Disclosure of Interests: Khojasta Talash: None declared, Marwan Bukhari Speakers bureau: Bristol-Myers Squib, UCB celltech, Roche/Chugai, Pfizer, Abbvie, Merck, Mennarini, Sanofi-aventis, Eli-Lilly, Janssen and Novartis.
DOI: 10.1136/annrheumdis-2019-eular.900

SAT0247

FEMALE SEX AND AGE AT DIAGNOSIS ARE ASSOCIATED WITH A DECREASED POSSIBILITY OF DRUG DISCONTINUATION IN GIANT CELL ARTERITIS: DATA FROM A MULTICENTER PROSPECTIVE COHORT OF 177 PATIENTS

Christina Tsalapaki¹, Argyro Lazarini¹, Maria Tektonidou², Petros Sfikakis², Vasiliki Dania³, Kyriaki Boki³, Evripidis Kaltsonoudis⁴, Paraskevi V. Voulgaris⁴, Gerasimos Evangelatos⁵, Alexios Iliopoulos⁵, Evangelos Theotikos⁶, Antonia Elezoglou⁶, Charalampos Papagoras⁷, Theodoros Dimitroulas⁸, Alexandros Garyfallos⁸, Dimitrios Boumpas⁹, Georgios Vosvotekas¹⁰, Dimitrios Vassilopoulos¹. ¹Joint Rheumatology Program, Clinical Immunology-Rheumatology Unit, 2nd Department of Medicine and Laboratory, University of Athens, School of Medicine, Athens, Greece; ²Joint Rheumatology Program, First Department of Propedeutic Internal Medicine, University of Athens, School of Medicine, Athens, Greece; ³Sismanoglio Hospital, Rheumatology Unit, Athens, Greece; ⁴Rheumatology Clinic, University of Ioannina, Ioannina, Greece; ⁵NIMTS Hospital, Rheumatology Unit, Athens, Greece; ⁶Asklepiion General Hospital, Rheumatology Unit, Athens, Greece; ⁷Democritus University of Thrace, Rheumatology Clinic, Thrace, Greece; ⁸Aristotle University, 4th Department of Medicine, Thessaloniki, Greece; ⁹Joint Rheumatology Program, 4th Department of Medicine, Attikon Hospital, University of Athens, School of Medicine, Athens, Greece; ¹⁰Private Practice, Thessaloniki, Greece

Background: Giant cell arteritis (GCA) usually requires long-term therapy with corticosteroids and/or Disease Modifying Anti-rheumatic Drugs (DMARDs) but the exact factors that are associated with drug discontinuation in these patients have not been well defined.

Objectives: To evaluate the baseline or on-treatment factors that influence drug survival in GCA.

Methods: Data were derived from an ongoing, multicenter, prospective cohort study of patients with GCA. During the 1st phase of the study, data regarding demographic and clinical characteristics at baseline, type of treatment, adverse events of therapy and co-morbidities were retrospectively collected and analyzed. Predictors of drug discontinuation were examined by univariate and multivariate logistic regression analyses.

Results: One hundred and seventy seven patients were included in the study; 70% (n=120) were women with a mean age at diagnosis of 74 ± 8.4 years. Diagnosis was established by temporal artery biopsy in 127 patients (72%), ultrasound of temporal arteries in 37 patients (21%) and large vessel imaging in 10 patients (6%). At diagnosis, the median ESR and CPR were 103 mm/h and 63 mg/L, respectively. All patients were treated initially with pos corticosteroids (median daily starting dose: 41 mg), while 12 (7%) of patients received pulse steroids. At the 1st patient evaluation (median follow-up from diagnosis: 3 years), the median daily steroid dose was 5 mg, while in 34% (n= 62) and 8% (n= 14) of patients a synthetic or biologic DMARD had been added, respectively. During that period, 24% (n=43) of patients had discontinued corticosteroids and 18% (n=32) all treatments. By univariate analysis, DMARD use was associated with a higher possibility for corticosteroid discontinuation (OR=2.3, p=0.017) but by multivariate logistic regression analysis only female sex (OR=0.49, p=0.07) and age at diagnosis (OR=0.94, p=0.01) were associated with a decreased risk for corticosteroid discontinuation. Similar results were found for discontinuation of all drugs [female sex (OR=0.4, p=0.05) and age at diagnosis (OR=0.9, p=0.046)].

