DMARDs (VNS monotherapy= 3.76±1.77 vs. VNS and biologic DMARD= 3.21±1.44, $p<0.54$). No difference in the adverse events profile between the two groups was seen.

Table 1 Two Year Efficacy of VNS Treatment. Mean DAS28-CRP at primary study baseline (month -3-5) and at visits over 2 years of long term follow up (months 0-24). **Conclusions:** The data presented here demonstrate that VNS in subjects with RA is associated with a substantial reduction in disease activity that is sustained for 24 months without untoward safety signals. In addition, the data suggest that biological DMARDs can be initiated safely in combination with VNS treatment, though this requires further study in larger cohorts. These results support further development of VNS devices as an alternative therapeutic approach for RA treatment, which potentially can safely be combined with biologic DMARDs.

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SAT0241

PATIENTS' PERSPECTIVE ON THE EFFICACY AND RISKS OF GLUCOCORTICOIDS IN RA – AN INITIATIVE UNDER THE GLORIA PROJECT

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Background: The Glucocorticoid Low-dose Outcome in Rheumatoid Arthritis Study (GLORIA) is an international investigator-initiated pragmatic randomised trial designed to study the effects of low dose glucocorticoids (GC) in elderly patients with Rheumatoid Arthritis (RA). The research team is also committed to promote a better understanding of the risks and benefits of these drugs among health professionals and patients. In order to achieve these goals, it is important to assess the current concepts and concerns of patients regarding GC.

Objectives: In this study, we evaluated the beliefs about GC in RA patients who are, or have been treated with GC.

Methods: Patients with RA from three different countries (United States (US), Portugal, and the Netherlands) completed an online survey which was presented in their native language.

Members of People with Arthritis and Rheumatism, and national associations were involved in the development of the questionnaire. In Europe, patients were invited to participate through national patients' organizations, and SurveyMonkey[®] was used to disseminate the online surveys. In the US, patients were invited to participate and surveyed through MediGuard.org.

Patients with RA from three different countries (United States (US), Portugal, and the Netherlands) completed an online survey which was presented in their native language.

Members of People with Arthritis and Rheumatism, and national associations were involved in the development of the questionnaire. Participants were asked to agree or disagree with statements on a 5-point scale. In Europe, patients were invited to participate through national patients' organizations, and SurveyMonkey[®] was used to disseminate the online surveys. In the US, patients were invited to participate and surveyed through MediGuard.org.

Results: Data was collected from 314 RA patients with exposure to GCs (table 1). Mean education level was around 15 years and duration of GC exposure was

skewed (median 48 months [inner quartiles 8, 120]). The majority of US patients had received prednisone and in Europe, prednisolone. The majority of participants in all three regions had already read articles or pamphlets on the benefits or harms related to GC therapy.

Regarding GC risk, about half of the European patients stated that they had already suffered a serious adverse event (SAE) due to GC. US patients were not asked if they suffered GC-related SAE due to regulatory reporting rules, but 82% showed concern about experiencing an SAE from GC use.

Regarding GC efficacy, high levels of endorsement were found for the three questions asked: more than 78% of patients considered that GC were very useful in their case, more than 61% considered that GC were effective even in low doses, and more than 60% agreed that GC improved RA symptoms within days.

Table 1 Patient beliefs about GC from surveys in USA, Portugal and The Netherlands.

	USA	Portugal	Netherlands
Number of patients	139	131	44
Education, mean ± SD (years)	14 ± 3	15 ± 5	16 ± 4
GC prescribed (%)			
Prednisone	78	18	33
Prednisolone	11	73	62
Methylprednisolone	9	10	5
Other	1	0	0
Treatment duration, median (inner quartiles, months)	24 (5-84)	120 (36-180)	36 (4-108)
Have you read any articles or pamphlets on the benefit or harm of GC therapy? (%)	72	64	75
Level of agreement for statements (%)*			
If I take GC, I am concerned that I may suffer a serious adverse event	82 (55)	NA	NA
I have suffered serious adverse events due to GC	NA	48 (18)	51 (23)
GC are or have been very useful to me	79 (56)	86 (55)	85 (69)
At a dose of less than or equal to 7.5 mg of prednisolone/day, GC:			
- are very effective in the control of signs and symptoms of RA	61 (38)	78 (41)	69 (44)
- improve RA symptoms within days	60 (41)	74 (45)	69 (49)

* Total of 'slightly agree' and 'agree' in parentheses the percentage of 'agree';

SD: Standard Deviation; GC: Glucocorticoid; RA: Rheumatoid Arthritis; NA: Not Asked.

Conclusions: Patients with RA exposed to long-term GC report a high prevalence of SAE or fear thereof. This is accompanied by a strong conviction that GC are very useful and effective for the treatment of their RA, even at low dosages.

