

# Anti RNP in systemic lupus erythematosus

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To the Editor,

Systemic lupus erythematosus (SLE) is a heterogeneous disease<sup>1</sup>; the analysis of the autoantibodies profile in a SLE patient may help to predict the clinical manifestations<sup>2,3</sup>. Anti-RNP presence have been associated to Raynaud's phenomenon, myositis, esophageal dysmotility and absence of nephritis in studies with a mixed population comprising both SLE and mixed connective tissue disease patients<sup>4,5</sup>. Anti-RNP together with anti-Sm are directed against spliceosome proteins, providing the appearance of an ANA with speckled pat-

tern<sup>6,7</sup>. Spliceosome is an intracellular structure that removes the intronic sequences of the pre-messenger RNA and links protein coding sequences to form mature RNA<sup>6</sup>. Anti-RNP presence is higher in blacks and Asians<sup>8,9</sup> and it is associated with HLA DR2 and 4<sup>9</sup>. The positivity is as high as 62% in black patients of African origin<sup>10</sup>.

As the clinical and serological associations of anti-RNP in lupus may vary with the patient's ethnic and geographical distribution, we studied the presence of anti-RNP in a SLE population from Southern Brazil. All included patients fulfilled at least four classification criteria for SLE of the American College of Rheumatolo-

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**TABLE I. COMPARISON OF SLE CLINICAL PROFILE IN 295 PATIENTS ACCORDING TO THE PRESENCE OF ANTI-RNP**

	Anti-RNP positive	ANTI-RNP negative	p
Photosensitivity	63/81 – 77.77%	164/212 – 77.35%	0.93
Oral ulcers	31/78 – 39.74%	105/208 – 50%	0.12
Raynaud	50/80 – 62.50%	86/208 – 41.34%	0.001
Malar rash	41/78 – 52.56%	102/209 – 48.80%	0.57
Discoid lesions	18/80 – 22.50%	21/211 – 9.95%	0.005
Serositis	13/82 – 15.85%	41/213 – 19.24%	0.49
Glomerulonephritis	36/86 – 43.90%	93/212 – 43.86%	0.99
Leukopenia	35/81 – 43.20%	62/211 – 29.38%	0.02
Lymphopenia	21/80 – 26.25%	28/208 – 13.46%	0.009
Hemolytic anemia	9/81 – 11.11%	15/211 – 7.10%	0.26
Thrombocytopenia	14/81 – 17.28%	46/213 – 21.59%	0.41
Arthritis	59/82 – 71.95%	128/211 – 60.66%	0.07
Myositis	3/81 – 3.70%	10/210 – 4.76%	1.00
Convulsions	7/81 – 8.64%	24/213 – 11.26%	0.51
Psychosis	5/80 – 6.25%	12/213 – 5.63%	0.84
Antiphospholipid syndrome	11/82 – 13.41%	18/212 – 8.49%	0.20
Pulmonary hypertension	4/79 – 5.06%	9/202 – 4.45%	0.76
Anti-Ro	35/81 – 43.20%	66/212 – 31.13%	0.055
Anti-La	18/81 – 22.22%	38/212 – 17.92%	0.40
Anti-Sm	51/81 – 62.96%	20/213 – 9.38%	<0.0001
Anti-dsDNA	35/81 – 43.20%	72/212 – 33.96%	0.14
Anticardiolipin IgG	9/81 – 11.11%	31/213 – 14.55%	0.44
Anticardiolipin IgM	12/81 – 14.81%	34/213 – 16.03%	0.79
Lupus anticoagulant	13/75 – 17.33%	28/197 – 14.21%	0.52

gy<sup>11</sup>. The charts were reviewed for demographic data (sex, age at diagnosis, ethnic background and disease duration), clinical and serological findings. The clinical findings included were in the criteria listed by the American College of Rheumatology for SLE classification (1997) and defined by them<sup>11</sup>. Furthermore, the presence of Raynaud phenomena, myositis, pulmonary hypertension and secondary antiphospholipid syndrome was examined.

Auto antibodies considered for analysis were: anti-Ro/SS-A, anti-La/SS-B, anti-RNP, anti-Sm, aCl IgG, aCl IgM (by ELISA using Orgentec Kits®); anti-dsDNA (by immunofluorescence technique (IFT) using Crithidia luciliae as a substrate). Lupus anticoagulant was examined through a screening test, the dRVVT (dilute Russell viper venom test) and confirmed by RVVT.

The sample had 295 patients with mean age at diagnosis of 37.1 ± 11.9 years; 17 were men and 278 women and the median disease duration was of 6 years. In this sample 65.9% were of African origin (blacks and mulattos) and 34.1% were Caucasian.

The prevalence of anti-RNP was 89/295 (27.8%). No differences were observed regarding gender in anti-RNP positive and negative patients (p=0.87). The associations with clinical and serological profile are in Table I.

In the present analysis, we found associations of anti-RNP with Raynaud phenomena, leukopenia and lymphocytopenia, rash discoid and anti Sm antibodies.

No association (either positive or negative) could be found with renal disease. Racial and environmental modulations are other possible explanations for the variability found in these associations.

To conclude, we can say that in our population the anti-RNP antibodies should alert the clinician to a more careful surveillance regarding hematologic manifestations, discoid lesions and Raynaud's phenomena.

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