Conclusion: In this large GCA cohort, only one out of four patients managed to discontinue corticosteroids and 20% all treatments ~3 years after diagnosis. Multivariate analysis revealed only female sex and age at diagnosis as independent factors for treatment discontinuation.

Acknowledgement: Supported by grants from the Greek Rheumatology Society and Professional Association of Rheumatologists.

Disclosure of Interests:

None declared

DOI: 10.1136/annrheumdis-2019-eular.6298

SAT0248

CLINICAL OUTCOMES OF PATIENTS WITH GIANT CELL ARTERITIS TREATED WITH TOCILIZUMAB IN REAL-WORLD CLINICAL PRACTICE

Sebastian Unizony¹, Jinglan Pei², Paris Sidiropoulos², Jennie H. Best², Christine Birchwood², John H. Stone¹. ¹Massachusetts General Hospital, Harvard Medical School, Boston, United States of America; ²Genentech, Inc., South San Francisco, United States of America

Background: Previously, the GACTA study demonstrated the superiority of subcutaneous (SC) tocilizumab (TCZ) plus prednisone vs prednisone alone in achieving sustained glucocorticoid (GC)-free remission in patients with giant cell arteritis (GCA).¹

Objectives: To evaluate the effectiveness and safety of SC and intravenous TCZ in real-world clinical practice.

Methods: We performed a retrospective analysis of GCA patients treated with TCZ at a single center (MGH) between 2010-2018. Annual relapse rate, time to disease relapse, number of relapses, prednisone use, and adverse events (AE) before and after TCZ initiation were assessed. Disease relapse was defined as the re-appearance of clinical manifestations of GCA (e.g., cranial symptoms) that required treatment modification.

Results: A total of 60 GCA patients were included in the analysis. **Table 1** depicts the baseline characteristics and the treatments received by this cohort. The median (IQR) disease duration before TCZ use was 0.6 (0.2-1.6) years. Fifty-eight patients (96.7%) received concomitant prednisone (mean [SD] dose: 30 [18.3] mg daily) at the time of TCZ initiation. Patients received TCZ for a median (IQR) period of 0.5 (0.3-1.4) years. Before TCZ treatment, 43 patients (71.7%) had ≥1 relapse (median [IQR] time to relapse 0.5 [0.3-0.7] years). After TCZ initiation, 18 patients (30.0%) had ≥1 relapse (estimated median [IQR] time to relapse 2.1 [0.6-2.3] years). The annual rate of relapse was lower on TCZ than while off TCZ (RR=0.40 [95% CI, 0.25-0.63]; P=0.0001) (**Table 2**). Twenty-seven patients (46.6%) successfully tapered off prednisone during TCZ treatment. The incidence of AEs and serious AEs (SAE) was similar before and after TCZ initiation (**Table 2**). During TCZ treatment, however, 38 of 81 AEs were considered related or possibly related to prednisone. An AE led to TCZ discontinuation in 5 patients. No deaths occurred during the study period.

Conclusion: In this retrospective analysis, TCZ improved clinical outcomes in patients with GCA as indicated by a reduced incidence of relapses and by the ability to discontinue prednisone. The occurrence of AEs and SAEs (many due to GC) did not differ substantially while patients were on or off TCZ. These real-world findings support the previously reported efficacy and safety profile of TCZ in patients with GCA.

REFERENCE

[1] Stone JH, et al. N Engl J Med. 2017;377(4):317-328.

