Table 2. Clinical outcomes and adverse events

	Off TCZ (N = 60)	On TCZ (N = 60)
Follow-up time, median (IQR), years	0.60 (0.20-1.57)	0.59 (0.32-1.50)
Patients with ≥ 1 relapse, n (%)	43 (71.7)	18 (30.0)
1 relapse	19 (31.7)	11 (18.3)
2 relapses	11 (18.3)	3 (5.0)
3 relapses	4 (6.7)	3 (5.0)
≥ 4 relapses	9 (15.0)	1 (1.7)
Time to relapse, median (IQR), years	0.5 (0.3-0.7)	2.1 (0.6-2.6)
Annual relapse rate (95% CI), events per year*	1.43 (0.95-2.13)	0.57 (0.33-0.98)
Patients with ≥ 1 AE, n (%)	29 (48.3)	32 (53.3)
AEs attributed to GC†	52 / 59 (88.1)	38 / 81 (46.9)
AEs attributed to TCZ‡	NA	27 / 81 (33.3)
AEs attributed to GC or TCZ†	52 / 59 (88.1)	53 / 81 (65.4)
Patients with ≥ 1 SAE, n (%)	6 (10.0)	6 (10.0)
SAEs attributed to GC	7 / 8 (87.5)	4 / 9 (44.4)
SAEs attributed to TCZ	NA	3 / 9 (33.3)
SAEs attributed to GC or TCZ	7 / 8 (87.5)	6 / 9 (66.7)

AE, adverse event; CI, confidence interval; GC, glucocorticoids; IQR, interquartile range; NA, not applicable; SAE, serious AE; TCZ, tocilizumab.

* Rates are estimated from a Poisson regression model with ongoing treatment (TCZ and GC combinations), age, smoking history, new or relapsing GCA as covariates, and random patient effect.

† Multiple occurrences of AEs are included. Denominators are all AEs or SAEs.

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SAT0249 IGA VASCULITIS ASSOCIATED WITH INFLAMMATORY BOWEL DISEASE. A RETROSPECTIVE STUDY

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Background: Leukocytoclastic vasculitis has been reported in patients with inflammatory bowel disease (IBD) but cases of IgA vasculitis (IgAV) in IBD are considered rare.

Objectives: The purpose of this study was to describe the baseline characteristics and outcome of a series of patients with IBD and IgAV.

Methods: Biopsy-proven IgAV-patients with prior history of IBD were identified retrospectively at Mayo Clinic, Rochester, MN. Data were abstracted from direct medical chart review. Each IBD-IgAV case was matched to two controls with IgAV but without IBD history based on age, sex and baseline renal function at time of IgAV onset.

Results: A total of 9 patients were included in the study group (6 male, 3 female). Crohn's disease (CD) and Ulcerative Colitis (UC) were present in 7 and 2 patients, respectively. The mean length of time between IBD diagnosis and onset of IgAV was 17.3 $\pm$ 19.9 years. For patients on biologic treatment for IBD, the mean length of time between initiation of biologic and onset of IgAV was 3.3 $\pm$ 3.8 years (range 0-12 years). Active IBD at IgAV-onset was present in 56% (5/9) of patients.

Tumor necrosis factor inhibitors (TNFi) were the most frequent biologics used for IBD (8, 89%); infliximab was the most common (7, 78%). At IgAV-onset, only 5 patients were on treatment with TNFi; two subsequently discontinued, two switched to another TNFi (adalimumab), and one patient continued. At last follow-up, two of three patients that remained on TNFi had full resolution of IgAV despite ongoing TNFi use. Comparison of baseline characteristics between cases with IBD-IgAV and matched non-IBD IgAV controls is demonstrated in Table 1. No differences were seen in regards to development of end-stage renal disease, resolution of hematuria and/or proteinuria, time to complete IgAV response or first IgAV relapse.

Conclusion: Baseline characteristics and outcomes of patients with IBD-IgAV are similar to those with IgAV without IBD. Development of IgAV is not limited to patients with clinically active IBD. Whether use of TNFi is related to the pathogenesis of IgAV in some patients with IBD remains unclear. Further research into the pathophysiologic connection between IBD and IgAV is needed.

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Table 1: Baseline characteristics of patients with inflammatory bowel disease and immunoglobulin-A vasculitis compared to patients with IgAV without inflammatory bowel disease

Characteristic	IBD-IgAV (n=9)	Non-IBD IgAV (n=18)	p-value
Age at IgAV diagnosis, years*	40.8 $\pm$ 17.5	40.7 $\pm$ 17.4	0.96
Male sex	6 (67%)	12 (67%)	1.00
Length of follow-up, years*	1.3 $\pm$ 2.6	1.1 $\pm$ 1.2	---
Creatinine, mg/dL*	1.0 $\pm$ 0.4	0.9 $\pm$ 0.3	0.59
eGFR (ml/min/1.73m ²)*	91.4 $\pm$ 31.6	96.4 $\pm$ 22.9	0.72
C-reactive protein, mg/L*	23.3 $\pm$ 12.2	27.0 $\pm$ 27.4	0.62
Renal involvement	7 (78%)	10 (56%)	0.26
Microscopic hematuria	5 (56%)	7 (39%)	0.41
Proteinuria	7 (78%)	8 (44%)	0.10
Palpable purpura	9 (100%)	18 (100%)	1.00
Abdominal Ischemic symptoms	3 (33%)	2 (11%)	0.161
Disease status at IgAV diagnosis			
Active IBD	5 (56%)	-	-
TNF- α inhibitor at diagnosis	2/5 (40%)	-	-
o Ever TNF- α inhibitor	5/5 (100%)	-	-
Inactive IBD	4 (44%)	-	-
TNF- α inhibitor at diagnosis	3/4 (75%)	-	-
o Ever TNF- α inhibitor	3/4 (75%)	-	-

IBD, inflammatory bowel disease; IgAV, immunoglobulin-A vasculitis; eGFR, estimated glomerular filtration rate; TNF- α , tumor necrosis factor- α inhibitor.

*Mean $\pm$ standard deviation

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SAT0250 MUSCLE INVOLVEMENT REVEALED BY 18F-PET-CT IN POLYMYALGIA RHEUMATICA

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Background: Imaging techniques in polymyalgia rheumatica (PMR) have revealed mainly bursitis, tenosynovitis, capsulitis and enthesitis. This is also the case for 18F-FDG-PET-CT. This technique, which has shown its interest in the diagnosis of PMR, allows an assessment of the metabolic activity of the entire musculoskeletal system and in particular of muscle structures.

Objectives: The objective of this work was to evaluate muscle damage using 18F-FDG-PET-CT in PMR.

Methods: This is a retrospective study including patients with PMR (ACR/EULAR 2012 criteria) seen in our department, who had an 18F-FDG PET-CT examination between September 2012 and November 2018. A control group consisting of subjects without rheumatological manifestations who had such an examination as part of neoplastic research or neoplastic disease control was also evaluated. PET evaluation included 17 sites suggestive of PMR, as previously reported [1], leading to a global PET score ranging from 0 to 51. Muscle hypermetabolism areas were similarly rated according to the same Goerres classification [2] (0 = no uptake; 1 = slight uptake, less than liver; 2 = uptake like liver; 3 = uptake higher than liver). Muscle activity sites have been identified. A comparison of PMR patients with and without muscle involvement was performed using the Mann Whitney or Fisher's exact test.