**Background:** Temporal arteritis is the most common systemic vasculitis in patients aged >50 years, the most serious complications of which is visual loss. The arterial biopsy is the diagnostic gold standard; alongside the classic finding of transmural infiltrate and giant cells, other abnormalities have been described, of which it is not yet known whether they identify specific clinical subsets. PET is not yet used for diagnosis, but it can be suggestive in patients with high clinical suspect; it may be useful for assessing the extent of the disease in already diagnosed patients and for ruling out alternative diagnoses as infections and neoplasms. More recently, PET has been used to assess disease metabolic activity.

**Objectives:** The aim of our study is to evaluate, in patients with histologically confirmed temporal arteritis, correlations between pathological subsets, metabolic activity and different clinical behavior.

**Methods:** We have recovered the medical records of patients with the diagnosis of temporal arteritis made in our Rheumatology service from January 2007 until now. We selected those satisfying the ACR 1990 criteria and, finally, those with a positive biopsy. We analyzed age at onset of symptoms, diagnostic delay, presence of PMR, fever, constitutional symptoms, headache, temporal artery induration, visual loss; we analyzed CRP, ESR, plasmatic Hb, PLT count, hypococomplementemia, ANA, aC1, ANCA. The referring pathologist, who didn't know the history of patients, re-examined all the biopsies performed, focusing on: transmural, perivascular, limited to small vessels or vasa vasorum infiltrate, presence of giant cells, macrophages, eosinophils, neutrophils, lymphocytes, plasmacells, stenosis, thrombosis, fibrinoid necrosis. The nuclear doctor retrieved the PET images by re-assigning each patient the relative PET VAS score. Descriptive analysis was performed: absolute and percentage frequencies were calculated for categorical variables and mean, standard deviation, range and percentiles for quantitative variables. The relationship between variables was tested by the chi-square test and the Mann-Whitney rank test. All analyses were performed with the STATA software 14.2.

**Results:** We analyzed 46 patients (F 32, M 14), average age at onset of symptoms of 75.75 yrs and average diagnostic delay of 3.2 months. Headache was reported by 37 patients, fever and asthenia by 26 and 24 respectively, jaw claudication and PMR by 18 and 7. 11 patients had visual loss while 4 and 2 respectively reported amaurosis and diplopia. Temporal artery induration was described in 24 patients. Tests of systemic inflammation were abnormal (mean CRP 85 mg/L, ESR 72.7 mm /1h); the finding of hypococomplementemia (1/26 patients), ANA, aC1 and ANCA positivity (2/31, 1/27 and 1/25) was negligible. Histological analysis showed the prevalence of transmural infiltrate (100% of patients), giant cells (67%) and lymphocytes/macrophages (85% both); small vessel vasculitis (59%) and the presence of plasma cells (53%), neutrophils (48%) and eosinophils (22%) were less represented. Perivascular infiltrate was described in 29% of patients, with negligibly of vascuLTis limited to the adventitia and of the vasa vasorum (2 and 0/46). Intimal hyperplasia and necrosis (22 and 14/46) prevailed of transmural infiltrate and giant cells, the presence of lymphocytes, macrophages and small vessel vasculitis. No correlation was observed between clinical findings, biological and metabolic activity, apart from the increased presence of giant cells in PET positive patients. Visual loss is slightly more common in PET negative patients. This may be due to severity of the cases which needed early steroid treatment.

**Disclosure of Interests:** Francesco Girelli: None declared, Silvia Asiolli: None declared, Riccardo Galassi: None declared, Daniela Tirotta: None declared, Chiara Bellini: None declared, Simone Bernardi Paid instructor for: Paid instructor for Pharmaceuticals in 2013, Lucia Gardelli: None declared, Linda Petriti: None declared, Elisabetta Fabbrini: None declared, Paolo Muratori: None declared.