Table 1.. Patient baseline characteristics and treatments

	Total Patients (N = 60)
Age, mean (SD)	69.4 (9.4)
Female sex, n (%)	43 (71.7)
Caucasian ethnicity, n (%)	53 (88.3)
Clinical manifestations at disease onset	
Headache, n (%)	47 (78.3)
Scalp tenderness, n (%)	26 (43.3)
Jaw claudication, n (%)	31 (51.7)
Amaurosis fugax, n (%)	11 (18.3)
Transient blurry vision, n (%)	18 (30.0)
Diplopia, n (%)	2 (3.3)
PMR symptoms, n (%)	32 (53.3)
ESR (mm/hr), mean (SD)	72.6 (33.6)
CRP (mg/L), mean (SD)	76.1 (72.6)
Positive TA biopsy, n (%)	26 (43.3)
Prednisone dose (mg/day) at disease onset, mean (SD)	54 (19.0)
Use of other immunosuppressants before TCZ initiation, n (%)	15 (25)
Duration of disease before TCZ initiation (years), median (IQR)	0.6 (0.2-1.6)
Prednisone dose (mg/day) at TCZ initiation, mean (SD)	30 (18.3)
Received TCZ IV, n (%) [†]	22 (37.3)
Received TCZ SC, n (%) [†]	43 (72.9)
Duration of TCZ treatment (years), mean (SD)	0.9 (1.0)
Duration of TCZ treatment (years), median (IQR)	0.5 (0.3-1.4)

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; IV, intravenous; PMR, polymyalgia rheumatica; SC, subcutaneous; TA, temporal artery; TCZ, tocilizumab.

Table 2. Clinical outcomes and adverse events

	Off TCZ (N = 60)	On TCZ (N = 60)
Follow-up time, median (IQR), years	0.60 (0.20-1.57)	0.59 (0.32-1.50)
Patients with ≥ 1 relapse, n (%)	43 (71.7)	18 (30.0)
1 relapse	19 (31.7)	11 (18.3)
2 relapses	11 (18.3)	3 (5.0)
3 relapses	4 (6.7)	3 (5.0)
≥ 4 relapses	9 (15.0)	1 (1.7)
Time to relapse, median (IQR), years	0.5 (0.3-0.7)	2.1 (0.6-2.6)
Annual relapse rate (95% CI), events per year*	1.43 (0.95-2.13)	0.57 (0.33-0.98)
Patients with ≥ 1 AE, n (%)	29 (48.3)	32 (53.3)
AEs attributed to GC†	52 / 59 (88.1)	38 / 81 (46.9)
AEs attributed to TCZ‡	NA	27 / 81 (33.3)
AEs attributed to GC or TCZ†	52 / 59 (88.1)	53 / 81 (65.4)
Patients with ≥ 1 SAE, n (%)	6 (10.0)	6 (10.0)
SAEs attributed to GC	7 / 8 (87.5)	4 / 9 (44.4)
SAEs attributed to TCZ	NA	3 / 9 (33.3)
SAEs attributed to GC or TCZ	7 / 8 (87.5)	6 / 9 (66.7)

AE, adverse event; CI, confidence interval; GC, glucocorticoids; IQR, interquartile range; NA, not applicable; SAE, serious AE; TCZ, tocilizumab.

* Rates are estimated from a Poisson regression model with ongoing treatment (TCZ and GC combinations), age, smoking history, new or relapsing GCA as covariates, and random patient effect.

† Multiple occurrences of AEs are included. Denominators are all AEs or SAEs.

