

**Methods:** Observational retrospective study on consecutive patients of our GCA fast track clinic. The gold standard diagnosis was the opinion of the clinical doctor after at least 6 months of follow-up. US recorded videos, of every explored vessel temporal (common trunk, frontal and parietal branches), subclavian and axillary arteries were reviewed and the IMT of each of them was measured in systole and diastole peaks for comparison. We define an ultrasound result as positive with cut-off values of IMT  $\geq 0.34$  mm for frontal and parietal branches,  $\geq 0.42$  mm for the common trunks of TA, and  $\geq 1$  mm for the axillary and subclavian arteries. Demographic data of the included patients were also collected.

**Results:** We have included 72 cases, 36 with GCA diagnosis and 36 without GCA (controls). The mean values of age and sex, as well as the IMT in systole and diastole of each vessel are shown in Table 1. There were not significant differences in sex but patients without GCA were younger ( $p < 0.01$ ). The US IMT measurements at the systolic and diastolic times showed statistically significant differences in all the explored vessels, as in patients with GCA as in the control non GCA group. All the IMT measured in diastole showed higher and statistically significant values than those measured in systole, with a mean increment of measurement of 5.3% and 6.5% in TA and 6.4% and 5.6% in LV, respectively in the GCA and control group. This result can be of clinical relevance because if we used diastolic measures, instead of systolic measures, 5/36 (13.8%) cases in controls had halo sign in one isolated vessel (2 cases in parietal right, 1 in common trunk right, 1 in subclavian right, 1 in subclavian left). However, in GCA patients, the number of patients with halo sign did not change, but the number of pathological vessels was increased when the measured was performed in diastole (1 frontal right branch, 1 common trunk right, 4 frontal left branches, 5 common trunks left and 2 axillary right), so this could have influence in the assessment of the disease.

**Conclusion:** There are significant differences between the IMT measured in systolic and diastolic peaks, with higher values in diastole. The differences are relatively small but may increase the number of false positives (13.8%) in controls, and the number of affected vessels in the GCA group. This should be considered in the diagnosis and assessment of GCA.

**Table 1. Changes in the IMT values in systole and diastole in GCA and non GCA patients**

Category	No GCA			GCA		
	Systole	Diastole	p	Systole	Diastole	p
N	36	36		36	36	
Sex ♂ / ♀ (n)	14/22	14/22		17/19	17/19	
Age years (mean $\pm$ SD)	74.4 $\pm$ 9.4	74.4 $\pm$ 9.4		80.8 $\pm$ 6.6	80.8 $\pm$ 6.6	
TA Frontal (right)	0.23 $\pm$ 0.56	0.24 $\pm$ 0.60	0.001	0.36 $\pm$ 0.15	0.39 $\pm$ 0.16	0.001
TA Frontal (left)	0.21 $\pm$ 0.44	0.23 $\pm$ 0.46	0.001	0.37 $\pm$ 0.18	0.39 $\pm$ 0.18	0.010
TA Parietal (right)	0.21 $\pm$ 0.40	0.23 $\pm$ 0.41	0.001	0.42 $\pm$ 0.21	0.44 $\pm$ 0.21	0.001
TA Parietal (left)	0.21 $\pm$ 0.45	0.23 $\pm$ 0.43	0.001	0.38 $\pm$ 0.20	0.40 $\pm$ 0.21	0.001
TA Common (right)	0.25 $\pm$ 0.76	0.26 $\pm$ 0.74	0.001	0.48 $\pm$ 0.26	0.52 $\pm$ 0.28	0.001
TA Common (left)	0.23 $\pm$ 0.50	0.23 $\pm$ 0.50	0.001	0.44 $\pm$ 0.20	0.44 $\pm$ 0.20	0.001
Axillary (right)	0.57 $\pm$ 0.14	0.60 $\pm$ 0.14	0.001	0.76 $\pm$ 0.30	0.82 $\pm$ 0.29	0.001
Axillary (left)	0.54 $\pm$ 0.13	0.57 $\pm$ 0.13	0.001	0.72 $\pm$ 0.29	0.76 $\pm$ 0.32	0.010
Subclavian (right)	0.64 $\pm$ 0.15	0.68 $\pm$ 0.15	0.001	0.79 $\pm$ 0.27	0.85 $\pm$ 0.30	0.001
Subclavian (left)	0.61 $\pm$ 0.16	0.64 $\pm$ 0.16	0.001	0.80 $\pm$ 0.33	0.85 $\pm$ 0.34	0.001

GCA= giant cell arteritis; N= number of patients; SD= standard deviation; TA temporal artery. Measures are showed in mm.

