was the worst prognostic phenotype of LV-GCA. Extent of LVLs by imaging should be considered when determining the treatment strategy for GCA.

REFERENCES:

[1] Sugihara T, et al. *Arthritis Res Ther*. 2020;22(1):72

Acknowledgements: The authors would like to acknowledge Mitsuki Isobe (Sakakibara Heart Institute), Yoshihiro Arimura (Kichijoji Asahi Hospital), and all the investigators in the Japan Research Committee of the Ministry of Health, Labour, and Welfare for Intractable Vasculitis (JPVAS). In addition to the authors, the following investigators and institutions participated in this study: Department of Internal Medicine, Juntendo University Koshigaya Hospital (Shigeto Kobayashi); Niigata Rheumatic Center (Satoshi Ito); Niigata Prefectural Shibata Hospital (Noriyuki Homma).

Disclosure of Interests: takahiko sugihara Speakers bureau: TS has received honoraria from Abbvie Japan Co., Ltd., AsahiKASEI Co., Ltd., Astellas Pharma Inc., Ayumi Pharmaceutical, Bristol Myers Squibb K.K., Chugai Pharmaceutical Co., Ltd., Eli Lilly Japan K.K., Mitsubishi-Tanabe Pharma Co., Ono Pharmaceutical, Pfizer Japan Inc., Takeda Pharmaceutical Co., Ltd., and UCB Japan Co. Ltd., Grant/research support from: TS has received research grants from AsahiKASEI Co., Ltd., Daiichi Sankyo, and Ono Pharmaceutical, Haruhito A. Uchida Grant/research support from: HAU belongs to the Department of Chronic Kidney Disease and Cardiovascular Disease which is endowed by Chugai Pharmaceutical, MSD, Boehringer Ingelheim, and Kawanishi Holdings., Hajime Yoshifuji Speakers bureau: HY has received lecture fees from Chugai Pharmaceutical Co., Ltd. and Nihon Medi-Physics Co., Ltd., Yasuhiro Maejima Speakers bureau: YM has received honoraria from Chugai Pharmaceutical Co., Ltd., Taio Naniwa Speakers bureau: TN has received lecture fees from Chugai Pharmaceutical Co., Ltd., Grant/research support from: TN has received research grants from Chugai Pharmaceutical Co., Ltd., Yasuhiro Katsumata Speakers bureau: YK has received honoraria from Chugai Pharmaceutical Co., Ltd., Glaxo-SmithKline K.K., Sanofi K.K., Pfizer Japan Inc., and Asahi Kasei Pharma Corp., Takahiro Okazaki Grant/research support from: TO has received research grants from Chugai Pharmaceutical Co., Ltd., Eisai Pharmaceutical, and Actelion, Jun Ishizaki: None declared, Yohko Murakawa Speakers bureau: YM has received honoraria from Abbvie, Astellas, Ayumi Pharmaceutical, Bristol Myers Squibb, Chugai Pharmaceutical, Eisai Pharmaceutical, Janssen Pharmaceutical, Kissei Pharmaceutical, Nippon Kayaku, Pfizer Pharmaceutical, Takeda Pharmaceutical, UCB Pharmaceutical, Grant/research support from: YM has received research grant support from Asahi Kasei Pharma, AbbVie Japan, Chugai Pharmaceutical, Daiichi Sankyo, Eisai Pharmaceutical, Mitsubishi Tanabe Pharma, Nippon Kayaku, Gilead Sciences Inc, Janssen Pharmaceutical, and Teijin Pharma., Noriyoshi Ogawa: None declared, Hiroaki Dobashi: None declared, Tetsuya Horita: None declared, Yoshiya Tanaka Speakers bureau: YT has received consulting fees, speaking fees, and/or honoraria from Daiichi-Sankyo, Astellas, Pfizer, Mitsubishi-Tanabe, Bristol-Myers, Chugai, YL Biologics, Eli Lilly, Sanofi, Janssen, UCB, Grant/research support from: YT has received research grants from Mitsubishi-Tanabe, Takeda, Bristol-Myers, Chugai, Astellas, Abbvie, MSD, Daiichi-Sankyo, Pfizer, Kyowa-Kirin, Eisai, Ono., Shunsuke Furuta: None declared, Tsutomu Takeuchi Speakers bureau: TT has served on speakers' fees for AbbVie, Bristol-Myers Squibb, Chugai, Mitsubishi Tanabe, Pfizer, Astellas, Daiichi Sankyo, Eisai, Sanofi, Teijin, Takeda, and Novartis., Consultant of: TT has received consulting fees from Astra Zeneca, Eli Lilly, Novartis, Mitsubishi Tanabe, AbbVie, Nippon Kayaku, Janssen, Astellas, Taiho, Chugai, Taisho Toyama, GlaxoSmithKline, and UCB., Grant/research support from: TT has received research grants from Astellas, Chugai, Daiichi Sankyo, Takeda, AbbVie, Asahi Kasei, Mitsubishi Tanabe, Pfizer, Eisai, AYUMI, Nippon Kayaku, and Novartis., Yoshinori Komagata Speakers bureau: YK has received speakers' fees from Bristol-Myers Squibb, Eli Lilly, Janssen, Novartis, Daiichi Sankyo, AbbVie, Nippon Shinyaku, Towa., Consultant of: YK has received consulting fees from Chugai, Kyowa Hakko Kirin, Asahi Kasei, UCB, Yoshikazu Nakaoka Speakers bureau: YN has received lecture fees from Astellas, Takeda, Daiichi Sankyo, Actelion, and Japan Blood Products Organization (JB)., Consultant of: YN has received consulting fees and/or lecture fees from AbbVie and Chugai, Grant/research support from: YN has received research grants from Chugai and Bayer Yakuhin, Ltd, masayoshi hiragai Speakers bureau: MH has received speaker's fee from AbbVie Japan GK, Ayumi Pharmaceutical Co., Boehringer Ingelheim Japan, Inc., Bristol Myers Squibb Co., Ltd., Chugai Pharmaceutical Co., Ltd., Eisai Co., Ltd., Eli Lilly Japan K.K., GlaxoSmithKline K.K., Kissei Pharmaceutical Co., Ltd., Pfizer Japan Inc., Takeda Pharmaceutical Co., Ltd., and Teijin Pharma Ltd., Consultant of: MH is a consultant for AbbVie, Boehringer-Ingelheim, Bristol Myers Squibb Co., Kissei Pharmaceutical Co., Ltd. and Teijin Pharma., Grant/research support from: MH has received research grants from AbbVie Japan GK, Asahi Kasei Corp., Astellas Pharma Inc., Ayumi Pharmaceutical Co., Bristol Myers Squibb Co., Ltd., Chugai Pharmaceutical Co., Daiichi-Sankyo, Inc., Eisai Co., Ltd., Kissei Pharmaceutical Co., Ltd., Mitsubishi Tanabe Pharma Co., Nippon Kayaku Co., Ltd., Sekiui Medical, Shionogi & Co., Ltd., Taisho Pharmaceutical Co., Ltd., Takeda Pharmaceutical Co., Ltd., and Teijin Pharma Ltd.

