Disclosure of Interest: K. Price Employee of: Bristol-Myers Squibb, Y. Doleh Employee of: Bristol-Myers Squibb, M. Eaddy Consultant for: Xcenda, A. Ogbonnaya Employee of: Xcenda, H.-C. Shih Employee of: Xcenda, H. Ahmad Shareholder of: Bristol-Myers Squibb, Employee of: Bristol-Myers Squibb, L. Lamerato Grant/research support from: Centers for Disease Control, National Cancer Institute, Policy Analysis, Inc, Outcomes Research Solutions, Xcenda, eMAXHealth, Merck Pharmaceuticals, Pfizer Pharmaceuticals, Reagan Udall Foundation, AstraZeneca, Employee of: Henry Ford Health System, University of Michigan-Dearborn, A. Szymialis Shareholder of: Bristol-Myers Squibb, Employee of: Bristol-Myers Squibb
DOI: 10.1136/annrheumdis-2017-eular.1794

SAT0189 FACTORS INFLUENCING THE PRESCRIPTION OF TOCILIZUMAB ALONE OR IN COMBINATION WITH DMARDS IN RHEUMATOID ARTHRITIS PATIENTS IN A REAL LIFE SETTING. POOLED ANALYSIS OF 3 OBSERVATIONAL STUDIES

A. Saraux[1], A. Cantagrel[2], B. Combe[3], R.-M. Flipo[4], I. Idier[5], C. Baffie[6], J. Tebib[7]. *[1]Rheumatology, University Hospital, Brest; [2]Rheumatology, University Hospital, Toulouse; [3]Rheumatology, University Hospital, Montpellier; [4]Rheumatology, University Hospital, Lille; [5]Medical, Chugai Pharma France, Paris la Defense; [6]Statistics, Roche SAS, Boulogne; [7]Rheumatology, University Hospital, Lyon, France*

Background: Tocilizumab (TCZ) as monotherapy (Mono) is nowadays a standard treatment in rheumatoid arthritis (RA) for patients in whom methotrexate (MTX) is inappropriate[1].
Objectives: To describe factors influencing the use of TCZ in Mono or in combination with DMARDs (Combo) in real-life practice in RA patients (pts).
Methods: *Analysis:* pooled data of 3 prospective, multicentre, observational studies (PEPS n=610, Spare-1 n=307, Act-solo n=577). *Patients:* RA pts requiring TCZ treatment according to their physician. *Treatment:* TCZ as prescribed in real life. *Endpoint:* Evaluation of factors influencing the use of TCZ in Mono or in Combo. *Data collected:* demographic characteristics, past medical history, RA characteristics and history including previous RA treatments, TCZ treatment strategy (Mono or Combo). *Statistical analysis:* Pts fulfilling inclusion and non-inclusion criteria and with ≥1 TCZ infusion were analyzed. 1- descriptive analysis 2- Univariate and multivariate analysis to determine factors influencing the use of TCZ in Mono. Variables with more than 20% of missing data were excluded from the multivariate model.
Results: 1494 pts (3 studies) were analysed at inclusion. Pts' characteristics: 56% of the pts were >55 years old, 79.6% female, mean RA duration 11.1±9.3 years, 83.0% positive for rheumatoid factors and/or ACPA, 77.3% with erosive disease on X-rays, mean ESR 29.9±23.1mm, mean CRP 19.9±26.2 mg/l mean DAS 28-ESR 5.21±1.22, mean HAQ-DI 1.56±0.68, and mean pain VAS 61.5±23.2. Past RA treatment included csDMARDs in 98.5% and biologics in 77.8% (median=2 [1–6]). TCZ was initiated as Mono in 36.4% of pts and in Combo in 63.6%, with MTX in 83.3% of Combo pts (mean dose 15.7±4.4mg/week). Corticosteroids were used in 74% of pts (mean dose 10±7mg/day). Variables associated with a TCZ prescription in Mono were age (≥65 years), number of previous bDMARD, use/dose of steroids, ESR/CRP values, VAS global activity (physician and pt), pain VAS and HAQ-DI. In the multivariate analysis, variables associated with a TCZ prescription in Mono were age ≥65 years (OR=1.71 [1.30 - 2.24], p<0.001), number of previous bDMARD (1 bDMARD, OR=1.35 [0.94 - 1.92], 2 bDMARD OR=1.82 [1.28 - 2.60], ≥2 bDMARD, OR=1.19 [0.82 - 1.71] p=0.006), higher pain VAS (OR=1.09 [1.04 - 1.15], p=0.001), higher ESR value (OR=1.07 [1.02 - 1.12], p=0.013).
Conclusions: This pooled analysis suggests that physicians preferably prescribe TCZ alone in older patients, heavily treated before, with higher inflammatory markers and higher pain VAS. This use might be explained by physicians' reluctance to prescribe the association in frailer patients and complementary data on comorbidity factors will be analysed to support this hypothesis.
References:
[1] JS. Smolen et al, Ann Rheum Dis doi:10.1136/annrheumdis-2013–204573.
Disclosure of Interest: A. Saraux: None declared, A. Cantagrel: None declared, B. Combe Consultant for: Chugai Pharma France, R.-M. Flipo Consultant for: Chugai Pharma France, I. Idier Employee of: Chugai Pharma France, C. Baffie Employee of: Altizem for Roche SAS, J. Tebib: None declared
DOI: 10.1136/annrheumdis-2017-eular.2916

