from life-threatening conditions to chronic relapsing long-term diseases as a result of significant advances in immunosuppressive therapy. Structured clinical assessment using Vasculitis Damage Index (DVI) should form the basis of a treatment plan and be used to document progress.

Objectives: To investigate the Vasculitis Damage Index and clinical manifestations in localized and systemic granulomatosis with poliangiitis in Mexican patients

Methods: We enrolled 61 patients with GPA according to The American College of Rheumatology (ACR) criteria at a referral hospital during the period from 2005 to 2015. Clinical and laboratory data, organ involvement and the Vasculitis Damage Index (VDI) were recorded at baseline. Patients were divide into systemic and localized form for their analysis.

Results: They were 61 GPA (34 men and 27 women) mean age 42 years old at diagnosis. Systemic form was observed in 53% and localized form 47%. Chronic sinusitis was the most frequent manifestation in 33% followed by otologic in 26%. Subglottic stenosis 4 patients, alveolar hemorrhage 1%. Of the patients with the systemic form 22 presented focal and segmental glomerulonephritis and 10 patients (32%) rapidly progressive glomerulonephritis. Distal-symmetric polyneuropathy and cranial neuropathy were present in 24%; scleritis 24.5% and proptosis in 18%, palpable purpura 26.2% and ulcers in 9 patients (14.8%). The VDI score in the systemic form was 3.8 and in the localized 2.6, p= NS. The disease related damage was pronounced in kidneys and upper airways. The majority of patients in the induction to remission phase received steroids plus cyclophosphamide, 7 patients also received plasmapheresis and in maintenance phase they were treated with methotrexate or azathioprine.

Conclusions: In this cohort of patients with GPA, a high chronic damage was found which was similar in both systemic and localized forms of this vasculitis. The VDI was more prominent in kidneys and upper airways in GPA patients

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AB0586 EVALUATION OF ASSSOCIATION BETWEEN ANTIPHOSPHOLIPID ANTIBODIES OR LUPUS ANTICOAGULANT POSITIVITY AND SEVERITY OF VASCULAR INVOLVEMENT IN TAKAYASU ARTERITIS PATIENTS

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Background: Takayasu Arteritis is a rare large-vessel vasculitis variant that affects the aorta and its main branches and the pulmonary arteries. Antiphospholipid syndrome is characterized by obstetric and thrombotic complications in the presence of antiphospholipid antibodies, which consist of anticardiolipin antibody, lupus anticoagulant and anti -β2 glycoprotein I. The association of antiphospholipid antibodies and Takayasu arteritis is very rare and few cases decumented it, while others argued aganist such association.

Objectives: This study was planned to find out the prevelance of immunoglobulin-IgM/G anti-cardiolipinantibodies, anti beta 2 glycoprotein- 1antibodies and lupus anticogulant and evaluate the relationship between these antibodies and disease severity/complications in Takayasu arteritis patients.

Methods: 53 patients with Takayasu arteritis patients were enrolled in this study. We obtained blood samples to detectIgM/G anti-cardiolipin antibodies, anti beta 2 glycoprotein 1 antibodies and lupus anticogulant (LA) levels from all patients during their routine control. ImmunoglobulinIgM/G anti-cardiolipin antibody, anti beta 2 glycoprotein 1 antibodies were measured by using a standardized enzyme-linked immunosorbent assay (ELISA) and lupus anticogulant was measured using the diluteRussell's viper venom time (dRVVT).

Results: No patients was positive forIgM/G anti-cardiolipin antibody. Seven were positive immunoglobulin IgM anti beta 2 glycoprotein 1 antibodies, three were positive immunoglobulin IgG anti beta 2 glycoprotein 1 antibodies, one waspositive LA. All of the antibody titters were low. TA patients who had antibody positivity had longer disease duration ($p<0.05$). Antibody and LA positive patients had superior mesenteric artery and celiac artery involvement more frequently then the antibody negative patients ($p<0.05$).

