Objectives: To analyze sVCAM-1 in TA patients treated with TCZ in a prospective clinical trial and compare these results to age-matched healthy controls (HC) as well as DMARDs. In addition, MRI analyses of aortic wall thickening and enhancement might serve as a morphologic correlate of serologic disease activity.

Methods: 29 TA patients were prospectively followed between 2016 and 2019 (27 females, mean $\pm$ SD: 39.2 $\pm$ 13.9 years) at the Department of Rheumatology of the University Hospital of Bern, Switzerland. At baseline, patients were without treatment (n=8) or treated with TCZ (n=13), GC (n=4), GC + methotrexate (MTX) (n=1), infliximab (IFX) + MTX (n=1), GC + IFX (n=1), MTX (n=1). Three follow-ups were performed after an average of 12, 24, 34 $\pm$ 3 months. sVCAM-1 was measured in serum using a commercially available ELISA kit (R&D Systems, Germany). Results were compared to 29 sera of matched HC (27 females, 40.8 $\pm$ 15.1 years). MRI of aortic changes were scored on a scale of 0 (no thickening and vessel wall enhancement) to 3 (maximal thickening and vessel wall enhancement). Cumulated sVCAM-1 concentrations from each MRI scoring group (0-3) were compared to HC in order to determine if sVCAM-1 from high aortic MRI inflammation was significantly elevated.

Results: At baseline, significantly increased serum concentrations of sVCAM-1 (ng/ml) were observed in TA patients without treatment (n=8, 537.3 $\pm$ 130.1, $p=0.002$) vs. HC (336.0 $\pm$ 76.1). A smaller difference was found between patients under treatment and HC (n=21, 468.2 $\pm$ 105.3 vs. 405.1 $\pm$ 82.5, $p=0.04$). Follow-ups in the TCZ group showed 518.9 $\pm$ 138.9 vs. 417.5 $\pm$ 46, $p=0.12$ (n=6) after 12 months, 436.6 $\pm$ 77.3 vs. 399.6 $\pm$ 113.1, $p=0.46$ (n=8) after 24 months and 407.1 $\pm$ 27.1 vs. 381.8 $\pm$ 60.4, $p=0.47$ (n=4) after 36 months. In contrast, patients under DMARD therapy showed values of 505.8 $\pm$ 126.4 vs. 395.6 $\pm$ 60.2 $p=0.04$ (n=8) after 12 months, 437.8 $\pm$ 76.2 vs. 396.4 $\pm$ 91.9, $p=0.24$ (n=12) after 24 months and 440 $\pm$ 43.3 vs. 323 $\pm$ 50 $p=0.03$ (n=8) after 36 months. MRI analyses showed that group 0 (no inflammation, n=7) had significant increased values compared to HC (474.7 $\pm$ 106.8 vs. 356.9 $\pm$ 48.8, $p=0.02$), and group 3 (maximal inflammation, n=11) was also elevated (461.8 $\pm$ 98.4 vs. 379.3 $\pm$ 88.9, $p=0.05$).

Conclusion: The results suggest that sVCAM-1 is a biomarker of disease activity in patients with TA. The results at follow-up show that sVCAM-1 decreased more rapidly under treatment with TCZ compared to treatment with DMARDs. Remarkably, sVCAM-1 concentrations did not correlate with disease activity as assessed with MRI.

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POS0342 DISCRIMINANT VALUE OF ULTRASOUND OF THE WALL OF LARGE VESSELS IN THE DIAGNOSIS OF GIANT CELL ARTERITIS

I. González-Mazón¹, E. Fernández-Fernández², I. Monjo³, E. De Miguel³.

¹Marqués de Valdecilla University Hospital, Rheumatology, Santander, Spain;

²La Paz University Hospital, Rheumatology, Madrid, Spain; ³La Paz University Hospital, Rheumatology, Madrid, Spain

Background: The EULAR recommendations for the use of imaging in large vessel vasculitis in clinical practice suggest the performance of diagnostic imaging tests in all patients with suspected GCA and ultrasound is the recommended first-line test in patients with suspected cranial GCA (c-GCA)¹. However, a priority imaging modality has not been established in cases of suspected GCA involving large vessels (LV-GCA). Since patients with LV-GCA often undergo extensive screening programs for suspected infection or malignancy before the diagnosis of GCA is established, our knowledge in this field needs to be improved.

Objectives: The aim of this study is to investigate the wall affection of vasculitis of the proximal arm and neck arteries in patients with suspected GCA, polymyalgia rheumatica (PMR), atherosclerosis and non-GCA patients.

Methods: Retrospective available ultrasound examination from all the patients referred to our GCA fast-track clinic in the last three years were collected. All patients had ultrasound of cranial and large vessel examination (axillary, subclavian and carotid arteries). GCA diagnosis was confirmed by the doctor after a follow-up between six months to three years. Every patient had videos and pictures of every referred vessel. Examination was performed by two rheumatologists with long experience in GCA ultrasound from the same Rheumatology

Department with a Esaote Mylab Twice with probe of 13 MHz (2017-2019) and a Esaote Mylab X8plus with a probe of 15 MHz (2019-2020). IMT and hypoechoic halo measures were taken.

