patients with PR3-ANCA, patients with MPO-ANCA presented a lower prevalence of toxic syndrome (42.9% vs. 55.6%, p=0.021), arthralgias (34.3% vs. 59.9%, p=0.008), arthritis (8.6% vs. 26.5%, p=0.026), pulmonary involvement (cavitated/infiltrated nodules, p=0.007 and p=0.05), and anaemia (57.1% vs. 77.8%, p=0.05). Renal disease was less severe in patients with MPO-ANCA (creatinine ≥1.58 mg/dL 14.3% vs. 30.4%,p=0.04). Subglottic stenosis and sensoryneural deafness were more frequent in patients with MPO-ANCA (20% vs. 6.8%, p=0.05% and 34.3% vs. 18%, p=0.03). The mean BVAS at baseline was lower in patients with MPO-ANCA (16.3□8.8) and negative ANCA (12.5□6.7) than in patients with PR3-ANCA (19.6□8.9), p=0.029. Patients with negative ANCA had less frequently toxic syndrome, fever, arthritis, subglottic stenosis, kidney disease, and peripheral neuropathy, and more frequently orbital masses. Disease relapses were less frequent in patients with MPO-ANCA than in patients with PR3-ANCA (37.1% vs. 48.8%, p=0.04), but more frequent than in patients with negative ANCA (20.8%), p=0.002. Patients with MPO-ANCA and negative ANCA received less frequently oral cyclophosphamide. No significant differences were found related to death in patients with MPO-ANCA and PR3-ANCA. Patients with negative ANCA had the lower mortality

Conclusions: A small percentage of patients with GPA present MPO-ANCA or negative ANCA. In our series, patients with MPO-ANCA were older at the disease onset, presented limited or less severe organic disease than patients with PR3-ANCA, lower percentage of relapses and lower requirement of aggressive therapies. Patients with negative ANCA had the best prognosis. Our findings are similar to those recently published,[1] although our patients with MPO-ANCA were older. Classification of GPA patients considering ANCA specificity can improve the treatment stratification and reduce adverse events.

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AB0697 GIANT CELL ARTERITIS WITH NORMAL INFLAMMATORY MARKERS AT DIAGNOSIS

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Background: GCA is an inflamatory vasculitis affecting medium and large-sized arteries, that can result in arteritic anterior ischaemic optic neuropathy. C-reactive protein (CRP) level and erythocyte sedimentation rate (ESR) are usually elevated at GCA diagnosis, but inflammatory-marker negative disease does occur.
Objectives: To analyse the clinical and histological findings of patients with biopsy-proven GCA and negative inflammatory-markers at diagnosis.
Methods: multicenter-longitudinal retrospective study that included patients with biopsy-proven GCA recruited at 10 Hospitals from Spain (REVAS Study). Statistical analysis was performed using SPSS vs. 21.
Results: 418 patients: 290 (69.4%) females (ratio F/M: 2.3/1) were included. The mean age at diagnosis was 75.5±7 (53–92). The most frequent symptoms at diagnosis were recent onset headache (81%), toxic syndrome (47%) and rheumatic polymyalgia (44.5%). Jaw claudication, cranial hyperesthesia and amaurosis fugax were reported by 44.5%, 31.8% and 16.5% of patients, respectively. A total of 84 patients suffered permanent vision loss. Fourteen patients (3.3%) had normal ESR (<40 mm/h) and CRP (<5 mg/L) at diagnosis. No significant differences were found related to age at disease onset in these patients. Most patients (85%) reported headache; 42.9% jaw claudication, 28.6% cranial hyperesthesia and 42% rheumatic polymialgia. Fever was less frequent in patients with negative inflammatory-markers (7.7% vs. 40.4%, p=0.020), as well as toxic syndrome (21.4% vs. 51.5%, p=0.031). In contrast, patients with negative inflammatory-markers had more frequently amaurosis fugax (35.7% vs. 16.8%, p=0.03) and optic ischaemic neuropathy (50% vs. 18.7%, p=0.009). Temporal arteries were abnormal (thickened and/or pulse less) in 78.6% of patients with negative inflammatory-markers vs. 37% of patients with elevated ESR and/or CRP. Anaemia was less common in patients with negative inflammatory-markers (28.6% vs. 81.5%, p<0.001, mean haemoglobin 12.9±1.1 g/dL). No significant differences were found related to temporal artery biopsy findings, although patients with normal ESR and CRP showed giant cells in 74.3% of cases vs. 62%.

