References:

- [1] Jennette JC, Xiao H, Hu P. Complement in ANCA-Associated Vasculitis. *Seminars in Nephrology* 2013; 33 (6): 557 – 564
- [2] Schreiber A, Xiao H, Jennette JC et al. C5a receptor mediates neutrophil activation and ANCA-induced glomerulonephritis. *J Am Soc Nephrol* 2009; 20(2): 289-298.
- [3] Gou SJ, Yuan J, Chen M et al. Circulating complement activation in patients with anti-neutrophil cytoplasmic antibody-associated vasculitis. *Kidney Int* 2013; 83(1): 129-137.

Disclosure of Interests: Anastasiia Zykova Grant/research support from: This work was supported by the 5-100 Project, Sechenov University, Moscow, Pavel Novikov Grant/research support from: This work was supported by the 5-100 Project, Sechenov University, Moscow, Nikolai Bulanov Grant/research support from: This work was supported by the 5-100 Project, Sechenov University, Moscow, Andreas Kronbichler Grant/research support from: This work was supported by unrestricted grant from the Austrian Society of Rheumatology., Evgeny Gitel: None declared, Oxana Novikova: None declared, Mayra Bulanov: None declared, Elizaveta Safonova: None declared, Sergey Moiseev Grant/research support from: This work was supported by the 5-100 Project, Sechenov University, Moscow

DOI: 10.1136/annrheumdis-2020-eular.4733

SAT0269

TAKAYASU ARTERITIS AND INFLAMMATORY BOWEL DISEASE. COEXISTENCE AMONG NORWEGIAN PATIENTS

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Background: Takayasu arteritis (TAK) and inflammatory bowel disease (IBD), consisting of ulcerative colitis (UC) and Crohn's disease (CD), are uncommon diseases. The incidence rates of TAK is 22.0/million and IBD 193/million in Norway (1, 2). UC in patients with TAK has previously been reported with a prevalence between 0.21% and 6.4% and may be explained by shared genetic disposition (3). Epidemiological data are mainly based on one Asian study (3), one Canadian study (4) and case reports.

Objectives: We investigated the coexistence of TAK and IBD and compared clinical characteristics between TAK patients with- and without IBD.

Methods: We analysed the prevalence of TAK with IBD in a population-based cohort of patients based on the Norwegian Systemic connective tissue disease and Vasculitis Registry (NOSVAR). The diagnoses of TAK were based on either 1990 ACR classification criteria (5) or the modified Ishikawa diagnostic criteria (6). The distribution of vasculitis was recorded in accordance with the angiographic classification of the 1994 International TAK Conference in Tokyo (7). The diagnoses of IBD were based on gastroenterological investigations and conclusions recorded in hospital charts.

Results: Among 116 patients with TAK, living in Southern part of Norway (2.5 mill inhabitants) 5 patients (4.3%) had IBD, 3 with UC and 2 with CD. All patients had onset of IBD prior to TAK and intestinal involvement of colon (Table 1). Moreover, patients with TAK and IBD were younger at TAK onset than in those without IBD (Table 2).

Table 1. Patients with the combination of Takayasu arteritis (TAK) and Inflammatory bowel disease (IBD); ulcerative colitis (UC) or Crohn's disease (CD)

Age at disease onset (years)							
Patients	IBD	Gender F/M	IBD extension	IBD TAK	Vascular complication	Distribution of vasculitis (4)	
1	UC	F	Total	12	27	Stenosis and Occlusions	I
2	CD	F	Colon	32	34	Stenosis	V
3	UC	M	Total	11	16	Stenosis and Aneurisms	IIb
4	CD	K	Colon	30	40	Stenosis	I
5	CD	K	Ileocecal+colon	20	22	Stenosis	IIb