Results: We analyzed 300 examinations and selected 196 baseline cases of four different pathologies (29 PMR, 40 atherosclerosis, 71 GCA of which 14 LV-GCA without cranial involvement and 56 non-GCA non-PMR controls). Intima-media thickness (IMT) with the statistical significance between different diseases and controls are showed in the Table 1.

Table 1. Ultrasound IMT and halo measures in large vessels of different diseases and controls

	Non-GCA n=56	GCA n=71	Atherosclerosis n=40	PMR n=29	LV-GCA n=14
Axillary right # *	0.67 $\pm$ 0.19	0.95 $\pm$ 0.30	0.70 $\pm$ 0.18	0.65 $\pm$ 0.13	1.03 $\pm$ 0.33
Axillary left # **	0.61 $\pm$ 0.12	0.92 $\pm$ 0.29	0.72 $\pm$ 0.19	0.66 $\pm$ 0.13	0.99 $\pm$ 0.25
Subclavian right # *	0.70 $\pm$ 0.15	1.00 $\pm$ 0.31	0.86 $\pm$ 0.29	0.70 $\pm$ 0.20	1.09 $\pm$ 0.36
Subclavian left # *	0.62 $\pm$ 0.14	0.95 $\pm$ 0.27	0.70 $\pm$ 0.14	0.64 $\pm$ 0.18	1.05 $\pm$ 0.25
CCD right #	0.79 $\pm$ 0.22	0.97 $\pm$ 0.26	1.01 $\pm$ 0.30	0.81 $\pm$ 0.10	0.91 $\pm$ 0.29
CCD left #	0.81 $\pm$ 0.16	0.99 $\pm$ 0.22	0.98 $\pm$ 0.32	0.82 $\pm$ 0.20	0.97 $\pm$ 0.17

GCA= Giant cell arteritis (cranial, large vessel and mixed forms); Non-GCA = Patients suspected but not confirmed as GCA; Atherosclerosis = patients with high arteriosclerosis risk without suspected GCA; PMR = Patients with polymyalgia rheumatica without GCA; LV-GCA= Large vessel vasculitis GCA without cranial involvement. # $p<0.01$ GCA vs non-GCA and GCA vs PMR* $p<0.01$ GCA vs Atherosclerosis; ** $p<0.05$ GCA vs Atherosclerosis

Conclusion: GCA ultrasound of large vessels shows significant differences with other diseases. The differences seem to support the use of ultrasound in the fast-track clinic of GCA not only in c-GCA but also in LV-GCA.

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POS0343 RITUXIMAB IS SUPERIOR TO PLACEBO IN POLYMYALGIA RHEUMATICA

D. Marsman¹, N. Den Broeder², F. Van den Hoogen³, A. Den Broeder³, A. Van der Maas³. ¹Sint Maartenskliniek, Rheumatology, Nijmegen, Netherlands; ²Sint Maartenskliniek, Rheumatology, Ubbergen, Netherlands; ³Sint Maartenskliniek, Rheumatology, Nijmegen, Netherlands

Background: Corticosteroids remain the cornerstone of polymyalgia rheumatica treatment, but their use has several disadvantages such as long treatment duration and glucocorticoid-related adverse events.^{1,2} Data on evidence based effective glucocorticoid-sparing agents are negative or absent.²

Objectives: Because B-cells may be involved in the pathogenesis of polymyalgia rheumatica, we evaluated the efficacy of rituximab in polymyalgia rheumatica.

Methods: In a 21-week double-blind placebo controlled exploratory study, 47 polymyalgia rheumatica patients (recently diagnosed n=38 / relapsing on prednisolone $\geq$ 7.5mg/day n=9) fulfilling the 2012 EULAR/ACR criteria, were randomized 1:1 to intravenous rituximab 1 x 1000mg (n=23) or placebo (n=24), with a 17-week long glucocorticoid co-treatment. Primary outcome was glucocorticoid-free remission at week 21. Secondary outcomes were glucocorticoid $\leq$ 5mg/day and adverse events. Several post-hoc analyses were done for robustness of results.

Results: Glucocorticoid-free remission was achieved in 48% (rituximab) versus 21% (placebo), one-sided 95%-CI 4% to 100%; $p=0.049$, and glucocorticoid $\leq$ 5mg/day in 100% versus 54% (one sided 95%-CI 20% to 100%; $p=0.005$). Post-hoc analysis showed efficacy mainly in recently diagnosed patients: glucocorticoid-free remission in 58% versus 21% (one-sided 95%-CI 10% to 100%; $p=0.02$); glucocorticoid $\leq$ 5mg/day in 100% versus 47% (one-sided 95%-CI 29 to 100%; $p<0.001$). No significant differences were observed regarding other outcomes (Table 1), except for less morning stiffness after rituximab.