SAT0190 ESTIMATE OF CLINICAL AND ANTIDESTRUCTIVE EFFECTS OF RITUXIMAB IN RHEUMATOID ARTHRITIS

A.V. Pivanova, G. Lukina, Y. Sigidin, A. Smirnov, K. Kuzikyants, A. Kuznetsova. *Nasonova Research Institute of Rheumatology, MOSCOW, RUSSIA, Moscow, Russian Federation*

Background: Rheumatoid arthritis (RA) is a chronic inflammatory disease characterized by synovial hyperplasia, mononuclear cell infiltration, bone erosion and joint destruction. Antirheumatic treatment plays a important role in controlling the inflammation of rheumatoid arthritis and in minimizing joint damage. Rituximab - it is a chimeric monoclonal antibody that targets the CD20 molecule expressed on the surface of B cells. It has been successfully used to treat rheumatoid arthritis, and it is worth noting that his antidestructive effect sometimes does not meet the clinical.
Objectives: to assess clinical and antidestructive effect of Rituximab (RTX) in patients with rheumatoid arthritis (RA).
Methods: 108 patients (pts) with RA, most of them were middle-age women with high disease activity (mean DAS28 6,1±1,04, RF-positive 77%, ACCP-positive 83%) treated with RTX (1000 mgx2 or 500 mgx2). Clinical effect was evaluated by EULAR criteria; radiological progression by SVH method.
Results: 104 patients were treated by RTX (500 x2 or 1000 x2), had good response: after 48 week of treatment clinical improvement was achieved in 65% pts, good and moderate response by EULAR criteria in 23% and 42% pts accordingly. Noteworthy, after 12 months of treatment RTX radiological progression was absent in 50% pts with high disease activity.
Conclusions: RTX treatment slowed joint damage without clinical improvement. Clinical and antidestructive results did not always coincide which suggests different mechanisms of clinical and antidestructive effects of anti-B-cell therapy
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.6524

