Conclusion: Our study has shown similar graft and patient survival as well as renal outcome in renal transplant due to the three types of RPGN. Renal transplantation could be the best option for patients with end stage renal disease due to RPGN regardless of systemic manifestations.

Figure 2. Patient survival.

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THU0321 URINARY T CELLS IDENTIFY ACTIVE RENAL ANCA ASSOCIATED VASCULITIS

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Background: ANCA-associated vasculitis (AAV) causes necrotizing crescentic glomerulonephritis (NCGN) which is a major contributor to morbidity and mortality in AAV. Since therapy relies on cytotoxic agents with potentially severe adverse effects, a reliable non-invasive biomarker of disease activity is needed to determine the right balance between over- and undertreatment. Using the urine space as a window into the local inflammatory milieu of the kidney, quantification of urinary leukocytes using flow cytometry has become an upcoming marker of various other inflammatory kidney diseases. As recent studies have illuminated the emerging role of T regulatory (Treg) and Th17 cells in the pathogenesis of AAV, these could represent a viable non-invasive biomarker more closely displaying the underlying pathogenic processes than metabolites or epiphenomena of inflammation.

Objectives: To quantify urinary T cells in active renal AAV and assess their biomarker characteristics.

Methods: Using flow cytometry, T-lymphocytes and their subsets were quantified in peripheral blood and urine samples from patients with active AAV with or without NCGN, in stable remission with previous NCGN and healthy controls. Concentrations of urinary soluble metabolites and cytokines (Monocyte-attracting protein 1 (MCP-1), sCD163, sCD25 and C5a) were measured using Multiplex analysis. Results were verified in a separate validation cohort.

Results: Patients with renal active AAV (n = 30) showed significantly higher urinary cell counts of total T cells, CD4+, CD8+, Treg and Th17 subsets than disease (n = 21) and healthy controls (n = 8). Patients with active renal AAV also showed a significantly higher percentage of Tregs in urine than in blood. While Tregs allowed a robust discrimination between active renal AAV and disease controls (receiver operator characteristics (ROC): area under the curve (AUC) 0.93, sensitivity 79%, specificity 95%) quantification of all T cells proved to be slightly more accurate (ROC: AUC 0.95, sensitivity 92%, specificity 95%). Soluble markers showed a slightly inferior discrimination (MCP-1 ROC: AUC 0.90, sensitivity 60%, specificity 100%, sCD163 ROC: AUC 0.92, sensitivity 96%, specificity 85%) while sCD25 and C5a were far less accurate.

Conclusion: Urinary T cells are significantly elevated in active renal AAV and the increased frequency of Tregs in urine suggests active migration into inflamed glomeruli and thereby the urine rather than mere bleeding of ruptured capillaries. These cells show great potential for a non-invasive biomarker close to the local inflammatory milieu. Particularly the total count of urinary T cells showed slightly superior biomarker characteristics than previously established soluble markers. Further studies are needed to confirm these results and show potential prognostic value of these cellular markers.

Fig. 1 Boxplots of urinary T cells in ANCA associated vasculitis, dotted line indicates optimal cut-off to determine renal disease activity
Kruskal-Wallis with post-hoc Dunn's, p-value summary: * = 0.05 – 0.01 ** = 0.01 – 0.001, *** ≤ 0.001
GN: glomerulonephritis, Rem: stable remission with previous GN

Disclosure of Interests: None declared

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THU0322 EFFECTIVENESS OF COMBINED LOCAL COLCHICINE AND STEROIDS IN ORAL ULCERS OF BEHÇET'S DISEASE

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Background: Behçet's disease (BD) is a chronic, multi-system vasculitic disease. It is characterized with relapsing episodes of oro-genital ulcers accompanied by cutaneous lesions, ocular symptoms, arthropathy, vascular thrombosis, central nervous system, gastrointestinal & cardiopulmonary involvements. Oral ulcers are frequently the first disease manifestation. Oral and genital ulcers cause pain and interfere with the quality of life. They may lead to difficulty in swallowing and walking. Most of them can be managed with topical glucocorticoids. Up till now there is no study discussed the effect of combined local therapy on oral ulcers in BD.^{1,2}

Objectives: To evaluate the effectiveness of combined local therapy (colchicine, steroid, antibiotic and anesthetic) on oral ulcers in BD

Methods: This study included 44 Patients who had Behçet's disease (according to International Study Group criteria) with active oral ulcers (at least three times in the previous 12-month period) Patients were excluded if they had active major organ involvement in the last 6 months. Patients with depilating diseases also were excluded.

