performed using antibodies directed against fibroblasts (CD90, vimentin), myofibroblasts (alpha smooth muscle actin {ASMA}) and vascular smooth muscle cells (desmin). Staining of prolyl-4-hydroxylase (P4H) and myosin were also performed. Stainings were quantified using the ODPviewer® software (Kamax Innovative System, France) by two double-blind observers. Moreover, co-expression of CD90 with different stainings (ASMA, myosin and P4H) was also performed. Cells in culture were identified by immunocytochemistry using anti-CD90 and markers of activity such as myosin and P4H. Functional assays were performed using culture cells obtained from GCA patients' BATs (n=4) and controls (n=4). The BATs were dissected to separate the adventitia from the other two layers. Each dissected fragment was seeded separately. The proliferation was studied using a bromodeoxyuridine incorporation test under different conditions: DMEM, fetal calf serum (FCS), PDGF and supernatants of cells in culture. The study of migration was performed using a scratch test under the same conditions.

Results: CD90 was significantly higher in GCA than controls in adventitia and intima. Vimentin and ASMA were significantly higher in the 3 tunics of patients with GCA. Expression of desmin was only present in the media for both groups with no significant difference. P4H was present in adventice significant differences between both groups. The adventitial and intimal CD90+ cells co-expressed P4H, ASMA and myosin more importantly during GCA. Cultured cells from BATs' adventices expressed CD90 and did not express desmin. These cells had a greater myosin staining during GCA and had an increased proliferation ability during GCA in the presence of FCS, PDGF and fibroblast supernatants of control or GCA patients. The migration rates of these cells were also significantly increased during GCA in the presence of FCS or PDGF.

Conclusion: Vascular remodeling during GCA would start in the adventitial layer with a key role of fibroblasts. Adventitial fibroblasts could be activated into myofibroblasts and acquire proliferative and migratory abilities. They would participate in the intimal hyperplasia, responsible for ischemic complications. Their inhibition could be a therapeutic way to limit these complications. The activating signal of adventitial fibroblasts into myofibroblasts is not yet known during GCA and requires further studies.

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THU0313

COMPARATIVE STUDY OF CLINICAL, ANALYTICAL AND VASCULAR ¹⁸F-FDG UPTAKE EVOLUTION IN PATIENTS WITH GIANT CELL ARTERITIS TREATED WITH METHOTREXATE VS TOCILIZUMAB

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Background: Glucocorticoids remain to be the cornerstone therapy in giant cell arteritis (GCA). However, relapses are common when the prednisone dose is tapered. Thus, additional therapies are required in relapsing GCA. The most widely used conventional immunosuppressive drug is methotrexate (MTX) which efficacy is modest. Consequently, in some cases biological therapy is needed. Among them, the most frequently used is the recombinant humanized anti-IL6 receptor antibody tocilizumab (TCZ).¹

Objectives: To compare clinical evolution, normalization of acute phase reactants and normalization of vascular ¹⁸F-FDG uptake assessed by PET/CT in patients with GCA treated with MTX vs TCZ.

Methods: Comparative multicentric study of 23 patients with GCA treated with MTX vs 36 patients with GCA treated with TCZ who had a baseline and follow-up PET/CT scan. We assessed clinical improvement (no improvement/partial/complete), normalization of acute phase reactants (CRP ≤ 0.5mg/dL and/or ESR ≤ 20 mm/1st hour) and reduction of ¹⁸F-FDG uptake in PET/CT (no reduction/partial/complete normalization). Images were evaluated qualitatively by experienced nuclear medicine physicians. Prednisone tapering was also assessed. Statistical analysis was performed with SPSS. Student's t test or Mann-Whitney U test was used to compare continuous variables, and Chi-squared test or Fisher's exact test for categorical variables as appropriate.

Results: We included 23 patients with GCA treated with MTX (20 women/3 men); mean age 65.6 ± 7.9 years; and 36 patients treated with TCZ (27 women/9 men); mean age 67.5 ± 8.3 years. Clinical, analytical and vascular ¹⁸F-FDG uptake evolution is shown in the TABLE. After one year of treatment, the percentage of patients who experienced complete clinical improvement was higher in those who received TCZ (88.9% vs 44.4%; p=0.003). Normalization of acute phase reactants was also more frequent in patients who received TCZ (92.6% vs 47.6%; p=0.001). In regard with reduction of vascular ¹⁸F-FDG uptake, complete normalization was only achieved in 25% of patients who received TCZ and 14.3% of those who received MTX.