SAT0191 PATTERNS OF INTERSTITIAL LUNG DISEASE IN RHEUMATOID ARTHRITIS AND ABATACEPT. MULTICENTER STUDY OF 63 PATIENTS

C. Fernández-Díaz[1], S. Castañeda[2], C. Ojeda[3], A. Olivé[4], P. Carreira[5], T. Perez-Sandoval[6], M. Retuerto[7], E. Cervantes[8], S. Rodriguez[4], B. Robles[9], B. Hernandez[3], A. Urruticoechea[10], O. Maiz[11], D. Palma[12], L. Arboleya[13], G. Bonilla[14], I. Rodriguez[15], C. Delgado[16], R. Exposito[17], A. Ruibal[18], J. Blanco[19], M. Rodriguez[20], J. Bernal[21], P. Vela[21], B. Alvarez[22], C. Fito[23], J. Narvaez[24], M. Moreno[25], M. Lopez[26], N. Mena[27], S. Romero[28], C. Aguilera[29], S. Ordoñez[30], I. Villa[31], V. Mora[1], M. Gonzalez-Gay[1], J. Hernandez[1], R. Blanco[1]. *[1]HUMV, Santander; [2]H.Princesa, Madrid; [3]H Macarena, Sevilla; [4]H Trias, Barcelona; [5]H 12 Octubre, Madrid; [6]H; [7]H., Leon; [8]H, Santiago; [9]H. P Hierro, Madrid; [10]H, Ibiza; [11]H, Donostia; [12]H, Lorca; [13]H, Oviedo; [14]H. Paz, Madrid; [15]H, Vigo; [16]H., Zaragoza; [17]H., Laredo; [18]H., Araba; [19]H.Basurto, Bilbao; [20]H., Ourense; [21]H., Alicante; [22]H, Araba; [23]H, Pamplona; [24]H.Bellvitge, Barcelona; [25]H.Arrixaca, Murcia; [26]HVHebron, Barcelona; [27]H, Malaga; [28]H, Pontevedra; [29]H, Sevilla; [30]H, Lleida; [31]H, Sierrallana, Spain*

Background: Disease modifying antirheumatic drugs (DMARDs) such as methotrexate (MTX), leflunomide (LFN) or antiTNFα have been implicated in development/exacerbation of Interstitial lung disease (ILD)of rheumatoid arthritis (RA). Several radiological patterns of ILD have been described: i) usual interstitial pneumonia (UIP), ii) nonspecific interstitial pneumonia (NSIP), iii) obliterating bronchitis (OB), and iv) Organized pneumonia (OP)
Objectives: To assess the response to Abatacept (ABA) in these patterns of ILD
Methods: Multicenter study of RA-ILD treated with ABA. ILD was diagnosed by high-resolution CT scan (HRCT) and classified in radiological patterns (Travis et al). We consider 3 subgroups: a) UIP, b) NSIP and c) "other" (OB, OP or mixed). ABA was used at iv or sc standard dose. We assessed: a) Dyspnea (Medical Research Council-modified scale; significant variations≥1); B) Respiratory function tests; significant changes≥10% in forced vital capacity (FVC) and DLCO≤10%, c) HRCT, d) DAS28. A comparative study was performed for the quantitative (U-Mann-Whitney) and qualitative variables (Fisher test) between the baseline and 3, 6 and 12 months.
Results: We included 63 patients (27 women/36 men), mean age; 63.1±9.6 years. At ABA onset the RA had a median evolution of 6.8 [2–13.6] years and the ILD of 1 [0.3–3.03]. RA was seropositive in 85.7%. The diagnosis of ILD was confirmed by biopsy (n=18). The ILD was related to DMARDs: MTX (4), etanercept (3), adalimumab (3), certolizumab (2), Infliximab (1). ABA was used in monotherapy (26) or combined with other DMARDs (37); LFN (15), Cyclosporin (1), sulfasalazine (4), MTX (6), hydroxychloroquine (10), azathioprine (4), chloroquine (1). Table 1 shows the evolution in the available cases. A significant improvement in dyspnea and HRCT was observed in the NIU type. DLCO remained stable in most patients regardless of the radiological pattern. The activity of RA (DAS28) also improved.

