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O-01

An 11β-hydroxysteroid dehydrogenase type 1 (HSD-1) inhibitor to improve the benefit-risk profile of prednisolone in patients with polymyalgia rheumatica (PMR)

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Background. HSD-1 differentially regulates intracellular glucocorticoid levels in the immune system and glucocorticoid toxicity target organs. SPI-62 is a potent HSD-1 inhibitor.
Objective. To observe whether SPI-62 can both maintain prednisolone efficacy and reduce prednisolone toxicity in patients with PMR.
Methods. Patients with PMR and who received prednisolone 10 mg/day continued treatment for 4 weeks without dose reduction. They additionally received SPI-62 6 mg/day or matching placebo (PBO-PSL10) for 2 weeks each. In sequential cohorts, during SPI-62 treatment the prednisolone dose was 10 (SPI-PSL10), 15 (SPI-PSL15), or 20 mg/day (SPI-PSL20). Relapse was defined when the investigator further increased the prednisolone dose to manage a participant's PMR symptoms. Participants completed daily numeric rating scales for pain, stiffness, and fatigue intensity, and pain chronicity, and Health Assessment Questionnaire-Disability Index during trial visits. Inflammatory, bone, and lipid biomarkers were measured at Baseline, Week 2, and Week 4. Insulin resistance (HOMA-IR) was calculated as fasting glucose×insulin. Trial interpretation is based on descriptive statistics. Hepatic HSD-1 inhibition was monitored via urinary metabolites: (tetrahydrocortisol+allotetrahydrocortisol)/tetrahydrocortisone.
Results. SPI-62 achieved 94.7±4.6% hepatic HSD-1 inhibition. Relapse incidence and descriptive statistics for other parameters are presented (Table I). PMR therapeutic control was reduced with SPI-PSL10 and SPI-PSL15, but not SPI-PSL20, compared to PBO-PSL10. Substantial improvements were observed on bone, lipid, and glycemic control parameters related to prednisolone toxicity with each SPI-62-containing regimen.
Conclusion. In patients with PMR the combination of SPI-62 and prednisolone 20 mg, compared to prednisolone 10 mg alone, showed an improved benefit-risk profile comprised of similar efficacy and less evidence of prednisolone toxicity.
Key words: glucocorticoids, corticosteroids, clinical trial, drug toxicity, polymyalgia rheumatica

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Table I. Descriptive statistics

			Change from Baseline							
	Baseline		PBO-PSL10		SPI-PSL10		SPI-PSL15		SPI-PSL20	
N			38		13		12		11	
PMR relapse			0 (0%)		5 (38%)		0 (0%)		0 (0%)	
Parameter	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Pain intensity	2.76	2.02	-0.15	1.00	2.18	2.09	2.54	2.91	0.27	0.79
Stiffness intensity	2.47	2.20	-0.24	1.10	1.73	2.45	1.25	2.05	-0.18	0.98
Fatigue intensity	3.20	2.56	-0.73	1.50	1.64	1.50	0.50	1.31	-0.09	0.70
Pain chronicity	1.44	1.13	-0.13	0.67	0.64	0.67	1.10	1.45	-0.10	0.57
HAQ-DI total	0.68	0.61	-0.16	0.33	0.33	0.46	0.37	0.67	0.05	0.17
ESR (mm/h)	9.75	6.16	2.41	7.39	12.91	16.82	5.43	8.55	1.73	3.72
CRP (mg/L)	3.18	--	0	2.74	6	6.08	3.6	3.04	1	4.60
Fibrinogen (g/L)	3.69	0.72	-0.52	0.81	0.92	0.94	1.89	1.07	0.36	1.61
Interleukin 6 (ng/L)	4.90	6.07	-1.33	0.75	5.16	1.29	2.70	1.14	-1.27	1.37
Osteocalcin (µg/L)	15.03	4.87	-0.36	3.36	6.06	3.65	4.11	3.35	3.35	4.96
PINP (µg/L)	42.4	11.6	-2.64	6.85	14.77	11.50	13.75	13.14	10.24	11.14
CTX (ng/L)	484	129	20.6	96.3	-81.4	76.2	-49.5	92.5	-52.1	70.9
HOMA-IR	1.92	3.20	0.15	0.25	-0.14	0.46	-0.25	0.40	-0.04	0.49
Cholesterol (mM)	5.85	0.98	-0.02	0.10	-0.20	0.18	-0.45	0.16	-0.22	0.18
Triglycerides (mM)	1.44	0.71	-0.06	0.05	-0.23	0.10	-0.06	0.09	-0.20	0.10

SD: standard deviation; HAQ-DI: Health Assessment Questionnaire - Disability Index; ESR: erythrocyte sedimentation rate; PINP: procollagen type I N-propeptide; CTX: collagen type I C-telopeptide, beta cross-linked; C-reactive protein (CRP) is described by median and median absolute difference. Interleukin-6, homeostatic model assessment of insulin resistance (HOMA-IR), cholesterol, and triglycerides are described by least squares means and standard errors.

O-02

Definitions of and instruments for disease activity, remission, and relapse in polymyalgia rheumatica: a systematic literature review

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Background. In polymyalgia rheumatica (PMR), relapses occur in up to 55%, with periods of remission in between. However, there is no consensus on the measurement instruments to define different disease states, leading to varying definitions in clinical studies.
Objective. To perform a systematic literature review on definitions and instruments used to measure remission, relapse, and disease activity in PMR, to inform an OMERACT project to endorse instruments for these outcomes.
Methods. A search of Pubmed/MEDLINE, EMBASE, CINAHL, Cochrane, and Epistemonikos was performed May 2021 and updated August 2023. Studies published in English were included recruiting people with isolated PMR. Study selection and data extraction was performed independently by two investigators with disagreements resolved through discussion. Data extracted encompassed definitions of disease activity, remission and relapse, and instruments used.
Results. From 5,718 records, we included 26 articles on disease activity, 36 on remission, and 53 on relapse; 64 studies were observational and 15 interventional, with none using qualitative methods. Some heterogeneity was found regarding definitions and instruments encompassing the domains pain, stiffness, fatigue, laboratory markers, and patient and physician global assessment (Fig. 1). Instruments for clinical signs were often poorly described. Whilst measurement properties of the polymyalgia rheumatica activity score (PMR-AS) have been assessed, data to support its use for measurement of remission and relapse is limited.
Conclusion. Remission, relapse, and disease activity have been defined heterogeneously in clinical studies. Instruments to measure these disease states need validation. Qualitative research is needed to better understand the concepts of remission and relapse in PMR.
Key words: polymyalgia rheumatica, relapse, remission, disease activity, systematic literature review

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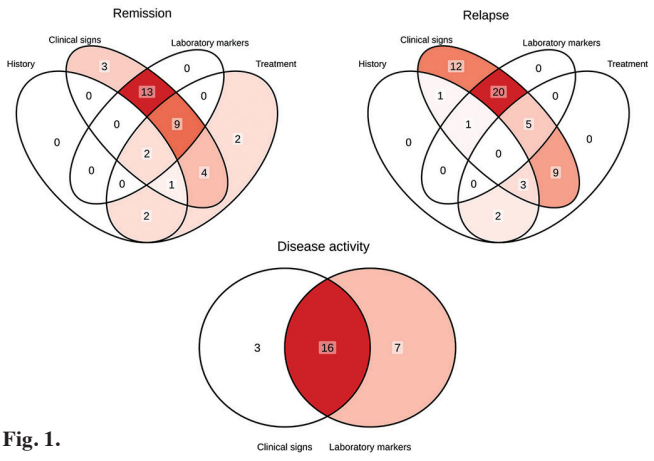

Fig. 1.

O-03

Diagnostic performance of head and neck FDG-PET/MR in new-onset GCA

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Background. 18F-FDG-PET/CT imaging measures metabolic activity, while MRI demonstrates morphological changes. Combining these modalities may result in a hybrid diagnostic test with high diagnostic accuracy for giant cell arteritis (GCA).
Objective. To evaluate the diagnostic performance of hybrid FDG-PET/MR in new-onset GCA at baseline and after 10 days glucocorticoid treatment.
Methods. Patients with suspected new-onset GCA, treated <3 days with glucocorticoids, were prospectively included. Standard diagnostic workup included vascular ultrasonography and whole-body FDG-PET/CT. Head and neck FDG-PET/MRI after FDG-PET/CT was performed using the same FDG administration. FDG-PET/CT and FDG- PET/MRI was repeated after 10 days of treatment in patients with GCA. The final clinical diagnosis was blinded for FDG-PET/MRI result. Head and neck arteries were independently assessed on FDG-PET, T1-weighted contrast-enhanced (T1WCE) MRI, and time-of- flight (TOF) MRI (Table I) by two experienced nuclear medicine physicians (NMPs) blinded for final clinical diagnosis and imaging timepoint (baseline or day 10).
Results. Baseline characteristics are shown in Table II. FDG-PET/MRI sensitivity was 95% and specificity 90% for GCA. Two patients had no FDG-uptake but showed vessel wall thickening or stenosis on MR imaging. Three of the 14 GCA patients who had repeated scan day 10 (range 8 to 13) did not show any signs of GCA on FDG-PET/MRI despite a positive baseline FDG-PET/MRI, thus resulting in a sensitivity of 79%. NMPs showed full agreement (Cohens kappa 1.0) regarding the final diagnosis.
Conclusion. Preliminary FDG-PET/MRI results shows high diagnostic accuracy for GCA. The sensitivity of FDG-PET/CT and FDG-PET/MRI decreases after 10 days of glucocorticoid treatment.
Key words: diagnostic imaging, giant cell arteritis, FDG PET/MRI

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Table I.

FDG PET	Grade 0 No vascular uptake
	Grade 1 Vascular uptake > background (considered pathologic)
	Grade 2 Vascular uptake >> background (considered pathologic)
T1WCE MR	Grade 0 No mural enhancement or thickening
	Grade 1 Slight mural enhancement but no thickening
	Grade 2 Significant mural enhancement and thickening (considered pathologic)
	Grade 3 Mural and perivascular enhancement and thickening (considered pathologic)
TOF MR	No stenosis
	Stenosis (considered pathologic)

Table II.

	GCA (N=20)	No GCA (N=10)
Age, years (mean, SD)	72, 62-78	65 (58-74)
Gender, female (N)	14	8
CRP, mg/L (mean, SD)	33, 15-52	40 (21-52)
Cranial symptoms present (N)	19	7
US positive (N)	17	0
Conventional baseline PET/CT positive (N)	18	0
GC treatment at time of baseline PET/CT (N)	12	6
>Days of GC treatment (median, range)	1 (0-3)	0 (0-2)

O-04

Efficacy and safety of upadacitinib in patients with giant cell arteritis (SELECT-GCA): a double-blind, randomized controlled phase 3 trial

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Background. Upadacitinib (UPA) is a strong inhibitor of IL-6 and IFN-γ, both implicated in GCA pathogenesis.
Objective. To assess the efficacy and safety of UPA vs placebo (PBO), plus a glucocorticoid taper regimen, in GCA.
Methods. Patients received UPA 7.5mg or 15mg (UPA15) daily plus a 26-week glucocorticoid taper or PBO with a 52-week glucocorticoid taper. Eligible patients were diagnosed with new-onset or relapsing GCA, and taking prednisone ≥20 mg daily at baseline. The primary endpoint was sustained remission, defined as absence of signs/symptoms of GCA from weeks 12 through 52 and adherence to the protocol-defined glucocorticoid taper.
Results. 428 patients were randomized and treated. Baseline characteristics were balanced across groups, with 70% and 30% of patients having new-onset and relapsing GCA, respectively. The primary endpoint of sustained remission at week 52 was achieved with UPA15 vs PBO (46% vs 29%, p=0.0019). The study met 9 out of 11 multiplicity-controlled secondary endpoints with UPA15. UPA15 resulted in a decreased risk for flare through 52 weeks vs PBO (Fig. 1). Safety outcomes over 52 weeks were generally similar among the UPA groups and PBO (Table I), including rates of venous thromboembolism. Numerically higher rates of serious infections were observed in the PBO group, and no major adverse cardiovascular events were reported in the UPA groups.
Conclusion. UPA15 with a 26-week glucocorticoid taper demonstrated superior efficacy compared to PBO with a 52-week glucocorticoid taper. No

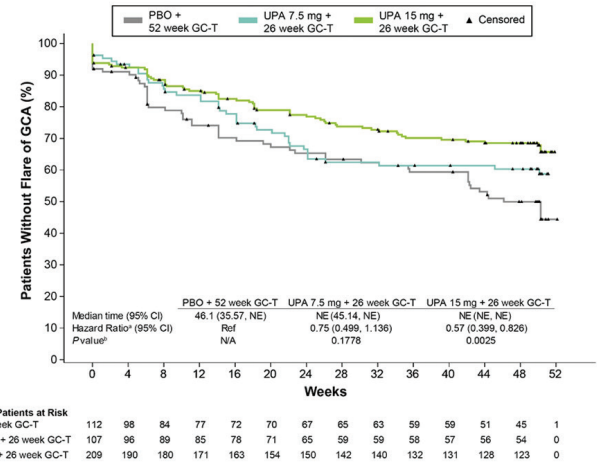

Fig. 1. Kaplan-Meier curves for time to first disease flare through week 52.
GCA: giant cell arteritis; GC-T: glucocorticoid taper; NE: not estimable; PBO: placebo; UPA: upadacitinib.
Patients who never met the "flare-free" criteria required prior to the assessment of disease flare were considered to have flares at baseline. Patients who met the "flare-free" criteria but did not have flares were censored at the last assessment prior to week 52.
[a]Hazard ratios were estimated using the Cox proportion hazard model.
[b]p-values were calculated using the log-rank test.

Table I. Exposure-adjusted event rates for adverse events of interest through week 53[a,b].

Events (E/100 PY)	PBO + 52-week GC-T (N = 112; PY = 94.3)	UPA 7.5 mg + 26-week GC-T (N = 107; PY = 88.5)	UPA 15 mg + 26-week GC-T (N = 209; PY = 178.1)
Any TEAE	706 (748.6)	597 (674.8)	1456 (817.7)
Serious TEAE	40 (42.4)	25 (28.3)	65 (36.5)
AE leading to study drug discontinuation	31 (32.9)	27 (30.5)	41 (23.0)
Serious infection	12 (12.7)	7 (7.9)	14 (7.9)
Opportunistic infection[c]	1 (1.1)	0	4 (2.2)
Herpes zoster	4 (4.2)	4 (4.5)	13 (7.3)
Malignancy (excluding NMSC)	2 (2.1)	0	4 (2.2)
NMSC	2 (2.1)	1 (1.1)	5 (2.8)
VTE (adjudicated)[d]	4 (4.2)	4 (4.5)	10 (5.6)
MACE (adjudicated)[e]	2 (2.1)	0	0
Bone fracture	6 (6.4)	9 (10.2)	15 (8.4)
Renal dysfunction	3 (3.2)	0	4 (2.2)
Retinal detachment	3 (3.2)	1 (1.1)	4 (2.2)
Anemia	3 (3.2)	3 (3.4)	15 (8.4)
Hepatic disorder	6 (6.4)	2 (2.3)	13 (7.3)
CPK elevation	0	0	6 (3.4)
Lymphopenia	0	1 (1.1)	4 (2.2)
Neutropenia	1 (1.1)	0	0
Deaths	2 (2.1)	0	2 (1.1)[f]

AE: adverse event; CPK: creatine phosphokinase; E: event; GC-T: glucocorticoid taper; MACE: major adverse cardiovascular event; NMSC: nonmelanoma skin cancer; PBO: placebo; PY: patient-years; TEAE: treatment-emergent adverse event; UPA: upadacitinib; VTE: venous thromboembolism.
There were no cases of vision loss. One event of quadrantanopia was reported in the UPA 15 mg group and resolved after 4 days without interruption of study drug: the event was considered not to be caused by the study drug.
[a]All events are reported as TEAEs, which are defined as any adverse events with an onset date that is on or after the first dose of study drug, and no more than 30 days after the last dose of study drug.
[b]No events of tuberculosis, gastrointestinal perforation, or lymphoma were reported in any of the treatment groups.
[c]Opportunistic infections exclude herpes zoster and tuerculosis.
[d]Includes pulmonary embolism and deep vein thrombosis.
[e]Defined as cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke.
[f]One non-treatment-emergent death also occurred 60 days after the last dose of study drug in the UPA 15 mg group.

new safety signals were identified with UPA. Overall, UPA15 represents a potential new oral targeted therapy for GCA.
Key words: giant cell arteritis, upadacitinib, jak-inhibitor, randomized controlled trial, JAK-inhibitor, glucocorticoids

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O-05

Giant cell arteritis care in England and Wales: first data from the updated National Early Inflammatory Arthritis Audit

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Background. The UK National Early Inflammatory Arthritis Audit (NEIAA) is a mandated rheumatology audit in England and Wales. In April 2023 GCA was added to NEIAA's included diseases.
Objectives. 1. Describe the characteristics of people with GCA enrolled in NEIAA. 2. Report data on care quality: time from referral to rheumatology assessment, time to diagnosis, and duration of symptoms at first review. 3. Compare these data to those for patients with other conditions enrolled into NEIAA (RA, other non-GCA LVV, other vasculitis).
Methods. Data obtained for patients enrolled from April to November 2023. We used descriptive statistics to summarise patient characteristics and care. Primary outcome: time from referral to rheumatology assessment.
Secondary outcomes: 1) time from referral to diagnosis; 2) patient reported duration of symptoms.
Results. 2308 patients were recruited (61 with GCA [2.6%], 2223 RA [96%], 12 non-GCA LVV [0.5%] and 12 Other Vasculitis [0.5%]); (Table I). GCA patients were older, more likely to be female and white, and less likely to be in work. Time to assessment was shorter for GCA (median 6 days, IQR [2,15]) than for RA (18 days, [11,23]) and non-GCA LVV (8 days, [3,22]) but longer than for Other Vasculitis (5 days, [0,16]). GCA patients experienced the shortest diagnostic delays. Other Vasculitis patients reported shorter symptom durations (67% <1 month vs 52% for GCA). 10 GCA patients (17%) reported symptoms for >3 months prior to assessment.
Conclusion. Times to assessment and diagnosis of GCA, LVV and other vasculitis were shorter than for RA, likely indicating appropriate prioritisation. On average these times are still longer than is desirable in the management of a potentially sight-threatening condition.

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Table I.

		Total (N=2,308)	GCA (N=61)	RA (N=2,223)
Age [median (IQR)]		61 (50,72)	76 (69,81)	61 (50,71)
Age Group [n (%)]	**0-60**	1,110 (48.1%)	5 (8.2%)	1,098 (49.4%)
	61-70	567 (24.6%)	15 (24.6%)	542 (24.4%)
	71-80	470 (20.4%)	23 (37.7%)	442 (19.9%)
	>80	161 (7.0%)	18 (29.5%)	141 (6.3%)
Gender [n (%)]	**Female**	1,480 (64.2%)	42 (68.9%)	1,428 (64.3%)
Ethnicity [n (%)]	**White**	1,867 (80.9%)	59 (96.7%)	1,787 (80.4%)
	Black	67 (2.9%)	<5 (<8.2%)*	64 (2.9%)
	Asian	218 (9.4%)	<5 (<8.2%)*	217 (9.8%)
	Mixed	13 (0.6%)	<5 (<8.2%)*	13 (0.6%)
	Other	103 (4.5%)	<5 (<8.2%)*	102 (4.6%)
Patients in paid work >20h/week [n (%)]		990 (44.5%)	12 (19.7%)	969 (45.3%)
Time from referral to assessment, days [median (IQR)]		18 (10,31)	6 (2,15)	18 (11,32)
Time from referral to assessment, days [mean (sd)]		28 (34)	14 (19)	28 (34)
Time from referral to diagnosis, days [median (IQR)]		26 (15,46)	7 (1,21)	27 (15,47)
Time from referral to diagnosis, days [mean (sd)]		35 (114)	16 (33)	36 (116)
Duration of symptoms prior to assessment	**<1 month**	211 (9.2%)	31 (51.7%)	171 (7.7%)
	1-3 months	723 (31.5%)	19 (31.7%)	699 (31.6%)
	>3 months	1,361 (59.3%)	10 (16.7%)	1,341 (60.7%)

O-06

Granulocyte-macrophage colony-stimulating factor drives interleukin-6 production by macrophages in polymyalgia rheumatica

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Background. Polymyalgia rheumatica (PMR) is an inflammatory rheumatic disease featured by subacromial-subdeltoid (SASD) bursitis (1). Glucocorticoid-sparing therapies are urgently needed, necessitating a better understanding of PMR immunopathogenesis. Previous studies (2-4) suggest monocytes/macrophages might be crucial in PMR, but their phenotype and functions have not been assessed.
Objective. We characterized monocytes/macrophages in PMR and identified tissue factors that drive monocytes towards a pro-inflammatory phenotype.
Methods. Monocytes were investigated by flow cytometry in peripheral blood (PMR, n=22; healthy controls, n=20) and SASD bursa fluid (PMR, n=9). Macrophages in SASD bursa tissue were assessed by immunohistochemistry (PMR, n=12; controls, n=10) and immunofluorescence (PMR, n=3). We assessed the effect of cytokines present in PMR-affected tissue on macrophage differentiation in vitro (PMR, n=7; healthy controls, n=7).
Results. Monocytes were increased in the blood of PMR patients and activated in bursa fluid. Macrophages dominated in PMR-affected tissue, expressing various cytokines, including IL-6, IL-1β and IL-23. GM-CSF, M-CSF and IFN-γ were increased compared to control tissue. GM-CSF induced expression of IL-6 by in vitro differentiated macrophages, and this effect was boosted in the presence of M-CSF and IFN-γ.
Conclusion. Monocytes are expanded and activated in PMR. Macrophages in tissue produce various pro-inflammatory cytokines, including IL-6. GM-CSF, M-CSF and IFN-γ may drive the pro-inflammatory functions of these macrophages. Macrophages may thus constitute targets of therapy in PMR.