Conclusions: In this study that there were no association between the antibodies positivity and vascular involvement or disease complications and severity in TA patients. In conclusion we can not suggest the routine evaluation of antiphospholipid antibodies or lupus anticoagulant test during the follow-upTakayasu arteritis patients. These antibodies may only be measured in the presence of clinical suspicion.

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AB0587 ANTINEUTROPHILIC CYTOPLASMIC ANTIBODY-ASSOCIATED VASCULITIS AND HYPOCOMPLEMENTEMIA: CLINICAL IMPACT AND OUTCOME

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Background: Although their pathophysiology are still largely unknown, there are growing evidences that complement (C) alternative pathway activation is implicated in antineutrophilic cytoplasmic antibody-associated vasculitis (AAV) pathogenesis.

Objectives: The aim of our study was to evaluate the clinical characteristics and outcome of AAV patients, according to their serum C levels at diagnosis.

Methods: A retrospective monocentric study carried out in Caen University Hospital led to identify proteinase-3 (PR3) or myeloperoxidase (MPO)-ANCA AAV patients (via an ELISA technique). All patients with available C3 and C4 levels (by nephelemetry) at diagnosis were included, except for eosinophilic granulomatosis with polyangiitis (EGPA), which has a different pathophysiology. AAV were classified between granulomatosis with polyangiitis (GPA) or microscopic polyangiitis (MPA), and limited or severe forms according to respectively European Medicines Agency vasculitis algorithm and WGET group. Patients were categorized in the hypocomplementemia group if the C3 or C4 level at diagnosis was below the lower limit of the normal range (respectively 750–1400 mg/l and 100–340 mg/l). Categorical variables were reported as percentages and compared using Fisher's tests. Continuous variables were expressed as means and analyzed using Student's t-test. Associations between survival, renal survival and relapse-free survival, and low serum C levels were evaluated by the log-rank test. A p-value <0.05 was considered to be statistically significant.

Results: Among the 157 AAV patients identified, 81 were excluded (8 EGPA, 73 without C3 and C4 determinations before treatment initiation). On the 76 AAV included (43 GPA, 33 MPA), median age at diagnosis was 65 years (M/F, 38/38). Clinical presentations included constitutional symptoms (56, 73.7%), pulmonary (52, 68.4%), renal (50, 65.8%), rheumatologic (43, 56.6%), and ear, nose or throat (37, 48.7%) involvements, without statistical differences between groups. Twelve (15.8%) deaths and 41 relapses in 25 (32.9%) patients were noted (median follow-up: 38 months). Four patients (5.3%) had hypocomplementemia: 1 patient had isolated low C3 level, 1 had isolated low C4 level, and 2 had both low C levels. All 4 patients had renal involvement. The C level, controlled in 1 patient, became normal 1 month later. No thrombotic microangiopathy (TMA) features were found on the 2 performed kidney biopsies.

	Hypocomplementemia group (n=4)	Normal complement level group (n=72)	p value
Male %	50	50	1
Age at diagnosis (years), median ± SD	71±9	65±16	0.18
BVAS, median ± SD	21±8	18±7	0.26
Granulomatosis with polyangiitis %	75	57	0.64
PR3-ANCA %	50	60	1
Limited vasculitis %	0	28	0.57
End-stage renal disease %	75	15	0.02
Death %	50	14	0.12
Relapse %	25	36	1

Survival and renal survival were significantly lower in the hypocomplementemia group ($p=0.0011$ and $p<0.001$, respectively), but relapse-free survival was similar ($p=0.1$).

Conclusions: Hypocomplementemia at AAV diagnosis may be responsible for worse survival and renal prognosis. This particular phenotype may confer resistance to common immunosuppressive approaches as in thrombotic microangiopathy caused by abnormalities in the regulation of the C system. These results also argue for larger studies and for investigating C pathway targeting.

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AB0588 CONCOMITANT ASSOCIATION OF GIANT CELL ARTERITIS AND MALIGNANCY: A MULTICENTER RETROSPECTIVE CASE-CONTROL STUDY

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