clinical features including histology, block 2: laboratory and treatment factors, block 3: demographic and traditional risk factors). Variables included in the final multivariate model were selected on the following basis: P-value <0.1 in univariate or block regression or otherwise deemed clinically significant. 

Results: 881 patients were included of which 626 (71.1%) were females. Mean age was 73 years (SD 9), 490 patients (55.6%) died during the study period (1 January 1972 – 31 December 2012). Characteristics and mortality for the GCA-cohort compared to matched controls have been published previously [3]. Within the GCA-cohort we found that presenting with visual disturbance (any) or scalp necrosis was associated with increased risk of death in univariate analysis (Figure 1). However, in multivariate analysis the traditional risk factors (age, smoking, hypertension and previous cardiovascular disease) were more strongly associated with risk of death. Among laboratory parameters only Hemoglobin (Hb) levels were significantly associated with risk of death with increasing Hb-levels indicating decreased risk. Neither temporal artery biopsy result nor initial or maximal (before first tapering) Prednisolone dose were found to be associated with risk of death. Results from univariate and the final multivariate models are presented in Figure 1.

Conclusion: In our large cohort of GCA-patients the risk of death was found to be predominantly predicted by age (HR 2.81) and traditional risk factors (smoking (HR 1.61), hypertension (HR 1.48) and previous cardiovascular disease (HR 1.26)). Visual disturbance (HR 1.40), visual loss in particular (HR 2.37), and scalp necrosis (HR 3.42) were found to be the clinical features most associated with risk of death. However, we note that our material lacked information about extra-cranial (large vessel) vasculitis, which may also carry increased risk of death.

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POS0340

VASCULAR IMAGING IN PATIENTS WITH REFRACTORY TAKAYASU ARTERITIS TREATED WITH TOCILIZUMAB: ANALYSIS FROM A RANDOMIZED CONTROLLED TRIAL

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Background: In the TAKT study, a randomized controlled trial of tocilizumab (TCZ) in patients with refractory Takayasu arteritis (TAK) in Japan, the primary end point of time to relapse after induction of remission with glucocorticoid (GC) treatment showed a trend favoring TCZ over placebo (hazard ratio 0.41 [95.41% confidence interval, 0.15-1.10; p=0.0569]) but the double-blind period was too short for imaging evaluation.

Objectives: To independently evaluate vascular imaging in a post hoc analysis of radiographs from the TAKT study.

Methods: Computed tomography images from patients in the TAKT study were evaluated by three independent radiologists who were not involved in the original trial. Patients who received TCZ and had computed tomography images available (n=28) were included. Assessments were made in 22 arteries for the change from baseline in wall thickness (primary end point), dilatation/aneurysm, stenosis/occlusion, or wall enhancement for at least 96 weeks after the start of tocilizumab treatment. Patient-level assessments were also conducted.

Results: Among 28 patients who received at least one dose of TCZ and for whom images were available, 86.7% of 22 arteries had improved/stable (no progression) wall thickness at week 96. The proportions of patients with no progression, partially progressed, or newly progressed lesions were 57.1%, 10.7%, and 28.6% for wall thickness, and the proportions without progressed lesions were 92.9% for dilatation/aneurysm and 85.7% for stenosis/occlusion (Figure 1). Patients with newly progressed lesions, reflecting more refractory disease, were receiving glucocorticoid doses that could not be reduced below 0.1 mg/kg/day at week 96.

Conclusion: Approximately 60% of patients with TAK treated with tocilizumab did not experience progression in wall thickness. Few patients experienced progressive dilatation/aneurysm or stenosis/occlusion. Wall thickness progression likely resulted from refractory TAK. Patients who experience this should be monitored regularly by imaging, and additional glucocorticoid or immunosuppressive treatment should be considered to avoid vascular progression.

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Figure. Evaluation of arteries per patient (n=28)

Figure 1.
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 ± SD: 39.2±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 ± 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±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, 533±313.1, p=0.002) vs. HC (396±307.6). A smaller difference was found between patients under treatment and HC (n=21, 468±210.3 vs. 405±182.5, p=0.04). Follow-ups in the TCZ group showed 519±138.9 vs. 417±54.6, p=0.02 (n=12) after 12 months, 436.6±72.7 vs. 399.6±113.1, p=0.06 (n=24) after 24 months and 407±1271 vs. 381±86.0, p=0.04 (n=26) after 36 months. In contrast, patients under DMARD therapy showed values of 505.8±126.4 vs. 395±60.2, p=0.004 (n=8) after 12 months, 437±66.2 vs. 396±19.1, p=0.24 (n=12) after 24 months and 440±73.4 vs. 323±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±106.8 vs. 356.9±48.8, p=0.02), and group 3 (maximal inflammation, n=11) was also elevated (461±98.8 vs. 379.3±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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