Table 1

	Baseline			3month			6month			12month		
	UIP	NSIP	Others	UIP	NSIP	Other	UIP	NSIP	Other	UIP	NSIP	Other
MMRC. n(%)				n=26	n=16	n=16	n=24	n=16	n=15	n=13	n=10	n=8
No changes	n=29	n=17	n=17	18 (65.4)	12 (75)	13 (81.3)	17 (70.8)	12 (75.9)	11(73.3)	7 (53.8)	7 (70)	6 (75)
Improvement				**9(34.6)****	3 (18.8)	3 (18.7)	**5(20.8)***	3 (23.1)	4 (26.7)	**3 (23.1)***	3 (30)	2 (25)
Worsening				0	1 (6.2)	0	2(8.4)	0	0	3 (23.1)	0	0
CVF. n (%)				n=12	n=4	n=2	n=6	n=7	n=3	n=10	n=5	n=7
No changes	n=27	n=17	n=16	10 (83)	3 (75)	2 (100)	5 (83.3)	1 (14.3)	3 (100)	10(100)	3 (60)	2 (28.6)
Improvement				2 (16.7)	0	0	1 (16.7)	**4 (57.1)****	0	0	2 (40)	**3 (28.6)***
Worsening				0	1(25)	0	0	2 (28.6)	0	0	0	2 (28.6)
DLCO. n (%)				n=7	n=4	n=2	n=5	n=3	n=3	n=7	n=3	n=6
No changes	n=23	n=12	n=13	5 (71.4)	4 (100)	1 (50)	3 (60)	2 (66.7)	2 (66.7)	5 (71.6)	2 (66.7)	3 (33.3)
Improvement				2 (28.6)	0	1 (50)	1 (20)	0	0	2 (28.6)	0	2 (5)
Worsening				0	0	0	1 (20)	1 (33.3)	1 (33.3)	0	1 (33.3)	1 (16.7)
HRTC. n (%)				n=6	n=3	n=1	n=6	n=5	n=5	n=9	n=5	n=3
No changes	n=29	n=17	n=17	3 (50)	2 (66.7)	0	3 (50)	2 (40)	2 (40)	4 (44.4)	2 (40)	1 (33.3)
Improvement				**2 (33.3)***	1 (33.3)	1 (100)	1 (16.7)	2 (40)	**3 (60)***	**3 (33.3)***	2 (40)	**2 (66.7)***
worsening				1 (16.7)	0	0	2 (33.3)	1 (20)	0	2 (22.2)	1 (20)	0
DAS28	n=29	n=16	n=16	n=17	n=8	n=8	n=14	n=13	n=9	n=14	n=10	n=9
median[IQR]	5.3 [4.4-6.3]	5.17[3.5-5.9]	5.21[3.8-5.9]	3.0 [2.24.8]	3.1[2.24.8]	**2.3[1.9.4.4]***	3.7[2.24.9]	**3.1[2.6.4.6]***	**2.8[1.3.3.7]***	**3.8[2.5.5.1]***	3.1[2.3-3.9]	**3.1[2.3.4.5]***
CRP (mg/dl),	n=29	n=14	n=16	n=17	n=5	n=9	n=14	n=12	n=10	n=14	n=9	n=9
median[IQR]	3.1 [0.8-8.7]	1.7 [0.9-4.9]	1.7 [1.0-4.8]	1.2 [0.3-5.4]	3.3[0.9-7.0]	1.5[0.6-3.9]	1.9[0.4-10.1]	**0.8 [0.4-2.1]***	**0.6 [0.5-1.9]***	4.2[0.8-13.0]	1.5 [0.4-7.6]	1.5[0.7-4.6]
ESR (mm/1st h),	n=29	n=15	n=16	n=20	n=5	n=9	n=16	n=12	n=10	n=13	n=10	n=10
median[IQR]	32[11.5-49.0]	41.0[30.0-63.0]	26.5[16.8-51.3]	16.5[10-40.8]	52.0[25.5-71.5]	19[10.5-29.5]	43[10.5-66.5]	**18.5 [8.0-29.8]***	18.5[9.0-44.5]	43.0[10.5-63.5]	**31.0 [18.0-54.0]***	27.0[14.3-38.0]
Prednisone mg	n=26	n=17	n=14	n=18	n=11	n=8	n=5	n=12	n=9	n=9	n=8	n=8
median[IQR]	7.5[5.0-11.3]	10.0[5.0-25.0]	7.8[4.4-15.0]	5.0[5.0-10.0]	7.5[5.0-25.0]	7.5[5.0-13.8]	5.0[5.0-10.0]	**5.0[2.5-7.5]***	**5.0 [3.8-7.8]***	5.0[2.5-18.8]	**5.0 [3.1-7.5]***	3.8 [2.5-6.9]

The comparisons are with respect to the baseline visit. * p <0.05; ** p <0.01.For the qualitative variables only the variable "improvement" is calculated.

