

Conclusions LIT responds appropriately in both directions to changes in physician (T2T) as well as patient relevant (DA and health status) outcomes among Spanish SLE patients.

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73 RHEUMATOID FACTOR IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background Rheumatoid factor (RF) in SLE is found in around 25% of patients. Authors associate it with cutaneous involvement, Sicca, anti Ro (+) and a protective role in glomerulonephritis (GMN), suggested by the finding of less frequent and less severe nephritis in lupus patients with RF(+) as in patients with RA.

The proposed protective mechanisms of RF include preventing the complement from joining immune-complexes (IC) and avoiding the IC deposit in the glomerulus.

We aimed to determine the clinical and immunological profile of SLE patients with and without RF and to determine the association between RF and GMN.

Methods Descriptive, retrospective study of SLE (SLICC 2012) patients selected from Rheumatology department of Güemes Hospital, from January 2015-August 2018.

We collected data on demographics, cutaneous-articular, renal, hematological and CNS involvement. Immunoserology: RF IgM by immunoturbidimetric (+)>30 UI/ml, anti Ro, anti La, anti Sm, anti RNP (ELISA), ANA (Hep 2), Anti DNA (Crithidia lucilliae), C3, C4, aPL at least one

of the following: LI, anti 2GP1 IgG/IgM, Anti aCL IgG/IgM.

Statistical Analysis: Epiinfo version 7.2.0.1.

Results We reviewed 147 clinical histories, 107 with RF ordered. Female 93/107 (86.9%), mean age 41.6 years (SD ±13.8). Disease duration 98 months (SD ±80.6). Tobacco exposure 10/107 (9.35%). RF (+) 22/107 (20.5%), RF median titer 228.8 IU/ml (31–2825 IU/ml).

RF level in patients with GMN was 142 IU/ml (39–191), without GMN 258 IU/ml (31–2825) p=0.46.

RF level in patients with anti Ro (+) was 248 IU/ml (31–2825), Ro (-) 52.5 IU/ml (31–74) p=0.37.

RF (-) subgroup showed statistically significant association with female sex, discoid lesions and aPL presence. RF(+) was associated with Ab Ro, La and patients with RA. (Table 1).

Not found significant association with: rash, photosensitivity, oral ulcer, joint pain, arthritis, serositis, neurologic and hematological involvement.

In those with GMN, we found a significant association with Ab Ro(+) in the absence of RF (table 1).

Conclusions Similar RF frequency was found as the published literature. There was no association with nephritis but the mean RF titer was higher in patients without GMN.

The detection of Ab clusters associated with RF may be useful to suggest absence of renal involvement.

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74 RNA-SEQUENCING TO DISSECT THE ROLE OF SELECTIVE HDAC6 INHIBITION ON B CELL SIGNALING AND GERMINAL FORMATION IN LUPUS NEPHRITIS

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Background Autoantibody production by plasma cells (PC) play a pivotal role in pathogenesis in of lupus nephritis (LN). The mechanism(s) of how B cells become pathogenic PC secreting autoantibody in SLE is incompletely characterized. In the current study, sought to determine if selective histone deacetylase (HDAC)6 inhibition would abrogate abnormal B cell activation in SLE.

Methods We treated 20-week-old NZB/W lupus mice with the selective HDAC6 inhibitor ACY-738 for four weeks beginning at 20 weeks-of age; at early disease. After 4 weeks-of-treatment, we used RNA-seq to determine the genetic signatures of splenocytes with and without treatment and applied signaling pathway computational analysis to reveal multiple pathways associated with B cell activation and differentiation in SLE that were modulated by HDAC6 inhibition.

Results Plasma cell development was abrogated as well as GC were greatly reduced. Additionally, kidney pathology was greatly reduced. When gene signature was compared to human lupus patients, the HDAC6 inhibitor treated mice showed several inflammatory pathways were decreased.

Conclusions Taken together, these studies suggest that HDAC6 inhibition decreased B cell activation signaling pathways and reduced PC differentiation in LN, suggesting that HDAC6 inhibition may represent an effective target to treat SLE.

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Abstract 73 Table 1 Subgroup characteristics

	RF (+) n 22 (%)	RF (-) n 85 (%)	p
Female	16 (73)	77 (90)	0.02
Tobacco	4 (18)	6 (7)	0.07
Discoid	0 (0)	9 (10)*	0.049
GMN	6 (27)	28 (33)	0.31
Ro	18 (82)*	35 (41)	0.0003
La	11 (50)*	16 (18)	0.002
Sm	6 (27)	18 (21)	0.27
RNP	7 (32)	27 (32)	0.49
DNA	13 (59)	41 (48)	0.50
Low complement	17 (77)	66 (77)	0.47
SICCA	7 (32)	17 (20)	0.12
aPL +	0 (0)	11/71 (15)*	0.04
SAF	2 (9)	7 (8)	0.43
RA	6 (27)*	4 (4)	0.002
S. Sjogren	3 (14)	6 (7)	0.17

	RF negative		p
	Ro +	Ro-	
La	13/35 (37%)	2/50 (4%)	0.00006
GNF	16/35 (46%)	12/50 (24%)	0.02
AR	2/35 (6%)	2/50 (2%)	0.36