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ORIGINAL ARTICLE

# Clinical and serological associations of anti-ribosomal P0 protein antibodies in systemic lupus erythematosus

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**Abstract** The purpose of this study is to investigate the clinical and serological associations of anti-ribosomal P0 protein antibodies (anti-Rib-P0) in patients with systemic lupus erythematosus (SLE). The sera of 470 patients with SLE and 124 patients with primary Sjogren's Syndrome (pSS) were collected. Line immunoassay (LIA) was used to detect anti-Rib-P0 and other related antibodies. A complete laboratory evaluation and clinical examination were also performed in each SLE patient. The prevalence of anti-Rib-P0 in SLE patients was significantly higher than that in pSS patients (35.74 vs 6.45%) ($P < 0.001$). There was a significantly lower prevalence of cardiac involvement in anti-Rib-P0-positive SLE patients compared to anti-Rib-P0-negative SLE patients ($P = 0.019$); no significant associations of anti-Rib-P0 antibodies with encephalopathy manifestations and other vital organs involvement were observed. Anti-nucleosomes, anti-dsDNA, anti-Histones, anti-SmD1, and anti-U1snRNP were significantly associated with serum anti-Rib-P0 antibodies positivity in SLE patients (all $P < 0.05$). The sensitivity and specificity of the anti-Rib-P0 antibodies to diagnose SLE were 35.74 and 93.55%, respectively. There is a higher prevalence of anti-Rib-P0 in SLE patients. Anti-Rib-P0 positivity may indicate lower cardiac involvement for SLE patients. It may serve as an important complementary parameter in SLE, in addition to anti-dsDNA, anti-SmD1, and anti-nucleosomes.

**Keywords** Anti-ribosomal P0 protein antibodies · Ribosomal RNP · Systemic lupus erythematosus

## Introduction

Systemic lupus erythematosus (SLE) is a chronic inflammatory and autoimmune disease characterized by the presence of autoantibodies directed against a variety of nuclear and cytoplasmic antigens. It cannot only affect multiple organs, but also has a large spectrum of clinical presentations. Assessment of the autoantibody profile is fundamental and important process in the diagnosis, pathogenesis, and clinical management of SLE [1].

The targets of anti-ribosomal antibodies are three highly conserved P proteins located on the 60S subunit of ribosomes [2, 3]. These three ribosomal proteins (P0, P1, P2) are organized in a pentamer consisting of one copy of P0 and two copies each of P1 and P2, with the molecular weights of 38, 19, and 17 kD. The immunodominant epitope of SLE patients is a shared sequence at the carboxy (C)-termini of the P proteins [4–6]. Autoantibody directed against three phosphorylated protein (P proteins) components of ribosomes is present in the sera of SLE patients and is highly specific for this disease. The reported prevalence of anti-ribosomal P protein antibodies (anti-Rib-P) in SLE population ranges from 6 to 46%, which is higher in Asian patients than Afro-Americans and

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Caucasians [7–12]. Anti-ribosomal P0 protein antibody (anti-Rib-P0) is one of the three subunits of anti-Rib-P; several previous studies have addressed the diagnostic value of anti-Rib-P0; sera anti-Rib-P0 is highly specific for SLE, therefore having a significant clinical value in the diagnosis of SLE [13, 14]. However, comprehensive analyses on the clinical associations of anti-Rib-P0 which mediate SLE-related organ injuries in the Chinese population remain largely unknown.

The aim of our study is to evaluate the clinical and serological associations of anti-Rib-P0 in a large cohort of Chinese SLE patients and clarify the correlation of anti-Rib-P0 with vital organs damage in SLE.

## Materials and methods

### Study subjects

This study is approved by the ethical committees in the First Affiliated Hospital of Bengbu Medical College. Methods were carried out in accordance with the approved guidelines. All subjects were enrolled after informed consent had been obtained. Four hundred and seventy patients with SLE (441 females, 29 males; mean age 35.63 ± 13.23 years, range from 14 to 70 years) were recruited from the Department of Rheumatology and Immunology at the First Affiliated Hospital of Bengbu Medical College from 2013 to 2015. All patients fulfilled the American College of Rheumatology (ACR) criteria for SLE [15]; drug-induced SLE patients were excluded. From the same hospital, 124 patients with primary Sjögren's syndrome (pSS) (121 females, 3 males; mean age 46.44 ± 12.65 years, range from 20 to 81 years) who fulfilled the preliminary European League Against Rheumatism Criteria of Vitali et al. [16] were chosen as controls. Clinical features were defined according to the ACR criteria [15], encephalopathy was diagnosed according to the ACR guidelines [17]. Cardiac involvement involves the pericardium, valves, myocardium, and coronary arteries. Interstitial lung disease was diagnosed by CT scan. Demographic data, clinical data, and laboratory data were collected from hospital records or by self-designed questionnaire and reviewed by experienced physicians.