#### REFERENCES: NIL.

**Acknowledgements:** NIL.

**Disclosure of Interests:** None Declared.

**DOI:** 10.1136/annrheumdis-2023-eular.3038

POS0737

#### AGE AND SEX BIAS OF ULTRASOUND SCORES FOR DIAGNOSIS OF GIANT CELL ARTERITIS: INSIGHTS FROM THE PROSPECTIVE, LONGITUDINAL HAS-GCA STUDY

**Keywords:** Ultrasound, Imaging, Vasculitis

K. Van der Geest<sup>1</sup>, A. Sebastian<sup>2,3,4</sup>, E. Conticini<sup>5</sup>, S. Inness<sup>3</sup>, J. Jackson<sup>3</sup>, A. Kayani<sup>2</sup>, M. Khurshid<sup>6</sup>, G. Klinowski<sup>7,8</sup>, P. Macchioni<sup>7</sup>, D. Prieto-Peña<sup>9</sup>, C. Salvarani<sup>7,8</sup>, M. Tariq<sup>2</sup>, A. Tomelleri<sup>10</sup>, B. Dasgupta<sup>2,3</sup>. <sup>1</sup>University of Groningen, University Medical Center Groningen, Rheumatology and Clinical Immunology, Groningen, Netherlands; <sup>2</sup>Mid and South Essex University Hospital Groups, Southend University Hospital, Rheumatology, Westcliff-on-sea, United Kingdom; <sup>3</sup>University of Essex, School of Sport, Rehabilitation and Exercise science, Colchester, United Kingdom; <sup>4</sup>University Hospital Limerick, Rheumatology, Limerick, Ireland; <sup>5</sup>University of Siena, Rheumatology Unit, Department of Medicine, Surgery and Neurosciences, Siena, Italy; <sup>6</sup>University Hospitals Dorset NHS Foundation Trust, Rheumatology, Dorset, United Kingdom; <sup>7</sup>Azienda USL - IRCCS - di Reggio Emilia, Rheumatology, Reggio

Emilia, Italy; <sup>8</sup>University of Modena and Reggio Emilia, Rheumatology, Modena, Italy; <sup>9</sup>Marqués de Valdecilla University Hospital, Rheumatology, Santander, Spain; <sup>10</sup>San Raffaele Hospital, Unit of Immunology, Rheumatology, Allergy and Rare Diseases, Milan, Italy

**Background:** Giant cell arteritis (GCA) is an age-linked, critically ischaemic vasculitis where ultrasound (US) serves as an important tool enabling urgent diagnosis and therapy. US scores have been developed to quantify the arterial inflammation in GCA [1,2], but optimal diagnostic cut-off points and potential bias of age and sex on these values remain un-elucidated.

**Objectives:** To compare the diagnostic accuracy of previously reported US scores for GCA, and to determine whether these scores require correction for age and sex.

**Methods:** The HAS-GCA study, with a prospective, multicentre, cohort design, recruited 229 consecutive patients with suspected, new-onset GCA from 7 sites located in the UK, Italy, Spain and the Netherlands. All cases underwent bilateral US evaluation of the three temporal artery segments (common superficial, parietal, frontal) and axillary arteries. Intimal-medial thickness (IMT) was measured in each segment. Three ultrasound scores were calculated based on the IMT: halo count (HCount) [1], Halo Score (HScore) [2] and the OMERACT GCA US Score (OGUS) [2]. The definition of halo was based on previously reported IMT cut-off values [3]. The reference standard was the final clinical diagnosis after 6 months follow-up. ROC with AUC analysis was performed. Optimal cut-off points were determined by Youden Index. Group comparisons were made by Mann Whitney U test and correlations by Spearman's rank correlation coefficient. P values  $< 0.05$  were considered statistically significant.