Patients were randomly divided into two equal groups; group I received combined local therapy (lidocaine HCL 2.0% gel mixed with grinded tablet of 5mg prednisone and grinded tablet of 0.6mg colchicine). Group II received combine local therapy (lidocaine HCL 2.0% gel mixed with grinded tablet of 5mg prednisone). Local treatments were applied to the lesions 3 times per day until healing of the ulcer (advised not to eat or drink for 30 minutes after application). All other topical medications were stopped during this study.

All patients were assessed with Oral ulcer severity score (OUSS), Behçet's Disease Quality of Life score (BD-QoL), Medical Outcomes Study Questionnaire Short Form 36 Health Survey (SF-36) at baseline and after 24 weeks.

Results: Thirty eight patients had completed this study; (20 in Group I & 18 in Group II). There were no significant differences between the 2 groups in both demographic data & educational status. At baseline there was no significant difference between both groups regarding all assessment measures. There was significant improvement (P<0.05) in both groups regarding OUSS, BD-QoL, SF-36 after 24 weeks. There was significant better improvement

($P < 0.05$) in group I than in group II in all assessment measures (except ulcer free periods and sites). The results of the study are summarized in table 1 & figure 1.

Table 1. A comparison of the individual ulcer characteristics in both groups at baseline and after 24 weeks

Ulcer characteristics	Group I baseline	Group II baseline	Group I after 24 weeks	Group II after 24 weeks
Number	13.6 $\pm$ 3.5	12.9 $\pm$ 2.3	8.2 $\pm$ 1.5 [*] †	10.3 $\pm$ 1.7 [*]
Size	11.8 $\pm$ 3.3	12.5 $\pm$ 2.4	7.6 $\pm$ 1.8 [*] †	9.6 $\pm$ 1.2 [*]
Duration	6.7 $\pm$ 1.1	6.6 $\pm$ 1.7	4.5 $\pm$ 1.9 [*] †	5.5 $\pm$ 1.3 [*]
Ulcer-free period	4.7 $\pm$ 0.9	5.1 $\pm$ 1.5	5.7 $\pm$ 1.1 [*]	5.9 $\pm$ 0.7 [*]
Pain	8.7 $\pm$ 1.2	8.6 $\pm$ 1.4	5.3 $\pm$ 1.5 [*] †	6.7 $\pm$ 1.1 [*]
Site	4.9 $\pm$ 0.8	5.1 $\pm$ 1.2	3.8 $\pm$ 0.6 [*]	4.2 $\pm$ 0.9 [*]

^{*}Significant improvement after 24 weeks of the study

[†]Significant difference between the two studied groups

Conclusion: Combined local therapy (colchicine, steroid, antibiotic and anesthetic) is an effective method in management of oral ulcers in BD.

References:

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Figure 1: SF-36 (Total score) at baseline and after 24 weeks in both groups

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THU0323 MYELOID BIOMARKERS IN GIANT CELL ARTERITIS AND POLYMYALGIA RHEUMATICA – TWO INDEPENDENT COHORTS

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Background: Giant cell arteritis (GCA) commonly overlaps with polymyalgia rheumatica (PMR). The incidence of GCA among PMR patients is between 16 and 21%, and both diseases are treated with long-term glucocorticoids (GCs). Patients with GCA suffer from inflammation of their large-sized arteries, whereas PMR is characterized by synovial inflammation. A key question for every physician dealing with a PMR patient is whether or not the patient also has GCA. Symptoms of GCA are often non-specific and difficult to identify. Compared to healthy controls (HCs), newly-diagnosed GCA and PMR patients display a change in leukocyte composition with a shift towards the myeloid lineage, evidenced by elevated monocyte and neutrophil counts. Persistence of this myeloid bias (during and after treatment) is in congruence with mounting evidence that GCs do not sufficiently suppress the vascular/synovial inflammation, contributing to a relapsing disease course. Yet, it may be difficult to readily identify the myeloid bias in the blood, and therefore easily detectable biomarkers are required to monitor this myeloid bias in GCA and PMR patients.