**DOI:** 10.1136/annrheumdis-2022-eular.3247

---

**Table 1. Optimal IMT cut-off values for cranial and extracranial arteries**

<table>
<thead>
<tr>
<th>Artery</th>
<th>Side</th>
<th>Patients without GCA</th>
<th>Patients with GCA</th>
<th>Cut-off (mm)</th>
<th>AUC (CI 95%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common temporal artery mm, mean (SD)</td>
<td>Right</td>
<td>0.33 (0.06)</td>
<td>0.68 (0.28)</td>
<td>0.43</td>
<td>0.997 (0.988 - 1)</td>
<td>100</td>
<td>97.1</td>
</tr>
<tr>
<td>temporal artery mm, mean (SD)</td>
<td>Left</td>
<td>0.35 (0.11)</td>
<td>0.57 (0.21)</td>
<td>0.45</td>
<td>0.966 (0.905 - 1)</td>
<td>92.3</td>
<td></td>
</tr>
<tr>
<td>Frontal branch mm, mean (SD)</td>
<td>Both</td>
<td>0.34 (0.08)</td>
<td>0.63 (0.25)</td>
<td>0.44</td>
<td>0.984 (0.959 - 1)</td>
<td>94.7</td>
<td>95.1</td>
</tr>
<tr>
<td>Parietal branch mm, mean (SD)</td>
<td>Left</td>
<td>0.27 (0.05)</td>
<td>0.41 (0.18)</td>
<td>0.34</td>
<td>0.985 (0.962 - 1)</td>
<td>96.1</td>
<td></td>
</tr>
<tr>
<td>Common carotid mm, mean (SD)</td>
<td>Right</td>
<td>0.27 (0.05)</td>
<td>0.43 (0.18)</td>
<td>0.34</td>
<td>0.989 (0.976 - 1)</td>
<td>96.6</td>
<td></td>
</tr>
<tr>
<td>Subclavian mm, mean (SD)</td>
<td>Left</td>
<td>0.27 (0.05)</td>
<td>0.41 (0.16)</td>
<td>0.36</td>
<td>0.987 (0.976 - 1)</td>
<td>97.6</td>
<td></td>
</tr>
<tr>
<td>Axillary mm, mean (SD)</td>
<td>Left</td>
<td>0.27 (0.05)</td>
<td>0.42 (0.17)</td>
<td>0.36</td>
<td>0.991 (0.980 - 1)</td>
<td>98.3</td>
<td></td>
</tr>
<tr>
<td>Common carotid mm, mean (SD)</td>
<td>Both</td>
<td>0.86 (0.17)</td>
<td>0.89 (0.29)</td>
<td>1</td>
<td>0.974 (0.949 - 0.999)</td>
<td>90.3</td>
<td>92.6</td>
</tr>
<tr>
<td>Subclavian mm, mean (SD)</td>
<td>Left</td>
<td>0.82 (0.15)</td>
<td>1 (0.42)</td>
<td>1.2</td>
<td>0.982 (0.961 - 1)</td>
<td>90.9</td>
<td>96.2</td>
</tr>
<tr>
<td>Axillary mm, mean (SD)</td>
<td>Both</td>
<td>0.81 (0.16)</td>
<td>0.96 (0.36)</td>
<td>1.1</td>
<td>0.977 (0.961 - 0.993)</td>
<td>90</td>
<td>94</td>
</tr>
<tr>
<td>Common carotid mm, mean (SD)</td>
<td>Both</td>
<td>0.76 (0.18)</td>
<td>0.99 (0.44)</td>
<td>1</td>
<td>0.967 (0.970 - 1)</td>
<td>100</td>
<td>99.3</td>
</tr>
<tr>
<td>Subclavian mm, mean (SD)</td>
<td>Left</td>
<td>0.67 (0.17)</td>
<td>0.93 (0.35)</td>
<td>1.1</td>
<td>0.991 (0.975 - 1)</td>
<td>100</td>
<td>98.3</td>
</tr>
<tr>
<td>Axillary mm, mean (SD)</td>
<td>Both</td>
<td>0.69 (0.16)</td>
<td>0.99 (0.49)</td>
<td>1</td>
<td>0.998 (0.995 - 1)</td>
<td>100</td>
<td>99.3</td>
</tr>
<tr>
<td>Common carotid mm, mean (SD)</td>
<td>Left</td>
<td>0.67 (0.17)</td>
<td>0.99 (0.49)</td>
<td>1</td>
<td>0.998 (0.995 - 1)</td>
<td>100</td>
<td>99.3</td>
</tr>
</tbody>
</table>

**Discussion:** Ultrasound (US) is a valid imaging tool to detect signs of giant cell arteritis (GCA). Although the halo sign has always been considered the most useful finding for GCA diagnosis, modern high frequency transducers are able to precisely measure the intima-media thickness (IMT) of cranial and extracranial arteries. However, data on optimal cut-off values for IMT to differentiate patients and controls in clinical practice are limited.

**Objectives:** To determine the optimal cut-off value for IMT of cranial and extracranial arteries in patients with suspected GCA.

**Methods:** This is a retrospective observational study of patients referred to our US fast-track clinic with suspected GCA. All patients underwent bilateral US examination of the cranial and extracranial (carotid, subclavian and axillary) arteries within 24 hours per protocol. The exam was performed using an Esacote MyLabb with a 12-18 MHz frequency transducer for cranial arteries and an 8-14 frequency transducer for extracranial arteries. The IMT was measured in gray scale mode and the presence of a non-compressible halo sign was checked in all arteries. The gold standard for GCA diagnosis was clinical confirmation by the referring rheumatologist after 6 months follow-up. Mean IMT values of each artery were compared between patients with and without GCA by independent samples T-Test. Receiver operating characteristics analysis was performed and the Youden index was used to determine the optimal cut-off value for IMT of each artery.

**Results:** Of the 157 patients with suspected GCA (67.5% female, mean age 73.7 years) referred to the fast-track clinic, 47 (29.9%) had GCA clinical confirmation after 6 months. 41 (87.2%) patients with GCA had positive US findings (61.7% had cranial involvement, 44.7% extracranial involvement and 19.1% a mixed pattern of cranial and extracranial arteries). The following IMT cut-off values showed the highest diagnostic accuracy: 0.44mm for the common superficial temporal artery; 0.34 mm for the frontal branch; 0.36 mm for the parietal branch; 1.1 mm for the carotid artery; 1 mm for the subclavian and axillary arteries. The area under the ROC curve of the IMT for a clinical diagnosis of GCA was 0.984 (95% CI 0.999 - 1) for common superficial temporal artery, 0.989 (95% CI 0.978 - 1) for frontal branch, 0.991 (95% CI 0.980 - 1) for parietal branch, 0.977 (95% CI 0.961 – 0.993) for carotid, 0.99 (95% CI 0.979 - 1) for subclavian and 0.996 (95% CI 0.991 - 1) for axillary arteries (Table 1).
Conclusion: Different IMT cut-off values for each artery are necessary to establish a correct US diagnosis of GCA. These proposed IMT cut-off values may help to improve the diagnostic accuracy of US in clinical practice.

Disclosure of Interests: None declared


AB0595 INFANTILE TAKAYASU: CLINICAL FEATURES AND LONG TERM OUTCOME

A Miller Barmak1,2, F Sztajinbok3, S Özen4, Z Balik4, A Borzutzky3, L Fogel5, G Goldberg2, Y Butbul2,3,4, 5Rambam Medical Center, Pediatrics B, Haifa, Israel; 5Rambam Medical Center, Rheumatology Unit, Haifa, Israel; 3Federal University of Rio de Janeiro, Pediatric Rheumatology Unit, Rio de Janeiro, Brazil; 4Hacettepe University, Pediatric Rheumatology, Ankara, Turkey; 2Pontifical Catholic University of Chile, Department of Pediatric Infectious Diseases and Immunology, Santiago, Chile; 2Washington University in St. Louis, Pediatrics, St. Louis, United States of America; 3Kaplan Medical Center, Pediatric Rheumatology Unit, Rehovot, Israel; 4Rambam Medical Center, Pediatric Rheumatology Unit, Haifa, Israel; 5Technion - Israel Institute of Technology, The Ruth and Bruce Rappaport Faculty of Medicine, Haifa, Israel.