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SAT0242

EFFECTIVENESS OF CONVENTIONAL DMARD THERAPY IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED UNDER A TREAT TO TARGET MODEL – LESSONS FROM A REAL-LIFE COHORT

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Background: Rheumatoid arthritis (RA) is a common chronic inflammatory disease. It is characterized by progressive, joint damage, impaired joint function and pain, the disease causes disability and reduces quality of life. Treat-to-target (T2T) is an acknowledged management strategy for RA; it proposes that the therapeutic target in RA should be a state of remission or low disease activity. There are two types of pharmacological therapy available: biological DMARDs that are considered highly expensive for our countries and conventional DMARDs which have demonstrated effectiveness and is a low-cost treatment (1,2).

Objectives: The aim of this study was to describe global change in Disease Activity Score 28 (DAS28) using a T2T strategy during three years in a cohort of patients receiving conventional DMARDs.

Methods: A descriptive cohort study was conducted. Medical records of patients from specialized in RA center were reviewed during 2015–2017; those patients were followed-up under T2T standards and a multidisciplinary approach. Clinical follow-up was according to DAS28: every 3–5 weeks (DAS28 >5.1), every 7–9 weeks (DAS28 ≥3.1 and ≤5.1), and every 11–13 weeks (DAS28 <3.1). Therapy had to be adjusted with DAS28 >3.2 unless patient's conditions don't permit it; We divided patients in four groups: remission (REM), low disease activity (LDA), moderate disease activity (MDA) and severe disease activity (SDA) patients and the aim of the study was to look at what percentage of patients reached LDA or REM. Descriptive epidemiology was done, we calculated means, and standard deviations for continuous variables and categorical variables were presented as rates.

We analyzed normality for DAS28, in order to compare disease activity at beginning and the end of follow-up.

Results: During 3 years we included 1953 patients were 39% were in low disease activity, 47% in moderate disease activity and 14% were in severe disease activity, 84% were female, mean age was 60 years $\pm$ 12. At baseline mean DAS28 was 4.45 $\pm$ 0.90 with a median of 4.3 at three years the mean DAS28 was 3.83 $\pm$ 1.08 with a median of 3.60. At the end of follow-up 46% of population achieved remission and 25% achieved low disease activity; at overall 71% improved disease activity, see table 1. In our study DAS28 was not normally distributed, thus we performed a Wilcoxon test in order to compare the mean DAS28 at baseline/36 months showing statistical significance ($P<0.05$).

ACTIVITY LEVEL	BASELINE		3 YEARS FOLLOW-UP	
	n	%	n	%
REM			898	46
LDA	759	39	488	25
MDA	912	47	528	27
SDA	282	14	39	2

Conclusions: Patients treated with conventional DMARD therapy and under a T2T model achieve favorable results in regards of disease activity. This is real life evidence that can support the advantages of treating RA patients with a multidisciplinary team under a T2T model with a low-cost treatment.

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SAT0243

INCIDENCE OF THROMBOEMBOLIC EVENTS IN THE TOFACITINIB RHEUMATOID ARTHRITIS, PSORIASIS, PSORIATIC ARTHRITIS AND ULCERATIVE COLITIS DEVELOPMENT PROGRAMMES

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Background: Tofacitinib is an oral Janus kinase (JAK) inhibitor that preferentially inhibits signalling by JAK3 and JAK1, with functional selectivity over JAK2. Potential increased risk of venous thromboembolic events (VTE) in patients (pts) with rheumatoid arthritis (RA) has been reported for a JAK 1/2 inhibitor.¹

Objectives: To assess VTE risk with tofacitinib in pts with RA, psoriasis (PsO), psoriatic arthritis (PsA) and ulcerative colitis (UC).

Methods: Data from Phase (P)2 (RA, PsO, UC) and P3 (RA, PsO, PsA, UC) randomised clinical studies of tofacitinib as monotherapy or in combination with conventional synthetic (cs)DMARDs were reviewed. Two cohorts were defined; 1) the placebo (PBO)-controlled cohort: pts randomised to tofacitinib 5 or 10 mg BID, or PBO up to Month (M)3 in RA, PsO and PsA studies, and pts randomised to tofacitinib 10 mg BID or PBO for the 9-week induction period in UC studies; 2) the dose-comparison cohort: pts randomised to tofacitinib 5 or 10 mg BID, adalimumab (ADA) 40 mg SC Q2W (RA and PsA only) or methotrexate (MTX) 20 mg QW (RA only) throughout the P2/3 studies for RA (up to M24), PsO (up to M12) and PsA (up to M12), and for the 12-month P3 UC maintenance study. First deep vein thrombosis (DVT) and pulmonary embolism (PE) events were identified using the MedDRA embolic and thrombotic SMQ preferred terms restricted to the respiratory, thoracic, mediastinal and vascular disorder System Organ Classes; incidence rates (IRs; pts with events/100 pt-years) were based on single events occurring during treatment or $\leq$ 28 days after the last dose or up to the cohort cut-off date. IRs for PE in RA pts were compared with Corrona Registry data (May 2017 cut-off).