Conclusions: ABA appears to be effective in ILD associated-RA, including the pattern of poor prognosis (UIP).

References:
[1] Travis WD et al. J Respir Crit Care Med 2013 188:733–748.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.3492

SAT0192 SARILUMAB SUPPRESSES THROMBOSIS-RELATED GENE EXPRESSION IN CIRCULATING BLOOD CELLS IN MTX-IR AND TNF-IR PATIENTS WITH ACTIVE RHEUMATOID ARTHRITIS

C. Paccard[1], J. Msihid[1], A. Brisacier[1], A. Damask[2], C. Paulding[2], M. Zilberstein[3], A. Boyapati[2]. *[1]Sanofi R&D, Chilly-Mazarin, France; [2]Regeneron Pharmaceuticals, Inc, Tarrytown; [3]Sanofi R&D, Bridgewater, United States*

Background: Bone and joint damage due to chronic inflammation in the synovium of patients with RA is mediated by IL-6 and other cytokines. IL-6 mediates effects inside the joint and systemically and is blocked by sarilumab, a human mAb blocking the IL-6R?. Safety and efficacy of SC sarilumab (150 or 200 mg q2w) was evaluated in combination with MTX in patients with RA and inadequate response (IR) to MTX (MOBILITY; NCT01061736) or csDMARDs in patients with RA and IR or intolerance to ≥1 TNFi (TARGET; NCT01709578).

Objectives: To compare gene expression patterns in circulating blood cells after treatment with sarilumab vs placebo in patients from MOBILITY and TARGET.

Methods: Total RNA was isolated from whole blood collected at baseline (predose) and wk 2 posttreatment in patients from MOBILITY (placebo, n=58; sarilumab 150 mg q2w, n=60; sarilumab 200 mg q2w, n=46) and TARGET (placebo, n=19; sarilumab 150 mg q2w, n=27; sarilumab 200 mg q2w, n=16). Gene expression data were generated using microarray analyses (Agilent 8 × 60k platform). Data were processed using the limma package (R Bioconductor). Background correction and quantile normalization were performed and probes with low expression were filtered; 48,109 probes were analyzed. Effect of sarilumab vs placebo on gene expression was assessed in each study using linear mixed models. *P* values were adjusted using the Benjamini-Hochberg procedure to control false discovery rate (FDR; 5% threshold).

Results: After *P* value adjustment to control FDR at 5%, 2 genes implicated in thrombosis and atherosclerosis (thrombomodulin [THBD] and platelet endothelial cell adhesion molecule 1 [PECAM-1]) were downregulated after treatment with sarilumab 200 mg q2w vs placebo in both studies (Table). These genes decreased with a fold-change (FC) ≤0.8 in both studies ($P<0.001$). An additional gene associated with coagulation, von Willebrand factor (vWF), was significantly decreased in MTX-IR but not TNF-IR patients. Numerical decreases in gene expression between sarilumab 150 mg q2w and placebo did not reach significance.

Table 1. Select Genes Regulated by Sarilumab 200 mg q2w at Week 2

	MOBILITY Sarilumab 200 mg q2w + MTX		TARGET Sarilumab 200 mg q2w + csDMARDs	
Gene	*P* value	Fold reduction[a]	*P* value	Fold reduction[a]
THBD	6.3×10^{-7}	0.67	3.9×10^{-6}	0.58
PECAM-1	1.6×10^{-9}	0.78	2.1×10^{-4}	0.78
vWF	1.3×10^{-10}	0.58	0.55	0.91

[a]Fold reduction vs placebo.