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O-07

Higher baseline FDG musculoskeletal uptake at PET/CT is associated with a higher remission rate in polymyalgia rheumatica: a retrospective 3-year observational study

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Objective. The aim of our study was to evaluate if increased baseline joint and vascular uptake of FDG could predict the clinical outcomes of patients with PMR followed-up for three years.
Methods. Eighty-three patients with PMR were included. At first visit (V0), patients underwent clinical, laboratory assessments and PET/CT; follow-up visits were performed every 6 months with a mean duration of 3 years. Clinical outcomes included the total number of relapses, remission rate, cumulative glucocorticoid (GC) dosage, GC discontinuation, need for methotrexate (MTX), and death. Regression analysis was conducted using as predictors the presence of large vessel vasculitis [LVV], the total joint score [TJS], total vascular score [TVS], baseline clinical and laboratory parameters.
Results. LVV was present in 32 (39%) PMR patients with grade 2 and in 10 (12%) with grade 3. At third year, 35% of PMR patients experienced at least one polymyalgic relapse and 10% showed at least one GCA relapse. The highest remission (92%) rate was achieved at the 24th month.
The baseline TJS of PMR patients was inversely associated with polymyalgic relapses (p=0.04) and with a higher likelihood of achieving remission at 6 months (p=0.04). Baseline TVS in PMR patients was directly but marginally associated with MTX prescription at follow-up (p=0.01).
Conclusion. High PET/CT-detected musculoskeletal inflammation may be a positive prognostic factor in PMR whereas higher TVS values might predict the need for immunosuppressive treatment with the limitation of a potential indication bias for the latter prognostic factor.
Key words: polymyalgia rheumatica, giant cell arteritis, PET/CT, imaging, clinical outcomes

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O-08

Reliability of the OMERACT Giant Cell Arteritis Ultrasonography Score (OGUS): results of a patient-based exercise involving experts and non-experts in vascular ultra-sonography

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Objective. To test the reliability of the OMERACT Giant cell arteritis (GCA) Ultrasonography Score (OGUS) and other composite scores in a patient-based exercise involving experts and non-experts in vascular ultrasonography.

Methods. Six GCA patients were scanned twice (2 rounds separated ≥3 hours) by 12 experts and 12 non-experts. Non-experts received 90 minutes of theoretical and 240 minutes of practical training between rounds 1 and 2. Ultrasonography was conducted on temporal arteries (common superficial, frontal and parietal branches) and axillary arteries bilaterally to calculate the OGUS, the Southend score and the Halo count. Inter- and intra-reader reliability were assessed by intraclass correlation coefficient (ICC).

Results. Mean age of GCA patients was 78±5.1 years, 2 (33.3%) were female, and all were in clinical remission. Expert inter-reader ICC of the OGUS was 0.60 in both rounds, 0.40 in round 1 and 0.51 in round 2 for the Southend score, and 0.45 and 0.52, respectively for the Halo count. Median ICCs for intra-reader reliability were 0.86, 0.73 and 0.65 for the OGUS, Southend score and Halo count, respectively. For non-experts, inter-reader ICCs in round 1 were 0.20 for the OGUS, 0.20 for a normalized Southend score (=score divided by available segments) and 0.35 for a normalized Halo count. After training, inter-reader reliability ICCs improved to 0.52, 0.29 and 0.54, respectively.

Conclusion. Inter-reader reliability was fair to moderate, and intra-reader reliability was good for OGUS, Southend score, and Halo count among experts. Inter-reader reliability of non-experts in vascular ultrasonography improved after the training.

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P-01

A case report of giant cell arteritis diagnosed by temporal artery biopsy

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Background. Giant cell arteritis (GCA) is highly prevalent in elderly patients and usually presents with fever.
Case report. A 71-year-old female patient was admitted to the hospital in October 2021 presenting Blurred vision with intermittent headache and fever for more than 2 months. The right neck blood vessels smell and murmur; Tenderness in the right temporomandibular joint (+). Leukocytes 9.8 10^9/L, hemoglobin 90 g/L, platelets 491 10^9/L, erythrocyte sedimentation rate 104.0mm /h, globulin 47.90 g/L↑, RF normal, hypersensitive C-reactive protein 15.85 mg/L↑, immunofixed protein electrophoresis no abnormalities, urine routine normal, ANA, ENA and ANCA were negative. Carotid arteriovenous vertebral artery (color ultrasound) showed bilateral carotid arteriosclerosis and multiple plaque formation, combined with stenosis of the right external carotid artery (67%), bilateral temporal artery ultrasound showed no abnormalities, and arteriovenous abnormalities of both upper limbs were not found (Fig. 1). Histopathologic findings after temporal artery biopsy (Fig. 2) is consistent with late changes in giant cell arteritis. Final diagnosis: giant cell arteritis; plasma cell tumor to be ruled out.
Learning points for clinical practice. When elderly patients present fever, headache and blurred vision, physicians should pay close attention to the patient's temporal artery, internal artery, and monitor ESR and CRP, and consider the possibility of GCA.
Key words: GCA, temporal artery biopsy

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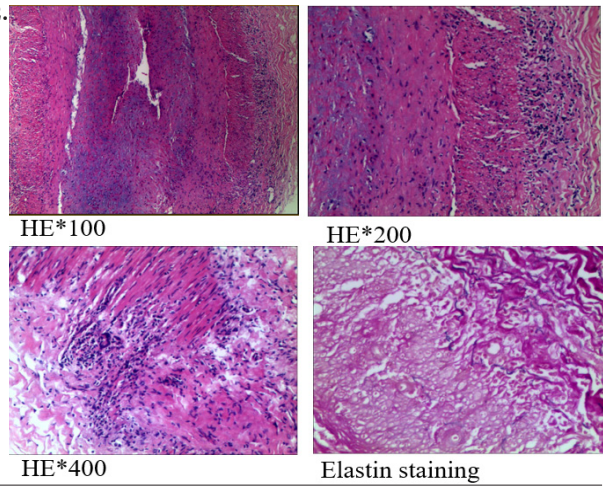
Fig. 1.

Fig. 2.



P-02

A preliminary study on the role of cytokine network imbalance in the pathogenesis of polymyalgia rheumatica

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Background. Polymyalgia rheumatica (PMR) is an inflammatory rheumatic disease that is common in elderly people. The pathogenesis of immune-mediated cytokine network imbalance is not fully understood.
Objective. To explore the potential role of cytokine network imbalance in the pathogenesis of PMR.
Methods. In this study, protein microarray technology was employed to screen differentially expressed cytokine profiles in the serum of patients with polymyalgia rheumatica (PMR). Enzyme-linked immunosorbent assay (ELISA) was used to verify and preliminarily explore the potential role of cytokine network imbalance in the pathogenesis of PMR.
Results. Our results showed that a total of 5 cytokines namely interleukin- 6(IL-6), Interleukin-4(IL-4), Interleukin-13 (IL-13), growth-regulated oncogene (GRO), and Vascular Endothelial Growth Factor (VEGF) were identified by protein chip of 8 newly diagnosed PMR patients before and after treatment. The results of ELISA indicated that the serum levels of IL-6, IL-13, GRO, and VEGF in 20 PMR patients decreased significantly after the remission. The serum levels of IL-6, GRO, and VEGF in the 20 PMR patients before the treatment were significantly higher as compared to those in the 12 healthy controls, but not significantly different from those in the active rheumatoid arthritis (12 patients). There was a positive correlation between the expression of GRO and VEGF. Correlation analysis indicated that the serum levels of IL-6, IL-4, IL-13, GRO, and VEGF were not significantly correlated with clinical activity parameters in 20 PMR patients.
Conclusion. In conclusion, the various cytokines IL-6, IL-4, IL-13, GRO, and VEGF were found involved in the imbalance of the PMR cytokine network. The alteration in the cytokine network composed of pro-inflammatory factors, anti-inflammatory factors, chemokines, and growth factors was involved in the pathogenesis of PMR.

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P-03

Aging in newly diagnosed patients with polymyalgia rheumatica

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Background. Polymyalgia Rheumatica (PMR) is the most common inflammatory rheumatic disease in older adults, and aging has been postulated to be central to its pathophysiology.
Objective. To assess the prevalence of the physical phenotypes of aging in those newly diagnosed with PMR.
Methods. Patients were recruited over a 12-month period from a multicentre fast track PMR clinic. Frailty status was assessed using the SHARE-FI tool. Cognitive status was assessed using the Montreal cognitive assessment tool (MOCA), with a cut-off of <26 used to define cognitive impairment. Bone mineral density was assessed using DEXA, whilst sarcopenia was assessed using the SARC-F questionnaire. Mood, anxiety, fatigue and pain were assessed using the PHQ-9, GAD-7, FACIT-F and pVAS screening tools respectively. Disease activity was assessed using the PMR-activity score (PMR-AS).
Results. 79 newly diagnosed patients (48% (n=38) female) were recruited. At baseline, 40.5% (n=32) patients were frail, 27.8% (n=22) were pre-frail, and 31.6% (n=25) were robust. Increased frailty status was associated with a significant difference in median FACIT-F (p=0.00), pVAS (0.028), PHQ-9 (0.00) and GAD-7 (0.014) scores. 74.4% of patients had evidence of cognitive impairment at baseline. There was no significant difference between age, sex, BMI or disease activity across the different frailty or cognitive categories. 23%(n=18) had evidence of sarcopenia. 43 patients had baseline DEXA scans, with 21.4% (n=9) demonstrating osteoporosis, and a further 30% (n=13) osteopenia (Table I).

Table I. Breakdown of each aging physical phenotype in those newly diagnosed with PMR.

Assessment tool (Characteristic)	Category	N (%)
MOCA (cognition)	Normal	20 (25.6)
	Mild	53 (67.9)
	Moderate	4 (5.1)
	Severe	1 (1.3)
SHARE-FI (frailty)	Robust	25 (31.6)
	Pre-frail	22 (27.8)
	Frail	32 (40.5)
DEXA (BMD)	Normal BMD	21 (49)
	Osteopenia	13 (30)
	Osteoporosis	9 (21)
SARC-F (sarcopenia)	Normal	61 (77)
	Sarcopenia	18 (23)
Urinary Continence	Continent	59 (75)
	Incontinent	20 (25)
Polypharmacy	<5 regular medications	43 (54)
	≥5 regular medications	36 (46)

Conclusion. Our study provides preliminary evidence for an accelerated physical aging phenotype in PMR. Further studies are needed to explore the role of inflammaging and immunosenescence in the pathophysiology of PMR.

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P-04

Assessment of damage indices in Takayasu's Arteritis

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Background. Damage is the irreversible consequence of disease or its treatment.
Objective. To evaluate 2 indices of damage assessment in Takayasu's arteritis (TAK).
Methods. Patients with TAK enrolled in a combined multicenter, observational cohort. Damage at baseline and last visit were assessed using the Vasculitis Damage Index (VDI) and the Large-Vessel Vasculitis Index of Damage (LVVID).
Results. The study included 350 patients with TAK, mean±SD age 40.3 (13.4) years, 91% female, median (25th, 75th) disease duration 69 (1.6, 274) weeks. 89% had ≥1 follow-up, median (25^{th}, 75^{th}) 4.5 (2.1, 8.0) years. Damage was present at first visit in 83% on VDI, 89% on LVVID with most items captured in the peripheral vascular and cardiac categories (Table I). At last follow-up, damage items were noted in 95% on VDI and 95% on LVVID, mostly in the peripheral vascular and cardiac categories (Table I). New damage was captured in 52% patients on VDI and 53% on LVVID (Table I). Items of disease-related damage captured only on 1 of the indices are in Table II. 19% of items VDI and 23% on LVVID were never applicable to any patient with TAK.
Conclusion. During follow-up of TAK, new damage items, are observed in 50%, including new cardiovascular damage. LVVID captures new damage items in the cardiovascular category more comprehensively than VDI. Many items on both indices are not applicable to TAK. Based on these results, LVVID can be modified and streamlined to more efficiently measure damage in TAK and focus on disease-related damage.

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Table I. Items of damage in patients with Takayasu's arteritis at baseline and last visit.

	VDI			LVVID		
Organ System	Baseline, N=350 N(%)	Follow-up, N=313 N (%)	New items, N=313 N (%)	Baseline, N=350 N(%)	Follow-up, N=313 N (%)	New items N=313 N (%)
Peripheral vascular	292 (83)	304 (97)	55 (18)	245 (70)	280 (89)	83 (46)
Cardiac	148 (42)	190 (61)	58 (18)	155 (44)	186 (59)	63 (20)
Musculoskeletal	22 (6)	45 (14)	26 (8)	21 (6)	43 (14)	27 (9)
Ocular	18 (5)	50 (16)	37 (12)	11 (3)	44 (14)	37 (12)
Ear, Nose, and Throat	3 (1)	4 (1)	0 (0)	2 (0.6)	2 (0.6)	0 (0)
Gastrointestinal	6 (2)	8 (3)	3 (1)	3 (1)	5 (2)	2 (1)
Neuropsychiatric	31 (9)	39 (12)	12 (4)	34 (10)	38 (12)	12 (4)
Endocrine	N/A	N/A	N/A	10 (3)	22 (7)	15 (5)
Hematology/Oncology	N/A	N/A	N/A	7 (2)	15 (5)	8 (3)
Skin	12 (3)	20 (6)	7 (2)	11 (3)	22 (7)	13 (4)
Pulmonary	19 (5)	28 (9)	13 (4)	N/A	N/A	N/A
Renal	6 (2)	10 (3)	6 (2)	N/A	N/A	N/A
Other	29 (8)	55 (18)	34 (11)	95 (27)	129 (41)	70 (22)

VDI=Vasculitis Damage Index; LVVID=Large-Vessel Vasculitis Index of Damage
N=number, %=percent, N/A=not applicable

Table II. Items of disease-related damage captured only on 1 of the damage indices in patients with Takayasu's arteritis at last visit.

	VDI		LVVID	
Organ System	AT LAST VISIT	NEW ITEMS	AT LAST VISIT	NEW ITEMS
Peripheral vascular, N (%)[a]	**304 (97)**	**55 (17)**	**280 (89)**	**83 (46)**
Arterial thrombosis/occlusion	---	---	127 (41)	20 (6)
Renal artery stenosis	---	---	89 (28)	10 (3)
Pulse loss	310 (98)	15 (5)	---	---
Second episode absent pulse in one limb	38 (12)	11 (3)	---	---
Major vessel stenosis	261 (83)	16 (5)	---	---
Aortic aneurysm	---		45 (14)	11 (3)
Angioplasty alone	---		25 (8)	6 (2)
Angioplasty with stent	---		63 (20)	16 (5)
Bypass	---		70 (22)	10 (3)
Other, N (%)	**55 (18)**	**34 (11)**	**129 (41)**	**70 (22)**
Damage requiring surgical intervention	---	---	57 (18)	22 (7)
Ocular	**50 (16)**	**37 (12)**	**44 (14)**	**37 (12)**
Retinal change	21 (7)	16 (5)	---	---

N = number, % = percentage, VDI=Vasculitis Damage Index, LVVID=Large-Vessel Vasculitis Index of Damage, --- =Not applicable (ie item not queried on index)

[a]Cardiovascular category on LVVID

P-05

Assessment of factors affecting pregnancy outcomes in Takayasu Arteritis

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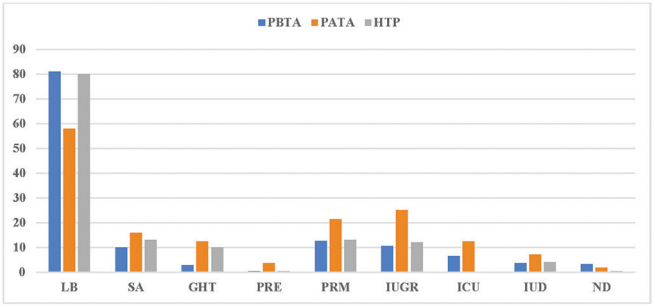
Background. Despite the lack of data about the effect of delivery type on feto-maternal outcomes and disease course in patients with Takayasu Arteritis (TAK), cesarean section is generally preferred in TAK with aortic involvement to prevent increase in intraabdominal pressure during vaginal birth.
Objective. We aimed to assess the feto-maternal outcomes and the choice of delivery type on patients.
Methods. TAK patients having at least one pregnancy history were included. Spontaneous abortion (SA), prematurity (PRM) and ıntrauterine growth retardation (IUGR) were defined according to obstetrics and perinatology guidelines. (1-2). Outcomes of TAK patients were compared with the aged matched healthy Turkish population.
Results. A total of 270 pregnancies of 106 patients were analyzed. Among pregnancy complications, SA rates in PATA were higher than PBTA but not statistically significant. Gestational hypertension rates were statistically significantly higher in PATA. Among fetal complications, PRM rates were higher in PATA but there was no statistical difference. Live birth rates were statistically significantly lower in PATA. IUGR rates were statistically significantly higher in PATA. Birth complication rates were similar between normal vaginal delivery and cesarean sections (Fig. 1).
Conclusion. We found that the incidence of fetal and maternal complication rates of PATA was higher than the HTP (3-4). While LB rate was lower, IUGR and GHT were more common in PATA. The result of our study does not support a greater preference for cesarean section after the diagnosis of TAK. As the rates of birth complications were similar between both modes of delivery. (Table I).
Key words: Takayasu arteritis, pregnancy complications, fetal complications

Table I. The characteristics and outcomes of Takayasu arteritis patients.

Patients, n	106		
İnfertility, n	6		
Age, years, mean± SD	44.8 ± 10.2		
Disease duration, years, median(IQR)	7.5(3-14)		
Gestational age, years, median(IQR)	25(21-30)		
Angiographic class, n(%)			
Type 1 Type 2 Type 3 Type 4 Type 5	46 (46) 10 (10) 2 (2) 4 (4) 38(38)		Not performed
Immunosuppressive treatment during pregnancy, n (%)			
Glucocorticoids csDMARD bDMARD	26 (47.3) 26 (47.3) 7 (9.19		
	PBTA	**PATA**	***p*-value**
Pregnancy, n(%)	214(79.3)	56(20.7)	Not performed
Cesarean delivery, n(%)	53(24.8)	26(46.4)	
Normal vaginal delivery, n(%)	123(57.5)	10(17.9)	
Pregnancy complications, n(%) Spontanous abortion Gestational hypertension Preeclampsia	 23(10.7) 6(2.8) 1(0.5)	 9(16.1) 7(12.5) 2(3.6)	 0.272 **0.003** 1.0
Fetal complications, n(%) Live birth Prematurity Intrauterin growth retardation Intensive care unıt Intrauterin death Neonatal death	 174(81.3) 27(12.6) 33(10.7) 14(6.5) 8(3.7) 7(3.3)	 33(58.9) 12(21.4) 9(16.1) 7(12.5) 4(7.1) 1(1.8)	 **0.001** 0.095 **0.006** 0.138 0.279 1.0
Birth complications, n(%)	Normal vaginal delivery 4(3%)	Cesarean delivery 2(2.5%)	 1.0
	Pregnancies on immunosuppressive	Pregnancies without immunosppressive	
Pregnancy complications, n(%)	16(41)	0(0)	**0.002**
Fetal complications, n(%)	16(41)	5(31.3)	0.498

PBTA: Pregnancy before Takayasu arteritis, PATA: Pregnancy after Takayasu arteritis



LB: Live birth, SA: Spontanous abortion, GHT: Gestational hypertensıon, PRE: Preeclampsia, PRM: Prematurity, IUGR: Intrauterine growth retardation ICU: Intensive care unit, IUD: Intrauterine death, ND: Neonatal death, PBTA:Pregnancy before Takayasu arteritis, PATA:Pregnancy after Takayasu arteritis, HTP : Healthy Turkish population

Fig. 1. Comparison of maternal and fetal pregnancy outcomes with the heathy Turkish population.