DOI: 10.1136/annrheumdis-2021-eular.824

POS0337

SOUTHEND PRE-TEST PROBABILITY SCORE AND HALO SCORE AS MARKERS FOR DIAGNOSIS AND MONITORING OF GCA: EARLY RESULTS FROM THE PROSPECTIVE HAS-GCA STUDY

A. Sebastian^{1,2}, A. Tomelleri³, A. Kayani¹, M. Tariq¹, D. Prieto-Peña⁴, S. Inness², J. Jackson², K. Van der Geest⁵, B. Dasgupta^{1,2}. ¹Mid and South Essex University Hospital Groups, Southend University Hospital, Rheumatology, Westcliff-on-sea, United Kingdom; ²University of Essex, School of Sport, Rehabilitation and Exercise science, Colchester, United Kingdom; ³IRCCS San Raffaele Hospital, Unit of Immunology, Rheumatology, Allergy and Rare Diseases, Milan, Italy; ⁴Marques de Valdecilla University Hospital, Rheumatology, Santander, Spain; ⁵University of Groningen, University Medical Center Groningen, Rheumatology and Clinical Immunology, Groningen, Netherlands

Background: EULAR recommends doppler ultrasound (US) as the first line imaging in patients with Giant Cell Arteritis (GCA) suspect. Traditionally, US non-compressive halo sign has been used for diagnosis but prospective studies on response and disease monitoring are lacking

Objectives: The HAS GCA study has the objective of prospectively assessing role of US in diagnosis, prognosis and monitoring in newly diagnosed GCA. We report early baseline and up to month 3 data on our current recruitment in a study that has suffered disruption from the pandemic

Methods: HAS GCA (IRAS#264294) is an ongoing, prospective, multi-centre study recruiting from referrals of suspected GCA to fast track clinics. The objective is to recruit 270 patients, including 68 GCA patients. Based on the Southend GCA clinical pre-test probability score (SPTPS)¹, patients were stratified in to low, intermediate and high risk categories². Temporal and axillary US Halo Scores were calculated from the halo thickness and extent in bilateral temporal arteries, parietal and frontal branches and axillary arteries. These individual scores were summed (TA Halo Score x1 plus; AA Halo Score x3) to generate a Total Halo Score (THS)³.