Conclusion: Patients with GCA who received TCZ experienced a more rapid and effective clinical and analytical improvement than patients who received MTX. Besides, prednisone tapering was quicker in patients with TCZ. However, no significant differences were found in complete normalization of ¹⁸F-FDG vascular uptake between both treatments.

Abstract THU0313 –Table 1.

TABLE

	MTX (n=23)	TCZ (n=36)	P
Age, mean ± DS	65.6 ± 7.9	67.5 ± 8.3	0.39
Sex (woman), n (%)	20 (87.0)	27 (77.1)	0.50
Complete clinical improvement, n/N (%)			
- 6 months	9/23 (39.1)	28/35 (80.0)	0.002
- 12 months	8/18 (44.4)	24/27 (88.9)	0.003
- 18 months	7/11 (63.6)	21/22 (95.4)	0.03
- 24 months	7/9 (77.8)	17/18 (94.4)	0.25
Normalization of ESR and/or CRP, n/N (%)			
- 6 months	6/23 (26.1)	30/35 (85.7)	< 0.01
- 12 months	10/21 (47.6)	25/27 (92.6)	< 0.01
- 18 months	10/11 (90.9)	23/23 (100.0)	0.32
- 24 months	9/9 (100.0)	19/19 (100.0)	0.99
Normalization of ¹⁸ F-FDG PET/CT, n/N (%)			
- 6 months	0/11 (0.0)	0/8 (0.0)	0.99
- 12 months	0/14 (0.0)	3/13 (23.0)	0.10
- 18 months	0/5 (0.0)	3/11 (27.3)	0.51
- 24 months	1/7 (14.3)	1/4 (25.0)	0.99
Dose of Prednisone (mg/day), median [IQR]			
- 6 months	7.5 [5.0-15.0]	5.0 [0.3-5.0]	< 0.01
- 12 months	5.0 [5.0-10.0]	0.0 [0.0-5.0]	< 0.01
- 18 months	5.0 [3.0-6.0]	0.0 [0.0-3.3]	0.03
- 24 months	2.5 [0.0-3.0]	0.0 [0.0-0.0]	0.07

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THU0314 OUTCOMES OF PATIENTS TREATED WITH TOCILIZUMAB OR ABATACEPT AS STEROID-SPARING AGENTS WITH GIANT CELL ARTERITIS

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Background: Giant cell arteritis (GCA) is a common form of systemic vasculitis. The current mainstay of GCA management is glucocorticoid (GC) therapy. Recently, at least 2 biological therapies [tocilizumab (TCZ)

Abstract THU0314 –Table 1.

Table 1. Characteristics of the patients included in the study

	Total number of patients (33)	%
Demographic characteristics		
Female/Male	21/12	
Age (years) (mean, SD)	73.6 ±8.7	
Clinical characteristics at the onset of GCA		
Fever (n)	12	36.4
Fatigue (n)	18	54.5
Headache (n)	28	84.8
Scalp tenderness (n)	8	24.2
Jaw claudication (n)	15	45.4
Vision loss (n)	16	48.5
PMR (n)	11	33.3
Diagnosis		
Biopsy proven	27/32	84.4
PET positivity	12/29	41.4
Eco-color-doppler positivity	30/31	97
GC therapy		
Previous methylprednisolone pulses (n)	7	21.2
Oral prednisone (n)	33	100
Dose of oral prednisone (mg/day) (mean, SD)	49.7 ±15.1	
Previous immunosuppressive therapies		
Methotrexate (n)	11	33.3
Mean dose (mg/week) (mean, SD)	15.6 ±1.7	
Mycophenolate (n)	6	18.1
Mean dose (g/day) (mean, SD)	2.2 ±0.4	

and abatacept (ABA)] have been proven to be effective in the management of CGA in randomized controlled trials. Nevertheless, their use as steroid sparing agents might need further investigation.

Objectives: We aimed to investigate the steroid-sparing effect of biological therapies, namely TCZ and ABA, in a cohort of GCA patients when compared to standard GC treatment.