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P-06

Biological treatment may be used as first steroid-sparing option in a subgroup of young Takayasu Arteritis patients

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Background. There is no data regarding in which patients biologic immunosuppressive (bIS) treatment should be chosen in Takayasu's arteritis (TAK).
Objective. We aimed to assess the characteristics of TAK patients who needed biologic treatment during follow-up.
Methods. Patients fullfilling the ACR 1990 criteria for TAK and who received conventional ISs (cISs) or bISs were included in this retrospective multicentre study. Data were collected from patient files.

Results. We included 329 patients (F/M: 284/45) in the study. The number of the patients who received bISs was 113 (34%) (89 TNF inhibitors, 24 tocilizumab) and who received only cISs was 216 (66%) during follow-up. Mean age was 43.0±13.5 years, mean follow-up duration was 78.7±65.8 months. Patients who received bISs were younger than patients who received cISs (36.8±11.3 vs 46.2±13.2 years, $p<0.01$) at last visit assessment. The frequency of constitutional symptoms at baseline visit was higher in bISs group (85% vs 66%, $p<0.01$). Baseline erythrocyte sedimentation rate (bISs vs cISs: 66.7±33.5 vs 45.0±29.1 mm/h, $p<0.01$) and CRP (19 (0.3-280) vs 12.5 (0.2-286) mg/L, $p=0.002$) were higher in patients receiving bISs. Number of relapses were higher in patients who needed bISs (Table I).
Conclusion. In this study, TAK patients with biologic treatment need during follow-up had more frequent constitutional symptoms and higher acute phase reactants with a higher relapse rate compared to patients receiving cIS treatment. Our results may suggest that in young TAK patients with prominent acute phase reactants and constitutional symptoms at diagnosis, biologic treatment may be used as first sparing option.

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Table I. The comparison of the characteristics of TAK patients who received cIS and bIS treatments.

	cIS (n=216)	bIS (n=113)	p
Age	46.2±13.2	36.8±11.3	**<0.01**
Gender, F, n(%)	186 (86)	98 (87)	0.80
Age at symptom onset	34.9±12.9	25.4±8.3	**<0.01**
Age at diagnosis	38.0±13.1	27.9±8.6	**<0.01**
Baseline clinical characteristics			
Constitutional symptoms, n(%)	143 (66)	96 (85)	**<0.01**
Claudication, n (%)	156 (72)	71 (63)	0.08
Carotydinia, n (%)	47 (22)	31 (27)	0.28
Cerebrovascular event, n (%)	17 (8)	3 (3)	0.07
Coronary artery disease, n (%)	19 (9)	3 (3)	0.08
Angiographic type 5, n(%)	84 (45)	49 (45)	0.99
Baseline disease activity			
PGA, active disease, n(%)	196 (96)	95 (93)	0.35
Kerr, active disease, n(%)	187 (93)	91 (92)	0.72
ITAS 2010	11.6±4.6	11.3±6.3	0.39
Laboratory			
ESR, baseline, mm/h	45.0±29.1	66.7±33.5	**<0.01**
CRP, baseline, mg/L	12.5 (0.2-286)	19 (0.3-280)	**0.002**
Follow-up duration, months	73.0±58.3	73.2±53.2	0.97
Number of relapses	0 (0-3)	1 (0-5)	**<0.01**

P-07

Carotidynia is the main predictor of future relapses in a large early inception cohort in Takayasu Arteritis

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Background. There is only retrospective and limited data for the long-term prognosis of Takayasu's Arteritis (TAK).
Objective. We aimed to present the results of a Takayasu Inception Cohort settled for long-term, prospective follow-up of only newly-diagnosed patients with TAK.
Methods. Patients fullfilling the ACR 1990 criteria for TAK and diagnosed in the last 12 months were included. Data were recorded in an electronic database of an international "Takayasu's Arteritis Registry". Data is compared with an historical Turkish cohort previously published.
Results. The study included 215 patients with TAK (age: 38.4±13.3 years, F/M: 181/34). The follow-up duration was median 46 (2-124) months. Compared to our retrospective cohort limb claudication (66.4% vs 48%) was more frequent, pulselessness (36 % vs 88%) was less in the inception cohort. Carotidynia was present only in the inception cohort. Mucocutaneous symptoms seem to be a feature of newly-diagnosed disease (17% vs 8.8%). 126(59%) patients had follow-up data. Remission was observed in 79%, at least one relapse was observed in 26% of the patients. Carotidynia was more frequent in patients with relapse (32% vs 10%, $p=0.015$). Also, carotidynia was associated with relapse in logistic regression analysis (OR (95% CI): 4.13(1.26-13.52), $p=0.019$).
Conclusion. Our results suggest that, in an inception cohort, signs and symptoms of 'systemic inflammation' is more prominent in newly diagnosed TAK patients. Whereas vascular extent and damage accumulates during the disease course. In spite of IS treatments 26% of patients relapse within 4 years after diagnosis. Carotidynia was found the main predictor factor for future relapses.

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P-09

Characteristics associated with long-term glucocorticoid use in patients with new onset polymyalgia rheumatica

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Background. Early recognition of PMR patients requiring GC>1year may identify candidates for GC sparing therapies.
Objective. Identify characteristics at 6 months in new-onset PMR associated with GC >1year
Methods. PMR patients were identified from Medicare claims with no PMR/GCA history, age ≥50years, ≥1 inpatient/≥2 outpatient PMR claims ≥30 – <365 days apart, initiating GC (prednisone-equivalent) 7.5–25mg/day ≤30 days after first-inpatient or first-outpatient to ≤30 days after second-outpatient code, had GC ≥200mg in first ≤30 days, ≥4 months continuous GC and enrollment ≥1year prior to first diagnosis. Exclusions: seropositive RA/other rheumatic disease/multiple sclerosis/organ transplant, malignancy treatment or conventional synthetic immunomodulators(csIM)/IL-6Ri, ≤1 year prior to first-inpatient)/second-outpatient code-6months after GC initiation (baseline) (GC-cohort). Patients initiating csIM ≤6 months after GC, meeting other criteria were analyzed separately (csIM-cohort). Characteristics at 6 months between patients on-GC vs. off-GC @1 year were compared.
Results. GC/csIM-cohorts included 4,748/318 patients, respectively. In csIM-cohort, Methotrexate (MTX) use was [200/318(62.9%)]. 3,038(64.0%)/183(57.5%)/111(55%) patients in GC-cohort/csIM-cohort/csIM-cohort MTX initiators were on-GC@1year, respectively. At 6 months in both cohorts, patients on-GC vs. off-GC@1 year had significantly higher GC use and significantly more were on GC ≥5mg (Table I).
In csIM-cohort, initial GC dose and more recent GC initiation were also associated with GC use@1year (Tables I-II). Adding csIM-cohort MTX initiators to GC-cohort, found MTX use was significantly associated with GC@1 year: fewer patients on-GC vs. off-GC@1year received MTX within 6 months [111/3149(3.5%) vs 89/1799(4.9%); $p=0.015$].
Conclusion. Evaluation of GC dose at 6 months may be useful to identify patients who may benefit from GC sparing therapy.
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Table I. Key demographic and clinical characteristics at 6 months by GC status at 1 year.

Characteristics[a]	Overall GC cohort (N=4748)	Discontinued GC at 1 year			Overall csIM[c] cohort (N = 318)	Discontinued GC at 1 year		
		Yes (N=1710)	No (N=3038)	p-value[b]		Yes (N = 135)	No (N = 183)	p-value[b]
Key demographics								
Age, years, Mean (SD)	77.5 (7.1)	77.3 (7.0)	77.6 (7.2)	0.138	75.9 (6.9)	74.9 (7.1)	76.7 (6.7)	0.013
Gender Female, n (%)	3081 (64.9)	1090 (63.7)	1991 (65.5)	0.214	210 (66.0)	88 (65.2)	122 (66.7)	0.783
Race, n (%)				0.017				0.394
White	4346 (91.5)	1550 (90.6)	2796 (92.0)		290 (91.2)	121 (89.6)	169 (92.3)	
Black	145 (3.1)	47 (2.7)	98 (3.2)		11 (3.5)	<11[d]	<11[d]	
Other	257 (5.4)	113 (6.6)	144 (4.7)		17 (5.3)	<11[d]	<11[d]	
Calendar year at GC initiation, n (%)				0.454				0.014
2016	451 (9.5)	150 (8.8)	301 (9.9)		23 (7.2)	<11[d]	Redacted[d]	
2017	1400 (29.5)	495 (28.9)	905 (29.8)		103 (32.4)	Redacted[d]	70 (38.3)	
2018	1584 (33.4)	577 (33.7)	1007 (33.1)		96 (30.2)	42 (31.1)	54 (29.5)	
2019	1313 (27.7)	488 (28.5)	825 (27.2)		96 (30.2)	52 (38.5)	44 (24.0)	
Clinical								
Initial GC dose, mg/day Mean (SD)	14.8 (4.4)	14.7 (4.3)	14.8 (4.4)	0.362	15.3 (4.4)	14.7 (4.4)	15.7 (4.4)	0.026
Mean daily GC dose from month 5 to 7, mg/day,								
Mean (SD)	10.4 (6.0)	9.9 (6.2)	10.7 (5.8)	<0.001	12.8 (8.4)	12.4 (8.8)	13.0 (8.2)	0.169
≥5 mg/day, n (%)	4031 (84.9)	1382 (80.8)	2649 (87.2)	<0.001	292 (91.8)	120 (88.9)	172 (94.0)	0.101
≥7.5 mg/day, n (%)	3214 (67.7)	1062 (62.1)	2152 (70.8)	<0.001	246 (77.4)	95 (70.4)	151 (82.5)	0.011
GC dose at 6 months, mg/day				<0.001				0.002
Mean (SD)	6.7 (7.4)	5.0 (6.7)	7.7 (7.6)		8.3 (8.9)	7.3 (10.0)	9.1 (7.9)	
Median (IQR)	5.0 (0.0, 10.0)	3.8 (0.0, 8.0)	6.3 (1.6, 10.0)		7.5 (0.0, 12.5)	5.0 (0.0, 10.0)	9.0 (3.3, 14.0)	
≥5 mg/day, n (%)	2643 (55.7)	720 (42.1)	1923 (63.3)	<0.001	199 (62.6)	70 (51.9)	129 (70.5)	<0.001
≥7.5 mg/day, n (%)	1956 (41.2)	499 (29.2)	1457 (48.0)	<0.001	163 (51.3)	56 (41.5)	107 (58.5)	0.003
Cumulative GC dose, mg				<0.001				0.002
Mean (SD)	1966.6 (814.5)	1827.2 (801.0)	2045.1 (811.6)		2278.7 (1018.3)	2137.1 (1043.6)	2383.2 (989.2)	
Median (IQR)	1820.0 (1430.0, 2340.0)	1710.0 (1327.8, 2190.0)	1902.1 (1505.3, 2420.0)		2070.6 (1588.8, 2658.3)	1920.0 (1442.5, 2501.5)	2215.0 (1743.0, 2793.5)	
Charlson Comorbidity Index Score (Quan18), Mean (SD)	3.4 (2.4)	3.4 (2.4)	3.4 (2.4)	0.946	3.7 (2.6)	3.5 (2.4)	3.9 (2.7)	0.358
≥1 visit to a rheumatologist with a PMR diagnosis code from GC initiation to 6 months, n (%)	2665 (56.1)	987 (57.7)	1678 (55.2)	0.098	244 (76.7)	103 (76.3)	141 (77.0)	0.875
Outpatient office visits with PMR diagnosis code from GC initiation to 6 months, Mean (SD)	4.2 (2.3)	4.2 (2.2)	4.2 (2.4)	0.637	5.5 (2.8)	5.5 (2.8)	5.4 (2.7)	0.829
Diagnosis for seronegative RA during baseline period, n (%)	831 (17.5)	301 (17.6)	530 (17.4)	0.892	140 (44.0)	62 (45.9)	78 (42.6)	0.558

[a]Region, reason for enrolling in Medicare, baseline diagnosis of asthma, atopic dermatitis COPD, Crohn's disease, psoriasis, ulcerative colitis, baseline HCRU (in patients days, emergency room visits, outpatient office visits, unique prescription medications filled) were also analyzed and were not significant. [b]T-test was used to compare normally distributed continuous variables, Wilcoxon signed-rank test for non-normally distributed continuous variables, chi-squared test for categorical variables and Fisher's exact test to compare categorical variables for small sample size. [c]methotrexate, leflunomide, azathioprine. [d]To protect patient privacy and avoid potential identification of patients, only results with >11 patients are reported and data are redacted when there are >11 patients but such results would allow derivation of the number of patients when <11 are reported. COPD: chronic obstructive pulmonary disease; csIM: conventional synthetic immunomodulators; GC: glucocorticoids; IQR: inter-quartile range; PMR: polymyalgia rheumatica; RA: rheumatoid arthritis; SD: standard deviation.

Table II. Baseline period comorbidities and frailty at 6 months by GC status at 1 year.

Characteristics	Overall GC cohort (N=4748)	Discontinued GC at 1 year			Overall csIM cohort (N = 318)	Discontinued GC at 1 year		
		Yes (N=1710)	No (N=3038)	p-value[b]		Yes (N = 135)	No (N = 183)	p-value[b]
Baseline period comorbidities/events leading to relative contraindication to GC, n (%)								
Diabetes	1404 (29.6)	498 (29.1)	906 (29.8)	0.612	120 (37.7)	48 (35.6)	72 (39.3)	0.491
MACE[a]	166 (3.5)	64 (3.7)	102 (3.4)	0.488	11 (3.5)	<11[c]	<11[c]	0.765
Hypertension	4131 (87.0)	1472 (86.1)	2659 (87.5)	0.156	285 (89.6)	116 (85.9)	169 (92.3)	0.063
Unstable angina	46 (1.0)	14 (0.8)	32 (1.1)	0.428	2 (0.6)	<11[c]	<11[c]	>0.999
Cardiac dysrhythmia	170 (3.6)	59 (3.5)	111 (3.7)	0.717	6 (1.9)	<11[c]	<11[c]	0.701
Heart failure	667 (14.0)	231 (13.5)	436 (14.4)	0.422	44 (13.8)	21 (15.6)	23 (12.6)	0.446
Low bone density (Osteoporosis or osteopenia)	1528 (32.2)	530 (31.0)	998 (32.9)	0.189	107 (33.6)	45 (33.3)	62 (33.9)	0.919
Fracture event	243 (5.1)	86 (5.0)	157 (5.2)	0.835	11 (3.5)	<11[c]	<11[c]	0.537
Osteonecrosis	22 (0.5)	Redacted[c]	<11[c]	0.070	1 (0.3)	<11[c]	<11[c]	0.425
Glaucoma	137 (2.9)	41 (2.4)	96 (3.2)	0.132	8 (2.5)	<11[c]	<11[c]	0.012
Ocular event (cataract surgery)	365 (7.7)	133 (7.8)	232 (7.6)	0.861	26 (8.2)	11 (8.1)	15 (8.2)	0.988
Steroid myopathy	128 (2.7)	42 (2.5)	86 (2.8)	0.444	10 (3.1)	<11[c]	<11[c]	>0.999
Hospitalized infection	648 (13.6)	230 (13.5)	418 (13.8)	0.766	42 (13.2)	25 (18.5)	17 (9.3)	0.016
Psychiatric conditions (psychosis, mania, agitation, mood lability, catatonia, depersonalization, delirium)	3039 (64.0)	1066 (62.3)	1973 (64.9)	0.073	221 (69.5)	95 (70.4)	126 (68.9)	0.771
Frailty at 6 months, n (%)								
Any frailty (CFI 0.20 – 1)	1946 (41.0)	672 (39.3)	1274 (41.9)	0.076	149 (46.9)	62 (45.9)	87 (47.5)	0.775
Mild frailty (CFI 0.20 – 0.29)	1553 (32.7)	533 (31.2)	1020 (33.6)	0.090	126 (39.6)	Redacted[c]	Redacted[c]	0.730
Moderate to severe frailty (CFI 0.30 – 1)	393 (8.3)	139 (8.1)	254 (8.4)	0.781	23 (7.2)	<11[c]	Redacted[c]	0.918

Comorbidities were defined using diagnosis, procedure, or drug codes. Frailty was assessed by a validated claims-based frailty index and defined using a published threshold (Kim DH et al. *J Gerontol A Biol Sci Med Sci* 2018; 73: 980-7; Halawa OA et al., *Ophthalmology* 2023; 130: 646-54). [a]Includes myocardial infarction, stroke, percutaneous coronary intervention, and cardiac artery bypass surgery, [b]Test was used to compare normally distributed continuous variables, Wilcoxon signed-rank test for non-normally distributed continuous variables, chi-squared test for categorical variables and Fisher's exact test to compare categorical variables for small sample size: [c]To protect patient privacy and avoid potential identification of patients, only results with >11 patients are reported, and data are redacted when there are >11 patients but such results would allow derivation of the number of patients when <11 are reported. CFI: daims-based frailty index; csIM: conventional synthetic immunomodulators; GC: glucocorticoids; MACE: major adverse cardiovascular event; PMR: polymyalgia rheumatica, SD: standard deviation.

P-10

Clinical and economic burden of polymyalgia rheumatica in patients with an inadequate response to glucocorticoids or unable to taper glucocorticoids in a real-world setting

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Background. Nearly 50% of PMR patients experience a disease flare upon glucocorticoids (GC) taper/discontinuation.

Objective. To assess clinical and economic outcomes in PMR patients with inadequate response to GC/unable to taper GC.

Methods. A new-onset PMR cohort from Medicare claims included patients aged ≥50 years without GCA history, ≥1 inpatient/≥2 outpatient claims for PMR ≥30 days (D) to <365D, GC initiation 7.5–25mg/D <30D after 1st inpatient or from 1st outpatient code to 30D after 2nd code with GC dose ≥200mg in first ≤30D GC use ≥4 months, enrollment ≥1 year prior to index, defined as latter of meeting diagnosis/GC criteria, and ≥2 years follow-up. Exclusion criteria: Seropositive RA/other rheumatic disease/organ transplant/multiple sclerosis/malignancy treatment or conventional immunomodulatory/IL-6Ri drugs during baseline. Patients were divided into IR and non-IR to GC or GC taper. GC-related AEs (GC-AEs) incidence rates and all-cause healthcare resource utilization (HCRU) costs were reported

Table I. Incidence rates of selected GC-AEs and all-cause HCRU costs in MR patients (n=6054) stratified by IR-GC/GC taper status.