Mann Whitney U test and Fisher's exact test were used to compare baseline features between GCA and controls. Wilcoxon signed rank test was used to evaluate disease features at baseline and at 3 months in GCA patients. Sensitivity (Sn) and Specificity (Sp) were calculated, where applicable. P value <0.05 is statistically significant

Results: Ninety-three patients (29 GCA, 64 controls) have been recruited thus far: 18 completed 3-month follow up assessment; 4 were lost to follow up (2 died, 2 withdrew consent due to pandemic). Demographics, clinical features, and US results are shown (Table 1).

Table 1. Baseline features of GCA patients and controls.

	GCA (n=29)	Controls (n=64)	P-value
Age, median (IQR)	75 (71-80)	67 (61.25 – 75.0)	0.001
Female, n (%)	15 (42)	50 (78)	0.01
SPTPS category, n (%)			
Low risk	0 (0)	31 (48)	<0.001
Intermediate risk	7 (24)	25 (39)	0.24
High risk	22 (76)	8 (13)	<0.001
Halo score (HS), median (range)			
Temporal artery HS	10 (1-21)	1 (0-9)	<0.001
Axillary artery HS	12 (0-18)	6 (0-18)	<0.001
Total HS	21 (2-38)	6 (0-19)	<0.001
Clinical features, n (%)			
Temporal headache	21 (72)	40 (63)	0.48
Scalp tenderness	17 (59)	31 (48)	0.38
Jaw claudication	19 (66)	4 (6)	<0.001
PMR symptoms	16 (55)	6 (9)	<0.001
Constitutional symptoms	17 (59)	18 (28)	0.006
Visual disturbance	18 (62)	38 (59)	1
Vision loss	7 (24)	4 (6)	0.03

Among GCA patients, 23 had cranial, 2 large-vessel and 4 mixed phenotypes (cranial plus large vessel) disease.

Jaw claudication (66%) and polymyalgic symptoms (55%) were the dominant features in GCA patients. Median age 75 years in GCA (42% females) and 67 years in controls (78% females). GCA and controls were stratified by SPTPS to Low risk (0% vs 48%; Sn-undefined, Sp-97), Intermediate risk (24% vs 39%; Sn-100, Sp-100) and High risk (76% vs 13%; Sn-95, Sp-88). Optimal SPTPS cut-off point was ≥ 12 (Sn-93, Sp-86); ≥ 10 (Sn-100 & Sp-69).

Median THS was 21 in GCA and 6 in controls. Optimal cut-off Halo Score in diagnosis was THS ≥ 5 (Sn-90, Sp-98), AAHS ≥ 11 (Sn-55, Sp-80), THS ≥ 18 (Sn-72%, Sp-98%). Among the 18 patients who completed 3-months follow up,

median TAHS, AAHS and THS reduced from 10 to 2.5, 12 to 6 and 21 to 10, respectively (Figure 1).

Figure: HS comparing GCA vs non GCA and baseline Halo Score vs 1 and 3 months follow up

AA, Axillary artery; GCA, Giant cell arteritis; TA, Temporal artery

Conclusion: Along with SPTPS, Halo Score successfully discriminates GCA from non GCA mimics. HS is effective in showing 3-month response and may be a useful marker to monitor GCA disease activity.

REFERENCES:

- [1] Laskou F et al. A probability score to aid the diagnosis of suspected giant cell arteritis. *Clin Exp Rheumatol*. 2019
- [2] Sebastian A et al. Probability-based algorithm using ultrasound and additional tests for suspected GCA in a fast-track clinic. *RMD Open*. 2020
- [3] Sebastian A et al. Halo score (temporal artery, its branches and axillary artery) as a diagnostic, prognostic and disease monitoring tool for Giant Cell Arteritis (GCA). *BMC Rheumatol*. 2020

Disclosure of Interests: Alwin Sebastian: None declared, Alessandro Tomelleri: None declared, Abdul Kayani: None declared, Mohammad Tariq: None declared, Diana Prieto-Peña: None declared, Sue Inness: None declared, Jo Jackson: None declared, Kornelis van der Geest Speakers bureau: Roche, Bhaskar Dasgupta Speakers bureau: Roche, GSK, BMS, Sanofi, Abbie, Grant/research support from: Roche

DOI: 10.1136/annrheumdis-2021-eular.1038

POS0338

STERIOD-SPARING EFFECT OF ANAKINRA IN GIANT-CELL ARTERITIS: A CASE SERIES WITH CLINICAL, BIOLOGICAL AND ICONOGRAPHIC LONG-TERM ASSESSMENTS

S. Deshayes¹, K. Ly², V. Rieu³, G. Maigné¹, N. M. Silva¹, A. Manrique⁴, J. Monteil⁵, H. De Boysson¹, A. Aouba⁴ on behalf of the French Study Group for Large Vessel Vasculitis (GEFA). ¹CHU de Caen Normandie, Internal Medicine, Caen, France; ²CHU Dupuytren, Internal Medicine, Limoges, France; ³CHU Estaing, Internal Medicine, Clermont-Ferrand, France; ⁴CHU de Caen Normandie, Nuclear Medicine, Caen, France; ⁵CHU Dupuytren, Nuclear Medicine, Limoges, France

Background: The treatment of giant cell arteritis (GCA) relies on corticosteroids but is burdened by a high rate of relapses and adverse effects. Anti-interleukin-6 treatments show a clear benefit with a significant steroid-sparing effect, but late relapses occur after treatment discontinuation. In addition to interleukin-6, interleukin-1 also appears to play a significant role in GCA pathophysiology.