Table 2. Comparison of Takayasu arteritis (TAK) with- and without IBD

	TAB/IBD	TAK/non-IBD	p
Number of patients (n%)	5	111	

Table 2. Comparison of Takayasu arteritis (TAK) with- and without IBD

	TAB/IBD	TAK/non-IBD	p
Females (n%)	4	90 (94)	
Age at TAK onset (years)	27	39 (17)	0.043
Disease duration (years)	1.1	1.4	0.46
Arterial distribution (n%)			
	I	2	17 (15)
	IIa	0	30 (27)
	IIb	2	16 (14)
	III	0	0
	IV	0	2 (2)
	V	1	29 (26)
Arterial complications* (n%)			
	Stenosis	5	85 (85)
	Occlusions	1	30/110 (27)
	Aneurisms	1/5 (20)	19/132 (14)
	Carotidynia	2	29 (26)
	Myocardial infarction	1	5/106 (5)
	Deceased (n%)	0	23 (16)

*Based on CTA, MRA or PET/CT.

Conclusion: The presence of IBD in Norwegian patients with TAK is higher than expected by chance. This is comparable to previously reported data from Asian patients (3). Strikingly, onset of IBD preceded TAK in all our cases. The patients with IBD were younger at onset of TAK, but vascular distribution and complication rate did not seem to differ between TAK with and without IBD.

References:

- [1] Gudbrandsson B, Molberg Ø, Garen T, Palm Ø, Arthritis Care Res (Hoboken), 2017 Sept;69:1384-1390
- [2] Moum B, Vatn MH, Ekbohm A, Fausa O, Aadland E, Lygren I, Sauar J, Schulz T, Digestion, 1995;56: 377-81
- [3] Terao C, Matsumura T, Yoshifuji H, Kirino Y et al, Arthritis Rheumatism, 2015; May 67: 2226-32
- [4] Sy A, Khalidi N, Dehghan N, Barra L, Carrette S, Semin Arthritis Rheum. 2016 Feb;45:475-82
- [5] Arend WP, Michel BA, Bloch DA, Hunder GG, Calabrese LH, Edworthy SM, et al. Arthritis Rheum 1990;33:1129–34
- [6] Sharma BK, Jain S, Suri S, Numano F. Diagnostic criteria for Takayasu arteritis. *Int J Cardiol* 1996;54 Suppl:S141–7.
- [7] Hata A, Noda M, Moriaki R, Numano F. Angiographic findings of Takayasu arteritis: new classification. *Int J Cardiol* 1996;54 Suppl:S155–63.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.2034

SAT0270

TOCILIZUMAB IN REFRACTORY TAKAYASU ARTERITIS. OPEN-LABEL NATIONAL MULTICENTER STUDY OF 53 PATIENTS OF CLINICAL PRACTICE

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Background: Tocilizumab (TCZ) was recently approved for Takayasu Arteritis (TAK) in Japan based on the results of the TAKT trial (1). However, data in clinical practice in Europe and America are scarce (2).

Objectives: To assess efficacy and safety of TCZ in TAK of clinical practice in Spain.

Methods: Observational, open-label multicentre study of 53 TAK patients treated with TCZ due to refractoriness or adverse events of previous therapy. Outcomes variables were improvement of clinical features, acute phase reactants and glucocorticoid-sparing effect.

Results: 53 patients (46w/7m); mean age, 40.6±14.6 years at TCZ onset. TCZ was started after a median of 12 [3.0-48.0] months from TAK diagnosis. In addition to systemic corticosteroids and before TCZ they received conventional immunosuppressant drugs (n=42) and biologic therapy (n=14). TCZ was prescribed as standard I.V. (n=42;

79.2%) or subcutaneous (n=11; 20.8%). The initial dose was 8mg/kg/IV/4 weeks or 162mg/SC/week, respectively. TCZ was used in monotherapy or combined with immunosuppressants (n=32; 60.4%): methotrexate (n=27), azathioprine (n=2), cyclosporine (n=3). Main clinical features at TCZ onset were: malaise (n=30), limb claudication (n=22), headache (n=18), fever (n=14), abdominal pain (n=10), and chest pain (n=9). Most of the patients experienced a rapid and maintained clinical, analytical improvement (TABLE). After a median follow-up of 18.0 [7.0-45.0] months, TCZ was discontinued in 20 patients due to: sustained remission (n=6), relapse (n=6), adverse event (n=5), gestation (n=3). Most relevant adverse side effects were serious infections: pneumonia (n=2), herpes zoster (n=1), abdominal sepsis (n=1).