Rate of selected GC-AE*, Events/100 PY (95% CI)†	Months >6–18	Months >18–30	Months >30–42	Months >42–54
IR-group vs. Non-IR-group‡				
Hospitalized infections (1st event)	13.26 (11.89–14.74) vs. 9.24 (8.20–10.37)	8.92 (7.62–10.38) vs. 5.33 (4.26–6.60)	7.12 (5.46–9.13) vs. 5.54 (3.98–7.52)	8.17 (4.35–13.97) vs. 5.78 (2.50–11.40)
Hospitalized infections (all events based on GC use)	25.71 (23.81–27.72) vs. 17.00 (15.59–18.50)	24.74 (22.57–27.05) vs. 12.97 (11.27–14.85)	18.71 (16.01–21.74) vs. 12.72 (10.29–15.55)	19.08 (13.05–26.94) vs. 15.16 (9.39–23.18)
Diabetes mellitus	4.67 (3.71–5.81) vs. 2.90 (2.22–3.72)	3.62 (2.66–4.81) vs. 2.19 (1.39–3.28)	2.60 (1.49–4.22) vs. 2.45 (1.27–4.29)	5.33 (1.96–11.60) vs. 1.08 (0.03–6.01)
MACE	2.38 (1.75–3.17) vs. 1.88 (1.36–2.52)	1.78 (1.16–2.61) vs. 2.16 (1.41–3.17)	3.08 (1.90–4.70) vs. 1.57 (0.72–2.98)	2.33 (0.48–6.81) vs. 0.95 (0.02–5.28)
Fracture (1st event)	4.56 (3.78–5.46) vs. 3.47 (2.85–4.19)	4.72 (3.80–5.80) vs. 3.31 (2.48–4.33)	2.46 (1.54–3.72) vs. 2.16 (1.24–3.51)	1.83 (0.38–5.33) vs. 2.19 (0.45–6.39)
Fracture (all events based on GC use)	8.10 (7.05–9.26) vs. 5.47 (4.68–6.34)	8.73 (7.46–10.15) vs. 5.46 (4.38–6.73)	7.11 (5.49–9.07) vs. 5.36 (3.83–7.29)	4.77 (2.06–9.40) vs. 2.17 (0.45–6.33)
Osteonecrosis	0.15 (0.04–0.39) vs. 0.19 (0.07–0.41)	0.21 (0.06–0.53) vs. 0.25 (0.07–0.64)	0.00 (0.00–0.41) vs. 0.00 (0.00–0.50)	0.00 (0.00–2.21) vs. 0.00 (0.00–2.67)
Steroid myopathy	3.22 (2.56–4.00) vs. 1.00 (0.68–1.43)	1.97 (1.39–2.72) vs. 0.63 (0.30–1.17)	1.24 (0.62–2.21) vs. 0.95 (0.38–1.96)	1.21 (0.15–4.38) vs. 0.74 (0.02–4.10)
Glaucoma	1.56 (1.12–2.12) vs. 1.78 (1.34–2.31)	1.66 (1.14–2.34) vs. 1.12 (0.67–1.78)	0.44 (0.12–1.12) vs. 0.54 (0.15–1.38)	1.19 (0.14–4.31) vs. 0.73 (0.02–4.04)
Heart failure	7.40 (6.30–8.65) vs. 4.31 (3.35–5.18)	5.25 (4.19–6.50) vs. 4.59 (3.52–5.87)	4.98 (3.52–6.83) vs. 3.52 (2.23–5.29)	4.93 (1.98–10.16) vs. 3.23 (0.88–8.28)
Gastrointestinal event	0.66 (0.39–1.06) vs. 0.29 (0.13–0.55)	0.90 (0.52–1.44) vs. 0.25 (0.07–0.65)	0.34 (0.07–0.99) vs. 0.27 (0.03–0.98)	0.00 (0.00–2.27) vs. 0.00 (0.00–2.68)
Ocular event	0.08 (0.01–0.27) vs. 0.00 (0.00–0.12)	0.00 (0.00–0.19) vs. 0.00 (0.00–0.23)	0.00 (0.00–0.40) vs. 0.00 (0.00–0.49)	0.00 (0.00–2.20) vs. 0.00 (0.00–2.66)
Low bone density	7.66 (6.38–9.12) vs. 6.54 (5.45–7.77)	5.21 (4.00–6.68) vs. 4.26 (3.08–5.74)	5.07 (3.37–7.32) vs. 4.40 (2.69–6.79)	2.95 (0.61–8.62) vs. 2.38 (0.29–8.60)
Hypertension	16.13 (11.52–21.96) vs. 12.06 (8.57–16.48)	9.49 (5.71–14.82) vs. 8.67 (4.74–14.55)	7.39 (2.97–15.22) vs. 7.87 (2.89–17.14)	10.68 (1.29–38.58) vs. 0.00 (0.00–26.37)
Cardiac dysrhythmia	1.15 (0.76–1.66) vs. 1.31 (0.93–1.80)	1.07 (0.64–1.66) vs. 0.74 (0.37–1.33)	1.55 (0.83–2.65) vs. 0.73 (0.24–1.71)	0.66 (0.02–3.69) vs. 0.79 (0.02–4.41)
Unstable angina	0.23 (0.09–0.51) vs. 0.23 (0.09–0.47)	0.21 (0.06–0.55) vs. 0.45 (0.18–0.93)	0.80 (0.32–1.64) vs. 0.41 (0.09–1.21)	0.00 (0.00–2.26) vs. 0.00 (0.00–2.75)
Psychiatric conditions (sensitive)	15.44 (12.66–18.66) vs. 13.95 (11.68–16.54)	13.40 (10.48–16.87) vs. 10.82 (8.15–14.08)	6.68 (3.83–10.69) vs. 7.91 (4.83–12.22)	6.23 (1.29–18.21) vs. 2.07 (0.05–11.54)
Psychiatric conditions (specific)	8.48 (6.47–10.92) vs. 6.14 (4.67–7.92)	6.46 (4.53–8.94) vs. 5.07 (3.31–7.42)	4.45 (2.30–7.77) vs. 4.33 (2.16–7.74)	3.94 (0.48–14.22) vs. 4.12 (0.50–14.90)
All-cause HCRU cost§, $ mean (SD) [Median (IQR)]	25504 (52647) [6821 (1652–27691)] vs. 20723 (47913) [4751 (1325–19147)]	25259 (57124) [5905 (1445–24951)] vs. 19,208 (39,698) [3716 (1006–17518)]	21818 (56792) [3792 (1075–20105)] vs. 17649 (40628) [3093 (780–15223)]	22179 (72516) [2455 (707–11880)] vs. 15718 (40961) [1808 (462–9722)]
All-cause HCRU cost§, $ Mean difference (95% CI)	4781 (2384–7179)	6051 (3179– 8922)	4168 (264–8073)	6461 (-1291–14213)

*For GC-AEs incidence rate, only the first event was counted except hospitalized infections and fractures.
†Exact Poisson methods were used to estimate event rates/100 PY.
‡IR to GC/GC taper was defined as presumed flare (GC dose ≥7.5 mg/D at 6 months or GC use >12 months) or relapse (GC used after discontinuation [>60D gap]).
§Mean costs associated with inpatient hospitalization, outpatient visits, emergency room visits and prescriptions per person in each 6-month period; costs were adjusted to 2020 US dollar using Cost Price Index.
AE: adverse event; CI: confidence interval; D: day; GC: glucocorticoid, HCRU: healthcare resource utilization; IQR: interquartile range; IR: inadequate responder; MACE: major adverse cardiac event; PMR: polymyalgia rheumatica; PY: patient years; SD: standard deviation.

Table II. Incidence rates of GC-AEs and costs by steroid dose quartile among IR-GC/GC taper in the 6-18 months interval after the index date.

Rates of selected GC-AEs*, Events/100 PY (95% CI)†	Overall	Dose quartiles (Minimum:maximum cumulative GC dose)			
		Quartile 1 (390:2305)	Quartile 2 (2305:3378)	Quartile 3 (3380:4743)	Quartile 4 (4747:29000)
Hospitalized infections	13.26 (11.89–14.74)	12.12 (6.45–20.72)	12.62 (9.48–16.47)	12.20 (9.89–14.88)	14.26 (12.23–16.52)
Diabetes mellitus	4.67 (3.71–5.81)	5.60 (1.52–14.33)	3.73 (1.86–6.68)	3.83 (2.37–5.86)	5.49 (4.01–7.35)
MACE	2.38 (1.75–3.17)	6.12 (1.99–14.29)	2.53 (1.09–4.99)	2.17 (1.15–3.71)	2.15 (1.33–3.29)
Fracture	4.56 (3.78–5.46)	2.80 (0.58–8.17)	3.51 (1.97–5.80)	3.99 (2.73–5.64)	5.42 (4.21–6.86)
Osteonecrosis	0.15 (0.04–0.39)	0.00 (0.00–3.44)	0.00 (0.00–0.86)	0.25 (0.03–0.90)	0.16 (0.02–0.56)
Steroid myopathy	3.22 (2.56–4.00)	0.94 (0.02–5.25)	2.38 (1.14–4.37)	2.01 (1.15–3.27)	4.50 (3.39–5.85)
Glaucoma	1.56 (1.12–2.12)	0.93 (0.02–5.19)	1.40 (0.51–3.04)	1.61 (0.86–2.76)	1.63 (1.01–2.50)
Heart failure	7.40 (6.30–8.65)	7.02 (2.58–15.27)	7.12 (4.65–10.43)	7.16 (5.29–9.46)	7.71 (6.09–9.62)
Gastrointestinal event	0.66 (0.39–1.06)	0.00 (0.00–3.53)	0.97 (0.26–2.47)	0.63 (0.20–1.47)	0.64 (0.28–1.26)
Ocular event	0.08 (0.01–0.27)	0.00 (0.00–3.44)	0.23 (0.01–1.29)	0.00 (0.00–0.46)	0.08 (0.00–0.43)
Low bone density	7.66 (6.38–9.12)	10.21 (4.10–21.03)	5.12 (2.80–8.59)	5.70 (3.82–8.19)	9.55 (7.53–11.96)
Hypertension	16.13 (11.52–21.96)	9.92 (0.25–55.27)	20.76 (9.96–38.18)	12.29 (5.89–22.60)	17.52 (10.55–27.37)
Cardiac dysrhythmia	1.15 (0.76– 1.66)	2.06 (0.25–7.45)	1.49 (0.55–3.23)	1.08 (0.46–2.12)	1.00 (0.52–1.75)
Unstable angina	0.23 (0.09–0.51)	0.00 (0.00–3.52)	0.24 (0.01–1.33)	0.25 (0.03–0.92)	0.24 (0.05–0.70)
Psychiatric conditions (sensitive)	15.44 (12.66–18.66)	14.20 (3.87–36.36)	16.47 (10.06–25.44)	15.11 (10.46–21.12)	15.40 (11.39–20.35)
Psychiatric conditions (specific)	8.48 (6.47–10.92)	3.55 (0.09–19.78)	8.94 (4.46–15.99)	11.10 (7.18–16.38)	6.95 (4.41–10.43)
All-cause HCRU cost‡, $ mean (SD)	25504 (52647)	30765 (70279)	21835 (69436)	22146 (44978)	29306 (44941)
Median (IQR)	6821 (1652–27691)	4122 (1181–25316)	3760 (1045–16944)	4605 (1204–21230)	11811 (2999–37168)

*For GC-AEs incidence rate, only the first event was counted except hospitalized infections and fractures.
†Exact Poisson methods were used to estimate event rates/100 PY.
‡Sum of costs associated with inpatient hospitalization, outpatient visits, emergency room visits and prescriptions; costs were adjusted to 2020 US dollar using Cost Price Index.
AE: adverse event; CI: confidence interval; GC: glucocorticoid; HCRU: healthcare resource utilization; IQR: interquartile range; IR: inadequate responder; MACE: major adverse cardiac event; PY: patient years; SD: standard deviation.

from 6 months after index over 12-months intervals. Subgroup analysis of the IR-group estimated outcomes by cumulative GC dose quartiles.
Results. All-cause HCRU costs and most GC-AEs incidences were higher in IR vs. non-IR group (Table I). In IR-group, all-cause HCRU costs and most GC-AEs decreased over time; there was GC dose-response relationship with most outcomes (Table II).
Conclusion. The long-term impact of GCs is significant even after GC discontinuation which underscores the need for GC-sparing therapies.
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P-11

Clinical profile of visual symptoms in giant cell arteritis - insights from a tertiary center cohort

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Background. Giant cell arthritis (GCA) is the most common form of systemic vasculitis in adults. It typically affects the extracranial branches of the carotid artery, and visual symptoms are one of its most feared complications.
Objective. To evaluate visual symptoms prevalence and clinical correlations in a cohort of GCA patients.
Methods. Cross-sectional single-center study including patients followed in our University Hospital fulfilling the 1990 ACR and/or 2022 ACR/EULAR GCA Classification Criteria. Demographic features and clinical and serological characteristics were compared between the patients with and without visual symptoms, using the Chi-squared and Mann-Whitney U-test. A p-value <0.05 was considered statistically significant.
Results. 37 GCA patients were included; 23 (62.2) were female, and the median age at diagnosis was 72 (IQR 10.5) years. Visual symptoms were registered in 32.4% (n=12), including diplopia (n=5; 41.6%), and unilateral (n=4; 33.3%) and bilateral (n=5; 41.6%) amaurosis. The GCA-visual symptoms group was associated with male sex (66.7% vs 24.0%, p=0.027), age ≥70 years (100.0% vs 56%, p=0.007) and higher BVAS at baseline (6.5 [IQR 6.8] vs 2.0 [IQR 2.0]). These group also presented a lower prevalence of Polymyalgia rheumatica (8.3% vs 52%, p=0.013) and a lower prevalence of relapse (8.3% vs 56%, p=0.015) (Table I).
Conclusion. In our cohort of GCA patients, visual symptoms are significantly associated with the male sex, older age (≥70 years) and higher disease activity at baseline. Additionally, these patients showed a lower prevalence of Polymyalgia rheumatica and experienced fewer relapses, suggesting a distinct clinical profile within GCA.

Table. I. Frequency of GCA-associated clinical manifestations, comorbidies, and serological characteristics in patients with and without visual symptoms.

	Whole Cohort n = 37 (100%)	Visual Symptoms n = 12 (32.4%)	Without Visual Symptoms n = 25 (67.6%)	*p*-value
Age at GCA diagnosis median (IQR)	72.0 (10.5)	79.5 (12.8)	70.0 (10.5)	**<0.001**
Age ≥ 70 years at GCA diagnosis [n (%)]	26.0 (70.3)	12 (100.0)	14.0 (56.0)	**0.007**
Male [n (%)]	14.0 (37.8)	8.0 (66.7)	6.0 (24.0)	**0.027**
Clinical features [n (%)]				
- Constitutional symptoms	22 (59.5)	6 (50.0)	16 (64.0)	0.265
* Fever	6 (16.2)	1 (8.3)	5 (20.0)	0.640
* Weight loss	15 (40.0)	4 (33.3)	11 (44.4)	0.350
* Asthenia	11 (29.7)	1 (8.3)	10 (40.0)	0.060
* Anorexia	11 (29.7)	4 (33.3)	7 (28.0)	1.000
- Cranial symptoms	33 (89.2)	12 (100.0)	21 (84.0)	0.282
* Headache	30 (81.1)	10 (83.3)	20 (80.0)	1.000
* Scalp sensitivity	6 (16.2)	2 (16.7)	4 (16.0)	1.000
* Jaw claudication	18 (48.6)	7 (58.3)	11 (44.0)	0.414
- Polymyalgia rheumatica	14 (37.8)	1 (8.3)	13 (52.0)	**0.013**
- Abnormalities in the temporal artery	20 (54.1)	8 (66.7)	12 (48.0)	0.343
Other common comorbidities and clinical manifestations [n (%)]				
- Arterial hypertension	26 (70.3)	11 (91.7)	15 (60.0)	0.064
- Dyslipidemia	18 (48.6)	5 (41.7)	13 (52.0)	0.556
- Obesity	1 (2.7)	0 (0)	1 (4.0)	-
- Diabetes mellitus	7 (18.9)	2 (16.7)	5 (20.0)	1.000
Laboratory features [median (IQR)]				
- Hemoglobin (g/dL)	11.6 (1.5)	11.5 (2.6)	11.6 (1.4)	0.860
- Leukocytes (x10^9/L)	9.1 (2.9)	10.2 (2.1)	8.7 (2.3)	0.115
- Platelets (x10^9/L)	316.0 (119.3)	345.0 (128.0)	304.5 (99.3)	0.466
- C-Reactive Protein (mg/dL)	6.8 (8.1)	7.7 (7.0)	6.4 (7.5)	0.905
- Erythrocyte sedimentation rate (mm/h)	71.5 (32.0)	70.0 (24.5)	71.5 (42.3)	0.704
BVAS baseline median (IQR)	3.0 (3.5)	6.5 (6.8)	2.0 (2.0)	**0.002**
Relapse [n (%)]	10.0 (27.0)	1.0 (8.3)	9.0 (36.0)	**0.015**
Prednisolone in mg/day or equivalent induction dose median (IQR)	60.0 (240.0)	500.0 (815.0)	60.0 (20.0)	**<0.001**

IQR: Interquartile range; GCA: Giant Cell Arteritis

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P-12

Clinical utility of serum markers of disease activity in isolated polymyalgia rheumatica

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Background. A significant unmet need in polymyalgia rheumatica (PMR) is the lack of a disease specific serum biomarker for diagnosis, and monitoring of disease activity.
Objective. To evaluate readily available laboratory tests as markers of disease activity in isolated PMR.
Methods. Full blood count, liver function tests, c-reactive protein (CRP), erythrocyte sedimentation rate (ESR), serum fibrinogen, haptoglobins and immunoglobulins were tested on steroid naïve patients recruited from a multicentre fast track clinic, at baseline, subsequent remission, and in the event of disease relapse.
Results. 55 patients (52.7% (n=29) female) were included, with a total of 36 relapses.
Comparing baseline with remission bloods, there was a statistically significant increased mean difference in platelet count (p=0.001), fibrinogen (p<0.0001), ESR (p<0.001), haptoglobin (p<0.0001), alkaline phosphatase (p<0.001), CRP (p<0.0001) and IgG (p<0.001). There was also a significant decreased Hb between baseline (p<0.001) and relapse (p=0.07) versus remission. When comparing relapse to remission bloods, there was a statistically significant increased mean difference in fibrinogen (p<0.0001), ESR (p<0.0001), haptoglobin (p<0.0001) and CRP (p<0.0001) during relapse. A ROC analysis was performed with fibrinogen (1.000), ESR (0.986), Haptoglobin (0.976), CRP (0.986) and alkaline phosphatase (0.854) demonstrating high sensitivity and specificity for the detection of increased disease activity at baseline versus remission (p=0.000) (Tables I-II).
Conclusion. We have demonstrated the clinical utility of using serum fibrinogen, haptoglobin and other readily available serum markers in the diagnosis, and detection of relapse in an Irish isolated PMR cohort. Further studies, incorporating such markers into a diagnostic algorithm are warranted to enhance diagnostic accuracy and detection of relapse in isolated PMR.

Table I. Normally distributed variables tested at baseline, remission and relapse.

Variable	Time point 1	Time point 2	Mean Difference	Post-Hoc P value	Post- Hoc 95% CI
Haemoglobin (g/L)	Baseline	Remission	-0.99	<0.001	(-1.53, -0.45)
	Relapse	Remission	-0.62	0.07	(-1.23,0.05)
	Remission	Relapse	-0.37	0.41	(-1.05, 0.31)
Platelet count (10^9/L)	Baseline	Remission	57.57	0.001	[21.75, 93.39]
	Relapse	Remission	27.4	0.32	[-17.14, 71.94]
	Remission	Relapse	30.17	0.26	[-15.06, 75.40]
Lymphocyte count (10^9/L)	Baseline	Remission	-0.08	0.89	[-0.48, 0.32]
	Relapse	Remission	-0.35	0.21	[-0.85, 0.14]
	Remission	Relapse	0.28	0.40	[-0.23, 0.78]
Haptoglobin (g/L)	Baseline	Remission	1.02	<0.0001	[0.66, 1.39]
	Relapse	Remission	0.73	<0.0001	[0.28, 1.17]
	Remission	Relapse	0.29	0.28	[-0.16, 0.75]
IgG (g/L)	Baseline	Remission	2.03	<0.001	[0.90, 3.18]
	Relapse	Remission	-0.06	0.99	[-1.60, 1.49]
	Remission	Relapse	2.09	0.006	[0.51, 3.66]
IgA (g/L)	Baseline	Remission	0.66	0.10	[-0.01, 1.41]
	Relapse	Remission	-0.55	0.42	[-1.58, 0.48]
	Remission	Relapse	1.21	0.019	[0.16, 2.23]
Albumin (g/L)	Baseline	Remission	-1.64	0.17	[-3.80, 0.52]
	Relapse	Remission	-1.11	0.59	[-3.81, 1.58]
	Remission	Relapse	-0.52	0.90	[-3.28, 2.23]
Alkaline Phosphatase (IU/L)	Baseline	Remission	20.78	<0.001	[9.79, 31.76]
	Relapse	Remission	2.02	0.94	[-11.69, 15.74]
	Remission	Relapse	18.75	0.005	[4.74, 32.77]

Table II. Variables with skewed distribution tested at baseline, remission and relapse.

Variable		Baseline	Relapse	Remission	P-Value
C-reactive protein (g/dL)	Median Min-Max (IQR)	23.00 1.00-121.00 (29.08)	13.04 2.01-66.32 (25.79)	1.85 1.00-9.19 (2.91)	<0.0001
	Mean Rank	92.02	78.81	31.99	
Fibrinogen (g/L)	Median Min-Max (IQR)	4.4 3.50-8.00 (0.84)	4.09 3.60-6.50 (0.90)	3.20 1.63-4.60 (0.55)	<0.0001
	Mean Rank	92.12	79.79	31.46	
Neutrophil: lymphocyte Ratio	Median Min-Max (IQR)	3.2 0.73-15.00 (1.75)	4.85 1.90-13.60 (3.56)	4.05 1.46-22.80 (3.01	0.007
	Mean Rank	52.64	78.33	67.94	
Platelet:Lymphocyte Ratio	Median Min-Max (IQR)	190.00 105.00-2900.00 (109.2)	215.50 93.60-367.14 (146.90)	155.70 63.10-562.00 (115.08)	0.097
	Mean Rank	67.72	79.08	53.92	
Erythrocyte Sedimentation Rate (mm/hr)	Median Min-Max (IQR)	35.00 9.00-120.00 (19.50)	29.50 13.00-57.00 (18.00)	9.50 2.00-35.00 (10.50)	<0.0001
	Mean Rank	92.64	79.21	31.25	
Alanine transferase (IU/L)	Median Min-Max (IQR)	17.00 5.00-47.00 (8.50)	18.00 9.00-190.00 (11.25)	18.00 7.00-45.00 (8.00)	0.69
	Mean Rank	59.93	58.85	65.10	
IgM (g/L)	Median Min-Max (IQR)	0.98 0.28-3.54 (0.56)	0.99 0.39-2.00 (0.25)	0.95 0.32-3.29 (0.61)	0.69
	Mean Rank	57.76	56.47	56.51	

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P-13

Concomitant shoulder osteoarthritis in PMR - Does it matter?

