CLINICAL AND SEROLOGICAL CORRELATIONS OF AUTOANTIBODIES DIRECTED AGAINST RNP-C IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background Autoantibodies to RNP-C protein, along with RNP-A, is a component of the U1RNP macromolecular complex. Antibodies to U1-RNP are typically associated with mixed connective tissue disease and other systemic autoimmune rheumatic diseases. Autoantibodies to RNP-C or their clinical significance have not been thoroughly studied in systemic lupus erythematosus (SLE). The goals of this study were to determine the frequency of anti-RNP-C autoantibodies in a SLE cohort and identify demographic, clinical, and serologic correlations.

Methods Patients fulfilling the ACR or SLICC Classification Criteria for SLE were enrolled in a local cohort. Demographic, clinical information (disease activity SLEDAI-2K; damage SLICC/ACR Damage Index (SDI)), and sera were collected at time of enrollment. Antibodies to anti-RNP-C were determined by a line immunoassay using a purified, full-length recombinant protein Euroimmun GmbH, Luebeck, Germany. Univariable and multivariable analysis were performed to determine associations between the prevalence of high positive anti-RNP-C and demographic (age, sex, race/ethnicity), clinical features (SLICC/ACR classification criteria, SLEDAI-2K and SDI total scores and subscales from SLEDAI-2K), medications, and other autoantibodies.

Results 138 SLE patients were included; 89.1% were female with a mean age of 46.1 years (SD 18.1 years) and disease duration of 13.7 years (SD 11.6 years). The prevalence of anti-RNP-C antibodies was 19.6% (27/138); 25.9% (7/27) were male. Univariable analysis demonstrated that patients fulfilling a higher total SLICC criteria (Odds Ratio, OR, 1.4 [95% Confidence interval, CI: 1.1–1.7]), particularly maculopapular rash (OR 4.0 [95% CI: 1.3–11.9]) and pericardial effusion (OR 6.3 [95% CI: 1.3–29.9]), or a higher SLEDAI score (OR 1.2 [95% CI: 1.0–1.3]) were more likely to be anti-RNP-C positive. Also, patients with higher immunological SLICC subscales (OR 1.8 [95% CI: 1.2–2.5]), anti-dsDNA (OR 7.6 [95% CI: 2.6–21.9]), anti-Sm (OR 29.7 [95% CI: 9.8–89.9]), anti-RNP (OR 44.2 [95% CI: 12.0–163.4]), anti-nucleosome (OR 9.4 [95% CI: 2.5–35.0]), anti-Ribosomal P (OR 7.4 [95% CI: 2.5–22.5]), or anti-RNP-A (OR 153.8 [95% CI: 35.8–660.5]) were more likely to be anti-RNP-C positive. Multivariable analysis demonstrated that patients who were anti-RNP-A positive (OR 78.5 [95% CI: 6.5–941.2]) were more likely to be anti-RNP-C positive while those who were female (OR 0.2 [95% CI: 0.1–0.7]), had longer disease duration (OR 0.9 [95% CI: 0.9–1.0]), or were on steroids (OR 0.3 [95% CI: 0.1–0.7]) were less likely to be anti-RNP-C positive. Multivariable analysis demonstrated that patients who were anti-RNP-A positive (OR 78.5 [95% CI: 6.5–941.2]) were more likely to be anti-RNP-C positive while those who were female (OR 0.2 [95% CI: 0.1–0.7]), had longer disease duration (OR 0.9 [95% CI: 0.9–1.0]), or were on steroids (OR 0.3 [95% CI: 0.1–0.7]) were less likely to be anti-RNP-C positive.

Conclusions Anti-RNP-C antibodies were common (19.6%) in our SLE cohort. In SLE, they were associated with anti-RNP-A antibodies, a finding which is in keeping with the concept of inter-molecular epitope spreading. Most notably, anti-RNP-A antibodies were more likely seen to be seen in males with SLE. A thorough study of male SLE patients is needed.

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