Conclusions: In patients with active RA, sarilumab may decrease thrombosis-related gene expression in circulating immune cells. Additional analysis of the serum levels of thrombosis risk proteins is needed to test the hypothesis that sarilumab treatment decreases levels of thrombosis risk factors.

Acknowledgements: This study was sponsored by Sanofi Genzyme and Regeneron Pharmaceuticals, Inc. Editorial support was provided by MedThink SciCom and funded by Sanofi Genzyme and Regeneron Pharmaceuticals, Inc.

Disclosure of Interest: C. Paccard Shareholder of: Sanofi R&D, Employee of: Sanofi R&D, J. Msihid Shareholder of: Sanofi R&D, Employee of: Sanofi R&D, A. Brisacier Shareholder of: Sanofi R&D, Employee of: Sanofi R&D, A. Damask Shareholder of: Regeneron Pharmaceuticals, Inc, Employee of: Regeneron Pharmaceuticals, Inc, C. Paulding Shareholder of: Regeneron Pharmaceuticals, Inc, Employee of: Regeneron Pharmaceuticals, Inc, M. Zilberstein Shareholder of: Sanofi R&D, Employee of: Sanofi R&D, A. Boyapati Shareholder of: Regeneron Pharmaceuticals, Inc, Employee of: Regeneron Pharmaceuticals, Inc

DOI: 10.1136/annrheumdis-2017-eular.5183

SAT0193 RA PATIENTS WITH INFLAMMATORY ANEMIA BENEFIT FROM INCREASED HEMOGLOBIN AND DECREASED FATIGUE UNDER TOCILIZUMAB THERAPY

C. Specker[1], H. Kellner[2], P. Kästner[3], C. Volberg[4], V. Braunewell[5], I. Schwarze[6], M. Aringer[7], M. Sieburg[8], M.W. Hofmann[9], J.P. Flacke[10], H.-P. Tony[11], G. Fliedner[12]. *[1]Kliniken Essen Süd, Essen; [2]Schwerpunktpraxis für Rheumatologie und Gastroenterologie, München; [3]MVZ Ambulantes Rheumazentrum Erfurt, Erfurt; [4]Rheumazentrum Neuss, Neuss; [5]Schwerpunktpraxis Rheumatologie, Mönchengladbach; [6]Praxis für Internistische Rheumatologie, Leipzig; [7]Medizinische Klinik III, Rheumatologie, Technischen Universität Dresden, Dresden; [8]Rheumatologische Gemeinschaftspraxis, Magdeburg; [9]Rheumatologie, Chugai Pharma Europe Ltd., Frankfurt; [10]Rheumatologie, Roche Pharma AG, Grenzach-Wyhlen; [11]Rheumatologie/Immunologie der Medizinischen Klinik und Poliklinik II, Universitaetsklinikum Wuerzburg, Wuerzburg; [12]Rheumapraxis, Osnabrueck, Germany*

Background: According to WHO definition approximately 15% of all patients with rheumatoid arthritis (RA) suffer from anemia (hemoglobin <13 g/dl for men and <12 g/dl for women). Interleukin 6 (IL-6) takes an active part in the pathogenesis of this inflammatory anemia.

Objectives: The 6th interim analysis of the non-interventional ICHIBAN study (NCT01194401) evaluated the occurrence of inflammatory anemia, characterized the patient population with anemia, and observed the response during intravenous Tocilizumab therapy (TCZ i.v.). Patients were subgrouped according to their anemic/non-anemic status at baseline.

Methods: Since 2010 the ICHIBAN study collects clinical data of the routine use of TCZ i.v. in RA patients. The observation period for each patient is up to two years. At the due date of the current interim analysis (Dec 10, 2015) 2999 patients were enrolled. 902 patients have completed the maximal 104 weeks observation period (Group W104).

Results: At baseline, the proportion of patients with anemia (acc. to WHO definition) was 21.4% (men) and 22.0% (women) in the group W104.