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Background. Polymyalgia rheumatica (PMR) is an inflammatory rheumatic disease of the shoulder and pelvic girdle. In the majority of centres, baseline shoulder radiograph forms part of the initial diagnostic workup.
Objective. To evaluate the prevalence of concomitant shoulder osteoarthritis (OA), and its impact on patient reported outcomes in those with PMR.
Methods. Those with a new diagnosis of PMR, underwent a bilateral shoulder radiograph on their initial consultation, and patient reported outcomes including fatigue using FACIT-F, mood using PHQ-9, anxiety using GAD-7, pain using p-VAS, and overall health related quality of life and disability using the Health Assessment Questionnaire-Disability Index (HAQ-DI) were collected.
Results. A total of 43 patients (48.8% female) were recruited. 29 (67.4%) patients had evidence of degenerative change of their acromioclavicular joints on shoulder radiograph. Notably, no patient had evidence of erosive change, or crystal arthropathy. The mean age in those with degenerative changes was 70.21 (SD 7.87), and 73.29 (SD 7.17) in those without (Table I).
There was a statistically significant higher pVAS (p=0.0049) and HAQ pain score (p=0.0041) in those with degenerative changes. There was also a statistically significant lower FACIT-F score in those with evidence of degenerative change (p=0.0376). There was no statistically significant difference between HAQ-DI or HAQ- global patient scores between the groups.

Table I. Characteristics of those with, and without degenerative change on shoulder X-ray.

Characteristic	Degenerative change on bilateral shoulder X-Ray	Normal bilateral Shoulder X-Ray	p-value
Sex, female, n (%)	14 (48.3)	7 (50)	
Age, mean (SD)	70.21 (7.87)	73.29 (7.17)	0.2236
p-VAS score, mean (SD)	5.10 (2.34)	2.86 (2.28)	0.0049
FACIT-F, mean (SD)	29.97 (11.29)	37.71 (10.61)	0.0376
HAQ-DI, mean (SD)	0.93 (0.53)	0.73 (0.57)	0.2684
HAQ pain score, mean (SD)	62.93 (27.04)	36.07 (27.47)	0.0041
HAQ- global score, mean (SD)	41.55 (25.74)	29.64 (20.80)	0.1395

Conclusion. This study, demonstrates the high prevalence of concomitant acromioclavicular joint OA in those with PMR, and for the first time highlights the impact that this has on patient reported pain and fatigue, which has potential implications for disease management.
Key words: PMR, osteoarthritis, pain, fatigue, patient reported outcomes

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P-14

Cytokine signature of newly diagnosed steroid naive PMR patients

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Background. There is a significant unmet need for a reliable serum biomarker of disease activity, to aid the diagnosis and the identification of relapse in Polymyalgia Rheumatica (PMR).
Objective. To evaluate the levels of 7 serum cytokines in a cohort of newly diagnosed, treatment naive, pure PMR patients.
Methods. Patients with active, untreated pure PMR, were recruited from a Fast Track PMR clinic. The following circulating cytokines were measured using ELISA: MCP-1 (CCL-2), interleukin-6 (IL-6), CXCL9 (MIG), MIP-1 alpha (CCL3), Tumour necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β) and interleukin-17A (IL-17A).
Results. A total of 15 consecutive patients (33% female), with a mean age of 71.71 (SD 5.97) were included. In reference to defined manufacturer parameters, at diagnosis serum MCP-1 levels were elevated in 14/15 (93%), with a median level of 732.5pg/ml (571, 921). Serum IL-6 levels were elevated in 10/15 (67%) with a median level of 11.85 pg/ml (6.05, 31.8). CXCL9 was elevated in 14/14 (100%), with a median level of 2184pg/ml (1180, 3897.5). Levels of MIP-1 alpha were elevated in 4/14 (28.6%) patients, with a median level of 91.1 pg/ml (76.5, 105). IL-1b was elevated in 2/15 (13.3%) with a median level of 0.445pg/ml (0.27, 0.815). TNF-α was elevated in 1/15 (6.7%) with a median level of 15.35pg/ml (12.85, 16.8). Finally, IL-17A was only available in 6 patients, with 5/6 (83%) demonstrating increased levels, with a median IL-17A level of 3.02 (2.89,3.27).
Conclusion. We have demonstrated the potential utility of serum CXCL-9, MCP-1 and IL-17A in the diagnosis of those with pure PMR.

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P-15

Difficulty in squatting and standing up (DISS) score should be not overlooked as an important evaluation index for PMR: data from the EAST PMR study

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Background. Polymyalgia Rheumatica (PMR) is characterized by pain and morning stiffness in the shoulder, neck, and pelvic girdle. These patients usually have difficulties in raising their upper limbs and squatting. PMR activity disease score (PMR-AS) includes EUL (ability to elevate the upper limbs). However, difficulty in squatting and standing up (DISS) score is ignored.
Objective. We aimed to know the role of DISS score in the assessment of PMR during the Efficacy and safety of tofacitinib in patients with Polymyalgia Rheumatica (EAST PMR).
Methods. Patients with newly diagnosed PMR were randomized to tofacitinib (5mg bid) group and Glucocorticoids (Pred 15mg/day, gradually tapered) group. All PMR patients underwent clinical and laboratory examinations at 0, 4, 8, 12, 16, 20, and 24 weeks. DISS score due to hip muscle involvement was scored as 0=no difficulty, 1=can squat and stand with the help of others or upper body strength, 2=can squat with the help of others or upper body strength, but cannot stand, 3=cannot squat and stand. PMR-AS score and DISS score were both calculated.
Results. 35 patients and 32 patients completed the 24 weeks-intervention respectively. The baseline of PMR-AS and DISS scores were comparable in two group. At weeks 12 and 24, all patients in both groups had PMR-AS<10. PMR-AS, CRP, and ESR as well as DISS score were all significantly decreased at weeks 12, and 24 in both groups (Fig. 1).
Conclusion. DISS score is a useful measurement as PMR-AS score in the assessment of therapeutic response in PMR patients.
Key words: polymyalgia rheumatica, PMR-AS score, DISS score, tofacitinib, glucocorticoids

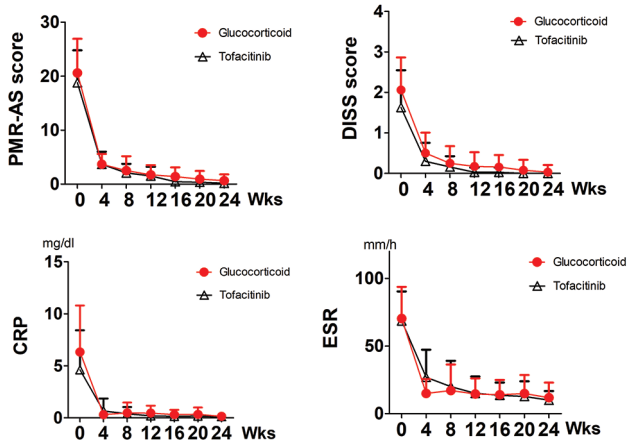

Fig. 1.

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Effectiveness of IL-6 receptor inhibitors versus methotrexate or any conventional immunomodulators in patients with steroid refractory polymyalgia rheumatica

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Background. A retrospective study (based on Medicare data) reported the effectiveness of interleukin-6-receptor inhibitors (IL-6Ri) vs conventional synthetic immunomodulators (csIM) in glucocorticoid (GC)-refractory PMR. Residual differences in direct/propensity-score match prompted adjustment to matching and exclusion criteria.
Objective. This analysis compared IL-6Ri to common csIMs (main cohort) or methotrexate alone (MTX-subgroup); a sensitivity cohort excluded GCA history. Primary outcomes were GC-discontinuation and minimal/no GC at 1 year.
Methods. Adults ≥50 years old with 1-inpatient/2-outpatient PMR Medicare claims (3/29/16–6/30/20) were included. Patients were on ≤25mg GC, started IL-6Ri/csIM as 2nd/3rd line (2L/3L) therapy (index), and had 180 days continuous enrollment prior-index (baseline-period).
Results. The main and sensitivity cohorts had 415 (203 MTX) and 451 matched pairs, respectively. Most baseline covariates were balanced between arms. MTX(2L) and leflunomide (3L) were the common index csIMs. Significantly more IL-6Ri vs csIM/MTX patients discontinued GC or were on minimal/no GC at 1-year (p<0.05). Results were significant in 2L/3L for the main (Table I) and sensitivity cohorts (GC discontinuation:49.7% vs 35%, p=0.004[2L]/45.5% vs 28.4%, p<0.001[3L]; minimal/no GC:55.2% vs 37.2%, p<0.001[2L]/47.4% vs 32.1%, p<0.001[3L]), but only in 2L for the MTX-subgroup (Table II). Mean cumulative GC dose was lower with IL-6Ri vs csIM/MTX (Tables I-II).
Time-to-event analyses favored IL-6Ri over csIM/MTX for GC-discontinuation (HR[95%CI], main cohort:1.28[1.02-1.60], p=0.031; sensitivity cohort:1.30[1.05-1.61], p=0.018; MTX-subgroup:1.29[0.95-1.75], p=0.109) and minimal/no GC at 1-year (HR[95%CI], main cohort:1.28[1.03-1.58], p=0.025; sensitivity cohort:1.29[1.04-1.58], p=0.018; MTX-subgroup: 1.41[1.05-1.89], p=0.022).

Table I. Characteristics and outcomes at 1 year in PMR patients receiving IL-6Ri or csIM as 2L and 3L therapy after propensity score match in the main cohort*

Characteristics	2L			3L		
	IL-6Ri[a] (N = 187)[c]	csIM[b] (N = 187)[c]	SMD[#]	IL-6Ri[a] (N = 228)[c]	csIM[b] (N = 228)[c]	SMD[#]
csIM Use, n (%)			NA			NA
Index methotrexate	0 (0.0%)	162 (86.6%)		0 (0.0%)	41 (18.0%)	
Index leflunomide	0 (0.0%)	19 (10.2%)		0 (0.0%)	162 (71.1%)	
Index azathioprine	0 (0.0%)	<11		0 (0.0%)	25 (11.0%)	
Prior methotrexate	-	-		177 (77.6%)	177 (77.6%)	
Prior leflunomide	-	-		41 (18.0%)	43 (18.9%)	
Prior azathioprine	-	-		< 11	< 11	
Direct Match and/or PS Match Covariate						
Age at index[d], years, Mean (SD)	75.7 (6.8)	76.1 (6.1)	0.06	74.0 (5.9)	74.5 (5.5)	0.09
Sex, Female, n (%)	136 (72.7%)	136 (72.7%)	0.00	162 (71.1%)	174 (76.3%)	0.12
Baseline daily GC dose category (mg), n (%)			0.16			0.06
<2.5	13 (7.0%)	16 (8.6%)		24 (10.5%)	26 (11.4%)	
2.5–<5	24 (12.8%)	25 (13.4%)		54 (23.7%)	54 (23.7%)	
5–<10	65 (34.8%)	72 (38.5%)		109 (47.8%)	111 (48.7%)	
10–<15	51 (27.3%)	44 (23.5%)		30 (13.2%)	26 (11.4%)	
15–<20	19 (10.2%)	14 (7.5%)		<11	<11	
20–25	<11	<11		<11	<11	
>25	<11	Redacted				
Index[d] GC dose category (mg), n (%)			0.10			0.12
<2.5	<11	<11		<11	<11	
2.5–<5	Redacted	Redacted		19 (8.3%)	15 (6.6%)	
5–<10	45 (24.1%)	53 (28.3%)		80 (35.1%)	87 (38.2%)	
10–<15	44 (23.5%)	40 (21.4%)		59 (25.9%)	57 (25.0%)	
15–<20	43 (23.0%)	41 (21.9%)		31 (13.6%)	30 (13.2%)	
20–25	33 (17.6%)	33 (17.6%)		31 (13.6%)	34 (14.9%)	
GCA	26 (13.9%)	26 (13.9%)	0.00	<11	<11	0.00
Race, n (%)[e]			0.12			0.04
White	161 (86.1%)	163 (87.2%)		212 (93.0%)	214 (93.9%)	
Black	<11	12 (6.4%)		<11	<11	
Other	Redacted	12 (6.4%)		<11	<11	
CCI, Mean (SD)[e]	2.4 (2.0)	2.4 (1.8)	0.01	2.5 (2.0)	2.5 (1.8)	0.03
Time from first PMR diagnosis to index date[d], Median (IQR)[e]	295.0 (133.0, 717.5)	358.0 (147.0, 811.0)	0.04	704.0 (278.8, 1,438.5)	644.5 (294.8, 1,333.3)	0.01
Seronegative RA[f], n (%)[e]	59 (31.6%)	55 (29.4%)	0.05	160 (70.2%)	159 (69.7%)	0.01
Outcomes at 1 year			**P-value[g]**			**P-value[g]**
GC discontinuation, n (%)	92 (49.2%)	70 (37.4%)	0.022	103 (45.2%)	66 (28.9%)	<0.001
No or minimal GC, n (%)	102 (54.5%)	77 (41.2%)	0.010	108 (47.4%)	74 (32.5%)	0.001
Cumulative GC dose (mg/per person-week)			0.264			0.107
Mean (SD)	49.1 (37.1)	54.3 (42.1)		46.6 (39.2)	49.7 (33.6)	
Median (IQR)	40.5 (21.2, 64.5)	43.0 (25.6, 71.4)		37.2 (24.5, 60.9)	42.4 (28.0, 63.1)	

GC dose is presented as prednisone equivalents, minimal GC defined as prednisone equivalent dose ≤2 mg/day; [#]adjusted for baseline covariates with SMD >0.1; *IL-6Ri and csIM patients were PS matched for both 2L and 3L on region, calendar year, baseline GC dose, COPD, seronegative RA, and outpatient office visits; for 2L only on Crohn's disease, psoriasis, ulcerative colitis, and baseline inpatient days; and for 3L only on age, index day GC dose category and asthma: Region, original reason eligible for Medicare, calendar year at index, duration of baseline GC therapy, baseline comorbidities (asthma, AD, COPD, Crohn's disease, psoriasis, ulcerative colitis), baseline HCRU (inpatient, outpatient, emergency room visits) were not significantly different between the cohorts.
[a]sarilumab, tocilizumab; [b]methotrexate, leflunomide, azathioprine; [c]To protect patient privacy and avoid potential identification of patients, only results with >11 patients are reported, and data are redacted when there are >11 patients when such results would allow derivation of the number of patients when <11 are reported; [d]Index date for 3L cohort was the start date of IL-6Ri or new csIM after prior csIM. For 2L cohort, index was the start date of IL-6Ri or csIM with no prior use of IL-6Ri or csIM; [e]PS match only; [f]Seronegative RA was allowed, given patientts can meet classification criteria for both PMR and seronegative RA and may represent initial misdiagnosis or use of ICD-10 codes to obtain reimbursement for off-label use in PMR; [g]Pearson Chi Square test for categorical, and Wilcoxon rank sum test for continuous variables.
2L: second line; 3L: third line; AD: atopic dermatitis; CCI: Charlson comorbidity index; COPD: chronic obstructive pulmonary disease; csIM: conventional synthetic immunomodulators; GC: glucocorticoid; GCA: giant cell arteritis; HCRU: healthcare resource utilization; IL-6Ri: interleukin-6 receptor inhibitors; IQR: interquartile range; MTX: methotrexate; NA: not analyzed/applicable; PMR: polymyalgia rheumatica; PS: propensity score; RA: rheumatoid arthritis; SD: standard deviation; SMD: standardized mean difference.

Table II. Characteristics and outcomes at 1 year in PMR patients receiving IL-6Ri or MTX as 2L and 3L therapy after propensity score match in the MTX-subgroup analysis*

Characteristics	2L				3L			
	IL-6Ri (N=162)[a]	MTX (N=162)[a]	SMD	P-value[b]	IL-6Ri (N=41)[a]	MTX (N=41)[a]	SMD	P-value[b]
Prior csIM (3L only), n (%)								
Methotrexate	-	-	-	-	32 (78%)	Redacted	-	-
Leflunomide	-	-	-	-	<11	37 (90.2%)	-	-
Azathioprine	-	-	-	-	Redacted	<11	-	-
Direct Match and/or PS Match Covariates								
Recency of prior csIM use (3L only), days								
1–60	-	-	-	-	18 (43.9%)	20 (48.8%)		
61–180	-	-	-	-	<11	<11	0.53	0.0705[3]
180+	-	-	-	-	Redacted	Redacted		
Age at index[c], years, mean (SD)	75.8 (7.0)	75.7 (6.0)	0.01	0.835[1]	75.0 (6.8)	74.7 (4.7)	0.06	0.780[1]
Sex, Female, n (%)	118 (72.8%)	118 (72.8%)	0.00	>0.999[3]	26 (63.4%)	31 (75.6%)	0.27	0.230[3]
Baseline daily GC dose category (mg), n (%)			0.11	0.968[2,3]			0.34	0.715[2,3]
<2.5	<11	11 (6.8%)			<11	<11		
2.5–<5	18 (11.1%)	22 (13.6%)			<11	<11		
5–<10	62 (38.3%)	61 (37.7%)			21 (51.2%)	24 (58.5%)		
10–<15	43 (26.5%)	39 (24.1%)			<11	<11		
15–<20	16 (9.9%)	14 (8.6%)			<11	<11		
20–25	<11	<11			<11	<11		
>25	<11	<11						
Index[c] GC dose category (mg), n (%)			0.11	0.964[2]			0.47	0.506[2]
<2.5	<11	<11			<11	<11		
2.5–<5	Redacted	Redacted			<11	<11		
5–<10	41 (25.3%)	47 (29.0%)			15 (36.6%)	13 (31.7%)		
10–<15	38 (23.5%)	36 (22.2%)			13 (31.7%)	<11		
15–<20	35 (21.6%)	36 (22.2%)			<11	<11		
20–25	29 (17.9%)	28 (17.3%)			<11	<11		
GCA	23 (14.2%)	23 (14.2%)	0.00	>0.999[3]	0	0	-	-
Race[d], n (%)			0.14	0.435[2,3]			0.27	0.513[2,3]
White	138 (85.2%)	142 (87.7%)			39 (95.1%)	36 (87.8%)		
Black	<11	<11			<11	<11		
Other	Redacted	<11			<11	<11		
CCI[d], Mean (SD)	2.5 (2.1)	2.3 (1.7)	0.06	0.683[1]	2.5 (2.0)	2.5 (1.5)	0.00	0.480[1]
Time from first PMR diagnosis code to index date[c], Median (IQR) [d]	295.5 (142.5, 682.0)	357.0 (129.8, 793.0)	0.07	0.539[1]	621.0 (268.0, 1256.0)	577.0 (293.0, 1295.0)	0.02	0.982[1]
Seronegative RA[d], n (%)	48 (29.6%)	45 (27.8%)	0.04	0.713[3]	29 (70.7%)	28 (68.3%)	0.05	0.810[3]
Outcomes at 1 year								
GC discontinuation, n (%)	80 (49.4%)	57 (35.2%)	NA	0.010	14 (34.1%)	15 (36.6%)	NA	0.817
No or minimal GC, n (%)	90 (55.6%)	63 (38.9%)	NA	0.003	15 (36.6%)	15 (36.6%)	NA	>0.999
Cumulative GC dose (mg)			NA	0.795			NA	0.613
Mean (SD)	2,757.1 (3,771.0)	4,223.0 (11,555.2)			2,548.5 (3,011.2)	3,978.4 (7,461.4)		
Median (IQR)	1,367.3 (693.8, 3,429.9)	1,470.0 (637.4, 3,530.0)			1,593.0 (729.0, 3,585.0)	1,557.5 (870.0, 4,100.0)		

GC dose is presented as prednisone equivalents, minimal GC defined as prednisone equivalent dose ≤2 mg/day; *This subset was drawn from IL-6Ri and csIM PS matched pairs in the main cohort. Region, Original reason eligible for Medicare, Calendar year at index, Duration of baseline GC therapy, Baseline Comorbidities (asthma, AD, COPD, Crohn's disease, Psoriasis, ulcerative colitis), baseline HCRU (inpatient, outpatient, emergency room visits) were also analyzed and were not significant.
[a]To protect patient privacy and avoid potential identification of patients only results with >11 patients are reported and data are redacted when there are >11 patients when such results would allow derivation of the number of patients when <11 are reported; [b]Due to small sample size; p-value was evaluated in addition to SMD to evaluate if the Cox-model required adjustment for residual cofounders; [c]Index date was the date IL-6Ri or csIM was initiated; [d]PS match only; [1]Wilcoxon rank sum test; [2]Fisher's exact test; [3]Pearson's Chi squared test.
2L: second line; 3L: third line; AD: atopic dermatitis; CCI: Charlson comorbidity index; COPD: chronic obstructive pulmonary disease; csIM: conventional synthetic immunomodulatory therapy; GC: glucocorticoid; GCA: giant cell arteritis; HCRU: healthcare resource utilization; IL-6Ri: interleukin-6 receptor inhibitor; IQR: interquartile range; MTX: methotrexate; NA: not analyzed/applicable; PMR: polymyalgia rheumatica; PS: propensity score; RA: rheumatoid arthritis; SD: standard deviation; SMD: standardized mean difference.