Table.

	Basal (N=53)	Month 1 (N=53)	Month 3 (N=46)	Month 6 (N=44)	Month 12 (N=34)
Clinical improvement, n/N(%)					
Complete		17/53 (32.1)	19/46 (41.3)	23/44 (52.3)	26/34 (76.5)
Partial		30/53 (54.6)	26/46 (56.5)	18/44 (40.9)	8/34 (23.5)
No improvement		6/53 (11.3)	1/46 (2.2)	3/44 (6.8)	0/34 (0.0)
Analytical markers, n/N(%)					
ESR (mm/1 st h), median [IQR]	35.0 [16.0-52.0]	7.5 [3.0-14.0] *	3.5 [2.0-8.0] *	5.0 [2.0-6.0] *	5.0 [2.0-8.5] *
CRP (mg/dL), median [IQR]	1.7 [0.6-3.5]	0.21 [0.05-0.6] *	0.14 [0.05-0.5] *	0.14 [0.04-0.4] *	0.10 [0.03-0.30] *
Hb (g/dL), mean ± SD	12.3±1.5	12.8±1.2*	12.9±1.3*	12.9±1.4*	12.9±1.4*
Prednisone dose (mg/ day), median [IQR]	30.0 [15.0-50.0]	20.0 [10.0-37.5] *	10.0 [5.0-20.0] *	5.0 [5.0-12.5] *	5.0 [0.0-7.5] *

*Wilcoxon test p < 0.001.

Conclusion: TCZ appears to be effective and safe in patients with refractory TAK in clinical practice.

References:

- 1 Nakaoka Y et al. Ann Rheum Dis. 2018;77:348-354
- 2 Loricera J et al. Clin Exp Rheumatol. 2016; 34: S44-53.

Disclosure of Interests: D. Prieto-Peña: None declared, Monica Calderón-Goercke: None declared, Pilar Bernabéu: None declared, Paloma Vela-Casasempere: None declared, J. Narváez: None declared, Carlos Fernández-López: None declared, Mercedes Freire González: None declared, Beatriz González-Alvarez: None declared, Roser Solans-Laqué: None declared, Jose Luis Callejas-Rubio: None declared, Norberto Ortego: None declared, Carlos Fernández-Díaz Speakers bureau: Bristol Myers Squibb, Esteban Rubio Romero: None declared, SALVADOR GARCÍA MORILLO: None declared, Mauricio Minguez: None declared, Cristina Fernández-Carballido Consultant of: Yes, I have received fees for scientific advice (Abbvie, Celgene, Janssen, Lilly and Novartis), Speakers bureau: Yes, I have received fees as a speaker (Abbvie, Celgene, Janssen, Lilly, MSD, Novartis), Eugenio de Miguel Grant/research support from: Yes (Abbvie, Novartis, Pfizer), Consultant of: Yes (Abbvie, Novartis, Pfizer), Paid instructor for: yes (AbbVie, Novartis, Pfizer, MSD, BMS, UCB, Roche, Grunental, Janssen, Sanofi), Speakers bureau: yes (AbbVie, Novartis, Pfizer, MSD, BMS, UCB, Roche, Grunental, Janssen, Sanofi), Sheila Melchor: None declared, Eva Salgado-Pérez: None declared, Beatriz Bravo: None declared, Susana Romero-Yuste: None declared, J. Salvatierra: None declared, Cristina Hidalgo: None declared, Sara Manrique Arja: None declared, C. Romero-Gómez: None declared, Patricia Moya: None declared, Noelia Alvarez-Rivas: None declared, Javier Mendizabal: None declared, Francisco Miguel Ortiz Sanjuan: None declared, I. Pérez de Pedro: None declared, Javier Loricera: None declared, Santos Castañeda: None declared, Miguel A González-Gay Grant/research support from: Pfizer, Abbvie, MSD, Speakers bureau: Pfizer, Abbvie, MSD, Ricardo Blanco Grant/research support from: AbbVie, MSD, Roche, Consultant of: Abbvie, Eli Lilly, Pfizer, Roche, Bristol-Myers, Janssen, UCB Pharma and MSD, Speakers bureau: Abbvie, Eli Lilly, Pfizer, Roche, Bristol-Myers, Janssen, UCB Pharma. MSD

