All patients underwent baseline magnetic resonance angiography (MRA) and a follow-up MRA at least one year after baseline per a standardized imaging protocol. The presence of angiographic lesions, defined as stenosis, occlusion, or aneurysm, was evaluated by visual inspection by a single reader who was blinded to clinical status. Angiographic lesions were evaluated in 4 segments of the aorta and in 13 branch arteries. On follow up angiography, the development of new lesions was recorded, and existing lesions were characterized as improved, worsened, or unchanged.

Results: 782 arterial territories were evaluated from 46 patients with LVV (TAK=28; GCA=18). Baseline characteristics were as follows: Age [TAK=24.8 years (18.6-34.9), GCA=64.8 years (57.8-73.9)], Female gender [TAK=21 patients (78%), GCA=16 patients (84%)], Disease duration [TAK=2.3 years (0.6-4.9), GCA 1.2 years (0.4-2.9)], Active clinical disease [TAK=12 patients (44%), GCA 12 patients (63%)]. The median time from initial MRA to follow up MRA was 2.4 years (1.5-3.1) for GCA and 1.6 years (1.3-3.3) for TAK.

There were 159 territories affected at the baseline visit in 41 patients [TAK: 108 territories in 26 patients, GCA: 51 territories in 15 patients]. The development of new territory involvement was infrequent and only occurred in patients with TAK (8 new lesions out of 352 baseline unaffected territories (2.3%) in 5 patients).

At follow up, existing arterial lesions improved in 25 (15.7%) territories, worsened in 6 (3.8%) territories, and stayed the same in 128 (80.5%) territories. There were no significant differences in angiographic progression of disease between the two diseases: improved - TAK 19 (17.6%), GCA 6 (11.8%); worsened - TAK 5 (4.6%), GCA 1 (1.9%); unchanged - TAK 84 (77.8%), GCA 44 (86.3%). Change in the branch arteries was more dynamic than change in the aorta (**Figure**).

Improvement in angiographic disease was observed in 8 (17%) patients (TAK=6, GCA=2). Worsening of disease was seen in 3 (7%) patients (TAK=2, GCA=1). In 5 (11%) patients (TAK=5, GCA=0), there were areas of improvement and other areas of worsening disease within the same patient.

Conclusion: Dynamic change in arterial lesions is observed in patients with TAK and GCA. Improvement and worsening of arterial lesions can be observed over time, even within the same patient. This observation suggests that both local factors at the level of the artery and systemic factors (e.g. treatment response) are likely associated with angiographic progression. The development of new angiographic lesions was infrequent, and only occurred in patients with TAK. These data may inform future guideline recommendations for angiographic monitoring in LVV.

References: N/A

Figure.

Disclosure of Interests: None declared

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SAT0252 INCREASED MORTALITY FOR INDIVIDUALS WITH GIANT CELL ARTERITIS: A POPULATION-BASED STUDY

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Background: Individuals with giant cell arteritis (GCA) are at increased risk of serious morbidity including cardiovascular disease and stroke. Yet the risk of mortality among individuals with GCA have produced conflicting reports¹.

Objectives: Our aim was to evaluate excess all-cause mortality among individuals with GCA relative to the general population over time.

Methods: We performed a population-based study in Ontario, Canada, using health administrative data among all individuals 50 years and older. Individuals with GCA were identified using a validated case definition (81% PPV, 100% specificity). All Ontario residents aged 50 and above who do not have GCA served as the General Population comparators. Deaths occurring in each cohort each year

were ascertained from vital statistics. Annual crude and age/sex standardized all-cause mortality rates were determined for individuals with and without GCA between 2000 and 2018. Standardized mortality ratios (SMRs) were calculated to measure relative excess mortality over time. Differences in mortality between sexes and ages were also evaluated.

Results: Population denominators among individuals 50 years and older with GCA and the General Population increased over time with 12,792 GCA patients and 5,456,966 comparators by 2018. Annual standardized mortality rates among the comparators steadily declined over time and were significantly lower than GCA mortality rates (Figure). Annual GCA mortality rates fluctuated between 42-61 deaths per 1000 population (with overlapping confidence intervals) during the same time period. SMRs for GCA ranged from 1.28 (95% CI 1.08,1.47) at the lowest in 2002 to 1.96 (95% CI 1.84, 2.07) at the highest in 2018. GCA mortality rates and SMRs were highest among males and younger age groups.

Conclusion: Over a 19-year period, mortality has remained increased among GCA patients relative to the general population. GCA mortality rates were higher among males and more premature deaths were occurring at younger age groups. In our study, improvements to the relative excess mortality for GCA patients over time (mortality gap) did not occur. Understanding cause-specific mortality and other factors are necessary to inform contributors to premature mortality among GCA patients.

References:

[1] Hill CL, et al. Risk of mortality in patients with giant cell arteritis: a systematic review and meta-analysis. *Semin Arthritis Rheum.* 2017;46(4):513-9.

Figure.

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SAT0253 ANTI-NEUTROPHIL CYTOPLASMATIC ANTIBODIES PREDATE SYMPTOM ONSET OF ANCA-ASSOCIATED VASCULITIS. A CASE-CONTROL STUDY

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Background: Presence of anti-neutrophil cytoplasmic autoantibodies (ANCA) is important for the diagnosis of ANCA-associated vasculitis (AAV) and reflects on-going immune processes. The timing of the antibody development and its contribution to disease is not well established.

Objectives: To investigate the presence of proteinase 3 (PR3)- and myeloperoxidase (MPO)-ANCA in blood samples collected from healthy individuals who subsequently developed AAV.

Methods: The Swedish National Patient Register of inpatient care and the Swedish Cause of Death Register were used to identify individuals assigned ICD codes for AAV (1) in the discharge summary or cause of death, respectively. The resulted cohort was then linked to the registers of 4 different biobanks to identify those with available predating blood samples. Diagnoses of AAV were confirmed and time point for onset of symptoms was identified by reviewing all available case records (1); 68 were classified as granulomatosis with polyangiitis (GPA), 14 as microscopic polyangiitis (MPA), and 4 as eosinophilic GPA (EGPA). The 86 cases (36 males, 50 females) had a mean (SD) age of 51.9 (16.9) years at sampling, with ≥1 sample (26% plasma, 74% serum samples). The sampling time point before onset of symptoms was mean (SD); 4.4 (3.1) years. Serum and plasma control samples (n=198; 82 males, 116 females; mean age (SD); 52.0 (16.5) years) were identified and matched for sex, age and date of sampling. The samples were first screened for ANCA using high sensitive ELISA (ORGANTEC diagnostika, Germany) and samples close to or above cut-off level were further analysed for capture PR3- and capture MPO-ANCA (ELISA; SVAR Life Science,