Conclusion. IL-6Ri was a more effective steroid-sparing agent than csIM/MTX, with potential to reduce cumulative GC exposure in PMR.
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Effectiveness of interleukin-6 receptor inhibitors versus conventional synthetic immunomodulatory therapy for treatment of frail patients with polymyalgia rheumatica

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Background. Frailty is associated with aging and inflammation and is more prevalent in PMR vs. the general population. Patients with PMR and frailty may benefit from IL-6 receptor inhibitors (IL-6Ri) as IL-6 is involved in pathogenesis of both. A retrospective study showed a higher proportion of patients on IL-6Ri vs. conventional synthetic immunomodulators (csIM) discontinued GC at 1 year (HR [95%CI]: 1.28 [1.02–1.60]) (main cohort).
Objective. To compare effectiveness of IL-6Ri vs. csIM as second (2L) and third (3L) line treatment for frail patients with PMR.
Methods. A subgroup of PMR patients with frailty from Medicare claims data was evaluated. IL-6Ri and csIM PMR patients were direct and propensity score (PS) matched on multiple factors. PS matched pairs with claims-based frailty index (CFI) ≥ median (0.2) were compared. Outcomes were: primary: time-to-GC discontinuation (>60-day gap), minimal (≤2mg/day of prednisone equivalent)/no GC; secondary: cumulative GC dose.
Results. Of 187 2L/228 3L PS matched pairs, 89 (35 2L/54 3L) had CFI≥0.2. Patient characteristics were generally balanced (Table I). IL-6Ri vs. csIM initiators were significantly more likely to discontinue GC (HR [95%CI] 2.32 [1.35–3.99], p=0.002), achieve minimal/no GC (HR [95%CI] 2.23 [1.34–3.71], p=0.002), and had less GC (mean, 3017.6mg vs. 5764.0mg, p=0.025) (Table II).
Conclusion. Compared with csIM, IL-6Ri had a greater GC-sparing effect in frail patients with PMR. The effect size in frail patients appears to be larger vs. main cohort, suggesting patients with frailty may benefit more from IL-6Ri.
Originally presented at EULAR 2024. Funding by Sanofi and Regeneron.

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Table I. Distribution of patients' characterstics by exposure group for combined cohort.

Characteristics*	IL-6Ri N=89†	csIM N=89†
Age, Years[a,b]	77.2 (6.6)	76.0 (6.5)
Sex, Female[a,b]	70 (78.7%)	69 (77.5%)
Race[b]		
White	81 (91.0%)	77 (86.5%)
Black	<11	<11
Other	<11	<11
Living Region[b]		
West	19 (21.3%)	22 (24.7%)
Midwest	Redacted	Redacted
Northeast	22 (24.7%)	18 (20.2%)
South	35 (39.3%)	33 (37.1%)
Unknown	<11	<11
Reason for Medicare Enrollment[b]		
Age 65 or older	72 (80.9%)	61 (68.5%)
Disabled	17 (19.1%)	28 (31.5%)
Daily GC Dose[#] during Baseline (mg)[c]		
Mean (SD)	8.1 (5.0)	8.4 (5.4)
Median (IQR)	7.4 (4.4, 9.9)	7.3 (4.9, 10.1)
Daily GC Dose[#] Category during Baseline[a,b]		
<2.5 mg	<11	<11
2.5 mg to <5 mg	19 (21.3%)	19 (21.6%)
5 mg to <10 mg	39 (43.8%)	40 (45.5%)
10 mg to <15 mg	11 (12.4%)	17 (19.3%)
15 mg to <20mg	<11	<11
20 mg to 25 mg	<11	<11
Days GC Treatment during Baseline[b]	131.7 (49.3)	139.7 (42.4)
Daily GC Dose[#] on Index Date (mg)[c]		
Mean (SD)	11.1 (6.4)	11.7 (6.3)
Median (IQR)	10.0 (5.0, 15.0)	10.0 (5.8, 16.5)
Daily GC Dose[#] Category on Index Date[a]		
<2.5 mg	<11	<11
2.5 mg to <5 mg	<11	<11
5 mg to <10 mg	30 (33.7%)	32 (36.0%)
10 mg to <15 mg	17 (19.1%)	20 (22.5%)
15 mg to <20 mg	14 (15.7%)	15 (16.9%)
20 mg to 25 mg	18 (20.2%)	17 (19.1%)
Time Since Last csIM Use to Index (3L only), Days[a]		
1–60	16 (29.6%)	Redacted
61–180	11 (20.4%)	<11
180+	27 (50.0%)	30 (55.6%)
Charlson Comorbidity Index[b]	3.4 (2.4)	3.3 (2.3)
Time from First PMR Diagnosis to Index Date (Days)[b]	1233.5 (1184.1)	1249.2 (1196.1)
Comorbidities during Baseline		
Asthma[b]	14 (15.7%)	11 (12.4%)
Atopic dermatitis[b]	<11	<11
Chronic obstructive pulmonary disease[b]	13 (14.6%)	<11
Crohn's disease[b]	<11	<11
Giant cell arteritis[a,b]	<11	<11
Psoriasis[b]	<11	<11
Seronegative rheumatoid arthritis[‡,b]	51 (57.3%)	50 (56.2%)
Number Inpatient Days during Baseline[b]	2.1 (6.2)	0.9 (2.6)
Number Emergency Department Visits during Baseline[b]	0.7 (1.1)	0.8 (1.6)
Number Outpatient Office Visits during Baseline[b]	11.7 (6.0)	11.5 (5.5)
Index Date Calendar Year[b]		
2016	<11	<11
2017	26 (29.2%)	30 (33.7%)
2018	23 (25.8%)	21 (23.6%)
2019	26 (29.2%)	22 (24.7%)
2020	<11	Redacted

Index date was the date IL-6Ri or csIM was initiated; Baseline was defined as the 180 days prior to index date.*Unless otherwise stated continuous variables are reported as Mean (SD) and categorical as n (%); †To protect patient privacy and avoid potential identification of patients only results with >11 patients are reported and data are redacted when there are >11 patients when such results would allow derivation of the number of patients when <11 are reported; ‡Seronegative RA was allowed, given patients can meet classification criteria for both PMR and seronegative RA and may represent initial misdiagnosis or use of ICD-10 codes to obtain reimbursement for off-label use in PMR; §Prednisone equivalents; [a]Covariates used as direct match criterion; [b]Covariates in PS model; [c]Covariates not included in direct match or PS model.
All matches were done in the original analysis and matched pairs were kept both or neither.
csIM: conventional synthetic immunomodulators; GC: glucocorticoids; IL-6Ri: interleukin-6 receptor inhibitors; IQR: interquartile range; PMR: polymyalgia rheumatica; PS: propensity score; RA: rheumatoid arthritis; SD: standard deviation; 3L: 3rd line.

Table II. Outcomes in PS matched IL-6Ri and csIM frail patients with PMR*.

	IL-6Ri (N=89)	csIM (N=89)	P-value[†]	HR (95% CI)	P-value
GC Discontinuation, n (%)	44 (49.4%)	19 (21.3%)	<0.001	2.32 (1.35–3.99)	0.002
GC Discontinuation or Minimal GC Dose, n (%)	47 (52.8%)	23 (25.8%)	<0.001	2.23 (1.34–3.71)	0.002
Cumulative GC Dose[‡] (mg)			0.025		
Mean (SD)	3017.6 (4327.1)	5764.0 (14690.3)			
Median (IQR)	1620.0 (695.0, 3620.0)	2140.0 (1189.3, 5558.0)			

*Patients were direct matched on age, gender, baseline daily GC dose category, index day daily GC category, history of GCA, and recency of csIM (3L only) and then PS matched for both 2L and 3L on region, calendar year, baseline GC dose, COPD, seronegative RA, and outpatient office visits; for 2L only on Crohn's disease, psoriasis, ulcerative colitis and baseline inpatient days; and for 3L only on age, index day GC dose category and asthma; †Pearson's Chi-squared test, Wilcoxon rank sum test; ‡Prednisone equivalents.
CI: confidence interval; COPD: chronic obstructive pulmonary disease; csIM: conventional synthetic immunomodulators; GC: glucocorticoids; GCA: giant cell arteritis; HR: hazard ratio; IL-6Ri: interleukin-6 receptor inhibitors; IQR: interquartile range; PMR: polymyalgia rheumatica; PS: propensity score; RA: rheumatoid arthritis; SD: standard deviation; 2L: 2nd line; 3L: 3rd line.

P-18

Giant cell arteritis and myelodysplastic syndrome: a case of concurrent diagnosis

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Background. Associations of autoimmune and/or inflammatory diseases have been reported in myelodysplastic syndromes (MDS). We present a case of giant cell arteritis (GCA) associated with MDS.
Case. An 80-year-old woman was presented to our hospital with a 3-month history of fatigue, weight loss and headache. On her physical examination, there was scalp tenderness. Laboratory studies showed normocytic anemia and elevated acute phase reactants (Table I). The computed tomography scans of the abdomen and chest showed no abnormalities. A positron emission tomography scan demonstrated fluorodeoxyglucose uptake in the descending thoracic aorta consistent with early atherosclerotic changes. A bone marrow biopsy was performed due to the presence of anemia. Ophthalmologic examination revealed no findings other than cataract. Due to the tenderness of the scalp, a temporal ultrasound was performed, revealing a halo sign suggestive of GCA (Fig. 1). A temporal artery biopsy was performed, and treatment with methylprednisolone (MP) at a dose of 1 mg/kg was initiated. Her clinical symptoms and high serum level of CRP improved after the MP treatment and the patient was discharged. One month later, temporal artery biopsy revealed chronic granulomatous vasculitis and bone marrow biopsy revealed MDS with multilineage dysplasia. The patient was referred to hematology and treatment with azacitidine was initiated.
Learning points for clinical practice. GCA and MDS are diseases commonly seen in the elderly. While they can coexist coincidentally, secondary vasculitis to MDS has also been reported. In patients suspected of vasculitis, before attributing anemia to chronic inflammation, MDS should be considered in the differential diagnosis.
Key words: giant cell arteritis, myelodysplastic syndrome

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Table I. Laboratory data at the time of admission.

Hgb	8 g/dL	AST/ALT	16/14 IU/L
WBC	16,6 x10^3/µL	LDH	202 IU/L
Lymphocyte	0,8 x10^3/µL	Creatinine	0,46 mg/dL
Neutrophil	10,3 x10^3/µL	ANA	neg
MCV	91 fL	ENA	neg
Platelet	220 x10^3/µL	RF	neg
CRP	217 mg/L	Anti-CCP	neg
Sedimentation	89 mm/h	ANCA	neg
Ferritin	1034 ng/L	B12	1472 ng/L

ALT: Alanine aminotransferase; ANA: anti-nuclear antibodies; ANCA: anti-neutrophil cytoplasmic antibodies; AST: Aspartate aminotransferase; CRP: C reactant protein; ENA: antibodies to extractable nuclear antigens; CCP: antibodies to cyclic citrullinated peptide; Hgb: Hemoglobin; LDH: Lactate dehydrogenase; RF: rheumatoid factor; WBC: White blood cell.

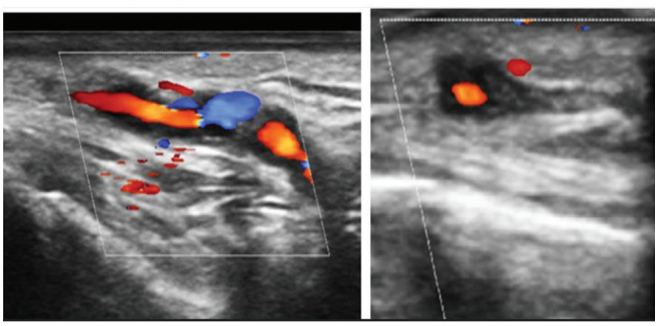

Fig. 1. Halo sign.

P-20

Intimomedial mucoid degeneration of the temporal artery. A rare vascular disease mimicking giant cell arteritis

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Background. The ultrasound "halo" sign is considered virtually pathognomonic of Giant Cell Arteritis in the setting of a correlating clinical presentation. "False halos" have been reported associated with arterial thickening in other conditions including atherosclerosis and lymphoma. In this case arterial wall thickening mimicking a halo on ultrasound was due to a rare vascular condition.
Case report. A 63-year-old female presented with right sided forehead tenderness, associated headache and a tender lump. No significant PMR, ischaemic or visual symptoms. CRP 2mg/L, ESR 30mm/hr. Other indices normal. A painful thickened pulsatile area of right frontal branch artery was noted with tenderness of the artery and scalp adjacent to this. No symptoms on the left side.
Temporal artery ultrasound performed (Fig. 1). The symptomatic area imaged as a focal asymmetrically thickened area of the frontal branch. The artery imaged normally proximal and distal to this, as did the right common superficial, parietal and left sided branches.
Temporal artery biopsy showed mucoid change with eccentric thickening of the artery and aneurysmal change in keeping with intimomedial mucoid degeneration (Fig. 2). This condition causes predisposition to arterial aneurysm formation, and typically presents with acute pain of the artery involved. This lady had developed an aneurysm which was excised at the time of the biopsy, relieving her symptoms.

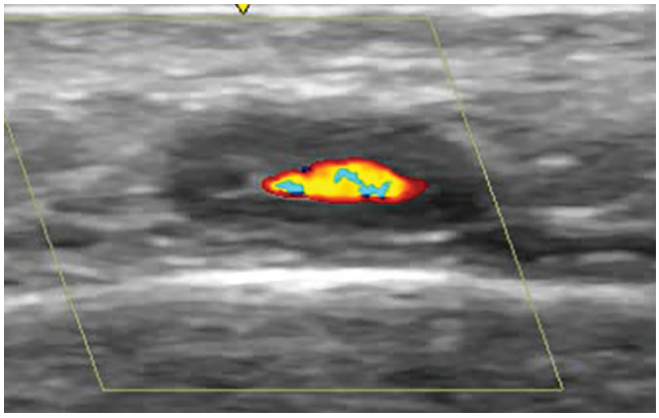

Fig. 1.

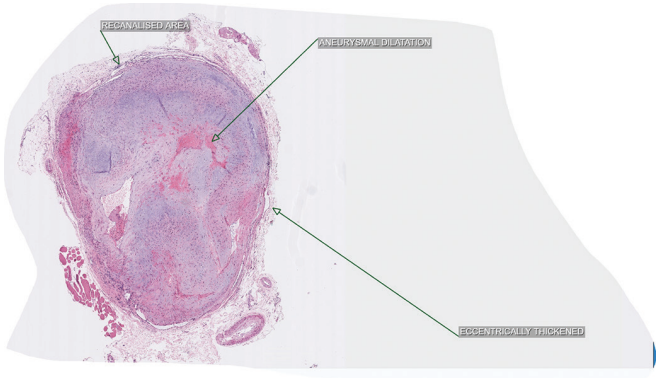

Fig. 2.

Learning points. This is the first reported case of intimomedial mucoid degeneration affecting the temporal artery and highlights the need for additional investigations and tissue diagnosis in the setting of atypical clinical and imaging findings.
Key words: GCA, ultrasound, false halo

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P-21

One-year longitudinal outcomes of an Irish pure PMR cohort

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Background. The longitudinal outcomes of those with pure Polymyalgia Rheumatica (PMR) are poorly understood, owing largely to the fact that in the majority of countries PMR is diagnosed and managed in primary care.
Objective. To establish for the first time the longitudinal outcomes of an Irish population with pure PMR.
Methods. Patients were recruited to this multicentre longitudinal cohort study from a newly established fast track PMR clinic, where those fulfilling the 2012 EULAR/ACR Provisional PMR classification criteria were invited to participate. Those with symptoms or imaging confirmation of cGCA/LV-GCA were excluded.
Results. 65 patients (50.8% females), with a mean age at diagnosis of 70.44 (SD 7.15) were recruited. Four patients, including one death, and three subsequent cancer diagnoses were excluded from the final analysis. At one year, 23/61 (27.7%) patients remained on glucocorticoid therapy, with a mean dose of 3.282 (SD 2.038). A total of 13/61 (21.3%) were commenced on steroid sparing therapy – tocilizumab. There was a total of 33 disease relapses, in 20/61 patients (32.7%). 12 patients experienced 2 or more relapses. The mean time to disease relapse was 6.55 (3.69) months, with a mean steroid dose at the time of relapse of 6.652 (SD 4.168). Increased duration of early morning stiffness (p=0.04), and higher PMR-AS (p=0.05) at disease presentation were significantly associated with disease relapse. Adverse events at one year are displayed in Table I.
Conclusion. We report the longitudinal outcomes of an Irish PMR population, highlighting the disease burden and emphasising the need for specialist input.
Key words: PMR, isolated PMR, outcomes, adverse events, relapse, glucocorticoid, tocilizumab

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Table I. Adverse events experienced over one year longitudinal follow-up.

Adverse Event	Number (%)
Lower Respiratory Tract Infection (LRTI)	11 (18)
LRTI requiring hospitalisation	4 (6.5)
Mood Disturbance	10 (16.4)
Sleep Disturbance	2 (3.3)
Osteoporotic fractures	2 (3.3)
Achilles Tendon rupture	1 (1.6)
Shingles	1 (1.6)
Diabetes Mellitus	2 (3.3)
Ischaemic Cardiac Event	2 (3.3)
Transient Ischaemic Attack	1 (1.6)

P-22

Outcomes of vascular interventions in Takayasu arteritis patients: a tertiary centre experience from Turkey

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Background. Stenosis, occlusion, dilatation and/or aneurysm can develop in the vessel wall of patients with TAK due to inflammation. Vascular interventions may be required in the presence of symptomatic signs of arterial stenosis and severe aneurysm.
Objective. This study aimed to evaluate outcomes and complications of vascular interventions in patients with TAK.
Methods. Patients who underwent vascular interventions before and after the diagnosis of TAK, were evaluated retrospectively. The indication for vascular interventions, the stage of the disease, the type of vascular intervention, early and late results were recorded (Fig. 1, Table I).

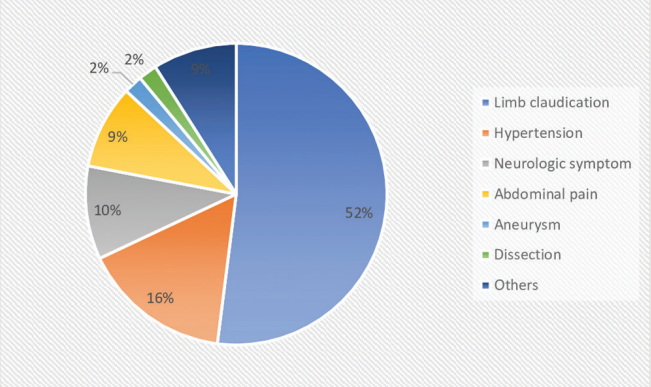

Fig. 1. Indications for vascular interventions.

Table I. The characteristics of Takayasu arteritis (TAK) patients (n=58).

Type of procedure, n (%)	
Stent	73 (64%)
Balloon angioplasty	17 (14,9%)
Bypass graft	19 (16,7)
Endarterectomy	2 (1,8%)
TEVAR	3 (2,6%)
Treatment, n (%)	
Glucocorticoids	54 (41,2%)
High dose	6 (5,9%)
csDMARD	52 (46,8%)
bDMARD	15 (13,5%)
Type of vascular involvement, n (%)	
Tip 1	20 (18%)
Tip 2a	2 (1,8%)
Tip 2b	3 (2,7%)
Tip 3	1 (0,9%)
Tip 4	12 (10,8%)
Tip 5	73 (65,8%)
Time to the last visit after the procedure, median (IQR), months	110 (IQR:35-161)

csDMARD: conventional synthetic disease-modifying antirheumatic drugs; bDMARD: biological disease-modifying antirheumatic drugs.

Results. In 54 patients, 114 lesions (110 stenosis and occlusions, four aneurysms or dissections) were included. Among the patients, 57.6% (n=66) underwent procedures while under immunosuppressive therapy following a diagnosis of TAK. Nine (7.9%) vascular interventions resulted in early failure. One hundred two procedures (89.5%) had available control imaging. Restenosis occurred in 52 (48.6%) lesions during follow-up. The median time to restenosis was 23 (IQR: 6–60) months. There was no difference in

restenosis rate between interventions before and after TAK diagnosis. The risk of restenosis was highest in the subclavian artery compared to other vessels. Our study also revealed a significant correlation between hyperlipidemia and restenosis (p=0.04).
Conclusion. Vascular interventions can be safely performed in patients with TAK. However, considering that 42.1% of vascular procedures are performed before diagnosis, it is crucial for the relevant departments to raise awareness and refer patients for rheumatologic evaluation. Additionally, managing hyperlipidemia may be a key strategy to prevent restenosis in patients with TAK.