Conclusions: typically patients with GCA present with elevated inflammatory-markers (ESR and CRP) at disease onset. However, a few percentages of patients (4%–5%) have normal ESR and CPR at diagnosis.[1] In our series, 3.3% of patients had negative inflammatory-markers at diagnosis. These patients had fewer constitutional symptoms and more visual symptoms (amaurosis fugax and permanent visual loss) than patients with elevated ESR and/or CRP. Our result are similar to those published in the laiterature. Abnormal temporal arteries on physical examination may help to diagnosis

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AB0698 A COMPARISON OF THE EFFECTIVENESS OF MYCOPHENOLATE MOFETIL OR METHOTREXATE IN COMBINATION WITH PREDNISOLONE VERSUS PREDNISOLONE ALONE IN THE TREATMENT OF LARGE VESSEL VASCULITIS

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Background: The mainstay of treatment for large vessel vasculitis is glucocorticoids. Immuno-suppressants, including mycophenolate mofetil (MMF) and methotrexate (MTX) are used as steroid-sparing agents.[1] A previous study at our centre showed MMF to have a steroid sparing effect in 97% of patients and to reduce C-reactive protein (CRP) in 80%.[2]
Objectives: This study was undertaken to compare the efficacy of MMF or MTX combined with prednisolone or prednisolone alone in the treatment of large vessel vasculitis.
Methods: Patients with large vessel vasculitis (LVV) confirmed on positron emission tomography (PET) scan and those meeting ACR criteria for a diagnosis of giant cell arteritis (GCA), treated with prednisolone alone, prednisolone with MMF or prednisolone with MTX started within 3 months of prednisolone being commenced and with a minimum follow up of 24 months were included in a retrospective single centre study. CRP and prednisolone doses were recorded at baseline, after 3, 6, 9, 12, 18 and 24 months of treatment and area under the curve (AOC) calculated for CRP and prednisolone doses. Median AOC prednisolone dose for patients treated with MMF or MTX was then compared with that of patients treated with prednisolone alone. A quantile regression model was also constructed to compare prednisolone dose between the 3 treatment groups, adjusted for CRP. StataCorp. 2013. *Stata Statistical Software: Release 13*. College Station, TX: StataCorp LP was used for all statistical calculations.
Results: 65 patients were included in the study, 41 with GCA and 24 LVV. 49 were female and 16 male. Mean age at diagnosis was 68; range 21 to 87. 37 patients were treated with prednisolone alone; 35 had GCA and 2 LVV. 20 were treated with MMF and prednisolone; 4 with GCA and 16 LVV. 8 were treated with MTX and prednisolone; 2 had GCA and 6 LVV. The AOC for prednisolone and CRP were not normally distributed across the cohort, and non-parametric methods were therefore used for comparisons. Median AOC prednisolone dose for the prednisolone only group was 68.0, (interquartile range (IQR) 17.7, n=37), for the MMF treated group 70.8 (IQR 28.7, n=20) and for the MTX treated group 67.8 (IQR 20.4 n=8). Median AOC CRP was highest in the group treated with prednisolone alone (58.9, IQR 34.5) compared to MMF (43.8, IQR 26.5) and MTX (49.3 IQR 67.5) but there were no statistical differences between median AOC prednisolone dose or CRP in either the unadjusted or regression models.
Conclusions: No significant difference was shown between the groups. MMF is as effective as MTX and prednisolone alone in the treatment of LVV. However, there are limitations to the study. The patient group was small. There was no randomisation to treatment group; treatment choice was based on clinician preference. There was potential bias in that patients perceived to be more difficult to treat may have been given MMF or MTX in addition to prednisolone, and there was a higher proportion of patients with LVV compared to GCA in the MMF and MTX treated groups.

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