Methods: We retrospectively collected data from GCA patients who attended the S.G. Bosco Hospital, Turin, Italy, who were treated with TCZ, both intravenous (IV) and subcutaneous (SC), and/or ABA SC (8 mg/kg/month, 162 mg/week, and 125 mg/week respectively). These therapies were prescribed as first line agents or as second line when patients were refractory/intolerant/contraindicated to standard immunosuppressive therapies. Complete response to the treatment was defined as a clinical and serological remission after 12 months of therapy; partial response was defined as clinical or serological remission after 12 months of therapy.

Results: This retrospective study included 33 GCA patients [mean age 74 (range 85-57), females 63%, mean follow-up from GCA diagnosis 44.4

±33.5 months). Table 1 resumes the characteristics of the GCA patients included in the study.

Twenty-eight patients out of 33 (85%) received one biologic agent. Five patients (15%) needed a therapeutic switch (one patient from TCZ to ABA, and 4 patients from ABA to TCZ). Patients were treated as follow: 9 with TCZ IV, 11 with TCZ SC, and 18 with ABA. Among the TCZ IV group, all patients experienced a response (57% complete response, and 43% partial response). Among the TCZ SC group, 83% experienced a response (67% complete response, and 16% partial response). Among the ABA group, 86% experienced a response (36% complete response and 50% partial response). After 12 months of therapy, 100% of patients in TCZ groups, both IV and SC, and 64.2% of ABA group were treated with low doses of oral prednisone (≤ 7.5 mg/day) as maintenance. We noticed a significant reduction of inflammatory parameters [C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR)] after 12 months of therapy with TCZ [TCZ IV group: mean baseline CRP (mg/dl) 1.9 ± 2.3 , mean CRP after 12 months of therapy 0.3 ± 0.2 ; mean baseline ESR (mm/h) 58.1 ± 25.6 , mean ESR after 12 months 9.5 ± 4.2 ; TCZ SC group: mean baseline CRP 4.5 ± 3.8 , mean CRP after 12 months 0.2 ± 0.2 ; mean baseline ESR 51.9 ± 27 , mean ESR after 12 months 6.5 ± 6]. When compared to standard GC regimen [1], in patients treated with TCZ, both IV and SC, we estimated a median steroid-sparing effect quantifiable in 30 mg/daily in the first month and an overall steroid-sparing effect of 15 mg/daily when assessed in 12 months.

Conclusion: This retrospective study confirms the efficacy of biological therapies in the management of CGA. Besides, in our experience TCZ allowed a significant reduction of GCs use, especially in the first month of therapy, when compared to standard GCs based regimens.

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THU0315 SEVERITY AND RESPONSE TO INDUCTION THERAPY IN NEW AND RELAPSING ANCA ASSOCIATED VASCULITIS PATIENTS – REAL WORLD PRACTICE DATA

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Background: ANCA-associated vasculitis (AAV) presents clinically with variable severity of disease and current clinical guidelines give varying advice on initial treatment to induce remission. Achieving remission quickly is important to gain disease control and avoid cumulative organ damage from treatment related adverse events.

Objectives: This study examined real world practice of AAV treatment in Europe to understand the AAV severity spectrum and the response to therapy over 12 months.

Methods: 929 incident and 268 relapsing EU AAV patients receiving care from 399 physicians were studied. Patients were referred between 2014-17 and data collected retrospectively at baseline and 1, 3, 6 and 12 months following induction therapy. Birmingham Vasculitis Activity Score (BVAS) was collected in only 12% of patients and severity was defined as mild (localized disease with no systemic symptoms), moderate (Mod, systemic disease with lung and/or kidney involvement) or systemic (rapidly progressive systemic disease with lung and/or kidney involvement).

Results: AAV disease severity varied in both incident (mild 12.2%, moderate 54.3%, severe 33.6%) and relapsing (corresponding values 9.7, 64.6 and 25.7) patients at start of treatment. Comorbidity at time of

Abstract THU0315 –Table 1

	1 month			3 months			6 months			12 months		
	Mild	Mod	Severe	Mild	Mod	Severe	Mild	Mod	Severe	Mild	Mod	Severe
Full	55.3	21.0	13.5	59.4	47.2	28.7	82.6	64.7	45.0	82.8	70.7	53.5
Partial	42.2	74.1	67.9	38.4	48.8	56.8	14.5	32.0	40.6	17.2	26.0	35.7
No	2.5	4.9	18.6	2.2	4.0	14.5	2.9	3.3	14.4	0	3.3	10.8