide (20 mg/d) and then by sulfasalazine (2 × 1000 mg/d). In parallel, oral GCs were initiated in the T2T group at 20 mg/d and tapered every 5 days (15–12.5–10–7.5–5–2.5–0 mg/d). The decision to take the next step in the DMARD modification and addition of oral GCs was taken depending on the achievement of LDA (low disease activity: DAS28 <3.2) at the 4-weekly visits. Injections of up to five tender and/or swollen joints with triamcinolone (small joints 10 mg, intermediate 20 mg, and big joints 40 mg; max. 100 mg/visit) was allowed in the T2T group.

The primary outcome measure was ACR 50 response after 24 weeks. The analysis was intention-to-treat.

Results: Three patients dropped out during the study (n=2 T2T, n=1 fixed dose). ACR 50 was achieved in 16 out 21 T2T patients (76.2%) as compared to 8 out of 22 fixed dose patients (36.4%; Chi^2: 5.355, p=0.020). ACR 20 and 70 responses were achieved in 90.5% and 71.4% of the T2T patients and 59.1% and 27.3% of the fixed dose patients, respectively (p=0.045 and p=0.010, resp.). Mean reduction in DAS28 were significantly and HAQ-DI markedly greater in the T2T group than in the fixed dose group (DAS28: −3.9 [SD 1.2] vs. −2.2 [SD 1.5], p<0.0006; HAQ-DI: −0.63 [SD 0.58] vs. 0.20 [SD 0.67], p<0.14). All but five of the T2T patients required only one modification of csDMARD and one additional course of oral GC. 10.2 joints (mean) were infiltrated with triamcinolone in the T2T group (av. dose 14.1 mg/injection). The adverse event rate was similar for both groups (T2T n=51; fixed dose n=55).

Conclusions: Treat to target management with optimisation of csDMARDs, oral and intra-articular glucocorticoids of RA patients in parallel to additional CZP treatment was safe and substantially improves disease activity and the patient related outcome HAQ-DI in comparison to additional CZP to a fixed dose of csDMARDS.

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FRI0139 COMPARATIVE SAFETY OF ABATACEPT IN RHEUMATOID ARTHRITIS WITH COPD: A REAL-WORLD POPULATION-BASED OBSERVATIONAL STUDY

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Background: In the ASSURE trial (NCT00048932) comparing abatacept with placebo for the treatment of RA, there was an increased incidence of respiratory serious adverse events (SAEs; COPD exacerbation/worsening, bronchitis and pneumonia) in those receiving abatacept among the subgroup of 54 patients with a history of chronic obstructive pulmonary disease (COPD).[1]

Objectives: To assess whether patients with RA and a history of co-morbid COPD treated with abatacept in a real-world, observational setting, have an increased risk of respiratory SAEs compared with similar patients treated with other biologic (b)DMARDs or the targeted synthetic DMARD tofacitinib (tofa).

Methods: The Truven MarketScan® Commercial and Supplemental Medicare databases were used to identify adult patients diagnosed with RA and COPD who were treated with abatacept, another bDMARD or tofa between January 2007 and December 2015. Other bDMARDs included adalimumab, anakinra, certolizumab, etanercept, golimumab, infliximab, rituximab and tocilizumab. A prevalent new-user study cohort design[2] was used in which each new user of abatacept was time- and propensity score-matched to two new users of other bDMARDs or tofa. Patients were required to have ≥6 months of continuous health plan enrolment before cohort entry and were followed up until the end of enrolment in the database or 31 December 2015. Propensity scores of abatacept treatment were estimated from the baseline covariates using a conditional logistic regression model separately in incident new users and prevalent new users. Patients with score ranges common to both abatacept and the comparator cohorts were included. An as-treated analysis based on the Cox proportional hazard regression model was used to estimate the hazard ratios (HRs) of respiratory SAEs associated with abatacept compared with other bDMARDs or tofa, further adjusted for confounders found to be unbalanced despite matching on propensity scores.

Results: A total of 9746 patients with RA and COPD initiated bDMARD or tofa therapy and included 1807 new users of abatacept matched to 3547 new users of another bDMARD or tofa. The matched cohort was followed for up to 9 years (mean 2.0 years); 53% were incident users. For users of abatacept relative to other bDMARDs or tofa, the adjusted HRs (95% CI) of respiratory SAEs were: hospitalisation for COPD exacerbation: 0.57 (0.30, 1.05); hospitalisation for pneumonia/influenza: 1.39 (0.91, 2.12); outpatient pneumonia/influenza: 1.04 (0.85,