### Extraction of serum and measurement of autoantibodies

Blood samples were obtained from 5 ml of whole blood of all enrolled SLE and pSS patients and then stored at − 20 °C until analysis. The anti-Rib-P0 and other antibodies (anti-nucleosomes, anti-dsDNA, anti-Histones, anti-SmD1, anti-U1snRNP, anti-SSA/Ro60, anti-SSA/Ro52, anti-SSb/La, anti-Scl70, anti-Centromere and anti-Jo1) were determined by line immunoassay (LIA). Reagents were purchased from Human Gesellschaft fur Biochemica und Diagnostica mbH, Wiesbaden, Germany. The detection was performed according to the manufacturers' instructions.

### Statistical analysis

All results were presented as mean ± SD or median (interquartile range, IQR) if they were not in normal distribution. The Student's $t$ test was used for comparison of ECLAM score indices, expressed as mean ± SD. Clinical and serological parameters were analyzed using conventional chi-square test or Fisher's exact test. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) statistical software for Windows, version 10.01 (SPSS Inc., IL, USA). All results were considered significant at the 0.05 level.

## Results

The general features of study subjects are shown in Table 1. We examined two groups of subjects: 470 SLE patients and 124 pSS controls. Their mean age (± SD) was 35.63 ± 13.23 and 46.44 ± 12.65 years, respectively. Females accounted for 94% of the SLE group and 98% of the pSS controls. The disease duration of SLE and pSS was (4.01 ± 4.62) years and (4.07 ± 4.93 years), respectively. The prevalence of nephritis, encephalopathy, cardiac involvement, and gastrointestinal vasculitis in SLE patients was significantly higher than that in pSS patients (all $P < 0.05$). The prevalence of anti-Rib-P0 in SLE patients was significantly higher than that in pSS patients (35.74 vs 6.45%) ($P < 0.05$).

The comparison of the clinical findings between the antibody-positive and antibody-negative groups demonstrated a much lower prevalence of cardiac involvement in anti-Rib-P0-positive patients ($P = 0.019$). No significant differences in

**Table 1** The general features of study subjects

| Parameters | SLE patients ($n$ = 470) | pSS patients ($n$ = 124) | $P$ value |
|---|---|---|---|
| Age (year) | 35.63 ± 13.23 | 46.44 ± 12.65 | 0.000 |
| Sex (male/female) | 29/441 | 3/121 | 0.119 |
| Disease duration (year) | 4.01 ± 4.62 | 4.07 ± 4.93 | 0.887 |
| Nephritis (yes/no) | 186/284 | 0/124 | 0.000 |
| Encephalopathy (yes/no) | 18/452 | 0/124 | 0.019 |
| Cardiac involvement (yes/no) | 96/373 | 2/122 | 0.000 |
| Thrombocytopenia (yes/no) | 98/372 | 17/107 | 0.075 |
| Interstitial lung disease(yes/no) | 65/405 | 12/112 | 0.234 |
| Gastrointestinal vasculitis (yes/no) | 17/453 | 0/124 | 0.030 |
| Anti-ribosomal P0 protein antibodies(+/−) | 168/302 | 8/116 | 0.000 |

the prevalence of other vital organ damages were observed between the anti-Rib-P0-positive and anti-Rib-P0-negative SLE patients (Table 2).

Associations of anti-Rib-P0 antibodies with categorical laboratory parameters of SLE patients were also analyzed, and the results showed that anti-Rib-P0 antibodies were associated with anti-nucleosomes, anti-dsDNA, anti-Histones, anti-SmD1, and anti-U1snRNP (Table 3).

The sensitivity and specificity of the anti-Rib-P0 antibodies to diagnose SLE were 35.74 and 93.55%, respectively. The seroprevalence of antibodies and clinical accuracy was shown in Table 4.

Anti-nucleosomes, anti-dsDNA, anti-Histones, anti-SmD1, and anti-Rib-P0 showed a higher seroprevalence and area under the curve (AUC) than the combination diagnostic of these five antibodies (Table 5).

## Discussion

SLE is a chronic, multifaceted rheumatic disease characterized by the generation of autoantibodies predominantly directed against nuclear proteins and nucleic acids. Anti-Rib-P antibodies are serological markers for SLE described in 13–20% of patients; it might contribute to the cognitive impairment which is frequently observed in SLE patients [18].