Objectives: We report herein the efficacy of anakinra, an interleukin-1 receptor antagonist, in 6 GCA patients exhibiting corticosteroid dependence or resistance, specifically analyzing the outcome of aortitis in 4 of them, and including the long-term follow-up of 2 previously described patients (1).

Methods: This retrospective study analyzed the cases of all GCA patients treated with anakinra from the French Study Group for Large Vessel Vasculitis. Patients had to satisfy the following two criteria to be enrolled in this retrospective study. First, their diagnosis of GCA should be based on the fulfillment of at least 3 criteria of the American College of Rheumatology (ACR) for GCA or on the satisfaction of 2 of these criteria along with the demonstration of LVI on imaging. Second, patients should have received anakinra because of corticosteroid dependence or resistance.

Corticosteroid dependence was defined as ≥ 2 relapses or the combination of 2 of the following criteria: a daily dose of oral prednisone >20 mg/day (or 0.3 mg/kg) at 6 months; a daily dose of oral prednisone >10 mg/day (or 0.2 mg/kg) at 12 months; and/or a treatment maintained >24 months because of a relapsing disease course. Corticosteroid resistance was defined as persistent increased inflammatory parameters at month 3 despite a steroid dosage over 0.5 mg/kg.

Results: After a median duration of anakinra therapy of 19 [18–32] months, all 6 patients exhibited complete clinical and biological remission. Among the 4 patients with large-vessel involvement, 2 had a disappearance of aortitis under anakinra, and 2 showed a decrease in vascular uptake. After a median follow-up of 56 [48–63] months, corticosteroids were discontinued in 4 patients, and corticosteroid dosage could be decreased to 5 mg/day in 2 patients. One patient relapsed 13 months after anakinra introduction in the context of increasing the daily anakinra injection interval to every 48 hours. Three patients experienced transient injection-site reactions, and 1 patient had pneumonia.

Figure 1. Steroid dosages before and after the introduction of anakinra in 6 patients with giant-cell arteritis and corticosteroid dependence or resistance. The black arrow indicates the time of anakinra introduction.

Conclusion: In this short series, anakinra appears to be an efficient and safe steroid-sparing agent in refractory GCA, with a possible beneficial effect on large-vessel involvement.

REFERENCES:

- [1] Ly K-H, Stirnemann J, Liozon E, Michel M, Fain O, Fauchais A-L. Interleukin-1 blockade in refractory giant cell arteritis. *Joint Bone Spine* 2014;81:76–8.

Disclosure of Interests: Samuel Deshayes: None declared, Kim LY: None declared, Virginie Rieu: None declared, Gwénola Maigné: None declared, Nicolas Martin Silva: None declared, Alain Manrique: None declared, Jacques Monteil: None declared, Hubert de Boysson Speakers bureau: Roche-Chugai, Grant/research support from: Roche-Chugai, Achille Aouba Grant/research support from: SOBI

DOI: 10.1136/annrheumdis-2021-eular.1623

POS0339

PREDICTORS OF DEATH IN PATIENTS DIAGNOSED WITH GIANT CELL ARTERITIS IN WESTERN NORWAY 1972-2012

L.K. Brekke¹, B. T. Svanes Fevang², J. Assmus³. ¹Hospital for Rheumatic Diseases, Rheumatology, Haugesund, Norway; ²Haukeland University Hospital, Rheumatology, Bergen, Norway; ³Haukeland University Hospital, Centre for Clinical Research, Bergen, Norway

Background: Giant cell arteritis (GCA) is the most common systemic vasculitis in adults, and the number of incident cases worldwide is projected to increase [1]. Evidence as to whether or not GCA confers a mortality risk is conflicting, and many studies have been limited by inadequate or lacking adjustment for confounders.

Objectives: To investigate possible predictors of death in a large and well characterized Norwegian cohort of GCA-patients.

Methods: This is a hospital-based retrospective cohort study including patients diagnosed with GCA during 1972-2012. Patients were identified through computerized hospital records using the International Classification of Diseases coding system. Clinical information was extracted from patients' medical journals. Further details about the inclusion process have been published previously [2]. Information on time of death was obtained from the Norwegian Cause of Death Registry. We investigated predicting factors using Cox regression. Selected variables were first analyzed in univariate and block regression models (block 1: