The mean time from onset of GCA to intracranial involvement was 17 months (± 38). All patients had neurologic symptoms, 33% (n:3) had a stroke and 22% (n:2) had a transient ischemic attack. IC-GCA was diagnosed by cranial imaging in 8 patients and by autopsy in one. Cranial imaging modalities used included magnetic resonance angiography (n=8), CT angiography (n=2) and cerebral angiography (n=2). Intracranial vasculitis most commonly affected the internal carotid artery 78% (N=7) followed by the, vertebral artery 56% (n=5), posterior cerebral artery 56% (n=5), middle cerebral artery 44% (n=4), anterior cerebral artery 33% (n=3) and posterior inferior cerebral artery 11% (n=1). Stenosis was present in 89%, occlusion in 33%, dilatation in 11%, and wall thickening in 11%. All patients received glucocorticoids. For treatment of intracranial disease, additional agents included: cyclophosphamide (67%), Tocilizumab (22%). Despite treatment, outcomes for patients with IC-GCA were poor. Five of nine patients died during a mean length of follow-up of 2.4 months. **Conclusion:** Although rare, IC-GCA is associated with significant morbidity and mortality. It occurs predominantly in men, presenting with ischemic cerebrovascular events. Current treatment strategies appear to be of limited efficacy for IC-GCA.

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Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2019-eular.3548

SAT0231 AGE AND THE CLINICAL FEATURES OF ADULT IGA VASCULITIS

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Background: IgA vasculitis (IgAV) is a common vasculitis of adult population, yet the disease in adults is still poorly defined.
Objectives: The aim of our study was to evaluate the potential influence of age on the presentation of adult IgAV.
Methods: We analyzed medical records of adult, histologically proven IgAV cases, diagnosed and followed at our secondary/tertiary rheumatology center between January 2010 and December 2018. We stratified IgAV cases into four groups, based on the patient age at presentation (18-39, 40-59, 60-79 and ≥ 80 years). The clinical features were compared between the groups.
Results: During the 108-month observation period we identified 265 new IgAV cases (60.0% males, median (IQR) age 64 (45–77) years). Skin, joint, gastrointestinal tract (GIT) and renal involvement developed in 265, 103 (38.9%), 80 (30.2%), and 118 (44.5%) cases, respectively. Younger adults (≤ 40 years) more frequently reported an infection prior to IgAV, and developed arthritis or GIT involvement. Severe renal involvement was less common under the age of 40 years. The incidence of skin limited IgAV and of necrotic purpura increased with patient age. Follow-up data were available for 189 patients. During a median (IQR) follow-up of 12 (6–24) months, 29 (15.3%) patients relapsed. Relapsing disease was more frequent in younger adults (≤ 40 years), compared to older ones.

Table 1. Age and clinical features of adult IgAV

Clinical characteristics	Age interval			
	18-39	40-59	60-79	≥ 80
Number of cases	49	69	96	51
Male gender (%)	63.3	68.1	62.5	41.2
Prior infection (%)	57.1	36.2	28.1	21.6
Generalized purpura (%)	49.0	52.2	50.0	45.1
Skin necroses (%)	14.3	43.5	55.2	62.7
Skin limited IgAV	16.3	18.8	35.4	41.2
Joint involvement (%)	63.3	49.3	30.2	17.6
Arthritis (%)	28.6	14.5	14.6	2.0
GIT involvement (%)	42.9	34.8	21.9	27.5
Severe GIT involv. (%)	14.3	7.2	7.3	2.0
Renal involvement (%)	36.7	43.5	50.0	43.1
Severe renal involv. (%)	2.0	15.9	14.6	11.8
Elevated serum IgA	22.6	45.6	56.5	48.6
Relapses (% and number)	25.0 (9/36)	13.2 (7/53)	18.6 (13/70)	0 (0/30)

Legend: Generalized purpura: purpura extending above the waistline; GIT: gastrointestinal tract; severe GIT involvement: bloody diarrhea or ileus or bowel perforation; severe renal involvement: nephritic syndrome with acute renal failure or nephrotic syndrome;

Data on age related variations in the clinical presentation of IgAV are presented in Table 1.
Conclusion: We found subtle age related differences in the presentation and during follow up of adult IgAV.
Disclosure of Interests: : None declared
DOI: 10.1136/annrheumdis-2019-eular.2155

SAT0232 DISTRIBUTION OF MACROPHAGE SUBSETS IN TEMPORAL ARTERY BIOPSIES OF PATIENTS WITH GIANT CELL ARTERITIS

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Background: Giant cell arteritis (GCA) is a granulomatous vasculitis characterized by recruitment of T-cells and monocytes to the vessel wall. Upon recruitment, monocytes are activated and differentiate into macrophages. These lesional macrophages take on different phenotypes depending on cues from the microenvironment. These macrophages are known to produce pro-inflammatory cytokines, chemokines, matrix-metalloproteinases (MMPs), and growth factors contributing to amplification of the inflammatory response, vessel wall injury and remodeling. However, current knowledge regarding macrophage subsets in GCA lesions is limited.
Objectives: To examine the distribution of macrophage subsets in GCA lesions.
Methods: Immunohistochemistry was performed on consecutive sections of paraffin-embedded temporal arteries of biopsy-confirmed GCA patients (n = 11) with antibodies to selected markers of inflammation and tissue remodeling (Table 1). Positive cells were scored in three layers of the vessel wall (adventitia, media-intima, inner-intima) using a semi-quantitative scoring system. Expression by macrophages was confirmed by double staining with the macrophage transcription factor PU.1.

Table 1. Immunohistochemistry panel. AB019

Pro-inflammatory	Tissue remodeling
IL-6	CD206 (MR)
IL-1 β	IL-10
IL-12	FR- β
IL-23	MMP-12
CD64	MMP-9
	MMP-2

Results: The pro-inflammatory marker CD64 and pro-inflammatory cytokines (IL-12, IL-23 and IL-1 β) were strongly expressed by macrophages throughout the vessel wall, most prominently in the adventitia. However, subsets of these CD64+ macrophages also showed concomitant expression of the tissue remodelling markers CD206 and FR- β in specific compartments of the lesions. More specifically, CD206+ macrophages were mainly found along the media borders which co-localized with positive MMP-9 staining. Interestingly, FR- β positivity was also found in the inner-intima of TABs with a high degree of intimal hyperplasia. FR- β expression was significantly higher in the inner-intima region in TABs with

Figure 1. Spatial distribution of macrophage subsets in temporal artery of GCA affected vessel.