an amputation had been performed or not despite a primary successful procedure.

Results: In this cohort of 19 patients, 16 were male (84%), all were tobacco active users, and 21% cannabis users. Median follow-up was 19.9 months [11.0–36.9]. Iloprost infusion had been tested for 84.2% of the patients before revascularisation, and 63.2% had a dihydropyridine, 73.3% a statin and 100% at least aspirin. Endovascular procedure was exclusive for 14 patients, and bypass surgery for 1 patient. Four patients had combined procedures. Globally, 44 procedures including 7 bypasses (15.9%) and 37 endovascular surgeries (84.1%), due to multiple procedures for some patients. Primary patency of endovascular procedures was 89.4% and their patency duration was 221 ± 173 days. For bypass surgery, the patency duration was higher 827 ± 408 days ($p=0.273$). Looking at the territory, aorto-iliac revascularizations gave better results on long term 1830 days followed by upper limb 751 ± 402 days, femoro-popliteal procedures 347 ± 196 days and finally infra-popliteal ones with 178 ± 134 days.

Stability or improvement was observed after 52.3% of the revascularizations. Among the failures, 21.2% were associated with a minor amputation, and 11.3% with a major amputation. No predicting factors of failure could have been determined.

Conclusions: We report the largest series of revascularization in TAO. As expected, proximal artery revascularisation of lower limbs were associated with a higher and a longer patency rate than femoro-popliteal procedures and even more than infra-popliteal revascularisations. Surprisingly, upper limb revascularisation had a better outcome than for lower limbs. Efficacy of revascularizations are difficult to assess because such procedures are often proposed as a salvage option. However, it is worth to note that more than 50% of the patients had a benefit from the revascularisation.

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SERUM ANGIOGENESIS BIOMARKERS PREDICT DISEASE OUTCOME IN GIANT CELL ARTERITIS

Y. van Sleen, M. Sandovici, W. Abdulahad, J. Bijzet, A. Boots, E. Brouwer.
Rheumatology and Clinical Immunology, University Medical Center Groningen, Groningen, Netherlands

Background: Giant cell arteritis (GCA) is characterised by inflammation of the medium and large vessels. Two forms of GCA are described, C(cranial)-GCA and LV(large vessel)-GCA, which can be present either separately or co-exist in a patient. Clinical features, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are used for disease diagnosis and monitoring, but are not disease specific. Relapses are common during and after treatment, therefore new biomarkers are needed for diagnosis and disease course prediction.

Objectives: As the majority of infiltrating cells in the vessels of GCA patients are macrophages the aim of this project was to identify and compare levels of macrophage products in the serum of GCA patients as potential biomarkers.

Methods: Forty-one newly diagnosed GCA patients (temporal artery biopsy proven C-GCA and FDG-PET scan-positive LV-GCA) were recruited before start of glucocorticoids. Disease course was monitored and time to glucocorticoid treatment free remission was documented. Thirty age- and sex matched healthy controls (HCs) and 13 infection controls (bladder or lung infection) were also included. Serum concentrations of interleukin (IL)–6, serum amyloid A (SAA), soluble CD163 (sCD163), calprotectin, YKL-40 (human cartilage glycoprotein-39), vascular endothelial growth factor (VEGF), and angiotensin-1 and 2 were determined by ELISA or Luminex assay.

Results: IL-6, SAA, sCD163, calprotectin, YKL-40, VEGF and angiotensin-2 levels are increased in GCA and infection controls compared to HCs. IL-6 levels correlated strongly with CRP, ESR and SAA, all markers of the Acute Phase Response (APR), in GCA. Interestingly, YKL-40, angiotensin-2 and calprotectin levels showed only weak or no correlation with APR biomarkers, while they were strongly correlated with the APR in infection controls. Monocytes in peripheral blood correlated with APR biomarkers in GCA, whereas neutrophils correlated with the APR in infection controls. Patients with overlapping C-GCA and LV-GCA displayed a significantly stronger APR than patients with C-GCA or LV-GCA alone. High VEGF and angiotensin-1, but low angiotensin-2 levels at baseline predicted a shorter time to treatment free remission. This is in contrast to markers of the APR, which did not significantly predict time to treatment free remission.