**Results:** A final diagnosis of GCA was made in 84 patients, whereas 145 cases received an alternative diagnosis (i.e. non-GCA patients). HCount, HScore and OGUS were significantly higher in GCA patients than non-GCA patients. In non-GCA patients, all three scores were significantly higher in men than women, and showed a statistically significant, positive correlation with age. In GCA patients, the US scores showed a positive correlation (HCount and OGUS) or strong trend for a positive correlation (HScore) with age; and the HCount was significantly higher in men. All scores effectively discriminated GCA from non-GCA patients as indicated by ROC analysis: AUC for halo count 0.936 (0.899-0.974), Halo Score 0.894 (0.848-0.939) and OGUS 0.937 (0.902-0.973). The optimal diagnostic cut-off points were: 2 for HCount (sens 86%, spec 92%), 17 for HScore (sens 76%, spec 91%) and 0.80 for OGUS (sens 87%, spec 93%). These diagnostic cut-off points differed in sensitivity and specificity within different age- and sex-defined groups with the highest sensitivity and lowest specificity observed among old ( $>81$ yr) male patients. Alternatively, age- and sex-stratified cut-off points could be established for the US scores (Table 1).

**Conclusion:** All previously reported US scores show excellent diagnostic accuracy for GCA, but are prone to over-diagnose GCA in older males. Our data highlight the need for age- and sex-specific diagnostic cut-off points. The excellent comparative performance of the simple HCount in GCA diagnosis suggests an exciting role in future clinical practice.

#### REFERENCES:

- [1] van der Geest KSM, et al. Annals of the Rheumatic Diseases 2020 Mar;79(3):393-399.
- [2] Dejaco C, et al. Annals of the Rheumatic Diseases 2022 doi: 10.1136/ard-2022-223367
- [3] Schafer VS, et al. Rheumatology 2017 Sep 1;56(9):1632.

**Table 1. Ultrasound scores in different age and sex groups. Age cut-offs were based on 25th percentile and 75th percentile of age in female and male patients with GCA.**

Category	Score	AUC in ROC analysis	Optimal cut-off point	Sensitivity (%)	Specificity (%)
Women	Halo count	0.897	2	75	98
Age <67	Halo Score	0.883	12	83	90
	OGUS	0.925	0.66	83	85
	Women	Halo count	0.927	1	88
age 67-79	Halo Score	0.873	16	72	93
	OGUS	0.933	0.80	80	98
	Women	Halo count	0.938	2	92
Age >79	Halo Score	0.889	16	92	88
	OGUS	0.928	0.83	92	94
	Men	Halo count	0.919	1	100
Age <74	Halo Score	0.877	15	88	74
	OGUS	0.911	0.87	75	97
	Men	Halo count	0.972	3	94
Age 74-81	Halo Score	0.837	20	72	100
	OGUS	0.972	0.76	100	88
	Men	Halo count	0.958	4	100
Age >81	Halo Score	0.885	19	88	83
	OGUS	0.896	0.90	100	83

**Acknowledgements:** K. van der Geest and A. Sebastian share first authorship.  
**Disclosure of Interests:** Kornelis van der Geest Speakers bureau: Roche, Grant/research support from: AbbVie, Alwin Sebastian: None declared, Edoardo Conticini: None declared, Sue Inness: None declared, Jo Jackson: None declared, Abdul Kayani: None declared, Muhammad Khurshid: None declared, Giulia Klinowski: None declared, Pierluigi Macchioni: None declared, Diana Prieto-Peña: None declared, Carlo Salvarani: None declared, Mohammad Tariq: None declared, Alessandro Tomelleri: None declared, Bhaskar Dasgupta Consultant of: Roche, Chugai, Sanofi, Grant/research support from: Roche, Sanofi, AbbVie, and GlaxoSmithKline.  
**DOI:** 10.1136/annrheumdis-2023-eular.5139

**POS1603 TREAT-TO-TARGET STRATEGIES IN GIANT CELL ARTERITIS AND POLYMYALGIA RHEUMATICA: A SYSTEMATIC LITERATURE REVIEW INFORMING AN INTERNATIONAL TASKFORCE**

