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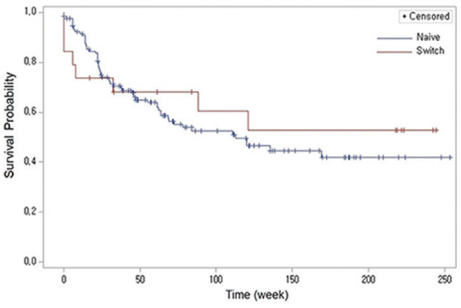

Figure 1. Drug survival probability of patients with RA treated with CT-P13

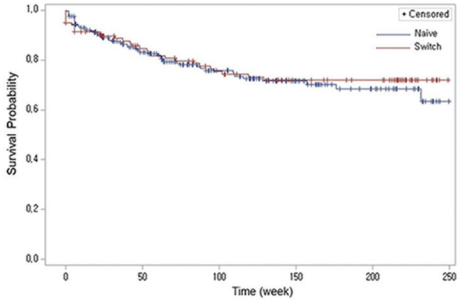

Figure 2. Drug survival probability of patients with AS treated with CT-P13

Disclosure of Interests: Tae-hwan Kim: None declared, Shin-Seok Lee: None declared, Won Park Consultant for: Celltrion, Inc, Yeong Wook Song: None declared, Chang-Hee Suh Consultant for: Celltrion, Inc, Soo-Kyoung Kim Shareholder of: Celltrion Healthcare, Employee of: Celltrion Healthcare, YoungNam Lee Shareholder of: Celltrion Healthcare, Employee of: Celltrion Healthcare, DaeHyun Yoo Grant/research support from: Celltrion, Inc., Consultant for: Celltrion, Inc.
DOI: 10.1136/annrheumdis-2019-eular.4546

SLE, Sjögren's and APS – clinical aspects (other than treatment)

SAT0172

ANTIBODIES TO BOTH RO52 AND RO60 DEFINE A SUBSET OF SJÖGREN'S MOST SUITABLE FOR CLINICAL TRIALS OF AGENTS TARGETING LYMPHOPROLIFERATIONS

Berkan Armagan[1,2], Susan Robinson[2], Adriana Bazoberry[2], Thomas Grader-Beck[2], Alan Baer[2]. [1]*Hacettepe University, Faculty of Medicine, Rheumatology, Ankara, Turkey*; [2]*Johns Hopkins University, School of Medicine, Rheumatology, Baltimore, MD, United States of America*

Background: Anti-SSA antibodies comprise reactivity to two distinct proteins, Ro52 and Ro60, encoded by separate genes and found on separate ribonucleoprotein particles. Specific testing for Ro52 and Ro60 antibodies is now clinically available, yet the phenotypic correlates of Ro52 and Ro60 reactivity profiles have not been well defined.
Objectives: To determine the phenotypic correlates of antibody reactivity to Ro52 alone, Ro52 + Ro60, and Ro60 alone in patients being evaluated for Sjögren`s syndrome (SS).
Methods: We initially studied 840 patients seen at the Hopkins Sjögren`s Syndrome Center with suspected or established SS. Each had serum tested for antibodies to recombinant Ro52 (Inova Quanta Lite ELISA) and Ro60 (**IVTT im***munoprecipitation*). We then validated our findings in a second cohort consisting of 194 patients, each with testing for antibodies to recombinant anti-Ro52 and anti-Ro60 by a chemiluminescent assay (Inova Bioflash). Statistical analyses were performed using JMP pro 13. The Chi-square or Fisher exact test was used to compare the groups.
Results: The discovery cohort of 840 patients included 751 (89%) women, with a mean age of 58.5±13.5 years. 371 (44%) patients met the ACR/EULAR classification criteria. There were 311 with anti-Ro52 +Ro60, 108 with anti-Ro60 alone, 95 with anti-Ro52 alone, and 326 with neither antibody. The 311 patients with anti-Ro52+Ro60 reactivity had a distinctive phenotype, with a markedly increased prevalence of ANA≥ 1:320, RF, IgG≥1560 mg/dL, and SS-B positivity ($p<0.008$ for all inter-group comparisons) and an increased prevalence of focus score ≥1 and hypoechoic lesions on parotid gland ultrasonography which trended toward statistical significance. These differences were also validated in the second cohort, with the exception of focus score and parotid gland hypoechoic lesions, possibly as a result of smaller group numbers.The Ro52 and Ro60 alone groups were equivalent to each other in their phenotypic associations, except for RF, which was higher in the Ro52 alone group. Measures of lacrimal and salivary gland function and the prevalences of extraglandular manifestations did not show consistent differences between the groups or the two cohorts.
Conclusion: Testing anti-Ro52 and anti-Ro60 in patients with suspected or established SS identifies a unique subset, namely those with both Ro52 and Ro60 antibodies, distinguished by a much higher prevalence of B-cell activation markers and glandular inflammation as measured by focus score and hypoechoic lesions. This subset may be most suitable for inclusion in clinical trials where the therapeutic agent targets glandular lymphoproliferation.