Conclusions: In this study, we show that markers of angiogenesis are better predictors of disease outcome than APR biomarkers. It appears that levels of calprotectin, YKL40 and angiotensin-2 are increased through other than APR pathways during GCA compared to acute infection. Monocytes rather than neutrophils appear to drive the APR in GCA. This response is stronger in GCA patients with both cranial and systemic symptoms.

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Osteoporosis

THU0469

QUANTIFYING THE TREATMENT WITH GLUCOCORTICOID AS A RISK FACTOR FOR THE OCCURRENCE OF OSTEOPOROSIS AND FRACTURES IN PATIENTS WITH RA

E. Wiebe, R. Biesen, K.N. Zeiner, D.C. Freier, S. Hermann, G.-R. Burmester, F. Buttgerit.
Department of Rheumatology and Clinical Immunology, Charité – University Medicine Berlin, Berlin, Germany

Background: Rheumatoid arthritis (RA) is associated with increased systemic bone loss, leading to a high risk for fragility fractures. The etiology of increased fracture risk in RA is multifactorial and comprises next to general risk factors also RA-specific risks, most prominently chronic inflammation, seropositivity and glucocorticoid (GC) use¹. Yet, there is evidence that GCs may, by adequately suppressing systemic inflammation, also have a positive effect on BMD and fracture risk in RA².

Objectives: The purpose of this study was to investigate the prevalence of osteoporosis and fragility fractures in RA patients and to characterise, among other risk factors, the role of GC dose, cumulative dose (GCCD) and duration as well DMARD treatment on bone health.

Methods: Rh-GIOP is an ongoing prospective observational study collecting and analysing disease- and bone-related data from patients with chronic rheumatic diseases treated with GCs. In this cross-sectional analysis, we evaluated the initial visit of 238 patients with RA. Descriptive analyses were performed, with values displayed as mean/standard deviation and median/range for continuous variables. For subgroup analyses, non-parametric tests were used.

Results: Of 238 patients with RA (79.4% women, mean age: 63.6 ± 12.5 years), 155 were seropositive and 83 seronegative. Seronegative patients were numerically older (66.8 ± 12.1 vs 61.8 ± 12.1 years) and more often in menopause (78.3% vs 61.8%, ns) than seropositive, while the latter had longer disease duration (median: 4.0 vs 11.0 years, $p=0.03$). Overall, osteoporotic BMD was more frequent at femoral sites, with 21% of patients having T-Scores < -2.5 . Osteoporotic BMD was more common in seronegative patients (ns), although no difference in the frequency of fragility fractures ($n=18/24.1\%$ vs $n=28/23.2\%$) was found.

All patients received GCs (mean dose: 5.0 ± 6.8 mg, mean GCCD 15.1 ± 19.3 g, mean duration 7.7 ± 8.2 years) with seropositive patients having numerically higher GCCD, longer duration of GC therapy and more often current GC doses above >10 g/day. Biological DMARDs were more frequently used in seropositive patients ($n=20/24.1\%$ vs $n=67/43.2\%$, $p=0.02$). Anti-osteoporotic therapies between both groups did not differ.

Neither current GC doses nor GCCD nor DMARD therapy had a statistically significant and independent effect on BMD or fragility fractures in either RA group.

Conclusions: Osteoporosis and fragility fractures remain a challenge in the management of RA, being determined by multiple interacting factors. Our data confirm that GCs may not *per se* increase fracture risk and decrease BMD in RA but rather, that optimal management of disease activity with or without GCs may be beneficial to bone health. Interestingly however, despite higher cumulative GC doses and duration, seropositive RA patients did not have lower BMD or higher prevalence of fragility fractures compared to seronegative patients. Further prospective data is warranted to better characterise the role of GCs and DMARDs in regard to osteoporosis and fracture risk in RA patients.

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