**Keywords:** Remission, Vasculitis, Treat to target

E. Hysa<sup>1</sup>, M. Bond<sup>2</sup>, L. Ehlers<sup>3</sup>, D. Camellino<sup>4</sup>, L. Falzon<sup>5</sup>, C. Dejaco<sup>2,6</sup>, F. Buttgerit<sup>3</sup>, D. Aletaha<sup>7</sup>, A. Kerschbaumer<sup>7</sup>. <sup>1</sup>San Martino Polyclinic, University of Genoa, Italy, Laboratory of Experimental Rheumatology and Academic Division of Clinical Rheumatology - Department of Internal Medicine, Genoa, Italy; <sup>2</sup>Hospital of Bruneck (ASAA-SABES), Department of Rheumatology, Bruneck, Italy; <sup>3</sup>Charité - Universitätsmedizin Berlin, Corporate Member of Freie, Universität Berlin and Humboldt-Universität zu Berlin, Department of Rheumatology and Clinical Immunology, Berlin, Germany; <sup>4</sup>Local Health Trust 3, Division of Rheumatology, Genoa, Italy; <sup>5</sup>University of Sheffield, University of Sheffield, South Yorkshire, United Kingdom; <sup>6</sup>Medical University Graz, Department of Rheumatology, Graz, Austria; <sup>7</sup>Medical University of Vienna, Division of Rheumatology, Department of Medicine III, Vienna, Austria

**Background:** Polymyalgia rheumatica (PMR) and Giant cell arteritis (GCA) are chronic inflammatory diseases. Despite the progress made in the management of these conditions, new unmet needs have emerged particularly in terms of prevention of disease- and treatment-related complications. A treat-to-target (T2T) strategy, which has been well established in other rheumatic diseases, has not yet been developed for PMR and GCA.

**Objectives:** To retrieve current evidence on T2T strategies in PMR and GCA to inform an international task force (TF) developing T2T recommendations.

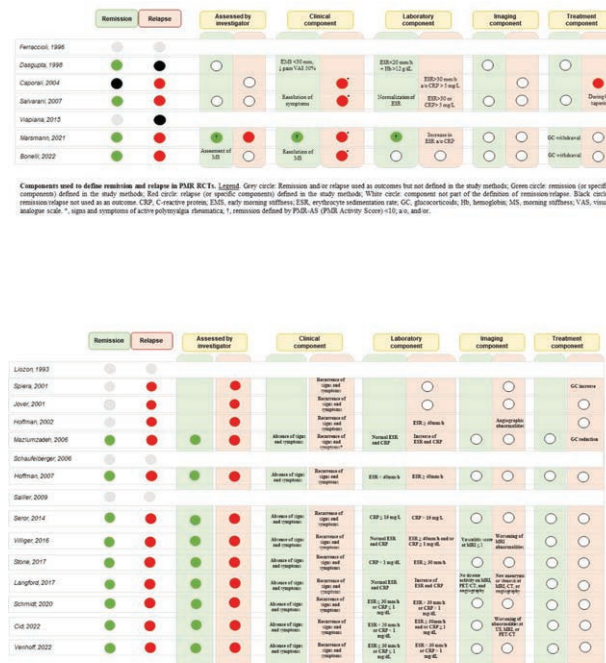
**Methods:** A systematic literature review (SLR) was conducted. Medline, EMBASE, Cochrane Library and clinicaltrials.gov (from inception until May 2022), as well as EULAR/ACR abstract databases were searched (2019-2021). Randomized controlled trials (RCTs) and non-randomized interventional studies published in English and answering at least one of the eleven PICO questions on treatment targets and outcomes, were identified (Table 1). The study selection process, data extraction, data synthesis and risk of bias assessment were conducted independently by two investigators.

