improvement as well as worsening in GC toxicity, and consists of two subscores. With the Cumulative Worsening Score (CWS), only worsening toxicities are counted. The CWS is always a positive number or, if no toxicity has occurred, 0. With the Aggregate Improvement Score (AIS), both improvement and worsening are considered. If the overall GC toxicity has improved, the AIS is negative.

Objectives: To evaluate the GTI in a prospective cohort of GC-treated vasculitis patients.

Methods: Vasculitis patients were included if starting GC or having a flare requiring increased GC. Doses were calculated in prednisone (PRED) equivalents. Nearly all patients had received GC (often for years) baseline (V1). Data for the 9 GTI domains were collected at V1 and follow-up (V2). These included: medications for hypertension, hyperglycemia, and hyperlipidemia; body mass index; bone mineral density; and data pertaining to muscle strength, skin toxicity, neuropsychiatric effects, and infections. Data for the blood pressure, glucose control, and lipid domains were inferred from treatment changes. We used multivariate logistic regression models to examine the relationship of PRED dose to the likelihood of CWS worsening and AIS improvement.

Results: 37 patients (25F; 12M) of mean age 66.6 years (range: 40.5–81.7) were included. Diagnoses: GCA (n=7), PMR (14), AAV (10), EGPA (4), and PAN (2).

Table 1. Prednisone Dosing

Mean Cumulative PRED Dose V0 & V1 (mg)(range)	7888 (30-55,800)
Mean Cumulative PRED Dose V1 & V2 (mg)(range)	3762 (108-14,207)
Days Between V0 & V1 (range)	1095 (2-7,443)
Days Between V1 & V2 (range)	613 (335-1,111)
Mean Daily PRED Dose Before V1(mg)(standard deviation, SD)	6.1 (6.2)
Mean PRED dose V1 (mg)(SD)	34.2 (36.5)
Mean PRED dose V2 (mg)(SD)	3.1 (2.9)

Thirty-four (92%) patients had changes in GC toxicity between V1-V2. The mean CWS & AIS were 34.8 and 11.1, respectively. Twenty-eight patients (76%) had CWSs >0, indicating worsening toxicity. Twenty-one (57%) had AISs >0, also consistent with overall GC toxicity worsening. However, 13 (35%) had negative AISs, indicating improvements in GC toxicity. BMD measurements worsened by at least 3% in 16 (43%) patients and improved by at least 3% in 9 (24%). Cumulative PRED doses correlated strongly with the CWS. For every 1000mg of PRED between V1 & V2, the risk of CWS increase – worsening of GC toxicity – rose by 79% (Table 2).

Table 2. GTI Score Relationships to PRED Dose

PRED Dose	Total (n=37)	CWS		AIS	
		Did not worsen (n=9)	Worsened (n=28)	Improved (n=13)	Did not improve (n=24)
V1-V2 (mg/d)(SD)	6.3 (4.5)	4.8 (5.9)	6.9 (4.0)	5.5 (4.6)	6.8 (4.5)
Cumulative PRED V1&V2 (mg; SD)	3761 (2660)	2027 (2165)	4319 (2592)	3147 (2047)	4094 (2925)
		OR (95% CI)*			
		CWS		AIS	
Daily Mean Dose V1-V2** (per 1mg)		1.15 (0.92-1.43)		0.93 (0.78-1.11)	
Cumulative PRED V1&V2 (per 1000mg)**		1.79 (1.06-3.03)		0.85 (0.63-1.16)	

V0 = Date when PRED was started, before baseline (V1)

*OR for CWS refers to risk of worsening. OR for AIS refers to risk of improvement.

**Adjusted for age, sex.

Conclusion: The GTI captured changes in GC toxicity over time well, both improvement and worsening. Cumulative PRED dose correlated strongly with increase in the Cumulative Worsening Score.

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NEUTROPHIL-, MONOCYTE-, EOSINOPHIL- AND BASOPHIL-LYMPHOCYTE RATIOS: ASSOCIATIONS WITH TRADITIONAL INFLAMMATORY MARKERS AND DISEASE ACTIVITY MEASURES AT BASELINE AND 6 MONTHS AFTER A bDMARD AMONG BIOLOGIC-NAÏVE RHEUMATOID ARTHRITIS PATIENTS

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Background: Neutrophil-lymphocyte (NLR) and monocyte-lymphocyte (MLR) ratios have been not only suggested as systemic inflammatory markers, but also associated with disease activity in some inflammatory diseases. Eosinophil-lymphocyte (ELR) and basophil-lymphocyte (BLR) ratios have been studied to a lesser extent.

