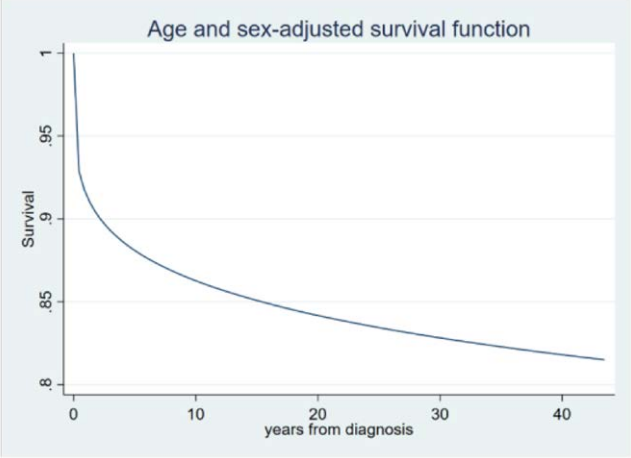

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Vasculitis – large vessel vasculitis

AB0352 ANTICARDIOLIPIN ANTIBODIES AND ACTIVITY OF GIANT CELL ARTERITIS

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Background: Anticardiolipin antibodies (aCL) can be detected in newly diagnosed GCA as reactive antibodies to endothelial lesions. Their prognostic role, as a marker of disease activity, has not been extensively studied in GCA.
Objectives: Our aim was to determine whether aCL IgG might represent laboratory marker of active GCA.
Methods: We included patients with new clinical diagnosis of GCA supported by histology or imaging between September 2011 and July 2019, who completed at least a 48-week follow-up at our secondary/tertiary rheumatology center. Follow up visits with predetermined clinical and laboratory tests, including aCL IgG, were performed 12, 24, 48, and 96 weeks after diagnosis. GCA relapse was defined as worsening or new disease activity after initial remission. Other reasons for the disease-related symptoms, elevated inflammatory markers (C reactive protein or erythrocyte sedimentation rate) had to be excluded. aCL IgG were determined in the patients' sera samples at baseline and at follow up visits, using an in-house solid phase enzyme-linked immunosorbent assay[1]. A value above the 99th percentile of the healthy control population was taken as significant.
Results: During the observation period we identified 288 newly diagnosed GCA patients. Two hundred and twelve GCA patients (66.5% females, median (IQR) age 73.9 (67.0–78.7) years) fulfilled the study inclusion criterion, among them 145 patients completed the 96-week follow up visit. At baseline, 129/212 (60.8%) GCA patients had positive aCL IgG. During in total 781 follow up visits, we recorded 77 (9.9%) episodes of active/relapsing GCA (clinical, laboratory or combined in 4 (5.2%), 48 (62.3%), 25 (32.5%) episodes, respectively). aCL IgG were present at 155/781 (19.8%) measurements (at 24/77 episodes of relapsing/active and 131/573 episodes of quiescent GCA). The correlation between active/relapsing GCA and aCL IgG positivity was only weekly positive (r coefficient=0.094; p= 0.015).
Conclusion: The role of aCL IgG as a biomarker for GCA activity seems to be rather limited.
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AB0353 ADRENAL INSUFFICIENCY AFTER GLUCOCORTICOID TREATMENT OF GIANT CELL ARTERITIS

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Background: Adrenal insufficiency is frequently neglected and underappreciated, potentially severe complication of systemic glucocorticoid therapy.
Objectives: We aimed to evaluate the prevalence of glucocorticoid induced adrenal insufficiency in giant cell arteritis (GCA).
Methods: We analysed adrenal function data in a cohort of GCA patients diagnosed between July 2014 and July 2019, in whom discontinuation of methylprednisolone therapy was planned. Adrenal function was tested by Corticotropin stimulation test (CST). To perform the CST, methylprednisolone was substituted with hydrocortisone (20mg qd in three divided doses) for one to four weeks before the test. Adrenal insufficiency was defined as cortisol level <450 nmol/l measured 30 minutes after the corticotropin injection; additionally, the result of the CST was defined as borderline when the cortisol level 30 minutes after corticotropin injection was between 450 nmol/l and 500 nmol/l.
Results: Adrenal function was tested in 74/215 GCA patients before definite methylprednisolone withdrawal (after a median 13.5 (12.9 – 22.4) months of glucocorticoid therapy). The mean (SD) methylprednisolone dose, prior to substitution with hydrocortisone and subsequent CST, was 3.1 (1.6) mg. Adrenal insufficiency was detected in 36/74 patients (48.6%); additionally, 10/74 patients (13.5%) had a borderline CST result. Seventeen patients with either adrenal insufficiency or borderline CST result, had a repeated CST after median (IQR) 11.6 (8.9; 12.6) months. Adrenal insufficiency persisted in 11/17 (64.7%) patients, and 1/17 patients had a borderline CST. A third CST was performed in 4/12 patients with abnormal second CST after median (IQR) 8.3 (6.9; 10.6) months. Adrenal function recovered in one patient, while the adrenal insufficiency persisted in the remaining 3 patients.
Conclusion: Adrenal insufficiency is a common and potentially long-lasting glucocorticoid induced adverse event in GCA patients.
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AB0354 FDG-PET-DETECTED LARGE VESSEL VASCULITIS DOES NOT PREDICT DISEASE OUTCOME IN PATIENTS WITH GIANT CELL ARTERITIS AND POLYMYALGIA RHEUMATICA

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Background: Giant cell arteritis (GCA) and polymyalgia rheumatica (PMR) are tightly associated inflammatory conditions of the elderly [1]. Both disorders can exhibit an increased articular and vascular uptake of 18-fluorodeoxyglucose (18-FDG) at positron emission tomography (PET)/computed tomography (CT) scan [2].
Objectives: This study evaluated if large-vessel vasculitis (LVV) detected by PET/CT in patients with PMR and/or cranial GCA had a negative prognostic value.
Methods: 108 patients (35 men and 73 women) with a median age of 74 years (range 50-92 years) were prospectively enrolled in our centre over 4 years. PMR was diagnosed by Bird et al. criteria and GCA by the ACR criteria. Six patients died shortly after the first visit (V_0) and six were lost at follow-up. Of the remaining 96 patients, 77 were classified as PMR, 6 as GCA and 13 were affected by both diseases.
At V_0, patients underwent a clinical, laboratory and PET/CT evaluation, and were stratified according to the presence or not of LVV. Follow-up visits

were performed every 6 months for a median of 40 months. Disease outcomes were: prednisone (PDN) use and its cumulative dosage, need of methotrexate (MTX), number of relapses, patients' death, and PMR disease activity score (PMR-DAS). The independent variables were age, sex, disease duration, fever, C-reactive protein (CRP) concentration, platelet count (PLT), presence of cranial GCA, degree of joint and vascular uptake of FDG, and presence of LVV. The predictive role of LVV was tested by multiple regression.

Results: LVV was seen in 47 patients (49 %), 31 with PMR, 6 with GCA and 10 with both diseases. Patients with or without LVV did not significantly differ in terms of demographic and laboratory parameters except for a non-significant higher number of PLT in patients with LVV. Clinical and laboratory parameters at V_0, stratified per disease and considered together, did not significantly change between PET+ and PET- patients (table 1). Lastly, none of the independent variables, including LVV, could predict disease outcomes.

Conclusion: The presence of a PET-detected LVV at diagnosis does not seem a negative prognostic factor in PMR and GCA. As a consequence, routine investigation by PET/CT of patients with PMR and GCA is not indicated to predict disease outcome.

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Table 1. Clinical, laboratory and imaging features between PET+ and PET- patients at V_0

Features at V_0	PET+ patients	PET- patients	p
Morning stiffness (min)	30 (0-480)	60 (0-360)	0.20
Haemoglobin (g/dL)	12.3±1.5	12.6±1.5	0.28
Platelets (x 10^3/mm^3)	349 (108-643)	297(159-571)	0.08
C-reactive protein (mg/dL)	35.5 (3.4-149)	36.2 (2-149)	0.54
Erythrocyte sedimentation rate (mm/h)	62.5 (10-120)	57.5 (10-120)	0.29
Total Vascular Score at PET	20 (4-41)	6 (0-12)	0
Total Joint Score at PET	18 (5-30)	18 (5-32)	0.77

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AB0355 THE VALUE OF PLATELET-TO-LYMPHOCYTE AND NEUTROPHIL-TO-LYMPHOCYTE RATIOS AS INFLAMMATORY MARKERS IN BIOPSY-PROVEN GIANT CELL ARTERITIS

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Background: Platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR) have been shown to correlate with disease activity in various rheumatic diseases. High PLR and elevated platelet count are potentially useful in diagnosing some systemic vasculitides, eg. giant cell arteritis (GCA)[1] and has been suggested predictive for diagnosing GCA, even in the absence of characteristic temporal artery involvement[2].

Objectives: To evaluate the role of PLR and NLR as inflammatory markers in the diagnosis of GCA in patients referred to temporal artery biopsy (TAB).

Methods: During 10 months, all patients referred to TAB at the Department of Ophthalmology (Rigshospitalet, Copenhagen, Denmark) in suspected GCA were included. Immediately prior to TAB, ultrasound of bilateral temporal arteries was performed at the Center for Rheumatology and Spine Disease (Rigshospitalet, Copenhagen, Denmark) by rheumatologists experienced in vascular ultrasound. Ultrasound signs of GCA were a positive Halo sign or compression sign. Patients had C-reactive protein (CRP), erythrocyte sedimentation ratio (ESR), platelets, white blood cells with differential counts, hemoglobin and platelets measured. Final clinical diagnosis, based on rheumatological expert opinion, and fulfilment of ACR1990 classification criteria for temporal arteritis at six months was noted, with clinical diagnosis being the reference standard.

Results: 106 patients were included and had a TAB evaluated. Forty-five (42%) had a clinical diagnosis of GCA at 6 months of which 28 (62%) also were TAB positive. US was performed in 74 (70%), of these 20 (67%) of the GCA patients had a positive ultrasound for GCA (termed US GCA patients). There was no significant difference in mean age or gender distribution between GCA and non-GCA patients. ESR, CRP and platelets were significantly higher in GCA than non-GCA patients (p<0.001). PLR was significantly higher in GCA than non-GCA patients (p<0.007), while NLR was not (p=0.076). For US GCA patients vs. US non-GCA patients, platelets were significantly higher in the US GCA group (p=0.025). Both PLR and NLR were significantly higher in the US GCA group compared to the US non-GCA group (p= 0.003 and p=0.007, respectively).

Conclusion: In this cohort of patients, suspected of GCA and referred to TAB, PLR but not NLR was significantly higher in GCA patients than in non-GCA patients. In patients with US findings of GCA, both PLR and NLR were significantly higher in GCA compared to non-GCA. An elevated PLR could be considered an additional feature in diagnosing GCA.

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	GCA (n=45)	Non-GCA (n=61)	*P*-value
Age (years)	73.6 (7.5)	72.6 (10.4)	0.575
Females, n (%)	34 (76)	38 (62)	0.148
Fulfilling ACR1990 criteria, n (%)	36 (80)	16 (26)	**<0.001**
TAB positive for GCA, n (%)	28 (62)	0 (0)	**<0.001**
Ultrasound positive for GCA*, n (%)	20 (67)	9 (20)	**<0.001**
Sedimentation rate (mm/hr)	58 (30)	33 (31)	**<0.001**
Haemoglobin (mmol/L)	7.68 (0.92)	7.96 (1.05)	0.154
CRP (mg/L), median (IQR)	38 (15-92)	8 (2-40)	**<0.001**
White blood cells (10^9/L)	9.54 (3.18)	8.99 (3.63)	0.407
Lymphocytes (10^9/L)	1.72 (0.78)	1.87 (0.93)	0.367
Monocytes (10^9/L)	0.71 (0.37)	0.77 (0.41)	0.441
Neutrophils (10^9/L)	6.85 (2.94)	6.01 (3.02)	0.157
Basophils (10^9/L)	0.05 (0.04)	0.05 (0.05)	0.464
Eosinophils (10^9/L)	0.15 (0.14)	0.18 (0.21)	0.410
Platelets (10^9/L)	400 (154)	289 (111)	**<0.001**
Neutrophil-to-lymphocyte ratio	4.91 (3.22)	3.87 (2.52)	0.076
Platelet-to-lymphocyte ratio	287 (189)	193 (147)	**0.007**

*Values are mean and standard deviation unless notedComparison between groups by independent samples t-test (continuous variables), χ^2-test (categorical variables) and Mann-Whitney for CRP (not normal distributed continuous variable)ACR=American College of Rheumatology; CRP=C-reactive protein; GCA=giant cell arteritis; IQR=interquartile range; n=number; SD=standard deviation; TAB=temporal artery biopsy*Ultrasound not done in 32 patients (15 with GCA and 17 without)*

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AB0356 VENOUS INVOLVEMENT IN BEHÇET'S DISEASE

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Background: Behçet's disease (BD) is a vasculitic multisystem inflammatory disorder. It may also involve the skin, mucosa, eyes, blood vessels, joints, gastrointestinal system, and central nervous system.

Objectives: In this study, we aimed to present venous involvement data in patients followed up with a diagnosis of BD.

Methods: The clinical, demographic, laboratory and medication data of 394 patients who were followed up with a diagnosis of BD in our