Disclosure of Interests: Sebastian Unizony Grant/research support from: F. Hoffmann-La Roche, Genentech, Consultant for: Kiniksa, Sanofi, GSK, Jinglan Pei Employee of: Genentech, Paris Sidiropoulos Employee of: Genentech, Jennie H. Best Shareholder of: Genentech, Employee of: Genentech, Christine Birchwood Employee of: Genentech, John H. Stone Grant/research support from: F. Hoffmann-La Roche, Genentech, Xencor, Consultant for: Chugain, F. Hoffmann-La Roche, Genentech, Xencor
DOI: 10.1136/annrheumdis-2019-eular.1239

SAT0249 IGA VASCULITIS ASSOCIATED WITH INFLAMMATORY BOWEL DISEASE. A RETROSPECTIVE STUDY

Michel Villatoro Villar¹, Cynthia S. Crowson^{1,2}, Ashima Makol¹, Kenneth J. Warrington¹, Steven R. Ytterberg¹, Matthew Koster¹. ¹Mayo Clinic, Rheumatology, Rochester MN, United States of America; ²Mayo Clinic, Health Sciences Research, Rochester MN, United States of America

Background: Leukocytoclastic vasculitis has been reported in patients with inflammatory bowel disease (IBD) but cases of IgA vasculitis (IgAV) in IBD are considered rare.

Objectives: The purpose of this study was to describe the baseline characteristics and outcome of a series of patients with IBD and IgAV.

Methods: Biopsy-proven IgAV-patients with prior history of IBD were identified retrospectively at Mayo Clinic, Rochester, MN. Data were abstracted from direct medical chart review. Each IBD-IgAV case was matched to two controls with IgAV but without IBD history based on age, sex and baseline renal function at time of IgAV onset.

Results: A total of 9 patients were included in the study group (6 male, 3 female). Crohn's disease (CD) and Ulcerative Colitis (UC) were present in 7 and 2 patients, respectively. The mean length of time between IBD diagnosis and onset of IgAV was 17.3 $\pm$ 19.9 years. For patients on biologic treatment for IBD, the mean length of time between initiation of biologic and onset of IgAV was 3.3 $\pm$ 3.8 years (range 0-12 years). Active IBD at IgAV-onset was present in 56% (5/9) of patients.

Tumor necrosis factor inhibitors (TNFi) were the most frequent biologics used for IBD (8, 89%); infliximab was the most common (7, 78%). At IgAV-onset, only 5 patients were on treatment with TNFi; two subsequently discontinued, two switched to another TNFi (adalimumab), and one patient continued. At last follow-up, two of three patients that remained on TNFi had full resolution of IgAV despite ongoing TNFi use. Comparison of baseline characteristics between cases with IBD-IgAV and matched non-IBD IgAV controls is demonstrated in Table 1. No differences were seen in regards to development of end-stage renal disease, resolution of hematuria and/or proteinuria, time to complete IgAV response or first IgAV relapse.

Conclusion: Baseline characteristics and outcomes of patients with IBD-IgAV are similar to those with IgAV without IBD. Development of IgAV is not limited to patients with clinically active IBD. Whether use of TNFi is related to the pathogenesis of IgAV in some patients with IBD remains unclear. Further research into the pathophysiologic connection between IBD and IgAV is needed.

REFERENCES

- [1] Saint Marcoux B, De Bandt M. Vasculitides induced by TNF α antagonists: a study in 39 patients in France. Joint, bone, spine : revue du rhumatisme. 2006;73(6):710-3.

- [2] Sy A, Khalidi N, Dehghan N, Barra L, Carrette S, Cuthbertson D, et al. Vasculitis in patients with inflammatory bowel diseases: A study of 32 patients and systematic review of the literature. Seminars in arthritis and rheumatism. 2016;45(4):475-82.
- [3] Ko JS, Uberti G, Napekoski K, Patil DT, Billings SD. Cutaneous manifestations in inflammatory bowel disease: a single institutional study of non-neoplastic biopsies over 13 years. Journal of cutaneous pathology. 2016;43(11):946-55.
- [4] Ozen S, Pistorio A, Iusan SM, Bakaloglu A, Herlin T, Brik R, et al. EULAR/PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: Final classification criteria. Ann Rheum Dis. 2010;69(5):798-806.
- [5] Laresche C, Locatelli F, Biver-Dalle C, Nachury M, Heyd B, Koch S, et al. Severe Henoch-Schönlein purpura complicating infliximab therapy for ulcerative colitis. Cutis. 2017;99(1):E20-e2.

Table 1: Baseline characteristics of patients with inflammatory bowel disease and immunoglobulin-A vasculitis compared to patients with IgAV without inflammatory bowel disease

Characteristic	IBD-IgAV (n=9)	Non-IBD IgAV (n=18)	p-value
Age at IgAV diagnosis, years*	40.8 $\pm$ 17.5	40.7 $\pm$ 17.4	0.96
Male sex	6 (67%)	12 (67%)	1.00
Length of follow-up, years*	1.3 $\pm$ 2.6	1.1 $\pm$ 1.2	---
Creatinine, mg/dL*	1.0 $\pm$ 0.4	0.9 $\pm$ 0.3	0.59
eGFR (ml/min/1.73m ²)*	91.4 $\pm$ 31.6	96.4 $\pm$ 22.9	0.72
C-reactive protein, mg/L*	23.3 $\pm$ 12.2	27.0 $\pm$ 27.4	0.62
Renal involvement	7 (78%)	10 (56%)	0.26
Microscopic hematuria	5 (56%)	7 (39%)	0.41
Proteinuria	7 (78%)	8 (44%)	0.10
Palpable purpura	9 (100%)	18 (100%)	1.00
Abdominal Ischemic symptoms	3 (33%)	2 (11%)	0.161
Disease status at IgAV diagnosis			
Active IBD	5 (56%)	-	-
TNF- α inhibitor at diagnosis	2/5 (40%)	-	-
o Ever TNF- α inhibitor	5/5 (100%)	-	-
Inactive IBD	4 (44%)	-	-
TNF- α inhibitor at diagnosis	3/4 (75%)	-	-
o Ever TNF- α inhibitor	3/4 (75%)	-	-

IBD, inflammatory bowel disease; IgAV, immunoglobulin-A vasculitis; eGFR, estimated glomerular filtration rate; TNF- α , tumor necrosis factor- α inhibitor.

*Mean $\pm$ standard deviation

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.7413

SAT0250 MUSCLE INVOLVEMENT REVEALED BY 18F-PET-CT IN POLYMYALGIA RHEUMATICA

Daniel Wendling¹, Maxime Sondag¹, Nicolas Giraud¹, Mickaël Chouk¹, Hatem Boulahdour², Clément Prati¹, Frank Verhoeven¹. ¹CHRU, Rheumatology, Besançon, France; ²CHRU, Nuclear Medicine, Besançon, France

Background: Imaging techniques in polymyalgia rheumatica (PMR) have revealed mainly bursitis, tenosynovitis, capsulitis and enthesitis. This is also the case for 18F-FDG-PET-CT. This technique, which has shown its interest in the diagnosis of PMR, allows an assessment of the metabolic activity of the entire musculoskeletal system and in particular of muscle structures.

Objectives: The objective of this work was to evaluate muscle damage using 18F-FDG-PET-CT in PMR.

Methods: This is a retrospective study including patients with PMR (ACR/EULAR 2012 criteria) seen in our department, who had an 18F-FDG PET-CT examination between September 2012 and November 2018. A control group consisting of subjects without rheumatological manifestations who had such an examination as part of neoplastic research or neoplastic disease control was also evaluated. PET evaluation included 17 sites suggestive of PMR, as previously reported [1], leading to a global PET score ranging from 0 to 51. Muscle hypermetabolism areas were similarly rated according to the same Goerres classification [2] (0 = no uptake; 1 = slight uptake, less than liver; 2 = uptake like liver; 3 = uptake higher than liver). Muscle activity sites have been identified. A comparison of PMR patients with and without muscle involvement was performed using the Mann Whitney or Fisher's exact test.