Objectives: To correlate NLR, MLR, ELR and BLR with inflammatory markers, other laboratory markers and disease activity measures in a portuguese sample of rheumatoid arthritis (RA) patients.

Methods: We conducted a retrospective study including all RA patients under the first bDMARD (*biological disease-modifying antirheumatic drug*), followed in a tertiary university hospital and registered in the Rheumatic Diseases Portuguese Register (*Reuma.pt*). Results of blood tests (C-reactive protein [CRP], erythrocyte sedimentation rate [ESR], serum amyloid A [SAA], rheumatoid factor [RF], anti-citrullinated protein autoantibodies [ACPA], haemoglobin, platelets, 25-hydroxide-vitamin D [25OHvitD]), leukocyte formula ratios (NLR, MLR, ELR, BLR) and disease activity and function measures (disease activity score 28 [DAS28 4V, 3V, 4V-CRP, 3V-CRP], clinician and simple disease activity scores [CDAI, SDAI], swollen and tender joint count [SJC, TJC], physician and patient visual analogical scale [VAS]) at baseline and at 6 months of bDMARD therapy were collected. Correlations between variables were evaluated by Spearman rank test (significance level at $p < 0.05$), using SPSS 25.0 software.

Results: 435 patients were included: 81.2% female, mean age at baseline 53.3 ± 11.4 years, mean disease duration 12.6 ± 8.8 years, 72.2% RF positive, 77.1% ACPA positive, 52.5% erosive disease.

At baseline, NLR correlated positively with CRP ($r = 0.343$; $p < 0.001$), ESR ($r = 0.124$; $p = 0.031$), RF title ($r = 0.185$; $p = 0.009$), platelets count ($r = 0.137$; $p = 0.018$), DAS28(4V) ($r = 0.183$; $p = 0.002$), DAS28(3V) ($r = 0.159$; $p = 0.007$), DAS28(4V-CRP) ($r = 0.235$; $p < 0.001$), DAS28(3V-CRP) ($r = 0.222$; $p < 0.001$), SDAI ($r = 0.178$; $p = 0.024$), SJC ($r = 0.120$; $p = 0.040$), TJC ($r = 0.139$; $p = 0.018$) and patient VAS ($r = 0.133$; $p = 0.026$). At baseline, we also found a negative correlation between NLR and 25OHvitD levels ($r = -0.286$; $p = 0.012$). Baseline MLR showed a positive correlation with baseline CRP ($r = 0.138$; $p = 0.017$), RF ($r = 0.156$; $p = 0.027$) and patient VAS ($r = 0.135$; $p = 0.023$). Similarly, at baseline, ELR correlated with CRP ($r = 0.115$; $p = 0.048$) haemoglobin levels ($r = 0.126$; $p = 0.029$) and DAS28(4V-CRP) ($r = 0.126$; $p = 0.037$); and BLR associated positively with DAS28(4V-CRP) ($r = 0.138$; $p = 0.022$), TJC ($r = 0.121$; $p = 0.038$) and patient VAS ($r = 0.131$; $p = 0.028$). At six months of a bDMARD, NLR correlated positively with: CRP ($r = 0.340$; $p < 0.001$), ESR ($r = 0.140$; $p = 0.018$), SAA ($r = 0.350$; $p = 0.008$) and physician VAS ($r = 0.186$; $p = 0.023$). MLR also exhibited a positive association with CRP ($r = 0.161$; $p = 0.007$). Similarly, at six months, ELR correlated positively with CRP ($r = 0.120$; $p = 0.043$), RF ($r = 0.135$; $p = 0.046$), CDAI ($r = 0.180$; $p = 0.027$) and SDAI ($r = 0.183$; $p = 0.025$); and BLR correlated positively with platelets count ($r = 0.139$; $p = 0.020$).

Conclusion: Although weak to moderate in strength, we found significant correlations, between leukocyte formula ratios (NLR, MLR and ELR) and, not only, inflammatory markers but also disease activity measures, predominantly at baseline but also at 6 months of bDMARD. Interestingly, is of note the negative association between NLR and 25OHvitD at baseline. This study reinforced the suggested place of NLR and MLR (but also of ELR) as inflammatory markers and possible disease activity measures in RA. More studies are needed to validate this data.

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