Table. The comparison of the differences according to anti-Ro subtypes in 2 different cohorts

	Discovery cohort (n=840)						Validation cohort[4] (n=194)
	Group A: anti-Ro60 alone n=108 (%)	Group B: anti-Ro60 with anti-Ro52 n=311 (%)	Group C: anti-Ro52 alone n=95 (%)	Group D: All negative n=326, (%)	All group comparison	Two group comparison	Two group comparison
					p value[3]	p value[c]	p value[c]
ANA ≥ 1:320	25/106 (25.6)	228/297 (76.7)	36/91 (39.5)	41/318 (12.9)	<0.0001	B vs A, <0.0001 B vs C, <0.0001 B vs D, <0.0001 A vs C, 0.0156	B vs A, 0.0004 B vs C, <.0001 B vs D, <.0001 A vs C, 0.5266
Positive RF	11/104 (10.6)	178/306 (58.2)	27/88 (30.7)	20/321 (6.2)	<0.0001	B vs A, <0.0001 B vs C, <0.0001 B vs D, <0.0001 A vs C, 0.0005	B vs A, 0.0003 B vs C, 0.0009 B vs D, <.0001 A vs C, 0.8772
IgG ≥ 1560 mg/dL	14/100 (14.0)	135/298 (45.3)	12/87 (13.8)	16/306 (5.2)	<0.0001	B vs A, <0.0001 B vs C, <0.0001 B vs D, <0.0001 A vs C, 0.9675	B vs A, 0.0005 B vs C, 0.0019 B vs D, <.0001 A vs C, 0.8841
SSB antibody positivity	16/108 (14.8)	166/311 (53.4)	17/95 (17.8)	23/326 (7.1)	<0.0001	B vs A, <0.0001 B vs C, <0.0001 B vs D, <0.0001 A vs C, 0.3529	B vs A, 0.0031 B vs C, <.0001 B vs D, <.0001 A vs C, 0.0265
FLS with FS ≥ 1	5/78 (6.4)	50/150 (33.3)	17/63 (26.9)	38/288 (13.2)	<0.0001	B vs A, <0.0001 B vs C, 0.3624 B vs D, <0.0001 A vs C, 0.0008	B vs A, 0.1328 B vs C, 0.2027 B vs D, 0.0006 A vs C, 0.8771
Parotid hypoechoic lesions	4/14 (28.6)	41/68 (60.3)	3/14 (21.4)	3/38 (7.9)	<0.0001	B vs A, 0.0298 B vs C, 0.0079 B vs D, <0.0001 A vs C, 0.6625	B vs A, 0.3351 B vs C, 0.1146 B vs D, <.0001 A vs C, 0.5356
Extraglandular manifestations	38/108 (35.2)	159/311 (51.1)	41/95 (43.2)	100/326 (30.7)	<0.0001	B vs A, 0.0042 B vs C, 0.1740 B vs D, <0.0001 A vs C, 0.2450	NS in all groups

ANA, antinuclear antibody; RF, rheumatoid factor; IgG, immunoglobulin G; FLS, focal lymphocytic sialadenitis; FS, focus score
[a] Number and percentages of immunological features of validation cohort patients was not shown
[b] A p value <0.05 was considered significant
[c] A p value <0.008 was considered significant

Disclosure of Interests: Berkan Armagan: None declared, Susan Robinson: None declared, Adriana Bazoberry: None declared, Thomas Grader-Beck: None declared, Alan Baer Consultant for: I have no conflicts of interest within the past 12 months. In 2017, I served on advisory boards for Novartis and AbbVie.
DOI: 10.1136/annrheumdis-2019-eular.3387

SAT0173

RENAL GALLIUM SCAN CORRELATED WITH INFLAMMATION IN RENAL HISTOLOGY OF PATIENTS WITH LUPUS NEPHRITIS

Tsu-Yi Hsieh, Yi-Ming Chen, Wei-Ting Hung, Hsin-Hua Chen, Kuo-Lung Lai, Ching-Tsai Lin, Yi-Da Wu, Wen-Nan Huang, Chih-Wei Tseng, Yi-Hsing Chen. *Taichung City, Section of Allergy, Immunology and Rheumatology, Department of Internal Medicine, Taichung, Taiwan, Republic of China*

Background: Lupus nephritis (LN) is the leading cause of mortality in lupus patients. But there is only one image assessment method, the histopathology through invasive renal biopsy.
Objectives: This study aimed to investigate the clinical value of the non-invasive image assessment method: renal gallium scan, in renal histological parameters of LN in a cohort of one single tertiary referral center.
Methods: Between 2006 and 2018, a hospital-based observational study was conducted to enroll 266 biopsy-proved and 40 repeated-biopsied LN patients who underwent renal gallium scan before biopsy. The classification and scoring of LN were assessed according to the International