DOI: 10.1136/annrheumdis-2020-eular.2445

SAT0271

THE "GOLD STANDARD" DIAGNOSTIC TEST FOR GCA IS THE WHOLE GCA FAST TRACK PATHWAY COMBINED

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Background: We have been developing a rheumatologist-led ultrasound driven giant cell arteritis (GCA) fast-track pathway (FTP), which in year 3 had the following structure:

1. **Rapid access to rheumatology assessment (RAS)** to establish clinical probability of GCA (CP-GCA). No referral criteria required
2. **Temporal and axillary artery ultrasound (TAUS)** if GCA not excluded with RAS. TAUS considered positive if bilateral (halo score $\geq 2/8^{1,2}$ at >1 temporal artery)
3. **Second test in selected patients:** GCA diagnosed in those with mod-high CP-GCA and +ve TAUS and excluded in those with low CP-GCA and -ve TAUS. All others had biopsy (TAB) or large vessel imaging (LVI), presentation depending
4. **Protocolised withdrawal of prednisolone:** Patients only treated for GCA if ≥ 1 of: high CP-GCA, +ve TAUS, TAB or LVI
5. **Rapid access** if symptoms recurred on steroid withdrawal for RAS + TAUS

Objectives:

- To compare security of GCA diagnosis, sight loss rate and TAB rate in Year 3 to previous years
- To assess sensitivity and specificity of all components of the Yr 3 FTP for diagnosis of GCA

Methods: As in Yr 2, TAUS was performed by VQ with an Esaote Mylab7, 6-15MHz probe for axillaries, 22MHz for temporal arteries (TAs). In Yr 1 VQ used a GES8 with ML6-15 for axillaries, 18MHz probe for TAs. Year 3 audit data were compared to previous audits

Results:

Year 3 Luton GCA FTP compared to previous years

	Traditional Model	Year 1	Year 2	Year 3
Audit period	2015 12 months	1/1/16-31/3/17 15 months	1/4/17-31/3/18 12 months	1/4/18-31/3/19 12 months
Pathway structure	No rapid access to RAS TAB, no TAUS service	Patients seen ad-hoc for RAS + TAUS TAB requested in all appropriate cases to compare TAUS to TAB	Rapid access for RAS + TAUS, 2 slots/wk 2nd test in selected patients	Rapid access for RAS + TAUS, 3 slots /wk 2nd test in selected patients Protocolised withdrawal prednisolone
No. referrals with suspected GCA/yr	NK	50.4	70	90
Cases GCA /yr	9	24	18	25
Cases GCA excluded /yr	NK	26.4	52	65
% GCA patients with imaging/TAB +ve GCA	44.4	63.3	83.3	92
% GCA patients with clinical GCA (high CP-GCA, no +ve test)	55.6	36.7	16.6	8
% referred where GCA excluded on clinical grounds alone	NK	11.1	5.7	5.6
% referred where TAUS performed and TAB avoided	NA	NA	38.6	50.0
% referred who had TAB	NK (total 27)	84.1	42.8	31.1
% referred where TAUS, then LVI performed instead of TAB	NA	4.8	12.8	13.3
Mean days on prednisolone for GCA before TAUS (median, range)	NA	10.6 (7, 0-81)	4.4 (2, 0-54)	5.9 (3, 0-57)
% GCA patients with permanent sight loss due to GCA	33	23.3	11.2	8

NA: Not applicable NK: not known