Background: Takayasu arteritis (TA) is a large vessel vasculitis rarely reported in children, and its incidence is extremely low in infants. Most articles on pediatric TA have not focused on infants. We present the largest case series of infantile TA aiming to characterize demographic and clinical data and compare it with existing data on older children.

Objectives: Characterize demographic and clinical data regarding TA and compare it with existing data on older children.

Methods: We conducted an international multi-center retrospective cohort study. Epidemiological and clinical data were collected from patient charts by doctors from six centers.

Results: Twelve patients (50% female) meeting the ACR criteria of TA were included. Median age of symptom onset was 11 months, with a diagnostic delay of 4 months and median time of follow up of 7.5 years. The most common symptoms at presentation were hypertension, BP difference between upper and lower limbs, and fever. The arteries most commonly involved at diagnosis were the abdominal aorta, renal artery, and superior mesenteric artery. Different medications used included steroids, conventional and biological DMARDs, and immunosuppressive therapies. Half of the patients received biologic agents of which infliximab had the highest complete remission rate (40%). Other medications resulting in complete remission were cyclophosphamide (40%) and methotrexate (38%). Invasive procedures were needed in 58% of patients. The most common complications were cardiac (50%), strokes (42%) and serious infections (33%). None of the patients died.

Conclusion: This study presents the largest series of infantile TA. Compared to reported series on older children, infants with TA were more likely to receive biologic agents, develop complications and require invasive interventions.

Disclosure of Interests: None declared


AB0596 AORTIC MANIFESTATIONS IN GIANT CELL ARTERITIS: SINGLE CENTRE 10-YEAR EXPERIENCE

F Coath1, A Sharma2, R Enshad3, J Mo3, J Davies2, D Basquita3, 1Southend University Hospital, Mid and South Essex NHS Foundation Trust, Rheumatology, Southend-on-Sea, United Kingdom; 3Southend University Hospital, Mid and South Essex NHS Foundation Trust, Cardiology, Southend-on-Sea, United Kingdom; 2Southend University Hospital, Mid and South Essex NHS Foundation Trust, Radiology, Southend-on-Sea, United Kingdom.

Background: Disease stratification in GCA is an urgent need, with patients categorised into cranial and large- vessel GCA (LV-GCA) subgroups. LV-GCA may have worse outcomes than GCA. Vascular disease, poor response to glucocorticoids (GC) and aortic involvement.

Objectives: We report a single centre experience using clinical, imaging and treatment outcomes from a specialist clinic.

Methods: 134 patients with LV-GCA were identified over a 10-year period at Southend University Hospital (2012-2022). Medical records were reviewed retrospectively for baseline demographics, clinical presentation, inflammatory markers, imaging (vascular ultrasound, PET-CT, echocardiography), vascular damage and treatment.

Results: There was a female predominance (female n=91). Age at presentation ranged from 46 to 86 years (median 70 years). Co-morbidities implicated in aortic disease included hypertension (n=60), hypercholesterolaemia (n=29), diabetes (n=14), aortic valve disease (n=5) and aortic coarctation (n=2), including coronary artery and carotid artery disease (n=19). Constitutional disturbance was most frequently observed presentation (70%, n=94), and the only feature for 11 patients. This was followed by cranial symptoms (62%, n=83), polymyalgia (53%, n=71), ischaemic symptoms i.e., visual disturbance or tongue/jaw claudication (24%, n=32) and cardiovascular presentations (7%, n=9). The latter included limb claudication, stroke, and aortic aneurysm. Although LV-GCA refers to extra-cranial disease, 12% patients (9%) had isolated cranial and/or ischaemic symptoms at initial presentation.

Inflammatory markers were typically elevated at presentation, C-reactive protein ranged from 1-425mg/L and ESR 1-130mm. Vascular ultrasound was used at diagnosis in 93 patients, with positive temporal arterial findings in 50% (n=38) and positive axillary findings in 75% (n=57). PET-CT data was available for 125 patients, of which 113 were positive for LV-GCA. The posterior auricular artery was seen in 77%, with 7 ascending and 1 abdominal aortic aneurysm observed. Transthoracic echocardiogram was available for 46% (n=62). Four (6.5%) patients had a dilated aortic root when indexed to height as per British Society of Echocardiography (BSE) guidelines SOV (mm/m^3) > 21.8mm in males and > 20.7mm in females). Values for our patients were 22.6 and 21.2mm/m^3 for the female patients and 29.2 and 25.2mm/m^3 for the male patients. Furthermore, 32 patients showed some extent of diastolic dysfunction as per BSE criteria (52%). All patients received GC as part of their treatment, 60% (n=82) needing one or more DMARDs and 17% (n=23) Tocilizumab for relapsing disease. DMARDs used included Leflunomide (n=63), Methotrexate (n=16), Mycophenolate mofetil (n=5) and Azathioprine (n=1]. One patient received cyclophosphamide.

Conclusion: By combined imaging modalities, 11 patients (8%) had evidence of ascending aortic damage. Grade 1 diastolic dysfunction can be age related, so this may be association rather than causation. Over half of patients had not undergone echocardiogram evaluation, so there may be a hidden burden of disease. Many patients required GC-sparing therapy, showing GC alone are often not enough to halt disease progression, and vascular damage was relatively reduced compared to historical reports. The authors feel GCA services should include standardised protocols for early DMARDs, continuing thorough assessment for LV-GCA and vascular damage, including echocardiography, progressing to cross-sectional imaging if indicated.

References:

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2022-eular.3598

AB0597 FDG-PET/CT IN THE DIAGNOSE AND FOLLOW UP OF TAKAYASU VASCUITIS

B. Gudbrandsson1, O. Palm1, O. Molberg1, H. Gilvet1, M. Revheim1, 1Oslo University Hospital, Rheumatology, Oslo, Norway; 2Rikshospitalet, Rheumatology, Oslo, Norway; 3Oslo University Hospital, Division of Radiology and Nuclear Medicine, Oslo, Norway; 4Rikshospitalet, Division of Radiology and Nuclear Medicine, Oslo, Norway.

Background: Takayasu vasculitis (TAK) is a chronic disease, where clinical and serological markers as CRP/ESR may fail to predict development of new vascular lesions in the disease course (1). Similarly, 18F-fluorodeoxyglucose positron emission tomography/computed tomography (FDG-PET/CT) studies show conflicting results on the association between vessel uptake of FDG and clinical and laboratory finding. A study on new FDG-PET activity scoring system, PETVAS was newly published but has not been validated in other cohorts (2). To date there are limited data on FDG-PET/CT finding at time of diagnoses before treatment induction and 18-FDG uptake and development of new stenosis during follow up.

Objectives: The goal of this study was to see; 1) FDG-PET/CT uptake in newly diagnosed patients before any treatment start 2) FDG-PET/CT uptake and development of new vascular lesions during follow up magnetic resonance angiography (MRA) 3) assess PETVAS score before and after treatment induction. Methods: All patients in a population-based TAK cohort with FDG-PET/CT at the time of diagnosis and treatment induction were identified. Disease activity was assessed with the NIH activity score (1). Patients had to have clinical, laboratory and MR-angiography prior to/or right after FDG-PET/CT and a minimum of one follow up MRA. The clinical report from the FDG-PET/CT finding at time of diagnoses before treatment induction and 18-FDG uptake and development of new stenosis during follow up.

Disclosure of Interests: None declared