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P-23

Phenotypes of patients with giant cell arteritis comparing clinical presentation with diagnostic imaging results

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Background. Giant cell arteritis (GCA) is a vasculitis of the medium to large sized vessels. Two subtypes can be distinguished on imaging: cranial GCA (C-GCA) and extracranial large vessel GCA (LV-GCA), which often overlap. It is unclear whether clinical presentation reflects vessel involvement on imaging.
Objective. This study describes cranial and constitutional symptoms of patients with C-GCA, LV-GCA and C/LV-GCA based on imaging.
Methods. GCA patients who underwent both ultrasound and FDG-PET/CT were included from the prospective Hospital Group Twente (ZGT) GCA Early in Twente (GET) cohort and the University Medical Center Groningen (UMCG) GCA/PMR/SENEX (GPS) cohort. Diagnosis was confirmed after 6 months follow-up and subtype was stratified by vessel involvement on imaging. Symptoms per subtype were described grouped in cranial (headache, scalp tenderness, jaw claudication and/or vision loss) and constitutional (fever, fatigue, weight loss) symptoms.
Results. In total, 71 patients were included. 12 patients had C-GCA, 15 had LV-GCA and 43 had C/LV-GCA. All C-GCA patients, 76.3% of the LV-GCA patients and 79.1% of the C/LV-GCA patients had cranial symptoms. Constitutional symptoms were present in 75.0% of C-GCA patients, 80.0% of LV-GCA patients and 83.7% of C/LV-patients. Most patients presented with both cranial and constitutional symptoms. Clinical symptoms reflect GCA subtype classified on imaging in 47.9% of patients. The majority of patients had C/LV-GCA based on imaging.
Conclusion. Symptoms do not always correspond with subtype and therefore categorization of GCA patients based on clinical presentation only should be avoided without confirmation on imaging.

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P-24

Prevalence and management of patients with comorbidities and frailty in new-onset polymyalgia rheumatica (PMR)

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Background. High comorbidity burden and frailty in PMR may increase glucocorticoid (GC) toxicity risk and warrant personalized management (earlier specialist care, screening, less GC exposure).
Objective. Describe prevalence and management of comorbidities, resulting from or worsening due to long-term GC use, and frailty in PMR.
Methods. Patients aged ≥50 years, without PMR or GCA history, ≥1 inpatient or ≥2 outpatient PMR ≥30 – <365 days (D) apart were identified from Medicare claims. GC initiation 7.5–25mg/D <30D after 1st inpatient or from 1st outpatient code to 30D after 2nd code with GC dose, ≥200mg in 1st ≤30D, continuous ≥4 months. Key exclusion criteria included seropositive RA, other rheumatic disease, conventional immunomodulatory drugs/interleukin-6 receptor inhibitor during baseline. Outcomes included prevalence of comorbidities and frailty (claims-based frailty index ≥0.2), GC use, rheumatology care, and Dual-energy X-ray absorptiometry (DXA) screening.
Results. Of 5,499 patients, at baseline 72.5% had ≥2 comorbidities, and 33.8% were frail. Comorbidities/frailty increased over 3 years; 4-fold in moderate-severe frailty (Table I). More patients with comorbidities/frailty were on long-term GCs (Table II). DXA obtained ≤2 years prior to ≤3 months after GC in 42.8% of patients. In ≤3 years, 36.7% of patients did not visit rheumatology regardless of comorbidity/frailty.
Conclusion. Many new-onset PMR patients had/developed comorbidities/frailty, possibly due to GC, PMR activity, aging or other factors. Despite increased vulnerability to GC toxicity, no substantial difference was observed in GC use. BMD screening and rheumatology referral need improvement.
Originally presented at EULAR 2024; Funding by Sanofi and Regeneron.

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Table I. Prevalence of comorbidities and frailty over time.

n (%)	Baseline period[a] (N=5,499)	Year 1[b] (N=5,499)	Year 2[c] (N=3,362)	Year 3[d] (N=1,608)
Comorbidity				
Hypertension	4,676 (85.0)	4,890 (88.9)	3,046 (90.6)	1,470 (91.4)
Psychiatric conditions (psychosis, mania, agitation, mood lability, catatonia, depersonalization, delirium)	3,283 (59.7)	3,756 (68.3)	2,473 (73.6)	1,230 (76.5)
Diabetes	1,456 (26.5)	1,766 (32.1)	1,160 (34.5)	558 (34.7)
Low bone density (Osteoporosis or osteopenia)	1,171 (21.3)	2,057 (37.4)	1,414 (42.1)	735 (45.7)
Heart failure	605 (11.0)	967 (17.6)	685 (20.4)	363 (22.6)
Hospitalized infection	513 (9.3)	1,029 (18.7)	792 (23.6)	439 (27.3)
Ocular Event (cataract surgery)	289 (5.3)	565 (10.3)	559 (16.6)	348 (21.6)
Fracture event	171 (3.1)	395 (7.2)	355 (10.6)	201 (12.5)
Cardiac dysrhythmia	139 (2.5)	243 (4.4)	191 (5.7)	123 (7.6)
Major adverse cardiovascular event (MACE)[e]	131 (2.4)	271 (4.9)	228 (6.8)	144 (9.0)
Glaucoma	119 (2.2)	201 (3.7)	154 (4.6)	80 (5.0)
Steroid myopathy	66 (1.2)	251 (4.6)	182 (5.4)	89 (5.5)
Unstable angina	35 (0.6)	64 (1.2)	54 (1.6)	32 (2.0)
GI event	23 (0.4)	41 (0.7)	33 (1.0)	20 (1.2)
Osteonecrosis	15 (0.3)	31 (0.6)	23 (0.7)	12 (0.7)
Frailty				
Any Frailty (CFI 0.20–1)	1,857 (33.8)	2,667 (48.5)	1,906 (56.7)	968 (60.2)
Mild Frailty (CFI 0.20–0.29)	1,579 (28.7)	1,970 (35.8)	1,317 (39.2)	635 (39.5)
Moderate to Severe Frailty (CFI 0.30–1)	278 (5.1)	697 (12.7)	589 (17.5)	333 (20.7)

[a]Baseline period is defined as 12 months prior to GC initiation; [b]year 1 is defined as day 365, [c]year 2 as day 730, and [d]year 3 as day 1095 following GC initiation; [e]Includes myocardial infarction, stroke, percutaneous coronary intervention, and cardiac artery bypass surgery.
CFI: claims-based frailty index; GI: gastrointestinal.

Table II. Proportion of patients on GC and GC dose over time based on baseline comorbidity and frailty status.

	1 Year[a] (N=3,513)		2 Years[b] (N=1,561)		3 Years[c] (N=629)	
	On GC[d,e] n (%)[f]	GC dose (mg/day) Median (IQR)	On GC[d,e] n (%)[f]	GC dose (mg/day) Median (IQR)	On GC[d,e] n (%)[f]	GC dose (mg/day) Median (IQR)
Comorbidity						
No comorbidity	150 (58.1)	4.9 (2.8, 8.6)	69 (41.3)	4.1 (2.0, 7.8)	29 (36.3)	3.9 (2.0, 6.8)
≥1 comorbidity	3,363 (64.2)	5.0 (2.8, 9.0)	1,492 (46.7)	4.8 (2.5, 7.9)	600 (39.3)	4.8 (2.1, 7.4)
≥2 comorbidities	2,574 (64.6)	5.0 (2.8, 9.0)	1,154 (47.9)	4.8 (2.5, 8.3)	461 (40.5)	4.8 (2.2, 8.0)
≥3 comorbidities	1,416 (64.8)	5.0 (2.8, 9.3)	652 (50.2)	4.9 (2.8, 8.7)	265 (44.0)	4.8 (2.0, 8.0)
Hypertension	3,004 (64.2)	5.0 (2.8, 9.0)	1,348 (47.4)	4.8 (2.5, 7.9)	542 (40.1)	4.8 (2.2, 7.6)
Psychiatric conditions[g]	2,127 (64.8)	5.0 (2.8, 9.3)	949 (48.3)	4.8 (2.6, 8.8)	394 (42.2)	4.8 (2.1, 7.9)
Diabetes	932 (64.0)	5.0 (2.9, 8.9)	428 (47.5)	4.9 (2.5, 8.1)	150 (36.6)	4.5 (2.2, 7.5)
Low bone density (Osteoporosis or osteopenia)	764 (65.2)	5.0 (2.7, 8.7)	322 (45.5)	4.8 (2.8, 7.7)	134 (40.5)	4.9 (1.9, 8.3)
Heart failure	398 (65.8)	5.8 (3.4, 9.7)	165 (49.7)	5.0 (3.1, 9.7)	66 (46.8)	4.8 (2.1, 9.2)
Hospitalized infection	335 (65.3)	5.5 (3.4, 9.7)	155 (56.8)	4.9 (3.2, 8.8)	53 (47.7)	4.9 (2.5, 9.7)
Ocular Event (cataract surgery)	181 (62.6)	4.8 (2.7, 7.8)	91 (49.2)	4.1 (2.2, 7.2)	40 (40.4)	3.9 (2.1, 7.2)
Fracture event	117 (68.4)	5.3 (3.2, 9.7)	58 (54.7)	4.9 (2.5, 7.3)	18 (46.2)	4.9 (2.7, 7.5)
Cardiac dysrhythmia	87 (62.6)	4.9 (2.5, 7.7)	43 (48.9)	4.8 (2.9, 7.3)	21 (46.7)	5.0 (2.2, 8.3)
Major adverse cardiovascular event (MACE)[h]	81 (61.8)	5.5 (2.7, 9.7)	38 (50.0)	4.8 (3.4, 9.6)	14 (48.3)	3.9 (2.8, 9.1)
Glaucoma	80 (67.2)	4.4 (2.4, 6.9)	38 (53.5)	4.8 (3.1, 9.7)	15 (55.6)	4.9 (4.1, 6.8)
Unstable angina	24 (68.6)	5.6 (2.9, 9.7)	<11	7.2 (2.7, 10.0)	<11	2.2 (1.9, 4.4)
GI event	17 (73.9)	5.0 (3.8, 8.3)	<11	9.7 (6.9, 9.8)	<11	8.4 (4.2, 10.2)
Osteonecrosis	<11	4.5 (1.0, 8.5)	<11	4.8 (4.8, 10.1)	<11	10.8 (10.8, 10.8)
Frailty						
No/Pre-frail (CFI 0–0.19)	2,316 (63.6)	4.9 (2.6, 8.5)	1,016 (44.4)	4.3 (2.4, 7.4)	435 (38.0)	4.3 (2.0, 7.0)
Any Frailty (CFI 0.20–1)	1,197 (64.5)	5.4 (3.1, 9.7)	545 (50.7)	4.9 (2.9, 9.0)	194 (41.8)	4.9 (2.4, 8.5)
Mild Frailty (CFI 0.20– 0.29)	1,006 (63.7)	5.2 (2.9, 9.5)	460 (49.7)	4.9 (2.9, 9.2)	166 (40.6)	5.0 (2.4, 8.5)
Moderate to Severe Frailty (CFI 0.30–1)	191 (68.7)	6.4 (4.0, 9.7)	85 (56.7)	5.0 (3.4, 7.5)	28 (50.9)	4.7 (2.5, 9.3)

[a]Year 1 is defined as day 365, [b]Year 2 as day 730, and [c]Year 3 as day 1095 following GC initiation; [d]Discontinuation of GC was defined using a 60 day gap; [e]Categories with <11 patients are not reported to avoid potential identification of patients; [f]The denominator to calculate the % is based on the number of patients available at the particular timepoint for each characteristics; [g]e.g. psychosis, mania, agitation, mood lability, catatonia, depersonalization, delirium; [h]Includes myocardial infarction, stroke, percutaneous coronary intervention, and cardiac artery bypass surgery.
CFI: claims-based frailty index; GC: glucocorticoids; GI: gastrointestinal.

P-26

RARE CASE OF TAKAYASU ARTERITIS: BILATERAL SUBCAPSULAR RENAL HEMORRHAGE IN AN ELDERLY MALE

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Background. Takayasu arteritis (TAK) is a rare chronic inflammatory vasculitis of large arteries, primarily affecting young women. It involves a cell-mediated inflammatory process with granulomatous inflammation, typically targeting the aorta ant its main branches. Diagnosing TAK is challenging due to non-specific clinical signs.
Case report. We report a unique case of a 60-year-old male presenting with limb pain, weight loss, decreased appetite, and fatigue. Laboratory tests showed elevated ESR (54mm/h) and CRP (129 IU/ml), with negative results for rheumatoid factor (RF), anti-nuclear antibody (ANA), and anti-neutrophil cytoplasmic antibody (ANCA).
Viral tests, including hepatitis B and C, and LIOFERON-TB, were negative. CT angiography revealed panaortitis with narrowing of the distal descending thoracic aorta, the proximal celiac artery, subtotal narrowing of the superior and inferior mesenteric arteries, and occlusion of both external iliac arteries. Severe stenosis of the right and left subclavian arteries was noted. Additionally, the patient exhibited a rare manifestation of TAK: bilateral subcapsular renal hemorrhage without urinary tract obstruction and no history of trauma or significant medical issues.
Learning points. In conclusion, this exceptional case of Takayasu arteritis, marked by the extremely rare presentation of bilateral subcapsular renal hemorrhage without impaired renal function, underscores the critical need for heightened awareness and consideration of TAK in differential diagnoses. The patient's successful treatment with glucocorticoid pulse therapy, continued oral glucocorticoids (60mg prednisolone), immunosuppressive therapy (Infliximab, Cyclophosphamide), and anticoagulant therapy highlights the importance of recognizing and managing such uncommon manifestations. This case expands TAK's clinical spectrum and necessitates vigilance in atypical presentations.
Key words: Takayasu arteritis, subcapsular renal hemorrhage, rare manifestation

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P-28

Study on the changes of CXCL10 level in serum of isolated rheumatic polymyalgia

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Background and Objective. Polymyalgia rheumatica is a chronic inflammatory rheumatic disease characterized by pain and stiffness in the neck, shoulders, and pelvic muscles of elderly people. Chemokines are small molecular proteins that induce the chemotaxis of various immune cells. CXC chemokine ligand 10 is a CXC family chemokine which is induced by interferon gamma (IFN-γ) and synthesized and secreted by various immune cells (such as monocytes, macrophages, T cells, B cells, NK cells, etc.) and non-immune cells (endothelial cells, epidermal cells, fibroblasts, etc.). In order to study the role of chemokines in the pathogenesis of PMR, this study first screened chemokines that may be related to the pathogenesis of PMR using protein chips, combined with previous research literature, CXCL10 is selected and then expanded the sample size using enzyme-linked immunosorbent assay to verify the changes in serum CXCL10 levels between PMR and control groups.
Methods. Serum samples from 6 patients with PMR before and after six month treatment, 6 patients with active rheumatoid arthritis (RA), and 6 healthy controls were collected from the Yijishan Hospital of Wannan Medical College from September 2019 to December 2020. The protein chip technology was used to detect the significantly different chemokine CXCL10 in each group. In addition, serum samples from another 28 patients with active PMR, 26 patients with remission PMR, 24 patients with active RA, and 24 healthy controls were collected from January 2021 to July 2023. The levels of CXCL10 in each group were verified by enzyme-linked immunosorbent assay (ELISA), and the correlation between serum CXCL10 levels and clinical activity parameters of PMR was analyzed.
Results. Protein chip screening showed significant differences in CXCL10 levels before and after treatment in PMR. ELISA verification found that the peripheral serum CXCL10 levels in the active PMR group (87.30±54.55 pg/ml) were significantly higher than those in the PMR remission group (0.00±0.00 pg/ml) (p<0.001), and also significantly higher than those in the active RA group (10.45±9.17 pg/ml) (p=0.003) and healthy control group (6.25±4.84 pg/ml) (p<0.001). Correlation analysis showed that the serum CXCL10 levels in PMR patients were significantly positively correlated with serum ferritin (r=0.450, p=0.024). No correlation can be observed between CXCL10 level and ESR, CRP and PMR-AS
Conclusion. CXCL10 may play a role in the pathogenesis of isolated PMR, and its main biological effect may be to transmit information and induce the migration of immune cells, rather than being a direct pro-inflammatory factor. CXCL10 may be helpful for the differential diagnosis of PMR and RA, especially serum active RA.

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P-29

SUCCESSFUL PREGNANCY OUTCOME IN A FEMALE PATIENT WITH TAKAYASU ARTERITIS TREATED WITH TOCILIZUMAB

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Background. Takayasu's Arteritis (TAK) is a large-vessel systemic vasculitis that affects mainly young women of childbearing age. Pregnancy is an important challenge in the management of TAK.
Case description. We present a case of a 33-year-old African woman, presenting with acute ischemic stroke – aphasia and right sided hemiparesis – with occlusion of the proximal left medial cerebral artery. She was treated with alteplase with recanalization and neurologic improvement. She reported mild constitutional symptoms and had raised inflammatory markers. Angiography and ultrasound revealed bilateral occlusion of common carotids (Fig. 1-2), left vertebral stenosis and left subclavian artery severe stenosis. PET-FDG showed a thoracic and abdominal aortitis. The diagnosis of Takayasu Arteritis was established and treatment started with prednisolone 60 mg with tapering and weekly SC tocilizumab (TCZ).

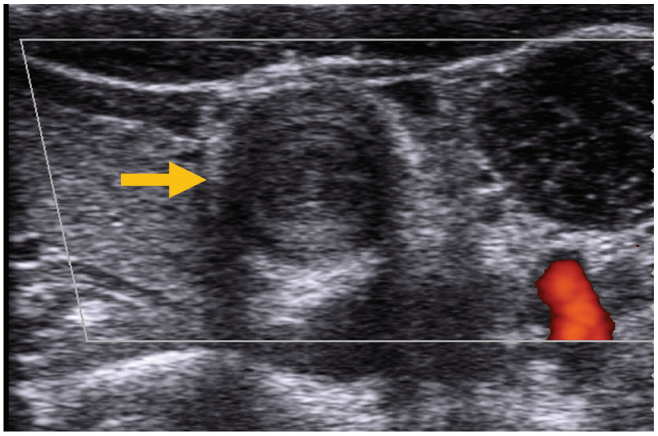

Fig. 1. Vasculitic occlusion (yellow arrow) of left common carotid artery on vascular ultrasound.

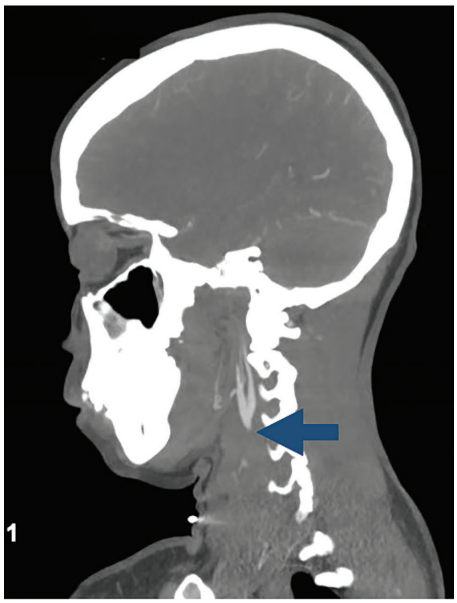

Fig. 2. CT-angiography showing complete occlusion of left common carotid artery (blue arrow), with permeabilization of the carotid bifurcation through anastomosis of the external carotid artery.

Three-months after stroke, she had a positive pregnancy test; obstetric ultrasound showed a 9-week viable pregnancy. It was decided to keep treatment with weekly SC TCZ and prednisolone 5mg daily. Vasculitis follow-up was performed with vascular ultrasound. At 39 weeks of gestation the patient underwent an elective cesarian section, newborn weighing 2720g and Apgar score 9/10/10. Six-months after delivery, we report good maternal and infant outcomes.
Discussion. In this patient, the benefits of TCZ in TAK control were considered to overcome the potential pregnancy related risks. Few clinical data about the safety and efficacy of TCZ in pregnant women with TAK are available. The control of disease activity during pregnancy is important, since it is associated with poor maternal and fetal outcomes.
Key words: Takayasu arteritis, pregnancy, tocilizumab

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P-30

The importance of fatigue and patient reported outcomes in predicting disease relapse in those with pure PMR

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Background. A significant unmet need in the management of those with Polymyalgia Rheumatica (PMR) is the lack of a disease stratification tool to guide therapy from disease outset.
Objective. To explore baseline patient reported outcomes as potential prognostic factors for disease outcomes at one year in those with pure PMR.
Methods. Participants, presenting to a fast track PMR clinic, with a new diagnosis of pure PMR, were recruited to this multicentre longitudinal cohort study. At baseline, patient reported outcomes including fatigue using FACIT-F, mood using PHQ-9, anxiety using GAD-7, pain using p-VAS, and overall health related quality of life and disability using the HAQ-DI screening tool were collected.
Results. 79 patients (48.1% female) were included. 45.6% (n=36) of patients experienced disease relapse over the one-year follow-up period. There was a significant difference in mean rank between relapse and non-relapse patients PVAS score (p=0.05) and HAQ health status score (p=0.02), with relapse patients having significantly higher scores. Relapse patients also had significantly lower FACIT-F scores (p=0.03), indicating a greater degree of fatigue. Relapse patients had significantly lower PHQ-9 scores (p=0.03). There was no statistically significant difference between HAQ-DI (p=0.27) or GAD-7 (p=0.30) scores in those with relapse, and those without (Table I).