**Results:** Of 7809 screened abstracts, 397 were selected for detailed assessment and 76 papers were finally included (31 RCTs, 8 subgroup/exploratory analyses of RCTs and 37 non-randomized interventional studies). No study comparing a T2T strategy against standard of care was identified. In PMR RCTs, treatment-related outcomes were most commonly used (90.9% of RCTs), specifically in terms of the glucocorticoids (GC) cumulative dose and GC tapering, followed by clinical, laboratory and safety targets (63.3% each). Conversely, the most frequently reported outcomes in RCTs in GCA were prevention of relapses (72.2%), remission, treatment, and safety (66.7 % each). Remission and relapses were variably defined in PMR and GCA RCTs but, in most cases, they comprised a combination of clinical and laboratory parameters (Figure 1). The following predictors of poor treatment response were identified: for GCA, data from RCTs yielded female sex, initial prednisone dose <30mg/day, bad baseline patient-reported outcomes, increased inflammatory markers after the achievement of clinical remission and absence of PMR symptoms at baseline as risk factors for treatment failure and an increased relapse rate. In PMR, no high-quality data predicting clinical outcomes were identified. Finally, in RCTs comparing the outcomes of GCA patients with new onset versus established disease, no differences were found, given that treatment was equal in both groups.

**Conclusion:** This SLR informed an international TF developing T2T recommendations in PMR and GCA. It provides up-to-date evidence while simultaneously highlighting the gaps in current knowledge about T2T strategies in these diseases.

**Table 1. Clinical key questions**

1. What are the treatment targets and outcomes in GCA/PMR, and how can they be measured (imaging, lab parameters, clinical, PRO)?
2. Is coming-off GC a treatment target in GCA/PMR, and how quickly should it be achieved?
3. What should be the frequency of monitoring disease state/ adapting therapy? How fast and to what extent should disease activity change before requiring treatment modification?
4. How do comorbidities influence T2T outcomes in GCA/PMR?
5. What are comorbidities related to uncontrolled disease activity?
6. Do targets need to be adapted based on the presence of comorbidities?
7. Is residual disease activity acceptable, and to what extent?
8. How can reaching disease targets, reducing/ preventing treatment side effects, and long-term consequences of disease be balanced in GCA/PMR? What is more important: control of disease activity or prevention of treatment related adverse effects?
9. Can treatment success be predicted?
10. What are the predictors of successful treatment reduction (e.g., duration on target)?
11. Do treatment targets differ over time (early vs. established disease)?



**Figure 1.** Components used to define remission and relapse in PMR and GCA RCTs

**Acknowledgements:** 1. Elvis Hysa and Milena Bond contributed equally to this work. 2. Grant support from Abbvie.

**Disclosure of Interests:** Elvis Hysa: None declared, Milena Bond: None declared, Lisa Ehlers: None declared, Dario Camellino Speakers bureau: speaker fees from Abiogen, BMS and GSK, Louise Falzon: None declared, Christian Dejaco Speakers bureau: consulting/speaker's fees from Abbvie, Eli Lilly, Janssen, Novartis, Pfizer, Roche, Galapagos, Sparrow and Sanofi, Consultant of: consulting/speaker's fees from Abbvie, Eli Lilly, Janssen, Novartis, Pfizer, Roche, Galapagos, Sparrow and Sanofi, Grant/research support from: grant support from Abbvie, Frank Buttgerit Speakers bureau: consultancy fees, honoraria and travel expenses from Abbvie, Novartis, Pfizer, Roche, and Sanofi, Consultant of: consultancy fees, honoraria and travel expenses from Abbvie, Novartis, Pfizer, Roche, and Sanofi, Daniel Aletaha Speakers bureau: grants, speaker fees, and/or consultancy fees from Abbvie, Amgen, Galapagos, Lilly, Janssen, Merck, Novartis, Pfizer, Sandoz, and Sanofi, Consultant of: grants, speaker fees, and/or consultancy fees from Abbvie, Amgen, Galapagos, Lilly, Janssen, Merck, Novartis, Pfizer, Sandoz, and Sanofi, Grant/research support from: grants, speaker fees, and/or consultancy fees from Abbvie, Amgen, Galapagos, Lilly, Janssen, Merck, Novartis, Pfizer, Sandoz, and Sanofi, Andreas Kerschbaumer Speakers bureau: consultancy fees, honoraria and travel expenses from AbbVie, Amgen, Bristol-Myers Squibb, Eli Lilly, Gilead, Janssen, Merck Sharp and Dohme, Novartis, UCB and Pfizer, Consultant of: consultancy fees, honoraria and travel expenses from AbbVie, Amgen, Bristol-Myers Squibb, Eli Lilly, Gilead, Janssen, Merck Sharp and Dohme, Novartis, UCB and Pfizer.

**DOI:** 10.1136/annrheumdis-2023-eular.3193