On comparison, RA patients with anemia showed, amongst others, increased inflammation parameters, a higher disease activity and higher rates of comorbidities. Already after 4 weeks with TCZ i.v. the proportion of patients with anemia improved to 12.1% (men) and 12.7% (women). After 104 weeks therapy the proportion of patients with anemia reduced further to 7.4% (men) and 8.4% (women). The relevant response parameters and laboratory values are shown in Table 1.

Despite the higher disease activity at baseline for anemic patients, the benefit was comparable for patients with and without anemia. DAS28-ESR values decreased on average by 2.9 (women) and 3.1 (men) in RA patients with anemia and by 2.7 (women) and 2.8 (men) in RA patients without, resulting in similar disease scores at the end of the observational period.

The effectiveness of TCZ i.v. was also confirmed by patient reported outcomes (PROs) via visual analogue scales (VAS). In particular, a reduction of the intensity of pain (>50%) and a reduction of fatigue (>38%) was observed (Table 1).

Table 1 Treatment response to TCZ in anemic patients (at baseline)

		Week 0 (Baseline)	Last visit with TCZ
% Anemia	male	21.4% (46/215)	7.4% (16/215)
	female	22.0% (151/687)	8.4% (58/687)
Hemoglobin [g/dl]	male	12.2 (11.8, 12.7)	13.8 (13.2, 14.7)
Median (Q1, Q3)	female	11.1 (10.3, 11.6)	12.7 (11.9, 13.4)
Erythrocytes [10^{12}/l]	male	4.5 (4.0, 4.7)	4.7 (4.3, 5.0)
Median (Q1, Q3)	female	4.1 (3.8, 4.4)	4.2 (4.0, 4.5)
CRP [mg/l]	male	42.1 (27.3, 71.2)	7.3 (1.5, 25.4)
Median (Q1, Q3)	female	30.0 (11.1, 61.0)	3.1 (1.0, 11.9)
ESR [mm/h]	male	46.5 (30.0, 68.0)	4.5 (2.0, 13.0)
Median (Q1, Q3)	female	42.0 (28.0, 74.0)	8.0 (4.0, 16.0)
DAS28-ESR			
Change from baseline	male		-3.1 ± 2.1
Mean ± SD	female		-2.9 ± 1.5
% Remission (< 2.6)	male	0.0% (0/46)	52.2% (24/46)
	female	0.0% (0/151)	45.0% (68/151)
Visual analogue scales			
Fatigue	male	62.0 (40.0, 79.0)	30.0 (10.0, 52.0)
Median (Q1, Q3)	female	65.0 (35.0, 85.0)	40.0 (15.0, 63.0)
Intensity of pain	male	70.0 (45.0, 83.0)	32.0 (10.0, 61.0)
Median (Q1, Q3)	female	65.0 (43.0, 86.0)	30.0 (12.0, 56.0)
Sleep disturbances	male	48.0 (19.0, 67.0)	12.0 (5.0, 50.0)
Median (Q1, Q3)	female	52.0 (21.0, 80.0)	38.0 (12.0, 66.0)

(CRP = C-Reactive Protein; ESR = Erythrocyte Sedimentation Rate; Q1, Q3 = 1st / 3rd quartile; SD = standard deviation)

Conclusions: At start of therapy, approximately one out of five patients documented in ICHIBAN showed anemia according to the WHO definition. During TCZ i.v. therapy a noticeable decrease in the rate of anemia and improved hemoglobin values were observed. These effects can already be seen after four weeks of treatment and continue up to the end of this study (i.e. 2 years). Despite the higher burden of disease at baseline in RA patients with anemia, TCZ i.v. therapy resulted in good clinical response rates and PROs.

Acknowledgements: We would like to thank all patients, their families, the investigators and the nurses who participated in this trial. This research was funded by Roche Pharma AG, Germany, and Chugai Pharma Europe Ltd., Germany. Support for third-party writing assistance for this abstract presentation