Conclusion: The finding of thoracic aortitis following aortic replacement surgery does not affect short-term post-operative mortality or morbidity. Corticosteroid therapy in the immediate post-operative period did not impact upon short-term outcomes.

REFERENCES:

- [1] Quimson L, Mayer A, Capponi S, et al. Comparison of aortitis versus non-inflammatory aortic aneurysms among patients who undergo open aortic aneurysm repair. *Arthritis Rheum* 2020;72:1154-59.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.1531

POS0805

TREATMENT OF GIANT CELL ARTERITIS WITH TOCILIZUMAB: A RETROSPECTIVE COHORT STUDY OF 119 PATIENTS

J. Rakholiya¹, M. Koster¹, H. Langenfeld², C. S. Crowson^{1,2}, A. Abril³, P. Bansal⁴, L. Mertz⁵, A. Rodriguez-Pla⁵, R. Sehgal⁴, B. Wang³, K. J. Warrington¹. ¹Mayo Clinic, Rheumatology, Rochester, United States of America; ²Mayo Clinic School of Health Sciences, Biomedical Statistics and Informatics, Rochester, United States of America; ³Mayo Clinic, Rheumatology, Jacksonville, United States of America; ⁴Mayo Clinic Health System, Rheumatology, Eau Claire, United States of America; ⁵Mayo Clinic, Rheumatology, Scottsdale, United States of America

Background: Giant cell arteritis (GCA) is an inflammatory condition of medium- and large-sized arteries. Prospective clinical trials have demonstrated the efficacy of tocilizumab (TCZ) for treatment of patients with GCA (1). However, there is a limited data on the use of TCZ in routine clinical practice.

Objectives: To evaluate the efficacy and safety of TCZ in a retrospective cohort study of patients with GCA treated with TCZ.

Methods: Patients with GCA treated with TCZ at 4 clinical centers of a single tertiary care institution (2000-2020) were identified. The diagnosis of GCA was confirmed by at least one of the following modalities: 1. Arterial biopsy 2. Large vessel imaging 3. Clinical diagnosis of GCA meeting ACR classification criteria and established by a rheumatologist. Patient demographics, clinical presentation, laboratory studies, treatment course and adverse events were abstracted from the medical record; only patients with at least 6 months of follow-up after TCZ initiation were included. Kaplan-Meier methods were used to estimate time to TCZ discontinuation and time to first relapse after TCZ discontinuation. Poisson regression models were used to compare relapse rates before and after TCZ initiation.

Results: The study cohort included 119 patients [61% female; mean (SD) age at GCA diagnosis 70.3 (8.2) years]. The majority of patients (89%) had a biopsy-proven and/or imaging-based diagnosis of GCA, while 13 (11%) had a clinical diagnosis of GCA. In addition to glucocorticoids, 40 (34%) patients received other immunosuppressive agents prior to TCZ. The method of initial TCZ administration was subcutaneous (162mg/ml) weekly in 48 (41%), subcutaneous every other week in 20 (17%), monthly 4mg/kg infusions in 34 (29%), monthly 8mg/kg infusions in 14 (12%) and non-standard dosing in 3 remaining patients. The median (IQR) duration from GCA diagnosis to TCZ initiation was 4.8 (1.2-22.0) months and the median (IQR) duration of TCZ treatment was 18 (11-28) months. The mean (SD) dose of prednisone at TCZ initiation was 31 (19) mg/day and was reduced to a mean (SD) dose of 3.9 (6.7) mg/day at TCZ discontinuation/last follow-up visit. The relapse rate per year decreased 43% from 0.77 to 0.44 after the initiation of TCZ (RR=0.57; 95% CI: 0.44-0.75; p<0.001). The mean (SD) ESR and CRP decreased from 22 (20) mm/hour to 6 (9.2) mm/hour and from 19.1 (25) mg/L to 5.4 (16.6) mg/L, respectively from TCZ initiation to TCZ discontinuation/last follow-up visit. At 2 years of follow-up, 67% of patients had discontinued glucocorticoids. At last follow up, 46 patients had discontinued TCZ, only 14 of which were due to adverse events. The median time to TCZ discontinuation was 2.9 years. Only 17% (95%CI: 10-24%) had discontinued by 1 year after TCZ initiation and 38% (95% CI: 26-47%) had discontinued by 2 years. The most common adverse events were infections and cytopenias. While on TCZ, 1 patient developed new onset vision loss related to GCA and 1 patient, without history of diverticulitis, had bowel perforation. Among those discontinuing TCZ, 61% had relapsed at least once by 1 year after TCZ discontinuation.