Objectives: The first objective of this study is to identify disease specific biomarkers for GCA and for PMR using myeloid serum/plasma markers. Next, we assessed whether (a profile of) these markers could be used to reflect the persistent myeloid profile.

Methods: Biomarkers were measured in two independent cohorts: Groningen, the Netherlands (GPS cohort) and Aarhus Denmark (Aarhus cohort). Both cohorts included treatment-naïve GCA and PMR patients, supplemented with age- and sex matched HCs (Table 1). Along with the GPS cohort, age-matched inflammatory controls were included. GCA-lookalike patients were added in the measurements for the Aarhus cohort. All patients started treatment with GCs, and follow-up samples were measured at 8 weeks for GCA patients. All measurements were performed by ELISA: sCD206, calprotectin, A1AT and elastase in serum samples, whereas PR3 and MPO were measured in plasma samples.

Table 1. Baseline characteristics of the two cohorts

	GPS cohort				Aarhus Cohort			
	GCA	PMR	HC	Infection	GCA	PMR	HC	GCA-lookalike
N	48	39	50	16	52	25	20	18
PET-CT:	32 5 11	0 29 10	NA	NA	48 4 0	0 25 0	NA	0 13 5
positive/ negative/ not done								
CRP, median (mg/L)	52	35	2	70	74	35	1	54

Results: Analyses of biomarkers in the two cohorts had mostly equivalent results. Compared to HCs, levels of sCD206, calprotectin, PR3 and A1AT were elevated in GCA, PMR and infection/GCA-lookalike patients. GCA patients had higher levels of sCD206 than PMR patients, but only in the Aarhus cohort this reached statistical significance (Aarhus: $p = 0.02$, GPS: $p = 0.17$). Treatment with GCs substantially affected the biomarker levels: in GCA patients of both cohorts, calprotectin and A1AT levels dropped, sCD206 levels remained high (unchanged), and elastase and PR3 levels increased. Next, we assessed whether the biomarkers correlated with inflammation and the myeloid bias in the GPS cohort. Particularly in GCA patients, A1AT levels correlated with inflammatory marker CRP. Elastase correlated significantly with neutrophil counts in both GCA ($R = 0.42$) and PMR ($R = 0.57$). Calprotectin correlated with neutrophil counts in both GCA and PMR, and with monocyte counts in GCA.

Conclusion: This is one of the first studies in GCA and PMR patients to study biomarkers in two independent cohorts. We consistently showed elevated levels of monocyte/macrophage and neutrophil products in both cohorts. Levels of sCD206 may help in discriminating GCA from PMR patients. The myeloid bias may be monitored using a combination of calprotectin and elastase levels. Additionally, sCD206 or calprotectin may serve as tissue inflammation markers under the cover of GC treatment, a notion to be further investigated using follow-up imaging data.

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THU0324 CYTOMEGALOVIRUS REACTIVATION AND HIGH INITIAL SERUM CREATININE ARE SIGNIFICANT PROGNOSTIC FACTORS FOR SUBSEQUENT SEVERE INFECTIONS IN PATIENTS WITH ANCA-ASSOCIATED VASCULITIS

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Background: There are several reports that cytomegalovirus (CMV) reactivation resulted in more co-infections affecting survival in rheumatic disease, and CMV reactivation can lead to infections in granulomatosis with polyangiitis patients by inducing CD4+CD28- T cell and depressing naïve T cell populations.¹⁻⁴ Despite this evidence, the prognostic value of CMV reactivation for severe infections in patients with connective tissue disease are still unknown.

Objectives: The aim of this study was to examine prognostic factors for severe infection during the early phase of treatment, especially in CMV reactivation, in patients with antineutrophil cytoplasmic autoantibody (ANCA)-associated vasculitis (AAV) who received initial high dose corticosteroid therapy (prednisolone > 0.8mg/kg/day).