Table I.

		Relapse N=36 (45.6%)	No Relapse N=43 (54.4%)	Mean Difference	P-Value
FACIT-F	Mean (SD)	32.6 (12.1)	38.2 (10.5)	-5.6	0.03
PHQ-9	Median Min-Max (IQR) Mean Rank	8 5-15 (5.5) 46.3	6 0-17 (14.5) 34.7	N/A	0.03
p-VAS	Median Min-Max (IQR) Mean Rank	4 0-6 (3.75) 45.4	1 0-2 (2.0) 35.5	N/A	0.05
HAQ-DI	Median Min-Max (IQR)	0.94 0.25-2.00 (1.09)	0.00 0.00-0.63 (0.47)	N/A	0.27
GAD-7	Median Min-Max (IQR)	6.5 2.5-15 (10.75)	4.3 0-13 (10.25)	N/A	0.30

Conclusion. In the quest to develop a disease stratification tool for those with pure PMR, our study findings emphasise for the first time, the importance of patient reported outcomes, in particular fatigue, pain and overall health scores.
Key words: PMR, patient reported outcomes, fatigue, pain, mood, relapse

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P-31

The incidence and clinical features of ischemic stroke in patients with giant cell arteritis

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Background and Objective. Ischemic stroke caused by Gian cell Arteritis (GCA) is a rare but a devastating condition. The aim of this study was to determine the incidence of ischemic stroke in patients with GCA and to characterize the features of GCA related strokes.
Methods. Retrospective analysis of the characteristics and incidence of ischemic stroke in a single-center cohort of 48 consecutive patients GCA patients (Table I); median age 71 (66–81) years-old at diagnosis, 50% female sex. Vascular Ultrasound was performed in 42 patients (88%), FDG-PET in 35 (73%) and CT-angiography in 20 (42%). Ischemic strokes were classified as Vasculitis-Related Strokes (VRS) and unrelated with vasculitis.
Results. During a median follow-up of 23 (12-44) months, 8 (17%) patients had ischemic stroke, 4 (8%) were VRS and 4 (8%) vasculitis-unrelated strokes. The overall incidence of ischemic stroke was 5.09 (95%CI 2.55–10.19) per 100 patient-years. All VRS occurred within 4 weeks after the diagnosis of GCA, while vasculitis unrelated strokes occurred at 33 (25–52) months after GCA diagnosis (p=0.014).

Table I. Clinical characteristics of the study population.

Variables	GCA patients n=48	GCA with Stroke n=8	GCA with no stroke n=40	p-value
Age at diagnosis	71 (66-81)	75 (70-77)	71 (64-81)	0,803
Female sex	24 (50%)	4 (50%)	20 (50%)	0,650
Cardiovascualr risk factors				
Hypertension	38 (79%)	7 (88%)	31 (78%)	0,464
Diabetes	11 (23%)	5 (63%)	6 (15%)	**0,010**
Hyperlipidemia	31 (65%)	5 (63%)	26 (65%)	0,595
Obesity	6 (14%)	1 (13%)	5 (13%)	0,545
Smoking	11 (23%)	1 (13%)	10 (25%)	0,618
Previous Stroke/MI	4 (8%)	0	4 (10%)	0,571
Vasculitis features				
Large-Vessel GCA	33 (69%)	4 (50%)	29 (73%)	0,199
Headache	36 (75%)	8 (100%)	28 (70%)	0,080
Jaw claudication	26 (54%)	5 (63%)	21 (53%)	0,452
Vision loss	14 (29%)	3 (38%)	11 (28%)	0,428
Constitutional symptoms	35 (73%)	5 (75%)	29 (73%)	0,630
Polymyalgia rheumatica	38 (79%)	6 (75%)	32 (80%)	0,536
Inflammatory anemia	29 (60%)	7 (88%)	22 (55%)	0,136
Treatment				
Methylprednisolone IV	10 (21%)	5 (63%)	5 (13%)	**0,006**
Methotrexate	13 (27%)	0	13 (33%)	0,062
Tocilizumab	25 (52%)	4 (50%)	21 (53%)	0,600

Results are expressed as median (IQR) for quantitative variables, n (%) for qualitative variables. GCA: giant cell arteritis; IV: intravenous; MI: myocardial infarction.

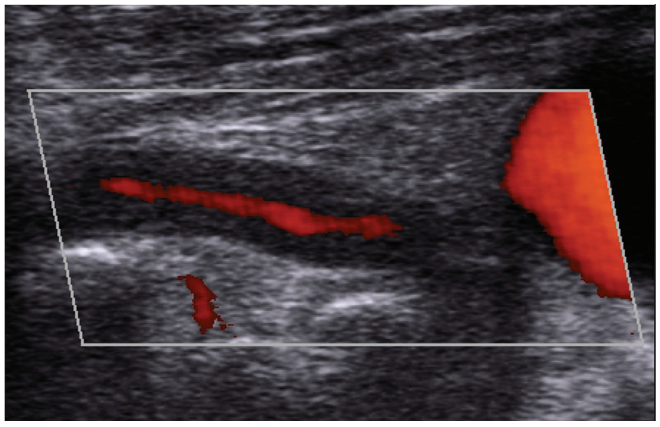

Fig. 1. Vascular ultrasound image depicting diffuse hipoecoic wall thickening of the right vertebral artery at the V1 segment in a patient with GCA and vertebrobasilar ischemic stroke.

All patients with VRS had vertebrobasilar territory infarctions and 1 patient had both vertebrobasilar and carotid infarctions. VRS all had vertebral artery vasculitis (Fig. 1), whereas this was reported in 3/44 (7%) of the remaining GCA patients (p=0.001). All patients with vasculitis unrelated strokes had carotid territory infarctions.
Conclusion. Despite the small population of this analysis, VRS tend to occur during the first weeks after GCA diagnosis and mainly affect the vertebrobasilar territory. Vertebral artery vasculitis may raise the suspicion for increased risk of VRS in GCA.
Key words: giant cell arteritis, stroke, ultrasound, large vessel vasculitis

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P-32

Ultrasound assessment of subclinical giant cell arteritis in patients with polymyalgia rheumatica in a Bulgarian population: a cross-sectional study

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Background. Polymyalgia rheumatica (PMR) and Giant cell arteritis (GCA) are closely related conditions. The prevalence of subclinical GCA in PMR has been of increased scientific interest in recent years.
Objective. To determine the prevalence of subclinical GCA in patients with PMR in a Bulgarian population using US and to compare mean IMT values of temporal and axillary arteries between PMR patients and a control group.
Methods. A cross-sectional single-center study of 37 patients with newly diagnosed PMR and 37 healthy controls. All participants underwent US of common superficial temporal arteries with their frontal and parietal branches and axillary arteries. Vessels were examined bilaterally in longitudinal and transverse scans using GSUS and CDUS. Subclinical GCA was considered in the presence of halo and compression sign. IMT was measured at the level of the largest non-atherosclerotic area in the two subgroups of PMR patients (with and without subclinical GCA) and healthy controls.
Results. Of 37 patients with PMR, 11 (29.70%) had subclinical GCA. US findings of vasculitis were present in at least 2 of the 8 arteries examined with predominant bilateral involvement (Fig. 1). No subclinical vasculitis was identified in the healthy controls. Mean IMT values in PMR patients with US GCA were: 0.56 ± 0.07 mm, 0.40 ± 0.06 mm, 0.44±0.04 mm, 1.53±0.09 mm for CSTA, PTA, FTA and AxiA, respectively. They were significantly higher than IMT in PMR patients without US GCA: 0.28±0.04 mm, 0.21±0.03 mm, 0.23±0.07 mm, 0.67±0.12 mm, respectively and healthy controls: 0.25±0.04 mm, 0.19±0.02 mm, 0.20±0.03 mm, 0.59±0.06 mm, respectively (Fig. 2).

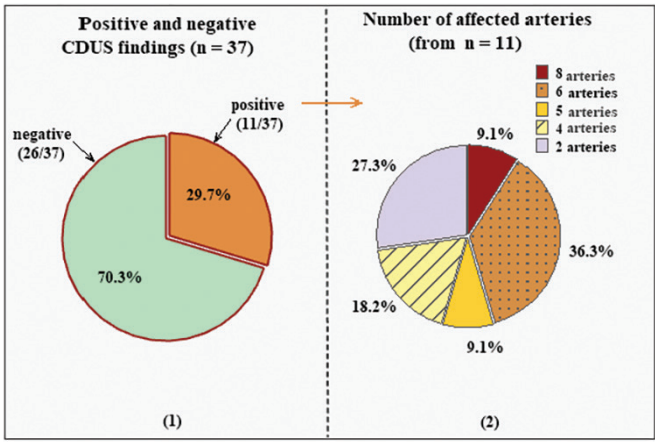

Fig. 1. Prevalence of positive CDUS findings (subclinical GCA) in PMR patients (1). Distribution of patients with positive findigs according to the number of affected arteries (2).

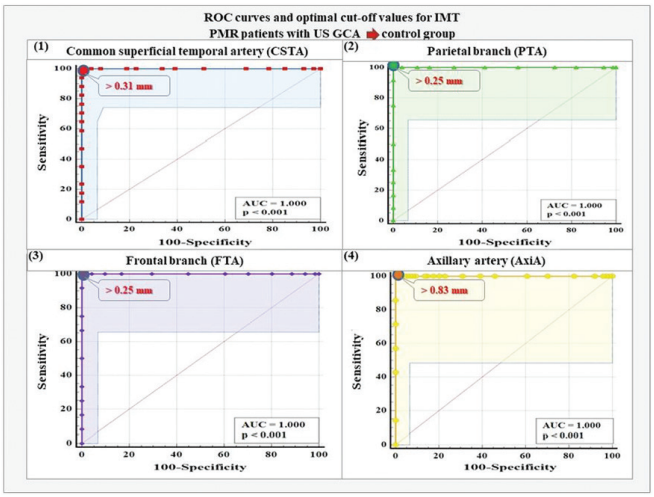

Fig. 2. ROC curves for IMT cut-off values of CSTA (panel 1), PTA (panel 2), FTA (panel 3) and AxiA (panel 4) distinguishing PMR patients with US GCA from healthy controls.

Conclusion. Over ¼ of the 'pure' PMR patients in a Bulgarian population had subclinical GCA. The evaluation of IMT for each artery may serve as a valuable US tool for early detection of subclinical vasculitis.

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[68Ga]Ga-DOTA-Siglec-9-PET/CT imaging of inflammation in polymyalgia rheumatica

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Background and Objective. In polymyalgia rheumatica (PMR), positron emission tomography-computed tomography (PET/CT) with [18F]FDG can detect inflammation but has low specificity. New radiotracers such as [68Ga]Ga-DOTA-Siglec-9 may improve the assessment of PMR. Siglec-9 is the ligand of vascular adhesion protein 1 (VAP-1), an endothelial adhesion molecule that translocates to the cell surface upon inflammatory stimuli. This study is the first to investigate the diagnostic value of [68Ga]Ga-DOTA-Siglec-9-PET/CT in patients with PMR.
Methods. Patients with active PMR underwent [68Ga]Ga-DOTA-Siglec-9-PET/CT. Concurrent clinical and laboratory evaluations, as well as musculoskeletal ultrasound of the shoulder and pelvic regions, were conducted. Pearson correlation analysis was used to examine the associations between relative tracer uptake and prednisolone dosage, as well as C-reactive protein (CRP) levels.
Results. This study recruited eight patients with active PMR. [68Ga]Ga-DOTA-Siglec-9-PET/CT revealed increased tracer uptake in the shoulder and pelvic girdle regions (Fig. 1). A significant negative association was found between prednisolone intake and tracer uptake in the shoulder region (r=-0.775, *p*=0.024). No significant association was found between prednisolone intake and tracer uptake in the pelvic region (r=-0.486, *p*=0.222), nor between CRP levels and tracer uptake. Increased tracer uptake in the aorta and subclavian artery was observed in half of the patients (Table I).
Conclusion. This study highlights the diagnostic potential of [68Ga]Ga-DOTA-Siglec-9-PET/CT for assessing disease activity in PMR, revealing increased, patient-specific tracer uptake in the shoulder and pelvic girdle regions. Additionally, the increased tracer uptake observed in the aorta and subclavian artery underscores the potential of [68Ga]Ga-DOTA-Siglec-9-PET/CT for identifying coexisting giant cell arteritis.

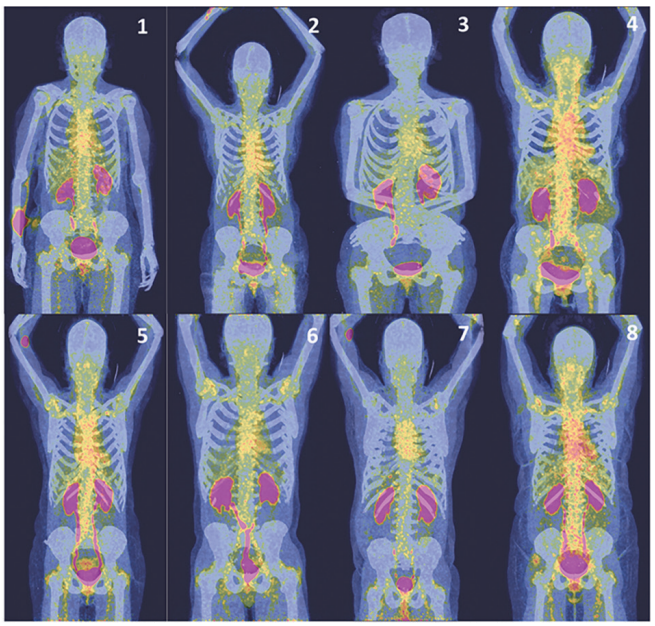

Fig. 1. [68Ga] Ga-DOTA-Siglec-9-PET/CT imaging findings in patients with active polymyalgia rheumatic.
The figure illustrates the [68Ga]Ga-DOTA-Siglec-9-Pet/CT results of eight patients with active polymyalgia rheumatica, showing increasing tracer uptake in the shoulder and pelvic regions.
Numbers 1-8 correspond to patient numbers in Table I.

Table I. Relative tracer uptake in [68Ga]Ga-DOTA-Siglec-9-PET/CT in the shoulder and pelvic regions of patients with active polymyalgia rheumatica.

Patient	Age (years)	Sex	Prednisolone in the last 7 days (mg)	C-reactive Protein (mg/l)	Shoulder	Pelvic	Aorta	Subclavian artery
1	79.5	female	120	131.0	8.82	12.78	14.06	13.11
2	78.7	female	500	44.3	11.95	25.56	15.21	11.42
3	78.8	female	120	57.9	12.89	15.40	15.25	11.05
4	79.7	female	0	40.0	13.88	10.75	26.13	17.63
5	66.3	female	70	30.3	15.03	18.40	19.60	20.34
6	59.8	male	0	31.8	19.42	16.28	16.22	14.68
7	61.6	male	0	9.5	20.22	24.47	19.72	12.83
8	66.9	female	0	15.0	25.44	18.24	27.92	24.32

The table shows the clinical and laboratory data of eight patients with active polymyalgia rheumatica, including age, sex, prednisolone dosage in the seven days before the scans, C-reactive protein levels, and relative tracer uptake in [68Ga]Ga-DOTA-Siglec-9-PET/CT in the shoulder and pelvic region, aorta, and subclavian artery. Pearson correlation analysis reveals a significant negative association between prednisolone dosage and tracer uptake in the shoulder (r=-0.775, p=0.024).
mg: milligram; l: liter.

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[68Ga]Ga-DOTA-Siglec-9-PET/CT imaging of vascular inflammation in giant cell arteritis

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Background and Objective. Positron emission tomography-computed tomography (PET/CT) with [18F]FDG has demonstrated utility in assessing disease activity in giant cell arteritis (GCA), albeit with limited specificity for inflammation. The radiotracer [68Ga]Ga-DOTA-Siglec-9 may improve GCA assessment due to its theoretically higher specificity for inflammatory activity. Siglec-9, a leukocyte ligand for the endothelial adhesion molecule vascular adhesion protein 1 (VAP-1), translocates to the endothelial surface upon inflammatory stimuli. This study is the first to evaluate the diagnostic value of [68Ga]Ga-DOTA-Siglec-9-PET/CT in GCA patients experiencing relapse.

Methods. Patients with relapsed GCA underwent [68Ga]Ga-DOTA-Siglec-9-PET/CT. Concurrent clinical and laboratory assessments and vascular ultrasound of the superficial temporal, subclavian, and axillary arteries were performed. Associations between relative tracer uptake in the aorta and subclavian artery, prednisolone intake, and C-reactive protein (CRP) levels were analyzed using Pearson correlation analysis.

Results. This study included eight patients with relapsed GCA. [68Ga]Ga-DOTA-Siglec-9-PET/CT showed localized increased tracer uptake in the aorta and subclavian artery (Fig. 1). Pearson correlation analysis revealed a significant negative association between prednisolone intake and tracer uptake in the aorta (r=-0.73, p=0.038) and subclavian artery (r=-0.77, p=0.027). No significant correlation was found between CRP levels and tracer uptake (Table I).

Conclusion. This study evaluated the diagnostic utility of [68Ga]Ga-DOTA-Siglec-9-PET/CT for assessing disease activity in GCA, revealing increased, patient-specific tracer uptake in the aorta and subclavian artery. Furthermore, the observed inverse association between prednisolone intake and tracer uptake suggests rapid, therapy-induced endothelial elimination of VAP-1. These results underscore the radiotracer's potential for accurately detecting, assessing, and localizing acute vascular inflammation in GCA.

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Table I. Relative tracer uptake in [68Ga]Ga-DOTA-Siglec-09-PET/CT in the aorta and subclavian artery in giant cell arteritis partients with relapse.

Patient	Age (Years)	Sex	Prednisolone dose in the last 7 days (mg)	C-reactive protein (mg/l)	Aorta	Subclavian Artery
1	75,4	male	140	11,2	16,01	11,71
2	91,0	male	42	8,7	19,55	12,21
3	68,2	female	7	114,6	23,90	18,24
4	55,9	female	70	6,1	24,63	15,92
5	75,8	female	0	23,0	25,06	19,46
6	79,7	female	0	40,0	26,13	17,63
7	82,6	male	35	32,2	29,04	19,68
8	68,9	male	0	33,6	30,74	18,77

The table displays clinical and laboratory data of eight patients with relapses of giant cell arteritis. It displays age, gender, administered prednisolone dose in the last 7 days prior to the scan, C-reactive protein level, and relative tracer uptake in [68Ga]Ga-DOTA-Siglec-09-PET/CT in the aorta and subcavian artery: Pearson correlation analysis indicates a significant negative association between prednisolone dosage and tracer uptake in the aorta (r=0.796, p=0.018) and subclavian artery (r=-0.77, p=0.027). mg: milligram; l: liter.

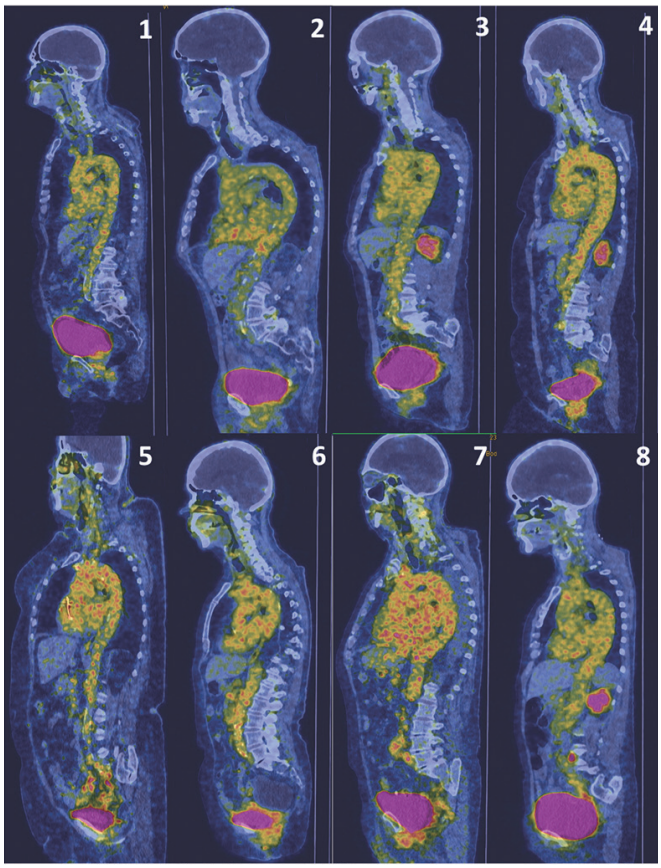

Fig. 1. [68Ga]Ga-DOTA-Siglec-9-PET/CT imaging findings in giant cell arteritis patients with relapse.
The figure illustrates [68Ga]Ga-DOTA-Siglec-9-Pet/CT results from eight patients with giant cell arteritis during relapsing disease activity. Patient-specific, elevated tracer uptakes are shown in the aorta and subclavian artery.
Numbers 1-8 correspond to patient numbers in Table I.