Conclusion: In this large single institution cohort of patients with GCA, TCZ use resulted in a significantly reduced relapse rate and reduction in glucocorticoid dosage. Overall, patients tolerated long-term use with only 12% discontinuing due to adverse events. However, over half of patients stopping TCZ had a subsequent flare; highlighting ongoing use may be required beyond two years in several patients with GCA to maintain remission.

REFERENCES:

- [1] Stone JH, et al. Trial of Tocilizumab in Giant-Cell Arteritis. *N Engl J Med*. 2017 Jul 27;377(4):317-328. doi: 10.1056/NEJMoa1613849. PMID: 28745999.

- [2] Calderón-Goercke M, et al. Tocilizumab in giant cell arteritis. Observational, open-label multicenter study of 134 patients in clinical practice. *Semin Arthritis Rheum*. 2019 Aug;49(1):126-135. doi: 10.1016/j.semarthrit.2019.01.003. Epub 2019 Jan 5. PMID: 30655091.

Disclosure of Interests: Jigisha Rakholiya: None declared, Matthew Koster: None declared, Hannah Langenfeld: None declared, Cynthia S. Crowson: None declared, Andy Abril: None declared, Pankaj Bansal: None declared, Lester Mertz: None declared, Alicia Rodriguez-Pla: None declared, Rahul Sehgal: None declared, Benjamin Wang: None declared, Kenneth J Warrington Grant/research support from: Research support: Kiniksa, Eli Lilly
DOI: 10.1136/annrheumdis-2021-eular.1674

POS0806

FINDINGS CONSISTENT WITH SUBCLINICAL VASCULITIS IN PATIENTS WITH NEW ONSET POLYMYALGIA: A SYSTEMATIC LITERATURE REVIEW AND A META-ANALYSIS OF COHORT DATA

D. S. Gozzoli¹, A. Hemmig², L. Hemkens^{3,4,5}, L. Werlen³, H. Ewald⁶, C. Berger⁷, D. Kyburz^{2,8}, S. Imfeld⁹, M. Aschwanden⁹, M. Stegert², D. Camellino¹⁰, M. A. Cimmino¹¹, C. Campochiaro¹², A. Tomelleri¹², L. Henckaerts¹³, D. Blockmans¹³, P. Moya¹⁴, H. Corominas¹⁵, R. Buchanan^{16,17}, C. Owen^{16,17}, Y. Van Sleen¹⁸, E. Brouwer¹⁸, H. Ymashita¹⁹, T. Daikeler². ¹University Hospital of Basel, Department of Internal Medicine, Basel, Switzerland; ²University Hospital of Basel, Department of Rheumatology, Basel, Switzerland; ³University Hospital of Basel, Department of Clinical Research, Basel, Switzerland; ⁴Stanford University, Meta-Research Innovation Center at Stanford (METRICS), Palo Alto, CA 94305, United States of America; ⁵Berlin Institute of Health, Meta-Research Innovation Center Berlin (METRIC-B), Berlin, Germany; ⁶University of Basel, Medical Library, Basel, Switzerland; ⁷University Hospital of Basel, Departments of Biomedicine and Internal Medicine, Translational Immunology and Medical Outpatient Clinic, Basel, Switzerland; ⁸University of Basel, Department of Biomedicine, Basel, Switzerland; ⁹University Hospital of Basel, Department of Angiology, Basel, Switzerland; ¹⁰"La Colletta" Hospital, Division of Rheumatology, Arenzano, Italy; ¹¹University of Genoa, Research Laboratory and Academic Division of Clinical Rheumatology, Department of Internal Medicine, Genoa, Italy; ¹²San Raffaele Scientific Institute, Unit of Immunology, Rheumatology, Allergy and Rare Diseases (UnIRAR), Milan, Italy; ¹³KU Leuven, University Hospitals Leuven, Department of General Internal Medicine, Department of Microbiology, Immunology and Transplantation, Leuven, Belgium; ¹⁴Hospital de la Santa Creu i Sant Pau, Rheumatology Unit, Department of Internal Medicine, Barcelona, Spain; ¹⁵Hospital Universitari de la Sant creu i Sant Pau, Servei de Reumatologia, Barcelona, Spain; ¹⁶Austin Health, Department of Rheumatology, Heidelberg, Victoria, Australia; ¹⁷University of Melbourne, Department of Medicine, Parkville, Victoria, Australia; ¹⁸University of Groningen, University Medical Center Groningen, Groningen, Netherlands; ¹⁹National Center for Global Health and Medicine, Division of Rheumatic Diseases, Tokyo, Japan

