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=286; 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 16 years (1.3-3.3) for TAK.

There were 150 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 (176%), 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=2, GCA=3), 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 frequent, and only occurred in patients with TAK. These data may inform future guideline recommendations for angiographic monitoring in LVV.

References: N/A

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SAT0253

ANTI-NEUTROPHIL CYTOPLASMIC ANTIBODY PREDATE SYMPTOM ONSET OF ANCA-ASSOCIATED VASCULITIS. A CASE-CONTROL STUDY


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). ANCA were classified as perinuclear (Pteinu), perinuclear (PANCA), and cytoplasmic (PANCA) (1). 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): 4.3 (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 (ORGANTECagnostika, Germany) and samples close to or above cut-off level were further analysed for capture PR3- and capture MPO-ANCA (ELISA; SVAR Life Science, Sweden).