An original and important finding of this study was that the prevalence of anti-Rib-P0 in SLE patients was significantly higher than in pSS controls. The sensitivity and specificity of the anti-Rib-P0 antibodies to diagnose SLE were 35.74 and 93.55%, respectively. In addition, anti-Rib-P0 antibodies were also found in the serum of patients with pSS; this is inconsistent with previous studies on the specificity of anti-Rib-P0 in SLE. The possible explanation is that some of pSS patients would fold to SLE after years of slow progression; therefore, the presence of anti-Rib-P0 in pSS may predict the possibility of future concurrent SLE.

There was a much lower prevalence of cardiac involvement in anti-Rib-P0 positive SLE patients compared to anti-Rib-P0 negative SLE patients, but those vital complications of SLE such as nephritis and encephalopathy were not associated with anti-Rib-P0 positivity. In addition, there was no significant correlation between neural symptoms and anti-Rib-P0; this result is consistent with previous studies [19, 20].

Giving the evidence for an association of anti-Rib-P0 with other specific autoantibodies for SLE, the frequency of anti-nucleosomes, anti-dsDNA, anti-Histones, anti-SmD1, and anti-U1snRNP were higher in anti-Rib-P0 positive patients than those of negative patients. However, we found no significant correlation of anti-Rib-P0 with anti-SSA/Ro60, anti-SSA/Ro52, anti-SSb/La, anti-Scl70, anti-Centromere, and anti-Jo1, respectively.

As the prevalence of anti-Rib-P0 antibodies in SLE varies according to several factors, of which the most important factor is the method for antibody detection/measurement, we

**Table 2** Comparison of different complications with anti-ribosomal P0 protein antibodies in SLE

| Parameter | | Anti-ribosomal P0 protein antibodies | | *P* value |
|---|---|---|---|---|
| | | + | – | |
| Nephritis | Yes | 67 | 119 | 0.919 |
| | No | 101 | 183 | |
| Encephalopathy | Yes | 7 | 11 | 0.777 |
| | No | 161 | 291 | |
| Cardiac involvement | Yes | 23 | 73 | 0.019* |
| | No | 145 | 228 | |
| Thrombocytopenia | Yes | 33 | 65 | 0.631 |
| | No | 135 | 237 | |
| Interstitial lung disease | Yes | 21 | 44 | 0.553 |
| | No | 147 | 258 | |
| Gastrointestinal vasculitis | Yes | 5 | 12 | 0.579 |
| | No | 163 | 290 | |

*Versus non-cardiac involvement patients

**Table 3** Associations of anti-ribosomal P0 protein antibodies with categorical laboratory parameters of SLE patients

| Parameter | | Anti-ribosomal P0 protein antibodies | | *P* value |
|---|---|---|---|---|
| | | + | – | |
| Nuclosomcs | Yes | 84 | 108 | 0.003 |
| | No | 84 | 194 | |
| dsDNA | Yes | 89 | 112 | 0.001 |
| | No | 79 | 190 | |
| Histones | Yes | 83 | 117 | 0.025 |
| | No | 85 | 185 | |
| SmD1 | Yes | 91 | 102 | 0.000 |
| | No | 77 | 200 | |
| U1snRNP | Yes | 98 | 123 | 0.000 |
| | No | 70 | 179 | |
| SSAro60 | Yes | 116 | 198 | 0.442 |
| | No | 52 | 104 | |
| SSAro52 | Yes | 89 | 161 | 0.944 |
| | No | 79 | 141 | |
| SSBla | Yes | 35 | 58 | 0.671 |
| | No | 133 | 244 | |
| Scl70 | Yes | 2 | 3 | 1.000 |
| | No | 166 | 299 | |
| Centromere | Yes | 5 | 16 | 0.243 |
| | No | 163 | 286 | |
| Jo1 | Yes | 2 | 3 | 1.000 |
| | No | 166 | 299 | |

+/−, with/without

**Table 4** The seroprevalence of antibodies and clinical accuracy

| Antibody | SLE | | | pSS | | | Sensitivity | Specificity | AUC |
|---|---|---|---|---|---|---|---|---|---|
| | Positive | Negative | Sero-prevalence | Positive | Negative | Seroprevalence | | | |
| P0 | 168 | 302 | 35.74% | 8 | 116 | 6.45% | 35.74% | 93.55% | 0.65 |
| Nucleosomes | 192 | 278 | 40.85% | 2 | 122 | 1.61% | 40.85% | 98.39% | 0.70 |
| dsDNA | 201 | 269 | 42.77% | 0 | 124 | 0.00% | 42.77% | 100.00% | 0.71 |
| Histones | 200 | 270 | 42.55% | 0 | 124 | 0.00% | 42.55% | 100.00% | 0.71 |
| SmD1 | 193 | 277 | 41.06% | 0 | 124 | 0.00% | 41.06% | 100.00% | 0.70 |
| U1snRNP | 221 | 249 | 47.02% | 11 | 113 | 8.87% | 47.02% | 91.13% | 0.69 |
| SSAro60 | 314 | 156 | 66.81% | 99 | 25 | 79.84% | 66.81% | 20.16% | 0.44 |
| SSAro52 | 250 | 220 | 53.19% | 102 | 22 | 82.26% | 53.19% | 17.74% | 0.36 |
| SSBla | 93 | 377 | 19.79% | 45 | 79 | 36.29% | 19.79% | 63.71% | 0.42 |
| Scl70 | 5 | 465 | 1.06% | 1 | 123 | 0.81% | 1.06% | 99.19% | 0.50 |
| Centromere | 21 | 449 | 4.47% | 13 | 109 | 10.48% | 4.47% | 89.52% | 0.47 |
| Jo1 | 3 | 467 | 0.64% | 1 | 123 | 0.81% | 0.64% | 99.19% | 0.50 |