Background: GCA is characterized by cranial symptoms but imaging techniques show that patients with non-specific symptoms such as systemic inflammation or PMR may have undiagnosed large vessel (LV) GCA¹. Although silent GCA in patients with clinically isolated PMR may have consequences for patients' outcome, little is known about its prevalence and characteristics of affected patients.

Objectives: To review data on the prevalence of silent GCA in newly diagnosed PMR patients without cranial GCA symptoms and to analyze which characteristics are associated with vascular involvement among PMR patients.

Methods: We systematically screened PubMed, Embase and Web of Science databases and included studies screening for GCA in steroid naïve PMR patients without cranial symptoms consistent with GCA. Authors of the publications that used PET for vasculitis screening were invited to share their individual patient data (IPD) for a meta-analysis. We sought to define patient characteristics that were associated with vasculitis using univariable mixed effects logistic regression models with vascular involvement as the outcome, missing values were imputed using multilevel joint modeling multiple imputation. To fit a multivariable model with the candidate predictors we excluded variables that were hypothesized to have less medical relevance for the outcome and highly correlated inflammation markers (ESR, Lc).

Results: Out of the 3047 studies screened independently by 2 authors (DG and TD), 13 fulfilled the inclusion criteria. These studies (published 1963-2019) reported on 543 PMR patients examined by temporal artery biopsy (n=175), ultrasound (n=110), PET or PET-CT (n=258). 115 PMR patients were diagnosed with GCA (21.2%), with prevalence ranging from 0-92%.

We collected IPD for 243 patients from 4 cohorts using PET and 3 using PET/CT for GCA diagnosis. The overall median age of patients was 72.3 years (IQR 66.4-78.0) and vasculitis was found in 65 patients (26.7%) (table 1).

Table 1.

	Overall	PMR	PMR+GCA
n (%)	243	178 (73.3)	65 (26.7)
Female sex (%)	146 (60.1)	98 (55.1)	48 (73.8)
Shoulder girdle pain (%)	236 (97.1)	174 (97.8)	62 (95.4)
Pelvic girdle pain (%)	174 (71.6)	127 (71.3)	47 (72.3)
Inflammatory back pain (%)			
No	107 (44.0)	83 (46.6)	24 (36.9)
Yes	106 (43.6)	70 (39.3)	36 (55.4)
Lower limb pain (%)			
No	87 (35.8)	61 (34.3)	26 (40.0)
Yes	81 (33.3)	68 (38.2)	13 (20.0)
Weight loss (%)	112 (46.1)	78 (43.8)	34 (52.3)
CRP (mg/l) (median [IQR])	46.0 [19.0, 77.7]	44.0 [16.9, 74.2]	52.0 [27.9, 85.0]
ESR (mm/h) (mean (SD))	65.2 (30.3)	62.7 (30.2)	72.3 (29.7)
Hemoglobin (g/dl) (mean (SD))	12.1 (1.5)	12.2 (1.5)	11.7 (1.6)
Thrombocytes (1e+09/ml) (mean (SD))	341.9 (106.3)	323.9 (103.2)	375.8 (104.6)

In the univariable analyses the following factors were most strongly associated with vasculitic PET findings: female sex (OR 2.31, CI 1.17-4.58), inflammatory back pain (OR 2.73, CI 1.32-5.64), temperature $>37^{\circ}$ (OR 1.83, CI 0.90-3.7), weight loss (OR 1.83, CI 0.96-3.51), thrombocytosis (i.e., patients with a thrombocyte count 1 SD above mean have an OR of 1.51, CI 1.05-2.18), anemia (i.e., 1 g/dl decrease in Hb below mean corresponds to an OR of 1.25, CI 1.00-1.56). Patients with lower limb pain were less likely to have vasculitis (OR 0.43, CI 0.19-0.95). The estimated ORs were very similar in the multivariable model although the 95% CIs became wider.

Conclusion: Although the prevalence across published studies showed substantial variation, 6 out of 13 studies reported a prevalence of silent GCA in 18-40% of all PMR patients. The exploratory analysis of the collected IPD identified female sex, inflammatory back pain, fever, weight loss, absence of lower leg pain, thrombocytosis and anemia as factors associated with LV-GCA. These findings should be validated in future prospective cohort studies. The presence or absence of these factors may further aid in diagnosing LV-GCA in PMR patients.

REFERENCES:

- [1] Buttgeriet F, Dejaco C, Matteson EL, Dasgupta B. Polymyalgia Rheumatica and Giant Cell Arteritis: A Systematic Review. *JAMA*. 2016 Jun 14;315(22):2442-58.

Acknowledgements: The study is funded by the "Schweizerische Stiftung für die Erforschung der Muskelkrankheiten (SSEM)".

Disclosure of Interests: Daniele Silvio Gozzoli: None declared, Andrea Hemmig: None declared, Lars Hemkens: None declared, Laura Werlen: None declared, Hannah Ewald: None declared, Christoph Berger: None declared, Diego Kyburz Grant/research support from: DK reports personal fees from Abbvie, Gilead, Lilly, Novartis and Pfizer, outside of the submitted work, Stephan Imfeld: None declared, Markus Aschwanden: None declared, Mihaela Stegert: None declared, Dario Camellino: None declared, Marco Amedeo Cimmino: None declared, Corrado Campochiaro Grant/research support from: personal fees from Roche, Alessandro Tomelleri: None declared, Liesbet Henckaerts: None declared, Daniel Blockmans Speakers bureau: Paid speaker for Roche, Consultant of: Paid consultant for Roche, Patricia Moya: None declared, Hector Corominas: None declared, Russell Buchanan: None declared, Claire Owen Speakers bureau: CO has received speaking honoraria from Roche, Janssen, Novartis and Pfizer, and meeting sponsorship from Roche, UCB and Janssen, Yannick van Sleen: None declared, Elisabeth Brouwer Speakers bureau: E. Brouwer as an employee of the UMCG received speaker fees and consulting fees from Roche in 2017, 2018 which were paid to the UMCG, Consultant of: E. Brouwer as an employee of the UMCG received speaker fees and consulting fees from Roche in 2017, 2018 which were paid to the UMCG, Hiroyuki Ymashita: None declared, Thomas Daikeler: None declared

DOI: 10.1136/annrheumdis-2021-eular.1803

POS0807

RETINAL VESSEL CALIBERS AS A NON-INVASIVE BIOMARKER OF INFLAMMATORY BURDEN IN PRIMARY SYSTEMIC VASCULITIS

O. Argyropoulou¹, E. Aissopou¹, A. Argyris¹, A. Goules¹, C. Mavragani², N. Tentolouris³, P. Sfrikakis³, P. Vlachoyiannopoulos¹, A. Tzioufas¹, A. Proterogerou¹. ¹School of Medicine, National and Kapodistrian University of Athens, Pathophysiology, Athens, Greece; ²School of Medicine, National and Kapodistrian University of Athens, Physiology, Athens, Greece; ³Medical School, National and Kapodistrian University of Athens, Athens, First Department of Propaedeutic Internal Medicine, Athens, Greece

Background: Primary Systemic Vasculitides (PSV) constitute a heterogeneous group of rare and potentially life-threatening autoimmune diseases, characterized by a varying degree of inflammatory response, leading to local or generalized vascular disease. Vessel involvement accounts for the micro- and macrovascular complications of the disease, along with the classic risk factors including, among others age and chronic use of steroids (1). Early identification of high-risk

patients for cardiovascular disease (CVD) development and the contribution of inflammation towards this adverse outcome are still unmet needs. Alterations of retinal microcirculation have been independently associated with increased CVD risk in the general population (2). The potential changes of retinal vasculature and their association with disease activity and the magnitude of inflammation have not been studied in PSV so far.

Objectives: To explore the effect of disease activity and inflammation on retinal microcirculation in PSV, classified according to vessel size as large, medium or small vessel vasculitides (LVV, MVV or SVV respectively), and polymyalgia rheumatica (PMR) without vasculitis.