Results: Up to M3 in the PBO-controlled cohort, DVT and PE each occurred in 2 pts receiving PBO (1 RA pt and 1 UC pt per event); no tofacitinib-treated pts had DVT or PE events (Table). In the dose-comparison cohort 2 DVT events occurred in tofacitinib-treated pts with RA (5 mg BID, n=1; 10 mg BID, n=1) and 1 DVT event in a pt with PsA (tofacitinib 10 mg BID) (Table). IRs (95% CI) were 0.1 (0.0, 0.3) for both tofacitinib doses in RA and 0.5 (0.0, 2.8) for tofacitinib 10 mg BID in PsA. Two DVT events occurred with MTX; none with ADA. Five PE events occurred in the dose comparison cohort, all in RA (5 mg BID, n=2; 10 mg BID, n=3). IRs were 0.1 (0.0, 0.4) for tofacitinib 5 mg BID and 0.2 (0.0, 0.4) for 10 mg BID. IRs for PE with tofacitinib in RA were similar to those reported by the Corrona Registry in RA pts treated with tofacitinib (0.1 [0.0, 0.4]), biologic DMARDs (0.2 [0.1, 0.3]) and csDMARDs (0.2 [0.0, 0.5]).

Table 1 Incidence rates for deep vein thrombosis and pulmonary embolism from randomised controlled studies across the tofacitinib clinical development programme

n/N (95% CI) PE	Placebo-controlled cohort ^a			Dose-comparison cohort ^b			Methotrexate 20 mg QW
	Tofacitinib 5 mg BID	Tofacitinib 10 mg BID	Placebo	Tofacitinib 5 mg BID	Tofacitinib 10 mg BID	Adalimumab 40 mg SC Q2W	
Rheumatoid arthritis							
DVT	0/1849 (0.0, 0.6)	0/2004 (0.0, 0.4)	1/1879 (0.0, 0.4)	1/1849 (0.1, 0.3)	1/2004 (0.1, 0.4)	8/227 (0.0, 1.9)	2/223 (0.0, 1.2)
PE ^c	0/1849 (0.0, 0.6)	0/2004 (0.0, 0.4)	1/1879 (0.0, 0.4)	1/1849 (0.1, 0.4)	2/2004 (0.2, 0.6)	8/227 (0.0, 1.9)	2/223 (0.0, 1.3)
Psoriasis							
DVT	0/123 (0.0, 1.0)	0/123 (0.0, 1.0)	0/123 (0.0, 2.1)	0/127 (0.0, 0.4)	0/1219 (0.0, 0.6)	—	—
PE ^c	0/123 (0.0, 1.0)	0/123 (0.0, 1.0)	0/123 (0.0, 2.1)	0/127 (0.0, 0.4)	0/1219 (0.0, 0.6)	—	—
Psoriatic arthritis^c							
DVT	0/28 (0.0, 6.4)	0/28 (0.0, 6.4)	0/28 (0.0, 6.4)	0/347 (0.0, 1.8)	1/144 (0.0, 2.8)	8/106 (0.0, 4.6)	—
PE ^c	0/28 (0.0, 6.4)	0/28 (0.0, 6.4)	0/28 (0.0, 6.4)	0/347 (0.0, 1.8)	1/144 (0.0, 2.8)	8/106 (0.0, 4.6)	—
Ulcerative colitis							
DVT	N/A	0/958 (0.0, 2.2)	1/282 (0.0, 1.1)	0/508 (0.0, 2.5)	0/196 (0.0, 2.4)	—	—
PE ^c	N/A	0/958 (0.0, 2.2)	1/282 (0.0, 1.1)	0/508 (0.0, 2.5)	0/196 (0.0, 2.4)	—	—

^aIncludes Months 0–3 of Phase 2/3 studies for RA, PsO and PsA, and the 9-week induction period of Phase 2/3 UC studies. ^bIncludes Months 0–24 of Phase 2/3 studies for RA, Months 0–12 for PsO and PsA, and the 12-month P3 UC maintenance study. ^cDose-comparison cohort for tofacitinib 5 mg BID and 10 mg BID compares patients randomised at baseline to tofacitinib 5 mg BID + patients randomised at baseline to PBO + tofacitinib 5 mg BID, and patients randomised at baseline to tofacitinib 10 mg BID + patients randomised at baseline to PBO + tofacitinib 10 mg BID, respectively. IR=patients with event/100 PY.

Conclusions: Analysis of DVT and PE across randomised clinical studies for RA, PsO, PsA and UC showed no evidence of an increased risk of these events with tofacitinib vs other therapies.

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