*AUC* area under the curve

investigated the anti-Rib-P0 antibodies by using LIA; the prevalence of anti-Rib-P0 antibodies in SLE patients was 35.7%. Previous studies showed that the prevalence of anti-Rib-P0 in a Chinese SLE cohort and a Caucasian SLE cohort was 33 and 22% by means of enzyme-linked immunosorbent assays (ELISA), respectively [19, 20].

Some limitations should be considered in the current study. First, the cardiac involvements in SLE patients are broad, it contains pericarditis, endocarditis, myocarditis, coronary vasculitis and other cardiac diseases, but in this study, it has pericarditis that had been reported only; any other cardiac involvements (endocarditis, myocarditis, and coronary vasculitis) of SLE patients were not recorded. Second, the measurement for anti-Rib-P0 is by LIA, which is a qualitative but not a quantitative determination method. Therefore, the value of anti-Rib-P0 and its association with clinical characteristics cannot be fully analyzed. Furthermore, this study is a retrospective study, we have no detailed clinical data of patients with positive

**Table 5** The distribution and seroprevalence of anti-nucleosomes, anti-dsDNA, anti-Histones, anti-SmD1, and anti-Rib-P0 antibodies in SLE patients

| Antibody | Positive | *N* | Seroprevalence | AUC |
|---|---|---|---|---|
| Nucleosomes | 192 | 470 | 40.90% | 0.70 |
| dsDNA | 201 | 470 | 42.80% | 0.71 |
| Histones | 200 | 470 | 42.60% | 0.71 |
| SmD1 | 193 | 470 | 41.10% | 0.71 |
| P0 | 168 | 470 | 35.70% | 0.65 |
| Nucleosomes + dsDNA | 159 | 470 | 33.80% | 0.67 |
| Smd1 + Nucleosomes | 123 | 470 | 26.20% | 0.63 |
| P0 + dsDNA | 89 | 470 | 18.90% | 0.60 |
| P0 + Nucleosomes | 84 | 470 | 17.90% | 0.59 |
| P0 + SmD1 | 91 | 470 | 19.40% | 0.60 |
| P0 + Histones | 83 | 470 | 17.70% | 0.59 |
| P0 + dsDNA + Nucleosomes | 67 | 470 | 14.30% | 0.57 |
| P0 + dsDNA + SmD1 | 62 | 470 | 13.20% | 0.57 |
| P0 + dsDNA + Histones | 68 | 470 | 14.50% | 0.57 |
| P0 + dsDNA + Nucleosomes + SmD1 | 53 | 470 | 11.30% | 0.56 |
| P0 + dsDNA + Nucleosomes + Histones | 65 | 470 | 13.80% | 0.57 |
| P0 + dsDNA + Nucleosomes + Histones + SmD1 | 52 | 470 | 11.10% | 0.55 |

*AUC* area under the curve

anti-Rib-P0 only; thus we could not compare the differences of clinical characteristics between having positive anti-Rib-P0 patients and those having negative anti-Rib-P0 patients.

Nevertheless, the present study also has its advantages: the study included a large Chinese population including 470 SLE patients and 124 pSS patients; the evaluation of these antibodies was by a method commonly used in clinical practice with high sensitivity and specificity.

## Conclusion

In conclusion, our results indicate the potential usefulness of anti-Rib-P0 as a specific marker in SLE patients. In addition, anti-Rib-P0 positivity may indicate lower cardiac involvement for SLE patients.

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**Author's contributions** H.-F.P. and Z.-J.L. designed the study; Y.-J.M., P.W., C.J., T.W., and L.-J.C. performed the experiments; Y.-J.M. and P.W. collected and analyzed the data; Y.-J.M. wrote the paper; H.-F.P. reviewed the paper.

**Compliance with ethical standards**

**Disclosures** None.

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