Methods: Fifty-nine patients, 43 with active disease [30 active vasculitis (12 LVV, 4 MVV, 14 SVV) and 13 PMR] and 16 with chronic inactive disease (13 vasculitis, 3 PMR) were studied. All patients were matched at 1:1 ratio with 59 controls, without underlying autoimmune/autoinflammatory disorder, neoplasia or infection, according to age, gender, CVD history, BMI, smoking, arterial hypertension, dyslipidemia, diabetes mellitus and treatment related to comorbidities. A total of 32 rheumatoid arthritis (RA) patients with mild to moderate inflammatory component as defined by standard of care acute phase reactants (ESR and CRP) and 16 chronic RA patients with normal ESR and CRP levels, matched 1:1 according to all the above parameters with 32 active and 16 inactive vasculitis/PMR patients respectively, served as disease controls. Digital retinal images were obtained and retinal vessel calibers were measured with a validated software to determine central retinal arteriolar and venular equivalents ratio (CRAE and CRVE respectively). For 16 patients with active vasculitis/PMR, retinal examination was performed in two time points (baseline and ≤ 6 months later).

Results: In the overall population, patients with active Vasculitis/PMR had increased CRVE (213.8 ± 21.7 vs 201.3 ± 17.1 , $p < 0.001$) and CRAE (180.0 ± 19.2 vs 164.1 ± 17.5 , $p < 0.001$) compared to healthy controls. Separating patients according to disease type, we found that in LVV, MVV and PMR, CRAE ($p \leq 0.05$) and CRVE ($p < 0.05$) were increased compared to control group, while in SVV only CRAE was increased ($p < 0.001$). Interestingly, chronic patients with disease in remission displayed higher CRAE compared to matched controls (179.8 ± 17.2 vs 169.1 ± 11.1 , $p = 0.006$). After immunosuppressive treatment for ≤ 6 months CRVE and to a lesser extent CRAE were reduced ($p = 0.048$ and 0.149 respectively) with a stronger statistical significance found in the Vasculitis group ($p = 0.026$ and 0.069 respectively). Pearson's linear correlation coefficient in active disease state (at baseline examination) revealed positive bivariate correlation only between CRVE with ESR and CRP. Patients with Vasculitis/PMR had also increased CRAE when compared to RA patients in both active and inactive disease status (182.8 ± 19.4 vs 170.1 ± 18.4 , $p < 0.001$ and 179.8 ± 17.2 vs 166.3 ± 17.9 , $p = 0.005$ respectively).

Conclusion: Systemic inflammation alters retinal microcirculation in both a reversible (venules) and irreversible (arterioles) way, independently of PSV form. Thus, common disease specific pathogenetic mechanisms related to inflammation may be implicated in vascular remodeling. Sequential follow-up of PSV patients will address whether retinal vessel calibers may serve as a biomarker of disease activity and CVD development.

REFERENCES:

- [1] Argyropoulou OD et al. *Curr Opin Rheumatol* 2017.
- [2] Shaohua G et al. *Current Atherosclerosis reports* 2020.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.1950

POS0808

CHARACTERISTICS OF GIANT CELL ARTERITIS FLARES AFTER SUCCESSFUL TREATMENT WITH TOCILIZUMAB: RESULTS FROM THE LONG-TERM EXTENSION OF A RANDOMIZED CONTROLLED PHASE 3 TRIAL

S. Unizony¹, S. Mohan², J. Han², J. H. Stone¹. ¹Harvard Medical School, Massachusetts General Hospital Rheumatology Unit, Boston, United States of America; ²Genentech, US Medical Affairs, South San Francisco, United States of America

Background: GiACTA investigated tocilizumab (TCZ) for the treatment of giant cell arteritis (GCA).

Objectives: To investigate disease flare characteristics after successful treatment with TCZ in GiACTA.

Methods: We report a post hoc analysis from part 2 of GiACTA. Part 1 was a 52-week, double-blind, randomized controlled period and part 2 was a 2-year open-label follow-up. In part 1, patients received TCZ 162mg subcutaneously every week or every other week with a 26-week prednisone taper or placebo plus a 26- or 52-week prednisone taper. Patients who were in remission at week 52 were to enter part 2 on no TCZ treatment. Part 2 treatment was at the investigator's discretion. We report characteristics of first disease flare in patients assigned to TCZ in part 1 who were in sustained remission at week 52 and experienced flare in part 2. Flare was defined as reappearance of cranial symptoms (headaches, jaw claudication, visual manifestations, scalp tenderness) or polymyalgia rheumatica (PMR) symptoms or elevation of erythrocyte sedimentation rate (ESR) ≥ 30 mm/h attributable to GCA that required treatment.